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Stress, Trauma and Personality correlates of Nightmare Frequency and Nightmare Distress

Ph.D Thesis

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September 2006

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Abstract

Four studies were conducted with the following samples a): people with frequent nightmares; b) people with sleep apnoea; c) fire-fighters and d) people with traumatic brain injury. Nightmare frequency was significantly elevated in these groups compared to the normal population. Correlations between nightmare frequency and various measures of psychopathology and individual differences were similar in size to those found in student samples. However, partialling out nightmare distress did not render nightmare frequency psychopathology correlations negligible. Indeed, in the fire-fighters study, only one fire-fighter reported having a problem with nightmares. Nightmare frequency is thus not an artifact of nightmare distress in these samples. Defining nightmares as having to wake up the sleeper made no difference to between subjects correlations with the individual difference and psychopathology variables, and to within subjects correlations with state mood. However, requiring the dream to be very unpleasant as opposed to just moderately unpleasant to be classed as a nightmare did result in increased sizes of correlations. Within subjects correlations of pre-sleep mood with the presence/absence of a nightmare that night were small in comparison to between subjects analyses. Nightmares are thus more likely to be caused by general trait or long-term poor well being than by acute poor well-being. None of the individual difference variables assessed in this study predispose individuals to have nightmares under conditions of high anxiety or high depression. This was despite the individual difference variables in many cases having significant between subjects correlations with nightmare frequency. Ratings of PTSD correlated with nightmare frequency in individuals who had undergone repeated exposure to trauma (emergency service workers), and in individuals who had had one severe trauma (individuals with traumatic head injury). Despite sleep fragmentation there was no indication of cessation of dreaming in the patients with apnoea, but approximately one third of the participants with brain injury had complete cessation of dreaming. These results are discussed in terms of theories of nightmare formation, and of the continuity of waking and sleeping cognition.

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Abstract

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Chapter 1

1

General Introduction

This thesis reports investigations of the aetiology of nightmares, and the correlates of nightmare frequency and of the distress caused by nightmares.

Definition of nightmares

The Diagnostic and Statistical Manual for Mental Disorders defines a nightmare as an 'extremely frightening dream' from which a person wakes up directly. The nightmare is remembered in detail and is said to 'usually involve threats to survival, security or self esteem' (American Psychiatric Association, 2000). Nightmares are thought to occur predominantly in the REM sleep stage in the latter part of the sleep cycle (Fisher et al, 1970; Hartmann, 1984), however, they can occur in NREM sleep (Hefez et al. 1987; van der Kolk et al. 1984). They are said to be accompanied by little if any autonomic arousal (Fischer, Byrne, Edwards and Kahn, 1970). However, night terrors, also referred to as 'pavor nocturnus', 'incubus attacks' or 'stage 4 nightmares' are quite rare and said to occur usually early in the night during slow wave NREM sleep. They are often accompanied by marked increases in autonomic arousal, marked fear, screaming and little or no recollection of a dream (Kales et al. 1980b). Night terrors have been viewed as a 'disorder of arousal' rather than one of sleep (Broughton, 1968).

Prevalence of nightmares.

Prevalence estimates of nightmare frequency in the general population have not been consistent. Hartmann (1984) reported that the average number of nightmares experienced by adults is estimated to be one or two per year. The American Sleep Disorders Association (1990) estimated that 5% of the adult population experience at least one nightmare per month. Klink and Quan (1987) found a prevalence of 8.1% for nightmares; however, Bixler et al (1979) found an estimate of 5.3% when using a similar definition of nightmares. Nielsen and Zadra (2000) estimated that 4-8% of the general population had a 'current problem with nightmares'. For instance, in a twin study, Hublin et al (1999) found that 3% had nightmares weekly, whereas 10% had nightmares monthly. In Spoormaker et al (in press) 2.2% of the general Dutch population suffered 'much' or 'very much' from nightmares and 7% suffered 'a little' from nightmares. It is likely that the inconsistency of these prevalence rates can be attributable to different criterion for defining a nightmare and the time scales used.

Research has also suggested that women tend to report more nightmares than men (Ohayon et al. 1996). DSM-IV describes the ratio as between 2-4: 1 (American Psychiatric association, 1994). It has also been reported that women have a higher dream recall frequency in comparison to men (Schredl and Palmer, 1998) which may lead to a higher recall of nightmares. However, Klink and Quan (1987) found an equal prevalence of nightmares among elderly males and females. This suggests along with other research that nightmare frequency may decrease with age (Salvio et al, 1992).

Inconsistency in how nightmares are defined

Much of the inconsistency in prevalence rates of nightmares is attributable to how various authors have defined and measured a 'nightmare'. The use of

inconsistent definitions of what it is that constitutes a nightmare has lead to problems in interpreting the nightmare literature. Some researchers have argued that a nightmare can be and should be distinguished from other disturbing dreams because it causes the sleeper to awaken. However, others have not included this waking criterion in their definitions. For example, Hartmann (1984) defined a nightmare as a long frightening dream that awakens the sleeper. Many others have utilised similar definitions, which encompass this waking criterion (Coalson, 1995; Feldman & Hersen, 1967; Hersen, 1971; Levin, 1994; Levin & Hurvich, 1995; Miller & DiPilato, 1983). However, many researchers have not used this waking criterion (Belicki, 1992a; Bixler et al., 1979; Blagrove et al, 2004; Chivers & Blagrove, 1999; Kales et al., 1980a; Klink & Quan, 1987; Salvio et al., 1992; Wood & Bootzin, 1990). For instance, Wood et al (1992) defined nightmares as 'frightening dreams with visual content and an elaborate story'. Some researchers provided no definition of a nightmare to subjects (Belicki & Belicki, 1986; Cernovsky, 1983; Dunn & Barrett, 1988; Haynes & Mooney, 1975; Hearne, 1991; Stepansky et al., 1988).

Use of the waking criterion in the definition of a nightmare

The waking criterion is used as it is assumed by many that waking from an unpleasant dream is an indirect measure of nightmare intensity. Although Zadra and Donderi (1993) found that 45% of bad dreams are found to have emotional intensities equal to or exceeding those of the average nightmare. Furthermore, Krakow, Kellner, Pathak & Lambert (1995) found that less that one forth of chronic nightmare sufferers report always awakening from their nightmares. Zadra and Donderi (1993) assessed the use of the waking criterion. They defined nightmares as disturbing dreams that awaken the sleeper and bad dreams as disturbing dreams that do not awaken the sleeper. They demonstrated that nightmare frequency had more significant correlations than bad dream frequency with measures of well-being. This suggests that people who experience bad dreams are low on measures of well being, but not to the extent demonstrated by those with nightmares. Therefore, this study seems to support the use of the waking criterion. They highlight that group studies which define nightmares using the waking criterion would classify bad dream only participants as control participants; hence, this may lead to an increase in the control group's mean scores on measures of psychopathology. However, those studies that do not use a waking criterion place may place bad dream only participants in the experimental nightmare group, thus decreasing the mean psychopathology scores of the experimental group. The authors argue that this may account for some of the inconsistent findings reported on the relationship between nightmare frequency and measures of psychopathology. Schreuder et al (2000) found that direct awakening from a bad dream resulted in higher total scores on the Symptom checklist-90 and higher scores on several posttraumatic complaints. However, subjects in this study had posttraumatic stress disorder and the majority experienced both types of dreams. In contrast, Kellner et al (1992) found no relationship between direct awakenings from a frightening dream and associated distress. Blagrove et al (2004) found that the frequency of unpleasant dreams (a combined measure of disturbing dreams that wake the sleeper and those that do not) is a better index of low well being than nightmares alone (with the waking criterion).

Validity of the waking criterion

Blagrove and Haywood (2006) assessed whether people believe they are able to judge whether the imagery or emotion of a dream caused them to wake up. It is

possible that some individuals may be inaccurate in the judgements they make about their sleep and that this may be a problem when expecting people to judge whether a nightmare woke them up or not. It is suggested that there may be a halo effect when people are deciding whether a nightmare woke them up, with awakening being erroneously attributed to a dream that had particularly unpleasant events or emotions. The authors investigated how certain people were in deciding that a nightmare woke them. In order to reduce the halo effect and attributions participants were asked to make awakening decisions about all dreams (whether pleasant or unpleasant). They found that judgements of being woken by a dream were made with high certainty particularly for very unpleasant dreams. Furthermore, judgements that dreams did not cause awakenings were made with moderate to high certainty. In a sub sample it was found that participants were significantly more certain in judgements that dreams had woken them than in judgements that dreams had not woken them. It was also found that dreams in general that were judged to cause awakening were significantly more unpleasant than dreams judged not to cause awakening. Thus, the authors conclude that if certainty can be used as evidence that people are accurate in attributing awakening to their dreams then it may be valid to include the waking criterion in the definition of a nightmare. Importantly, level of certainty was not related to state or trait anxiety or nightmare distress suggesting that these judgements are valid.

Blagrove and Haywood (2006) also highlight other possible advantages of using the waking criterion. One possibility is that the waking criterion provides a more exact and clear definition of what constitutes a nightmare. They argue that this may be important when using retrospective measures to measure nightmare frequency as these two categories of nightmares differ in the accuracy of retrospective frequency estimations. This could be the result of the difference in ease of making instances of

these two categories of nightmares available to memory (Tversky and Kahneman, 1974). It could also be the case that waking criterion nightmares produce more distress than non-waking nightmares (inclusive nightmares). Separate studies have assessed state distress resulting from waking criterion nightmares (Levin and Fireman, 2002b; Ohayon et al 1997) and resulting from inclusive definition nightmares (Kothe and Pietrowski, 2001; Krakow et al 2002), but no study has directly compared the two. However, trait nightmare distress has been found to correlate more strongly with prospective frequency of waking criterion nightmares than with prospective frequency of inclusive definition nightmares (Blagrove and Haywood, 2006; Blagrove et al. 2004). A further point of interest raised by these authors is whether waking criterion nightmares and inclusive definition nightmares respond differently to treatment. However, there has been no research on treatments in inclusive definition nightmares.

Problems with using the waking criterion

Blagrove et al (2006) caution that a possible confound in using the waking definition of nightmares is that frequent awakenings from sleep have been reported in people suffering from nightmares compared to controls in studies using inpatients (Hersen, 1971), participants, of which some had suffered trauma (Krakow et al. 1995) and insomniacs (Ohayon et al 1997). Furthermore, Germain and Nielsen (2003) found poorer general sleep in people with trauma related nightmares. Thus, while it may be the case that individuals in such clinical populations may be able to correctly judge when they have been awakened from a nightmare, the frequent awakening and nightmares may at times occur simultaneously leading to the erroneous judgement that the nightmare caused the awakening. Blagrove and Haywood (2006) state that

although subjective reports of frequent awakenings are increased in non-clinical populations with nightmares (Levin, 19094; Schredl, 2003) this has not been found polysomnographically for idiopathic nightmares (Germain and Neilsen, 2003).

Clearly it would be productive if all studies made the distinction between waking criterion and inclusive definition nightmares. This would allow for consistency across studies but also enable the exploration of the different correlates of these two types of dreams. Furthermore, Blagrove et al (2006) suggest that data from studies using both these definitions can be combined in order to make comparisons with studies that have not used the waking criterion as a necessity in their definition of a nightmare. It may then be possible to explain the inconsistencies surrounding the prevalence of nightmares in the general population as well as offering insight into the differences between nightmares that wake the sleeper and bad dreams that do not.

The role of emotion in the definition of a nightmare

A further problem with the defining of nightmares is that the majority of researchers have defined nightmares as frightening dreams (e.g. Bixler et al., 1979; Feldman & Hersen, 1967; Hartmann, 1984; Hersen, 1971; Kales et al., 1980a; Levin, 1998; Levin & Hurvich, 1995; Miller & DiPilato, 1983; Salvio et al., 1992; Wood & Bootzin, 1990). However, Zadra and Donderi (1993) found that 17% to 30% of the nightmares reported contained emotions other than fear such as anger and grief. Zadra et al (2004) conducted a content analysis of nightmares and found incidences of 61.1% for fear/ anxiety and 38.4% for other negative emotions (e.g. anger, grief) in nightmares.

Other studies have shown that nightmare sufferers also report many other negative emotions besides fear, such as anxiety (Perlis, Warbasse & Bootzin, 1990).

A similar conclusion was reached by Belicki et al. (1985). This research suggests that the criterion defining a nightmare should be broadened from a 'frightening dream' to include dreams that contain other distressing negative emotions. However, an agreed definition should be reached so that studies can be meaningfully compared.

Inconsistencies due to sampling differences

Another source of variability within the literature is that different studies have focused on different populations. The majority of studies have used undergraduates (Cellucci & Lawrence, 1978; Dunn & Barrett, 1988; Feldman & Hersen, 1967; Haynes & Mooney, 1975; Lester, 1968, 1969; Levin, 1989, 1998; Levin & Hurvich, 1995; Wood & Bootzin, 1990), others have used non clinical adults (Hearne, 1991; Kales et al., 1980a; Miller & DiPilato, 1983) or adults with a lifelong history of nightmares (Berquier & Ashton, 1992; Hartmann & Russ, 1979; Hartmann et al., 1981) or psychiatric inpatients (Hersen, 1971). Hence, it is possible, if not probable that the nature and strength of the relationship between nightmare frequency and measures of psychopathology will vary across some of these populations. Thus, it may be less meaningful to make comparison between studies conducted on very different populations.

The definition of frequent nightmares

Researchers often talk about people who suffer from 'frequent' nightmares, however, there is often a different consensus of what is meant by 'frequent'. Some classify participants into a frequent nightmare group if they report at least one nightmare a week (Dunn & Barrett, 1988; Feldman & Hersen, 1967; Hartmann et al., 1981; Levin, 1998), whereas, others classify participants into a frequent nightmare group if they report at least one nightmare a month (Belicki & Belicki, 1986; Berquier & Ashton, 1992; Hersen, 1971; Kales et al., 1980a; Levin, 1989). Levin and Fireman (2002a) found in their study that participants reporting three or more nightmares (i.e. one or more a week) reported significantly higher levels of psychological disturbance than those reporting two nightmares or less. Hence, they argue that this highlights the validity of a once per week diagnostic criterion rather than once a month, as it is a more meaningful indicator of dysfunctional nightmare behaviour. Therefore, it is not surprising that there any many inconsistencies within the nightmare literature when there is no agreed consensus as to what is meant by the term 'frequent nightmares'.

Retrospective verses prospective measures of nightmare frequency.

A further problem adding to the inconsistency within the nightmare literature is the use of differing methodologies. A major issue here is that until fairly recently the majority of studies only used retrospective self-report in order to assess nightmare frequency. The retrospective method has been shown to produce considerably lower estimates of nightmare frequency compared to retrospective reports. For instance, Wood and Bootzin (1990) used both retrospective measures of nightmare frequency and contemporaneous logs of the incidence of nightmares. Undergraduates were asked to estimate the number of nightmares they had experienced in the past month and the past year and also to record the number of nightmares they experienced over a period of 2 weeks. It was found that the retrospective self-reports produced a lower estimate of nightmare frequency by a factor of 2.5. They found that participants reported a 150% higher mean nightmare frequency using a prorated 2-week log than they reported on a 12-month retrospective estimate and a 91% increase over the prorated mean 1-month estimates. Blagrove et al. (2001) also found that dream logs produced a mean estimate of nightmare frequency (using waking criterion) that was 25% higher than the retrospective questionnaires. Similarly, Salvio et al. (1992) demonstrated that nightmares in the elderly were 10 times more prevalent, when assessed by dream logs, than had previously been estimated using retrospective self-reports.

Zadra and Donderi (2000) confirmed and extended such findings. They argued that there were various methodological shortcomings of Wood and Bootzin's study, firstly that their reporting period was only 2 weeks which was then prorated to 52 weeks for comparison to the 12 month retrospective reports. Hence, small variations in these two weeks would magnify the variability of a prorated 52-week estimate. Furthermore, Wood and Bootzin instructed their participants to record only nightmares and hence, this focus might have influenced participants' reports. Zadra and Donderi, therefore, used a 1-month dream log (in which all remembered dreams were recorded) also retrospective estimates of pleasant dreams (eg. flying dreams) were also measured together with disturbing ones. In this study nightmares were defined as very disturbing dreams that awaken the sleeper, whereas, bad dreams were defined as very disturbing dreams that do not awaken the sleeper. Hence, the authors were able to examine whether retrospective reports and daily logs produced different estimations of nightmares and bad dreams. It was found that the retrospective measures produced markedly lower estimations of both nightmare and bad dream frequency. Their results also suggested that bad dreams were even more prevalent than nightmares. When the 1 month log data were prorated over 52 weeks, it was found to give an annual frequency 162% higher than the mean 12 month retrospective estimate and 92% higher than the prorated mean 1 month retrospective estimates. The prorated annual bad-dream frequency estimated from the logs was 69% and 53% higher than the 12-month and the prorated 1-month estimated respectively. Hence, they found that the frequency of both nightmares and bad dreams were lower when retrospective measures were used; however, retrospectively measured nightmare frequency was lower than for bad dreams. The authors attempted to explain this latter finding by suggesting that fewer nightmares were recalled retrospectively as a result of the stronger negative dream content, as retrospective estimates of flying and lucid dreams were closer to dream log frequency data than were retrospective estimates of nightmares and bad dreams. However, Blagrove et al. (2004) argued that lucid and flying dreams are also rarer than nightmares and, hence, this may be a simpler explanation for the smaller difference between their retrospective and prospective estimates of frequency. However, in order to investigate the motivated forgetting hypothesis suggested by Zadra and Donderi, Blagrove et al. (2004) investigated whether the lower estimate of nightmare frequency obtained retrospectively (which they also found in their study) was related to the mean emotional tone of the participants' dreams. However, they concluded that the underestimation appeared to be due to a simple function of forgetting over time rather than a motivated forgetting explanation. Alternatively, it may be the case that keeping a log increases the frequency of the experiencing or reporting of unpleasant dreams.

A similar concern occurs with retrospective and log assessments of dream recall frequency. Zadra and Donderi (2000) found that a larger number of dreams were reported in the dream logs than were reported in retrospective estimates of dream recall frequency. When asked the number of dreams they recalled a week on the retrospective questionnaire the mean dream recall frequency was 5.82 (SD= 3.06). The mean number of dreams reported in the 4-week logs was 27.13 (SD14.24) or 6.78

dreams per week. Hence, they suggested that either retrospective reports underestimates the total number of dreams or that keeping a dream log increases dream recall, or both. Hence, prospective estimates can not necessarily be considered to produce the 'true frequency' compared to retrospective measures but rather it is more helpful to consider them as two different estimates.

Idiopathic and PTSD nightmares

It is important to distinguish between idiopathic (or ordinary) nightmares and posttraumatic nightmares. Posttraumatic nightmares can occur following exposure to a traumatic event. Nightmares can occur as part of the PTSD syndrome or as part of a posttraumatic stress reaction. Posttraumatic nightmares typically re-enact the traumatic event and occur repetitively. Kilpatrick et al (1998) found that approximately 60% of patients with PTSD experienced nightmares. A similar prevalence rate of 56% was reported by Schreuder et al. (2000) for PTSD nightmares in a sample of war veterans who continued to re experience their combat trauma from more than 40 years previously. However, idiopathic nightmares are those which are long standing and emerge in the absence of an identifiable external trigger. Hartmann (1996) compared posttraumatic and idiopathic nightmares and found that post traumatic nightmares usually occurred earlier in the night, whereas, idiopathic nightmares occurred in the last hours of sleep. Kales (1980a) found that average time of occurrence for idiopathic nightmares was 212 minutes after sleep onset. Hartmann also noted that posttraumatic nightmares were associated with more limb movements than idiopathic nightmares. However, Germain and Nielsen (2003) found that both idiopathic and posttraumatic nightmares were associated with an elevated number of periodic limb movements, in comparison to a healthy control group. This study also found that individuals with posttraumatic nightmares experienced a lower sleep

efficiency (longer and more frequent awakenings). PTSD nightmares will be examined in more detail later in this chapter as they are investigated in the third and fourth studies of this thesis.

Nightmare Distress

Nightmare distress refers to the impact that nightmares have on daily functioning, including fear of going to sleep and of meeting people who have been in one's nightmares. Belicki (1992) found that nightmare frequency and nightmare distress were only moderately correlated (r = 0.26). Thus, it seems that some people can have lots of nightmares and not be bothered by them, whereas others may experience relatively few nightmares but become very distressed by them. Spoormaker et al (2006) claim that nightmare distress seems to be almost like a trait variable with correlations to trait but not state anxiety (Belicki, 1992) and neuroticism (Levin and Fireman, 2002a). The variable has also been correlated with physical complaints (Kothe and Pietrowsky, 2001) and stress related symptoms (Zadra and Donderi 2000). Belicki (1992) found that only nightmare distress, and not nightmare frequency, was associated with heightened psychopathology. Wood and Bootzin (1990) reported a similar association between nightmare frequency and distress. Levin and Fireman (2002a) also found that prospective ratings of nightmare prevalence and trait nightmare distress were not significantly associated (r =0.21). Blagrove et al. (2004) also demonstrated that nightmare distress was a mediating variable for the relationships of nightmare frequency and measures of psychopathology. Furthermore, when nightmare distress was controlled for nightmare frequency (retrospective and prospective measures) was no longer related to any of the psychopathology or

personality variables. However, interestingly when nightmare distress was controlled for, weak but significant relationships between bad dream frequency and anxiety, depression and neuroticism all remained. Therefore, this latter finding supports the proposition that it is useful for investigators to distinguish between nightmares (waking criterion) and bad dreams (inclusive definition). However, this study used the Belicki (1992) Nightmare Distress Scale, which contains a response format based on frequencies and, thus, it has been argued that the format is likely to be confounded by nightmare frequency (Schredl et al. 2003). Therefore, the correlation between nightmare frequency and nightmare distress may be too high when this scale is used. However, a lower correlation was found with nightmare frequency when a nightmare distress scale was used with an intensity scale.

This research suggests that nightmare distress could be a confounding factor in many previous studies and may have stronger relationships with measures of psychopathology than nightmare frequency. However, the concept of nightmare distress is still vague. It remains to be evaluated as to whether nightmare distress is a mental complaint or personality trait. Furthermore, imagery treatments for nightmares work on the principle of reducing the frequency of nightmares, thus, it is unclear how nightmare distress would be affected by treatment. The latter studies reviewed suggest that it would be more effective to treat the distress associated with the nightmares rather than the frequency. However, it can reasonably assumed that imagery therapy and changing the ending of nightmares may decrease distress by empowering and increasing the a person's sense of control over their nightmares.

Individual difference correlates of nightmare frequency and nightmare distress

It has been suggested that nightmares may be indicative of an underlying psychopathology. However, there have been many conflicting results on whether the frequency of nightmares is correlated to waking psychopathology. This is because not only has there been a failure to replicate some findings, but that there has recently been raised the possibility that relationships between nightmare frequency and psychopathology are confounded by trait nightmare distress (Blagrove et al, 2004). The individual difference variables that have been investigated will now be reviewed.

Proneness to psychosis.

There is some evidence to suggest that frequent nightmares may have a specific association with schizophrenic spectrum disorders and in particular schizotypy (Hartmann, 1984; Kales et al. 1980a; Hartmann et al., 1981; Hartmann et al., 1987; Claridge et al., 1997; Levin, 1998; Levin & Raulin, 1991; Levin, 1990; Levin, Galin & Zywiak, 1991; Herz & Melville, 1980; Fennig et al., 1992).

Van der Kolk and Goldberg (1983) stated that most schizophrenics have at least one nightmare per month during periods of remission. Furthermore, it has been demonstrated that pre-psychotic patients often report nightmares (Stone, 1979). Kestenbaum (cited in Hartmann, 1984), in a longitudinal study found that nightmares were a frequent symptom in children that later went on to develop schizophrenia. Hartmann (1984) found that a significantly high proportion of persons who experienced one nightmare per week throughout their lifetime reported that a first or second degree relative suffered from schizophrenia. Such observations lead Hartmann to conclude that the nightmare group were genetically vulnerable to schizophrenia. Hartmann also found that 25% of his nightmare sufferers had at least one relative who had attempted or committed suicide.

Many investigators have proposed that nightmares and schizophrenia may share a common biology (Doty, 1989; West, 1962). Evidence to support this assertion is the observation that parkinsonian patients administered with L-dopa often have a intermittent phase of frequent nightmares, thus, suggesting that nightmares and schizophrenia are bio-chemically if not etiologically related (Hartmann, Skoff, Russ, & Oldfield, 1978).

Hartmann et al (1984) compared nightmare sufferers with two matched controls groups all recruited via newspaper advertisements. There were 12 subjects in each group (6 male and 6 female) all between 20 and 35 years of age. Inclusion in the nightmare group required a lifelong history of nightmares and a frequency of one nightmare per week. The first control group consisted of vivid dreamers and the second of those who had neither nightmares nor vivid dreams. It was found that the nightmare subjects demonstrated elevated scores on the psychosis scales of the MMPI but not on the neuroticism scales. The authors concluded that there was more pathology in the nightmare group, which took the direction of schizophrenia. Out of the 12 nightmare sufferers; two were diagnosed with schizophrenia, three with schizotypal personality and one with borderline personality. Hartmann concluded that his nightmare subjects 'very strikingly did not have the characteristics of neurotics'(p.73) and none could be diagnosed as having anxiety states, phobic states, or obsessive-compulsive disorder. Thus, this study clearly advocates that the types of psychopathologies specifically heightened in chronic nightmare sufferers are the

schizophrenia spectrum disorders and not neurotic disorders. Kales et al (1980a) also reached a similar conclusion. They found that the nightmare group had especially elevated scores on the paranoid and schizophrenia scales; however, they also found that all the clinical scales of the MMPI, including neurotic scale, were elevated.

Hartmann (1984) argues that there may be several reasons for these differences; firstly, Kales et al. (1980a) selected participants who reported 'twelve or more nightmares within the previous year but not necessarily with a lifelong history of nightmares, however, Hartmann et al. included only people who reported at least one nightmare per week and they had to be lifelong nightmare sufferers. Also, in contrast to Hartmann et al (1984), Kales et al. noted that 43% of their group reported a current or past insomnia. Hartmann pointed out that the MMPI profiles of insomniac patients 'almost always have elevated scores on the many MMPI scales, especially the neurotic scales'. Hence, the greater incidence of insomnia in Kales et al's study may have contributed to the increased score on the neurotic scales. A further difference between Kales et al's and Hartmann et al's study, is that in contrast to Hartmann et al's study, 60% of the nightmares sufferers in the Kales et al. study reported the occurrence of a major life event preceding the onset of the nightmare disorder. Thus, Hartmann (1984) argues that Kales et al's sample are more similar to the veterans with post traumatic nightmares reported in the Van der Kolk et al. (1980) study.

A large-scale study by Levin and Raulin (1991) assessed the relationship between frequent nightmares and schizotypal symptomatolgy. They used a sample of 669 undergraduate students (446 females and 223 males) who were administered 4 measures of schizotypy (Chapman, Chapman & Raulin, 1976; Raulin, 1984; Raulin,

Chapman & Chapman, 1978; Chapman, Chapman & Raulin, 1978). A nightmare was defined as 'a scary dream that awakens the dreamer from sleep'. They found that perceptual aberration, intense ambivalence and somatic symptoms were all significantly positively correlated to nightmare frequency, however, the fourth scale physical anhedonia was negatively correlated to nightmare frequency. Hence, these findings support the proposed link in aetiology between frequent nightmares and schizotypal traits and furthermore, point to specific disorders of thought. The authors interpreted their findings in terms of a disturbance of ego boundaries. They suggest that even in a non-clinical population, nightmare sufferers 'may lack the capacity to maintain a consistent, firm boundary around the mental representation of the self and to differentiate separate internal representations of different external objects' (pp 12). When the data were analysed separately according to sex, Levin and Raulin (1991) found that the female's data contributed primarily to the overall significance of the findings. It was suggested that this was because the females were more disturbed by their nightmares than the males and that these effects are readily generalised to their waking experiences. Hence, this is consistent with the assertion that nightmare distress may be a mediating factor in the relationship between nightmare frequency and measures of psychopathology (Belicki & Parry, 1987) and, hence, may be a confound in the relationship between schizotypy and nightmare frequency. In fact, research has suggested that there is a significant relationship between schizotypy and nightmare distress. Claridge et al. (1997) gave a non-clinical adult sample of 204 (117 females and 87 males) four measures of psychosis proneness, these were; the Schizotypal Personality (STA) and the Borderline Personality (STB) (STO; Claridge and Broks, 1984) and the Physical Anhedonia (PhA) and Social Anhedonia (SoA) (Chapman et al. 1976). They found that the STA was the only psychosis proneness

measure positively related to nightmare distress and it was the biggest single predictor. They found this was still the case even after controlling for neuroticism which otherwise had a strong influence throughout the data (this will be discussed in more detail later). Claridge et al. also found that overall women reported more nightmare distress, but in addition they found that irrespective of gender, nightmare distress increased as a function of self-evaluated feminine traits, as measured on the BEM Sex Role Inventory (BEM, 1973). Hence, this finding may offer some insight as to why Raulin and Levin found that females contributed disproportionately to the overall significance of the relationship between schizotypy and nightmare frequency. However, Claridge et al. (1997) did not include any measure of nightmare frequency. Hence, these studies appear to support the notion that nightmares are associated with the schizophrenic spectrum disorders, although, it is less clear to what extent these associations are due to increased nightmare frequency or to the emotional distress caused by the nightmares.

Schizotypy

Levin and Fireman (2002a) examined the role of nightmare frequency, state nightmare distress and various measures of psychopathology including schizotypy. 116 undergraduates (31 males and 85 females) completed a 21 day dream log. A nightmare was defined as a 'scary dream that awakens the dreamer from sleep'. The authors hypothesised that the relationship between nightmare frequency and psychological disturbance would be largely attributable to the level of state nightmare distress with one exception: schizotypy. They argued that for individuals scoring highly on this measure, that nightmares would occur more frequently and be experienced as more 'egosyntonic' and, hence, be experienced with less distress. Therefore, they hypothesised that for schizotypy nightmare frequency would contribute unique predictive variance, irrespective of the level of subjective distress. They found that individuals reporting nightmares on a weekly basis reported more psychiatric disturbances on most scales of the SCL-90-R as well as greater levels of dissociation and schizotypy than individuals reporting 2 or less nightmares over the 21 day logs. Both nightmare frequency and state nightmare distress were significantly related to most measures of psychological disturbance, it was found that these relationships were often stronger for the distress dimension. With the exception of the SCL-90-R phobia subscales, the state distress dimension was related to all measures of psychopathology, whereas, nightmare prevalence was not related to either measure of depression (SCL-90-R or BDI) nor to state or trait anxiety or pathological dissociation. Furthermore, when the shared variance was removed state nightmare distress was found to consistently account for more unique variance to the prediction of higher psychological disturbance than nightmare frequency. Both state nightmare distress and frequency were significantly associated with schizotypy (as measured by perceptual aberration, Magical Ideation and PER-MAG) although the correlations were stronger with nightmare distress. Nightmare frequency was not significantly related to the psychoticism scale on the SCL-90-R, however, the distress dimension was. The authors reported that nightmare frequency was not uniquely predictive of schizotypy and failed to contribute any significant variance to the Psychoticism scale of the SCL-90-R. Hence, the authors conclude that there is no evidence to suggest a specific relationship between nightmare frequency and psychosis proneness.

Berquier and Ashton (1992) also concluded that frequent nightmares were reflective of a 'global maladjustment rather than specific psychotic symptomology' (pp249). They used a sample of 30 non-clinical adults who experienced one nightmare or more per month since childhood. The mean age was 34.7 yrs (ranging from 19-63). They were compared with 30 low nightmare frequency control subjects with a mean age of 35.9 years. Selection criteria for the control group were the presence of less than two nightmares per year on average. Participants completed a dream dairy for 1 month. Subjects also completed the Minnesota Multiphasic Personality Inventory (Hathoway and McKinley, 1940) and the Eysenck Personality Questionnaire (Eysenck and Eysenck, 1975) and a drug use questionnaire. The findings demonstrated that the nightmare subjects scored significantly higher on the EPQ Neuroticism scale and on 8 of the MMPI clinical scales than did the control group. However, the nightmare sufferers were not shown to be significantly higher on the EPQ Psychoticism scale.

Hence, there seems to be some inconsistency in the findings from different studies. Hartmann et al's (1984) data reveal some important gender differences, which may in part explain these inconsistencies. The MMPI profiles demonstrate that male nightmare sufferers tended to have higher scores than the females on the psychotic scales (paranoia, psychasthenia and schizophrenia). The Rorschach tests also suggested more thought disorders and psychotic thinking among the males. Hence, Hartmann, concluded that the 'males could be considered slightly more disturbed on average than the females' (p.99). It may be if the sample is predominantly female then averaging may reduce the mean scores on these psychotic scales given the indication of a 'global psychopathology' (Berquier and Ashton, 1992).

Furthermore, Hartmann et al (1984) and Kales et al (1980a) relied on retrospective estimates of nightmare frequency, whereas, Berquier and Ashton used daily logs to measure nightmare frequency, and as mentioned previously nightmare frequency tends to be underestimated on retrospective reports.

Creativity and Fantasy Proneness

Evidence suggests that creativity is a heterogeneous concept compromised of a multiplicity of factors (Belcher, Rubovits and DiMeo, 1981; Richards, 1981) which has caused considerable debate in the literature regarding its definition and measurement (Treffinger, Renzulli and Feldhusen, 1971). The problem is made more difficult still by the need to distinguish the creative process from the creative product and convergent from divergent creativity. Levin, Gallin and Zywiak (1991) concluded that there does not appear to be overwhelming evidence that subjects who self-report frequent nightmares are significantly more creative than those that do not.

Hartmann et al (1981) found that lifelong nightmare sufferers have a potential for artistic achievement. Using a student sample and a self-rating scale of creativity Chivers & Blagrove (1999) did not find this. Levin and Fireman (2001-2002) found that nightmare frequency and nightmare distress were associated with higher levels of fantasy proneness, psychological absorption, and a guilty-dysphoric daydreaming style. These results were not due to higher levels of overall dream recall.

Thin and thick intrapsychic boundaries

Hartmann (1989) noted that frequent nightmare sufferers have striking personality characteristics that could be called "thin boundaries" in several senses. To measure thin and thick boundaries, a 145-item questionnaire, the Boundary Questionnaire, was developed. Nightmare sufferers and art students usually scored thin, while a group of naval officers usually had thick boundaries. Schredl et al (1999) found that persons with thin boundaries report dreams that are more negative (r=.31) and emotionally intense (r=.27). For adolescents Cowen & Levin (1995) found that boundary thinness was related to dream recall (r=.16), nightmare recall (r=.16) and distress due to nightmares.

Cognitive arousal

Hicks et al (2002) finds that cognitive arousability, as measured by the selfreport Arousal Predisposition Scale, is not related to dream recall frequency, but is positively related to dream unpleasantness and to frequency of night-terrors and fantastic or posttraumatic nightmares.

Neuroticism

Chivers and Blagrove (1999) reported no significant relationship between nightmare frequency and neuroticism. However, Zadra and Donderi (2000) found that both retrospective and prospective measures of nightmare frequency were correlated to neuroticism. Lang and O'Connor (1984) found significant correlations between the EPQ-N and both nightmare frequency and nightmare intensity. Furthermore, Berquier and Ashton (1992) also found neuroticism was significantly higher in the nightmare group compared to controls. Blagrove et al. (2001) also found that neuroticism (EPQ-N) was significantly correlated with nightmare frequency as measured both retrospectively and prospectively. However, such correlations become smaller when nightmare distress was partialled out.

Claridge et al. (1996) noted that neuroticism was highly correlated to several measures of proneness to psychosis and furthermore, they pointed out that when included alongside measures of psychosis proneness in factor analysis, N helped to define at least one of the major components of schizotypy. Thus, they stressed the importance of detangling these correlated influences of psychosis proneness and neuroticism in order to establish whether the relation between neuroticism and nightmares is significant in its own right or whether its effect is secondary to that of more primary psychotic traits. Thus, they looked at the extent to which any of the relations found with nightmare distress were due to general neuroticism rather than specifically the influence of schizotypy. They demonstrated, however, that the STA (Claridge and Broks, 1984) a measure of schizotypal personality was the only psychosis proneness measure that was positively related to nightmare distress and was its single biggest predictor, and this was the case even after controlling for N. Thus, they conclude that neuroticism may be considered as a 'very broad dimension that correlates with, but subsumes, a range of more specific, clinically relevant personality features'.

Borderline Personality

Claridge et al (1997) demonstrated a significant relationship between the distress engendered by nightmares and schizotypal personality traits (as reviewed above), however, in unpublished data from this study they also revealed a smaller but significantly positive correlation with the associated dimension of borderline personality (r = 0.39, N = 179; p <0.001). Claridge, Davies, Bellhouse and Kaptein (1998) aimed to establish the existence of a three-way relationship between reported adverse life events, borderline personality traits and susceptibility to nightmares.

They assessed 60 undergraduate females (mean age 20.4 years, range 18-30). Participants completed the nightmare distress scale (Belicki, 1992), a scale of borderline personality traits (STB; Claridge and Broks, 1984) and the Child abuse and Trauma (CAT) scale (which includes subscales covering three areas of life events: sexual abuse, punishment, and neglect/negative home environment). They found that these relationships were all positive and mostly very significant, with the exception of the punishment scale of the CAT, which generally wasn't related to the other variables. Stepwise regression analysis revealed that nightmare distress was predicted by a combination of borderline personality and the sexual abuse and neglect trauma scales. In a large sample of 203 women, a regression analysis revealed that borderline personality (again measured by the STB) continued to be a strong predictor of nightmare distress but this time only the abuse trauma scale remained in the equation $(R^2 \text{ was } 0.27)$. Thus, these authors conclude that borderline personality traits were strongly associated with the tendency to report early sexual and neglectful abuse, as well as distressing nightmares (the last two domains also being significantly correlated). However, these authors did not measure the frequency of nightmares so it is not clear if this variable is related to borderline personality. It was hypothesised in the current study that borderline personality would be significantly related to both distress and frequency dimensions.

- <u>Depression</u>

Having nightmares on a weekly basis was strongly associated with depressed mood (Miro and Martinez 2005), and an association between nightmare frequency and non-clinical depression is seen in Blagrove et al (2004) and Zadra and Donderi (2000). However, for clinical depression the situation becomes complicated, as Armitage et al (1995) shows that dream recall rates were extremely low in depressed patients (<20%) and decreased further when antidepressants were used. Also, most dreams were short and bland.

Nightmares as a predictor of suicide

Agargun and Kara (1998) reported in a case-control study that patients with major depression who experience at least two nightmares a week had higher suicide scores than depressed patients who never report having nightmares. There is also evidence to suggest an association between REM sleep abnormalities and suicidal behaviour in patients with major depression (Sabo et al. 1991) and in patients with psychosis (Keshavan et al. 1994).

Tanskanen et al (2001) examined the risk between the frequency of nightmares and the risk of suicide in a large, prospective population based study (N=36,211). The study began in 1972 and subjects were followed until 1995 or death. Information or deaths caused by suicide or other self-inflicted injury was obtained from the National Death Register. They found that the frequency of nightmare was directly related to the risk of suicide. They reported that for subjects reporting nightmares occasionally the adjusted relative risk of suicide was 57% higher than those without nightmares and that subjects reporting frequent nightmares at all. They also found that the frequency of reporting a nightmare was directly associated with more frequent symptoms of mental health problems such as insomnia, depression, life stress and anxiety. However, one limitation of this study is that the frequency of nightmares was investigated using only one question. This study clearly has important clinical implications as frequent nightmares may alert clinicians that a patient may be at risk of suicide (obviously the presence of frequent nightmares will be considered in the wider clinical profile of the patient along with other risk factors). Also the authors found that the prevalence of insomnia in those reporting frequent nightmares was significantly greater than those not reporting nightmares.

Nightmares, sleep disturbances and suicidal ideation

The relationship between depression and sleep problems is well established. For example, Breslau et al (1996) found that even after controlling for a history of depressive symptoms, previous insomnia is a significant predictor of subsequent depressive episodes. Perlis et al (1997) have demonstrated that patients who suffer from recurrent depressive symptoms have increased insomnia prior to the recurrence of Major Depressive Disorder. However, there has been little research assessing the impact of sleep problems on suicidal ideation. Insomnia, hypersomnia and subjective sleep quality have also been reported to be related to suicidal behaviour in patients with major depression (Agargun, Kara and Solmaz 1997; Agargun, Kara and Solmaz 1997) and panic disorder (Agargun and Kara, 1998). Research has demonstrated differences in sleep between depressed patients who are suicidal compared to those who are not. Depressed patients with suicidal ideation often display increased REM activity and longer waking periods when compared to depressed patients without suicidal ideation (Keshaven et al 1994; Yu et al. 2003).

Cukrowicz, Krakow and Joiner (2006) assessed nightmare frequency, insomnia, suicidality and depression in 222 university students. They found that insomnia and nightmares were significant predictors of depression, however, only nightmares significantly predicted suicidal ideation. This indicates that disruptions in sleep associated with disturbing dreams and nightmares play an important role in the development and maintenance of suicidal ideation. This was even found to be the case after controlling for the effects of depression on suicidality. This suggests that distressing cognitions and waking experiences may be re-experienced during sleep possibly leading to lower mood the following day and increased preoccupation with the nightmare. Also disturbing dreams may play a role in depression by reducing sleep quality. However, one limitation of these studies assessing suicidal behaviour is that they did not control for PTSD, although PTSD has been associated with suicidal behaviour and suicide attempts (Davidson, Hughes and Blazer, 1991). Furthermore, the role of nightmare distress has not be examined.

Anxiety

The empirical data regarding the relationship between nightmare frequency and anxiety is also inconsistent. Hersen (1971) found a significant relationship between nightmare frequency and anxiety together with heightened concerns about death in a clinical sample of 160 male inpatients. However, their diagnoses included primarily psychotic disorders.

Non-clinical populations with frequent nightmares were correlated with the presence of neurotic symptoms such as guilt and anxiety. Nightmare frequency has been correlated with anxiety-distraction, guilt-dysphoria and guilt-fear of failure in day dream patterns (Starker, 1974, 1984; Starker and Hasenfeld, 1976). Berquier and Ashton (1992) found that people who report frequent nightmares have higher levels of anxiety than people with few nightmares. In two different samples of college students (N=210 and 76 respectively) Haynes and Mooney (1975) using the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953) found low but statistically significant correlations ($\mathbf{r} = 0.17$ and 0.23) with a measure of retrospective nightmare frequency.

Nielsen et al (2000) assessed the association between anxiety and disturbing dreams in a sample of adolescents. 610 boys and girls rated their recall of both disturbing and normal dreams at both 13 and 16 years of age. The authors found elevated anxiety levels for 13 and 16 year olds who reported frequent disturbing dreams. Furthermore, at the age of 16 they demonstrated that the recall of disturbing dreams was associated with clinically symptomatic anxiety indicators taken from DSM-IIIR. Schredl et al. (1996) reported a significant relationship between the level of generalised trait anxiety and the frequency of bad dreams in 10-16 year olds.

Mindell and Barrett (2002) examined the relationship between nightmares and trait anxiety in elementary aged children. Children and their parents completed questionnaires assessing nightmare occurrence and anxiety. From both the parental reports and the children's reports, it was found that the children whose nightmares were rated as most frightening had significantly higher levels of trait anxiety than those whose nightmares were rated as less frightening. However, the relationship between nightmare prevalence and anxiety differed depending on whether the reports were made by the children themselves or by their parents. Based on the children's reports there was no significant difference between the anxiety scores of those who reported experiencing nightmares and those who did not experience nightmares. However, based on parental reports there was a relationship between nightmare prevalence and anxiety scores. The authors claim that the limited relationship especially for the children may have been affected by a ceiling effect as there was a 75% nightmare prevalence estimate based on the children's reports, however, this decreased to 49% when rated by the children's parents. Thus, the authors argue that given the high base rate of nightmare prevalence a relation with anxiety was difficult to obtain.

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Anxiety and nightmares: negative findings.

Some studies have not found a relationship between anxiety and nightmares. Wood and Bootzin (1990) found no relationship between nightmare frequency and self reported anxiety as measured by the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953). Using a retrospective measure of nightmare frequency (12 month reports) the correlation with anxiety was 0.13, when prospective logs were used this decreased to 0.04. They concluded that 'a genuine, though very low, association may exist between anxiety and nightmare frequency when retrospective reports are used'. Wood and Bootzin suggested that anxious individuals may not actually have more nightmares but they may be more likely to remember them and report them retrospectively. This assertion is supported by Clark and Teasdale (1982), who suggest that anxious individuals might have greater access to memories of anxietyrelated events, such as nightmares. In addition Wood and Bootzin also found that nightmare distress was not significantly correlated with scores on the TMAS.

Belicki (1992) also found no relationship between anxiety and nightmares. Fisher and Wilson (1987) assessed a sample of children in grades 1-12 and found a positive relationship between frightening TV content and overexcitement but not . anxiety.

Zadra et al (1995) investigated the relationship between trait anxiety and both nightmare frequency and bad dream frequency in an attempt to offer some clarity to how different definitions may affect the correlations reported. This study used a sample of 46 women (mean age 36.9 yrs) who completed a daily log of all their dreams for 14 consecutive days. Over this period 46 nightmares were reported and 113 bad dreams. The authors reported that there was no significant correlation between levels of self- reported anxiety and nightmare frequency (r = 0.23) only a modest correlation was found between trait anxiety and bad dream frequency (r = 0.31, p<0.05). The authors concluded that the relation between trait anxiety, nightmares and bad dreams is at best weak. However, they found that trait anxiety correlated negatively with the ratio of positive to negative dream affect, this suggests that the emotional tone of dreams is related to low well being. Hence, in this study it seems that emotional tone of the dream may be a better predictor of low well being than nightmare frequency.

State anxiety

Levin (1998) found that his subjects, who reported at least one nightmare per week, had significantly greater trait anxiety than controls, although only trends were found for depression and state anxiety. However, other authors have found that state anxiety was related to heightened mood (Cook, Caplan and Wolowitz, 1990; Stepansky et al. 1998; Zadra and Donderi, 2000; Zadra et al 2000; Levin and Fireman, 2000). Thus, current findings on the relationship between anxiety and nightmares are inconsistent. The heterogeneity may be explained by the findings of Kothe and Pietrowsky (2001). They assessed a sample of 41 non clinical adults who reported having the 'occasional nightmare'. Anxiety was measured by the 'State-Trait-Anxiety-Inventory' (STAI; Laux et al.,1981). They found that STAI scores after a night with a nightmare were significantly higher compared to those after a night without a nightmare. This suggests that state anxiety may be a mediating factor in the relationship between trait anxiety and nightmare frequency.

It has been suggested by various authors that the reasons for these inconsistencies concerning psychopathology and nightmare frequency may be a result of the different methodologies used. Blagrove and Haywood (2006) suggested that there is no systematic difference between correlations of anxiety with frequencies of waking criterion nightmares and non-waking criterion nightmares. They present the following table of all between subject's correlational studies involving nightmare frequency and anxiety.

Comparison of Pearson and Spearman correlations of nightmare frequency and anxiety, categorised according to whether study used waking criterion, and according to retrospective or log measure of nightmare frequency. Original table reproduced from Blagrove et al (2006).

Table I Pearson and Spearman correlation coefficients between nightmane frequency and trait	it and state anniety	and trait and state analiety	1. Clark
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Austan	Per traspective for a specific	Cangory of nightnane or had drasm	Prot i mextery	State and to by
Biagrave et al., 2004	R	W	0.16	
Zadra and Donders, 2000	R	w	0.27 and 0.37*	0.28 and 0.35*
(Starker and Hasenfeld, 1976)	R	w	(0.25)*	
Cook er al., 1990	8.	1	0.33	
Haynes and Mooney, 1975	R	t	0.17 and 0.23*	
Krakow er al., 2002	R	1	0.29 and 0.28 ⁴	
Miró and Marinez, 2005	R	I	0.23 and 0.06°	
Nguyen et al., 2002	R	1	0.26 and 0.19	
Schredter al., 1996	R	T	0_31	
Wood and Bootzin, 1990	R	1	0.13	
Zadra and Donden, 2000	R	BD	0.16 and 0.09	0.09 and 0.09*
Bingrove et al., 2004	P = 2 weeks	w	0.12	
Levin and Fireman, 2002a	P = 3 weeks	w	0.23	0.20
Zadra and Donders, 2000	F » ដោលដោ	W	0.32	0.27
Zadra er al., 1995	P ~ 2 weeks	W	0.23	
Blagrove et al., 2004	P = 2 weeks	1	0.15	
Krakow et al., 2002	P = 2 weeks	I	0.13 and 0.17 ⁴	
Wood and Bootzin, 1990	P = 2 weeks	I	0.04	
Wood et al. 1992	P = 3 weeks	T		0.29, 0.38 and 0.234
Wood et al., 1992	P = 3 weeks	I		AS 3 correlations non-significant ^b
Zadra and Donderi, 2000	P∞ imon¥u	BD	0.09	0.03
Zadra et al., 1995	P == 2 weeks	BD	0.31	

Studies are categorized as retrospective (R) or prospective (P); nightmanes categorized as using the waking criterion (W) or as inclusive definition nightmanes (I). Studies of frequency of had dreams, where, by definition it was not the dream that caused the person to wake, are labelled BD. Studies using inclusive definition nightmanes thus combine waking criterion nightmanes and had dreams.

*First coefficient is for nightmare frequency assessed for 1 year revespectively and second coefficient is for nightmare frequency assessed for 1 month retrospectively.

^b Measure used for correlation was anxious distractivite daydreaming style. As this is not a standard trait anxiety measure citation and coefficients are placed in parentheses.

"Coefficients are from study I and study II.

⁴ First coefficient refers to mights per week that a nightmare occurred and second coefficient refers to number of nightmares per week.

"First coefficient refers to frequency of nightmares for participants who have nightmares on a weekly basis, i.e. 1-7 times per week and second coefficient refers to frequency of nightmares for participants who have nightmares on a monthly basis, i.e. 1-3 times per month.

First coefficient is for global anticity and second coefficient is for anticity related to testing.

"Coefficients reported for three groups; first two correlations are for individuals who were in the proximity of the 1989 San Francisco earthquake and third correlation is for a non-earthquake control group. Anxiety assessed for time of occurrence of earthquake

*Coefficients not reported due to being non significant for each of the three groups usted Anniety assessed for the 24 h before the log started

Furthermore, inspection of this table suggests that there is no systematic difference between correlations with anxiety and frequency of nightmares whether measured retrospectively or prospectively. The average correlation for all the above studies between anxiety and nightmares measured retrospectively (excluding the Starker et al 1976 study) is r = 0.22 and the average correlation for the studies measuring nightmare frequency using logs is r = 0.23. However, these average correlations include studies using different criteria for nightmares (i.e. waking and non waking and all inclusive definitions). The average correlation with anxiety for the two studies using retrospective measures and waking criterion nightmares measured retrospectively is 0.26 and for the four studies using logs and the waking definition of nightmares is 0.21. Although the correlation is slightly higher for retrospective measures the difference is small and both average correlations are weak. The average correlation for the seven studies using an inclusive definition of nightmare and a retrospective measure of nightmare frequency is 0.22, whereas, when the inclusive definition is used with logs the average correlation with anxiety in the above 5 studies is 0.27. This suggests that the correlation with anxiety is slightly higher when logs are used, however, the correlations again are small. Thus, inconsistencies in the literature concerning the relationship with anxiety may be due to the different measures of anxiety utilised as well as the different samples used (e.g. the number of chronic nightmare sufferers). However, in general the literature suggests that the relationship between trait anxiety and nightmare frequency is at best weak. However, evidence suggests that this relationship is mediated by state factors (Koth and Pietrowsky, 2001).

Within subjects analysis with anxiety

Cellucci and Lawrence (1978) examined the relationship between daily fluctuations in anxiety and the occurrence of nightmares over an 8 week period among 29 undergraduates. They found no simple relationship between anxiety and the occurrence of nightmares. However, within subjects correlations revealed that a relationship does exist between these variables for a small subset of the sample. However, Cellucci and Lawrence did not use frequent nightmare sufferers in their study and it is possible that with such a population stronger within subjects correlations would exist.

Thus, study one aims to assess within subjects correlations of anxiety and depression in a population of frequent nightmare sufferers and to compare the magnitude of these correlations with between subjects correlations. It may be the case that only weak relationships are found with trait and state anxiety and nightmare frequency using between subjects correlations, whereas, within subjects correlations may reveal a strong association with anxiety and indeed other measures of general psychopathology for a subgroup of individuals. It may be that state anxiety and depression are correlated within subjects with unpleasant dreams only in those individuals with heightened psychopathology. Thus, in this way, psychopathology may modulate the relationship between state factors and the occurrence of nightmares.

A possible confound here, however, is that dream recall, and not just nightmare recall, may be increased under conditions of state anxiety. Cohen (1974a) found a small within-subjects effect of waking mood on dream emotional quality. He suggests that individuals with negative waking mood can mobilize defensive capacities during sleep. Dream recall was more likely following negative presleep mood, with this effect being greater for infrequent dream recallers. He suggests that

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this effect is mediated by dream salience, which is increased by negative presleep mood. Within subjects correlations for the presence or absence of a dream each night, therefore, will have to conducted to investigate this confound.

Effects of trait and state factors on nightmare frequency

Schredl (2003) proposes that the inconsistencies in the literature may be due to the fact that previous studies have not taken into account the intercorrelations between state and trait measures and that the magnitude of the correlation coefficients to nightmare frequency have not been directly compared.

Schredl argues that on the basis of the continuity hypothesis that one would expect state factors to play a more important role in explaining the variability in nightmare frequency than trait factors. Schredl (2003) tested the hypothesis that state factors explain the relationship between trait factors and nightmare frequency. Retrospective and prospective measures of nightmare frequency were used (the intercorrelation between these scales was r = 0.662; p<0.0001). The German version of the NEO-PI-R (Ostendorf and Angleitner, 1994) was used, measuring the Big Five personality measures (neuroticism, extraversion, openness to experience, agreeableness and conscientiousness). Three stress measures were used; the ATE-36 (Schmidt-Atzert, 1989), which assessed the occurrence of 19 negative events, which are part of daily hassles, the EBF-72/3 (Kallus, 1995) which comprises of 42 seven point items measuring the stress present in a number of areas such as social interactions, emotional stress etc and the SCL-90 R (Derogaties, 1986) with a retrospective interval of 7 days. A regression analysis found that 10% of the variance in nightmare frequency was explained by gender and neuroticism, a further regression showed that 6% of the variance was accounted for by boundary thinness, openness to experience and absorption (as well as gender). Gender and the three stress variables accounted for 17.5% of the total variance in a further regression and it was found that including the trait measures did not increase this variance. Thus, the proportions of the variance explained by the stress measures were considerably higher than that explained by the trait measures. This finding supports the hypothesis that the effects of trait factors are mediated by state factors. In this way persons who scores high on neuroticism experience stress more frequency and hence, have more nightmares. This study provides support for the continuity hypothesis of dreaming in that nightmares reflect negative waking experiences.

Summary of individual difference variables

The majority of the research reviewed above assessing the relationship between nightmares and psychopathology has used samples of students, healthy adults or people reporting frequent nightmares. These studies (reviewed below) have found only small to moderately sized relationships between nightmares and anxiety (Zadra and Donderi, 2000; Nielsen et al, 2000; Haynes and Mooney, 1975; Levin, 1998; Tanskanen et 2001) depression (Levin, 1998; Tanskanen et 2001) and symptoms of psychosis (Kales et al, 1980a; Levin, 1998). Other studies have found no relationship between psychological complaints and nightmares or bad dreams (Wood and Bootzin, 1990; Belicki, 1992; Spoormaker and van den Bout, 2005). Between subjects correlations in studies that have identified positive relationships between nightmares and psychopathology have generally been around about 0.3 suggesting relatively weak relationships. Furthermore, Cellucci and Lawrence (1978) found significant within subjects correlations with nightmare occurrence and anxiety in their longitudinal study. However, there have been no other studies that have addressed within subjects correlations of nightmares or bad dreams with any other measure of well-being.

Stress, dreaming and nightmares

Experimentally induced stress and dreaming

In laboratory experiments typically subjects are divided into two groups. One group are put in a high stress situation (e.g. given a sham intelligence test which is very difficult and can not be completed in the time limit) and the remaining participants are assigned to a low-stress condition (e.g. given an easy test to do). De Koninck and Koulack (1975) and Koulack et al (1985) have demonstrated that participants in the high stress condition reported an increased amount of incorporation of the stressor compared to participants in the low stress condition. Goodenough et al (1974, 1975) found an increase in dream anxiety and an alteration of dream content following stressful presleep films. These studies suggest that waking life stressors can affect dream content. However, Koulack (1991) and Schredl (2003) state that real life stressor are more likely to be incorporated into dream than experimentally induced stressors and, furthermore, individual differences are evident when determining what is stressful and what is not.

Waking life stress and dreaming

Berger, Hunter and Lane (1971) awakened hospital patients during REM sleep and collected dream reports for several nights while they were awaiting surgery as well as during the recovery period. They found that a large percentage of their dream content was related to the awaited surgery. Furthermore, the postoperative dream report showed a marked decrease in surgery-related content. Cartwright et al (1998) found that patient's level of depression was correlated with the subjective experience of negative emotion in early night REM sleep periods. Negative toned films presented prior to sleep also correlates with subject experience of negative toned dreams.

Cartwright (1991) studied volunteers who were going through a divorce at the time of their break up and again a year later. 31 of these were diagnosed with depression at the initial testing period and 18 were not depressed. She found that those who were depressed dreamt of their ex-partner more frequently and with stronger affect than those who were not depressed. Of the participants that were initially depressed she found that on follow up those who had incorporated the ex spouse into their dreams a year previously had significantly lower depression scores and were judged as doing well in getting over the divorce. Furthermore, those who showed recovery at follow up were found to have more highly developed dreams and fewer reports without recall than those who remained depressed as well as a shift towards more positive affect in dreams that directly incorporated divorce related elements. Cartwright (1991) concludes that there are clear differences in dream content and affect between those who do and those that do not get over their depression. It suggests that those who suffer depression in response to a stressful life event and whose dreams contain strong affect and incorporations of the stressor appear to remit more successfully from their depression. This study suggests that a direct incorporation of a stressor into one's dream may serve as a mechanism for coping with that stressor.

In general these data suggest that events, particularly those of a usual or stressful nature do affect the content of dreams. However, the influence of presleep stimuli can take a number of forms from direct incorporation of a stimulus, changes in dream activity or types of interactions or changes in the emotionality of dreams.

Stress and nightmares

The previous research suggests that negative life events can affect dream content and that waking stress will be associated with an increased frequency of nightmares. Schredl's (2003) study, described above, has demonstrated that waking stress is an important factor in explaining nightmare frequency. Chivers and Blagrove (1999) found that that nightmare frequency was significantly correlated with stress as measured by the General Health Questionnaire (which examines current life events rather than stable traits). Koulack and Nesca (1992) found that type A individuals have more nightmares than type B individuals, although there was no difference in dream recall frequency between the two groups. Type A behaviour patterns are seen as an indicator for experiencing high amounts of stress and are more likely to experience stress-related health problems such as coronary disease, compared to type B individuals (Glass, 1977). Hartmann (1984) reported periods of stress were a precipitating factor for increased nightmares in his nightmare sufferers.

These included many different stressors such as adolescence, relationship break ups, geographical moves etc. Cernovsky (1984) found correlations between life events and sleep disturbances especially the occurrence of nightmares. Krakow et al. (1993) reported that 12 out of 20 nightmare sufferers had reported a traumatic incident or stressful period prior to the nightmare. These included job loss, pregnancy, death of a loved one, divorce, physical abuse, burglary, rape, starting college or a new job. In line with this research Kales et al. (1980a) showed that 60% of their sample reported the occurrence of a major life event preceded the onset of their nightmares.

Köthe & Pietrowsky's (2001) reported that 31.6% of the nightmares were considered to be associated with a previous stressful occurrence and 75.5% of the nightmares were thought to reflect something within the participants' life. However, using the 'Social Readjustment Rating Scale' (SRRS; Holmes & Rahe, 1967) Kothe & Pietrowsky found no relationship between life events and frequency of nightmares. Such a negative finding was also reported by Belicki (1987). Belicki (1992a) and Cernovsky (1984b) also found no relationship between nightmare frequency and recent life stress. Picchioni et al (2002) examined the relationship between stress and nightmares as well as assessing the possibility that nightmares can serve a beneficial function in this relationship. 412 psychology students were separated into low, medium and high nightmare frequency groups as well as a low, medium and high nightmare intensity groups. Nightmare intensity was measured using the Nightmare Distress Questionnaire (Belicki, 1992). Comparisons were then conducted for daily stressors, life stressors, social support and coping. The authors found a significant relationship between nightmares (intensity and frequency) and daily stressors (using the Hassles and Uplifts Scale; Delongis et al. 1988). This suggests that even daily hassles can be associated with nightmares (as well as the more severe trauma reported, for example, by Wood and Bootzin, 1992). Furthermore, they found that the relationship between life stressors and nightmares (frequency and intensity) was also significant (using the college student version of the Social Readjustment Rating Scale; Holmes and Rahe, 1967). Therefore, this suggests that nightmares are associated with stress regardless of its severity. Interestingly, these authors also found that high levels of social support were associated with low levels of nightmare frequency and nightmare intensity. This is meaningful in light of the well-known finding that social support buffers the effects of stress, and thus it would be logical to expect that high

levels of social support would attenuate the relationship between stress and nightmares. However, their findings also suggest that this relationship between social support and stress could be mediated by nightmare distress as a high amount of social support could have produced a reduction in nightmare intensity.

Sexual trauma and nightmare frequency

In the life events domain, accessing information about some forms of personal trauma by explicit enquiry is problematic and there is growing interest in the complementary use of more indirect method; among these is the examination of individual sleep disturbances. This methodology is based on the considerable evidence that nightmares represent one way in which traumatised persons find an outlet for their distress. Although this method have considerable interpretative difficulties (Renneis, 1994) dreams and nightmare analysis if properly systemised could have some advantages for exploring particularly sensitive areas of life experience, such as child sexual abuse.

There is agreement within the literature that adults who have experienced childhood sexual trauma frequently report impaired sleep and frequent nightmares. Studies by Garfield (1987) and Ellenson (1985) found that there was more explicit violence in the nightmares of those who had been sexually abused. Some case reports have reported more sexual content (Burgess and Holmstrom, 1978; Kavaler, 1987). The presence of shadowy figures has been reported (Robinson, 1982), as have snakes and worms (Weiss et al, 1955; Barry and Johnson, 1958). Belicki and Cuddy (1996) argue that nightmares and unpleasant dreams are expected in survivors of sexual trauma given that the 'elements of a dream are derived from self-concept, world-view and emotional response'. Cuddy and Belicki (1992) assessed 539 women, 124 reporting prior sexual trauma (63 of these reporting physical as well as sexual abuse), 71 women reporting only physical abuse and 344 reporting no abuse. They found that the sexual abuse group reported more nightmares, more repetitive nightmares; more sleep terrors and greater difficulty falling asleep after a nightmare than did the no abuse group. The sexual and physical abuse group reported a mean of 13 and 9.7 nightmares in the prior year respectively; the no abuse group reported a mean of 6.8 nightmares in the prior year. The sexual and physical abuse groups reported a mean of 4.2 and 3.3 repetitive nightmares in the prior year. The sexual and physical abuse groups reported a mean of 4.2 and 3.3 repetitive nightmares. Furthermore, these figures were derived from retrospective estimates which has been shown to produce lower estimates of nightmare frequency compared to logs (Wood and Bootzin, 1990). However, this study demonstrates that there are relative differences in these distressing experiences.

Sexual trauma and nightmare content

In order to examine possible content differences in nightmares Cuddy and Belicki (1992) studied 66 women reporting sexual abuse, 33 reporting physical abuse and 70 reporting no abuse. They completed a detailed questionnaire about their abusive experience, various measures of well-being and a sleep experiences questionnaire in which they recorded the worse nightmares that they could recall having and a typical nightmare. They also completed a checklist where they noted the presence of various themes, emotions, and objects that had occurred in their nightmares. They found that the worse nightmare prove to be better at distinguishing between the physical and sexual trauma group. A discriminate function analysis was able to sort subjects in sexual versus physical versus no abuse group with 79%

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accuracy. The authors argue that this clearly indicates that the damage sustained from sexual abuse cannot be accounted for simply in terms of the effects of physical abuse. They found that the women who reported sexual abuse tended to have more sexual themes in their nightmares, and sexual activity was likely to be associated with such negative qualities as distrust, shame, guilt, jealousy, anger, or violence. They states that although violence, or the threat of violence was common in all women's nightmares, when explicit details were present, such as blood or dismemberment, the dreamer was likely to have been sexually abused. It was found that several types of dream characters and objects were more common in the dreams of those who had been sexually abuse. They were more likely to have a male stranger as the pivotal character of the dream. The characters were often faceless, shadowy, or described as somehow evil. Images of snakes and worms were slightly more common in this group as were reference to parts of the body (particularly references to sexual anatomy). The authors state that higher scores for anatomy occurred usually because sexual trauma survivors were more likely to describe the physical appearance of the dream characters. However, they highlight that nightmares typically did not replay the actual abusive event. This is consistent with Finkelhor's (1987) argument that many survivors of sexual trauma do not fit a typical PTSD profile. The nightmares represented the emotions of the dreamers to the event. This suggested that the event was not perceived as a sexual act but rather an act of extreme violence.

Stress and recurrent dreams and nightmares

The examination of recurrent dreams may also enhance our understanding of the functioning of dreaming. Zadra (1996) highlights that most recurrent dreams have

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negative content, arise during periods of stress and resolve when the stressor has been dealt with effectively. A number studies have found that 60%-85% of recurrent dreams are described as being unpleasant by the subject (Cartwright, 1978; Zadra and Donderi, 1992). Robbins and Tanck (1993) and Cartwright (1979) assessed retrospective reports accounts of childhood recurrent dreams. They found that 36%-90% of these were unpleasant or threatening in nature. Zadra (1996) conducted a content analysis of 163 recurrent dreams. These dreams were collected from the dream reports of 352 subjects that completed the McGill Sleep/Dream Questionnaire as part of our studies on dreams between 1990-1992. The recurrent dream selected had to reach the following criteria; the recurrent dream must have occurred for a period of at least 6 months, the content of the recurrent dream had to be rated by the subject as being 'always' or 'almost always' identical, the recurrent dream had to be described in enough detail to allow content analyses. The recurrent dreams were also classified occurring to whether they occurred in adulthood or childhood. It was found that 77% of repetitive dreams from adulthood and 81.1% of dreams from childhood. It was found that among the dreams containing negative affect, fear or apprehension was the most frequently reported emotion, occurring in 67 and 79% of the adult and childhood recurrent dreams respectively. The rest of these recurrent dreams contained other negative emotions, including sadness, anger, confusion and guilt. Using the Achievement Outcomes measure it was found that adult recurrent dreams were nine times more likely that childhood recurrent dreams to contain one or more failures. Approximately 42% of the adult and childhood recurrent dreams contained one or more misfortunes, they majority in which the dreamer was the recipient of this misfortune. For both adult and childhood recurrent dreams the most frequent theme is one where the dreamer is being chased. Dream theorists generally agree that the

repetitive dream is related to an unresolved conflict or stressor in the dreamer's life. This is supported by Robbins and Houshi (1983) who found that undergraduate students who reported having a recurrent dream also had significantly higher scores on the BDI and reported a significantly greater number of problems in their daily lives than did undergraduates who did not have recurrent dreams. Brown and Donderi (1986) examined the relationship of recurrent dreams and well-being in former recurrent dreamers, recurrent dreamers and non-recurrent dreamers. They found that the recurrent dream group scored consistently lower on measures of well-being that the other groups (they had higher scores on depression, anxiety, personal adjustment and life events stress. The content analyses of the dream reports also showed that the recurrent dreamers experienced more anxious, dysphoric and conflict orientated dream content than either of the two groups. Interestingly, past recurrent dreamers score higher on the well being measures and positive dream content than the non recurrent dreamers. Zadra (1996) suggests that the maintained cessation of a recurrent dream is associated with a positive rebound effect on well-being. The finding that recurrent dreamers score significantly lower on measures of well-being than nonrecurrent dreamers has been replicated (Zadra, O'Brien and Donderi, 1993). Specifically it was found that recurrent dreamers reported higher levels of anxiety, neuroticism, depression, somatic symptomatology and life events stress and significantly lower levels of personal adjustment. Zadra et al (1993) also included a past recurrent dreamers group, however, they found that they did not differ from non recurrent dreamers on any of the well-being measures or any of the dream content measures.

These studies provide support for the assertion that dream content changes during and following episodes of stress and that the content becomes more negatively charged. Furthermore, these negatively charged dreams are associated with attenuated well-being.

Zadra, Miller and Dondei (1994) extended these findings to examine the relationship between people who experience repetitive dream themes (although not exactly the same dream repeated as in recurrent dreams). They found that people with repetitive dreams themes were found to score significantly lower on measures of wellbeing than those without repetitive dream themes. Furthermore, it was found that this group with repetitive dream themes scored higher (better adapted) than those with recurrent dreams. Content analysis also found that the dreams of the repetitive dream group contained more negative dream elements than did the dreams of the control group, however, the frequency and intensity of these negative dream elements was not as great as that found in individuals with recurrent dreams. Thus, these results suggest that people with repetitive dream themes show deficits on measures of well-being but not the extent of those with recurrent dreams. Zadra states that these results form a pattern where 'scores on measures of psychological well-being are inversely related to the position of a dreaming experience on the repetition continuum (Domhoff, 1993). On the basis of this repetition continuum it is hypothesised that recurrent trauma dreams would score even lower on measures of well-being than people with either recurrent dreams or repetitive dream themes. Domhoff (1993) argues that the dreams, which make up his repetition dimension (i.e. traumatic dreams, recurrent dreams, repeated themes and frequent dream elements) all reflect attempts at resolving emotional conflicts. Thus, this hypothesis suggests that when the emotional issue has

been resolved this should lead to the cessation of any type of recurrent dream. Brown and Donderi (1986) provide support for this in their finding that the cessation of recurrent dreams in adulthood is correlated with an elevation in well being. However, the existence of repetitive dreams that are positive has not yet been investigated. These occur in approximately 10% of recurrent dreams. It is not clear whether people who report repetitive positive dreams should show a deficit on measures of well-being

These studies suggest that the theories on recurrent dreams are in line with theories suggesting that dreams attempt to resolve current emotional concerns (Breger, 1967; Greenberg and Pearlman, 1975; Cartwright, 1977; Fiss, 1986). However, there is the problem of inferring causality. Does dream content reflect or influence waking adjustment or indeed both?

Interestingly, anecdotal reports have found that old recurrent dreams can resurface when a new crisis is faced. For example, Kramer, Schoen and Kinney (1987) found that many of their Vietnam veterans re-experienced their old traumatic dreams when dealing with martial crisis, demonstrating that these dreams can resurface when new stressors are faced.

The data reviewed support the generic psychological position that dreams are related to waking states. It has been shown that the link between people's dream content and their current level of well being is particularly evident in dreams that make up Domhoff's (1993) repetition continuum. Furthermore, the fact that cessation of recurrent dreams in adulthood is associated with an increase on self-reported measures of well-being suggests that a change from repetitive to progressive dreams may reflect how well a person is adapting to waking life situations.

Nightmares and Dissociation

Agargun et al (2003a) examined the relationship of nightmares to dissociative experiences in 292 undergraduate students. The subjects completed retrospective estimates of nightmare frequency. They were interviewed for childhood traumatic events including childhood physical and sexual abuse. Participants completed the Van Dream Anxiety Scale, which establishes a reliable and valid measure of dream anxiety (Agargun et al. 1999) and also the Dissociative Experiences Scale (DES; Berstein and Putnam, 1986). They found that the subjects with nightmares had significantly higher scores of the DES than those without nightmares. Furthermore, they found that 55% of the subjects who reported nightmares often, 27% of the subjects who reported nightmares sometimes and 24% of the subjects that never reported nightmares, reported at least one traumatic childhood event. Chi square analysis found that the rate of childhood traumatic experiences was significantly higher in the 'often' nightmares group versus the 'sometimes' and 'never' nightmare group. The rate of physical abuse was also significantly higher in the 'often' nightmare group versus the 'sometimes' and 'never group'. This study also found that subjects with a history of physical and with a history of sexual abuse had higher mean DES scores than those who had not. This replicates previous findings demonstrating an association between traumatic events in childhood and dissociative symptomology (Speigel et al 1991; Saxe et al 1993). These results indicate a strong association between nightmares and childhood traumatic experiences. However, Agargun et al (2003b) examine the prevalence of nightmare disorder (ND) in patients with dissociative disorders. They compared those with and without nightmares on measures of depression, borderline personality, suicide attempts and self-mutilative behaviour as well as history of childhood traumatic events. A 57% prevalence of

nightmare disorder was found among patients with dissociative disorders. They found that those patients with nightmares had a higher rate of self-mutilative behaviour, a history of suicide attempt in the last year and borderline personality disorder than those without ND. The patients were not significantly different in terms of age, age at onset, BDI, DES and history of childhood traumatic events. The finding that there was no association between nightmare disorder and childhood traumatic events is in contrast to their previous study (Agargun et al. 2003a). They suggest that this difference may be due to the fact that the previous study used a sample of undergraduates and not a patient sample as in this study.

The function of nightmares

Hartmann (1973, 1991b) postulates that the function of dreaming is to 'make connections' between recent material and older memories and that during dreaming connections can be made much more broadly than in waking. He states that this making of connections is not a random process instead the theory asserts that the emotion of the dreamer guides the dreaming process to choose patterns in the memory net, related to the emotional concerns of the dreamer (Hartmann, 1996).

The problem arises, however, is that if this is happening in dreams, what is happening in nightmares. Hartmann (1991b) thus asks if there is a distinction between 'dreams that work and dreams that poison.' He proposes that there may be some occasions when trauma is so great that dreams 'may not be helpful', but that dreams will still be attempting to 'bring together that which is usually kept apart.' This theme is then taken up in more detail in his (1998) book Dreams and Nightmares: The new theory on the origin and meaning of dreams. This book explores the metaphorical connections that dreams provide in their function of connecting memories, and with metaphorical images that can be very discomfiting if the memories involve traumas.

In the light of this work, other related hypotheses will now be reviewed.

Revonsuo's (2000) Threat Simulation Theory (TST) suggests that nightmares may allow the dreamer to adapt to a stressful situation by rehearsing it so that it will be easier when they encounter the problem in waking life. This theory states that dreaming is an essential biological defence mechanism, evolutionarily selected for its capacity to repeatedly simulate threatening events. Revonsuo (2000) argues that during dreaming the cognitive mechanisms required for efficient threat perception and threat avoidance are rehearsed and that this leads to an increased probability of reproductive success during human evolution. One hypothesis from the TST is that real threatening events encountered by the individual during wakefulness should lead to an activation of the system, a threat simulation response, and thus, an increased frequency and severity of threatening events in dreams. Valli et al (2003) tested this hypothesis by analysing the content of dream reports from severely traumatized and less traumatised Kurdish children and non-traumatised Finnish children. They found that the severely traumatised children reported a significantly greater number of dreams and their dreams included a greater number of threatening events. The dream threats of these severely traumatised children were also more severe in nature than the threats of less traumatised or non-traumatised children. They found that almost 80% of the dreams reported by the children in the severe trauma group included at least one threatening event, while 56% of the dreams reported in the less traumatised group and 31% of the dreams reported in the non-traumatised group contained at least one

threatening event. However, it could be argued that alternative theories could explain these findings. For example, theories proposing that dreaming aids in the solving of emotional problems by helping a person to adjust psychological to the threat or stressor (Cartwright, 1996; Hartmann, 1998; Kramer, 1993). Valli et al (2003) argue that the post traumatic nightmare poses a problem for other theories whereas TST predicts that powerful negative stimuli during wakefulness will lead to a strong and long lasting threat simulation response.

This Disruption-Avoidance-Adaptation model of dream function was proposed by Wright and Koulack (1987) and is a similar principle to systematic desensitization therapy. It is suggested that nightmares may serve to reduce the salience of a stressor through its repeated exposure, and thus, gradually reduce the associated affect.

Picchioni et al (2002) suggest that the function of nightmares is to serve in a problem solving capacity during times of stress. They argue that nightmares could provide a realistic but harmless environment for the individual to explore potential ways to approach the stressful situation.

Kramer's mood regulatory theory of dreaming

Kramer's mood regulatory theory (1993) suggests that the function of dreaming is to process emotions and thus, regulate mood. Kramer argues that dreaming functions to protect sleep by absorbing the emotional surge that occurs during REM sleep and that nightmares represent a failure in this function. The theory suggests that dreaming is related to emotional alterations and in most people there is a decrease in negative mood from night to morning. However, in the case of nightmares they suggest that the dreaming process is not effective and so may lead to more negative mood states the following day.

Kramer argues that there are two discernable patterns of thematic dream patterns across the night. The first is of the 'progressive-sequential' type in which problem are stated and worked on and resolved, the second is the 'repetitive traumatic' type in which the problem is stated but cannot be resolved. Kramer argues that the effectiveness of dreaming varies from night to night but that the nature of the problem solving is emotional. It is suggested that if one has experienced progressivesequential dreaming than there may be a positive acceleration in the emotional state of the dreamer. However, dreaming is less successful following the repetitive-traumatic type dreaming. It is through this 'problem solving' or failure to 'problem solve' that affective alteration occurs. He suggests that it is through this attempt at 'emotional problem solving' that the dream succeeds or fails at 'containing the emotional surge'.

Memory reprocessing

Thus, it could be that the improvement in mood is related to the function of REM. The following evidence suggests that sleep have a functional role in the reprocessing of memories. Indursky and Rotenberg (1998) found that in depressed patients mood in the morning was worse than in the evening. However, for 20% of all nights, mood was estimated as more positive in the morning than in the previous evening. It was observed that when mood was estimated as being better in the morning, eye movement density in REM sleep increased from the first to the fourth cycle. However, in all other nights eye movement density was slightly higher in the first than in the subsequent cycles. Mood improvement was found to correlate positively with eye movement density in the fourth cycle and negatively with REM sleep duration in the first cycle. Indursky and Rotenberg (1997) suggest a possible functional difference between initial and final REM sleep periods. However, the casual nature of the study leads to a number of possibilities. Firstly, it is proposed that the increase in eye movement density from cycle to cycle, which is usual for healthy participants, corresponds to mental activity with increasing participation of the dreamer in dream events (Rotenberg, 1988) and it is this mental activity that Kramer (1993) argues is responsibility for mood restoration. The authors argue that several lines of evidence lend support to this idea. For instance, REM deprivation studies have been shown to cause emotional disturbances in healthy participants (Greenberg, 1972), while REM deprivation in depressed participants has been shown to cause mood improvement (Vogel, 1980). However, the authors acknowledge the alternative explanation for these findings; normal eye movements from cycle to cycle are accompanied by mood improvement brought about in sleep by an unknown cause. Indursky and Rotenberg's (1998) findings also suggest that REM sleep periods may have different functional meanings. Eye movements in the fourth cycle correlated positive with mood restoration, but eye movements in the first cycle correlated negatively with the same variable. It is also interesting to note that increased eye movement density in the first REM period is typical of untreated depression. Cartwright et al (1994) suggest this may be an attempt by the brain to compensate for depressive disorder, although, these attempts appear to be insufficient. However, they highlight that one rare occasion and predominantly in the last cycles, did REM sleep in depression become functionally sufficient to perform a compensatory function and restore mood. Thus, it is clearly clinically important to take these functional differences in REM cycles into account if using REM deprivation in the treatment of depression.

Sleep and Memory

There is an implicit assumption underlying theories which propose an emotional processing or problem solving function of dreaming and this involves the processing of memories. Recent developments in molecular genetics, neurophysiology and the cognitive neurosciences have highlighted a fundamental role of sleep in the reprocessing of memories (Smith, 1985, 1995; Hennevin et al. 1995; Stickgold, 1998; Maquet, 2001, Stickgold et al 2001). However, the proposition that either sleep or dreaming plays a role in the off line processing of memories remains hotly debated (Horne, 2000; Stickgold, 1998; Vertes and Eastman, 2000). Stickgold et al (2001) states that 'evolution assigned sleep a critical role in selectively consolidating, translocating, integrating and in some cases weakening memories encoded over prior periods of waking'. (Stickgold 2003, p18). Research has generally suggested that both REM and SWS may be play a role in the consolidation of memories, although it seems that the strength of the influence of each of these sleep stages depends on the type of memory. Research suggests that declarative memories benefit more from SWS, predominantly during early sleep. Smith's (1996) meta analysis concluded that REM sleep plays a critical role in the consolidation of procedural learning but not of declarative memory. Subsequent research has suggested that this situation is altered when declarative memories are strongly associated with affect. For example, Wagner et al (2001) found using the same earlynight late-night procedure as Phihal and Born (1997) that while early sleep is better overall for simple declarative memory retention that REM sleep augments recall of emotionally charged memories. Furthermore, they found that REM sleep might

actually increase the negative reaction to previously viewed pictures with negative content (Wagner et al 2002).

Born and Gais (2003) also assessed whether the benefit of declarative memory consolidation from REM sleep is greater for emotionally aversive than for neutral material. They compared the recall of emotional and neutral texts following early and late nocturnal retention periods filled with sleep or wakefulness. They found that neutral texts did not benefit from late sleep, but there was a marked increase in recall performance after late sleep for the highly emotional texts. The early retention periods did not differentially affect subsequent recall of neutral or highly emotional texts.

These studies suggest that REM sleep (and possible REM sleep dreaming) can contribute to the processing of affective memories (Greenberg et al. 1972; Grieser et al. 1972; Cartwright et al. 1975). Furthermore, research has shown increased REM densities and shortened REM latencies have been reported in patients with major depression (Kupfer and Foster, 1972; Cartwright, 1983), the state of bereavement (Cartwright 1983; Reynolds et al 1993) and in posttraumatic stress disorder (Ross et al. 1989). These findings suggest that REM sleep processes emotional memories. Furthermore, it has been suggested that changes in REM sleep might hinder the processing of traumatic memories during sleep, which could contribute to the development of PTSD (Stickgold 2002).

Dreaming and memory

It is reasoned that dreams presumably reflect the activation and recombination of memories and thus, may offer insight into which memory systems are activated during dreaming. Hartley (1791) first suggested that dreaming might alter the strength of associative memories. Stickgold (2003) also argues that 'dreaming can be seen as a probe into the cognitive brain mechanisms involved in this reprocessing of memories and emotion, and the investigation of dreaming becomes a logical extension of ongoing research into the nature and consequences of sleep-dependent memory reprocessing' (Stickgold 2003, pp18). A number of research paradigms have attempted to identify the relationship between dreaming and these underlying brain processes. However, it remains unknown as to whether dreaming is simply an epiphenomenon or whether the process of dreaming actually contributes to sleepdependent memory reprocessing.

Dreams do not replay episodic images

Evidence suggests that episodic memory replay does not occur in dreams. For example, Fosse et al (2003) found that while neocortical memory traces formed during recent waking events frequently contribute to dream scenarios; they only very rarely provide the integrated context of episodic memories. They found that only 1-2% of dreams reflect the same processes of episodic memory recall that occurs in waking life and that the remaining 98-99% of dream elements related to waking thoughts and events. Thus, the authors suggest that the brain sources for dream elements are not hippocampally mediated episodic memories, but cortical traces of discrete components of the episodic memory, which then presumable are combined with the associated semantic memories. They argue that 'we dream about what happened not what actually occurred'. They suggest that as these dream reports were collected from home recall studies and so contained NREM and REM sleep reports, that episodic memory replay may not occur in either of these sleep stages. This is in line with imaging studies which have found that the dorsolateral prefrontal cortex is deactivated in REM and NREM sleep (Maguet et al. 1996; Braun et al. 1997; Maguet et al. 1997; Nofzinger et al. 1997). These structures have been shown to be critical for episodic memory access (Squire, 1992b; Schater and Tulving 1994; Squire and Knowlton, 1994).

Stickgold et al (2000) have demonstrated robust incorporation of daytime experiences into sleep onset mentation. Experts, novices and amnesiacs were required to play a computer game called 'Tetris' for 7-9 hours over 3-4 days. The authors found that all subjects reported similar dream imagery of the game and the imagery was limited to those aspects of the experience that were most salient and to which subjects appeared to pay the most attention. Importantly, none of the reports had the characteristics of episodic memory. Interestingly the authors found that there was usually a 24-hour delay after novices start playing before hypnagogic incorporation occurs. Stickgold et al (2003) suggest, therefore, that the brain employs a complex algorithm to identify memories for sleep onset reprocessing. However, the expert players reported images of the game on the first night and did not require this delay. A possible reason for this, suggested by the authors is that many players report becoming 'hooked' on the game on the second day. This suggests a stronger degree of emotional involvement, which was not present of the first day. Interestingly, two of the five experts who reported Tetris images from earlier versions of the game which they had not played for at least one year instead of images from the new game. Thus, it is suggested that the imagery is not necessarily a replay of recent sensory input, but instead can incorporate older, strongly associated memories. Of the five patients with amnesia 3 reported hypnagogic Tetris images despite being unable to recall playing the game before the nights sleep. Thus, the frequency of subjects reporting game imagery was similar for normals and amnesiacs (64% vs. 60%). The content of these reports were also markedly similar to normals.

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The findings suggest that hypnagogic images do not arise from hippocampal memory systems and that they are recalled without the activation of this system. Thus, the authors argue that this study supports the notion that dream images do not depend on the declarative memory system (Squire 1992a). Although the study does not tell us whether this hypnagogic mentation contributes to the process of memory consolidation and integration it does suggest that these images may reflect the underlying neurocognitive system altering strengths, structures and associations among emotionally identified memories. Similar findings have been reported by Emberger (2001) using an Alpine Racer skiing simulator game. They found that images reported from the game tended to be to from scenes, which were high in emotional salience, such as crash sites or initial experiences on a steep hill. However, despite this subjects reported relatively flat affect during the dreams. This is in sharp contrast to the strong emotions associated with hypnagogic replay of traumatic events in patients with PTSD.

This study, together, with Stickgold's (2000) study, suggest that memories are initially replayed in near veridical form and that more weakly related images only appear later on and these initial images seem to be replayed without the accompanying strong emotion. This is in contrast to Hartmann et al's (2000) model of contextual images which states that traumatic memories are first introduced in dreams by weakly related images which over time become more veridical in order to allow gradual processing of a trauma. Stickgold's (2000) and Emberger's (2001) findings suggest a form of systematic desensitisation of dreaming in which the emotional memories are replayed but without the strong affect, however, this differs from Perlis and Nielsen's (1993) theory which suggests that the strong feeling is present together with the imagery, however, the motor and sympathetic components are lacking. The Alpine racer study also examined the functional changes in memories related to dreams. This offers insight as to whether these hypnagognic dreams are functional or rather just the residue of daily events. Subjects were allowed 2 hours of interrupted sleep before being awakened and dream reports were obtained when they began to fall back asleep. It was found that subjects no longer reported images from the Alpine Racer game or previous skiing experiences; instead they reported images more weakly associated with the game experience such as 'falling downhill'. These findings suggest that the memories had been altered during the initial REM sleep cycle and were now become integrated into a wider associative network with the neocortical semantic memory system. However, further studies are needed to support these latter findings. However, these findings offer a glimpse of the underlying neurocognitive processes and they suggest that hypnagogic dreaming initiates a process leading to the integration of daytime experiences into a wider network of semantic knowledge.

There is also evidence for both emotion enhanced REM and REM-facilitated retention of emotional salient memories. Studies have found that both depression (Cartwright et al 1998) and pre-sleep viewing of unpleasant films (Lauer et al, 1997) correlate with reports of negative emotion in early night REM sleep dreaming.

The above studies of dreaming have major implications as they have demonstrated the robust incorporation of waking memories into dreams under controlled experimental conditions. This has offered insight into the brain mechanisms underlying sleep-dependent memory reprocessing. Already these studies have suggested that the medial temporal lobe's declarative memory system does not play a part in this process. It has also allowed the examination of the shift in hypnagogic dream imagery seen when sleep onset reports are collected later in the night. It seems that the related images shift from near veridical images to more weakly associated images. Hence, these studies will allow for the monitoring of this sleep dependent process of memory consolidation and integration. However, it is still unclear if dreaming has a role in these processes per se or whether dreams merely reflect these processes.

Post Traumatic Stress Disorder (PTSD): Definition

It has been proposed that sleep disturbances and nightmares are a hallmark of Post Traumatic Stress Disorder (PTSD;Harvey et al. 2003; Ross et al. 1989). This is an anxiety disorder that was first included in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1980). According to DSM-IV, the essential feature of PTSD (Criterion A) is the development of characteristic symptoms following exposure to an extremely traumatic stressor. PTSD symptoms are further divided into three categories, reexperiencing (criterion B) of the trauma, avoidance (criterion C) of trauma-related stimuli and increased emotional arousal (criterion D). Reexperiencing includes symptoms such as flashbacks, intrusive memories, dreams and distress when exposed to stimuli that remind the person of the event. Avoidance includes 'pushing' thought or feelings out of one's mind, avoidance of people or places or the inability to recall certain aspects of the event. Arousal is characterised by increased jumpiness, irritability and sleep disturbances. PTSD should be diagnosed if the symptoms persist for at least 1 month. If the symptoms remit within 4 weeks after the traumatic event, the diagnosis of an acute stress disorder (ASD) is indicated.

Prevalence of PTSD

In the general population, a lifetime prevalence of PTSD was estimated to be between 1% and 9% (Helzer et al. 1987). In the largest population study conducted to date, Kessler and colleagues (1995) surveyed 2812 men and 3065 women. They found a population prevalence of PTSD to be 8% overall with 10% of women and 5% of men to have developed PTSD during their lifetime. In victims of significant trauma, 20-45% will develop PTSD (Conlon et al. 1999; Murphy et al. 1999). Among soldiers who have participated in battles a PTSD prevalence of 15-20% has been reported (Card, 1987).

Sleep and PTSD

Two aspects of sleep-related symptoms are characteristic of PTSD and thus an important concern of this thesis. These are the re-experiencing cluster (criterion B) concerning nightmares and the intrusion of traumatic memories into dreams and the hyperarousal cluster (Criterion D), which includes difficulties regulating (i.e. initiating and maintaining) sleep, increased body movements during sleep and frequent awakenings (Mellman et al. 1995).

Subjective reports of sleep disturbances in PTSD

Many of the studies assessing sleep disturbances have relied on subjective reports from self-reported symptoms, questionnaires and structured interviews. In such studies the most frequently reported complaint is of difficulty falling asleep, frequent awakenings, shorter sleep duration, restless sleep, daytime sleepiness, nightmares and anxiety dreams.

Reported difficulties of initiating and maintaining sleep

In Neylan et al's (1998) reanalysis of the National Veterans Vietnam Veterans Readjustment Study, 44% of veterans with PTSD rated themselves as having problems with sleep onset either 'sometimes' or 'very frequently', compared to 6% of veterans without PTSD and 5% of civilians. Furthermore, they found that 91% of veterans with PTSD related that they had difficulty maintaining sleep either 'sometimes' or 'very frequently', compared to 63% of veterans without PTSD and 53% of civilians. In Ohayon and Shapiro's (2000) study, based on a large community sample, they found that participants with PTSD reported more difficulties initiating sleep (41%) compared to those without PTSD (13%). Those with PTSD reported more disrupted sleep compared to those without PTSD (47% and 18% respectively). Those with PTSD were more likely to report waking up early in the morning than those without the disorder (43% and 13% respectively). Kuch and Cox (1992) reported that 119 (96%) of their Holocaust survivors reported sleep disturbances. Rosen et al (1991) found that 42 Holocaust survivors showed decreased sleep quality on all dimensions of the Pittsburgh Sleep Quality questionnaire in comparison to nontraumatised controls.

Arousal /awakening threshold

As stated above, patients with PTSD complain that they wake up during the night and have difficulty falling asleep gain. Thus, it would be expected that the arousal threshold in patients with PTSD would be decrease (i.e. they would be awakened from sleep more easily), however, this doesn't appear to be the case. A number of studies have objectively measured the awakening thresholds of patients with PTSD and found that they have elevated awakening thresholds. Two of these studies applied tones during SWS sleep (Dagan et al. 1991) and one during REM sleep (Schoen et al. 1984). Therefore, these studies suggest that contrary to subjective reports patients with PTSD are in fact more difficult to awaken from sleep than normals. Lavie et al (1998) also found that higher awakening thresholds in patients with PTSD were positively correlated to the level of depression and anxiety. The authors postulate that this finding reflects an 'active blocking mechanism invoked to suppress trauma-related anxiety provoking material during sleep (p.1063) and /or that there is an extreme inward directed attention due to an intense preoccupation and inability to disengage from traumatic memories. These authors also suggest that the suggestion of 'deeper' sleep in patients with PTSD is congruent with findings demonstrating an usually low rate of REM elicited dream recall in traumatised patients (Hefez et al. 1987; Kaminer and Lavie, 1989). Kramer et al (1984) also reported on a lower rate of REM elicited dream recall in a group of non-traumatised Vietman veterans. The suggestion that level of arousal may interact with memory processes to influence dream recall has been previously raised (Koulack and Goodenough, 1976). However, the arousal stimuli used in these studies has been neutral tones; thus, it is possible that individuals with PTSD would have lower arousal threshold for threatening stimuli.

PTSD and REM sleep

The dream disturbances of PTSD appear to be relatively specific for this disorder (Ross et al. 1994). It has been suggested that dysfunctional REM sleep may be involved in the pathogenesis of the posttraumatic anxiety dream (Ross et al. 1994; van der Kolk et al. 1984; Mellman et al. 1995). It is, therefore, important to consider

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the findings relating to abnormalities in REM sleep. Differences have been demonstrated in the amount of REM sleep, REM latency, density of REM and awakening from REM. However, again these findings have been mixed.

PTSD and REM latency and percentage

The percentage of REM sleep has varied between studies. Some studies have found a shorter percentage of REM sleep (Lavie et al. 1979; Hefez et al. 1987; Scholsberg and Benjamin, 1978; Glaubman et al. 1990). However, other studies have reported that PTSD is associated with more REM sleep (Engdahl et al. 2000; Ross et al. 1994a, 1994b) and other studies have found normal amounts of REM sleep (Mellman et al. 1995). Similarly some researchers have demonstrated prolonged REM latencies in patients with PTSD (Kato et al. 1996; Lavie et al. 1979; Hefez et al. 1987; Schlosberg et al. 1978; Glaubman et al. 1990). Others have found shortened REM latencies (Greenberg et al 1972; Reist et al. 1995; Kauffman et al. 1987). The differences in the occurrence of depressive co-morbidity in these samples may account for this divergence of findings. Affective disorders have been found to affect REM sleep, causing shorter REM latency and increased REM time in the first third of the night. A number of studies have attempted to control for co-morbidity. Mellman et al examined PSG studies of 25 patients with PTSD and compared them with 16 patients with Major Depression and 10 controls. They found that the amount of REM sleep was reduced in the PTSD group compared to the MD group. Furthermore, individuals with PTSD had both the highest and the lowest values for REM latency compared to the MD group. The authors suggested that a possible explanation for this extreme variability in REM latency in PTSD patients might arise from the coexistence of pressure for REM to occur, with heightened arousal at night inhibiting the onset of

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REM. Thus, it may be that the extreme variability of REM measures is relatively specific to PTSD.

PTSD and REM density

REM density refers to the number of eye movements per REM time. Pillar et al (2000) suggest that measuring REM density might serve as a surrogate measure of 'stormy' dreams, without the need to actively awaken subjects and interfere with their natural sleep. A number of studies have found that REM density is increased in patients with PTSD (Hefez et al. 1987; Mellman et al. 1995a, Mellman et al. 1995b; Hurwitz et al. 1998; Ross et al. 1994). Although again it should be noted that increased REM density is not specific to PTSD but occurs in other psychiatric disorders, particularly in MD. Ross et al. (1994) compared patients with PTSD and MD with patients with just MD. They found that REM density was similar in the two groups, leading to the conclusion that increased REM density is associated with PTSD rather than MD. However, Dow and colleagues (1996) found that both PTSD and MD and MD alone had increased REM density, and suggested that this finding in PTSD reflects actual depression.

PTSD and dream recall

Kramer et al (1984) compared dream recall rates after spontaneous awakenings from REM sleep in Vietnam veterans with and without disturbing NREM dreams. They found that those without disturbing dreams had a low rate of dream recall. This study, therefore, suggests that a low dream recall may represent a 'healthier adaptation' possible reflecting an avoidance of inner processes as a means of adapting.

Lavie and Kaminer (1991) assessed the dreams of 23 Holocaust survivors and 10 controls. The survivors were divided into two groups according to how successfully they had adjusted to post-war life on the basis of clinical interviews (assessing problems at work, family problems, social relations, mental problems, somatic complaints and general life satisfaction). Sleep assessments included four nights of polysomnography and during nights 1, 3 and 4 participants were awakened from all REM periods. They found that the control group recalled significantly more dreams that the well-adjusted holocaust survivors. The less well adjusted group held an intermediate position which was not significantly different from either. The welladjusted group not only did not recall any dream content but in marked contrast to the other group they did not recall dreaming at all. Dreams of the control group were found to be longer with higher scores for vividness, imagination and emotional elements than dreams of the two survivor groups. The well adjusted group had significantly higher scores than the other groups for denial of emotions towards the dream after awakening. Dream content analysis found that for all anxiety scores (anxiety measures related to death, injury, separation, guilt, shame and diffuse anxiety) the survivors had higher scores than the control group. There were no significant differences between well-adjusted and less well adjusted survivors. Less well-adjusted survivors had significantly higher general anxiety, guilt anxiety and diffuse anxiety compared to the dreams of controls. The less adjusted group had the highest general hostility score, which was significantly different from the control group but not from the well adjusted group. However, when the subscales of this component were examined it was found that the control group scored significantly higher than the other groups for outwardly directed aggression. However, the less

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adjusted group had significantly higher scores on inwardly directed aggression than both the well adjusted survivors and the control group. It was found that dreams 'that dealt with danger to existence' appeared significantly more often in the dreams of the two survivor groups in comparison to controls. Content related directly to the Holocaust appeared in 4% of the dreams of the well-adjusted, in 7.8% of the lessadjusted and none of the controls. However, controls and well-adjusted survivors dreamed more about their present life than their past. The percentage of REM awakenings that elicited anxiety dreams were 25%, 11% and 5% for the less adjusted, well-adjusted and the control group respectively. When these percentages were based on the total number of REM awakenings, which elicited some content, they were 49%, 32% and 6%. Anxiety dreams were defined as dreams in which there was actual danger to the life of the dreamer, a physical attack on the dreamer or escape behaviour of the dreamer to save his life. Also on awakening the dreamer had to indicate that he felt fear or anxiety because of the dream. They concluded that dreaming was modified in both groups of holocaust survivors. Both groups recalled fewer dreams than the control group. Furthermore, the dream content and structure was qualitatively different from that of non-traumatised controls. However, in terms of basic content of dreams it seems that the well-adjusted group did not differ from the less-adjusted group. Furthermore, even 40 years after the Holocaust content specifically related to this dreams were still present in dreams. However, there were important differences between these groups of survivors, which the authors believe could be related to their success in post war adjustment. They argue that this differences lies in the substantially lowered dream recall in the well adjusted survivors (recalling only 34% of dreams following REM awakenings) and in the indifference response of well adjusted survivors to their dreams on awakening. The authors point out that the

literature showed no comparable findings of such a low rate of dream recall in a group otherwise having normal sleep parameters (Cohen, 1970).

Repetitive post traumatic nightmares are not nightmares

Hartmann (1996) argues that repetitive post- traumatic nightmares are not nightmares and even suggests that they could be renamed as 'memory intrusion'. He postulates that they differ completely from idiopathic nightmares in their content, repetitive quality, time of occurrence and underlying biology. He argues that these dreams do not participate in the 'connecting' functioning of dreaming and that they tend to occur in certain types of people. The above research also suggests that PTSD nightmares are 'intrusive memories' and do not participate in the 'connecting' function of dreaming as this process may be dysfunctional.

Studies showing how profile of PTSD and idiopathic nightmares differ

DeFazio (1955) found that 67% of recent combat veterans from Vietnam had nightmares. Brill and Beebe's (1955) follow up study of World War II veterans indicated that the presence of nightmares decreased with time and that only those veterans with pre-existing low levels of stress tolerance continued to have nightmares 20 years after exposure to combat. Van der Kolk et al (1981) conducted a survey of nightmare frequency among patients from the Boston Veterns Administration Outpatient clinic. They found that of the 199 combat veterans, that 59% reported nightmares more than once a month, usually with combat-related content, compared with 27% of those who had not been in combat. The above studies of personality and idiopathic lifelong nightmare sufferers suggest that these nightmares are distinct from those who nightmares occuring as a consequence of trauma.

van der Kolk et al (1984) examined how the nightmares and personalities of individuals with persistent traumatic nightmares following combat differ from those of individuals who suffer from lifelong nightmares. The first group consisted of veterans with frequent nightmares (one a month or more) that began during or after their combat experience in Vietnam who had a definite diagnosis of PTSD and with the exception of alcohol abuse, no other DSM-III psychiatric disorders. The second group consisted of 10 veterans with no actual combat experience but were life long nightmare sufferers (and had had thee since childhood). The third group, like the first had extensive combat experience but did not report nightmares in the previous 8 years. All subjects underwent 3-5 hours of psychiatric interviews and interviews about their sleep as well as a number of other psychological tests. They found that the chronic traumatic nightmares of men who had been in combat differed from veterans with lifelong nightmares and no combat experience. The lifelong nightmare suffers had a dreamlike content and were rarely repetitive in nature and were not accompanied by body movements. Thus, it seems that the exact replay of a on actual scene is unique to PTSD, whereas, ordinary nightmares show great variation in their content. Incidentally, lifelong nightmare sufferers may report repetitive dreams, however, on questioning it seems that although settings, characters or themes may be the same (e.g. being chased by a monster figure) these are not the exact repetition of a traumatic event. Furthermore, these nightmares tended to occur between 5am and 7am. In contrast, of the subjects with PTSD had repetitive dreams that were usually exact replicas of actual combat events. These nightmares tended to occur between 1 am and 3 am and all these subjects reported that body movements accompanied their nightmares. Furthermore, it was found that both the PTSD group and the lifelong nightmare group experienced current difficulties but these difficulties differed in their

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nature. The lifelong nightmare group had a number of difficulties in social adjustment and even some psychotic features. In contrast, the PTSD group had less disturbances of that nature but showed evidence of affective disorders; they also showed evidence of the expression of aggression and little control over their destinies in comparison to the combat control group. No evidence of 'thin' boundaries was found in the childhoods of those in the PTSD group, however, evidence for 'thin' boundaries was found in the childhood of lifelong nightmare suffers. 47% of the PTSD group was found to have a 'good' premorbid adjustment on a global rating, 40% as 'average' and 13% as 'poor'. By contrast, only 10% of the lifelong nightmare sufferers were rated as having an 'average' childhood and adolescent adjustment and 90% were rated as 'poor'. Thus, this study supports the notion that the characteristics of those with PTSD nightmares differed from those with ordinary idiopathic nightmares. Furthermore, it has been argued that there are clear biological differences between these nightmares. It has been demonstrated that ordinary idiopathic nightmares nearly always occur during long stretches of REM sleep (Hartmann, 1970; Fisher et al. 1974). PTSD dreams can entail muscular activity and partial arousal and they can occur in NREM sleep and REM sleep. Also, the content of these PTSD nightmares can also be experienced as 'flashbacks' during waking. Thus, Hartmann () concludes that the PTSD nightmare is not strictly a nightmare but rather an 'encapsulated memory that can intrude suddenly in consciousness either during REM sleep (non-REM as well as REM) or during waking. It is not an REM sleep dream, although it may intrude upon a dream' (p109). Furthermore, post traumatic nightmares in some cases can be reduced by medications such as fluoxetine, monoamineoxydase inhibitors or other antidepressants. Whereas, idiopathic nightmares are typically not affected by these but in severe cases dopamine blockers may help (Hartmann, 1984).

Hartmann's research has also suggests that those who develop PTSD with repetitive nightmares are not more prone to have nightmares anyway. Hence, this does not offer support to the assertion that these nightmares are ordinary nightmares dealing with the traumatic incident.

Sleep-dependent memory systems and PTSD.

Stickgold et al (2001) argue that repetitive dreams (reflecting the repetitive replay of the episodic memory during sleep) demonstrate a failure in the memory processes system. They highlight that outside PTSD episodic memories are almost never replayed verbatim in dreams. Thus, the replay of an episodic memory during sleep would require the breakdown of the normal blockade of hippocampal outflow to the cortex, which prevents the normal integration and subsequent weakening of episodic memory. Stickgold et al (2003) postulate that it is this sequence that leads to PTSD.

They suggest when an emotional event occurs the amygdale links the episodic memory to the emotions experienced. Thus, when an event is recalled it is accompanied by the original sensations and emotions, however, eventually the relevant information is extracted from this memory and is transferred to semantic memory networks located in the brain's neocortex. This process has been referred by as 'interleaved replay' (McClelland et al, 1995). Thus, hippocampal memories are slowly and repetitively replayed from the hippocampus to the cortex where over time memories are incorporated into one's general semantic knowledge and become available for the understanding of future events. It is believed that once this transfer is complete there the hippocampal memory is weakened along with its links to associated affect. Therefore memory can be freed up for the storing of further episodic memories. However, it has been argued that occasional this extraction process fails and information from the episodic memory fails to be extracted, transferred and intergrated into the neocortex and with other similar experiences. Furthermore, the weakening of the episodic memory and its associated affect that typically occurs following transfer also fails to occur. Thus, if the episodic memory is a traumatic one then this could result in PTSD and recover would rely on the reestablishment of these failed processes of cortical memory consolidation.

The integration of memories is where sleep may be of key importance. Stickgold et al (1998) argue that the more complex components of memory integration appear to occur when the brain is 'off-line' during sleep. As mentioned previously evidence suggests that NREM sleep appears most critical for strengthening of hippocampal memories and REM sleep for neocortical memories (Plihal and Born, 1997). In addition, semantic memory in humans has been found to preferentially activate weak associations in REM sleep, but strong ones in NREM sleep. Stickgold et al (1999) found that semantic priming favours weaker associations after REM awakenings in sleep inertia procedures. Furthermore, solving anagrams is similarly enhances after REM compared with NREM awakenings. This supports the notion that REM favours more fluid thinking than NREM. Thus, the bizarre, hyper associative nature of dreams may result from the preferential activation of weak semantic associations together with a lack of input from the hippocampus. Furthermore, hyperactivation of limbic cortices and the amygdale could similarly explain the hyperemotional aspect of dreaming (Merritt et al. 1994). Stickgold et al (2003) argues that the preferential activation of weak associative links with the neocortex enhances the testing of semantic associations most likely to lead to the generation of new

relationships between older memories. These authors suggest that while hippocampal outflow to the cortex during NREM sleep may serve to reinforce old memories, blocking hippocampal outflow during REM sleep will help prevent the reinforcement of predictable over-learned patterns and favour the formation of new associative links necessary for understanding the meaning of events in our lives.

Therapists aim to help patients with PTSD integrate the traumatic event into their lives and understand what it means for them. However, progress can not be made until the traumatic event can be discussed without replaying the episodic memory with its sensory and affective intensity. This process is similar to the physiological process of REM sleep, which includes the integration of the 'extracted and abstracted' episodic memory in cortical semantic memory networks uninterrupted by the intrusive hippocampal replay of episodic memories. In both of the above processes integration is the key for a reduction in symptoms. Stickgold et al (2001) propose that sleep, particularly REM sleep is programmed for exactly this type of memory transfer and integration. Repetitive dreams (reflecting the repetitive replay of the episodic memory during sleep) demonstrate a failure in the memory processes system. Stickgold et al highlight that outside PTSD episodic memories are almost never replayed verdically in dreams. Thus, the replay of an episodic memory during sleep would require the breakdown of the normal blockade of hippocampal outflow to the cortex, which prevents the normal integration and subsequent weakening of episodic memory. Stickgold et al (2003) argue that it is this sequence that leads to PTSD.

What causes the breakdown in memory consolidation?

The sleep of PTSD sufferers is known to be more fragmented than normal and studies have suggested that these patients appear to have a high level of vigilance even when asleep (Mellman et al, 1995 and Mellman, 1997). The amount of REM sleep may also be reduced (Lavie et al 1979; Hefez et a; 1987; Glaubman et al 1990; Mellman et al 1995). Both of these observations could be explained by an increased release of adrenal adrenaline or brainstem norepinephrine (NE). Norepinephrine also regulates the REM-non REM cycle. During REM sleep, activity in the locus coeruleus and dorsal Raphe nucleus, brainstem structures that control levels of NE and serotonin in the brain, normally ceases (Hobson et al. 1975). Stickgold (2002) suggest that a failure to shut down these systems would lead to a dissociated neuromodulatory state and incomplete entry into REM sleep. Under these circumstances associated processes would be expected to shift towards stronger associations and away from the weak associations normally activated during REM. These authors propose that this would lead to a breach of the blockade of information flow from the hippocampus to the cortex, allowing the replay of traumatic memories. Therefore, the occurrence of PTSD may be in part the result of the inability of the brain to inhibit NE or serotonin release during REM sleep. Such an increase during sleep would block the activation of weak associations within the cortex necessary for the integration of atypical memories into normative association networks and would also disinhibit the blockade of normal hippocampal outflow (Buzsaki, 1996), leading to the recurrent re-enactment of traumatic memories. Furthermore, with integration blocked there would be no feedback to the hippocampus and hence it would fail to weaken the episodic memory of the trauma and its associated affect.

Brain imaging studies have also suggested that PTSD and REM sleep share functional pathways in the brain. Rauch et al (1996) and Shin et al (1997) have shown that alterations in the activity of the hippocampus, amygdala, anterior cingulate and possibly orbital frontal cortex, and visual cortex when PTSD patients are provoked with script driven imagery. Hobson et al (1998) have demonstrated that these are the very same regions that are activated during REM sleep. These studies support the notion that traumatic memories are reprocessed during REM sleep and also the notion that PTSD may alter the normal functioning of the brain during REM sleep.

Eye movement desensitisation therapy

Various studies have provided evidence for the efficacy of eve movement desensitisation and reprocessing therapy (EMDR) in the treatment of PTSD (see Servan-Schreiber, 2000; Shapiro, 1999 for reviews). This involves patients shifting their gaze back and forth between two fingers held up in front of the patient on either side of the midline or with extremely contralateral movements. Shapiro (1989) originally proposed that the directed eve movements involved in EMDR mimic the saccades of REM sleep; however, she did not provide details on how this clinical improvement might occur. Stickgold (2002) states that if REM supports the memory integration of traumatic events then it is sensible to propose that a shift towards this brain state may also be beneficial. It has been proposed that repeated saccadic eye movements could 'push start' brainstem induced REM mechanisms (Nelson, McCarley and Hobson, 1983) through the reciprocal pathways that normally lead to REM sleep. Furthermore, brain-imaging studies have suggested that eye movements in REM sleep and wakefulness activate similar cortical areas. Stickgold argues that it is the orienting responses induced when a patient is required to constantly shift their attention across the midline, which generates a REM-like state, facilitating cortical integration of traumatic memories. Thus, it is proposed that the constant reorienting of attention demanded by the alternating bilateral visual, auditory, or tactile stimuli of EMDR automatically activates brain mechanisms which facilitate this reprocessing. It

is argued that activation of these systems simultaneously shifts the brain into a memory processing mode similar to that of REM sleep. This REM-like state permits the integration of traumatic memories into associative cortical networks without the interference from hippocampally mediated episodic recall. Once this memory has been integrated, corticohippocampal circuits induce the weakening of the traumatic episodic memory and its associated affect. However, Stickgold (2003) highlight that they are not suggesting that the breakdown in this memory processing pathway is critical for the development and maintenance of PTSD but rather that is play some important role in the disease process.

Sleep deprivation therapy for PTSD?

Research has suggested that post learning sleep may be particularly involved in the long-term retention of emotional memories and that it could contribute to the development of PTSD. An intriguing study conducted by Wagner and colleagues (2006) examined the role of post learning sleep on the retention of neutral and emotional memory several year later in a sample of healthy adults. They found that sleep after learning compared with wakefulness enhanced memory for emotional texts after 4 years. No enhancement was observed for neutral texts. Thus, brief periods of sleep immediately following learning cause preservation of emotional memories over several years. These findings highlight the importance of sleep for establishing long lasting and perhaps even lifelong memory traces in the case of events of strong emotional impact. The authors propose that one reason why long-term effects of sleep were only found for emotional texts may be due to the deeper encoding level that is generally observed for emotional as compared to neutral material (Hamann, 2001).

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The authors suggest that sleep deprivation in the immediate aftermath of traumatic events could be a promising therapeutic measure to prevent PTSD. They suggest that sleep deprivation would disrupt sleep-dependent processes of neural reactivation assumed to be necessary for synaptic and network changes underlying the consolidation of new memory traces (Ribeiro and Nicolelis, 2004). They speculate that distinct sleep disturbances that frequently follow traumatic experiences (Caldwell and Redeker, 2005) might be regarded as naturally occurring mechanisms helping to prevent excessively deep consolidation of these experiences in memory.

Conclusions

This general introduction has provided evidence suggesting that dreaming reflects waking life experiences and also that that dreams are related to corresponding waking or psychological variables (Domhoff, 1996, 2003). Research was reviewed suggesting that dreams may have an adaptive function in that they might provide an opportunity for the dreamer to integrate affectively charged material with past similar material that has already reached a successful resolution (Hartman 2001). Furthermore, such claims were supported by evidence suggesting that emotional memories are processed during sleep and this may be reflected by dream content. Failures in such memory systems and the consequent development of PTSD were discussed. This thesis aims to assess the frequency of nightmares in various populations with waking stressors and possible deficits in well-being. This thesis has the following general aims.

1. To determine if the correlations between measures of psychopathology and nightmare frequency, specifically anxiety and depression, are increased in 'hard to reach' populations with trauma or increased daytime stress. Nightmare frequency, anxiety and depression will be assessed in patients with sleep apnoea and head injury and also in frontline emergency service workers as well as chronic nightmare suffers. It is anticipated that nightmare frequency will be significantly elevated in these groups due to increased daytime stress or other trauma. The question is then whether correlations of nightmare frequency with psychopathology will be stronger than in the student samples that are often used in the literature.

2. Nightmare distress has been found in student samples to be a major confound of nightmare frequency – psychopathology correlations, even to the extent of rendering such correlations negligible. This has led to the counterintuitive conclusion (Blagrove et al, 2004) that nightmare frequency per se is not related to waking psychopathology, and that waking psychopathology instead simply enhances memory for, sensitivity to and recall of nightmares. This study will use more highly stressed or psychopathological populations to ascertain whether in them nightmare distress similarly is the mediating variable between waking psychopathology and reported nightmare frequency.

3. To determine if stricter definitions of nightmares, either by the use of the waking criterion or by the requirement of how unpleasant the dream

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has to be to be classed as a nightmare, will result in nightmares being a better index of waking psychopathology.

4. To assess within subjects correlations of state mood with the presence/absence of a nightmare that night in order to determine if day-to-day associations between nightmares and anxiety and depression are larger in comparison to between subjects analysis.

5. To assess between subjects correlations of individual difference variables with the within subjects correlations in aim 4, so as to investigate whether there are individual difference variables that predispose some individuals to have nightmares under conditions of high anxiety or high depression.

6. To assess whether ratings of PTSD correlate with nightmare frequency in individuals who have undergone repeated exposure to trauma (emergency service workers), and in individuals who have had one severe trauma (individuals with traumatic head injury).

7. To assess whether some individuals who may be expected to have increased nightmare frequency may not do so due to complete cessation of dreaming, either due to disrupted sleep (sleep apnoea patients) or to brain injury.

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Chapter 2

Study 1: Personality, psychopathology and nightmares

Introduction

The general introduction has reviewed evidence concerning the relationships between nightmares and psychological well-being. That literature is common to each of the four studies reported in this thesis. Study 1, unlike the subsequent studies, also investigated the emotional profile of dreams. Research exploring the emotions present in dreams will thus firstly be reviewed.

Emotion and dreams

It is widely accepted that emotion is a common feature of the dream experience. Seligman and Yellen (1987) describe dream emotions as a 'limbic bath' that pervades the dream. Hartmann's (1996) theory of Contextualising Imagery, based on the notion that dreaming makes broad connections in the mind, suggests that this making of connections is not a random process but that the emotion of the dreamer guides the dreaming process to choose patterns in the memory net related to the emotional concern of the dreamer. He states:

'... emotion is the force that drives or guides the connecting process and determines which of the countless possible connections are actualised at a particular time and thus which images appear in the dream. Dream 'contextualise' the dominant emotion'

(Hartmann, 1996, p153).

Imaging studies and emotion

The neuromodulatory systems that are strongly altered in REM are all known to play a central role in emotions such as anxiety and fear in waking. Maquet (1997, 2000) has highlighted that the frontal and temporal coritical regions activated in REM are those that receive afferents from the amygdale, while those not being activated do not receive such input. The activated regions include Broadman areas 24 and 32 in the anterior cingulated, causal orbitiofrontal regions, medial prefrontal area 10, the insula, and the entorhinal and parahippocampal cortices (Braun et al, 1997; Braun et al, 1998; Maquet et al,1996, 1997; Nofzinger et al, 1997). These regions are held to be central components of the highest order control system for emotions (Bechara, Damasio & Damasio, 2000). Thus, it seems that the brain is predisposed during REM sleep towards complex emotional functioning.

Imaging studies have demonstrated specific activation of limbic and paralimbic regions of the forebrain in REM sleep compared to waking (Braun et al, 1997; 1998; Maquet et al, 1996; Nofzinger et al, 1997). Hobson et al (2003) argue that such findings imply that dream emotions may be the primary shaper of dream plots rather than following secondary to the dream plot, a point also made in the quote from Hartmann (1996) above.

Nofzinger et al (1997) concluded from their PET study that an important function of REM sleep is the integration of neocortical function with basal forebrain and hypothalamic motivational and reward mechanisms. However, PET studies have also demonstrated significant deactivation of a large area of the dorsolateral prefrontal cortex in REM (Braun et al. 1997; Maguet et al. 1996). Single photon emission computer tomography (SPECT) and fMRI have demonstrated a similar decrease in cerebral blood flow to frontal areas during REM (Madsen et al, 1991a; Lovblad et al,1999). However, these findings need to be replicated. The latter observations are of interest in terms of the cognitive deficits in memory, orientation and self-reflective awareness during dreaming. These new findings suggest that the forebrain activation and synthesis processes underlying waking and dreaming are very different. Thus, the combination of the preferential activation of subcortical and cortical limbic structures which mediate emotion and the relative inactivation of the lateral prefrontal cortex involved in directive thought, paints an overall impression of REM sleep (and REM dreaming) as an emotion driven cognition with deficits in orientation, memory, volition and directive thought. Such findings may suggest that REM sleep plays a role in the adaptive processing of emotional and motivational learning (Maquet et al, 1996; Nofzinger et al, 1997). This may explain the emotionality and salience of REM dreaming, a view supported by PET (glucose) studies conducted by Gottschalk et al (1991a), which found a correlation between content analysed dream anxiety and medial frontal activation.

The emotional spectrum of dreams

Surprisingly little work has been carried out on the emotional spectrum of dreams and also on how waking emotions may affect dreaming. The current literature regarding the emotional characteristics of REM sleep is contradictory. Some authors argue that REM mentation is mundane and lacking in strong emotion (Foulkes, 1999; Hall &Van de Castle, 1966). However, others have argued that REM mentation is intense and hyperemotional (Seligman & Yellen, 1987; Merritt et al, 1994; Hobson et al, 1998). Some researchers have argued that negative dream emotions outweigh positive ones (Hall & Van de Castle, 1966; Kramer et al, 1971; Winget & Whitman, 1971; Merritt et al, 1994) whereas others researchers have found a balanced proportion (Strauch & Meier, 1996; Fosse et al, 2001; Schredl & Doll, 1998) or even found that positive emotions outweigh negative ones (Stewart & Koulack, 1993). It is, however, probable that methodological differences between studies can account for many of the inconsistencies, as described below.

Specific attempts to elicit emotions are necessary

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Hall and Van de Castle (1966) conducted the first large scale dream content analysis of 1000 reports. They found the incidence of emotion in only one third of all dreams, which appears to be surprisingly low. However, Hall and Van de Castle (1966) did not specifically ask participants to report the occurrence of emotions. Subsequent studies have shown a similar tendency towards the under reporting of emotions (McCarley & Hoffman, 1981), unless the dreamer is specifically instructed to describe dream emotions (Blick & Howe, 1984). For example, McCarley and Hoffman (1981) found reports of emotions in only 15% of their dream reports, which is half that of the suspiciously low frequency reported by Hall and Van de Castle (1966).

In contrast, Merritt et al (1994) found a ten-fold increase in the reporting of emotion when participants were instructed to note the presence of specified emotions as and when they occurred on a line-to-line basis in their reports. These authors assessed the emotional profiles of dreams in twenty psychology students (aged 20-50; males = 6 and females = 14). Participants were required to transcribe ten dream reports on a form specifically designed to capture and assess emotion immediately from spontaneous awakenings. Subjects were also asked to record the intensity of each emotion they experienced, and dream bizarreness was scored by three independent judges.

It was found that only 11 of the 200 dream reports contained no reports of emotion. Thus, the remaining 189 reports (95%) contained a total of 809 incidents of emotion. Anxiety was the most commonly reported emotion followed by joy/elation and these two emotions were found to constitute 58% of all emotion reports. Anger, sadness and shame/guilt were reported much less frequently and affection/eroticism had the lowest report rate of any emotion in the sample, constituting only 6.4% of emotion reports. These authors found that negative emotions were more prevalent in dreams, with over two thirds (68%) of the emotion reports in their sample being negative emotions. Interestingly, they found that positive emotions were most likely to appear in the first quartile of the dream and negative emotions in the last two quartiles. They note that while 42% of reports containing emotions began with a positive emotion, only 24% ended with one. Furthermore, 58% of dreams began with a negative emotion and 76% of the final emotions were negative, suggesting that dream emotions tend to go 'from bad to worse' (Merritt et al, 1994, p56). However, this study was part of a class exercise where students, over a period of three weeks, were required to record ten dream reports from spontaneous awakenings in their home with a word count of at least 50 words. Participants were instructed to score their reports for emotions. Fosse et al (2001) argue that this type of spontaneous home recall design is likely to sample selectively the most dramatic and emotionally intense

dreams, thus neglecting reports of less salient emotions. Furthermore, the fact that the students were known to Merritt et al (1994) may have biased the findings. However, these findings do suggest that reports of emotions in dreams increase dramatically when a special attempt is made to elicit them.

The rating of emotions in dreams

A further methodological discrepancy between studies concerns how dream emotions are rated. Some studies require participants to provide their own estimation of dream emotion, whereas, in others, external judges are required to rate the transcribed dream reports for emotions. It has been suggested that when external judges have scored dream reports, emotions have been strongly underestimated compared to subject's own ratings. For example, Foulkes et al (1996) found that their subjects reported at least one emotion in 70% of their laboratory dreams; however, Synder (1970) found that only 30% of laboratory dreams were scored as emotionally charged by external raters. Furthermore, in home recall studies, such as Hall and van de Castle (1966), external judges scored about 0.35 emotions per dream diary, whereas dreamers themselves have been found to report about 3.6 emotions per diary (Dudley & Ransom, 1988; Merritt et al, 1994) or, in one study, up to 8 emotions per dream (Nielsen, Deslauriers & Baylor, 1991). Schredl and Doll (1998) found a balanced proportion of positive and negative emotions when participants rated their own dreams (42.6% negative versus 38.2% positive). However, when external judges rated these same dreams, negative emotions were found to outweigh positive ones (56.7% negative and 20.6% positive). They concluded that external judges generally underestimate the incidence of emotion but that they particularly underestimate positive emotions. Schredl (1991) reached a similar conclusion. He compared

estimates of two 4-point scales concerning emotional intensity (positive and negative emotions) to ratings made by external judges on similar scales. The results indicated that judges underestimated the intensity of positive emotions in particular but not the intensity of negative ones, as reported by the dreamer him/herself. Moreover, other studies have indicated that only about 50% or less of the emotions occurring in a dream, as measured by self-ratings, were correctly identified by judges (Stairs and-Blick, 1979; Borioli, Meier & Strauch, 1984; Foulkes et al, 1988).

Thus the use of external judges to rate dream emotions may explain the finding of a higher preponderance of negative emotions in dream reports. For example, Cartwright et al (1998), Strauch and Meier (1996) and Fosse et al (2001), using first person ratings, found a relatively balanced proportion of positive and negative emotions. However, notwithstanding this methodological criticism, Merritt et al (2004) found a greater preponderance of negative emotion compared to positive using first person ratings.

Explicitly mentioned dream emotion and overall dream emotional tone

Another critical point in evaluating this research regarding emotion in dreams is whether only explicitly mentioned emotions were elicited or whether moods (a positive or negative general atmosphere) were also included in the analysis. This is important since there is little agreement between psychologists on the terms 'affect', 'emotion', 'feelings' and 'moods' (Davidson, 1994). Snyder (1970), who analysed 635 laboratory dream reports, highlights that external judges were not able to rate such general moods by reading a dream report. Strauch and Meier (1989), analysing 500 laboratory dream reports, found explicitly mentioned emotions in 50% of their dream samples, but in an additional 23.1% of the dreams only moods could be detected. Furthermore, they found that dream moods were estimated as intense as explicitly mentioned emotions. They found that whereas two thirds of the emotions were negative, three out of four moods were positively toned. As a result, 30.9% of dreams were positive toned, 28.9% were negative toned, 10.2% contained both types of emotions (i.e. mixed) and in 28.4% no emotions were reported. Stairs and Blick (1979) asked their participants to report only the two most intense dream emotions; 44% were negative and 49% were positive. Therefore, it seems that when participants are asked to rate the overall emotional tone of their dreams that both moods and explicit emotions will form the basis of their judgement, even if they remain unmentioned. For example, in Stewart & Koulack (1993), participants were asked to estimate the dream affect quality on a 7 point scale ranging from extremely positive to extremely negative. The mean value of all subjects' dreams pointed slightly in the direction of positive emotions. Hence, these studies suggest that negative emotions may predominate if only explicitly mentioned dream reports are used.

Intensity of emotions

Another factor which may affect the measurement of explicit dream emotions concerns the intensity of emotion, particularly the intensity of negative dream emotions in comparison to positive ones. For example, McFalls, Roe & Blick (1980) reported that anxiety feelings, which often occurred in dream reports, were rated as less intense than other emotions such as surprise or interest. Furthermore, a number of studies have demonstrated that self-rated positive emotions were slightly more intense than negative ones (Strauch & Meier, 1989; Nielsen et al, 1991). Schredl and Doll (1998) argue that this may contribute to the overall estimation of negative emotions if only occurrence of emotion is measured.

Instrumental awakenings from REM sleep

Another possible confounding variable when looking at the emotional profile of dreams is the different methodologies used to elicit dream reports. Some studies have used instrumental awakening during REM sleep, whereas others have collected dream reports from spontaneous home recall studies. It has been argued that the latter reports can be extremely biased. Foulkes (1979) compared spontaneously reported home dreams to those elicited during laboratory awakenings from REM and found that spontaneously reported home dreams were an extremely biased subset of the most dramatic and emotionally unpleasant dreams. It is generally agreed that the REM awakening technique is the most appropriate method for obtaining dream reports although this paradigm may also influence dream content to a certain extent. Some studies have demonstrated that laboratory dreams contain less aggression and sexuality than do home dreams (Foulkes, 1979; Domhoff & Kamiya, 1964). Ruf (1973) found that laboratory dreams are less intense than dreams recalled at home, and Hartmann (1984) also found that nightmares were drastically reduced when subjects were polysomnographically recorded in the laboratory, although Weisz and Foulkes (1970) did not find any differences in emotional tone between home and laboratory dreams. Strauch and Meier (1996) reported that up to 50% of the dreams in their study contained at least one laboratory-related element. These findings illustrate the advantages and possible disadvantages of laboratory dream reports. However, some authors argue that the reliance on home dream reports may lead to a predominance of negative emotions in dreams (Fosse et al, 2001).

Dream recall: a confounding variable?

The relationship between dream recall and dream emotions also needs to be considered when elevating research on the emotional spectra of dreams. A number of studies have found a positive relationship between dream recall frequency and intensity of dream emotions (Schonbar, 1961; Belicki & Bowers, 1981, 1982; Schredl & Doll, 1998). Furthermore, Belicki et al (1978) found that high dream recall was associated with more positive dream emotions. This may be explained by the salience hypothesis (Cohen & MacNeilage, 1974) which suggests that the more intense a dream is the better it can be remembered. However, it may also be the case that high dream recallers are simply better trained at remembering and reporting dream content and dream emotions.

<u>Gender</u>

It may be that emotional profiles in dreams are influenced by gender. For example, it is generally believed that the expression of emotions in males and females is influenced by gender-learned appropriate behaviours. Some studies have found that women tend to have more intense dreams (Hall & Van de Castle, 1966; Winget, Kramer & Whitman, 1972; Merritt et al,1994) and more negatively toned ones (McElroy, 1952; Strauch & Meier, 1989) than do men. Lortie-Lussier et al (1992) studied the dreams of women and suggested that they are linked to the social roles of women and that new occupational roles are reducing stereotypical feminine dream imagery. However, in contrast, Van de Castle's (1982) study examining the roles of women and men in 1950 and 1980 found that there has been little change over a period of 30 years in what college students dream about and that sex differences in the 1980 dreams are the same as those in the 1950 dreams. However, Rubinstein and Krippner (1991) collected dreams of males and females in response to a television announcement and did not replicate previously reported gender differences such as in the amount of aggression, friendliness, sexuality, male characters, weapons or clothes.

However, Hall and van de Castle (1966) found that no single emotion showed a difference between men and women when analysed independently, and although Merritt et al (1994) found that women reported emotion 23% more frequently than men, this difference was not statistically significant. They also found no significant differences between any of the dream emotions in their samples of men and women. Also, Kahn and Hobson (2002) found that men and women did not differ significantly on the number of feelings they reported in their dreams or in the number of positive and negative feelings reported. They found that the feelings most often reported in dreams were the same for males and females, however, the rank order of these feelings was different. For women, the most often reported emotions were, in order of frequency: anger, affection, anxiety and joy, whereas, for males the most often reported feelings were; joy, affection, anxiety and anger. However, none of these differences reached statistical significance. Furthermore, Schredl and Doll (1998) found no evidence that women report more intense or more negatively toned dreams than men. They found that in the case of explicitly mentioned emotions there was a tendency for women to report emotions more often than do men but they suggest that this difference may be partly due to dream length, which was higher in women. Thus, the weight of the evidence suggests that there are few or no differences between the emotional spectrum of male and female dream reports.

Fosse et al (2001)

As study one is based on many of the same procedures used in the Fosse et al (2001) study (with the exception of REM awakenings) a detailed account of this study is now provided. As study one assessed the dreams of nightmare sufferers the REM awakening procedure was not considered suitable as the appearance of nightmares is drastically reduced in the laboratory (Hartmann, 1984).

Fosse et al (2001) assessed the emotional profile of nine healthy individuals sleeping in their own homes and monitored by ambulatory polysomnography. Participants were awakened 5 to 15 minutes into REM sleep across the night, for 3 consecutive nights. On awakenings participants were required to write down any mentation recalled from the pre-awakening period. For each line in the report they were asked to report the occurrence and intensity (ranging from 1-3) of the following emotions; anxiety/fear, anger, joy/elation, shame, sadness, surprise, love/eroticism, in addition to any other emotions they experienced.

65 of 88 reports (i.e. 74% of reports) had at least one emotion. There were 157 instances of emotion in total. 48% of the reports, or 65% of those containing emotions, contained two or more different emotions, and 16% (or 21% of those containing emotions), had at least three different emotions. When sorted into positive and negative emotions they found that of the 157 incidences of emotion, 52 (33%) were positive, 69 (44%) were negative and 36 (23%) were neutral. However, these authors noted that positive emotions were found in 49% of the reports and negative in only 42%. This difference between incidence and report prevalence reflected the fact that when occurring in a report, negative emotions showed a higher frequency than did positive emotions (1.73 vs. 1.25). These authors found that the difference between positive and negative emotions was not significantly different either on an overall incidence basis or a per report basis. Fosse et al also examined the overall intensity of positive and negative emotions in each report. Total negative intensity (TNE) and total positive intensity (TPE) were calculated as the sum of the highest intensity score for each discrete positive and negative emotion, respectively, in the report. It was found that the average TPE and TNE did not differ significantly either when tested on a per subject basis using average report scores or on a per report basis. They found that approximately half of the subjects had higher TPEs than TNEs (n = 5) and half had higher TNEs (n=4). Therefore, they concluded that positive and negative emotions occur in a balanced fashion.

Prevalence of discrete emotion types

In terms of prevalence of discrete emotions, joy/elation was found to be the most prevalent, occurring in 36% of the reports. The second most prevalent discrete emotion was surprise (24%), followed by anger (17%), anxiety/fear (11%), and sadness (10%). The difference in the occurrence of these emotion types was tested using the average report prevalence for each subject. Joy/elation was found to occur significantly more than anxiety /fear and also showed a trend towards a higher prevalence than anger. When these tests were conducted on a per-report basis, surprise was also found to be more prevalent than anxiety/fear.

Intensity of discrete emotions

26% of the reports had no emotions (intensity score of 0), 18% had an intensity score of 1 (low), 28% of 2 (medium) and 28% of 3 (high). Thus, about half the reports were given an emotional intensity score of medium or higher. On a persubject basis anxiety/fear was found to be less intense that both joy/elation and anger. On a per-report basis, significant overall differences were found in the intensity levels of joy/elation, surprise, anger and anxiety/fear. Anxiety/fear was found to be less intense than each of the other emotions. They report that the average intensity score for anxiety /fear (1.3) per report was one standard deviation below the mean of the average intensity score for joy/elation, anger, and surprise (2.1, SD = 0.7) combined. Therefore, the authors conclude that not only is anxiety/fear less prevalent than the other emotions but that when it does occur it is less intense. Joy/elation was found to be significantly less intense than surprise and had a tendency to be less intense than anger, despite that fact that it was the most prevalent emotion.

Inter-subject differences

Fosse et al tested total scores for positive and negative emotions and surprise in order to determine if they were stable across subjects. They found significant intersubject differences existed for TNE and surprise but no variation was found for positive emotions. They concluded that positive emotions are typical of most subjects but that both negative emotions and surprise are more typical of some but less so for other subjects. Fosse et al (2001) suggest that such variations in negative, positive and surprise emotions across subjects may be related to pre-sleep mood. It has been previously demonstrated that negative presleep mood can affect emotions in early REM sleep. For example, Cartwright et al (1998) found that mental activity in early (but not late REM sleep) was more negatively toned than in controls. Furthermore, Fosse et al suggest that other factors such as individual differences and personality may play a role in the variation of emotions between subjects. These factors will be assessed in the current study.

Change in emotion across the night

Fosse et al (2001) assessed the relationship between time spent asleep and the intensity of each discrete emotion. Thus, analyses were based on average scores per subject. Scores were also calculated for the first half of the night (less than 4 hours after sleep onset) and the second half of the night (more than 4 hours). They found a significant increase in surprise from the first to second half of the night (13% vs. 29% of reports). In contrast no significant change was found across the night for any of the other emotions. This was also the case when the data were analysis on a per-report basis. No change was found for TPE or TNE. This stability of positive and negative emotions across the night is perhaps surprising as many other mentation elements, such as, visual vividness, report length and bizarreness all increase considerably as the night progresses (Antrobus, Kondo & Reinsel, 1995; Fosse, 2000; Fosse, Stickgold & Hobson, 2001; Foulkes, 1962). However, the increase in surprise emotion throughout the night may draw parallels to other variations in dream mentation, particularly increases shown in dream bizarreness (Fosse, 2000). Fosse et al (2001) point out that bizarreness and emotion has been shown to co-vary in dreams (Merritt et al, 1994). Fosse et al (2001) suggest that these findings may indicate that different subsets of emotions are differentially related to and variously dependent on aspects of the hallucinatory scenario and to the dreamer's ability to think and reflect.

Summary of this chapter and chapter 1 as it relates to study 1

The frequency of nightmares has small to medium between subjects correlations with various individual difference measures, such as anxiety and boundariness. However, the findings of studies on nightmares, individual differences and waking life poor well-being, as described in chapter 1, have been hampered by inconsistencies in how nightmares are defined.

Study 1 attempts to assess relationships between nightmare frequency and individual difference and poor well-being variables while varying the definitions of nightmare used in the analyses. This will provide information on what aspects of nightmares are related to poor wellbeing.

This study will also take account of the confounding effects of nightmares distress in the apparent relationships between nightmare frequency and psychological well-being. Within subjects correlations will be used in addition to more common between subjects correlations with nightmare frequency.

Although there has been work on the specific emotions present in dreams, there has been little research into the specific emotions present in nightmares. This study will assess the emotions present in nightmares and bad dreams.

General aims and hypotheses of study 1

1. To assess relationships of nightmare frequency with measures of poor well-being, boundariness, creativity/fantasy proneness and cognitive arousal. It is hypothesised that the frequency of nightmares will be significantly related to all measures of psychological well-being, and to boundariness, creativity/fantasy proneness and cognitive arousal.

2. To assess the role of nightmare distress as a mediator in the above relationships with nightmare frequency. It was hypothesized that nightmare distress would be significantly related to all measures of well-being, and that partialling out nightmare distress would greatly diminish many of the correlations between nightmare frequency and poor well-being.

However, within these general aims the following questions will be assessed.

a) Does the degree of unpleasantness that is required for a dream to be classed as a nightmare affect the correlations with well-being?

b) Does the factor of degree of unpleasantness in the definition interact with the use of the waking criterion to effect size of correlations with well-being?

c) Is there any advantage in counting the number of nightmares over a period (whether defined with the waking criterion or not) as opposed to the number of nights on which at least one nightmare occurred? The questions above relate to between subjects correlations. A further class of questions involves the within subjects correlations between presence/absence of nightmare each night and state well-being just before and just after sleep. These questions are:

d) How do within subjects correlations between nightmare frequency and well-being compare in size to between subjects correlations?

e) Are these within subjects correlations affected by the use of the waking criterion or strictness of the unpleasantness criterion in defining nightmares?

f) Do the within subjects correlations themselves correlate significantly with individual difference variables such as neuroticism, boundariness, etc.?

Question f) has the novel aim of finding whether there are trait predisposing factors that interact with state anxiety or state depression to cause nightmares.

3. This study also aims to compare the emotional profiles of nightmares and bad dreams, and to compare the profile of dreams that are not nightmares or bad dreams with the normative data of Fosse et al (2001). Given that this sample consists predominantly of nightmares sufferers it is hypothesised that:

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g) the emotions in their dreams in general will be more negative than positive, but no prediction is made about the emotions in their nonnightmare non-bad dreams;

h) nightmares will have significantly more negative emotions, and that these will be significantly more intense, than in bad dreams.

i) nightmares and bad dreams will contain emotions other than fear/anxiety.

4. Given that mean dream emotional tone has only been found to have, at best, small correlations with waking life poor well-being, no predictions are made as to whether incidence or intensity of specific dream emotions, or Total Negative Emotions score, will be significantly associated with waking life poor well-being.

Chapter 3

Study 1: Personality, psychopathology and nightmares

Method

Participants

Part 1 – Questionnaire study

68 participants (male = 14, female = 54, mean age = 39.18, SD = 18.51; range = 18-82) were recruited from the general population through advertisements in the local newspaper and radio broadcasts. Advertisements asked for volunteers who had nightmares to assist in a research project examining the causes of nightmares. Care was taken in the wording of advertisements not to suggest that nightmares were distressing (e.g. it was asked 'do you have nightmares?' rather than 'do you suffer from nightmares?)' in order to avoid response bias. Participants were recruited from a wide geographical area as the study could be completed via the post. Participants were not paid for taking part in the study. The study included people who have frequent nightmares and those that do not. Participants were excluded if they had a sleep disorder or psychiatric illness. Participants were asked to record any medication they were taking at the time of the study. The study and its methods were approved by the departmental ethical committee (appendix 1).

Part two – Diary study

56 of the 68 participants (male = 12, female = 44; mean age = 41.05, SD = 19.08) also completed a 14-day dream diary.

Measures

Part one:

Nightmare Distress Questionnaire' (NDQ, Belicki, 1991)

The NDQ is a 13 item five-choice measure to assess waking distress due to nightmares. The NDS has been used in a number of studies with adequate reliability and validity (Belicki, 1992; Levin & Fireman, 2001; Madrid et al., 2000). Higher scores on this questionnaire indicate a greater distress caused by nightmares, such as fear of going to sleep in case a nightmare occurs, or and wishing to have therapy to treat nightmares.

State and Trait Anxiety Inventory (STAI-S; Spielberger, Gorsuch and Lushene, 1970).

Trait and state anxiety were measured by the Spielberger STAI-S (Spielberger, Gorsuch and Lushene, 1970). The STAI contains two self-report surveys, each with 20 items that assess worry, apprehension, tension and nervousness. The trait anxiety scale measures the 'general' level of anxiety and its stability over time and the state scale measures how one feels 'now'. The STAI has been used extensively and, construct validity, concurrent validity, and test-retest reliability have been established as adequate (Speiberger, Gorush, Lushene, Vagg and Jacobs,1983). Spielberger et al (1983) reported that the mean (and standard deviation) for trait anxiety was 35.55 (9.76) for males and 36.15 (9.53) for females in a normative samples of working adults aged 19-39. The mean (and standard deviation) for state anxiety was 36.54 (10.22) for males and 36.17 (10.96) for females in the same sample. Responses to the items on the trait and state scales are on a 4-point Likert-type scale ranging from 'not

at all', to 'very much so'. Each questionnaire is scored on a scale of 1 to 4 with a higher resulting score indicating a higher level of anxiety.

Symptom checklist (SCL-90-R;Derogitis, 1977)

The SCL-90-R is a valid multi-dimensional 90-item self-report symptom inventory designed to reflect the psychological symptom patterns of community, medical and psychiatric respondents. The SCL-90-R consists of nine primary symptom dimensions and three global indices of distress. The primary symptom dimensions are:

<u>Somatisation:</u> This is indicated by symptoms such as headache or upset stomach. These typically have a high prevalence in disorders of functional aetiology.

<u>Depression</u>: This is determined by a broad range of questions related to dysphoric mood and affect and cognitive and somatic correlates of depression.

<u>Anxiety:</u> This is manifested by symptoms such as nervousness, tension and apprehension.

Interpersonal sensitivity: This reflects feelings of personal inadequacy, as manifested by self-deprecation or discomfort during interpersonal interactions.

<u>Hostility:</u> This is demonstrated by negative thoughts, feelings or actions stemming from anger.



<u>Obsessive-compulsive symptoms</u>: These are related to patterns of thought or behaviours that are irresistible and unwanted in nature.

<u>Phobic anxiety:</u> This is indicated by irrational responses to situations or persons, leading to avoidance of those circumstances.

<u>Psychoticsim:</u> This dimension is aimed as recognising a spectrum of clinical entities ranging from withdrawn schizoid lifestyle to overt psychosis.

The SCL-90-R also has three global indices. Research has suggested that these reflect distinct aspects of psychological disorder (Derogatis, Yevzeroff and Wittelsberger, 1975). The global distress indices are:

<u>Global Severity Index (GSI)</u>: This index best denotes psychological distress and is the most sensitive single numeric indicator of a respondent's distress as it takes into account the intensity of distress and the number of symptoms.

<u>The Positive Symptom Distress Index (PSDI):</u> this reflects the average level of distress reported for the symptoms that were endorsed. As such it can be interpreted as a measure of symptom intensity.

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<u>The Positive Symptom Total: Th</u>is is a reflection of the number of symptoms endorsed by the respondent regardless of the level of distress reported. It can be interpreted as a measure of symptom breadth. The SC-90-R is a measure of current, point-in-time, psychological symptom status and is not a measure of personality. The internal consistency of the scale is high (ranging from 0.77 to 0.90) with high test-retest correlations (ranging from 0.78 to 0.90) (Derogatis, 1983). Each item is rated on a 5-point scale of distress (0-4) ranging from 'not at all' to 'extremely'. Psychological distress, as denoted by the Global Severity Index, is calculated by dividing the summed total score by the number of items. Within each symptom dimension, scores are averaged across the respective items in that dimension.

Derogatis (1994) suggests guidelines for identifying clinical cases on the SCL-90-R. It is suggested that a t-score on the General Symptom Index >63 or the presence of any two elevated clinical scales >63 has a good predictive value with adequate sensitivity and specificity.

Creative Experience Questionnaire (CEQ; Merckelbach, Horselenberg and Muris, 2001).

This is a 25-item self -report measure of fantasy proneness, each item has a yes /no format. It has been shown to demonstrate good test-retest stability (r = 0.95) and internal consistency (Cronbach's alpha = 0.72). It has been demonstrated to have predictive validity (Merckelbach, Horselenberg and Muris, 2001) and concurrent validity (Merckelbach, Horselenberg and Muris, 2001). Higher scores are an indicator of greater proneness to fantasy.

Arousal Predisposition Scale (APS; Coren, 1988).

This is a 12-item self-report measure of cognitive arousability. This questionnaire was developed to attempt to measure a predisposition or a trait of

cognitive hyperarousal that might predict tendencies towards disrupted sleep patterns. Coren (1990) has produced norms for the APS and has reported that the reliability is acceptable (Cronbach's alpha coefficient was found to be 0.83). A number of studies have provided additional support for the validity of the APS (Coren and Aks, 1991; Coren and Mah, 1993; and Hicks, Conti and Nellis, 1992).

Responses to the items on the APS are on a 5-point Likert-type scale: Always (scored 5), Frequently (scored 4), Occasionally (scored 3), Seldom (scored 2) and Never (scored 1). The responses on all the items can be summed to yield an overall APS score that could range from 12 (the lowest possible level of arousability) to 60 (the highest possible level of arousability).

Borderline Personality Scale (STB; Claridge and Broks, 1984).

The Schizotypal Personality scale taps into a variety of different areas of subclinical schizotypal symptomology. The STB subscale was used in the current study; this is largely a measure of emotional instability and personality characteristics of a general antisocial kind (Claridge and Broks, 1984). The STB is an 18- item selfreport measure based on the criteria for Borderline Personality Disorder set out in DSM-III (American Psychiatric Association, 1980). Factor analysis has demonstrated that the STB produces two factors; a Hopelessness factor, including items associated with dejection and purposelessness and thoughts concerned with suicide and self harm; and an Impulsiveness factor, including items associated with impulsive behaviours which are self-destructive in nature (e.g., alcohol abuse, spending money unwisely), and destructive behaviours directed against others and their property. Each item on the STB has a 'yes' / 'no' response format. Scores can range from 0-18, with higher scores being indicative of greater psychopathology.

The General Health Questionnaire (GHQ-12; Goldberg and Williams, 1988)

The GHQ-12 is a validated 12-item, self-report instrument for the detection of mental disorders in the community and non-psychiatric clinical settings. The emphasis in this questionnaire is on how the respondent's present state differs from his/her usual state. The psychometric literature concerning the GHQ-12 revealed adequate Cronbach's alpha coefficients, ranging from 0.78 to 0.97 (Goldberg et al. 1997; Koeter & Ormel, 1991; Schmitz, Kruse, & Tress, 1999). The GHQ-12 is scored as pathologic in case of a change in an individual's own normal functioning. The questionnaire has the following four-point response scale: 'not at all', 'same as usual', 'rather more than usual', or 'much more than usual'. In this study the bimodal GHQ scoring method (0-0-1-1) was applied, as recommended by Goldberg and Williams (1988). The resulting total scores range from 0 to 12, with higher scores indicating higher probability of mental health problems.

<u>The Eysenck Personality Questionnaire-Revised Short Scale (EPQ-R Short</u> Scale; EPQ-RS, Eysenck and Eysenck, 1991)

This is a 48 item self-report questionnaire. It assesses four dimensions of personality: Extraversion, Neuroticism, Lie (social desirability) and Pyschoticism. The neuroticism and lie scales were used in this study. The concept of neuroticism in part includes emotional lability and reactivity to stress (Roberts and Kendler, 1999; Watson, 2000). It is a stable and enduring trait (Santor et al 1997) that can act as a broad vulnerability for a wide range of distressing disorders (Costa and McCrae, 1992). Eysenck and Eysenck (1991) report that in a normative adult sample the mean neuroticism score was 5.75 (3.46) for males and 5.50 (2.92) for females. The lie scale

measures the tendency to manipulate responses for a desired outcome. It is also associated with the stable personality trait of 'social desirability'. This is the tendency to attempt to present oneself in a socially favourable light to gain approval (Eysenck and Eysenck, 1985; Gudjonsson, 1990b). Eysenck and Eysenck (1991) report that in a normative adult sample the mean score on the lie scale was = 3.58 (2.28) for males, 3.76 (2.38) for females. The reliability estimates of Lie and Neuroticism scales have been found to be satisfactory (Rafnasaaon et al. 2006). The validity of this instrument is discussed in the EPQ-R manual (Eysenck & Eysenck, 1991) with reference to 40 years of development, and a large number of psychometric and experimental studies involving the Eysenck Personality Scales. Each item on the neuroticism and lie scales has a 'yes' / 'no' response format. The scales range from 0 - 12 and higher scores are indicative of greater neuroticism or social desirability respectively.

Life events

A 'yes / no' format was used to determine the occurrence of adverse life events experienced in the previous year (appendix 2). These were: serious illness; accident or injury; caring for someone important to you who has had a serious illness; separation or divorce; serious problem in a relationship or with a family member; moving house; serious problems at work; minor problems at work; serious financial problems; victim of a serious crime; victim of a minor crime; legal difficulties; serious disappointment; continuous worry or stress lasting some months, and the death of a close family member or friend. The total score was the number of items that were reported as having occurred in the previous year.

Childhood events

Four questions were asked about childhood events (appendix 3). These questions were formulated based on the subscales of the Child Abuse and Trauma Scale (Saunders and Becker-Lausen, 1995). Thus, they assessed childhood neglect, childhood abuse (although sexual abuse was not specified) and unreasonable punishment, with higher scores indicating greater adversity during childhood. Participants responded to three of the questions on a 4-point likert scale with the following options; 'not at all', 'mildly' 'sometimes' 'very much'. These questions were:-

As a child did you ever feel unloved or unwanted?

As a child were you ever unreasonably punished?

Did you experience any types of abuse?

The final question, 'how happy was your childhood?' was responded to on a 5-point likert scale ranging from 'very happy' to 'very unhappy'. These items were summed to produce a measure of overall childhood adversity.

Boundary Questionnaire: short version (Kunzendorf, Hartmann, Cohen and Cutler, 1997)

The Boundary Questionnaire measures personality differences in boundary structure. The notion of thick and thin boundaries involves the degree of separateness (thick boundaries) versus connection (thin boundaries) between a broad range of mental functions, processes and entities (Hartman, 1998, p 32; Hartmann, 1989, 1991). The longer 138-item Boundary Questionnaire had a high test-retest and spilt half reliability and has been related to a number of other measures that confirm its validity (Hartmann, 1991). The 18 items of the short form of the Boundaries Questionnaire were selected from the longer 138-item Boundary Questionnaire based on their high face validity and high correlation with total scores on the 138 version. The test retest reliability of the 18 item version was been found to be satisfactory (r = 0.77; p< 0.01; Kunzendorf, Hartmann, Cohen and Cutler, 1997). Furthermore, Funkhouser et al (2001) assessed boundariness at the beginning and end of a 26-week study, and showed a very high test-retest correlation (r=.87).

Responses range from 0-4, where 0 indicates 'not true of me at all' and 4 indicates 'very true of me'. A higher score on this questionnaire refers to overall thinness of boundaries. The BQ-18 is scored by inverting the rating scales on items 5-7 and 16, and then adding the ratings for all the 18 items. Kunzendorf et al (1997) classified thick boundariness as a score of < 30 and thin boundariness as a BQ score of > 44.

Dream questionnaire

Participants were required to make a retrospective estimate of the frequency of their dream recall and the recall of nightmares and night terrors. Nightmares and night terrors were clearly defined. A night terror was defined as 'a sudden awakening in fear, but where you do not remember a dream'. Nightmares were defined as 'a very disturbing dream that you can recall clearly' (appendix 4).

Participants were asked:-

1. How many dreams do you have each week?

2. If you do not have one dream per week how many dreams do you have per month?'

3. How often, if at all, do you experience night terrors?'

. .

4. How many nightmares do you have a month?'

5. If you have nightmares less than once a month, how many do you have per year?

Participants were required to write their own responses for the above questions and were not constrained by any pre determined response format.

In order to obtain an estimation of how often participants were awakened by their nightmares the following question was used:

6. Now, please think about whether you are ever woken up by the emotions or events in your nightmares. Please state in what percentage of your nightmares does the emotion or the events of the nightmare wake you up. (Please circle one of the following)

0% 20% 40% 60% 80% 100% none of them all of them

This question allowed for the calculation of the proportion of nightmares in which the dreamer was awakened and the percentage of nightmares in which the dreamer was not awakened. Hence, comparisons could be made with studies in which researchers used the waking criterion to define nightmares (Zadra and Donderi, 2000), thus differentiating them from bad dreams.

A number of questions were included inquiring about current use of medication and previous history of sleep or psychiatric disorders.

<u>Part 2</u>

Dream and mood diary

A 14-day dream diary was used to provide a prospective measure of dream and nightmare frequency. Sleep diaries have been found to provide reliable estimates of daily sleep parameters comparable with wrist monitoring (Wilson et al, 1998) and electroencephalography (Rogers et al, 1993). The current 14-day diary was based on version used by Fosse, Stickgold and Hobson (2001). An example is shown below:-

Please write your dream report below. For each line of your report, indicate the appearance of any emotions. Use numbers from 1 to 3 to indicate the intensity of the emotions, where 1 is low,2 is medium, and 3 is high. Explain 'other' emotions underneath. Date Time	Anger	Anxiety/fear	Sadness	Shame	Joy/elation	Love/erotic	Surprise	Other

Participants were required to record the content of their dreams in the left hand side of the dream log. The eight columns on the right hand side denote a variety of possible dream emotions. If a particular emotion is experienced during a dream then a number between 1-3 is put in the appropriate column. The numbers signify how intensely the emotion was experienced (where 1 is low, 2 is medium and 3 is high). These numbers are placed opposite the line of the dream report in which the emotion

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was experienced. After recording dream content the following questions were asked about the preceding dream:-

1. Did the emotions or events of the dream wake you up?

2. How pleasant / unpleasant was the dream? Responses to this item were made on a 7-point dream hedonic tone scale (Foulkes et al. 1966) ranging from 1 (very unpleasant) to 7 (very pleasant).

Participants were asked to report if they consumed any alcohol before sleep and approximately how much (see appendix 5 for an example of the daily log).

State mood measures

Participants were also required to complete two Visual Analogue Scales (VAS) every morning and every evening for 14 consecutive days. These concerned 'happy/depressed' and 'relaxed/anxious'. For example, participants were asked to rate how anxious or relaxed they felt by putting a cross on the line overleaf.

very	quite	neither	quite	very
RELAXED				ANXIOUS
e.g. you may feel, serene, untroubled, peaceful, con				ay feel tense, nervous, aky, uneasy

These moods were rated on separate lines because it has been shown that these moods can vary independently (Bowen et al. 2004; Watson, 2000, pp.27, 45).

Instructions to participants

A detailed instruction sheet was provided to participants (appendix 6). This included instructions on completing the dream diary and mood scales. Examples were provided of a rated VAS and a dream entry with explicit commenting on the emotions experienced during the dream. If more than one dream a night was remembered participants reported it used extra sheets provided. Participants were informed that the researchers were interested in all their dreams and not just their nightmares, and were encouraged to report all information they could remember regardless of how unimportant or uneventful it may seem. Participants were reassured that all information would be treated with the utmost confidentiality and sensitivity.

Procedure

Participants responded to various advertisements stating that they would like to take part in the research. A letter was sent to them along with the questionnaires detailed above. A detailed instruction sheet and consent form was enclosed with a stamped addressed envelope. In addition to the information sheet, the contact details of the researchers were made available should there be any further questions. Participants were asked to complete the questionnaires and return them in the stamped addressed envelop provided. On the return of these questionnaires, participants were sent the dream dairies and asked to return them after the 14-day recording period in the stamped addressed envelop provided. This was to try to ensure that the diary was completed over consecutive days.

<u>Design</u>

Between subjects correlations will be conducted between dream / nightmare variables and waking individual difference and psychopathology/well-being variables. Within subjects correlations will also be conducted between dream / nightmare variables, dream emotional tone and waking longitudinal variables.

Chapter 4

Study 1: Personality, psychopathology and <u>nightmares</u>

Results

Descriptive data

The frequency of dreams, nightmares and bad dreams using the retrospective (questionnaire) and prospective (two-week logs) measures are presented in Table 1. The table shows two frequency ratings each for dreams, bad dreams, nightmares and unpleasant dreams on the logs. The one rating allows participants to record more than one dream a night. However, the second prospective ratings of dreams, bad dreams, nightmares and unpleasant dreams includes at most only one dream per night, This is indicated by (14) in the titles of these variables. In this study nightmares were defined as dreams rated as 1 (very unpleasant) or 2 (moderately unpleasant) on the hedonic scale and which awaked the sleeper. Bad dreams were defined as dreams which were rated as 1 (very unpleasant) or 2 (moderately unpleasant) on the hedonic scale but which did not awaken the sleeper. Unpleasant dreams are dreams rated as 1 or 2 on the hedonic scale but that may or may not wake the dreamer, that is, nightmares and bad dreams combined.

The frequency of nightmares, bad dreams and unpleasant dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale were also calculated using the log data, these were referred to as extreme nightmares, extreme bad dreams and extreme unpleasant dreams, and include the letter X or word 'extreme' in their titles. As above, the inclusion of (14) in their terminology means the number of nights with that type of dream is being assessed, those variables without a (14) have the total number of that type of dream being assessed.

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Table 1 - Descriptive data for the retrospective and prospective dream variables.

Data obtained from the two-week logs has been prorated in the following table to

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Frequency measure	Mean (SD)	Range
RDF	32.06 (25.14)	2-160 ³
RNM	2.79 (3.25)	0-15
RBD	2.40 (3.95)	0-22
RUD	5.15 (5.29)	0-22
RNT	0.43 (1.25)	0-9
ET	3.41 (0.83)	1.82 - 6.00
LDF	26.30 (10.46)	4-58
LNF	5.46 (5.50)	0-24
LBD	2.95 (3.07)	0-13
LUD	8.38 (6.61)	0-28
LXNM ¹	3.5 (3.50)	0-15
LXBD ¹	1.3 (0.47)	1-2
LXUD ¹	4.50 (4.0)	0-15
LDF (14) ¹	21.12 (5.89)	4-30.3
LNF (14) ¹	4.98 (4.77)	0-17
LBD (14) ¹	2.90 (3.03)	0-13
LUD (14) ¹	7.34 (5.50)	0-19
LXNM (14) ^{1, 2}	3.3 (3.1)	0-11
LXUD (14) ^{1, 2}	4.2 (3.60)	0-15
LNT	0.89 (0.76)	0-1

Note. RDF = retrospective dream frequency; RNM = retrospective nightmare frequency; RBD = retrospective bad dream frequency, RUD = retrospective unpleasant dream frequency; RNT = retrospective night terror frequency; ET = dream emotional hedonic tone; LDF = log dream frequency; LNF = log nightmare frequency; LBD = log bad dream frequency; LUD = log unpleasant dream frequency; LXNM = log extreme nightmares; LXBD = log extreme bad dreams; LXUD = log extreme unpleasant dreams; LDF (14) = log dream frequency 14; LNF (14) = log nightmare frequency 14; LBD (14) = log bad dream frequency (14); LUD (14) = log unpleasant dream frequency 14; LXNM (14) = log extreme nightmares (14); LXUD (14) = log extreme unpleasant dreams (14), LNT = log night terror frequency.

¹ Frequency variables with an X in their title refer to extreme dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale.

² Frequency variables with (14) in their tiles means that the measure includes at most only one dream per night. Frequency variables without (14) in their title allow for the recording of more than one dream per night.

³ The participant who is recorded as having 160 dreams per month (prorated from their retrospective weekly reported amount) also reported 21 dreams over the 14-day period of the diary. Although their retrospective figure appears to be an overestimate this figure has been left as reported as only rankings of the retrospective dream variables were used in statistical analyses.

Distribution of dream/nightmare/unpleasant dream frequency measures

Table 2 – Table 2 shows the frequency of dreams, nightmares and unpleasant dreams

from diaries.

Frequency	Dreams	Nightmares	Unpleasant	Dreams	Nightmares	Unpleasant
			dreams	$(14)^{1}$	$(14)^{1}$	dreams (14) ¹
0		15	8		15	8
1		9	8		9	9
2	1	8	7	1	9	7
3		8	4		9	4
4		6	6		7	10
5	3	4	6	4	1	6
6	2	1	6	2	3	5
7		2	5	2		3
8	6	1	1	10	3	2
9	3	1	3	7		2
10	9		1	5		
11	2	1		9		
12	7			5		
13	6		1	8		
14	4			3		
15	1					
16	2					
17	4					
18	1					
19	1					400
21	2					
25	1				· · · · · · ·	
27	1					· · · · · · · · · · · · · · · · · · ·

Note: ¹ Frequency variables with (14) in their tiles means that the measure includes at most only one

dream per night. Frequency variables without (14) in their title allow for the recording of more than one dream per night.

Retrospective measures

On the retrospective measure 45 (66%) participants reported one nightmare or more per month, 38 (49%) participants reported one bad dream or more per month and 53 (78%) of participants reported that they experienced at least one unpleasant dream per month.

Prospective measures.

73% (n= 41) of participants had at least one nightmare over the 2-week study period, 67.9% (n= 38) had at least one extreme nightmare, 67.9% (n = 38) had at least one bad dream. Thus, 85.7% (n = 48) reported experiencing at least one unpleasant dream during the two-week study period and 76.8% had at least one extreme unpleasant dream. All participants reported at least two dreams over the two week study period. 30.4% (n =17) reported at least one dream per night and 69.9% (n = 39) reported less than one dream a night. Table 3 - Shapiro-Wilk test statistics confirming that frequency variables are all non-normally distributed and, thus, non-parametric statistics will be used in the following analyses.

	Shapiro-Wilk statistic	df	Р
RUD	0.82	55	<0.01
RNM	0.79	55	<0.01
RBD	0.57	55	<0.01
LUD	0.87	55	<0.01
LUD (14) ¹	0.87	55	<0.01
LNM	0.82	55	<0.01
LNM (14) ¹	0.84	55	<0.01
LBD	0.94	55	0.01
LBD (14) ¹	0.94	55	0.01

Note. RUD = retrospective unpleasant dream frequency; RNM = retrospective nightmare frequency; RBD = retrospective bad dream frequency; LUD = log unpleasant dream frequency; LUD (14) = log unpleasant dream frequency 14; LNM = log nightmare frequency; LNM (14) = log nightmare frequency 14; LBD = log bad dream frequency; LBD (14) = log bad dream frequency (14).

¹ Frequency variables with (14) in their tiles means that the measure includes at most only one dream per night. Frequency variables without (14) in their title allow for the recording of more than one dream per night.

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Table 4 - Spearman's rho correlations between retrospective and prospective

frequency estimates.

	Retrospective	Retrospective	Retrospective unpleasant
	dream frequency	nightmare frequency	dream frequency
Log dream frequency	0.66 (<0.01)		
Log dream frequency (14) ¹	0.56 (<0.01)		
Log nightmare frequency		0.24 (0.07)	
Log nightmare frequency (14) ¹		0.43 (<0.01)	
Log extreme nightmares ²		0.51 (<0.01)	
Log extreme nightmares (14) ^{1,2}		0.52 (<0.01)	
Log unpleasant dream frequency	· · · · · · · · · · · · · · · · · · ·		0.57 (<0.01)
Log unpleasant dream frequency (14)		<u> </u>	0.58 (<0.01)
Log extreme unpleasant dreams ²			0.45 (<0.01)
Log extreme unpleasant dreams (14) ^{1,2}		<u></u>	0.43 (<0.01)

¹ Frequency variables with (14) in their tiles means that the measure includes at most only one dream per night. Frequency variables without (14) in their title allow for the recording of more than one dream per night.

² Frequency variables with 'extreme' in their title refer to extreme dreams defined only as dreams rated

1 (very unpleasant) on the hedonic scale.

 Table 4 shows moderately strong correlations between the retrospective and log

 nightmare and unpleasant dream variables.

Table 5 shows the descriptive data for the individual difference variables.

Table 5 - Descriptive data for the personality variables, and questionnaire and log psychopathology/well-being variables (N=67).

Variable or measure	Mean (SD)	Range
ND	35.18 (9.23)	17-57
APS	36.37 (9.62)	3-53
CEQ	9.94 (4.44)	0-21
STB	8.07 (4.24)	0-17
EPQ-N	7.66 (3.23)	0-12
EPQ-L	3.97 (2.71)	0-12
GHQ-12	3.90 (4.17)	0-12
BQ-18	34.67 (10.13)	13-53
STAI-T	49.78 (13.38)	28-79
STAI-S	47.96 (15.29)	23-77
SCL-GSI	1.19 (0.76)	0.10-3.54
SCL-D	1.44 (0.98)	0 -3.85
SCL-A	1.00 (0.94)	0 - 4
SCL-PST	48.61 (21.26)	8-90
SCL-PSD	1.91 (0.61)	0.85 - 3.58
LE	3.18 (2.49)	0-9
Child	3.98 (3.27)	0-13
Anxiety before sleep on logs	28.90 (11.44)	4.77-64.50
Anxiety after sleep on logs	32.84 (12.36)	6.15-63.46
Depression before sleep on logs	30.38 (11.34)	6.12-65.86
Depression after sleep on logs	33.22 (11.69)	7.54- 63.36

Note: ND = Nightmare distress; APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Eysenck Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-T = Trait anxiety; STAI-S = State anxiety; SCL-GSI = SCL- Global Severity Index; SCL-D = SCL depression, SCL-A = SCL-anxiety; SCL-PST= SCL positive symptom total; SCL-PSD = SCL positive symptom distress; LE = Sum of life events; Child = Sum of childhood items. The sample mean did not differ on BQ score from the student sample of Kunzendorf et al (1997), where 'thick' BQ score was defined as < 30 and 'thin' BQ score was defined as > 44. Using the normative samples of working adults aged 19-39 in Spielberger (1983, Table 2) trait anxiety was higher than for the normative sample (means = 35.55 (9.76) for males, 36.15 (9.53) for females), as was state anxiety (means = 36.54 (10.22) for males, 36.17 (10.96) for females). Using the normative samples in Eysenck and Eysenck (1991), ages 31-40, neuroticism was not higher than for the normative sample (means = 5.75 (3.46) for males; mean = 5.50 (2.92) for females), and the Lie scale did not differ from the normative sample (means = 3.58 (2.28) for males, 3.76 (2.38) for females). Derogatis (1994) suggested guidelines for identifying clinical cases on the SCL-90-R, operationally defined as a t-score on the General Symptom Index >63 or the presence of any two elevated clinical scales >63.

Life events and childhood events

Table 6 shows the number of life events during the previous year reported by participants.

Number of life events	Frequency	Cumulative percent
0	6	8.8
1	18	35.3
2	11	51.5
3	8	63.2
4	3	67.6
5	5	75.0
6	10	89.7
7	2	92.6
8	4	98.5
9	1	100

Table 6 - Frequency of participants reporting the listed life events in the previous year

<u>Note:</u> Listed life events were: Serious illness/injury/accident; Cared for someone who had a serious illness/injury/separation; Separation or divorce; Problems in relationship or with family members; Moved house; Serious problems at work; Minor problems at work; Serious financial problems; Victim of major crime; Victim of minor crime; Legal difficulties; Serious disappointment; Continuous worry /stress/major trouble; Death of family member or friend

Of the individual events, 11 (16.2%) reported a serious illness/accident or injury, 16 (23.3%) of participants reporting caring for someone with a serious illness/injury, 3 (4.4.%) reported experiencing separation or divorce, 26 (38.2%) reported problems with relationships or family members, 17 (25%) had moved house, 9 (13.2%) reported serious problems at work, 25 (36.8%) reported minor problems at work, 16 (23.5%) reported serious financial problems. No participants reported being a serious victim of crime, however, 11 (16.2%) reported being a victim of minor crime, 6 (8.8%) reported legal difficulties, 27 (39.7%) reported serious disappointment, 34 (50%) reported a continuous worry and 15 (22.1%) reported the death of a family member or close friend.

Table 7 reports the distribution of responses on the three questions about childhood.

 Table 7 - Frequency of participant's responses to statements regarding their

 childhood.

	Felt unloved/unwanted as	Were unreasonably	Experienced any
	a child	published	types of abuse
Not at all	31	34	50
Mildly	18	14	8
Sometimes	12	15	6
Very much	6	5	4

The responses to statements on happiness/unhappiness of childhood were:

9 (13.2%) reported that their childhood was 'very unhappy',

8 (11.8%) reported their childhood was 'unhappy',

18 (26.5%) said 'fair',

21 (30.9%) reported a 'happy' childhood and

12 (17.6%) reported a 'very happy' childhood.

Appendix Table 1 shows the intercorrelations between the well-being and

individual difference variables.

Gender and age

Appendix Table 8 shows the correlations and tests of difference for age and gender respectively. The table shows that age is not related to any of the dream type frequencies with the exception of bad dream frequency. Appendix Table 9 shows that there are no significant sex difference on dream frequency variables between males and females. However, there is a trend for females to report more dreams on the logs than males in the current study (Ms = 12.86 and 9.50 respectively over 14 nights). Appendix Tables 10 and 11 shows that there were no sex differences in any of the personality or psychopathology/well-being measures. Appendix Table 12 shows that age only had significant correlations with BQ score (thickening of boundaries with age) and with life events. As gender and age in general have only small relationships with the various measures of dream frequency, personality and psychopathology/well-being, they were not used as covariates in any of the analyses.

Retrospective frequency estimates and psychological well-being and individual differences

The following table shows Spearman's rho correlations between retrospectively measured bad dream, nightmare, unpleasant dream frequency, mean dream emotional tone (measured prospectively) and nightmare distress with measures of well-being and individual differences. In order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at is p is equal to or less than 0.01.

Table 8 - Spearman's rho correlations between retrospective frequency measuresand nightmare distress with psychopathology and personality variables

	RDF	RUD	RNM	RBD	ND	ET
ET	0.01	-0.39	-0.29	-0.30	-0.33	
	(0.92)	(<0.01)	(0.03)	(0.03)	(0.01)	-
ND	0.29*	0.59*	0.50*	0.26		-0.33*
	(<0.01)	(<0.01)	(<0.01)	(0.04)	-	(<0.01)
APS	0.30*	0.33*	0.28	0.31*	0.36*	-0.29
	(0.01)	(0.01)	(0.02)	(0.01)	(<0.01)	(0.03)
CEQ	0.20	0.31*	0.38*	0.19	0.36*	0.07
	(0.10)	(0.01)	(0.01)	(0.12)	(<0.01)	(0.58)
STB	0.10	0.38*	0.29	0.25	0.44*	-0.27
	(0.41)	(<0.01)	(0.02)	(0.04)	(<0.01)	(0.04)
EPQ-N	0.15	0.42*	0.37*	0.36*	0.44*	-0.36*
	(0.23)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.01)
EPQ-L	0.07	0.28	0.11	0.18	0.13	-0.26
	(0.55)	(0.02)	(0.38)	(0.13)	(0.29)	(0.05)
GHQ-12	0.02	0.28	0.24	0.21	0.37*	-0.12
	(0.88)	(0.02)	(0.05)	(0.08)	(<0.01)	(0.37)
BQ-18	0.22	0.27	0.24	0.16	0.35*	-0.10
	(0.07)	(0.03)	(0.05)	(0.20)	(<0.01)	(0.48)
STAI-S	0.01	0.69*	0.48*	0.38*	0.51*	-0.56*
	(0.97)	(<0.01)	(0.01)	(<0.01)	(<0.01)	(<0.01)
STAI-T	0.21	0.57*	0.46*	0.39	0.58*	-0.43*
	(0.14)	(<0.01)	(<0.01)	(0.05)	(<0.01)	(<0.01)
SCL-DEP	0.23	0.54*	0.41*	0.41*	0.33*	-0.27
	(0.08)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)
SCL-ANX	0.28	0.58*	0.47*	0.33*	0.43*	-0.34*
	(0.04)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(0.01)
SCL:GSI	0.10	0.59*	0.48*	0.33*	0.61*	-0.31
	(0.40)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(0.02)
SCL:PST	0.06	0.55*	0.42*	0.40*	0.50*	-0.31
	(0.61)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.02)
SCL:PSD	0.16	0.51*	0.46*	0.22	0.56*	-0.31
	(0.19)	(<0.01)	(<0.01)	(0.08)	(<0.01)	(0.02)
LE	0.10	0.38*	0.37*	0.29*	0.34*	-0.12
	(0.40)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(0.38)
Child	0.07	0.19	0.30*	0.08	0.44*	-0.24
	(0.59)	(0.11)	(0.01)	(0.50)	(<0.01)	(0.08)

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Note (table 8): ${}^{1}RDF$ = retrospective dream frequency; RUD = retrospective unpleasant dream frequency; RNM = retrospective nightmare frequency; RBD = retrospective bad dream frequency, ND = Nightmare distress; ET = dream emotional hedonic tone; APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N =Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety ; STAI-T = Trait anxiety; SCL-DEP = SCL depression, SCL-ANX = SCL-anxiety; SCL:GSI = SCL- Global Severity Index; SCL:PST= SCL positive symptom total; SCL:PSD = positive symptom distress; LE = Sum of life events; Child = Sum of childhood items. * p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed

Table 8 shows that retrospectively measured unpleasant dreams are significantly positively correlated to most measures of well-being with the exception of the GHQ-12 (r = 0.28; p = 0.02), the BQ-12 (r = 0.27; p = 0.03), Eysenck's lie scale (r = 0.28; p = 0.02) and childhood events (r = 0.07; p = 0.11). The latter variable is, however, significantly associated with retrospective nightmare frequency (r = 0.30; p = 0.01). Nightmares are significantly positively correlated with all measures of well-being with the exception of Eysenck's lie scale (r = 0.11; p = 0.38), the GHQ-12 (r = 0.24; p = 0.05) and the BQ-18 (r = 0.24; p = 0.05). Bad dream frequency has less significant correlations than either unpleasant dream frequency or nightmares frequency. It is not significantly correlated to the CEQ, the STB, the GHQ-12, the EPQ-L, the BQ-18, the STAI-T, the PSDI global scale of the SCL-90-R or childhood events. Dream frequency as measured retrospectively is not significantly associated with any of the well-being or individual difference variables, with the exception of the APS (r = 0.30; p = 0.01) and nightmare distress (r = 0.29; p < 0.01). Inspection of table 6 shows that in almost all cases unpleasant dreams, that is nightmares defined without the waking criterion, have higher correlations than do waking criterion nightmares. Furthermore, it can be observed that nightmare frequency has stronger correlations with the psychopathology measures than does bad dream frequency. This table also shows that nightmare distress is significantly positively related to all psychopathology variables (with the exception of Eysenck lie scale). Emotional tone (as measured

prospectively) is also significantly associated with the majority of individual difference and well-being measures. Nightmare distress has higher correlations with psychopathology than do the frequency variables or mean dream emotional tone. As would be expected, nightmare distress and emotional tone are significantly correlated with the frequency of all unpleasant dream types. Thus, partial correlations have been conducted below where nightmare distress has been partialled out of the relationship between frequency measures and psychopathology (table 9) and where frequency measures are partialled out of the relationship between nightmare distress and psychopathology measures (table 10). Note that Kendall correlations and Kendall partial correlations are used: a Kendall correlation has the same p values as a Spearman correlation on the same data, but Spearman's correlations are typically approximately 50% larger. Kendall's correlation is used as it provides the only nonparametric partial correlation. Again in order to reduce the possibility of type 1 errors as a result of multiple comparisons the level of significance was set at p = to or < t0.01.

Table 9 - Kendall's tau_b correlations of retrospective frequency measures with well-being and individual difference variables. The original correlations for each frequency dream type are presented on the left, and next to them on the right are the correlations when nightmare distress has been partialled out. Partialled out variables are indicated after hyphens in the top row.

	RUD	RUD -ND	ŔNM	RNM-ND	RBD	RBD-ND
APS	0.24*	0.14	0.21	0.12	0.23*	0.19
	(0.01)	(>0.05)	(0.02)	(>0.05)	(<0.01)	(<0.05)
CEQ	0.23*	0.13	0.24*	0.15	0.13	0.08
	(0.01)	(>0.05)	(0.01)	(>0.05)	(0.14)	(>0.05)
STB	0.27*	0.15	0.21*	0.10	0.18	0.13
	(<0.01)	(>0.05)	(0.01)	(>0.05)	(0.04)	(>0.05)
EPQ-N	0.32*	0.20	0.26*	0.15	0.26*	0.21*
	(<0.01)	(<0.05)	(<0.01)	(>0.05)	(<0.01)	(<0.01)
EPQ-L	0.18	0.15	0.09	0.06	0.14	0.12
	(0.04)	(>0.05)	(0.32)	(>0.05)	(0.12)	(>0.05)
GHQ-12	0.21	0.10	0.17	0.07	0.16	0.11
	(0.02)	(>0.05)	(0.05)	(>0.05)	(0.07)	(>0.05)
BQ-18	0.19	0.09	0.17	0.08	0.11	0.07
	(0.03)	(>0.05)	(0.05)	(>0.05)	(0.07)	(>0.05)
STAI-T	0.53*	0.44*	0.33*	0.21*	0.28*	0.21*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
STAI-S	0.40*	0.26	0.37*	0.25*	0.26	0.22
	(<0.01)	(<0.05)	(<0.01)	(<0.01)	(0.05)	(>0.05)
SCL-GSI	0.44*	0.31*	0.35*	0.22*	0.25*	0.19
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.05)
SCL-PST	0.40*	0.29*	0.30*	0.19	0.28*	0.23*
	(<0.01)	(<0.01)	(<0.01)	(<0.05)	(<0.01)	(<0.01)
SCL-PSD	0.38*	0.25*	0.35*	0.23*	0.16	0.09
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.06)	(<0.05)
LE	0.30*	0.22*	0.28*	0.20	0.23*	0.19
	(<0.01)	(<0.01)	(<0.01)	(<0.05)	(0.01)	(<0.05)
Child	0.14	-0.01	0.22*	0.11	0.05	-0.01
	(0.11)	(>0.05)	(0.01)	(>0.05)	(0.52)	(>0.05)

Note: RUD = retrospective unpleasant dream frequency; RNM = retrospective nightmare frequency; RBD =

retrospective bad dream frequency; -ND = nightmare distress partialled out; APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-T = Trait anxiety; STAI-S = State anxiety; SCL-GSI = SCL- Global Severity Index; SCL-PST= SCL positive symptom total; SCL-PSD = positive symptom distress; LE = Sum of life events; Child = Sum of childhood items.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

The above table shows that when nightmare distress has been partialled out correlations between nightmare frequency, bad dream frequency and unpleasant dream frequency become smaller. When nightmare distress is partialled out of the relationship between unpleasant dream frequency and well-being measures some of the associations become insignificant with the exceptions of trait anxiety, the three global indices of the SCL-90-R and life events. Similarly, when nightmare distress is partialled out of the relationship between nightmare frequency and psychopathology variables, only trait and state anxiety and two of the global indices of the SCL-90-R remain significant. Most of the previously significant correlations between bad dream frequency with well-being and personality measures remain significant when nightmare distress is partialled out with the exception of the arousal predisposition scale (APS), the global symptom index of the SCL-90-R and life events. Thus, it seems that retrospective frequency variables are associated with some of the wellbeing measures and individual difference variables independently of nightmare distress although all associations become smaller and some non significant when distress is controlled for. Again, unpleasant dream frequency has the strongest correlations with psychopathology when nightmare distress is partialled out.

Table 10 shows Kendall's tau_b correlations between nightmare distress and measures of well-being and personality, and the correlations when the various types of retrospective frequency measures are partialled out.

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Table 10 - Kendall tau_b correlations of nightmare distress variables with wellbeing and individual difference measures when retrospective frequency measures are partialled out. The original correlations with nightmare distress are presented on the left, and next to them on the right are the correlations when frequency variables are partialled out. Partialled out variables are indicated after hyphens in the top row.

- <u> </u>	ND	ND- RUDF	ND-RNM	ND-RBD
APS	0.27*	0.19	0.21*	0.24*
	(<0.01)	(<0.05)	(<0.01)	(<0.01)
CEQ	0.27*	0.19	0.20*	0.25*
	(<0.01)	(<0.05)	(<0.01)	(<0.01)
STB	0.32*	0.23*	0.26*	0.30*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
EPQ-N	0.34*	0.23*	0.27*	0.31*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
EPQ-L	0.10	0.02	0.07	0.08
	(0.27)	(>0.05)	(>0.05)	(>0.05)
GHQ-12	0.28*	0.21*	0.24*	0.26*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
BQ-18	0.25*	0.19	0.20	0.23*
	(<0.01)	(<0.05)	(<0.05)	(<0.01)
STAI-T	0.38*	0.19	0.29*	0.35*
	(<0.01)	(<0.05)	(<0.01)	(<0.01)
STAI-S	0.42*	0.30*	0.32*	0.39*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
SCL-GSI	0.44*	0.31*	0.35*	0.41*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
SCL-PST	0.35*	0.21*	0.27*	0.31*
	. (<0.01)	(<0.01)	(<0.01)	(<0.01)
SCL-PSDI	0.40*	0.28*	0.31*	0.38*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
LE	0.25*	0.14	0.16	0.22*
	(<0.01)	(>0.05)	(>0.05)	(<0.01)
Child	0.33*	0.30*	0.27*	0.33*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)

Note: ND = nightmare distress; -RUD = retrospective unpleasant dreams partialled out; -RNM = retrospective

nightmare frequency partialled out; -RBD = bad dream frequency partialled out; APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L =Eysenck Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-T = Trait anxiety; STAI-S = State anxiety; SCL-GSI = SCL- Global Severity Index; SCL-PST= SCL positive symptom total; SCL-PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items. * p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

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Inspection of the table overleaf shows that most of the relationships between nightmare distress and the well-being and personality variables remain significant when frequency variables are partialled out, with the expectation of correlations with life events and boundariness which became insignificant when retrospective unpleasant dream frequency and nightmare frequency are partialled out. Also, the APS, CEQ, trait and state anxiety become insignificant when unpleasant dream frequency is partialled out

Table 11 presents correlations of retrospective frequency measures when the mean emotional tone of the dream is partialled out.

Table 11 - Kendall's tau_b correlations of retrospective frequency measures with wellbeing and individual difference variables. The original correlations are for each frequency dream type are presented on the left and next to them are the correlations when mean dream emotional tone has been partialled out. Partialled out variables are indicated after hyphens in the top row

	RUD	RUD -ET	RNM	RNM-ET	RBD	RBD-ET
APS	0.24*	0.20	0.21	0.18	0.23*	0.20
	(0.01)	(<0.05)	(0.02)	(<0.05)	(0.01)	(<0.05)
CEQ	0.23*	0.23*	0.24*	0.24*	0.13	0.12
	(0.01)	(<0.01)	(<0.01)	(<0.01)	(0.14)	(>0.05)
STB	0.27*	0.23*	0.21*	0.18	0.18	0.15
	(<0.01)	(<0.01)	(0.01)	(<0.05)	(0.04)	(>0.05)
EPQ-N	0.32*	0.27*	0.27*	0.22*	0.26*	0.22*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
EPQ-L	0.18	0.14	0.09	0.06	0.14	0.11
	(0.04)	(>0.05)	(0.32)	(<0.05)	(0.12)	(>0.05)
GHQ-12	0.21	0.19	0.17	0.16	0.16	0.15
	(0.02)	(<0.05)	(0.05)	(>0.05)	(0.07)	(>0.05)
BQ-18	0.19	0.18	0.17	0.16	0.11	0.10
	(0.03)	(<0.05)	(0.05)	(<0.05)	(0.20)	(>0.05)
STAI-S	0.53*	0.48*	0.39*	0.34*	0.26	0.20
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.05)	(<0.05)
STAI-T	0.40*	0.34*	0.33*	0.28*	0.28*	0.23*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
SCL-ANX	0.40*	0.37*	0.30*	0.27*	0.30*	0.27*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
SCL-DEP	0.43*	0.39*	0.34*	0.31*	0.24*	0.21*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.01)
GSI	0.59*	0.57*	0.35*	0.32*	0.25*	0.22*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
PSI	0.55*	0.53*	0.30*	0.27*	0.28*	0.25*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
PSDI	0.51*	0.48*	0.35*	0.32*	0.16	0.12
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.06)	(>0.05)
LE	0.30*	0.29*	0.28*	0.27*	0.23*	0.22*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.01)
Child	0.14	0.10	0.22*	0.19	0.06	0.02
	(0.11)	(>0.01)	(0.01)	(<0.05)	(0.52)	(>0.05)

Note ¹ RUD = retrospective unpleasant dream frequency; RNM = retrospective nightmare frequency; RBD = retrospective bad dream frequency; -ET = mean dream emotional tone partialled out; APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-T = Trait anxiety; STAI-S = State anxiety; SCL-ANX = SCL-anxiety SCL-DEP = SCL depression; SCL-GSI = SCL- Global Severity Index; SCL-PST= SCL positive symptom total; SCL-PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items.

*p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed

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Inspection of table 11 shows that partialling out mean dream emotional tone does not greatly alter the size of the correlations with retrospective frequency measures. All previously significant correlations between retrospective unpleasant dream frequency and measures of well-being and individual differences remain when emotional tone is partialled out with the exception of the arousal predisposition scale (APS). Similarly all previously significant correlations with nightmare frequency remain significant when emotional tone is partialled out with the exception of the STB. The majority of bad dream correlations also remain significant with the exception of the APS . Thus, it seems to be that retrospective nightmare and unpleasant dream frequencies, rather than the average mood of all dreams, are significantly related to individual differences variables and well-being.

The following table shows the relationships between emotional tone and measures of individual differences and well-being when retrospective frequency variables are partialled out. Table 12 - Kendall tau b correlations of mean dream emotional tone variables with well-being and individual difference measures when retrospective frequency measures are partialled out. Partialled out variables are indicated after hyphens in the top row.

	ET	ET- RUD	ET-RNM	ET-RBD
ADC		-0.16	-0.18	-0.17
APS	-0.21			
	(0.03)	(>0.05)	(>0.05)	(>0.05)
CEQ	-0.05	0.01	0.01	-0.03
	(0.63)	(>0.05)	(>0.05)	(>0.05)
STB	-0.19	-0.13	-0.15	-0.16
	(0.04)	(>0.05)	(>0.05)	(>0.05)
EPQ-N	-0.25*	-0.18	-0.21*	-0.21*
	(0.01)	(>0.05)	(<0.01)	(<0.01)
EPQ-L	-0.18	-0.14	-0.17	-0.16
	(0.06)	(>0.05)	(>0.05)	(>0.05)
GHQ-12	-0.09	-0.04	-0.06	-0.06
	(0.36)	(>0.05)	(>0.05)	(>0.05)
BQ-18	-0.07	-0.02	-0.04	-0.05
	(0.48)	(>0.05)	(>0.05)	(>0.05)
STAI-S	-0.42*	-0.34*	-0.38*	-0.39*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
STAI-T	-0.34*	-0.27*	-0.30*	-0.30*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)
SCL-DEP	-0.19	-0.10	-0.14	-0.14
	(0.04)	(>0.05)	(>0.05)	(>0.05)
SCL-ANX	-0.23*	-0.14	-0.18	-0.19
	(0.01)	(>0.05)	(>0.05)	(<0.05)
SCL-GSI	-0.19	-0.05	-0.13	-0.15
	(0.04)	(>0.05)	(>0.05)	(>0.05)
SCL-PSI	-0.19	-0.06	-0.14	-0.15
	(0.04)	(>0.05)	(>0.05)	(>0.05)
SCL-PSDI	-0.22	-0.11	-0.16	-0.20
	(0.02)	(>0.05)	(>0.05)	(<0.05)
LE	-0.10	-0.02	-0.05	-0.06
	(0.32)	(>0.05)	(>0.05)	(>0.05)
Child	-0.18	-0.15	-0.14	-0.17
	(0.06)	(>0.05)	(>0.05)	(>0.05)
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Note. ET = mean dream emotional tone; -RUD = retrospective unpleasant dream frequency partialled out; - RNM = retrospective nightmare frequency partialled out; -RBD = retrospective bad dream frequency partialled out; ; APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-T = Trait anxiety; STAI-S = State anxiety; SCL-ANX = SCL-anxiety; SCL-DEP = SCL depression, SCL-GSI = SCL- Global Severity Index; SCL-PST= SCL positive symptom total; SCL-PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of

significance was set at p = <0.01). All values are two tailed.

Table 12 shows that when the strongest correlate of well-being, retrospective unpleasant dream frequency, is partialled out of the relationship being well-being and emotional tone all correlations become insignificant, with the exception of trait and state anxiety. Trait and state anxiety as well as neuroticism remain significantly associated with mean dream tone when retrospective nightmare frequency is partialled out. Thus, some variables, particularly trait and state anxiety appear to be related to the emotional tone of all dreams, although their correlations with unpleasant dream types are higher.

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Correlations between prospective nightmare and unpleasant dream measures and

individual difference variables.

Table 13 -Spearman's rho correlations between prospective frequency measures and

psychopathology variables.

	LDF	LUD	LUD	LXUD'	LXUD	LNM	LNM	LXNM	LXNM	BD	BD	LXBD ¹
			(14) ²		(14) ^{1,2}		(14)2	1	(14) ^{1,2}		(14)2	
ND	0.50*	0.51*	0.47*	0.42*	0.40*	0.49*	0.47*	0.40*	0.42*	0.38*	0.37*	0.17
	(<.01)	(0.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.22)
APS	0.40*	0.35*	0.31	0.39*	0.40*	0.32*	0.32*	0.35*	0.35*	0.22	0.21	0.18
	(<.01)	(0.01)	(0.02)	(<.01)	(<.01)	(0.01)	(0.01)	(0.01)	(<.01)	(0.12)	(0.13)	(0.19)
CEQ	0.26	0.20	0.17	0.31	0.31	0.12	0.11	0.25	0.25	0.29	0.28	0.20
	(0.05)	(0.14)	(0.20)	(0.02)	(0.02)	(0.40)	(0.42)	(0.06)	(0.06)	(0.03)	(0.04)	(0.14)
STB	0.25	0.29	0.27	0.40*	0.38*	0.25	0.22	0.27	0.30	0.29	0.29	0.36*
	(0.07)	(0.03)	(0.04)	(<.01)	(<.01)	(0.06)	(0.10)	(0.04)	(0.02)	(0.03)	(0.03)	(0.01)
EPQ-N	0.46*	0.46*	0.43*	0.50*	0.48*	0.49*	0.47*	0.43*	0.45*	0.24	0.24	0.22
	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.08)	(0.08)	(0.11)
EPQ-L	0.14	0.41*	0.41*	0.44*	0.44*	0.48*	0.49*	0.50*	0.49*	0.14	0.13	0.06
	(0.30)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.32)	(0.33)	(0.66)
GHQ-12	0.20	0.13	0.12	0.20	0.17	0.14	0.12	0.09	0.12	0.19	0.20	0.21
	(0.14)	(0.33)	(0.36)	(0.14)	(0.21)	(0.31)	(0.39)	(0.49)	(0.38)	(0.15)	(0.14)	(0.12)
BQ-18	0.18	0.18	0.17	0.29	0.26	0.09	0.07	0.13	0.16	0.34*	0.34*	0.35*
	(0.19)	(0.19)	(0.21)	(0.03)	(0.05)	(0.50)	(0.60)	(0.34)	(0.23)	(0.01)	(0.01)	(0.01)
STAI-S	0.07	0.41	0.41	0.58*	0.55*	0.42	0.41	0.48*	0.49*	0.22	0.22	0.33
	(0.72)	(0.04)	(0.04)	(<.01)	(<.01)	(0.03)	(0.04)	(0.01)	(0.01)	(0.28)	(0.28)	(0.11)
STAI-T	0.39*	0.43*	0.40*	0.50*	0.47*	0.39*	0.36*	0.39*	0.42*	0.38*	0.38*	0.41*
	(0.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.01)	(0.01)	(0.01)	(<.01)	(0.01)	(0.01)	(<.01)
SCL:GSI	0.30	.0.42*	0.39*	0.48*	0.45*	0.41*	0.37*	0.37*	0.40*	0.33*	0.34*	0.36*
	(0.03)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.01)	(0.01)	(0.01)
SCL:PST	0.24	0.36*	0.35*	0.47*	0.44*	0.39*	0.36*	0.38*	0.42*	0.20*	0.28	0.30
	(0.08)	(<.01)	(0.01)	(<.01)	(<.01)	(<.01)	(0.01)	(<.01)	(<.01)	(0.04)	(0.04)	(0.02)
SCL:PSDI	0.31	0.41*	0.39*	0.46*	0.43*	0.36*	0.33*	0.32	0.36*	0.39*	0.39*	0.32
	(0.02)	(<.01)	(0.01)	(<.01)	(<.01)	. (0.01).	(0.01)	(0.02)	(0.01)	(<.01)	(<.01)	(0.02)
SCL-DEP	0.23	0.31	0.27	0.40*	0.40*	0.31	0.27	0.32	0.35*	0.28	0.28	0.29
	(0.08)	(0.02)	(0.04)	(<.01)	(<.01)	(0.02)	(0.05)	(0.02)	(0.01)	(0.04)	(0.04)	(0.03)
SCL-ANX	0.28	0.42*	0.39*	0.54*	0.51*	0.43*	0.40*	0.45*	0.48*	0.26	0.27	0.36*
	(0.04)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.05)	(0.04)	(0.01)
LE	0.11	0.12	0.12	0.14	0.14	0.14	0.14	0.14	0.14	0.10	0.11	0.20
	(0.40)	(0.36)	(0.36)	(0.30)	(0.29)	(0.29)	(0.31)	(0.31)	(0.31)	(0.45)	(0.43)	(0.13)
Child	0.30	0.30	0.28	0.18	0.16	0.20	0.17	0.15	0.17	0.38	0.37*	0.09
	(0.03)	(0.03)	(0.03)	(0.16)	(0.22)	(0.14)	(0.20)	(0.28)	(0.20)	(0.04)	(<.01)	(0.49)
L												

Note (table 13): LDF = log dream frequency; LUD = log unpleasant dream frequency; LUD (14) = log unpleasant dream frequency 14; LXUD = log extreme unpleasant dreams; LXNM (14) = log extreme nightmares (14); LNM = log nightmare frequency; LNM (14) = log nightmare frequency (14); LXNM = log extreme nightmares; LXNM (14) = log extreme nightmares (14); LBD = log bad dream frequency; LBD (14) = log bad dream frequency (14); LXBD = log extreme bad dream; ND = Nightmare distress; APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Eysenck Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-T = Trait anxiety; STAI-S = State anxiety; SCL-GSI = SCL- Global Severity Index; SCL-PST= SCL positive symptom total; SCL-PSDI = positive symptom distress index; SCL-DEP = SCL depression, SCL-ANX = SCLanxiety LE = Sum of life events; Child = Sum of childhood items. * p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of

p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

¹ Frequency variables with 'X' in their title refer to extreme dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale.

² Frequency variables with (14) in their tiles means that the measure includes at most only one dream per night. Frequency variables without (14) in their title allow for the recording of more than one dream per night.

Table 13 shows the correlations between the prospective nightmare frequency estimates and the psychopathology variables. The table shows that:

- a) For nightmares and unpleasant dreams defined as very unpleasant (extreme), or defined as very or moderately unpleasant, correlations with the frequency measures are generally higher than with the number of nights measure which only counted one nightmare or unpleasant dream per night.
- b) For the very unpleasant dream definition of nightmares and unpleasant dreams, unpleasant dream frequency correlations were generally higher than correlations with nightmares, but these correlations were more similar if the very or moderately unpleasant definition was used. Furthermore, when the very unpleasant definition was used nightmares generally had higher correlations than bad dreams, but these correlations were similar when the very and moderately unpleasant criteria was used.
- c) In general, correlations tended to be higher with the very unpleasant rather than the very or moderately unpleasant criterion of nightmares or unpleasant dreams.

- d) In comparison with Table 8, the prospective dream frequency correlations were almost all larger than the retrospective DF correlations (14 larger, 1 smaller, 2 the same; Wilcoxon signed ranks test, z = 3.36, p=.001). The prospective NF and UD correlations were in general smaller than for the retrospective measures (3 larger, 11 smaller, 3 the same; 4 larger, 9 smaller, 4 the same, respectively). There was a difference between retrospective and prospective correlations for nightmare frequency (z = -2.14, p=.033) but not for unpleasant dreams (z = -1.39).
- e) Life events and adverse childhood mainly had only small or non-significant correlations with nightmare / unpleasant dream frequencies.

Partial correlations of nightmare distress and nightmare frequency with individual difference variables

Nightmare distress and nightmare/unpleasant dream frequencies are correlated with each other as well as with many of the individual difference variables. Partial correlations were thus conducted to ascertain the variance in individual difference measures attributable to each of these nightmare measures.

As the frequency definitions only including 'very unpleasant' dreams had the strongest relationships with measures of well-being and personality, these were assessed first in the following partial correlations. The following partial correlations used frequency measures, which allowed more than one nightmare / unpleasant dream per night to be recorded (little difference was found between these and measures onlycounting one dream per night).

Table 14 shows Kendall's tau_b correlation's of log frequency measures (only those dream variable rated as very unpleasant on the hedonic scale) with psychological well-being and individual difference measures before and after nightmare distress is partialled out. Table 14 - Kendall's tau_b correlations between nightmare/unpleasant dream /bad dream frequency variables (only those rated as very unpleasant on the hedonic scale) and individual difference measures before and after nightmare distress and emotional tone have been partialled out. Partialled out variables are indicated after hyphens in the top row. Only dreams rated as very unpleasant are classed as nightmares/unpleasant dreams/bad dreams in this analysis.

	LXNM	LXNM- ET	LXNM- ND	LXUD	LXUD- ET	LXUD- ND	LXBD	LXBD- ET	LXBD- ND
APS	0.26*	0.18	0.19	0.30*	0.23*	0.23*	0.13	0.07	0.10
	(0.01)	(<0.05)	(<0.05)	(<0.01)	(<0.01)	(<0.01)	(0.24)	(>0.05)	(0.05)
CEQ	0.18	0.18	0.11	0.22	0.25*	0.15	0.15	0.14	0.12
. <u> </u>	(0.08)	(>0.05)	(>0.05)	(0.03)	(<0.01)	(>0.05)	(0.18)	(>0.05)	(>0.05)
STB	0.21	0.13	0.12	0.31*	0.25*	0.23*	0.27*	0.23*	0.24*
	(0.04)	(>0.05)	(>0.05)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(<0.01)
EPQ-N	0.35*	0.27*	0.27*	0.38*	0.30*	0.30*	0.17	0.10	0.13
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.13)	(>0.05)	(>0.05)
EPQ-L	0.40*	0.36*	0.42*	0.34*	0.29*	0.36*	0.05	0.00	0.05
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.65)	(>0.05)	(>0.05)
GHQ-12	0.08	0.04	-0.01	0.16	0.13	0.08	0.16	0.14	0.13
	(0.46)	(>0.05)	(>0.05)	(0.11)	(>0.05)	(>0.05)	(0.16)	(>0.05)	(>0.05)
BQ-18	0.09	0.07	0.01	0.20	0.20	0.13	0.26	0.25*	0.24*
	(0.36)	(>0.05)	(>0.05)	(0.05)	(<0.05)	(>0.05)	(0.17)	(<0.01)	(<0.01)
STAI-S	0.38*	0.21	0.30*	0.43*	0.26*	0.35*	0.28	0.18	0.25*
	(0.01)	(<0.05)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.09)	(>0.05)	(<0.01)
STAI-T	0.30*	0.16	0.20*	0.37*	0.23*	0.27*	0.32*	0.24*	0.29*
	_(0.01)	(>0.05)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(<0.01)
SOM	0.29*	0.23*	0.21*	0.31*	0.25*	0.23*	0.10	0.05	0.06
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.34)	(>0.05)	(>0.05)
OCS	0.16	0.05	0.06	0.28*	0.18	0.20	0.27*	0.21*	0.24*
	(0.12)	(>0.05)	(>0.05)	(0.01)	(>0.05)	(<0.05)	(0.01)	(<0.01)	(<0.01)
IS	0.22	0.10	0.14	0.31*	0.20	0.23*	0.33*	0.27*	0.31*
	(0.03)	(>0.05)	(>0.05)	(<0.01)	(<0.05)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
DEP	0.24	0.17	0.15	0.33*	0.28*	0.25*	0.29	0.25*	0.26*
	(0.02)	(>0.05)	(>0.05)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(<0.01)
ANX	0.35*	0.28*	0.25*	0.42*	0.36*	0.33*	0.23	0.17	0.19
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(>0.01)	(0.04)	(>0.05)	(<0.05)
HOS	0.14	0.06	0.03	0.22	0.15	0.12	0.25	0.21	0.22*
	(0.19)	(>0.05)	(>0.05)	(0.03)	(>0.05)	(>0.05)	(0.02)	(<0.01)	(<0.01)
PhA	0.24	0.21*	0.13	0.34*	0.33*	0.24*	0.32*	0.30*	0.29*
	(0.02)	(<0.01)	(>0.05)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(<0.01)
PI	0.19	0.11	0.09	0.25*	0.18	0.15	0.23	0.18	0.20
	(0.07)	(>0.05)	(>0.05)	(0.01) -	(>0.05)	(>0.05)	(0.04)	- (>0.05)-	(<0.05)
Psy	0.25	0.23*	0.17	0.31*	0.31*	0.23*	0.22	0.20	0.19
	(0.02)	(<0.01)	(>0.05)	(<0.01)	(<0.01)	(<0.01)	(0.05)	(<0.05)	(<0.05)
GSI	0.28*	0.22*	0.17	0.37*	0.32*	0.27*	0.26	0.22*	0.23*
	(0.01)	(<0.01)	(>0.05)	(<0.01)	(<0.01)	(<0.01)	(0.02)	(<0.01)	(<0.01)
PST	0.28*	0.22*	0.19	0.35*	0.30*	0.27*	0.22	0.17	0.19
· · · · · · · · · · · · · · · · · · ·	(0.01)	(<0.01)	(<0.05)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(>0.05)	(<0.05)
PSDI	0.25*	0.16	0.14	0.36*	0.29*	0.26*	0.27*	0.22*	0.24*
	(0.01)	(>0.05)	(>0.05)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.01)	(<0.01)
LE	0.11	0.07	0.04	0.11	0.07	0.03	0.16	0.14	0.13
	(0.32)	(>0.05)	(>0.05)	(0.30)	(>0.05)	(>0.05)	(0.16)	(>0.05)	(>0.05)
	0.10	0.00	0.00		0.06	0.05	0.07	0.02	
Child	0.12	0.03	0.02	0.15	0.00	0.05	0.07	0.02	0.03

Note (table 14): LXNM = log extreme nightmares; LXUD = log extreme unpleasant dreams; LXBD = log extreme bad dream; -ET = mean dream emotional tone partialled out; -ND = nightmare distress partialled out. APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Eysenck Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety; STAI-T = Trait anxiety; SOM = SCL- somatisation; OCS = SCL- obsessive compulsive scale; IS = SCL- interpersonal sensitivity scale; DEP = SCL- depression; ANX =SCLanxiety; HOS = SCL- hostility; PhA =SCL-phobic anxiety; PI =SCL-paranoia; Psy = SCL-psychoticism; GSI = SCL- Global Symptom Index; PST= SCL- positive symptom total; PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items.

Frequency variables with 'X' in their title refer to extreme dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Table 14 shows that when nightmare distress is partialled out the log frequency measures defined as very unpleasant become smaller. When nightmare distress is partialled out of the relationships of unpleasant dream frequency with wellbeing and psychopathology all previously significant correlations remain, with the exception of the obsessive compulsive and the paranoid ideation scales of the SCL-90-R. This suggests that it is these extreme unpleasant dreams that are diagnostic of poor well-being rather than the mean emotion of all dreams. When nightmare distress is partialled out the previously significant relationships of nightmare frequency with the arousal predisposition scale and the three global scales of the SCL-90-R become insignificant. All significant correlations with bad dream frequency remain when nightmare distress is partialled out. Thus, these findings confirm the intuitive notion that poor waking well-being and some personality traits increase the frequency of unpleasant dream types and, furthermore, that this is independent of the distress associated with these dream types.

Partialling out mean dream tone seems to have a smaller effect on correlation sizes than does partialling out nightmare distress. When mean dream emotional tone is partialled out of the relationship between unpleasant dream frequency with wellbeing and individual differences all previously significant correlations remain

significant with the exception of the obsessive-compulsive, interpersonal sensitivity and paranoid ideation scales of the SCL-90-R. When mean dream emotion is partialled out of the relationship between nightmare frequency and bad dream frequency a few more previously significant correlations become insignificant. The following table shows the relationships of nightmare distress and mean dream tone when log extreme frequency measures are partialled out. Table 15 - Kendall's tau_b correlations of well-being and individual difference variables with nightmare distress and emotional tone when prospective frequency variables are partialled out. Only dreams rated as very unpleasant were classed as nightmares/unpleasant dreams/bad dreams for use in partialling out in this analysis.

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	ND	ND-	ND-	ND-	ET	ET-	ET-	ET-XBD
4.00	0.07*	XNM	XUD	XBD	0.01	XNM	XUD	0.10
APS	0.27*	0.21*	0.19	0.26*	-0.21	-0.09	-0.06	-0.18
	(<0.01)	(<0.01)	(<0.05)	(<0.01)	(0.03)	(>0.05)	(>0.05)	(>0.05)
CEQ	0.27*	0.23*	0.22*	0.26*	-0.05	0.05	0.09	-0.01
OTD	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.63)	(>0.05)	(>0.05)	(>0.05)
STB	0.32*	0.27*	0.25*	0.30*	-0.19	-0.10	-0.02	-0.12
EDO N	<u>(<0.01)</u> 0.34*	<u>(<0.01)</u> 0.26*	<u>(<0.01)</u> 0.25*	(<0.01) 0.33*	(0.04)	(>0.05)	(>0.05)	(>0.05)
EPQ-N			0.25* (<0.01)		-0.25*	-0.09 (>0.05)	-0.05	-0.21
EPQ-L	<u>(<0.01)</u> 0.01	<u>(<0.01)</u> -0.13	-0.11	(<0.01) 0.00	(0.01) -0.18	0.03	<u>(>0.05)</u> 0.01	<u>(<0.05)</u> -0.17
ErQ-L	(0.27)	-0.13 (>0.05)	-0.11 (>0.05)	(>0.00	-0.18 (0.06)	0.03 (>0.05)	(>0.01 (>0.05)	-0.17 (>0.05)
GHQ-12	0.28*	0.27*	0.24*	0.26*	-0.09	-0.06	0.00	-0.04
011Q-12	0.28* (<0.01)	(<0.01)	(<0.01)	0.26* (<0.01)	(0.36)	-0.08 (>0.05)	0.00 (>0.05)	-0.04 (>0.05)
BQ-12	0.25*	0.23*	0.20*	0.23*	-0.07	-0.02	0.06	0.01
JQ-12	(<0.01)	(<0.01)	(<0.01)	(<0.01)	-0.07 (0.48)	-0.02 (>0.05)	0.00 (>0.05)	(>0.01
STAI-S	0.38*	0.30*	0.28*	0.36*	-0.42*	-0.28*	-0.24	-0.37
UTAPU	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(>0.01)	(>0.24	(<0.01)
STAI-T	0.42*	0.36*	0.34*	0.40*	-0.34*	-0.23*	-0.18	-0.27*
~ I / LA A	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(>0.05)	(<0.01)
SOM	0.31*	0.24*	0.23*	0.30*	-0.19	-0.05	-0.02	-0.17
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(>0.05)	(>0.02	(>0.05)
OCS	0.33*	0.30*	0.26*	0.31*	-0.23*	-0.19	-0.11	-0.17
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(<0.05)	(>0.05)	(>0.05)
IS	0.31*	0.26*	0.23*	0.29*	-0.19	-0.19	-0.13	-0.19
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(<0.05)	(>0.05)	(<0.05)
DEP	0.33*	0.28*	0.25*	0.31*	-0.19	-0.08	-0.01	-0.11
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(>0.05)	(>0.05)	(>0.05)
ANX	0.43*	0.36*	0.34*	0.41*	-0.22	-0.06	0.00	-0.17
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.02)	(>0.05)	(>0.05)	(>0.05)
HOS	0.37*	0.35*	0.32*	0.35*	-0.10	-0.13	-0.07	-0.11
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.32)	(>0.05)	(>0.05)	(>0.05)
PhA	0.40*	0.35*	0.33*	0.38*	-0.18	0.00	0.09	-0.03
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.06)	(>0.05)	(>0.05)	(>0.05)
PI	0.37*	0.33*	0.32*	0.35*	-0.21	-0.11	-0.06	-0.13
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.03)	(>0.05)	(>0.05)	(>0.05)
Psy	0.32*	0.26*	0.25*	0.30*	-0.05	0.03	0.09	-0.04
·····	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.63)	(>0.05)	(>0.05)	(>0.05)
GSI	0.44*	0.39*	0.37*	0.42*	-0.19	-0.06	0.02	-0.12
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(>0.05)	(>0.05)	(>0.05)
PST	0.35*	0.29*	0.27*	0.33*	-0.25*	-0.06	0.00	-0.13
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(>0.05)	(>0.05)	(>0.05)
PSDI	0.40*	0.35*	0.32*	0.38*	-0.18	-0.11	-0.03	-0.15
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.06)	(>0.05)	(>0.05)	(>0.05)
LE	0.25*	0.23*	0.23*	0.23*	-0.09	-0.05	-0.05	-0.06
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.36)	(>0.05)	(>0.05)	(>0.05)
Child	0.33*	0.31*	0.30*	0.32*	-0.07	-0.14	-0.12	-0.17
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.48)	(>0.05)	(>0.05)	(>0.05)

Note (table 15): ND= nightmare distress; ET =mean dream emotional tone; -LXNM = log extreme nightmares partialled out; -LXUD = log extreme unpleasant dreams partialled out; -LXBD = log extreme bad dream partialled out

APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Eysenck Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety; STAI-T = Trait anxiety; SOM = SCL- somatisation; OCS = SCL- obsessive compulsive scale; IS = SCL- interpersonal sensitivity scale; DEP = SCL- depression; ANX = SCLanxiety; HOS = SCL- hostility; PhA = SCL-phobic anxiety; PI = SCL-paranoia; Psy = SCL-psychoticism; GSI = SCL- Global Symptom Index; PST= SCL- positive symptom total; PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items.

Frequency variables with 'X' in their title refer to extreme dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Table 15 shows that all relationships of individual difference variables with nightmare distress remain significantly correlated when log extreme frequency measures are partialled out.

Table 15 also shows that correlations of well-being and individual differences with emotional tone generally become much smaller when log frequency measures are partialled out. When unpleasant dream frequency is partialled out of the relationships of mean dream tone with well-being and individual difference measures, all correlations become (or remain) insignificant. When nightmare frequency and bad dream frequencies are partialled out nearly all correlations become (or remain) insignificant with the exception of trait anxiety.

The above partial correlations were also conducted for log frequency measures, using a criterion of including 'moderately unpleasant' and 'very unpleasant' dreams, in order to quantify the effects of different definitions of nightmares. The results are shown in Table 16. Table 16 - Kendall's tau_b correlations of frequency variables (using criterion of very unpleasant and moderately unpleasant dreams) with well-being and individual difference measures, before and after nightmare distress and emotional tone have been partialled out. Partialled out variables are indicated after hyphens in the top row.

	LUD	LUD –	LUD –	LNM	LNM-	LNM-	LBD	LBD-	LBD-
		ND	ET		ND	ET		ND	ET
APS	0.25*	0.17	0.15	0.22	0.13	0.11	0.15	0.08	0.09
	(0.01)	(>0.05)	(>0.05)	(0.02)	(>0.05)	(<0.05)	(0.13)	(>0.05)	(>0.05)
CEQ	0.13	0.03	0.12	0.07	-0.03	0.05	0.22	0.16	0.22
	(0.19)	(>0.05)	(>0.05)	(0.46)	(>0.05)	(>0.05)	(0.04)	(>0.05)	(<0.01)
STB	0.20	0.09	0.11	0.18	0.07	0.07	0.22	0.14	0.17
	(0.04)	(>0.05)	(>0.05)	(0.08)	(<0.05)	(>0.05)	(0.04)	(>0.05)	(>0.05)
EPQ-N	0.34*	0.24*	0.25*	0.38*	0.29*	0.30*	0.19	0.11	0.12
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.07)	(>0.05)	(>0.05)
EPQ-L	0.31*	0.30*	0.26*	0.37*	0.36*	0.34*	0.10	0.08	0.04
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.32)	(>0.05)	(>0.05)
GHQ-12	0.10	-0.01	0.06	0.11	0.01	0.07	0.14	0.07	0.12
	(0.32)	(>0.05)	(>0.05)	(0.30)	(>0.05)	(>0.05)	(0.18)	(>0.05)	(>0.05)
BQ-18	0.13	0.04	0.09	0.06	-0.04	0.02	0.26*	0.20	0.25*
	(0.19)	(>0.05)	(>0.05)	(0.52)	(>0.05)	(>0.05)	(0.01)	(<0.05)	(<0.01)
STAI-T	0.31	0.19	0.09	0.32	0.21	0.06	0.19	0.09	0.06
	(0.04)	(<0.05)	(>0.05)	(0.03)	(>0.05)	(>0.05)	(0.23)	(>0.05)	(>0.05)
STAI-S	0.31*	0.18	0.15	0.28*	0.15	0.07	0.30*	0.21	0.21
	(<0.01)	(>0.05)	(>0.05)	(0.01)	(>0.05)	(>0.05)	(0.01)	(<0.05)	(<0.05)
SCL:GSI	0.29*	0.15	0.39*	0.29*	0.15	0.39*	0.26*	0.16	0.21
	(0.01)	(>0.05)	(<0.01)	(<0.01)	(>0.05)	(<0.01)	(0.01)	(<0.05)	(<0.05)
SCL:PST	0.26*	0.15	0.32*	0.26*	0.15	0.36*	0.21	0.12	0.16
	(0.01)	(>0.05)	(<0.01)	(0.01)	(>0.05)	(<0.01)	(0.04)	(>0.05)	(>0.05)
SCL:PSDI	0.29*	0.16	0.36*	0.27*	0.14	0.29*	0.30*	0.21	0.25*
	(<0.01)	(>0.05)	(<0.01)	(0.01)	(>0.05)	(<0.01)	(<0.01)	(<0.05)	(<0.01)
LE	0.09	-0.01	0.04	0.10	0.01	0.04	0.08	0.01	0.05
	(0.37)	(>0.05)	(>0.05)	(0.32)	(>0.05)	(>0.05)	(0.46)	(>0.05)	(>0.05)
Child	0.22	0.11	0.15	0.15	0.03	0.04	0.28*	0.21	0.24*
	(0.02)	(>0.05)	(>0.05)	(0.13)	(>0.05)	(>0.05)	(0.01)	(<0.05)	(<0.01)

Note: LUD = log unpleasant dream frequency; LNM = log nightmare frequency; LBD = log bad dream frequency; -ND = nightmare distress partialled out; -ET = mean dream emotional tone partialled out.

APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale;

EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary

Questionnaire; STAI-T = Trait anxiety; STAI-S = State anxiety; SCL: GSI = SCL- Global Symptom Index;

SCL:PST= SCL- positive symptom total; SCL:PSDI = positive symptom distress index; LE = Sum of life events; Chil = Sum of childhood items.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Inspection of the above table shows that when nightmare distress is partialled out of the unpleasant dream frequency measure, including both moderately and very unpleasant dreams, correlations with well-being and individual differences become much smaller and all previously significant associations become insignificant with the exception of the neuroticism and social desirability scales. The majority of significant associations with log nightmare frequency also disappear with the exception of Eysenck's neuroticism and lie scales. Associations with log bad dream frequency all become insignificant (or remain) when nightmare distress is partialled out. As noted earlier when 'moderately unpleasant' dreams are included in the analysis nightmares do not generally have higher correlations with psychopathology than bad dreams. Comparing this table to table 14 shows that frequency measures only using 'very unpleasant' dreams, have stronger relationships with well-being. In the case of extreme unpleasant dreams, defined as very unpleasant, most correlations remain significant when nightmare distress is partialled out, which is in contrast to the above table where most correlations with unpleasant dream frequency, including moderately unpleasant dreams, become insignificant. The following tables shows the relationships of nightmare distress and emotional tone with measures of well-being and individual differences when these more loosely defined frequency measures are partialled out.

Table 17 - Kendall's tau_b correlations with nightmare distress and emotional tone when prospective frequency variables (using criterion of very unpleasant and moderately unpleasant dreams) are partialled out. Partialled out variables are indicated after hyphens in the top row.

	ND	ND- LUD	ND- LNM	ND- LBD	ET	ET- LUD	ET- LNM	ET-LBD
APS	0.27*	0.20	0.21*	0.24*	-0.21	-0.09	-0.09	-0.17
	(<0.01)	(<0.05)	(<0.01)	(<0.01)	(0.03)	(>0.05)	(>0.05)	(>0.05)
CEQ	0.27*	0.24*	0.26*	0.22*	-0.05	0.03	0.00	.002
:	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.63)	(>0.05)	(>0.05)	(>0.05)
STB	0.32 *	0.27*	0.28*	0.28*	-0.19	-0.09	-0.10	-0.13
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(>0.05)	(>0.05)	(>0.05)
EPQ-N	0.34*	0.24*	0.23*	0.30*	-0.25*	-0.07	0.01	-0.20
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.01)	(>0.05)	(>0.05)	(<0.05)
EPQ-L	0.10	-0.02	-0.04	0.08	-0.18	0.00	0.10	-0.16
	(0.27)	(>0.05)	(>0.05)	(>0.05)	(0.06)	(>0.05)	(>0.05)	(>0.05)
GHQ-12	0.28*	0.26*	0.26*	0.25*	-0.09	-0.04	-0.02	-0.05
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.36)	(>0.05)	(>0.05)	(>0.05)
BQ-18	0.25*	0.22*	0.25*	0.19	-0.07	-0.01	-0.04	-0.02
	(<0.01)	(<0.01)	(<0.01)	(<0.05)	(0.48)	(>0.05)	(>0.05)	(>0.05)
STAI-S	0.38*	0.30*	0.30*	0.35*	-0.42*	-0.31*	-0.29*	-0.39*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
STAI-T	0.42*	0.34*	0.35*	0.37*	-0.34*	-0.21*	-0.21*	-0.27*
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(<0.01)
SCL:PSDI	0.44*	0.37*	0.37*	0.40*	-0.19	0.07	0.13	-0.11
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(>0.05)	(>0.05)	(>0.05)
LE	0.25*	0.23*	0.23*	0.24*	-0.19	0.03	0.10	-0.13
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.04)	(>0.05)	(>0.05)	(>0.05)
Child	0.33*	0.27*	0.30*	0.27*	-0.22	0.02	0.03	-0.13
	(<0.01)	(<0.01)	(<0.01)	(<0.01)	(0.02)	(>0.05)	(>0.05)	(>0.05)

Note: ND= nightmare distress; ET =mean dream emotional tone; -LUD = log unpleasant dreams partialled out; -LNM = log nightmares partialled out; -LBD = log bad dreams partialled out. APS = Arousal Prediction Scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety; STAI-T = Trait anxiety; PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

As with the extreme frequency measures (Table 14) all significant relationships with nightmare distress remain when frequency measures partialled out, with the exception of the arousal prediction scale when unpleasant dream frequency is partialled out. Nearly all significant correlations with mean dream emotional tone become insignificant when frequency measures are partialled out with the exception of state and trait anxiety.

Summary of data on life events, childhood events and nightmare variables

Nightmare distress had the strongest relationship with adverse life events score. Retrospective frequency measures were significantly correlated with the life events score, even when nightmare distress was partialled out. Log frequency measures and dream emotional tone were not significantly correlated with life events score, nor with any of the individual life events items.

Nightmare distress was found to have the strongest correlations with the childhood variable, which combined the four items about characteristics of childhood. This sum of childhood items was also significantly correlated with retrospective nightmare frequency and log bad dream frequency, however, these became insignificant when nightmare distress was partialled out. There was no significant relationship between dream emotional tone.

frequency and occurrence

This section looks at the relationship of mood before and after sleep with the nightmare/unpleasant frequency variables and mean dream emotional tone. Levels of depression and anxiety were recorded before sleep and on awakening for the 14-day study period. Shapiro-Wilk tests of normality found that prospective measures of anxiety and depression were normally distributed with the exception of anxiety before sleep (Shapiro-Wilk statistic = 0.942, p= .01). Table 18 shows the means and (SDs) of pre- and post-sleep anxiety and depression. Anxiety and depression after sleep were found to be significantly higher than before sleep.

Table 18 – Means and (standard deviation) of prospective pre- and post-sleep anxiety and depression, from 14 day diaries

Pre-sleep	Post-sleep	Paired t(56)	р
28.91 (11.45)	32.84 (12.36)	4.73*	<0.001
30.38 (11.34)	33.22 (11.69)	4.54*	< 0.001
	28.91 (11.45)	28.91 (11.45) 32.84 (12.36)	28.91 (11.45) 32.84 (12.36) 4.73*

* p<0.01

Table 19 shows correlations between retrospective and prospective frequency measures, nightmare distress, mean dream tone and anxiety and depression well-being measures with mean anxiety before and after sleep. Table 19 - Spearman's rho correlations between mean prospective anxiety and mean prospective depression measures and retrospective and prospective frequency measures, nightmare distress, mean dream emotional tone, trait and state anxiety, and

SCL-anxiety and SCL-depression

Mean anxiety	Mean anxiety	Mean depression	Mean depression
before sleep	after sleep	before sleep	after sleep
0.33* (0.01)	0.28 (0.03)	0.24 (0.07)	0.28 (0.04)
0.19 (0.16)	0.19 (0.16)	0.07 (0.62)	0.18 (0.18)
0.57* (<0.01)	0.47*(<0.01)	0.42* (<0.01)	0.46* (<0.01)
0.48* (<0.01)	0.46* (<0.01)	0.41* (<0.01)	0.48* (<0.01)
0.55* (<0.01)	0.50*(<0.01)	0.42* (<0.01)	0.48* (<0.01)
0.45* (<0.01)	0.45*(<0.01)	0.39* (<0.01)	0.46* (<0.01)
0.50* (<0.01)	0.44*(<0.01)	0.38* (<0.01)	0.41* (<0.01)
0.42* (<0.01)	0.41* (<0.01)	0.35* (0.01)	0.43* (<0.01)
0.46* (<0.01)	0.42* (<0.01)	0.39*(<0.01)	0.45* (<0.01)
0.44* (<0.01)	0.41* (<0.01)	0.38* (<0.01)	0.44* (<0.01)
0.39* (<0.01)	0.39* (<0.01)	0.33* (<0.01)	0.41* (<0.01)
-0.59* (<0.01)	-0.66*(<0.01)	-0.52* (<0.01)	-0.59* (<0.01)
0.71* (<0.01)	0.69* (<0.01)	0.75* (<0.01)	0.75* (<0.01)
0.55* (<0.01)	0.54* (<0.01)	0.50* (<0.01)	0.50* (<0.01)
0.50* (<0.01)	0.35* (<0.01)	0.39* (<0.01)	0.43*(<0.01)
0.52* (<0.01)	0.48* (<0.01)	0.45* (<0.01)	0.49* (<0.01)
	before sleep $0.33*(0.01)$ $0.19(0.16)$ $0.57*(<0.01)$ $0.57*(<0.01)$ $0.48*(<0.01)$ $0.55*(<0.01)$ $0.45*(<0.01)$ $0.45*(<0.01)$ $0.42*(<0.01)$ $0.46*(<0.01)$ $0.44*(<0.01)$ $0.39*(<0.01)$ $0.55*(<0.01)$ $0.71*(<0.01)$ $0.55*(<0.01)$ $0.50*(<0.01)$	before sleepafter sleep $0.33^*(0.01)$ $0.28(0.03)$ $0.19(0.16)$ $0.19(0.16)$ $0.19(0.16)$ $0.19(0.16)$ $0.57^*(<0.01)$ $0.47^*(<0.01)$ $0.48^*(<0.01)$ $0.46^*(<0.01)$ $0.48^*(<0.01)$ $0.46^*(<0.01)$ $0.55^*(<0.01)$ $0.50^*(<0.01)$ $0.45^*(<0.01)$ $0.45^*(<0.01)$ $0.45^*(<0.01)$ $0.44^*(<0.01)$ $0.46^*(<0.01)$ $0.41^*(<0.01)$ $0.46^*(<0.01)$ $0.41^*(<0.01)$ $0.44^*(<0.01)$ $0.41^*(<0.01)$ $0.39^*(<0.01)$ $0.39^*(<0.01)$ $0.71^*(<0.01)$ $0.69^*(<0.01)$ $0.55^*(<0.01)$ $0.35^*(<0.01)$ $0.50^*(<0.01)$ $0.35^*(<0.01)$	before sleepafter sleepbefore sleep $0.33^*(0.01)$ $0.28(0.03)$ $0.24(0.07)$ $0.19(0.16)$ $0.19(0.16)$ $0.07(0.62)$ $0.57^*(<0.01)$ $0.47^*(<0.01)$ $0.42^*(<0.01)$ $0.48^*(<0.01)$ $0.47^*(<0.01)$ $0.42^*(<0.01)$ $0.48^*(<0.01)$ $0.46^*(<0.01)$ $0.41^*(<0.01)$ $0.55^*(<0.01)$ $0.50^*(<0.01)$ $0.42^*(<0.01)$ $0.45^*(<0.01)$ $0.50^*(<0.01)$ $0.45^*(<0.01)$ $0.45^*(<0.01)$ $0.44^*(<0.01)$ $0.38^*(<0.01)$ $0.42^*(<0.01)$ $0.41^*(<0.01)$ $0.35^*(0.01)$ $0.46^*(<0.01)$ $0.41^*(<0.01)$ $0.39^*(<0.01)$ $0.44^*(<0.01)$ $0.41^*(<0.01)$ $0.38^*(<0.01)$ $0.39^*(<0.01)$ $0.39^*(<0.01)$ $0.33^*(<0.01)$ $0.59^*(<0.01)$ $0.69^*(<0.01)$ $0.75^*(<0.01)$ $0.55^*(<0.01)$ $0.54^*(<0.01)$ $0.50^*(<0.01)$ $0.50^*(<0.01)$ $0.35^*(<0.01)$ $0.39^*(<0.01)$

Note: RUD = retrospective unpleasant dream frequency; RNM = retrospective nightmare frequency; ND= nightmare distress; LNM = log nightmare frequency; LUD = log unpleasant dream frequency; ; LNM (14) = log nightmare frequency (14); LUD (14) = log unpleasant dream frequency (14); LXNM = log extreme nightmares; LXUD = log extreme unpleasant dreams; LXNM (14) = log extreme nightmares (14); LXUD (14) = log extreme unpleasant dreams (14). ET = mean dream emotional tone. STAI-S = state anxiety; STAI-T =trait anxiety; SCL-DEP = SCL-depression; SCL-ANX = SCL- anxiety.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

¹ Frequency variables with 'X' in their title refer to extreme dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale.

² Frequency variables with (14) in their tiles means that the measure includes at most only one dream per night. Frequency variables without (14) in their title allow for the recording of more than one dream per night. Table 19 shows that prospective anxiety and depression had correlations that were ordered in size as follows: dream emotional tone > nightmare distress > log nightmare and unpleasant dream frequencies > frequency of log nightmare and unpleasant dreams with criterion of very unpleasant dream emotion > retrospective unpleasant dream frequency > retrospective nightmare frequency.

Correlations with nightmares and unpleasant dreams (defined as very unpleasant) are smaller than with those defined as moderately unpleasant and very unpleasant which is in contrast to correlations with measures of poor well-being and individual differences which were higher with these more strictly defined nightmares and unpleasant dreams.

State and trait anxiety and SCL anxiety and depression sub scales were all significantly associated with prospective measures of anxiety and depression. However, it is a concern, which will be further addressed in Tables 22 and 23, that correlations of prospective anxiety and depression do not differ from each other appreciably according to whether they are correlated with the individual difference STAI- and SCL-anxiety or SCL-depression measures.

Table 20 shows the relationship between frequency variables and mood before and after sleep when retrospective nightmare distress and prospectively measured emotional tone are partialled out. Table 20 – Kendall's tau_b correlations between frequency variables and anxiety and depression before and after sleep when nightmare distress and mean dream emotional tone are partialled out.

	Mean anxiety	Mean anxiety	Mean depression	Mean depression
	before sleep	after sleep	before sleep	after sleep
RUD	0.22 (0.02)	0.20 (0.03)	0.16 (0.08)	0.20 (0.03)
RUD-ND	0.05 (>0.05)	0.05 (>0.05)	0.03 (>0.05)	0.05 (>0.05)
RUD-ET	0.12 (>0.05)	0.09 (>0.05)	0.07 (>0.05)	0.10 (>0.05)
RNM	0.13 (0.18)	0.14 (0.12)	0.03 (0.75)	0.13 (0.14)
RNM-ND	-0.06 (>0.05)	-0.02 (>0.05)	-0.12 (>0.05)	-0.03 (>0.05)
RNM-ET	0.02 (>0.05)	0.02 (>0.05)	-0.07 (>0.05)	0.02 (>0.05)
LUD	0.39* (<0.01)	0.36* (<0.01)	0.30* (<0.01)	0.35* (<0.01)
LUD-ND	0.28* (<0.01)	0.26* (<0.01)	0.21* (<0.01)	0.25* (<0.01)
LUPD-ET	0.20 (<0.05)	0.13 (>0.05)	0.12 (>0.05)	0.14 (>0.05)
LNM	0.35* (<0.01)	0.33* (<0.01)	0.31* (<0.01)	0.35* (<0.01)
LNM-ND	0.23* (<0.01)	0.23* (<0.01)	0.22* (<0.01)	0.25* (<0.01)
LNM –ET	0.16 (>0.05)	0.11(>0.05)	0.15(>0.05)	0.16 (>0.05)
LXUD ¹	0.34* (<0.01)	0.32*(<0.01)	0.29* (<0.01)	0.35* (<0.01)
LXUD' -ND	0.24*(<0.01)	0.23*(<0.01)	0.21*(<0.01)	0.27*(<0.01)
LXUD-ET	0.14 (>0.05)	0.08 (>0.05)	0.11 (>0.05)	0.15 (>0.05)
LXNM ¹	0.29*(<0.01)	0.29* (<0.01)	0.26* (0.01)	0.31*(<0.01)
LXNM ¹ -ND	0.19 (<0.05)	0.20 (<0.05)	0.18 (>0.05)	0.23* (<0.01)
LXNM ¹ -ET	0.09 (>0.05)	0.07(>0.05)	0.09(>0.05)	0.12(>0.05)

Note: RUD = retrospective unpleasant dream frequency; RNM= retrospective nightmare frequency; LUD = log unpleasant dream frequency; LNM = log nightmare frequency; LXUD = log extreme unpleasant dreams; LXNM = Log extreme nightmares; -ET=mean dream emotional tone partialled out; -ND = nightmare distress partialled out. * p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

¹ Frequency variables with 'X' in their title refer to extreme dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale.

Table 20 shows that there is little difference between pre- and post-sleep correlations with the nightmare and unpleasant dream variables. When nightmare distress and emotional tone are partialled out Kendall's correlations with retrospective unpleasant dream frequency and nightmare frequency become very small.

Log frequency correlations, with nightmares or unpleasant dreams defined as moderately and very unpleasant, remain significantly correlated with anxiety and depression before and after sleep when nightmare distress is partialled out as does log unpleasant dream frequency defined as very unpleasant. However, log nightmares defined only as very unpleasant remain significantly correlated with depression after sleep when nightmare distress is partialled out. However, all correlations with frequency measures and anxiety and depression before and after sleep become much smaller and insignificant when mean dream emotional tone is partialled out.

Table 21 shows Kendall's tau_b correlations of nightmare distress and emotional tone with prospective anxiety and depression when frequency variables are partialled out.

	Mean anxiety	Mean anxiety	Mean depression	Mean depression after
	before sleep	after sleep	before sleep	sleep
ND	0.40* (<0.01)	0.35* (<0.01)	0.30* (<0.01)	0.35* (<0.01)
ND –RUD	0.35* (<0.01)	0.30* (<0.01)	0.26* (<0.01)	0.30* (<0.01)
ND- RNM	0.39* (<0.01)	0.32* (<0.01)	0.20 (<0.05)	0.33* (<0.01)
ND- LUD	0.30* (<0.01)	0.25* (<0.01)	0.21* (<0.01)	0.25* (<0.01)
ND-LNM	0.31* (<0.01)	0.26* (<0.01)	0.21*(<0.01)	0.25* (<0.01)
ND-LXUD ¹	0.33* (<0.01)	0.28*(<0.01)	0.23*(<0.01)	0.27*(<0.01)
ND-LXNM ¹	0.34*(<0.01)	0.29*(<0.01)	0.24*(<0.01)	0.28*(<0.01)
ET	-0.43* (<0.01)	-0.47* (<0.01)	-0.37* (<0.01)	-0.43* (<0.01)
ET-RUD	-0.40* (<0.01)	-0.44* (<0.01)	-0.34* (<0.01)	-0.40* (<0.01)
ET- RNM	-0.41*(<0.01)	-0.45* (<0.01)	-0.32* (<0.01)	-0.41* (<0.01)
ET- LUD	-0.27* (<0.01)	-0.35* (<0.01)	-0.25* (<0.01)	-0.30* (<0.01)
ET- LNM	-0.31* (<0.01)	-0.37* (<0.01)	-0.26* (<0.01)	-0.31* (<0.01)
ET-LXNM ¹	-0.34*(<0.01)	-0.39*(<0.01)	-0.29*(<0.01)	-0.33*(<0.01)
ET-LXUD ¹	-0.31*(<0.01)	-0.37*(<0.01)	-0.26*(<0.01)	-0.30*(<0.01)

Table 21: Kendall's tau_b correlations of nightmare distress and emotional tone with prospective anxiety and depression when frequency variables are partialled out.

Note: ND= nightmare distress; ET = mean dream hedonic tone; RUD = retrospective unpleasant dream frequency partialled out; -RMN= retrospective nightmare frequency partialled out; -LUD= log unpleasant dream frequency partialled out; -LNM = Log nightmare frequency partialled out; -LXUD= log extreme unpleasant dream frequency partialled out; -LXNM= log extreme nightmares partialled out;

¹ Frequency variables with 'X' in their title refer to extreme dreams defined only as dreams rated 1 (very unpleasant) on the hedonic scale.

Table 21 shows that significant relationships of nightmare distress and mean dream tone with anxiety and depression remain significant when retrospective and prospective unpleasant dream and nightmare frequency variables are partialled out.

<u>Caveat</u>

There is a concern about participants' ability to distinguish anxiety from depression given that Table 19 shows high correlations between the prospective anxiety and depression measures, and Table 26 shows that the prospective anxiety and depression measures do not show systematic differences in their correlations with STAI-anxiety and with SCL-depression and anxiety. Table 26 shows that SCLdepression and SCL-Anxiety are also highly correlated with each other, as well as with the STAI measures of anxiety.

Table 22 – Spearman's rho intercorrelations between anxiety and depression before and after sleep.

	Mean anxiety	Mean anxiety	Mean depression	Mean depression
	before sleep	after sleep	before sleep	after sleep
Mean anxiety before		0.87 (<0.01)	0.90 (<0.01)	0.87 (<0.01)
sleep			``´´	· · ·
Mean anxiety after	0.87 (<0.01)	<u> </u>	0.88 (<0.01)	0.94 (<0.01)
sleep				
Mean depression	0.90 (<0.01)	0.88 (<0.01)		0.92 (<0.01)
before sleep	0.90 (0.01)	0.00 (0.01)		0.72 (10.01)
Mean depression	0.87 (<0.01)	0.94 (<0.01)	0.92 (<0.01)	
after sleep	0.87 (<0.01)	0.94 (<0.01)	0.92 (<0.01)	-

p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of

significance was set at p = <0.01). All values are two tailed.

	State Anxiety	Trait Anxiety	SCL-	SCL-Anxiety
			Depression	
State anxiety	-	0.88 (<0.01)	0.88 (<0.01)	0.91 (<0.01)
Trait anxiety	0.88 (<0.01)	-	0.78 (<0.01)	0.70 (<0.01)
SCL-Depression	0.88 (<0.01)	0.78 (<0.01)	-	0.82 (<0.01)
SCL-Anxiety	0.91 (<0.01)	0.70 (<0.01)	0.82 (<0.01)	-

Table 23 – Spearman's rho correlations between retrospective anxiety and depression

p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Within subjects correlations of prospective anxiety and depression with prospective dream variables

This section examines within subjects correlations of pre- and post-sleep anxiety and depression with the occurrence / non-occurrence of a nightmare / an unpleasant dream / a dreams on that night, and also the correlation with dream emotional tone for that night. Once the magnitude of these within subjects correlations is established the following section will investigate whether individual differences in the size of these correlations is correlated with individual differences or psychopathology variables, which would indicate that the effect of waking life anxiety and depression on initiating nightmares / unpleasant dreams / dreams may be modulated by individual differences or psychopathology variables. Within subjects correlations of anxiety and depression with dream emotional tone

Within subjects correlations were first conducted between mean emotional tone of the dream and pre- and post-sleep anxiety, pre- and post-sleep depression, the change in anxiety across the night and change in depression across the night. Table 24 shows the descriptive statistics for these within subjects correlations (WS r).

Table 24 - Descriptive statistics for within subjects correlations (WS r) between mean dream tone and pre- and post-sleep anxiety and depression, and change across sleep in anxiety and in depression

	Mean	Median	SD	Range
WS r with ET and pre-sleep anxiety	-0.22	-0.20	0.33	-0.88 - 061
WS r with ET and post-sleep anxiety	-0.41	-0.44	0.38	-0.91 - 0.80
WS r with ET and pre-sleep depression	-0.15	-0.18	0.37	-0.98 - 0.72
WS r with ET and post-sleep depression	-0.39	-0.46	0.33	-0.89 - 0.70
WS r with ET and change between pre and post anxiety	-0.17	-0.14	0.37	-0.89 - 0.58
WS r with ET and change between pre and post depression levels.	-0.19	-0.23	0.40	-0.89 - 0.74

Note: WSr = within subjects correlation; ET = mean dream hedonic tone.

The above table shows that the correlations of dream tone are higher with post-sleep depression and anxiety than with pre-sleep anxiety and depression, suggesting that a more negative emotional tone of a dream is more strongly associated with higher levels of depression and anxiety on awakening rather than before sleep. Using paired t-tests these pre- post-sleep differences were both significant (anxiety: t(54) = 3.21, p=.002; depression: t (54) = 3.53, p=.001).

Within subjects correlations of anxiety and depression with the occurrence / non-occurrence of a nightmare and of an unpleasant dream

Within subjects analyses were conducted to assess the relationships between pre- and post-sleep anxiety and depression, and the change between pre- and postsleep anxiety and depression, with the occurrence / non-occurrence of a nightmares or an unpleasant dreams. As there are two levels of the occurrence / non-occurrence variable, and as the anxiety and depression variables were generally normally distributed, Pearson's biserial correlation was used.

The first set of analyses was conducted with the dichotomous variable presence of a nightmare versus no nightmare, and then with the dichotomous variable presence of an unpleasant dream versus no unpleasant dream. These analyses thus included nights where subjects reported no dream at all, as on those nights there is thus no nightmare.

Thus across all 14 nights analysis was conducted firstly using unpleasant dreams/nightmares with a hedonic tone of 1 (very unpleasant) or 2 (moderately unpleasant) (Table 25) and then including only unpleasant dreams/nightmares with hedonic tone of 1 (very unpleasant) (Table 26).

Table 25 - Mean Pearson biserial within subject correlations (WS r) between preand post-sleep anxiety / depression and the dichotomous variable presence/absence of a nightmare or unpleasant dream, where absence of a dream is counted as absence of a nightmare or unpleasant dream. Nightmare and unpleasant dreams have a hedonic tone of 1 (very unpleasant) or 2 (moderately unpleasant).

	Mean correlation	Median	SD	Range	
	coefficient				
WS r with nights with UD and	0.11	0.12	0.20	0.40 0.62	
anxiety before sleep	0.11	0.13	0.30	-0.49 – 0.63	
WS r with nights UD and	0.28	0.30	0.28	-0.44 - 0.63	
anxiety after sleep	0.28	0.50	0.28	-0.44 - 0.03	
WS r with nights with UD and	0.09	0.08	0.34	-0.75 -0.10	
depression before sleep	0.09	0.08	0.54	-0.750.10	
WS r with nights with UD and	0.23	0.24	0.29	-0.26 - 0.10	
depression after sleep	0.25	0.24	0.29	-0.20 - 0.10	
WS r with nights with UD and			· · ·		
change between morning and	0.17	0.18	0.31	-0.64- 0.70	
night anxiety					
WS r with UD and change					
between morning and night	0.14	0.18	0.36	-0.72 - 0.76	
depression levels.					
WS r with nights with NM	0.12	0.28	0.29	-0.31- 0.76	
and anxiety before sleep	0.12	0.20	0.29	0.51 0.70	
WS r with nights with NM	0.36	0.38	0.25	-0.25 - 0.76	
and anxiety after sleep	0.50	0.50	0.25	-0.23 - 0.70	
WS r with nights with NM	0.08	0.06	0.30	-0.47 - 0.78	
and depression before sleep		0.00	0.50	0.17 0.70	
WS r with nights with NM	0.30	0.35	0.32	-0.40 - 0.77	
and depression after sleep	0.50	0.55	0.52	0.10 0.77	
WS r with nights with NM					
and change between morning	0.17	0.17	0.29	-0.56 - 0.81	
and night anxiety				·····	
WS r with nights with NM			<u> </u>		
and change between morning	0.19	0.24	0.32	-0.550.81	
and night depression levels.					

Note: WS r = within subjects correlation; NM=presence/absence of a nightmare; UD= presence/absence of an unpleasant dream.

Table 26 - Mean Pearson biserial within subject correlations (WS r) between preand post-sleep anxiety / depression and the dichotomous variable presence/absence of nightmares or unpleasant dreams, where absence of a dream is counted as absence of nightmare/ unpleasant dreams. Nightmare/ unpleasant dreams have hedonic tone of 1 (very unpleasant).

	Mean	<u> </u>		· · · · · · · · · · · · · · · · · · ·
	correlation	Median	SD	Range
	coefficient			
WS r with nights with UD and	<u>^ 10</u>	0.15	0.00	0.44.0.50
anxiety before sleep	0.12	0.15	0.28	-0.44 - 0.59
WS r with nights with UD and	0.32	0.27	0.29	-0.44 - 0.69
anxiety after sleep	0.32	0.27	0.29	-0.44 - 0.09
WS r with nights with UD and	0.09	0.17	0.31	-0.33 - 0.99
depression before sleep	0.09	0.17	0.51	-0.33 - 0.99
WS r with nights with UD and	0.32	0.32	0.31	-0.33 - 0.99
depression after sleep	0.52	0.52	0.51	-0.55 - 0.95
WS r with nights with UD and		<u> </u>		
change between morning and night	0.15	0.10	0.34	-0.70 - 0.70
anxiety				
WS r with UD frequency and	····	- <u> </u>		
change between morning and night	0.18	0.21	0.34	-0.68 - 0.76
depression levels.				
WS r with nights with NM and	0.14	0.16	0.25	-0.18 - 0.90
anxiety before sleep	0.14	0.10	0.25	-0.10 - 0.90
WS r with nights with MN and	0.32	0.32	0.29	-0.41 - 0.90
anxiety after sleep	0.52	0.52	0.27	-0.41 - 0.90
WS r with nights with NM and	0.09	0.10	0.31	-0.47 - 0.77
depression before sleep	0.09	0.10	0.51	0.47 0.77
WS r with nights with NM and	0.30	0.29	0.30	-0.35 - 0.77
depression after sleep	0.50	0.27	0.50	-0.55 - 0.77
WS r with nights with NM and				
change between morning and night	0.14	0.10	0.28	-0.39 - 0.81
anxiety				
WS r with nights with NM and				
change between morning and night	0.18	0.22	0.29	-0.41 - 0.81
depression levels.				

Note: WS r = within subjects correlation; NM=presence/absence of a nightmare; UD= presence/absence of an unpleasant dream.

The second set of analyses was conducted with the dichotomous variable; dream that is a nightmare versus dream that is not a nightmare, and then with the dichotomous variable; dream that is unpleasant versus dream that is not unpleasant. These analyses thus did not include nights where subjects reported no dream at all.

This analysis of nights where there was a dream was conducted firstly using unpleasant dreams/nightmares with a hedonic tone of 1 (very unpleasant) or 2 (moderately unpleasant) (Table 27) and then including only unpleasant dreams/nightmares with hedonic tone of 1 (very unpleasant) (Table 28).

Table 27 - Mean Pearson biserial within subject correlations (WS r) between preand post-sleep anxiety / depression and the dichotomous variable presence/absence of nightmares or unpleasant dreams, using only nights where a dream did occur. Nightmares and unpleasant dreams have hedonic tone = 1 (very unpleasant) or 2 (moderately unpleasant).

	Mean			
	correlation	Median	SD	Range
	coefficient			
VS r with nights with UD and	0.14	0.14	0.36	-0.51 - 1.00
nxiety before sleep	0.14	0.14	0.50	-0.51 1.00
/S r with nights with UD and	0.31	0.33	0.34	-0.45 - 0.90
nxiety after sleep	0.51	0.55	0.54	-0.45 - 0.50
/S r with nights with UD and	0.10	0.13	0.36	-0.78 - 0.99
epression before sleep	0.10	0.15	0.50	-0.78 - 0.99
VS r with nights with UD and	0.23	0.30	0.33	-0.39 - 0.99
lepression after sleep	0.25	0.50	0.00	-0.37 - 0.77
VS r with nights with UD and				
hange between morning and	0.16	0.20	0.36	-0.63 - 0.80
ight anxiety				
VS r with UD frequency and				
hange between morning and	0.14	0.14	0.38	-0.63 - 0.80
ight depression levels.				
/S r with nights with NM and	0.14	0.13	0.33	-0.32 - 0.88
xiety before sleep	0.14	0.15	0.55	-0.52 - 0.00
/S r with nights with NM and	0.36	0.43	0.29	-0.40 - 0.88
nxiety after sleep	0.50	0.45	0.27	-0.40 - 0.00
'S r with nights with MN and	0.11	0.08	0.34	-0.58 - 0.80
epression before sleep	0.11	0.00	0.54	-0.00 - 0.00
/S r with nights with MN and	0.32	0.38	0.35	-0.43 - 0.77
epression after sleep	0.32	0.30	0.55	-0.45 - 0.77
/S r with nights with NM and				
hange between morning and	0.17	0.21	0.34	-0.57 - 0.89
ight anxiety				
/S r with nights with NM and	· · · · · · · · · · · · · · · · · · ·			
hange between morning and	0.20	0.26	0.35	-0.56 - 0.82
ight depression levels.				

Note: WS r = within subjects correlation; NM=presence/absence of a nightmare; UD= presence/absence of an unpleasant dream.

Table 28 - Mean Pearson biserial within subject correlations (WS r) between preand post-sleep anxiety / depression and the dichotomous variable presence/absence of nightmares or unpleasant dreams, using only nights where a dream did occur. Nightmares and unpleasant dreams have hedonic tone of 1 (very unpleasant).

	Mean correlation coefficient	Median	SD	Range
WS r with nights with UD and anxiety before sleep	0.16	0.10	0.34	-0.41 - 0.90
WS r with nights with UD and anxiety after sleep	0.34	0.28	0.35	-0.45 - 0.90
WS r with nights with UD and depression before sleep	0.11	0.16	0.35	-0.42-0.99
WS r with nights with UD and depression after sleep	0.35	0.38	0.36	-0.420.99
WS r with nights with UD and change between morning and night anxiety	0.14	0.12	0.40	-0.89-0.80
WS r with nights with UD and change between morning and night depression levels.	0.19	0.21	0.38	-0.840.76
WS r with nights with NM and anxiety before sleep	0.16	0.15	0.29	-0.24-0.90
WS r with nights with NM and anxiety after sleep	0.33	0.35	0.31	-0.40-0.90
WS r with nights with NM and depression before sleep	0.12	0.19	0.36	-0.47-0.80
WS r with nights with NM and depression after sleep	0.34	0.38	0.33	-0.47- 0.76
WS r with nights with NM and change between morning and night anxiety	0.13	0.08	0.33	-0.39-0.89
WS r with nights with NM and change between morning and night depression levels.	0.18	0.21	0.33	-0.41-0.82

Note: WS r = within subjects correlation; NM=presence/absence of a nightmare; UD= presence/absence of an unpleasant dream.

Comparisons of the magnitudes of the within subjects correlations

In order to analyse these within-subject correlations with inferential statistics the correlation coefficients were each converted to a Fisher's r coefficient. Shapiro-Wilk tests showed that almost all of the 32 within-subjects r's were normally distributed. These r co-efficients were then analysed by repeated measures ANOVA, one ANOVA for the anxiety correlations, and one for the depression correlations.

- a) whether anxiety or depression were assessed pre- or post-sleep,
- b) whether a nightmare or unpleasant dream was defined as very unpleasant, or as very or moderately unpleasant,
- c) whether the dichotomous variable (presence/absence of nightmare/unpleasant dream) is assessed for all 14 nights or only for those nights on which there was a dream; and
- d) whether the waking criterion is used (waking criterion nightmare versus inclusive unpleasant dream).

Sphericity was not violated and so no correlations for degrees of freedom were used in the following analysis.

Table 29 – Effects of within subjects factors on within subjects correlations reported in Tables 25 - 28, for anxiety and depression analysed separately.

· <u> </u>	WS correlations w	ith anxiety	WS correlations with depression		
	F(1,36)	р	F(1,37)	Р	
Within-subjects factor			. <u>, .</u>		
Pre-versus post-sleep	9.655	.004	15.028	<.001	
Very high versus high unpleasantness criterion	0.187	0.67	0.926	0.342	
Absence of NM/UD includes nights with no dreams versus only nights with dream.	3.128	.085	2.201	0.15	
Waking criterion versus	0.030	0.86	0.028	0.87	

Note: NM = nightmare; UD= unpleasant dream.

Due to the number of participants and large number of combinations of factors threshold for significance of p was set at .01 for all interactions between these factors. No interactions were found to be significant. In general there were no sex differences in these within-subjects correlations.

Table 29 shows that the strictness of defining nightmares, whether in terms of use of the awakening criterion or the excluding of moderately unpleasant dreams, did not affect within subjects correlations with anxiety or with depression. The table shows that presence / absence of a nightmare/unpleasant dream has a significantly greater association with anxiety and depression after sleep than before sleep. Thus, all factors were not significant except for the pre-sleep versus post-sleep factor.

Caveat regarding robustness of the within-subjects correlations

A caveat with the within subjects results so far is that some of the within subjects correlations had just one data point for presence of a nightmare / unpleasant dream, with all the other data points being for when no nightmare or unpleasant dream occurred. In order to determine if this paucity of sampling of presence of a nightmare / unpleasant dream caused a lower mean of the within subjects correlations, by increasing error variance, the following analyses were conducted.

Firstly, a single mean of all within subjects correlations with anxiety and with depression were calculated for all participants. This included all the different types of nightmare definition (e.g. very unpleasant dream emotion criterion with waking criterion; very and moderate unpleasant dream emotion criterion without waking criterion, etc.) If a participant did not have one of the specific types of nightmare definition, all the other within subjects correlations were averaged. The mean of all within subject correlations of mean emotional tone with anxiety and depression was also calculated for all participants. Table 30 shows the mean and median for all participants, for these two variables. 55 participants had produced diary ratings of dream emotional tone, however, as some of these had no nightmares or unpleasant dreams, the number of participants providing within subject correlations of

presence/absence of a nightmare / unpleasant dream is lower (n = 48), but

included all participants who experienced at least one nightmare or unpleasant dream.

Table 30 – Mean, median, SD and range of within-subject correlations between presence/absence of nightmare or unpleasant dream and anxiety and depression, and mean, median, SD and range of within subjects correlations between dream emotional tone and anxiety and depression.

	Mean	Median	SD	Range
WS correlations of	0.19	0.15	0.20	- 0.18 - 0.60
presence/absence of nightmare /				
unpleasant dream with anxiety				
and depression $(n = 48)$				
WS correlations of dream	- 0.29	- 0.30	0.23	- 0.89 - 0.21
emotional tone with anxiety and				
depression (n=55)				

The table shows that worse mood (anxiety and depression) is associated with nightmares and unpleasant dreams, and with more negative dream emotional tone.

This analysis was then conducted again but including only participants who reported two or more waking criterion nightmares rated as very unpleasant (hedonic tone = 1) during the 14 day study period. 24 participants met this criterion. It was reasoned that within subjects correlations might become larger as subjects would have more than one data point for presence of a nightmare for all their WS correlations, and hence error variance would be reduced. The descriptive statistics of these within subject's correlations are shown in Table 31. Table 31 - Mean, median, standard deviation and range of within-subject correlations for participants who reported two or more waking criterion nightmares rated as very unpleasant (hedonic tone = 1) during the 14 day study period. WS correlations are between presence/absence of nightmare or unpleasant dream and anxiety and depression, and between dream emotional tone and anxiety and depression.

	Mean	Median	SD	Range
WS correlations of	0.25	0.23	0.18	-0.03 - 0.60
presence/absence of				
nightmare / unpleasant dream				
with anxiety and depression				
(n =24)				
WS correlations of dream	-0.37	-0.40	0.21	-0.68 - 0.04
emotional tone with anxiety				
and depression (n=24)				

This table shows that the means and medians of this subsample are larger and the ranges smaller than for those of the whole sample.

These within subjects analyses were then conducted again but only for those participants who reported three or more waking criterion nightmare rated as very unpleasant over the 14 day log period. 13 participants met this criterion. The means and median's of these within subjects correlations are shown in Table 32. Table 32 - Mean, median, SD and range of within-subject correlations for participants who reported three or more waking criterion nightmares rated as very unpleasant (hedonic tone = 1) during the 14 day study period. Within subject correlations are between presence/absence of nightmare or unpleasant dream and anxiety and depression, and between dream emotional tone and anxiety and depression.

	Mean	Median	SD	Range
WS correlations of	0.24	0.15	0.20	0.02 - 0.60
presence/absence of				
nightmare / unpleasant				
dream with anxiety and				
depression (n =13)				
WS correlations of dream	-0.34	-0.34	0.24	-0.68 - 0.04
emotional tone with anxiety				
and depression $(n = 13)$				
NT 4				

Note: WS= within subject.

This table shows that the medians of the correlations for this subsample are comparable to the medians of the correlations for the whole sample of participants. Thus, there may be an effect of having too small a sample of participants if the criterion of number of nights with nightmares is set too high.

Tables 30 - 32 suggest that higher within subjects correlations are obtained by using the subsample of 24 participants, defined as having more than one waking criterion nightmare with very high unpleasantness. The inferential statistical analysis that compared the four factors of the set of within-subjects correlations was thus repeated using this subsample, while acknowledging that the sample size is small for a 4-way ANOVA. Shapiro-Wilk tests showed that the Fisher r transforms of the within subjects correlations for the 24 participants were normally distributed. The Fisher's r variables were analysed by repeated measures ANOVA; one ANOVA was conducted for anxiety and one for depression. The results are shown in Table 33. Sphericity was not violated for either ANOVA and so no corrections for dfs were used.

Table 33 - Table showing effects of repeated measures factors on within subjects correlations, for anxiety and depression analysed separately. Only data from 24 participants, defined as having more than one waking criterion nightmare with very high unpleasantness during the 14 day diary period, are included.

	Ws correlations with anxiety		Ws correlations with depressi	
	F(1,22)	Р	F(1,23)	Р
Within-subjects factor				
Pre-versus post-sleep	8.28	0.01	7.50	0.01
Very high versus high unpleasantness criterion	0.00	0.10	0.97	0.33
Waking criterion versus no waking criterion	1.61	0.22	0.39	0.54
Absence of NM/UPD includes		<u></u>		
nights with no dreams versus only nights with dream.	0.79	0.38	2.71	0.11

Note: NM = nightmare; UD= unpleasant dream.

Due to the number of participants and large number of combinations of factors threshold for significance of p was set at .01 for all interactions between these factors. No interactions were found to be significant. In general there were no sex differences in these within-subjects correlations. As with the previous ANOVA for the entire sample (shown in table 29), none of the factors were significant except for the pre-sleep versus post-sleep factor.

Within subjects correlations of anxiety and depression with the occurrence / nonoccurrence of a dream

The results in this section have shown that the within subjects correlations of state anxiety and state depression with presence/ absence of a nightmare or unpleasant dreams are generally small. However, as reviewed in the introduction there have been reports that increased state anxiety and state depression can increase the likelihood of dream recall: a check must therefore be made as to whether poor waking mood before sleep, in increasing nightmare and unpleasant dream occurrence, is actually increasing the occurrence of all dreams. Thus, to address this potential confound in the results, within-subjects correlations between pre sleep anxiety / depression and the presence/absence of a dream were computed, and the results are presented in Table 34.

Table 34 – Mean, median, SD and range of within-subjects Pearson correlations between pre-sleep anxiety and depression and presence / absence of a dream that night (n=51)

	Mean	Median	SD	Range
Pre-sleep anxiety	0.01	0.01	0.29	-0.72 - 0.57
Pre-sleep depression	-0.01		0.27	-0.54 - 0.57

It can be concluded from the above table that pre-sleep anxiety and depression do not affect dream recall. The only effect of pre-sleep mood is thus specifically on the presence of nightmares and unpleasant dreams, although this effect is small.

Comparison of within-subject and between-subject correlations of presence of nightmares with state anxiety and depression

Participants assessed their state anxiety and depression before and after sleep for 14 nights. Table 35 shows the mean of these state anxiety and state depression measures, and the between-subjects Kendall's correlations of those means with the number of nightmares and unpleasant dreams that occurred across the 14 nights. In this analysis only dreams rated as very unpleasant were categorised as nightmares or unpleasant dreams. This restriction of definition was used because between-subject correlations of well-being and personality variables with nightmare and unpleasant dream frequencies were higher when this restriction was used (see Table 13; however, note that this restriction did not lead to significant changes in within-subject correlations, see Tables 29 and 33).

Table 35 - Mean and standard deviation of state anxiety and state depression measures, and the Kendall's between-subjects correlations and (probabilities) of those means with the number of nights on which a nightmare (NM) or unpleasant dream (UD) occurred across the 14 nights. NM and UPD are defined as dreams scored as very unpleasant, the former waking up the sleeper, the latter defined without the waking criterion (n = 56).

	Mean	SD	NM	UPD
Presleep anxiety	28.91	11.45	0.29 (0.004)	0.33 (0.001)
Postsleep anxiety	32.84	12.36	0.30 (0.003)	0.31 (0.002) -
Presleep depression	30.38	11.34	0.26 (0.009)	0.28 (0.004)
Postsleep depression	33.22	11.69	0.31 (0.002)	0.34 (0.001)

Table 36 reports the mean, median and standard deviation of the withinsubject Kendall correlations between the state anxiety and state depression scores over 14 nights with the presence or absence of a nightmare / unpleasant dream on each night. For 37 participants a Kendall correlation could be calculated for each of the 4 within-subject combinations.

Table 36 - Mean, standard deviation and median of Kendall's within-subjects correlations between presence or absence of a nightmare (NM) or unpleasant dream (UD) and state anxiety and state depression variables across the 14 nights. NM and UD are defined as dreams scored as very unpleasant, the former waking up the sleeper, the latter defined without the waking criterion (n = 37).

Correlates	NM presence/absence			UD pres	e	
	Mean	SD	Median	Mean	SD	Median
Presleep anxiety	0.10	0.24	0.12	0.10	0.24	0.15
Postsleep anxiety	0.24	0.27	0.32	0.24	0.27	0.21
Presleep depression	0.07	0.28	0.06	0.07	0.29	0.06
Postsleep depression	0.26	0.23	0.28	0.28	0.22	0.28

In order to compare these means of the within-subject correlations with between-subjects correlations, the latter must only be calculated for the 37 participants used for the within-subject correlations shown in Table 36. Table 37 shows these between-subjects correlations. Note that the exclusion of 19 participants who were included in the results of Table 35 has resulted in a decrease in some of the between-subjects correlations, because the exclusion of participants restricts the range of the data as these 19 participants had no nightmares or no unpleasant dreams. Table 37 - Between-subjects Kendall's correlations and (probabilities) between means of state anxiety and state depression and number of nights on which a nightmare (NM) or unpleasant dream (UD) occurred across the 14 nights. NM and UD are defined as dreams scored as very unpleasant, the former waking up the sleeper, the latter defined without the waking criterion. Only participants included in Table 36 are included here (n = 37).

Correlates	NM	UPD
Presleep anxiety	0.24 (0.057)	0.23 (0.065)
Postsleep anxiety	0.27 (0.030)	0.22 (0.080)
Presleep depression	0.27 (0.030)	0.19 (0.119)
Postsleep depression	0.32 (0.012)	0.25 (0.046)

A comparison of Tables 36 and 37 shows that for pre-sleep mood betweensubjects correlations are far higher than within-subjects correlations, and for postsleep mood the between-subjects and within-subjects correlations are similar in size.

Predisposition for nightmares and unpleasant dreams

The aim of this section is to test the hypothesis that individual differences lead to some people being more susceptible to the occurrence of nightmares. To do this between-subject correlations were calculated between 17 psychopathology and individual difference variables and the 32 types of within-subject correlations of state anxiety or state depression with nightmare/unpleasant dream variables. The 32 types of within-subjects correlations occur due to the combinations of the following factors, all of which have two levels:

Mood: anxiety versus depression

Time of assessment: Pre-sleep versus post-sleep

Use of waking criterion: Nightmare versus Unpleasant dream

Dream emotional tone criterion: Very unpleasant versus very or moderately unpleasant

Frequency assessment: Number of occurrences over 14 nights versus Number of nights

All 32 combinations of within-subjects variables were utilised in case the predisposing factors only correlated significantly with some of the WS correlations. WS correlations were converted to Fisher's r values, and these Fisher's r values were correlated with each of the individual difference variables.

The 32 x 17 between-subjects correlations are shown in Appendix Table 13 (placed in appendix due to lack of significant results and large size). A summary of the correlations for each individual difference variable is presented in Table 38. The correlations involving pre-sleep anxiety or depression are summarised separately from the post-sleep mood variables because the former address whether the individual difference variables interact with pre-sleep mood to cause nightmares, whereas the latter address whether the individual difference variables interact with the presence of nightmares to cause more negative waking mood.

Table 38 – Summary of 32 between subjects correlations for each of 17 Individual difference variables. Summary data are the number of the 32 correlations that are significant at the level of p<.05, and, where one or more significant correlations occurred, the highest correlation co-efficient. Highest n for the correlations = 48.

<u> </u>	Correlations with pre-	Correlations with post-
	sleep WS correlations	sleep WS correlations
Individual	Number of significant	Number of significant
difference	positive correlations (and	positive correlations (and
variable	highest r value if	highest r value if
	significant)	significant)
ND	0	0
APS	0	0
CEQ	0	0
STB	1 (r = .31)	0
EPQ-N	0	0
EPQ-L	0	4 (r = .40)
GHQ-12	2 (r = .32)	0
BQ-18	0	0
STAI-S	0	0
STAI- T	0	0
SCL – GSI	0	0
SCL-PST	0	0
SCL-PSDI	0	0
Child	0	0
LE	0	0
RUD	0	0
RNM	0	0

Notes: WS = within subject; ND = nightmare distress; APS = arousal predisposition scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety; STAI-T = Trait anxiety; SCL- GSI = SCL- Global Symptom Index; SCL-PST= SCL- positive symptom total; SCL-PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items. RUD= retrospective unpleasant dream frequency; RNM=retrospective nightmare frequency.

For the cases where more than 1 correlation in one cell is significant it is acknowledged that the 32 correlations summarised in each cell are not independent from each other, as all the correlations have some data in common with some of the other correlations. Some correlations were significantly negative, but as only significantly positive correlations were predicted, and as it is difficult to interpret what significantly negative correlations mean, apart from being a chance effect, those correlations are not recorded.

However, none of the above correlations involving pre-sleep mood were significant at the 0.01 level. This stricter significant threshold was used in order to correct for the large number of correlations. Hence, the hypothesis that individual differences lead to some people being more susceptible to the occurrence of nightmares under conditions of state anxiety or depression was not confirmed. Therefore, it can be concluded that none of the individual difference variables used here interacts with state anxiety or state depression to cause nightmares. Also, none of the above correlations involving post-sleep mood were significant at the 0.01 significance threshold. Hence, individual differences do not lead to some people having a more negative state response to the occurrence of nightmares.

As stated in the text regarding Tables 30 - 32 above, better estimates of within subjects correlations may be provided by using the sub sample of 24 participants that had more than one waking criterion nightmare defined as very high unpleasantness. The correlations between within subjects correlations and well-being and personality variables were repeated with just this sub-sample. These correlations are shown in Appendix Table 14 and summarised in Table 39. Table 39 – Summary of 32 between subjects correlations for each of 17 Individual difference variables. Summary data are the number of the 32 correlations that are significant at the level of p<.05. n for all correlations = 24.

Correlations with pre-	Correlations with post-
sleep WS correlations	sleep WS correlations
Number of significant	Number of significant
positive correlations (and	positive correlations (and
highest r value if	highest r value if
significant)	significant)
0	0
0	0
0	0
0	0
0	0
0	8 (r = .61)
0	0
0	0
0	0
0	0
0	0
0	0
0	0
0	0
1 (r = .41)	0
0	0
0	0
	sleep WS correlations Number of significant positive correlations (and highest r value if significant) 0 0 0 0 0 0 0 0 0 0

Notes: WS = within subject; ND = nightmare distress; APS = arousal predisposition scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety; STAI-T = Trait anxiety; SCL- GSI = SCL- Global Symptom Index; SCL-PST= SCL- positive symptom total; SCL-PSDI = positive symptom distress index; LE = Sum of life events; Child = Sum of childhood items; RUD = retrospective unpleasant dream frequency; RNM= retrospective nightmare frequency.

For the cases where more than 1 correlation in one cell is significant it is acknowledged that the 32 correlations summarised in each cell are not independent from each other, as all the correlations have some data in common with some of the other correlations. Some correlations were significantly negative, but as only significantly positive correlations were predicted, and as it is difficult to interpret what significantly negative correlations mean, apart from being a chance effect, those correlations are not recorded.

The data of Table 39 again show that there are very few significant correlations between the personality variables and the within subjects depression correlations, or with the anxiety correlations. Pre sleep correlations did not remain statistically significant at the stricter significance threshold of 0.01. However, post sleep correlations remained significant after this correction for the large number of correlations. Overall, therefore, using a sub-sample that had somewhat larger and arguably more appropriate within subjects correlations, there was still no evidence that any of the personality variables were correlated significantly with the within subjects correlations. Therefore, it can be concluded that none of the individual difference variables used here interacts with state anxiety or state depression to cause nightmares or unpleasant dreams, and the individual differences used here do not lead to some people having a more negative state response to the occurrence of nightmares or unpleasant dreams.

Predisposition for increased dream recall under conditions of high anxiety or high depression

In order to determine whether for some subjects there is a predisposition to increase dream recall due to pre-sleep anxiety or depression, point biserial within subject correlations between anxiety and depression and presence / absence of a dream the following night were correlated with each of the personality variables. The within subjects correlations of the 51 participants who had a dream on at least one night but not on other nights were first converted into Fishers r values. The WS presleep depression correlation was normally distributed (Shapiro-Wilk statistic = 0.98, df = 51, p = 0.43) and the WS pre-sleep anxiety correlation was approximately normally distributed (Shapiro-Wilk statistic = 0.95, df = 51, p = 0.02). The Fishers r values were then correlated between subjects with each of the individual difference variables. For anxiety as a pre-sleep correlate of dream recall, all the between subjects rs had an absolute value less than 0.16, and were all highly non-significant. For depression as a pre-sleep correlate of dream recall, all these between subjects rs had an absolute value less than 0.17 and again were highly non-significant.

It can thus be concluded that pre-sleep mood not only has a negligible effect on dream recall, but that this effect is not increased for some participants due to some personality susceptibility. The only effect of pre-sleep mood is thus on the presence of nightmares, although this is small, and as with dream recall this effect is not modulated by personality.

The emotional profile of dreams

680 dreams were reported across the 14-day diary period. In these dreams 1396 emotions were reported, which is a mean of 2.05 emotions per dream. 107 dreams contained no emotions. Thus, 84.29% of all reports contained at least one instance of emotion.

Frequency and intensity of discrete emotions

The scoring method of Fosse et al (2001) was used, which records frequency (i.e. number of dreams in which the emotion was present) and intensity (i.e. score on scale of 1 - 3 for when the emotion occurs) of seven major emotions (Anxiety/fear; Anger; Shame; Sadness; Joy/elation; Love/eroticism, and Surprise). Other negative and positive emotions could be reported: in this study other negative emotions reported were frustration, stress, confusion, disgust, pity, jealousy, rejection and irritation. Other positive emotions reported were relief, excitement, interest and comfort. The data are calculated on a per subject basis, that is, the mean of each variable is calculated for each participant, and then the overall mean is calculated with each participant being treated equally, irrespective of each person's number of dreams. This equal representation method is described by Zadra et al (2006).

Two measures of emotion intensity were calculated. One measure assessed the intensity of an emotion only when it occurred (e.g., the sum of the intensity scores for anxiety divided by the number of times anxiety occurred. As intensity can be scored as 1, 2 or 3 this method results in a mean intensity ranging from 1 to 3). The second measure looked at the mean intensity of that emotion across all dream reports, thus, for instances when a dream was reported but not with that particular emotion that dream was included in the denominator for the calculation of the mean. (This method, for example, would calculate the sum of the intensities of anxiety and divide that sum by the total number of dream reports). Shapiro-Wilk tests show that most of the emotion variables are not normally distributed and so non-parametric statistics were used.

Table 40 shows the frequency and intensity (using both intensity measures detailed above) of discrete emotions experienced across all types of dreams in the 14day study period, calculated on a per subject basis as above. Table 40 – Mean frequency and intensity (standard deviation) of discrete emotions experienced in all dream types across the 14 day dream diary.

	Frequency (%)	Intensity when	Intensity across all
		emotion occurred	dream reports
Anxiety/fear	46% (0.23)	2.14 (0.60)	0.99 (0.58)
Anger	25% (0.24)	1.96 (0.66)	0.47 (0.46)
Shame	15% (0.23)	1.64 (0.66)	0.23 (0.32)
Sadness	26% (0.25)	2.06 (0.68)	0.53 (0.48)
Other negative	9% (0.15)	2.02 (0.72)	0.18 (0.34)
Joy/elation	25% (0.27)	1.99 (0.69)	0.47 (0.51)
Love/eroticism	13% (0.24)	1.94 (0.77)	0.22 (0.36)
Other positive	2% (0.05)	1.92 (0.74)	0.04 (0.10)
Surprise	32% (0.28)	1.85 (0.64)	0.32 (0.28)

The order of frequency of the emotion types was:

anxiety/fear > surprise > sadness > anger and joy/elation > shame > love/eroticism > other negative > other positive.

Wilcoxon tests showed that anxiety/fear occurred significantly more often than each of the other emotions (all zs > 3.1 and all ps < .01). Surprise was significantly more prevalent than all remaining emotions except sadness (all zs > 2.0and all ps < .05). Sadness was significantly more prevalent than love/eroticism and joy (both zs > 4.3, both ps < .01). Anger was significantly more prevalent than shame and love (both zs > 3.6, both ps < .01). Joy/elation was significantly more prevalent than love/eroticism (z = 0.4.68; p < .01), and there was no significant difference in frequency between love and shame (z = -1.14; p = 0.15). Anxiety/fear was the most intense emotion, followed by sadness. In general, discrete negative emotions were more intense than positive ones.

Total positive and total negative emotions

The scoring method of Fosse et al (2001) was again used. Scores of frequency of total positive or total negative emotions were calculated for each participant as the total number of dreams that had at least one positive, or one negative emotion respectively. Surprise was considered to be neutral and not included in the positive or negative emotion categories. Intensity of total positive and total negative emotions was calculated as first as the sum of intensity scores within each dream, which were then summed for each participant. Table 41 shows the frequency and intensity scores for the total negative and total positive emotions and surprise.

Table 41 – Mean frequency and intensity (standard deviations) of total negative and total positive emotions and surprise in all dreams across the 14 day dream diary.

	Frequency (%)	Intensity when	Intensity across all
		emotion occurred	dream reports
Total negative	64% (0.21)	2.03 (0.53)	1.21 (0.90)
emotions			
Total positive	19% (0.17)	2.00 (0.66)	0.40 (0.48)
emotions			
Total surprise	32% (0.28)	1.85 (0.64)	0.32 (0.28)
emotions			

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Negative emotions were found to be significantly more prevalent than positive emotions (Wilcoxon test, z = 5.74; p < .01). Indeed, only one subject reported a higher frequency of positive emotions compared to negative. Negative emotions were also significantly more prevalent than surprise (z = -4.64; p < .01). Surprise also occurred significantly more often than positive emotions (z = -3.31; p = <.01). These results are to be expected as the sample consisted predominantly of nightmare sufferers.

In cases where emotions did occur, negative and positive emotions did not differ in intensity (z = -0.12; p = 0.91). Furthermore, neither negative nor positive emotions differed in intensity to surprise (z = -1.32; p = 0.19 and z = -1.92; p = 0.055respectively). However, when the sum of intensity scores were divided by the number of dream reports, the intensity of negative emotions was significantly greater than that of positive emotions (z = -4.87; p < 0.001). Total negative emotions were also significantly more intense than surprise (z = -6.19; p < 0.01). Total positive were

The emotional profile of dreams and psychological well-being

The Spearman's rho correlations shown in Table 42 demonstrate that none of the correlations between frequency measures and measures of well-being or individual differences are significant.

Table 42 – Spearman rho correlations and (ps) between the frequency of negative,

positive and surprise in dreams with measures of well-being and individual

differences.

	FNE	FPE	FSE
ND	0.04 (0.77)	-0.04 (0.76)	0.11 (0.43)
APS	0.22 (0.10)	-0.27 (0.04)	-0.02 (0.99)
CEQ	-0.08 (0.54)	0.16 (0.24)	0.17 (0.21)
STB	0.22 (0.10)	-0.06 (0.66)	-0.13 (0.32)
EPQ-N	0.24 (0.08)	-0.09 (0.49)	-0.08 (0.53)
GHQ-12	0.00 (0.99)	0.15 (0.27)	0.05 (0.70)
BQ-18	-0.02 (0.88)	0.11 (0.41)	0.05 (0.73)
STAI-T	0.28 (0.05)	-0.14 (0.33)	-0. 06 (0.68)
STAI-S	0.27 (0.19)	-0.32 (0.11)	-0.15 (0.46)
SCL-GSI	0.05 (0.70)	0.02 (0.85)	0.02 (0.85)
SCL-PST	0.03 (0.85)	-0.13 (0.93)	0.10 (0.46)
SCL-PDS	0.17 (0.21)	-0.09 (0.49)	0.03 (0.81)
LE	0.01 (0.96)	0.66 (0.63)	0.19 (0.15)

Note: ND = nightmare distress; APS = arousal predisposition scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety; STAI-T = Trait anxiety; SCL- GSI = SCL- Global Symptom Index; SCL-PST= SCL- positive symptom total; SCL-PSDI = positive symptom distress index; LE = Sum of life events;. FNE = total frequency of negative emotion; FPE = Frequency of positive emotion; FSE =total frequency of surprise emotion.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

The Spearman's rho correlations shown in Table 43 demonstrate that the majority of correlations between emotion intensity measures and measures of wellbeing or individual differences are not significant, although adverse life events, adverse childhood experiences and the positive symptom index of the SCL-90-R are significantly associated with one of the measures of intensity of negative emotions. Furthermore, the intensity of surprise when it occurs is significantly associated with the positive symptom index of the SCL-90-R.

Table 43 - Spearman's correlations between individual difference variables and the total intensity of positive, negative and surprise emotions when they occur and per dream report.

		IPE/		INE	IDE	ICE
	INE/		ISE/	INE	IPE	ISE
	DRF	DRF	DRF			
ND	0.25	0.09	0.20	0.30	-0.01	0.22
_	(0.06)	(0.53)	(0.14)	(0.03)	(0.96)	(0.14)
APS	0.15	-0.15	0.09	0.27	0.14	0.20
	(0.28)	(0.27)	(0.53)	(0.04)	(0.34)	(0.18)
CEQ	0.24	0.22	0.23	0.10	0.11	0.27
	(0.07)	(0.09)	(0.09)	(0.44)	(0.47)	(0.07)
STB	0.18	-0.06	-0.04	0.26	0.08	0.28
	(0.17)	(0.64)	(0.79)	(0.05)	(0.56)	(0.06)
	0.22	-0.04	-0.03	0.19	-0.10	0.12
EPQ-N	(0.10)	(0.79)	(0.80)	(0.17)	(0.49)	(0.44)
GHQ-12	0.16	0.18	0.32	0.09	0.23	0.33
	(0.24)	(0.19)	(0.32)	(0.49)	(0.11)	(0.02)
BQ-18	0.21	0.19	0.13	0.21	0.24	0.32
	(0.12)	(0.16)	(0.33)	(0.13)	(0.10)	(0.03)
Trait	0.33	-0.05	0.04	0.22	0.01	0.28
Anxiety	(0.02)	(0.75)	(0.79)	(0.13)	(0.93)	(0.08)
State	0.06	-0.26	-0.11	0.39	0.08	0.03
Anxiety	(0.76)	(0.21)	(0.60)	(0.06)	(0.73)	(0.89)
SCL-GSI	0.19	0.08	0.13	0.27	0.20	0.33
	(0.16)	(0.57)	(0.36)	(0.05)	(0.17)	(0.02)
SCL-PSI	0.19	0.07	0.22	0.21	0.19	0.41*
	(0.15)	(0.60)	(0.11)	(0.13)	(0.19)	(<0.01)
SDL-PDI	0.21	0.00	0.13	0.41*	0.25	0.34
	(0.13)	(0.98)	(0.33)	(<0.01)	(0.09)	(0.02)
Life Events	0.40*	0.22	0.26	0.07	0.05	0.20
	(<0.01)	(0.10)	(0.05)	(0.63)	(0.72)	(0.18)
Child	0.33*	0.14	0.33*	0.13	0.01	0.13
	(0.01)	(0.30)	(0.01)	(0.36)	(0.95)	(0.40)

Notes: INE/DRF= intensity of negative emotions per dream report; IPE/DRF = intensity of positive emotions per dream report; ISE/DRF = intensity of surprise emotions per dream report; INE= intensity of negative emotions when it occurred; IPE = intensity of positive emotions when it occurred; ISE = intensity of surprise emotions when it occurred; ND = nightmare distress; APS = arousal predisposition scale; CEQ = Creative experiences questionnaire; STB = Borderline Personality Scale; EPQ-N = Neuroticism; EPQ-L = Lie Scale; GHQ-12 = General Health Questionnaire; BQ-18 = Boundary Questionnaire; STAI-S = State anxiety; STAI-T = Trait anxiety; SCL- GSI = SCL- Global Symptom Index; SCL-PST= SCL- positive symptom total; SCL-PSDI = positive symptom distress index; LE = Sum of life events; Child= sum of childhood events. *p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Relationships between emotions in dreams and pre- and post-sleep mood

In contrast to measures of well-being and individual differences, Table 44 shows that the frequency of negative and positive emotions (but not surprise) in all dreams is significantly related to mean anxiety and depression before and after sleep.

The intensity of negative and positive emotions that occur are not significantly related to pre- and post-sleep anxiety or depression. However, the difference between intensity of negative and positive per occurrence is significantly associated with post-sleep anxiety and pre-sleep depression and there is a strong trend for an association with pre-sleep anxiety and post-sleep depression (p = 0.02). The intensity of negative and positive emotions per report is not significantly related to anxiety and depression before or after sleep. However, again the difference between the intensity of positive and negative emotions per dream is significantly positively related to anxiety and depression before and after sleep. Thus, it seems that in general elevated levels of depression and anxiety before sleep is related to a higher frequency of negative emotions in dreams, and these in turn are related to elevated post sleep anxiety and depression.

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and positive emotions and mean anxiety and depression pre- and post- sleep.

	Pre-sleep anxiety	Post-sleep anxiety	Pre-sleep depression	Post-sleep depression
Freq NE	0.38* (<0.01)	0.42 *(<0.01)	0.37* (<0.01)	0.35* (0.01)
Freq PE	-0.28 (0.04)	-0.39* (<0.01)	-0.30* (<0.01)	-0.35* (0.01)
Freq SE	-0.21 (0.12)	-0.20 (0.13)	-0.18 (0.18)	-0.14 (0.29)
Difference in NE and PE	0.37* (<0.01)	0.45* (<0.01)	0.37* (0.01)	0.38* (<0.01)
Intensity NE per dream	0.29 (0.03)	0.26 (0.05)	0.21 (0.13)	0.21 (0.13)
Intensity PE per dream	-0.16 (0.23)	-0.27 (0.04)	-0.24 (0.07)	-0.27 (0.04)
Intensity SE per dream	0.01 (0.93)	-0.05 (0.71)	-0.08 (0.57)	-0.04 (0.76)
Difference in NE and PE per dream	0.44* (<0.01)	0.45* (<0.01)	0.34* (0.01)	0.36* (0.01)
Intensity NE per occurrence	0.28 (0.03)	0.27 (0.04)	0.24 (0.07)	0.36 (0.02)
Intensity PE per occurrence	-0.04 (0.78)	0.00 (0.99)	-0.04 (0.77)	0.05 (0.72)
Intensity SE per occurrence	0.15 (0.32)	0.10 (0.52)	0.06 (0.69)	0.12 (0.43)
Difference in NE and PE per occurrence	0.34 (0.02)	0.35* (0.01)	0.38* (0.01)	0.35 (0.02)

Note: Freq NE = frequency of negative emotion; Freq PE= frequency of positive emotion; Freq of surprise

emotion; NE= negative emotion; PE= positive emotion; SE =surprise emotion.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of

significance was set at p = <0.01). All values are two tailed.

Comparisons between nightmares, bad dreams, and non-unpleasant dreams

The following section addresses the frequency and intensity of emotions in nightmares and bad dreams.

Table 45 - Frequency (and standard deviation) of discrete emotions and overall negative, positive and surprise emotions in nightmares, bad dreams and all other dreams. All other dreams are defined as dreams with emotional hedonic tone > 2, i.e. dreams rated as mildly unpleasant to very pleasant.

	Nightmares	Bad dreams	All other
			dreams
nxiety/fear	82% (0.26)	61% (0.45)	33% (0.27)
Anger	39% (0.39)	42% (0.43)	19% (0.24)
hame	24% (0.39)	17% (0.35)	15% (0.26)
Sadness	47% (0.41)	34% (0.44)	21% (0.24)
)ther negative	6% (0.19)	15% (0.31)	10% (0.17)
oy/elation	13% (0.30)	8% (0.23)	30% (0.30)
ove/eroticism	17% (0.36)	10% (0.28)	14% (0.26)
)ther positive	3% (0.16)	1% (0.06)	2% (0.05)
Surprise	26% (0.36)	17% (0.33)	32% (0.30)
INE	85% (0.16)	80% (0.31)	54% (0.25)
ГРЕ	7% (0.11)		24% (0.19)

and all other dreams are shown in Figure 2.

Figure 2 - Frequency of discrete emotions in nightmares, bad dreams and all other

dreams.

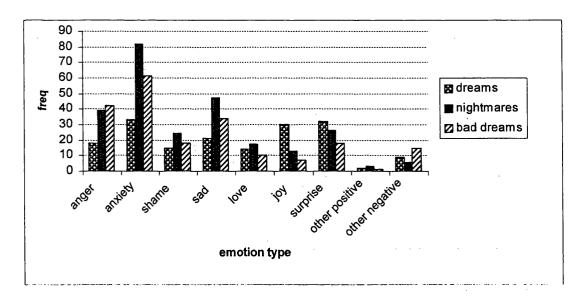


Table 45 and Figure 2 show that nightmares and bad dreams contain more instances of discrete negative emotions and less instances of positive emotions than other dreams. These descriptive statistics demonstrate that although anxiety/fear is the most prevalent emotion, other negative emotions occur frequently. This has implications for defining nightmares as containing solely anxiety or fear.

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Table 46 - Mean intensity (Standard deviations) of emotions when they are present,

for nightmares, bad dreams and all other dreams.

	Nightmares	Bad dreams	All other
			dreams
Anxiety/fear	2.61 (0.47)	2.28 (0.76)	1.81 (0.58)
Anger	2.23 (0.82)	2.00 (0.73)	1.73 (0.69)
Shame	1.90 (0.90)	1.98 (0.90)	1.54 (0.62)
Sadness	2.51 (0.60)	2.03 (0.84)	1.82 (0.67)
Other negative	2.67 (0.82)	2.28 (0.67)	1.89 (0.72)
Joy/elation	1.40 (0.70)	2.00 (1.00)	2.06 (0.66)
Love/eroticism	1.45 (0.69)	1.89 (0.93)	1.98 (0.81)
Other positive	2.00 (1.41)	2.00 (1.41)	2.00 (0.71)
Surprise	1.90 (0.72)	2.06 (0.88)	1.79 (0.68)
Total negative	2.47 (0.48)	2.10 (0.68)	1.80 (0.51)
emotions			
Total positive	1.43 (0.64)	2.33 (0.83)	1.79 (0.52)
emotions			

.....

Table 47 - Mean intensity (and standard deviations) of emotions per dream, for nightmares, bad dreams and all other dreams (e.g. the intensity of emotions was divided by the number of nightmares).

	Nightmares	Bad dreams	All other
			dreams
Anxiety/fear	2.16 (0.82)	1.39 (1.16)	0.39 (0.36)
Anger	0.81 (0.90)	0.87 (1.01)	0.17 (0.24)
Shame	0.40 (0.69)	0.34 (0.73)	0.10 (0.15)
Sadness	1.18 (1.06)	0.71 (1.04)	0.20 (0.23)
Other negative	0.18 (0.56)	0.34 (0.73)	0.12 (0.24)
Joy/elation	0.19 (0.54)	0.29 (0.76)	0.35 (0.38)
Love/eroticism	0.26 (0.61)	0.18 (0.55)	0.11 (0.19)
Other positive	0.04 (0.18)	0.03 (0.16)	0.04 (0.11)
Surprise	0.46 (0.65)	0.37 (0.75)	0.38 (0.50)
Total negative	4.61 (2.35)	3.84 (2.61)	1.00 (0.57)
emotions			
Total positive	0.48 (1.08)	0.51 (1.26)	0.51 (0.43)
emotions			

There are only a few significant differences between nightmares and bad dreams in Tables 45 - 47 and Figure 2. Anxiety/fear occurs significantly more often in nightmares than bad dreams, however, the frequency of all other emotions was not significantly different between bad dreams and nightmares. When anxiety occurs in nightmares it is more intense that when it occurs in bad dreams. There is also a trend for sadness to be more intense when it occurs in nightmares compared to bad dreams, however, this just missed statistical significance (p = 0.05). There is a trend for anxiety to be more intense in nightmares, however, this is not significant (p = 0.08). The intensity of anger per dream is higher in bad dreams than in nightmares.

Details of the Wilcoxon tests for these differences are reported in Appendix Table 15. In general, frequency and intensity of positive emotions were lower in nightmares and bad dreams than in all other dreams, and frequency and intensity of negative emotions were higher in nightmares and bad dreams than in all other dreams.

<u>Comparisons between emotions in non-nightmare non-bad dreams and the normative</u> data of Fosse et al (2001)

In the previous section the dreams rated as pleasant, neutral or just mildly unpleasant were compared to nightmares and bad dreams. This raises the question of how these non-nightmare non-bad dreams compare to the normative data in ordinary dreamers of Fosse et al (2001). Figures 3 and 4 show the frequency of emotions and their intensity when they occur in non-nightmare non-bad dreams from the current 1 study, and the data reported by Fosse et al. Figure 3 – Frequency of discrete emotions in dreams not rated as very or

moderately unpleasant, a comparison with data from Fosse et al (2001).

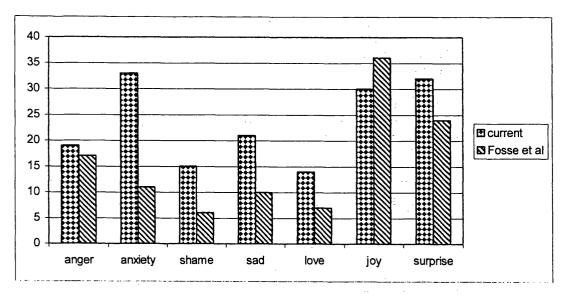
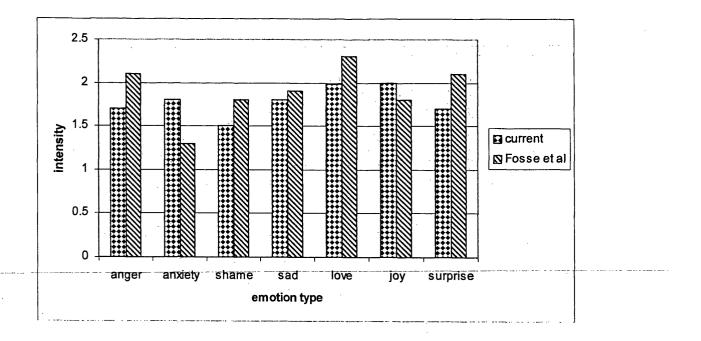


Figure 4 – Intensity of discrete emotions when they occur in dreams not rated as very or moderately unpleasant, a comparison with data from Fosse et al (2001).



Anxiety, shame and sadness occur more frequently in the non-nightmare non-bad dreams from the current study than in Fosse et al (2001), although only anxiety is more intense. It can be concluded that even the non-nightmare non-bad dreams of our sample are more negatively toned than for non-nightmare sufferers.

Chapter 5

Study 1: Personality, psychopathology and

<u>nightmares</u>

Discussion

Retrospective and prospective measures of nightmare frequency

The first aim of this study was to assess correlations of retrospective and prospective frequency variables with measures of psychological well-being and other individual difference variables. The current study found that retrospective measures produced significantly lower estimates of waking criterion nightmares defined as moderately or very unpleasant, compared to daily logs. However, if waking criterion nightmares were defined only as very unpleasant, the retrospective estimate was no longer significantly lower than the logs. Thus, when nightmares were defined more loosely, logs yielded a prevalence that was 95% higher than retrospective reports, however, a stricter definition on the logs yielded a frequency only 25% higher than the retrospective estimate. There was no significant difference between the frequency of dreams or bad dreams between prospective and retrospective methods.

These results offer support for previous studies showing lower frequency estimates produced by retrospective reports compared to logs. Blagrove et al (2004) found that logs produced a mean estimate of nightmare frequency (using the waking criterion) that was 25% higher than the questionnaires. Wood and Bootzin (1990) found that retrospective self-reports produced an estimation of nightmare frequency that was 2.5 times lower than the log estimate. Salvio et al (1992) also reported that nightmares were 10 times more prevalent than had previously been estimated using retrospective measures. Zadra and Donderi (2000) also found that retrospective reports produced lower estimates of nightmare frequency but that this difference was greater for nightmares compared to bad dreams. However, in contrast to the Zadra and Donderi study, the current study did not find a significant difference between the estimated frequency of bad dreams between prospective and retrospective measures.

Zadra and Donderi (2000) suggested that nightmares might produce lower estimates compared to bad dreams because of the stronger negative dream content of nightmares. However, Blagrove et al (2004) and the current study found that the lower estimates of nightmare frequency produced by retrospective reports may be a simple function of forgetting over time rather than a result of the stronger emotional content of nightmares. However, it may also be the case that keeping a log increases the frequency of nightmares.

In Zadra and Donderi's (2000) study it could be argued that estimates of bad dream frequency are not as low as nightmare frequency because their sample had many more bad dreams than nightmares (a mean of 0.92 nightmares a month compared to 2.45 bad dreams). Thus, participants may have had more access to instances of bad dreams in memory and so retrospective underestimation is less. However, in this study participants had more nightmares than bad dreams (a mean of 3.3 nightmares were reported per month and 1.3 bad dreams) and nightmare frequency was still underestimated in relation to bad dreams. Therefore, limited access to instances of these dream types does not seem to be able to account for the specific underestimation of nightmares.

There are a number of possibilities concerning the different estimations of nightmare frequency found between retrospective reports and daily logs in the current study. The first concerns a methodological problem. Participants were asked to estimate the percentage of their nightmares (not defined with a waking criterion) that wake them up. The scale ranged from 0-100% going up in intervals of 20. As intervals goes up by 20% this only allows for a very rough measurement of how many nightmares caused awakening. This could have led to errors in calculating retrospective bad dreams and nightmare frequency. Alternatively, it could be that participants have difficulty retrospectively estimating the number of dreams that specifically cause them to wake up. Blagrove and Haywood (2006) assessed whether people believe they are able to judge if the imagery or emotion of a dream caused them to wake up. They found that judgements of being woken by a dream were made with high certainty particularly for very unpleasant dreams. However, such judgements may not be as accurate when assessed retrospectively. Alternatively, it may be that keeping a log increases the number of unpleasant dreams or that ratings are more severe immediately after or on the same day that an unpleasant dream has occurred as the dream is still in mind. This contrasts with making a retrospective rating when the emotion of the dream is not so raw. Furthermore, individuals with nightmares are often highly distressed by their experiences and so the number of nightmares may be embellished in order to gain access to help. Logs often ask details about the dream (including sometimes content) which may lead to a secondary assessment of the dream experience, for example, writing down traumatic nightmare

scenes may lead one to re appraise the emotional intensity of the dream again and rate it as more severe /unpleasant. Thus, this study and previous research has demonstrated that retrospective and prospective measures produce different estimates of nightmare frequency, however, some of the above arguments caution that it should not be assumed that one measure is more accurate than the other. Future nightmare research would benefit from using both measures.

<u>Correlations between nightmare/bad dream/unpleasant dream frequencies and</u> well-being and individual difference variables.

The current study found that frequency of retrospective and log very unpleasant nightmares had higher correlations with psychological well-being measures than the frequency of retrospective and log very unpleasant bad dreams. However, when the definition of nightmares and bad dreams both included moderately and very unpleasant dreams, correlations with psychological well-being were similar.

The finding that very unpleasant nightmares had stronger correlates with wellbeing than do very unpleasant bad dreams is in line with Zadra and Donderi (2000). These findings were used as support for the waking criterion in the definition of nightmares, in that if researchers restrict their definitions to those disturbing dreams that waken the sleeper then the results may be confounded by having participants with bad dreams in the control group thus inflating the well-being of this comparison group. However, this study did not support the use of the waking criteria in that on both retrospective and prospective measures, unpleasant dream frequency, that is disturbing dreams that may or may not awaken the sleeper, have higher correlations with psychological well-being than do waking criterion nightmares or bad dreams alone. This is in line with the findings of Blagrove et al (2004) who found that the frequency of all distressing dreams, whether or not the negative emotion woke the dreamer was an even better index of psychopathology than nightmare frequency defined with the waking criterion only.

Nightmare frequency, nightmare distress and psychological well-being.

The current study assessed a number of measures of well being including, neuroticism, general symptoms of mental health, trait and state anxiety and borderline personality.

One aim of the current study was to assess whether other characteristics of unpleasant dream types might be better indicators of psychopathology than frequency variables.

Previous research has shown that whether a nightmare is reported is more dependent on an individual's global dream processes and attribution than by the phenomenal qualities of that specific nightmare (Levin & Fireman, 2002a).

Study one found medium to moderate correlations between retrospective and prospective frequency variables with measures of psychological well-being. This section will discuss between-subjects relationships of well-being with log measures defined as very unpleasant as these tended to be stronger than relationships with frequency measures defined as both moderately unpleasant and very unpleasant.

Nightmare distress was significantly positively associated with all measures of psychological well-being and was the strongest correlate of well being measures. Furthermore, all significant relationships with nightmare distress remained when frequency was controlled for (with the exception of the arousal predisposition scale). Unpleasant dream/ nightmare frequency measures were found to be better correlates of poor well-being than was mean emotional tone of dreams.

When nightmare distress was partialled out of the relationship between retrospective frequency measures and psychological well-being, trait anxiety, state anxiety, and two of the global scales of the SCL-90-R remained significantly correlated. Thus, with a few exceptions, poor waking psychological well-being was associated with higher unpleasant dream/nightmares as estimated retrospectively.

When nightmare distress was partialled out of the relationships of log unpleasant dream frequency with well-being variables, the correlations became smaller, although mostly they remained significant. When nightmare distress was partialled out of the relationships with log nightmare frequency and well-being measures, some of these become insignificant. However, correlations with neuroticism, state and trait anxiety, somatization and SCL- anxiety all remained significant. These findings support the intuitive hypothesis that poor waking well-being is associated with an increased frequency of 'very unpleasant' dreams and, furthermore, that this association is not solely a matter of how distressed one is by nightmares, a possibility supported by the results of Blagrove et al (2004).

Furthermore, all significant association between unpleasant dream frequency and well-being remained when mean dream emotional tone was partialled out, with the exception of the obsessive compulsive, interpersonal sensitivity and paranoid ideation scales of the SCL-90-R. When emotional tone was partialled out of the relationships between well-being and nightmare frequency, the arousal predisposition scale, state and trait anxiety and the positive symptom distress index of the SCL-90-R became insignificant. Thus, when the mood of all dreams is partialled out, the frequency of these extreme unpleasant dream types continues to be related to the majority of well-being measures. Furthermore, when the frequency of unpleasant dreams was partialled out of the relationships between mean emotional tone and wellbeing measures all associations become insignificant.

These partial correlations provide further evidence for the importance of unpleasant dream/nightmare frequency as a dream measure that is associated with waking poor well-being. In these correlations it was found that in general the unpleasant dream/nightmare partial correlations remained significant, whereas, the mean tone partial correlations did not. These results can be explained in terms of the distribution of mean emotional tone of dreams across the sample of dreams. The mean tone of dreams is slighter more negative for people who have poorer well-being. However, the bell-shaped curve of the mean tone values results in there being a large proportional increase in the extreme cases, nightmares/unpleasant dreams, for what is a relatively small change in mean tone. This supports the contention of Zadra and Donderi (2000) that it is the number of extreme types of dreams that correlate more highly with psychological well-being than mean dream emotional tone. However, the results of study one oppose those of Zadra and Donderi (2000) on the need to define the extreme dreams as not only unpleasant but also causing waking. These results support the general tenor of work over recent decades, which specify the frequencies of nightmares (Hartmann, 1984) and reactions to nightmares (Belicki, 1992a, b) as the main correlates of poor well-being.

The findings of this study are in accordance with Levin and Fireman (2002a) and Blagrove et al (2004) in that correlations between nightmare frequency and psychopathology become smaller when nightmare distress is controlled for, and that the relationships between psychopathology and nightmare distress were stronger than with frequency measures. Blagrove et al (2004) found that correlations between unpleasant dream frequency and well-being were maintained when nightmare distress was partialled out, whereas, correlations between nightmare frequency and well-being became insignificant. This study offers some support to these conclusions in that unpleasant dream frequency had higher and more significant correlations with wellbeing when nightmare distress was partialled out compared to nightmare frequency, however, the majority of significant correlations remained significant with nightmare frequency. However, when frequency measures were used which defined nightmares as very unpleasant and moderately unpleasant, as in Blagrove et al (2004), all correlations between unpleasant dream frequency and nightmare frequency measures become insignificant, with the exception of neuroticism and the social desirability

scale. However, the unpleasant dream and nightmare frequency measures including 'moderately unpleasant' dreams used in this study had similar correlations with psychopathology, whereas, in Blagrove et al's study unpleasant dream frequency was a higher correlate of well-being than nightmare frequency.

Levin and Fireman (2002a) also found that both nightmare frequency and nightmare distress were significantly associated with most measures of psychological disturbance (including the SCL-90-R), and that these relationships were often stronger for the distress dimension, although theirs was a state distress measure. However, Belicki (1992a) found that only nightmare distress and not frequency, was associated with the number of symptoms presented on the SCL-90-R. One possible explanation for this may be that arguably the volunteer sample used in this study is more extreme than the 85 undergraduate students used by Belicki (1992a) who received course credit for their participation. Furthermore, Belicki's participants reported fewer nightmares than the current study and relied upon a retrospective measure of nightmare frequency.

Schizophrenia spectrum

As previously reviewed there are some lines of evidence to suggest that frequent nightmares may have a specific association with schizophrenic spectrum disorders, and, in particular, schizotypy (Claridge et al, 1997; Fennig et al, 1992; Hartmann, 1984; Hartmann et al, 1981, 1987; Heinrichs & Carpenter, 1985; Herz & Melville, 1980; Kales et al, 1980; Levin, 1990, 1998; Levin, Galin & Zywiak, 1991; Levin & Raulin, 1991; Levin & Stiritz, 1998). Some investigators have suggested that nightmares and schizophrenia may share a common biology (Doty, 1989; Fischman, 1983; West, 1962). However, other studies argued that frequent nightmares are reflective of a 'global maladjustment rather than specific psychotic symptomology' (Berquier & Ashton, 1992). The results of the current study are in line with the assertion that frequent nightmares are associated with general psychopathology rather than specifically psychotic symptomatology: state and trait anxiety, neuroticism and all subscales of the SCL-90-R except hostility, paranoid ideation were correlated with the frequency of unpleasant dreams, and the highest correlations were for the anxiety scale rather than psychoticism, even when nightmare distress was partialled out..

Levin and Fireman (2002a) found that with the exception of the phobia subscale on the SCL-90-R, nightmare distress was significantly associated with all measures of psychopathology, however, in contrast to this study, they found that nightmare frequency was not related to either measure of SCL-90-R depression (or the BDI), state or trait anxiety or the psychoticism scale on the SCL-90-R.

It could be the discrepancies between the current study and Levin and Fireman (2002a) is a result of the more extreme sample recruited in this study. Levin and Fireman recruited undergraduate students who received course credits for their participation and were required to have at least 3-10 nightmares a year. However, this study advertised for those who suffer from frequent nightmares and those that do not to volunteer in a research study. It is possible, therefore, that the sample used in study one contained individuals who were more concerned by their nightmares. Levin and Fireman report that that using the guidelines (Derogatis, 1994) for identifying clinical cases on the SCL-90-R that 55.6% of their sample met the criteria for 'caseness', whereas, this study found that 69% of the sample met the same criteria for 'caseness'. Furthermore, Levin and Fireman (2002a) defined nightmares as 'a scary dream that awakens the dreamer from sleep', thus restricting this definition to waking criterion nightmares may have reduced relationships with psychopathology. Moreover, it is likely, with the use of this inclusion criterion, that individuals experiencing bad dreams but no nightmares could have ended up in one of the comparison groups, thus elevating psychopathology scores in those groups. Also, the definition of nightmares used in Levin and Fireman's (2002a) study suggests that fear/ anxiety must be the dominant emotion to be classified as a nightmare, thus very disturbing dreams dominated by other negative emotions would not be classified as a nightmare. Moreover, although these authors found no relationship with state and trait anxiety measures they found SCL-90-R anxiety was significantly higher in the high nightmare group. To summarise, however, the current study and Levin and Fireman's study both reached the same general conclusion; that there was no evidence to suggest a specific relationship between nightmare frequency and psychosis proneness.

The notions that frequent nightmares appear to be indicative of a general psychopathology rather than specific psychotic symptomatology is supported by Berquier and Ashton (1992). These authors found that their subjects scored significantly higher on the EPQ Neuroticism scale and on the 8 clinical scales of the MMPI. Frequent nightmare sufferers were not found to be significantly higher on psychoticism scales. It is perhaps important to note that similar samples were used in both these studies. Berquier and Ashton used a sample of 30 non-clinical adults with a mean age of 34.7 (ranging from 19-63). Similarly, study one consisted of a sample of non-clinical adults with a mean age of 39.18 (ranging from 18 – 82). Both studies also used advertisements to recruit participants.

Borderline Personality

This study hypothesised that borderline personality would be associated with both the frequency and distress dimension of nightmares/unpleasant dreams. The finding that borderline personality (as measured by the STB) was significantly associated with nightmare distress (r = 0.44; p <0.001) is in line with the findings of Claridge et al (1997) who also found a significant positive correlation between nightmare distress and borderline personality (r = 0.39, N = 179; p <0.001). Furthermore, the results from the current study are in accord with Claridge, Davies, Bellhouse and Kaptein (1998), who found that relationships of borderline personality, nightmare distress and childhood sexual abuse and neglect were all significantly and positively associated. The current study has extended previous findings by confirming a relationship between unpleasant dream frequency and borderline personality, which remains when nightmares distress and mean dream emotional tone are controlled for.

Childhood measures

Four questions were asked concerning participants perceptions of their childhood. These included feeling 'unloved or unwanted', being 'unreasonable published' or experiencing any type of abuse. Childhood items were significantly correlated with retrospective nightmare frequency and log bad dream frequency (defined as very unpleasant and moderately unpleasant); however, all correlations became insignificant when nightmare distress was partialled out. Nightmare distress was the strongest correlate of childhood items and these correlations remained when frequency variables were controlled for.

Therefore, it can be concluded that a history of childhood adversity is related to how distressed one is by nightmares but does not lead to an increased frequency of nightmares in adulthood. The lack of a relationship with nightmare frequency was not expected, given that childhood trauma increases the risk for the development of anxiety disorders (Bremmer et al, 1993; Kendler et al, 1992; Kessler et al, 1997; Yehuda, 2004), but the relationship with nightmare distress may result from participants with a history of childhood adversity having a generally increased emotional reactivity to all aversive stimuli. Previous studies have found that patients with childhood trauma and adult anxiety disorders show an increased sympathetic nervous system response to psychological stress during adulthood (Bremmer et al. 2003; Heim et al, 2002; Metzger et al, 1999). A very interesting study by Otte et al (2005) may offer insight into the relationship between nightmare distress and childhood trauma. They examined the salivary cortisol and 3-methoxy-4-hydroxyphenylglycol (MHPG, the major metabolite of noradrenaline) response to laboratory stress challenges in police academy recruits without current psychopathology during academy training, before actual critical incident exposure. They found that a history of childhood trauma in these recruits without current psychopathology was associated with an increased catecholamine response to simulated police-related stress. They propose that the mechanism by which childhood trauma exposure could increase the catecholamine response to adult stressors probably involves the amygdala. It has been suggested that prior trauma-induced activation of the amygdale enhances neuroendocrine and sympathetic nervous system responses to subsequent novel stressors through CRF pathways to the hypothalamus and locus ceruleus (Sanchez et al, 2001). Otte et al (2005) suggest that one possible explanation for their findings is that trauma exposure during childhood, a period of heightened plasticity of

corticolimbic neuronal systems, increases amygdala reactivity in adulthood to novel stressful or neutral situations, so that subsequent responses are affected.

The increased catecholamine response after childhood trauma could also be explained by the development of fear networks that contain information about stimuli, responses and meanings related to emotional events (Foa et al, 1989). Such networks are characterised by mental representations that are readily activated by many internal and external cues. Childhood trauma might lead to a lower threshold for interpreting stimuli as threatening, and thus attention may be biased towards searching for and identifying threatening information, leading to a higher catecholamine response (Foa et al, 1989). Such an explanation could also help to explain the relationship found in this study between life events and nightmare distress, which is described next.

Life events.

Participants were given a checklist of 14 life events in order to determine if they had experienced any of them within the previous year. 91.2% of the sample reported that they had experienced at least one of the life events listed in the previous year. The life events variable was the sum of the number of events that the person indicated had occurred.

Nightmare distress had the strongest relationship with life events, which remained when frequency measures were controlled for. Retrospective frequency measures were significantly correlated with life events but log frequency measures were not. When nightmare distress was partialled out retrospective unpleasant dream

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and bad dream frequency both remained significantly correlated with life events. Adverse life events score was not significantly correlated with dream emotional tone.

Although there does seem to be some relationship here with retrospective frequency a measure a caution is needed. It may be that this correlation is confounded by willingness or tendency to remember negative events, and that the non-significant relationship between log frequency measures and adverse life events is the truer finding than that of the retrospective nightmare and unpleasant dream measures. However, this issue is still open because of past findings relating nightmare frequency to waking stress.

For example, Chivers and Blagrove (1999) found that that nightmare frequency was significantly correlated with stress as measured by the General Health Questionnaire (which examines current life events rather than stable traits), and Hartmann (1984) reported periods of stress were a precipitating factor for increased nightmares in his nightmare sufferers. Cernovsky (1984) found correlations between life events and sleep disturbances especially the occurrence of nightmares. Picchioni et al (2002) found a significant relationship between nightmares (intensity and frequency and daily stressors (using the Hassles and Uplifts Scale; Delongis et al, 1988). Krakow et al (1993) reported that 12 out of 20 nightmare sufferers had reported a traumatic incident or stressful period prior to the nightmare. These included job loss, pregnancy, death of a loved one, divorce, physical abuse, burglary, rape, starting college or a new job. In line with this research Kales et al (1980) reported that of 60% of their sample reported the occurrence of a major life event preceded the onset of their nightmares. Köthe & Pietrowsky's (2001) reported that 31.6% of the nightmares were considered to be associated with a previous stressful occurrence and 75.5% of the nightmares were thought to reflect something within the participants' life. However, using the 'Social Readjustment Rating Scale' (SRRS; Holmes & Rahe, 1967), Köthe & Pietrowsky found no relationship between life events and frequency of nightmares. Such a negative finding was also reported by Belicki (1987). Furthermore, Belicki (1992a) and Cernovsky (1984) also found no relationship between nightmare frequency and recent life stress.

As a caveat here, it is accepted that the simple summing of number of life events ticked is a coarse procedure for the summarising of the life events items, and that the life events could have been weighted to take the severity of the life event into account. For example, the death of a close family member or friend is more severe than a minor problem at work. However, as individual differences in response to the life events are not known, and as there are individual differences for criteria used to tick some of the items, it was decided that the scale already has some coarseness in its application, and that a more sophisticated weighting formula would still retain some of these uncertainties.

Non-well being Individual Differences

In addition to well-being measures this study also assessed some nonpsychopathological measures of individual difference. These included the Arousal Predisposition scale, the Creative Experiences Questionnaire and the Boundaries Questionnaire.

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Arousal Predisposition Scale

This scale was significantly positively correlated to retrospective unpleasant dream and bad dream frequency and to prospective unpleasant dream and nightmare frequency measures, including dream frequency. However, all these correlations become insignificant when nightmare distress was controlled for, with the exception of log dream frequency and log unpleasant dreams rated as very unpleasant. These findings are in line with Coren (1988). He found significant correlations between the APS score and the frequency of nightmares, and Hicks, Fortin and Brassington (2002) reported that their high arousability group were significantly more likely to experience fantasy nightmares, post traumatic nightmares and night terrors than the low arousability group. However, these studies did not control for the influence of nightmare distress on these relationships. The current study thus extends these findings by suggesting that the influence of arousal is through nightmare recall but also in producing more very unpleasant dreams.

In contrast to the Hicks, Fortin and Brassington (2002), this study found that dream frequency was significantly positively correlated with total APS score. One reason for this may be the difference in reporting methods; this study used two-week diaries, whereas, Hicks et al. asked participants to respond on a 5-point likert scale to the retrospective question 'I remember my dreams', and on which participants may vary in what they consider to be very frequent or infrequent. It may be that this measure of cognitive arousal is significantly related to dream recall in this study as the scale was designed to predict sleep difficulties. Hence, those participants with high arousability scores may have more nocturnal awakenings and hence, remember more dreams. This notion is supported by Coren (1988) finding in two sample of university students that total APS scores was significantly positively related to frequent night awakening (r = 0.40 and r = 0.32). This is in line with the evidence that poor sleepers and insomniacs have higher dream recall, which is reviewed in the introduction to the next study.

Creativity

The creativity questionnaire was significantly positively related to retrospectively measured nightmare and unpleasant dream frequency but to none of the log measures. However, none of these retrospective correlations remained significant when nightmare distress was controlled for, whereas nightmare distress remained significantly related to creativity when all frequency measures were partialled out. The findings are in accordance with Hartmann (1984) and Belicki and Belicki (1982) who suggest a relationship between nightmares and creativity, however, the current study indicates that this relationship is mediated by nightmare distress.

Boundaries Questionnaire

In the current study the BQ-18 was only significantly related to log bad dream frequency and not to any of the retrospective frequency measures. However, this association did not remain when nightmare distress was partialled out. This is in contrast with Hartmann (1989) and Levin (1991) who also found that frequent nightmare sufferers reported evidence of thinner boundaries as measured by the Boundary Questionnaire. The correlation between nightmare distress and BQ remained significant when nightmare frequency and unpleasant dream frequency measures were partialled out. This study therefore shows that the tendency for permeable cognitive boundaries is related more strongly to nightmare distress rather than the frequency of nightmares.

The findings are in accordance with Belicki (1992a) in that she demonstrated a significant relationship between boundaries and nightmare distress. However, in line with the findings from the current study she did not find a significant relationship between boundaries and the frequency of nightmares..

In contrast to Hartmann, Elkins and Garg (1991), who demonstrated that thick boundaried participants report fewer dreams than thin boundaried subjects, this study found no significant association between dream frequency and thin boundaries (although there was a trend in this direction for retrospective estimates (p = 0.07)). This is in line with Beaulieu-Prévost & Zadra (in press) who report that although many studies have reported positive correlations between dream recall frequency and measures of absorption and psychological boundaries, a majority of these studies have relied exclusively on retrospective measures. They found that scores on measures of absorption and psychological boundaries are not related to log dream recall frequency, but rather to people's tendency to underestimate or overestimate their dream recall frequency on retrospective questionnaires.

Between subjects correlations of mood before and after sleep Mean anxiety and depression were generally found to be significantly higher in the morning compared to before sleep. Mean dream emotional tone was the strongest correlate with mean state anxiety and depression ratings before and after sleep. Nightmare distress was also significantly correlated with these state mood

measures. All these significant associations remained significant when frequency measures were controlled for. Retrospective frequency measures were not significantly related to mood before and after sleep. However, all log frequency measures, including dream frequency were significantly related to mood before and after sleep. These correlations remained significant when nightmare distress was partialled out, however, they generally became insignificant when emotional tone was partialled out.

These findings suggest that that mean state anxiety and depression levels before and after sleep are generally more related to the tone of all dreams rather than to the frequency of nightmares or unpleasant dreams. This is not in agreement with Zadra and Donderi's (2000) contention that it is the number of extremely unpleasant dreams/nightmares, rather than mean dream tone, that is the best correlate of poor waking mood.

Within subjects correlations of mood before and after sleep

The analysis of within subject correlations showed that the presence or absence of a nightmare or unpleasant dream has a significantly greater association with anxiety and depression after sleep than before sleep.

The importance of the post-sleep correlations is in line with Köthe and Pietrowsky (2001), who found that state anxiety scores after a night with a nightmare were significantly higher compared to those after a night without a nightmare. They reported that following a nightmare, participants felt more aroused, sad, anxious, less relaxed, less self-confident and less content than after a night without a nightmare. This finding is also in line with Kramer's (1993) Mood Regulatory theory. Kramer argues that dreaming functions to protect sleep by absorbing the emotional surge that occurs during REM sleep and that nightmares represent a failure in this function. Kramer and Roth (1980) found that the decrease in unhappiness from night to morning was related to whom, and to a lesser extent what we dream about. He suggests that dreaming is related to emotional alterations and in most people there is a decrease in negative mood from night to morning. However, in the case of nightmares they suggest that the dreaming process has not be effective and so may lead to more negative mood states the following day. The findings on the current study are in line with the notion that nightmares are related to significantly poor mood the following day.

Within subjects correlations between waking mood and presence/absence of a nightmare/unpleasant dream were not different if dreamless nights were included or not included in the analysis. This indicates that waking mood had no relationship with the presence of a dream, and, indeed, when within subjects correlations were computed between waking mood and presence/absence of a dream, these correlations were found to be negligible. Therefore, previous associations of low waking mood with dreaming (Cohen, 1974a, b) were not supported. Hence, it seems that the only effect of pre sleep mood is on the presence of nightmares/ unpleasant dreams, although this is small.

Cellucci and Lawrence (1978) used participants who reported two or more nightmares per week who recorded dreams and mood for 8 weeks. In their study participants were required to rate on a 10-point scale their general (average) and maximum anxiety for that day. Nightmares were defined as 'subjectively disturbing anxiety provoking dreams from which a person usually awakens. As well as indicating the presence of a nightmares, participants were required to rate the intensity of the nightmare on a 10-point scale. These authors found within subject support for a relationship between anxiety and nightmares, with daytime general anxiety correlating moderately (0.34 - 0.42), and maximum anxiety in the day correlating moderately (0.30 - 0.40) with presence of a nightmare the following night. These correlations are higher than in the current study. The difference between the findings of Cellucci and Lawrence (1978) and the current study could be a result of the different measures of anxiety used, general or maximum anxiety rather than state anxiety here. Also, their length of diary keeping was far longer.

This study was the first to address the issue of the nightmare waking criterion in a within subjects manner. In accordance with the between subjects results of Blagrove et al (2004) and Blagrove and Haywood (2006), this study found that use of the waking criterion did not increase the within subjects association with anxiety or depression when awake. Also, and moving further from the importance of strict definitions of nightmares hypothesised by Zadra and Donderi (2000), there was no significant difference between WS correlations defined with very unpleasant emotional tone versus defined with very or moderately unpleasant tone.

Comparison of Within Subject and Between Subjects correlations

Pre-sleep mood between-subjects correlations were far higher than withinsubjects correlations, whereas for post-sleep mood the between-subjects and withinsubjects correlations were similar in size. The latter result indicates that reactions to dreams can either be assessed immediately or as a mean reaction to a mean type of . dream, and that both methods indicate the effect of dream emotions on waking emotions. This accords with Wasserman and Ballif (1984-85), who had participants attribute their moods to 1 of 10 possible causes (e.g., interpersonal relationships, personal concerns) and to rate the influence of their dreams for 28 days. Across Ss and days, 11% of recalled dreams were perceived by Ss as the most influential cause of their moods as compared to other causes, and 35% of recalled dreams were considered moderately influential.

The results concerning pre-sleep variables and nightmares/unpleasant dreams, however, are more complicated. Significant correlations are only found with a lengthy sampling and averaging of data, rather than by matching each evening's data with the dream data of that night. A similar pattern of results, but regarding sexual dream content rather than nightmares, was obtained by Robbins et al (1985). They investigated the relationship between anxiety level and symbolic sexual dream content in 123 undergraduates. Anxiety, assessed by self-reports over a 10-day period, was positively related to the use of sexual symbolism identified from dream reports obtained over the same period. However, as in the longitudinal data from the current study, the appearance of symbolic dreams, was not preceded by elevated levels of anxiety. This paper also shows that negative emotion in waking life might be associated with non-negative dream content, just as Foulkes and Rechrtschaffen (1964) found that seeing a violent film did not result in violent dreams, but rather more vivid dreams. Furthermore, Proksch and Schredl (1999) found that the dreams of an acute divorce group were not, as expected, more negatively toned but instead showed more "primitivity" and "unsuccessful roles." These three papers show that dream emotions and events may not be continuous in a simple way with waking

emotions and events, and hence aid in understanding why the between subjects correlations between waking variables and nightmare/unpleasant dream frequencies are not large.

Between subjects correlations for pre-sleep variables are, however, larger than the within subjects correlations. What may be occurring is that dreams are affected not just by the immediately preceding day and evening but by many of the preceding days. Along these lines, Nielsen et al (2004) found that average ratings of correspondences between dreams and daytime events were high for predream days 1 and 2, low for days 3 and 4 and high again for days 5-7 (albeit only for participants who rated their confidence in recall of events as high and only for females). Delayed incorporations were more likely than immediate incorporations to refer to events characterized by interpersonal interactions and positive emotions. However, it should be noted that between subjects methods do allow for an influence of dreams on waking mood, and that the within subjects method used here is a purer method for elucidating the effects of waking mood on dream emotions. This method indicates that such effects are small.

Predisposition to nightmares

None of the individual difference variables used in this study interacts with state anxiety or state depression to cause nightmares or unpleasant dreams, and these individual differences did not lead to some people having a more negative state response to the occurrence of nightmares or unpleasant dreams. It had been hypothesised that variables that have been shown to be associated as a main effect with nightmare frequency would also have an interactive effect under conditions of high state anxiety or depression, or that variables shown to be associated with trait nightmare distress might interact with the presence of nightmares to increase state anxiety or depression. An enormous data analysis has resulted in the brief conclusion that none of these variables are predisposing factors for nightmare aetiology or for reaction to nightmares.

It was found that larger estimates of within subjects correlations were provided when only a sub-sample of participants were used who had more than one waking criterion nightmare defined as very high unpleasantness. However, even when using this sub-sample there was still no evidence that any personality variables modulated the within subjects correlation.

Prevalence and intensity of emotions in dreams and nightmares.

This study instructed participants to note the presence of specified emotions as and when they occurred on a line-to-line basis in their dream reports. They were required to rate the intensity of each reported emotion from 1 (low) to 3 (high) for every dream over 14 consecutive nights. The frequency of emotions was calculated as the total number of dreams with each emotion divided by the number of dreams. Emotion intensity was calculated per dream report and also only for instances when the emotion was reported. There was a mean of 2.05 emotions per dream, and 84.29% of all dream reports contained at least one instance of emotion.

The incidence of emotion in dream reports in this study is far higher than that reported by Hall and Van de Castle (1966) who found an average of 0.35 emotions per dream report. However, these authors did not specifically ask participants to report the occurrence of emotions. Merrit et al (1994), in a home dream recall study, instructed participants to note the presence of specified emotions as and when they occurred on a line-to-line basis in their reports (as in this study) and found an average of 3.6 emotions being reported per dream, which is higher than in this study. Furthermore, Merrit et al (1994) reported a prevalence rate of 95% for the occurrence of emotions in dreams, which is also higher than in the current study. There are a number of explanations for these differences. Firstly, Merritt et al (1994) had a small sample of only twenty students who completed the research as part of a class project. Participants were required to produce ten dream reports over the course of three weeks and score them for emotion. This may have led to a biased selection of only the most intense and dramatic dreams, therefore leading to an underreporting of less emotionally salient dreams. In contrast, this current study allowed participants to record as many dreams as they could recall per night, and as the study took place via post participants could be assured anonymity and experimenter bias was hopefully lessened.

Strauch and Meier (1996) used first person ratings and instrumental awakenings from REM sleep, finding an emotion in 72.5% of 500 REM reports from 44 subjects. Fosse et al (2001), using the same procedures, found emotion in 74% of 88 REM reports from 9 subjects. Thus, these prevalence rates of emotions are approximately 10% lower than the 84.29% found in this study. There are two possibilities for this finding. The first is that Fosse et al and Strauch and Meier both used REM instrumental awakenings, which reduces the possibility of participants forgetting dreams after awakening, particularly those that were not emotional salient. The second possibility is that nightmare sufferers may have more emotions in their dream reports or possibly that they remember more emotions due to increased negative content of dreams (in line with the salience hypothesis).

It should be noted, though, that as the current study relied on home recall it cannot be certain what underlying stage of sleep the dream reports are from. In Stickgold et al's (1994a) study, dream recall following spontaneous awakenings in the home was monitored by REMviews ('nightcap'). They observed that reports over 50 words long had a 76% probability of occurring following REM sleep arousals, and only a 24% chance of following NREM arousals. The current study had 64% of all reports being over 50 words long. Therefore, it is likely that a good proportion of the dream reports in this study were from sleep stages other than REM. Although these may have fewer emotions than REM dreams (Foulkes, 1966) the counteracting factors in the previous paragraph may still explain the high frequency of emotions in the home dream participants here.

Total prevalence of negative and positive emotions

Of the1396 instances of emotion, this study found that 64% were negative, 19% were positive and 17% were surprise, which is classed as neutral. Overall it was found that the frequency and intensity of total negative emotions was significantly greater than the prevalence and intensity of negative emotions. This is not surprising in a sample of subjects with frequent nightmares.

Those studies using first person ratings of emotion and instrumental awakenings from REM sleep have reported a relatively balanced proportion of total positive and total negative emotion (Strauch & Meier, 1996; Cartwright, Luten, Young, Mercer & Bears, 1998). However, Hall and van de Castle (1966) and Merritt et al (1994) found a higher proportion of negative emotions. Therefore, this suggests that home recall studies may overestimate the percentage of negative emotions. Foulkes et al (1979) compared dreams spontaneously recalled at home and those elicited under controlled laboratory condition. He found that home dreams constituted an extremely biased subset of the most dramatic and emotionally unpleasant dreams. Also, Fosse et al (2001) caution that when one departs from the strict sampling protocol of REM mentation, the resulting mentation report becomes increasingly biased towards including the most emotional and otherwise salient dreams. Thus, this may in part account for higher proportion of negative emotions in this study. However, the main reason for the high proportion of negative emotions in this study is that the sample consisted predominantly of frequent nightmare sufferers and so many more unpleasant dreams were reported. Moreover, 69% of this sample was found to meet the criteria for clinical 'caseness' on the SCL-90-R, thus, the continuity hypothesis would predict that these individuals would show an increased prevalence of negative emotions in dreams. In comparison the Fosse study and Strauch and Meier study used healthy participants. Nevertheless, although lab awakenings may in some circumstances be the gold standard when assessing the prevalence of emotions in normal dreamers, home diaries are arguably more reliable when assessing nightmare sufferers as nightmares are markedly reduced in the laboratory (Hartmann 1984).

This study found that anxiety was the most prevalent emotion, found in 46% of the reports. The second most prevalent emotion was surprise found in 32% of the reports, followed by sadness (26%), anger (25%), joy/elation (25%), shame (15%)

and love/eroticism (13%). This supports the continuity hypothesis; that individuals with poorer waking well-being will have increased negativity in dreams.

Total intensity of negative and positive emotions for all dreams

The intensity of negative emotions per dream report was significantly higher than the intensity of positive emotions or surprise emotions. However, when intensity of emotions were divided by the prevalence of these emotions, so as to assess intersity only for occasions when an emotion occurred, there was no difference between the intensity of positive, negative and surprise emotions. For both intensity measures, though, anxiety had the highest mean score.

The emotional profile of non-nightmare non-bad dreams

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As the sample in this study consisted primarily of frequent nightmare sufferers the data were analysed to explore the emotional profile of dreams rated 3 (slightly unpleasant) to 7 (very pleasant) on the hedonic scale, in order to make more meaningful comparison to previous research. However, negative emotions were still more prevalent than positive emotions (54% and 24% respectively). These findings are not in line with Fosse et al (2001) who found a balanced proportion of negative and positive emotions (44% and 49% respectively). This study also found a higher prevalence of most discrete emotions, especially the negative ones, compared to Fosse et al (2001). Fosse et al (2001) found significant inter subject differences in the total intensity of negative and surprise emotions but no comparable variation in positive emotions. They suggested that positive emotions were typical of most people but that negative emotions and surprise are more typical of some but less so for other subjects. Thus, the current study extends these findings by showing that this population has a very high levels of negative emotions.

Prevalence and intensity of emotions in dreams and their relationship with psychological well-being

As correlations of well-being individual difference variables with overall dream emotional tone were mainly small, ranging from .12 to .56 (Table 8), correlations with specific emotions in dreams were conducted in order to determine if the prevalence or intensity of negative or positive emotions or discrete emotions experienced during dreams had a larger relationship with psychological well-being or with mood before and after sleep.

None of the correlations between the frequency of negative and positive emotions and psychological well-being measures were significant. The intensity of negative emotions (when they occurred) was significantly correlated to the positive symptom distress index of the SCL-90-R and the intensity of surprise when it occurred was significantly related to the total positive symptom scale of the SCL-90-R. Intensity of surprise per report was significantly positively correlated with childhood events. The intensity of negative emotion per report was correlated with life events and childhood events. These correlations between surprise and well-being measures could be because people with poorer waking well-being have more bizarreness in their dreams. For example, Fosse et al (2001) found that the increase in surprise in their data was paralleled by an increase in bizarreness; furthermore, bizarreness and emotion have been reported to covary in dreams (Merritt et al. 1994). Total frequency and intensity of negative, positive and surprise emotion with mean mood before and after sleep.

The frequency of all negative emotions and the difference between negative and positive emotions was significantly positively associated with anxiety and depression before and after sleep. The total number of positive emotions was significantly inversely related to depression before and after sleep and anxiety after sleep.

These results suggest that higher scores on anxiety and depression before sleep increase the number of negative emotions and decrease the number of positive emotions, and these negative dream characteristics are also related to increased negative mood on awakening. These findings can be explained by the continuity hypothesis and are also in line with Kramer's (1993) mood regulatory theory.

These findings show that correlations with mood before and after sleep are more robust than those with trait well-being measures. Therefore, it could be suggested that although an individuals emotional status plays an role in dream emotionality, it seems that state factors are more important in determining the frequency and intensity of emotions in dreams, although these state factors must be averaged over a period of time rather than dealt with in a within subjects longitudinal manner.

Emotions in nightmares and bad dreams

This study assessed the prevalence and intensity of discrete emotion types in bad dreams and nightmares. It was hypothesised that nightmares would have significantly higher emotional intensities than bad dreams. Furthermore, it was predicted that nightmares and bad dreams would contain emotions other than fear.

Anxiety was significantly more prevalent in nightmares than in bad dreams (82% and 61% respectively). Shame and sadness were more prevalent in nightmares compared to bad dreams, although these differences were not statistical significant. Anger occurred approximately evenly in nightmares and bad dreams (39% and 42% respectively). When intensity was scored only for emotions that occurred, the intensity of anxiety was significantly greater in nightmares compared to bad dreams, and there was a trend for sadness to be more intense in nightmares although this was not significant (p = 0.05). These results are in accord with the same comparisons made by Zadra, Pilon and Donderi (2006), which will now be described.

Zadra, Pilon and Donderi (2006) assessed the variety and intensity of emotions in nightmares and bad dreams using a sample of 90 undergraduate students who recorded their dreams for 4 consecutive weeks and, for each dream recalled, noted the emotions present and their intensities on a 9-point scale. Of the 90 subjects 40% reported having had at least one nightmare and one bad dream over the 4 week study period. These authors divided participants into two groups depending on which dream types they reported on the logs. Participants who reported at least one nightmare and one bad dream were classified into the nightmare and bad dream group (NM+BD group). Participants who reported at least one bad dream but no nightmares were classified into the bad dream group (BD group). The emotional intensities of bad dreams and nightmares were calculated by averaging the ratings reported by each participant across all of the bad dreams or nightmares reported. Firstly, they found that the intensity of bad dreams from the BD group and bad dreams from the NM+BD group was not significantly different, and that there were non-significant differences between the BD and NM+BD groups for the emotion categories of fear, sadness, anger, frustration, confusion and other. (They were unable to conduct comparisons for the emotional categories of disgust and guilt due to the small proportion of subjects reporting these emotions.) This is very relevant to the current study as it suggests that the rating of emotional intensities for bad dreams is not affected by the experiencing of nightmares. Zadra et al suggest that it is therefore probable that the intensity rating of a bad dream reflects an independent assessment of that dream experience, and not a comparison of it to other disturbing dreams, including nightmares

Secondly, these authors reported that the mean emotional intensities for fear in nightmares were significantly greater than those reported in bad dreams. They were unable to conduct statistical comparisons for emotional intensities for nightmares and bad dreams across the remaining seven emotion categories (anger, sadness, frustration, disgust, confusion, guilt, other) due to the large proportion of students who did not report one or more of these emotions, but there was not any appreciable difference between nightmares and bad dreams in the incidence of these emotions.

The current study accords with these findings, and extends them by also assessing intensity of the individual emotions, finding there that anxiety/fear, when present in a nightmare, is more intense than when present in a bad dream.

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Waking criterion and emotional intensity

The waking criterion has been assumed to be a measure of nightmare intensity in that the sleeper is awakened from a nightmare because of the extreme intensity of the emotions experienced. Thus, if the emotional intensity is not sufficient to wake the sleeper then the dream is not classified as a nightmare. However, surprisingly there has been very little empirical evidence to support the notion that nightmares are more intense than bad dreams (disturbing dreams that do not waken the sleeper). In fact, Krakow et al's (1995) treatment studies highlight that less than 25% of chronic nightmare patients report always awakening from their nightmares. Furthermore, Zadra and Donderi (1993) found that approximately 45% of bad dreams have emotional intensities equal to or greater than those of the average nightmare in participants who have both bad dreams and nightmares. However, this current study has found that nightmares contain significantly more fear/anxiety than bad dreams and that this fear is significantly more intense. These findings also show a trend for a greater frequency and intensity of other negative emotions in nightmares compared to bad dreams. These data support Zadra and Donderi's (2000) contention that nightmares are emotionally more severe than bad dreams, but the data here do not support their contention that nightmare frequency is thus a higher correlates of poor psychological well-being than bad dream frequency.

Nightmares as frightening dreams

The general introduction highlights the debate over the definition of a nightmare as a 'frightening dream' (Bixler et al, 1979; Hartmann, 1984; Pietrowsky & Köthe, 2003; Wood & Bootzin, 1990). It has been argued that there is no empirical

evidence that the affective content of nightmares is restricted to fear alone. Anecdotal evidence suggests that although fear may be a dominant emotion of nightmares individuals also report experiencing intense unpleasant emotions other than fear (Belicki & Cuddy, 1991; Zadra & Donderi, 1993). A number of researchers have, thus, defined nightmares as disturbing dreams involving any unpleasant emotions (e.g., Blagrove et al 2004; Zadra & Donderi, 2000). However, these data, and the data of the current study, show that although fear is the dominant emotion in both nightmares and bad dreams these dreams also contain a variety of other unpleasant emotions. Similarly, Zadra, Pilon and Donderi (2006) found that thirty percent of nightmares and 51% of bad dreams contained primary emotions other than fear. Therefore, these findings should be considered in the definition of nightmares.

Summary of major/new findings

This study found that nightmare distress did not render nightmare frequencypsychopathology correlations negligible suggesting that nightmare frequency is not an artefact of nightmare distress.

The analysis of within subject correlations showed that the presence or absence of a nightmare or unpleasant dream has a significantly greater association with anxiety and depression after sleep than before sleep.

Pre-sleep mood between-subjects correlations were far higher than withinsubjects correlations, whereas for post-sleep mood the between-subjects and withinsubjects correlations were similar in size.

None of the individual difference variables assessed in this study predispose individuals to have nightmares under conditions of high anxiety or high depression. This was despite individual difference variables in many cases having significant between subjects correlations with nightmare frequency.

This study provides support for the assertion that waking criterion nightmares are more intense than non-waking criterion nightmares.

Summary and indications for the next study

This study found support for the continuity hypothesis in that poor waking well-being was significantly related to the prevalence of unpleasant dreams / nightmares and reactivity to them. However, the between subjects correlations with psychological variables were generally small, and the pre-sleep mood within subjects correlations were negligible: a new variable, sleep quality was thus introduced as a possible correlate of dream and nightmare frequency. In order to further explore the relationship with poor well-being it was decided to recruit a population that had an objective measurable psychopathology as well as deficits in subjective well-being. The population chosen was individuals with sleep apnoea and daytime sleepiness and snoring. Study two thus examines nightmare prevalence and its correlates in patients with sleep apnoea compared to sleepy/snoring controls.

As the measurement of discrete dream emotions was not found to have any advantage over dream emotional hedonic tone or nightmare/unpleasant dream frequency measures, discrete dream emotions were not to be assessed. As no measures of psychological predisposition to nightmares/unpleasant dreams were found, such predispositions were also not assessed in the next study.

Chapter 6

Study 2: Apnoea and nightmares

Introduction

Sleep apnoea

Sleep apnoea is a sleep disorder in which the sleeper repeatedly stops breathing during the night. The effects of untreated sleep apnoea can have marked detrimental effects on daily functioning. Patients mainly report diurnal symptoms, the most frequent and disruptive being excessive daytime somnolence (EDS). Disordered sleep combined with periods of cerebral hypoxia during apnoeic episodes can result in cognitive impairments (Brown, 1994), irritability, mood and personality changes (Engleman et al. 1994) and problems in personal relationships (Cartwright & Knight, 1987). Furthermore, patients with sleep apnoea have increased morbidity compared with the general population (Sharafkhaneh et al 2004; Smith et al 2002; Partinen and Guilleminault et al 1990) and have a greater incidence of inactivity, cardiovascular and other organ manifestations. The general introduction reviewed evidence suggesting that anxiety, depression and stress symptoms may affect the frequency of nightmares. As a result of the physical, psychological and social effects of sleep apnoea it is anticipated that nightmare frequency will be elevated in this group compared to sleepy/snoring controls.

This chapter will review evidence to support the assertion that sleep apnoea is associated with chronic sleep fragmentation (leading to excessive daytime sleepiness), increased psychopathology, cognitive impairments affecting daily functioning and decreased quality of life. Methodological shortcomings of this field of research will be summarised. Evidence will be reviewed assessing how apnoea, and various other sleep disorders affect dream recall and dream content. It is hypothesised that patients with apnoea will have an elevated nightmare frequency and more negatively toned dreams than sleepy/snoring controls.

Definition and measurement of obstructive sleep apnoea.

Obstructive sleep apnoea/ hypopnoea syndrome (OSAHS) can be defined as 'the coexistence of daytime sleepiness with irregular breathing at night' (Scottish Intercollegiate Guidelines Network, June 2003). When people with this condition fall asleep the muscle tone in the upper pharyngeal airway decreases resulting in narrowing of the upper airway. This leads to an increase in inspiratory effort, in an attempt to overcome this airway narrowing. In turn, this leads to a transient arousal from deep sleep to wakefulness or to a lighter sleep phase where normal airway muscular tone can be restored. This cycle can repeat itself hundreds of times throughout the night leading to the fragmentation of normal sleep architecture and the subjective experience of unrefreshing sleep. The collapse of the upper airway during sleep can be complete with total restriction of the airway (apnoea), or it can be partially restricted, resulting in hypoventilation.

An apnoea is defined as a pause in breathing for ten seconds, whereas a hypopnoea is a ten second event where breathing is continued but ventilation is reduced by at least 50% from the previous baseline during sleep. The apnoea/ hypopnoea index (AHI) is used to assess the severity of OSAHS; this measures the frequency of hourly apnoeas and hypopnoeas. OSAHS may be subdivided into varying degrees of breathing abnormality, depending on the AHI. Mild is considered to consist of an AHI of 5-14/hr, moderate of 15-30/hr and severe with an AHI of over 30 per hour. However, SIGN highlight that any cut off in AHI attempting to stratify the severity of OSAHS is arbitrary as severity can vary from night to night and symptoms from day to day in any individual. Clinically significant OSAHS is likely to be present when AHI> 15 events/hour asleep accompanied by daytime sleepiness and a minimum of two of the above features of the condition. Oximetry is often used as the first screening tool for OSAHS. These are spectrophometric devices that detect and calculate the differential absorption of light by oxygenated and deoxygenated haemoglobin in blood to produce a measure called the SpO₂. This is a measurement of the oxygen saturation of the arterial blood arriving at the fingertip or earlobe with each pulse beat. Commonly used oximetry methods include counting the number of oxygen desaturations (dips) per hour greater than an agreed value (often a 4% SpO₂ dip rate of more than 10 per hour)

Prevalence of obstructive sleep apnoea

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The SIGN guidelines estimate that the prevalence of OSAHS in middle-aged men (30- 65 years) is in the range of 0.3-4% (Stradling & Crosby, 1991; Young et al, 1993). The prevalence of OSAHS in middle-aged women has been studied less but it has been suggested that it is probably about half that of males, at around 0.5 - 1% (Bearpark et al, 1995). SIGN reports that approximately 50% of all patients with OSAHS are obese (BMI>30kg/m²).

Measurement of daytime sleepiness

The Epworth Sleepiness Scale (ESS; Johns, 1991) is a validated measure of daytime sleepiness in a variety of different situations. The maximum score is 24.

Patients can be subdivided clinically into the normal range (ESS<11), mild subjective daytime sleepiness (ESS =11-15), moderate subjective daytime sleepiness (ESS = 15-18) or severe subjective daytime sleepiness (ESS>18). Clinically the ESS is the best measure available to inform clinicians as to the patient's perception of their sleepiness, however, the correlation between the ESS and OSAHS severity is weak (Kingshott et al. 1998; Kingshott et al. 1995; Engleman et al. 1997). The standard objective measurement of sleepiness is the Multiple Sleep Latency Test (MSLT) (Carskadon et al. 1986). This involves a series of 20 minute naps administered over 2 hour intervals for a total of 8 hours. Patients are left to lie down in a darkened room connected to electroencephalographic (EEG) monitoring. The patient is told to try and go to sleep. The time required for EEG confirmed sleep is recorded and averages over the five sessions reflecting the patient's tendency to fall asleep. A MSLT of less than 5 minutes is considered pathological. Normal volunteers score with the range of 10 to 20 minutes.

Sleep fragmentation in healthy adults.

It is of relevance to this review to determine how sleep fragmentation (observed in patients with sleep apnoea) affects healthy adults without the confounds of co-morbid conditions associated with sleep apnoea. Research has suggested that daytime alertness can be impaired in both normal subjects and patients with sleep disorders, despite nocturnal sleep periods of normal duration (Carskadon et al, 1982; Bonnet, 1985; Stepanski et al, 1984). Bonnet (1985) hypothesised that in addition to sleep duration 'sleep continuity' must independently mediate recuperative sleep processes. Thus, it has been argued that disruption to sleep whether, 'external' (e.g. tones) or 'internal' (e.g. apnoea or periodic limb movements) may impair recuperation even though total sleep time may be unaffected. Research has examined the effects of experimentally induced arousals or awakenings using predominantly auditory stimulation. Experimental sleep fragmentation studies have aimed to determine the level of sleep disruption required to contribute to subsequent daytime sleepiness. The assumption of this research is that sleep must be uninterrupted for some minimum time period if it is to be restorative. Arousals or awakenings have been delivered at various rates and on different schedules (e.g. once per minute, once per five minutes, once per ten minutes) in order to determine the effects on daytime sleepiness. For example, Bonnet (1985) woke eleven young adults once every minute in a 6-night protocol. They reported performance defects and significantly reduced next day alertness after two nights of sleep fragmentation. Stepanski et al (1987) examined five healthy controls using EEG/EMG changes as the measure of arousal. In three conditions, arousals were administered once per 5 minutes, once per 10 minutes and once per minute for 4 hours, followed by solid sleep. The authors found significant decreases in MSLT scores after two nights of sleep fragmentation and no significant difference between conditions.

The majority of sleep fragmentation studies in healthy subjects have found significant impairments in daytime functioning. It has been consistently demonstrated that sleep fragmentation increases objective daytime sleepiness, as measured by the Multiple Sleep Latency Test (MSLT; Stepanski et al. 1987; Levine et al. 1987; Rochrs et al. 1994; Philip et al. 1994), single nap latencies (Bonnet, 1985; Bonnet, 1986; Bonnet, 1987; Bonnet et al. 1991) and Maintenance of Wakefulness Test (Martin et al. 1996). Bonnet and Arand (2003), in their review, concluded that thirteen of fourteen studies found significant increases in objective daytime sleepiness after various sleep fragmentation paradigms. These authors analysed the results of eight of these studies and found that sleepiness increased as the rate of fragmentation was increased (Pearson's r = 0.775, p<0.01). Increases on subjective measures of sleepiness have also been demonstrated using the Stanford Sleepiness Scale (Bonnet, 1986; Bonnet, 1987; Bonnet, 1989; Kingshott et al. 2000) and the Clyde Mood Scale (Bonnet, 1987; Bonnet, 1989; Bonnet, 1989). Furthermore, studies have demonstrated more negative mood following sleep fragmentation using the Clyde Mood Scale (Bonnet, 1987), the Profile of Moods States (Bonnet, 1991) and the UWIST mood adjective checklist (Martin et al, 1961; Kingshott et al. 2000). This deterioration in mood and the stress of excessive sleepiness supports the prediction of increased nightmare frequency and lower emotional tone of dreams in sleep apnoea.

However, some studies have failed to find differences in daytime sleepiness associated with different degrees of sleep fragmentation (Stepanski et al. 1987; Martin et al, 1996). For example, Levine et al (1987) found no increase in sleepiness, compared to uninterrupted sleep, when arousals were at the rate of one per ten minutes of sleep. On this basis it has been suggested that segments of sleep must be at least ten minutes in duration in order to be restorative (Downey & Bonnet, 1987; Levine et al. 1987), although this is controversial.

Sleep fragmentation compared to sleep deprivation

Some researchers have compared directly the effects of sleep fragmentation with sleep deprivation. Downey & Bonnet (1987) suggested that the one arousal per minute schedule of sleep fragmentation produces sleepiness comparable with total sleep deprivation. Levine et al (1987) found no significant difference in MSLT score between total sleep deprivation and 1-min sleep fragmentation (latencies were 2.2 and 4.1 minutes respectively). Bonnet at al. (1986) found that performance deficits were significantly greater following total sleep deprivation than following 1-minute sleep fragmentation on a number of addition problems and simple reaction times, although no significant differences were found on vigilance tasks or nap latencies. Spath-Schwalbe and Kern (1991) compared the effects of sleep deprivation and sleep fragmentation on hormones. They found that profiles of cortisol and ACTH peaked shortly after the initiation of sleep deprivation or sleep fragmentation and then showed the same pattern of inhibition followed by a later peak in both conditions. This pattern was significantly different from the normal sleep condition. Therefore, studies that have directly compared sleep fragmentation and sleep deprivation on hormones, objective sleepiness and psychomotor performance have, to date, provided strong evidence that a high frequency of sleep fragmentation is effectively the same as total sleep deprivation. Furthermore, sleep fragmentation has far higher detrimental effects on respiratory parameters, such as upper airway collapsibility, than does sleep deprivation (Series et al, 1994; Espinoza et al, 1991).

Sleep architecture and sleep fragmentation in apnoea

Sleep apnoea causes significant sleep fragmentation and considerably disturbs sleep architecture, causing EEG arousals of various durations, changes in sleep stage and frequent awakenings. This results in an almost complete deprivation of REM sleep and stages 3 and 4 of NREM sleep (Lamphere et al. 1990; Sforza et al. 1990). Stepanski et al (1984) reported that patients with sleep apnoea spent only 10.3% of their total sleep time in REM sleep, compared to 17.4% in healthy controls. Stage 1 sleep accounted for 47.9% of total sleep time in the apnoea group compared to only 15% in the control group.

Evidence for EDS in patients with sleep apnoea

Many studies have supported the finding that populations with excessive daytime sleepiness exhibit many instances of nocturnal sleep disruption (Guilleminault et al, 1976; Montplasisir, 1976; Zorick et al, 1978). Roth et al (1980) reported a correlation between nocturnal sleep disruption and daytime sleepiness in patients with sleep appoea and normal controls. They found that in patients with sleep apnoea only arousals associated with respiratory events correlated significantly with daytime sleepiness on the MSLT. More recently, Furuta et al (1999) found that the MSLT score showed significant negative correlations with the number of awakenings and with the apnoea/hypopnea index. These authors found no relationship between hypoxia in sleep and either MSLT or ESS. Other studies have also found an association between complaints of daytime sleepiness and an abnormal number of short EEG assessed arousals lasting between 2 and 14 seconds in patients with Upper Airway Resistance syndrome (Guilleminault et al, 1993; Guilleminault et al. 1991. Stepanski et al (1980) found that in patients with sleep apnoea (as well as with periodic leg movements, and insomnia, and in healthy controls), total number of arousals correlated with sleepiness index. Thus, research has consistently demonstrated that sleep apnoea leads to excessive daytime sleepiness, as measured subjectively and objectively.

This suggests that patients with sleep disordered breathing experience severe daytime sleepiness despite having often obtained 8-10 hours of total sleep. Sleep fragmentation also has a further effect: Phillipson et al (1980) found that sleep fragmentation impaired the ability of dogs to arise from sleep in response to hypercapnia and hypoxia. Thus, they concluded that sleep fragmentation is responsible not only for daytime sleepiness in sleep apnoea but also for changes in the acute response to airway occlusion resulting from sleep apnoea. This would further increase the hypothesised life stress caused by sleep fragmentation.

This section supports the notion that patients with sleep apnoea will have increased life stress as a result of excessive tiredness. Whether this is mediated by hypoxemia rather than sleep fragmentation would not affect this conclusion, but the evidence is that it is sleep fragmentation that directly causes daytime sleepiness (Roehrs et al, 1989; Colt et al, 1991; Bonnet and Arand, 2003.) The next section will review evidence that psychopathology is increased and quality of life is decreased in patients with sleep apnoea.

Sleep appoea and depression

A number of studies have found that patients with sleep apnoea have elevated levels of depression in comparison to healthy controls (Millman et al. 1989; Aiken et al. 1998; Cheshire et al. 1992; Guilleminault et al. 1977; Reynolds et al. 1984; Engleman et al. 1994; Derderian et al. 1988; Kales et al. 1985). Symptoms of anxiety and depression are also recognised in other respiratory diseases such as asthma, chronic obstructive pulmonary disease and sarcoidosis (Rimington et al 2001; van Manen et al 2002; Chang et al 2001).

Methodological problems

It is difficult to interpret the literature regarding the prevalence of depression in patients with sleep apnoea due the differences in methodologies, including different measures, control groups and also the cross sectional design of most of these studies. For example, some instruments measuring depression may contain somatic references and items related to sleep behaviours (e.g., fatigue, sleep disruption, task performance; see, for example, Millman et al, 1989). The BDI has been used to show the presence of depression in apnoea patients (e.g., Vandeputte and de Weerd, 2003; Watson et al, 1987) but also contains some somatic items and items referring to sleep. However, depressed mood due to apnoea has been shown by Reynold et al (1984), and Kales et al (1985) have shown depression in patients with severe sleep apnoea recommended for tracheotomy.

Many studies have utilised the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1940). This has 550 items covering a broad range of psychiatric symptoms, personality features and psychological difficulties. A number of studies have reported that patients with assorted disorders of initiating and maintaining sleep (DIMS) score higher on MMPI scales Depression and Hysteria (Bonnet & Arand, 1995; Kales et al. 1983). Levin et al (1984) also demonstrated that Paranoia scores differentiate mild from severe DIMS. It has been demonstrated that sleep apnoea patients have significantly elevated scores on at least some MMPI scales in comparison to normals, these are primarily, Depression, Schizophrenia and Hypochondriasis (Ramos & Espinar, 1992; Beutler et al. 1981). Beutler (1981) compared subjects with narcolepsy and sleep apnoea and found that both groups were significantly elevated on MMPI measures in comparison to normals. Kalogjere-Sackellares and Cartwright (1997) found that the MMPI profiles of patients with organic insomnia (e.g. sleep apnoea) did not differ from those with 'psychologicallybased' insomnia, and 79.3% of the cases contained at least 1 clinical scale in the

psychopathology range. They argue that their results support Kales et al's (1983) notion of the 'internalisation of psychological distress', as expressed in various constellations of neurotic symptoms of which depression is a common candidate as well as anxiety, somatic symptoms, passive aggressive or passive dependent character styles. Other studies have also shown an increased level of depression in patients with excessive daytime sleepiness. For example, Mosko et al. (1989) assessed patients with sleep disorders including sleep apnoea, narcolepsy and periodic limb movements. They found that approximately one quarter of all the patients assessed described themselves as depressed and between 58% and 71% of patients reported symptoms during the last 5 years that were consistent with episodes of major depression.

Comparison of psychological disturbance in patients with sleep apnoea and primary snorers

Aikens, Wallace & Mandelson's (1999) research is one of the few published studies to compare psychological disturbance in patients with sleep apnoea and primary snorers. They highlight that little is known about psychological disturbance in primary snorers and that this group would constitute an effective control group when assessing psychological factors in sleep apnoea. They hypothesised that primary snoring would be associated with less disturbance than observed in sleep apnoea, but with greater disturbance than seen in the general population. 98 patients were selected from the 499 patients who consecutively underwent overnight diagnostic polysomnography after referral to a sleep disorders centre. Forty-seven of these had received a diagnosis of primary snoring, based on an AHI of between 1.0 and 4.9. For each of these patients a patient with sleep apnoea was randomly selected from those with the same age and gender. The 47 selected sleep apnoea patients were similar to the unselected sleep apnoea patients in age, although they were more likely to be male. Subjects completed the sleep habits questionnaire and the MMPI. They were interviewed by a psychiatrist and underwent PSG followed by 4-trial MSLT. It was found that the sleep apnoea patients had a mean of 2.3 MMPI clinical elevations (i.e. in the disturbed range), which was significantly greater than the snorers group mean of 1.6 elevations. More than twice as many sleep apnoea patients than primary snorers fell into the disturbed range for the Depression scale (49% vs 25%) and on the Hysteria scale (35% versus 16%). The authors conclude that the sleep apnoea patients have 'relatively more inactivity, anergia, guilt, pessimism, and low self esteem accompanied by prominent somatic concerns' (pp357), although the snorers also had higher rates of disturbance than the MMPI reference norms. The authors suggest that primary snoring may be located midway along a general continuum between normalcy and sleep appoea in terms of symptoms and complications, although interpretation of differing MMPI elevations between these group of data is complicated by the presence of organic disease, in that some of the somatic consequences of sleep apnoea (fatigue, low energy, headaches) are similar to the criteria for diagnosing somatization disorder. It was, however, found that a higher total number of MMPI elevations were significantly associated with lower sleep efficiency, lower average blood saturations during non-REM, and greater wake time after sleep onset.

Negative findings for association between depression and apnoea

However, other studies have reported that patients with sleep apnoea do not show clinically significant levels of depression or that they have depressive symptoms no higher than control groups (Flemons et al, 1994, 1997; Gall & Isaac, 1993; Lee, 1990). Pillar and Lavie (1998) did not observe any relationship between depression and the severity of sleep apnoea in men, but did for women.

Pre and post CPAP studies and psychopathology.

Some studies have compared mood before and after treatment with CPAP. Reductions in depression have been shown by Derderian et al (1988), Li et al (2004), Ramos-Platon and Sierra (1992), Sanchez et al (2001) and Tsai (1997), and, in a placebo controlled cross over design (Engleman et al. 1994), although Bum-Hee et al (1999) found that CPAP and placebo CPAP groups both showed significant improvements in mood states (although their duration of CPAP was only 1 week and so the placebo effect may have diminished with greater time). Borak et al (1996) evaluated the effects of nocturnal CPAP on emotional and cognitive functioning in patients with sleep apnoea. They assessed 20 patients with severe sleep apnoea with a number of cognitive and psychological tests (depression, anxiety and mental stress) before, after 3 months and a year of CPAP treatment. They found that the majority of patients presented with symptoms of anxiety (14 patients), depression (11 patients) and mental stress (14 patients). Furthermore, mental stress and anxiety was significantly correlated with the AHI. There was a negative correlation between mental stress and duration of stage 2 NREM sleep. However, correlations between emotional status and severity of hypoxemia during sleep were weak and not significant. This suggests it is the sleep fragmentation rather than the hypoxemia that contributes to the emotional disorders in patients with sleep apnoea. However, these authors found that although cognitive functioning improved, there was no significant improvement in emotional status at 3 months or 1 year after CPAP. Borak et al suggest that this may be the result of the population studied, the patients in their study had severe apnoea (AHI of 67), whereas studies that did find an improvement, such as Bearpark et al (1987) and Engleman et al (1994), studied patients with less severe apnoea. However, a recent meta analysis of 4 randomised controlled trials consisting of patients with mild and severe sleep apnoea before and after treatment, found that Hospital Anxiety and Depression scores (HADS) and the Beck Depression Inventory (BDI) were significantly improved following treatment (National Health and Medical Research Council, 2000), although the improvement in mood according to Millman et al (1989) is only significant for those who initially had severe depression.

Confounding factors in mood and psychopathology relationships.

Bardwell, Berry, Ancoli-Israel & Joel (1999) highlight that much of the research examining sleep apnoea and mood has suffered from small sample sizes, lack of control groups and inconsistent or undisclosed criteria for diagnosing sleep apnoea. They highlight that patients often suffer from co-morbid conditions, for example, hypertension, which are rarely controlled and which have been associated with various psychological characteristics (Diamond, 1982; Dimsdale et al. 1986). Furthermore, they point out that previous research has often ignored the influence of weight and age, which are associated both with sleep apnoea and psychological factors, and found that after controlling for hypertension and age, almost none of the relationships between sleep variables and dysphoric mood remained significant. Bardwell et al. (1999) similarly suggest that depression and total mood disturbance are related more to age, weight and/or hypertension than they are to sleep apnoea. These papers do, however, offer some support to studies demonstrating that patients with sleep apnoea experience more anger than patients without sleep apnoea, and that patients with sleep apnoea have higher levels of fatigue.

In general these findings support the notion that patients with sleep apnoea experience elevated levels of general psychopathology in comparison to healthy controls and primary snorers. The following section reviews studies assessing general health and quality of life in patients with sleep apnoea.

General health and sleep appoea.

Recently, general health outcome measures have received growing recognition as relevant measurements for evaluating health care services and clinical trials. These general health evaluation questionnaires aim to quantify self-perceptions concerning general health and functional well-being. Using these measures, several studies have shown that sleep apnoea is associated with poor quality of life (QoL) (Baldwin et al, 2001; Bennet et al, 1999, using the Medical Outcomes Study Short form 36 Health Survey questionnaire of Jenkinson et al, 1996; Briones et al, 1996; D'Ambrosio et al. 1999; Gall et al 1993). There are also significant improvements in the health of patients with sleep apnoea following CPAP (Jenkinson et al, 1997; Smith and Shneerson, 1995).

Fornas et al. (1995) assessed the relationship between physiological disturbances, expressed as the number of respiratory events per hour and general health status and the degree of hypersomnolence. The authors assessed 103 consecutive patients admitted for sleep studies with suspected sleep apnoea. A group of 40 non-snoring healthy subjects were used as a control group. No significant differences were found in general health status when patients were divided into three groups according to the severity of the respiratory events during the night, but patients with sleep apnoea scored significantly worse on general health status in comparison to controls. This suggests that some individuals with severe apnoea may be more

tolerant and less distressed by their symptoms than some individuals with mild apnoea.

Interactions between QoL and mood in sleep apnoea.

Akashiba et al (2006), using the SF-36 QoL measure and the Zung self-rated depression scale (Zung, 1965), found a significant relationship between depression and QoL in patients with sleep apnoea, and argued that mood variables may play a more important role than Excessive Daytime Somnolence in QoL. Thus, it may be that sleep apnoea (through sleep fragmentation or reduction of sleep duration) affects mood, which in turn affects QoL.

This review of the literature suggests that mood and quality of life is often impaired in patients with sleep appoea. However, Lee (1990) argues that too much emphasis has been put on psychopathology, specifically depressive symptoms in patients with sleep apnoea. He argues that the symptoms presented in sleep apnoeic patients are often totally understandable and proportionate to the quality of life they experience as a result of this disorder. Patients are often forced to leave their jobs and stop driving due to the effects of daytime sleepiness. Some patients report personal, family or sexual problems. Many patients admit to personality changes such as irritability, which may also lead to loss of friendship and isolation. Since CPAP can prevent apnoeas and improve sleep architecture and abolish episodes of nocturnal hypoxia (Leech et al. 1992) it is reasonable to expect that patients established on CPAP may be able to return to work and continue to drive, etc., and these factors are liable to increase mood. Furthermore, patients no longer have the anxiety that may be triggered by fear of nocturnal approves and the consequences of approves on their general health (e.g. increased risk of cardiovascular illness).

BMI and mood

As the majority of patients with sleep apnoea are obese consideration needs to be given to the relationship between BMI and psychopathology. The review of this literature is not exhaustive but offers some insight as to whether obese individuals are at a higher risk for mental disorders and general psychopathology. Lamertz et al (2002) highlight that in most industrialised countries an elevated BMI is not only associated with increased risk for physical health but also with social stigma (Pi-Sunyer et al 1993) and other penalties (Wadden & Stunkard 1985), including mental disorders (Moore et al, 1997; Larsen, 1990) and elevated general psychopathology (Lamertz et al, 2002; Moore et al, 1997; Tiggemann et al, 1994), although it can be unclear how general psychopathology relates to defined DSM-IV diagnoses (Laessle et al. 1989). Indeed, only a few studies with representative samples of adults and adolescences have found significant (but weak) correlations between obesity and psychopathology when assessed by standardised measures (Istvan et al. 1992; Kohlmann & Weidner, 1996). Other studies have demonstrated lower rates of psychopathology in individuals with obesity or have not found elevated psychopathology (Krch et al 1997; Friedman et al. 1995).

Impairment of cognitive functioning

A number of studies have demonstrated that increased sleepiness in healthy normals and in patients with EDS, impairs cognitive functioning (Wilkinson, 1968; Levine et al, 1987; Rosenthal et al, 1993; Stepanski et al, 1987; Bonnet, 1985, Martin et al, 1996; Stepanski et al, 1987; Bonnet, 1989). However, as with mood, a clear pattern of deficits has not emerged. There are a number of reasons for these inconsistent findings. Investigators have used different methodologies, different control populations and different sample of patients.

Some studies have assessed the neurocognitive functions of primary snorers. It has been demonstrated that relative to normals primary snorers show deficits in focused attention (Verstraeten et al. 1997; Dealberto et al. 1996) and fine motor control (Verstraeten et al. 1997). Behavioural research has documented significant daytime cognitive and behavioural dysfunction in patients with sleep apnoea that seem to extend beyond that associated with simple sleepiness (Arens et al 2000; Marrone et al 1998).

A model of cognitive impairment in sleep apnoea

Beebe and Gozal (2002) have proposed a basic model, which provides a conceptual understanding of the behavioural and cognitive effects of sleep apnoea. They propose that patients with sleep apnoea have disrupted sleep and also intermittent hypoxemia and hypercarbia. They argue that these disturbances alter the efficacy of the restorative processes occurring during sleep and they also lead to a number of cellular and biochemical stresses that lead to disruption of functional homeostasis and altered neuronal and glial viability within particular brain regions. Beebe and Gozal propose that dysfunction of the prefrontal cortex (PFC) is a primary manifestation of the adverse cellular and biochemical events triggered by obstructive sleep apnoea. In their model this results in dysfunction of the 'executive system', which is composed of behavioural inhibition, working memory, contextual memory, analysis/ synthesis, set-shifting and self-regulation of affect and arousal.

Executive functioning impairments in patients with sleep apnoea

One measure of behavioural inhibition is the Stroop Colour-Word Interference task. Naegele et al (1995) found that adults with untreated sleep apnoea perform poorly on this task. Apnoea patients also make significantly more impulsive errors than controls on a maze completion task (Bedard et al 1991). Clinical measures of set shifting include perseveration in the Wisconsin Card Sorting Task (WCST, Heaton et al 1993), where, again, adults with untreated sleep apnoea have been found to perform poorly (Naegele et al 1995; Redline et al 1997). Self- regulation of affect and arousal refers to the internal modulation of affective states and arousal, although this is difficult to measure in the laboratory. The many reports of 'mood swings', irritability and affect lability are consistent with the notion of a weakness in this executive function. Continuous performance tests are often used as they require sustained vigilance on monotonous tasks over time. Poor performance on these tasks have been well documented in patients with untreated sleep apnoea, and deficits are more prominent towards the end of the tasks (Redline et al 1997). Therefore, this suggests that the problem is one of sustaining attention and effort rather than poor initiation or short-term maintenance of attention. Patients with untreated sleep apnoea also perform poorly on tasks involving working memory such as digit span, which involves the ability to repeat back digit strings and to mentally reverse them (Naegele et al 1995; Redline et al 1997); such working memory capacity is necessary for effective planning (Fuster, 1997). Studies have also demonstrated that patients with sleep apnoea perform poorly on verbal fluency, even when their language functions are otherwise intact (Bedard et al 1991; Neagele et al 1995): such verbal fluency indirectly measures mental flexibility and analytic/synthetic skills, which are thought to be important for creative, goal directed thought and problem solving.

Bebe and Gozal (1999) conclude from their review that, aside from sleepiness, executive dysfunction appears to be the most prominent area of cognitive impairment in untreated sleep disordered breathing, with deficits on this exceeding those on measures of visual and verbal ability (Bedard et al 1991) and long-term memory (Bedard et al 1991; Naegele et al 1995; Redline et al 1998; Haywood et al 1992).

Sleepiness and executive functioning

Research suggests that normal sleepiness cannot account for the executive functioning deficits displayed by patients with sleep apnoea. Executive functioning has been found to correlate better with blood gas abnormalities and sleep fragmentation than with either subjective or objectively measured sleepiness (Bedard et al 1991; Cheshire et al. 1992). Furthermore, Gozal et al. (2001a, b) demonstrated that children with sleep apnoea frequently display executive functioning deficits despite the fact that pre-pubertal children typically do not suffer daytime sleepiness unless their disease is moderate to severe. Some studies have also concluded that patients whose sleep has been normalised by CPAP may continue to show deficits on executive functioning tests (Bearpark et al. 1987; Bebard et al 1993; Feuerstein et al 1997; Naegele et al et al 1995). Gozal and Pope (2001) also found residual learning deficits long after sleep-disordered breathing had resolved. This persistence of executive dysfunction raises the possibility that neurological damage occurs as a result of sleep disordered breathing, and may in fact be only partially reversible.

Effects of cognitive impairments on daily life

The notion that patients with sleep apnoea may have cognitive deficits, particularly deficits in 'executive functioning', is important because of the consequences of these impairments on everyday life. Adults with obstructive sleep apnoea have been found to have occupational and social failures related to poor planning, disorganisation, diminished judgement, low motivation, rigid thinking and affective lability (e.g. Day et al. 1999; Doghramji 1993; Redline & Strohl, 1999). Furthermore, a number of studies have reported that sleep apnoea in children is associated with failure in school, behaviours similar to attention deficit disorder (Bower et al. 2002; Gozal 2000; Guilleminault et al 2000), and behaviours such as being unusually inattentive, hyperactive, impulsive, aggressive and rebellious (Dahl et al, 1996a,b). Bebe and Gozal (1999) highlight that executive functions allow people to make use of their basic skills in response to a complex challenging external environment. Cognitive impairments here that may result in social and occupational problems are also important because of their inevitable effects on mood. Thus, organic together with psychological factors may be responsible for low mood in these patients. This supports the hypothesis that patients with sleep apnoea will have increased life stress. The initial hypothesis of this chapter is that they will thus have more negatively toned dreams and a greater nightmare frequency. Evidence for this will now be reviewed.

Appoea and dreaming

Evidence has been reviewed above that sleep apnoea is associated with poor well-being and elevated general psychopathology. Research reviewed in chapter I has demonstrated that waking life stress causes negatively toned dreams and that these in turn affect daytime mood (Schredl, 1999 for review). This is stated in the continuity hypothesis of dreaming (Cartwright and Lamberg, 1992), which argues that dreams reflect the waking life concerns of the dreamer. The idea that dreams can convey information about the somatic concerns of the dreamer is still a prevalent concept in the literature. Sacks (1996) provided an anecdotal report of a person who had vivid dreams of becoming a statue and within weeks developed postencephalitic Parkinsonism. Saabini (1981) and van de Castle (1994) also provide hundreds of anecdotal accounts of dream content reflecting worsening symptoms or even seemingly forewarning of physical illness. Smith (1986) concluded in his review that dreams are sensitive to various health conditions, demonstrating that death and separation references in the dreams of medical patients had a prognostic value for clinical outcome. Dream content has also been related to the menstrual cycle (Bucci et al, 1991) and migraine (Heather-Greener et al, 1996). Studies that have assessed the emotional tone of dreams in patients with insomnia, and then apnoea, will now be reviewed.

Insomnia and the emotional tone of dreams

Ermann (1995) reported that the dream reports of patients with insomnia contained significantly more negatives in self-descriptions than healthy controls. The authors argue that this may be a reflection of the negative life events experienced by these patients. Moreover, negative sleep onset mentation in insomniacs was demonstrated by Antrobus and Saul (1980). Schredl (1991), in a small pilot study, found no heightened occurrence of problems in the dreams of insomniacs in comparison to controls, however, he did find that patients had a significantly reduced number of dream persons in comparison to controls. Schredl argues that this is in line with the continuity hypothesis as these patients in general described themselves as introverted. Schredl et al (1998) assessed 198 patients with primary insomnia and 722 healthy controls. All patients with insomnia and a subgroup of healthy controls (N=30) were evaluated polysomnographically for two consecutive nights. They completed a sleep questionnaire (SF-B) and were required to record their dream experiences after each night in the laboratory. On the sleep questionnaire patients reported considerably lower scores on sleep quality, feeling refreshed in the morning and emotional balance in the evening. Heightened scores were also found for sleep related psychosomatic symptoms, nocturnal awakenings, occupational stress and the occurrence of waking problems. Significantly increased scores were also reported for personal, occupational and health problems by patients with insomnia. In line with these problems, Schredl et al found that patients with insomnia rated their dream emotions as significantly more negative than controls. Dream content analysis showed that patients' dreams were characterised by heightened occurrences of aggression, problems, negatives in self-description and themes of depression, in comparison to controls. The authors also found an increased occurrence of health themes in the dreams of patients, which they argue reflects increased health concerns in waking life. This study supports the continuity hypothesis of dreaming in that waking life stressors, which were present in the group with insomnia, were reflected in their dreams.

Ohayon and Morselli (1994) and Hoffman et al (1996a) reported heightened nightmare frequency in patients with insomnia in comparison to healthy controls. Johnson and Spinweber's (1983) study of Navy enlisted men demonstrated consistent correlations between disturbing dreams and poor sleep quality. A similar conclusion was reached by Haynes and Mooney (1975) in their study of college students. Hence, such studies support the notion that, in those with poor sleep quality and insomnia, dream content is more negatively charged, possibly as a result of the increased waking stress due to daytime sleepiness.

Dreams of patients with sleep apnoea.

Hobson et al (1965) found significantly more respiratory themes in REM dream reports elicited after short apnoeas in healthy controls than in dreams after regular breathing. Santamaria et al (1998) found that the dreams of patients with sleep apnoea were predominantly negatively toned prior to CPAP therapy. However, they were unable to compare these with dreams following CPAP as dream recall frequency was drastically reduced.

Gross and Lavie (1994) assessed 33 patients with sleep apnoea who were already established on CPAP. They conducted dream awakenings during one night with a CPAP mask and one night without CPAP, awakenings were conducted with or without sleep apnoeas prior to awakenings. The authors found that the influence of breathing pattern on the content of dreams appeared to be stronger than the influence of CPAP. There was a tendency towards an increased rate of dream recall and longer dream reports in the apnoeic condition on untreated nights. Dream reports were also found to include significantly more characters, activities and social interactions when elicited during untreated nights and after awakenings preceded by sleep apnoeas. They found no direct incorporation of the apnoea into dream reports, but dream reports from the untreated nights and from the sleep approas appeared to be more negatively toned than after CPAP- nights and after awakenings without apnoeas. Moreover, dreams reported after regular breathing were more positive than dreams after awakenings proceeded by apnoeas. The significant difference in the emotional colouring of the dreams was found to be independent of dream length. Thus, the global emotional tone of the dreams appears to be altered by the apnoeic condition. They argue that the apnoeas influence psychological well-being in a negative way

and, in turn, affect the activation of memory elements involved in dream production (Foulkes, 1985). Hence, they argue that in this way the emotional tone of the dream generally becomes more negative although the dream may not contain direct references to the apnoeic stimuli. This finding was partly confirmed by Schredl (1998). He found that personal problems and occupational stress were reflected in the dreams of patients with sleep apnoea by increased negative feelings, aggression and occupational themes. However, his dream content analysis did not reveal a preponderance of negative emotions in the dreams of patients with sleep apnoea in comparison to healthy controls. Schredl (1991, 1998) found no heightened occurrence of breathing related dreams in patients with sleep apnoea in comparison to healthy controls (although there is the complicating factor of stimulus habituation, since sleep approved approved approved the night). Schredl (1998) concludes that the findings of Gross and Lavie of increased negative tone of dreams in patients with sleep apnoea may be an artefact. He argues that patients underwent a night without CPAP and this knowledge may have triggered anxieties about the possible effects of recurring sleep apnoeas.

Schredl et al (1999) assessed 44 patients with sleep apnoea and healthy controls. Participants were given the SF-B sleep questionnaire, measuring sleep quality, the feeling of being refreshed in the morning, emotional balance in the evening, tiredness in the evening, and sleep related psychosomatic symptoms. They were given several measures of waking stressors and a dream recall scale for the previous two weeks. Patients were then awakened and dream reports obtained towards the end of laboratory nights with standard polysomnography. Patients reported considerably lower scores for sleep quality, feeling refreshed in the morning, and emotional balance in the evening than did controls. Patients did not differ from controls in respect to partnership and family related problems but more often reported occupational problems and health problems. In terms of dream content the authors found that high Respiratory Disturbance Index (RDI) was related to more realistic and less intense dreams. Leisure time activities were more often found in these dreams. Furthermore, as RDI increases the negative emotions of the dream decrease (r = -0.352), low minimal blood oxygen saturation was associated with more positive emotions. These findings seem to suggest that the greater the severity of the apnoea the less negative and more neutrally toned the dream emotions. The authors also reported a significant reduction in dream bizarreness associated with a high RDI. They argue that the latter may be explained by arousal accompanying sleep apnoea in that these arousals interfere with the dream formation process. They suggest that the heightened occurrence of occupational themes related to low minimal blood oxygen saturation and high RDI is possibly a reflection of stress due to the severity of the sleep disorder. It could thus be suggested that individuals with severe apnoea do not have a sufficient amount of consolidated sleep to allow the formation of dreams with bizarre plots and negative emotions. Therefore, this suggests that nightmare frequency will be low in those with severe apnoea. Schredl et al's (1999) study also suggests that patients with severe apnoea will have more neutral and positively toned dreams.

Nightmares and snoring

There is evidence that snorers experience more negative dream content in the form of nightmares than healthy controls. This is of relevance here as the control group in the present study consists of sleepy snorers. Research has indicated that sleep is more fragmented in subjects who snore than in those who do not (Thoman, 1997). There is also evidence that daytime mood may be impaired in snorers in comparison to healthy non-snoring controls. For example, Vandeputte & Weerd (2003) demonstrated that 31% of patients who snored had some form of depression (i.e., rated mild or above using the Beck Depression Inventory). Thoman (1997) investigated 37 older women with snoring (age range 65-94) and found that snoring was positively correlated with the frequency of nightmares (r = 0.59; p<0.001), although snoring was not significantly correlated with depression scores (r = 0.23; p>0.05). The authors did not report whether an association existed between nightmares and respiratory disturbance index. This research suggests a significant relationship between snoring and nightmares and that sleep fragmentation may be associated with nightmares, although it is unclear what definition, if any, of a 'nightmare' was used.

DeGroen et al (1993) studied 98 male Dutch veterans, who had been exposed to excessive stress during World War II. 56% of this sample suffered from posttraumatic stress disorder. Unsurprisingly the authors found that PTSD was highly associated with the occurrence of anxiety dreams. However, they also found that snoring was strongly associated with anxiety dreams. This relationship was independent of the use of sedatives, antidepressants, smoking, alcohol and coffee consumption. Furthermore, they demonstrated that snorers with respiratory pauses appeared to have significantly more anxiety dreams compared to snorers without respiratory pauses. The difference between snorers without respiratory pauses and non-snorers was not significant. The authors argue that as habitual heavy snoring in combination with respiratory pauses is the most common symptom of sleep apnoea (Kapuniai et al. 1988) then it is reasonable to suggest that the occurrence of apnoeic episodes explains the relationship between snoring and anxiety dreams. (The authors propose that in subjects with a history of trauma or extreme stress the repression of traumatic memory content may fail during such apnoeic episodes resulting in the reliving of those experiences.) These findings lend support to the hypothesis here that nightmare frequency will be elevated in the group with sleep apnoea, and would also be high in the sleepy/snoring controls. However, it is acknowledged that there are obvious difficulties in generalising the DeGroen et al (1993) findings to other populations due to the fact that 56% of the veterans met the criterion for PTSD and all had experienced traumatic incidences in their past. Furthermore, anxiety dreams were undefined by the authors.

Nightmares in children with sleep apnoea.

Some studies have looked at nightmare frequency in children with sleep apnoea. Guilleminault et al (1996) assessed children with Sleep Disordered Breathing, including those with sleep apnoea and Upper Airway Resistance Syndrome (UARS). They reported that this group had significantly more nightmares / night terrors and sleep waking than would be expected in the general population. Owens et al (1997) assessed the incidence of parasomnias in a sample of children less than 6 years old (n = 81) and children between 6-12 years old (n = 83) who were diagnosed with sleep apnoea or behavioural-based sleep disorders (BSD). The children's Sleep Habits Questionnaire (SHQ) was completed by the parents of those children under six. The only item on the SHQ that differentiated between diagnostic groups was 'wakes alarmed by a scary dream': the behavioural-based sleep disorder group had a higher mean score on this item than did the apnoea group. For the children aged between six and twelve, no parasomnia was more common in the sleep appoea group than in the BSD group, although for the older sleep apnoea group incidence of bed wetting, sleep walking, rocking in sleep and unrecalled nightmares were significantly higher than

population norms. Also the older children with sleep apnoea were significantly more likely to be 'terrified during a dream' than population norms. Thus, the above evidence is suggestive that sleep apnoea is associated with an increased incidence of nightmares in comparison to healthy controls. However, this research may not be generalisable to adults as sleep apnoea manifests itself differently in children (for example, daytime sleepiness is a less common symptom in children).

Dream content and nightmares in adults with sleep apnoea

MacFarlane and Wilson (2006) examined the influence of specific sleep disorders (including sleep appoea) on dream content through retrospective analysis of questionnaire data of patients attending a Sleep Disorders Clinic. Data were collected from 124 subjects who demonstrated obstructive sleep apnoea, narcolepsy, an EEG arousal disorder during sleep, or periodic leg movements during sleep. The 42-item Whaler Physical Symptom Inventory was used to quantify somatic concerns. Dream content and dream frequency were assessed with a 37-item dream questionnaire. Ten symptom-dream pairs were selected as mutually relevant, for example reporting muscular weakness and dreaming of not being able to move, or reporting difficulty breathing and dreaming of choking or not being able to breathe. These pairs were analysed in order to determine whether those with a particular daytime somatic symptom report a related dream with a significantly greater frequency that those who are asymptomatic. They found that of the entire sample 84.6% (n = 105) reported having nightmares. This included 30 subjects with narcolepsy, 29 with obstructive sleep apnoea, 26 with increased cyclical alternating pattern (CAP) and 20 with periodic leg movements. The following table demonstrates the frequency at which nightmares occurred in each sleep-disordered group.

Table 48 – Data from MacFarlane and Wilson (2006) on dream content and nightmares in adults with sleep disorders

Frequency of reporting nightmares					
Never	Sometimes	Often	Always		
11	20	7	3		
15	19	6	1		
12	27	1	1		
21	19	1	0		
	Never 11 15 12	Never Sometimes 11 20 15 19 12 27	Never Sometimes Often 11 20 7 15 19 6 12 27 1		

Note: CAP = cyclical alternating pattern, PLMS = periodic limb movement syndrome.

It can be observed from Table 48 that the majority of patients with sleep apnoea report having nightmares 'sometimes'. However, it is not clear what the definition for 'sometimes' is. It seems that these patients do not report nightmares 'often' or 'always' in this study. However, it has been demonstrated that retrospective estimates produce lower estimates of nightmares compared to logs. The fact that 27 out of the 29 patients with sleep apnoea reported 'sometimes' having nightmares suggests that nightmare prevalence may be raised in this population.

These authors also found that patients with sleep apnoea do not dream of choking/ feelings of suffocation with a greater frequency than non-apnoeic patients. In fact they found that only 40% of patients with sleep apnoea report being bothered by difficulty breathing on the Whaler physical symptom inventory. However, in the sample as a whole difficulty breathing (reported by 17 patients) was significantly related to dreams of choking or not being able to breathe. Similarly, somatic

complaints of excessive perspiration were significantly related to dreams of perspiring. The other symptoms dream pairs were not significant. The authors suggest that this may be because the other symptom-dream pairs could not really be considered to be good matches when considering direct dream manifestation of somatic complaints (with the exception of reports of paralysis and dreams of not being able to move, which was not significantly correlated). For example, the authors correlated somatic complaints of 'poor health in general' with dreams of 'dying/being dead' and complaints of 'abnormal blood pressure' with dreams of 'heart pounding'.

The evidence above provides support to the notion that dream emotional tone may be more negative in some patients with sleep disorders and that nightmare frequency may be increased, even if the specific dream content does not match the disorder. The following section of this review will examine whether sleep disorders also affect dream recall in general.

Sleep disorders and dream recall

Schredl (1998b) found a heightened dream recall frequency in patients with narcolepsy, which he interpreted as an effect of disinhibited REM sleep regulation. Other studies have reported that patients with narcolepsy report vivid, frightening and disturbing dreams at night (Broughton et al., 1988; Montplaisir and Godbout, 1986). This is believed to reflect the physiological abnormality of REM sleep in narcolepsy.

Many authors have reported a significant positive correlation between dream recall frequency and frequency of nocturnal awakenings (Cory et al, 1975; Halliday, 1988; Schredl and Montasser, 1996-1997). Furthermore, a number of studies have found a significant relationship between poor sleep quality and dream recall frequency (Arand et al. 1972; Lugaresi et al, 1983; Borbely, 1984; Schredl et al. 1997). Schredl et al (1998) demonstrated that whereas dream recall frequency was significantly increased in patients with insomnia in comparison to controls, this difference disappeared when frequency of nocturnal arousals was partialled out. In contrast, partialling out the 'occurrence of problems' did not considerably reduce this difference between insomniac patients and controls. Thus, it can be concluded that dream recall frequency is heightened in patients with insomnia primarily due to increased nocturnal awakenings rather than daytime stress (measured by occurrence of problems). Schredl argues that this supports the arousal-retrieval model of dream recall (Koulack and Goodenough, 1976). Although this model acknowledges the influence of visual memory and salience of dream imagery it also emphasises the importance of sleep parameters in explaining the inter-individual variability of dream recall frequency. This model may also account for findings by Hartmann (1984) that some individuals who suffer from nightmares also have a pattern of prolonged and interrupted REM sleep. However, it could be that these individuals also have more interest and preoccupation with their dream life, a factor that has been found to increase dream recall (Halliday, 1992). Against this, Schredl et al (1998) found that increased dream frequency in patients with insomnia could not be attributed to a heightened interest in dreams as patients did not differ from controls on the variable 'engagement with dream during the day', which the authors used to estimate interest in dreams (Schredl, 1997).

However, two studies have not found an elevation in dream recall frequency in patients with insomnia (Schredl, 1991; Erman et al, 1993), although they both had small sample sizes and the latter had inadequate gender-matching and nonstandardised interview schedules. It is clear that in other patient groups, such as patients with clinical depression, variables other than nocturnal awakenings must be responsible for the dream recall differences between patients and controls. Although sleep quality is low in depressed patients, which includes frequent nocturnal awakenings, a number of studies have reported that dream recall frequency is also low (Riemann et al. 1990a; Schredl, 1995)

Dream recall and respiratory disturbance

Monday et al (1987) found a tendency for increased dream recall in subjects with asthma with nocturnal attacks in comparison to matched healthy controls, although this difference was not statistically significant. The number of REM awakenings for the asthmatic and control groups were 43 and 46 respectively, but asthmatic subjects reported 35 (81%) dreaming experiences versus 28 (61%) for controls. However, asthmatic subjects had a significantly higher vivid impression of being woken from a dream without any ability to remember any of the dream content than did controls. The authors suggested the term 'white dream' for this phenomenon of the vivid sensation of 'coming out from a dream' without being able to remember any content. However, none of the asthmatic subjects reported that they were dreaming when awakened from a nocturnal attack, thus the authors argued that hypoxemia at the time of nocturnal attacks could not be responsible for the poor dream recall as 'white dreams'; further evidence for this was that 'white dreams' were also reported by subjects who did not show significant (>4%) oxygen desaturations. The authors propose that poor dream recall by asthmatic subjects during nocturnal attacks could be because dreams leading to nocturnal attacks are strongly repressed. However, they also suggest that physiological distress at the time of the nocturnal attack makes the patient less attentive to the dream content.

Dream recall in sleep apnoea

Schredl et al (1998) found a slightly elevated dream recall frequency in patients with sleep apnoea in comparison to healthy controls. These authors compared dream recall estimates over a 2-week period of 236 patients with sleep apnoea with 722 healthy controls. They found that neither the Respiratory Disturbance Index (RDI) nor maximal decrease of blood oxygen saturation as severity measures were significantly correlated with dream recall frequency. They reported that the heightened dream frequency was best explained by the lower emotional balance of the patient group in the evening. They argue that this supports the salience hypothesis of dream recall (Cohen and MacNeilage, 1974) in that the more negative the pre-sleep mood the more negative the dream affect and the higher the probability of dream recall. They conclude that heightened dream recall in patients with sleep apnoea can be partly explained by the fractionated sleep profile (frequent arousals) and by this daytime stress.

Gross and Lavie (1994) reported that dream recall was better with REM awakenings after the occurrence of sleep apnoeas than with REM awakenings after regular breathing (using nights when patients were treated with CPAP). The authors argue that this finding is in line with the Functional State-Shift Model (Koukkou and Lehmann, 1983). This theory proposes that dream recall is dependent upon the difference between the functional state of the brain during sleep and during wakefulness. It is argued that the closer the distance between these two states the higher the dream recall. It is reasoned that sleep apnoea is characterised by numerous brief awakenings resulting in a higher state of brain arousal and that this elevated state

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produces the basis for improved dream recall. Schredl et al (1999) in a further study with sleep apnoea patients confirmed the previous findings of heightened dream recall in comparison to healthy controls. However, contrary to their previous research of 1998, Schredl et al (1999) found that the difference in dream recall between apnoea patients and control was not significantly reduced when 'frequency of nocturnal awakenings' or 'emotional balance in the evening' was partialled out. Therefore, they argued that frequent arousals due to sleep apnoeas lead to higher functional states during REM sleep and as a result dream recall is enhanced. Again this study lends support to the hypothesis that dream recall will be significantly elevated in the apnoea group.

Dream recall in patients with restless leg syndrome

Schredl (2001) investigated the dream recall frequency of 131 patients with restless leg syndrome in comparison to healthy controls. In RLS leg movements can be accompanied by short arousals and cause pronounced sleep fragmentation (Sforza et al. 1999). Patients reported considerably lower scores for sleep quality as well as for the feeling of being refreshed in the morning and for emotional balance in the evening, (the latter possibly reflecting the stress associated with having a sleep disorder). Dream recall frequency and the scores of 'engagement' in dreams did not differ significantly between the control group and the patient group. These findings do not support the arousal retrieval model, particularly since the authors report that the frequency of nocturnal awakenings was significantly higher in the patient group. However, dream recall frequency was found to be negatively associated with the number of periodic leg movements with arousal (PLMAI). Thus, as the severity of the PLM disorder increases the likelihood of reporting a dream decreases. Schredl argues that this suggests that the microarousals causing a distinct sleep fragmentation may actually interfere with the process of dreaming itself or with the process of dream recall.

Hypotheses:

Having reviewed the literature on apnoea and psychopathology, apnoea and sleep quality, apnoea and nightmare and dream frequency, and literature on the effects of sleep fragmentation and of other sleep disorders on dreaming, the following hypotheses have been derived for study 2.

- a) Levels of anxiety and depression will be significantly higher in the sleep apnoea group than the sleepy/snoring control group.
- b) There will be a higher frequency of nightmares in the apnoea group than in the sleep/snoring control group, and both groups will have a higher frequency of nightmares than the general population.
- c) Nightmare frequency will increase due to increased levels of disorder severity and waking psychopathology from the sleepy/snoring controls to the mild to the moderately severe apnoeics, however, those with the severest apnoea, despite having the most waking stress, will not have sufficient periods of consolidated sleep with which to support the formation of nightmares or dreams. Data from the apnoea and sleepy/snoring control groups will be combined for this analysis in order to provide data from people with a low diprate. The specific hypothesis is that, as the diprate increases, nightmare frequency will form an inverted U shaped curve, as a result of the combination between increasing waking distress but also increasing sleep fragmentation.

 d) The dreams of untreated sleep apnoea patients will be significantly more negatively toned than when they are treated with CPAP.

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Chapter 7

Study 2: Apnoea and nightmares

Method

Participants.

49 consecutive patients attending a sleep disordered-breathing clinic in a District General Hospital were recruited. They were referred by their GPs (or ENT specialists) and complained of daytime sleepiness and most had been told they were heavy snorers. The study and its procedures were approved by the departmental and local ethical committees (see appendix 20 and 21).

Those who were referred for sleep studies received a patient information sheet and consent form (see appendix 16 and 17). A diagnosis of Sleep Apnoea Syndrome (SAS) was made when they had significant daytime tiredness and a raised Epworth Sleepiness Score, together with a positive (limited channel) home sleep study showing a dip in (peripherally measured) oxygen saturation 10 or more times per hour (Embletta Portable Diagnostic System; ResMed, Abingdon, UK).

26 patients (21 males) were diagnosed with sleep apnoea (mean age 55.00; SD = 10.78). 23 (17 males) were classed as sleepy/snoring controls (mean age 50.14; SD = 8.76)

Mean 4% dip rate for the SAS group was 30.95 per hour (SD= 20.71; range 7.40-75.80) compared to 4.05 in the sleepy/snoring control group (SD= 2.88; range 0.60-9.40). This difference was statistically significant (t (44) = -6.33; p =0.001). SAS

patients had a mean BMI of 36.19 (SD= 5.9) compared to sleepy/snoring controls who had a mean BMI of 33.02 (SD= 7.80). This difference was not statistically significant (t (46) = -1.06; p =0.11) Mean Epworth sleepiness score was elevated in the apnoea group (14.73 SD= 5.39 and 10.47, SD = 4.61 respectively; t (43) = -2.85; p = 0.007).

Follow up.

10 of the 26 patients with SAS took part in a follow-up study after a minimum of three months on successful CPAP therapy. CPAP use resulted in abolition of almost all apnoeas, with a 4% dip rate in this group, measured by simply oximetry, after 1 month of treatment, improving from a mean of 37.73 to 0.54 events per hour.

Table 49 - Means (and standard deviations) for participants before and after CPAP therapy.

	Pre-CPAP	Post-CPAP
Age	58.5 (6.92)	-
Height	113.95 (18.05)	-
Weight	113.95 (18.05)	114.69 (20.57)
BMI	37.3 (6.71)	37.15 (7.44)
ESS	15.1 (3.54)	8.11 (4.81)

Note: BMI = Body Mass Index; ESS = Epworth Sleepiness Scale.

No significant difference was found in weight before and after CPAP (t = 1.108; p= 0.19) or BMI (t (7) = 1.43; p = 0.19). Epworth sleepiness scores were significantly lower following CPAP (t (8)= 4.08; p = 0.004).

Materials

Embletta Portable Diagnostic System (ResMed, Abingdon, UK).

The Embletta sleep recording system is a portable digital recorder that is a complete system for the diagnosis of sleep-disordered breathing. The Embletta's diagnostic signals include position and activity, leg movement, oxygen saturation, pulse, oral flow, event button and two respiratory effort signals through the XactTrace Respiratory Inductive Plethysmograph (RIP) sensors. Each sleep data trace was manually reviewed by a Chief Cardiorespiratory technician to ensure that the dip in oxygenation was due to an obstructive not central apnoea.

Oximetry

Oximetry is a spectrophometric device that detects and calculates the different absorption of light by oxygenated and deoxygenated haemoglobin in blood to produce a measure called the SpO_2 . This is a measurement of the oxygen saturation of the arterial blood arriving at the fingertip or earlobe with each pulse beat. The number of oxygen desaturations (dips) per hour was counted. Sleep apnoea was defined as a 4% SpO_2 dip rate of more than 10 per hour. Due to the use of a different machinery some patients had 5% SpO_2 dip rate values but these can used interchangeable with the 4% measure.

The Epworth Sleepiness Scale (ESS)

The Epworth Sleepiness Scale (ESS; Johns, 1991) is widely used in research and clinical practice and is a validated measure of the propensity of subjects to fall asleep in a variety of different everyday situations. The self-reported propensity to fall asleep has been used as a surrogate for daytime tiredness and the ESS has been used in a variety of sleep disorders. For example, a cut off score of 10 on the ESS gave a sensitivity of 93.5% and specificity of 100% in distinguishing narcoleptic patients from normative subjects (Johns, 2000) and a cut-off of 14 gave a sensitivity of 97% and a specificity of 100% (Parkes et al. 1998). ESS scores correlate with objective data obtained using the multiple sleep latency test (MSLT), a standardised objective test of sleepiness, which provides a mean sleep onset latency score based on scheduled sleep opportunity across the day (Carskadon et al. 1986). The reliability of the ESS is reported to be similar to that of the MSLT-derived measures (n=87, r = 0.81, p<0.001). Clinically the ESS is the best measure available to inform clinicians as to the patient's perception of their sleepiness, however, the correlation between the ESS and OSAHS severity is weak (Kingshott et al. 1998; Kingshott et al. 1995; Engleman et al. 1997) and it is not recommended as a screening tool.

The scale requires participants to rate how likely they would be to doze off in 8 situations, based on their usual way of life in recent times, using a scale from 0 (would never dose) to 3 (high chance of dozing). The maximum score is 24. Patients can be subdivided clinically into the normal range (ESS<11), mild subjective daytime sleepiness (ESS = 11-15), moderate subjective daytime sleepiness (ESS = 15-18) or severe subjective daytime sleepiness (ESS>18).

Sleep and mood diary

A 'yes' 'no' format was used to determine the presence of a dream and an awakening as a result of dream emotions or events. Participants rated the overall mood of the dream on a 7-point hedonic scale (Foulkes et al. 1996) ranging from 1

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(very unpleasant) to 7 (very pleasant). The mid point of the scale included an option for 'neither pleasant or unpleasant or mixed' (see appendix 18 for example).

Participants were also required to complete two Visual Analogue Scales (VAS) every morning for the ten consecutive days. VAS scales questions about 'happy/depressed' for low mood and 'relaxed/anxious' for anxiety. These moods were rated on separate lines because it has been shown that these moods can vary independently (Bowen et al. 2004; Watson, 2000, pp.27, 45). These scales and the questions above were identical to those used in study one.

Participants were instructed to complete their sleep diary for 10 consecutive days immediately after waking. Sleep diaries filled out immediately on waking, have been shown to be highly correlated with objective estimates of sleep (Espie et al 1989; Wilson et al. 1998).

Procedure

Participants were approached following their initial appointment at a Sleep Disorder Breathing Clinic at a district general hospital and invited to take part in the research. The aims of the study were explained those who agreed to participate. This information was also provided in writing (appendix 16). Written consent was obtained if participants were willing to take part (appendix 17). It was emphasised that this research was entirely separate from the treatment received at the hospital and that refusal to take part would in no way affect the standard of care they received. If sleep apnoea was suspected during the initial consultation, patients were required to attend a follow up hospital appointment approximately two weeks later in order to collect the equipment for their home sleep study (Embletta Portable Diagnostic System; ResMed, Abingdon, UK). During this two-week period participants were asked to complete a 10-day sleep and mood diary. The Epworth Sleepiness Scale and other demographic data were obtained from the initial consultation appointment. Participants were asked to bring their completed diaries with them when they attended their follow up appointment. Those diagnosed with sleep apnoea (as a result of their home sleep study and clinical interview) received an in-hospital auto-titration to maximise CPAP pressure. They were advised to use their machine (Sullivan S6, Sydney, New South Wales) as much as possible when asleep. Overnight oximetry was performed at 1 month, whilst using CPAP, to confirm the abolition of desaturations. Weight, BMI and Epworth Sleepiness Scores were also recorded during this follow- up hospital session.

Those with sleep apnoea established on CPAP therapy were written to after a minimum of three months after starting treatment (see appendix 19 for copy of letter). Participants were sent a 10-day sleep diary (identical to the one they had previously completed) and asked to return this to the hospital after they had completed it (in the stamped addressed envelop provided).

<u>Design</u>

A between subjects design was used to compare the sleep apnoea patients with patients who received a negative diagnosis of sleep apnoea (i.e., sleepy/snoring controls). The independent variable in this first part of the design was whether patients had received a diagnosis of sleep apnoea. The dependent variables of interest were frequency of dreams, number of nightmares, emotional tone of the dreams and scores on anxiety and depression scales. Dip rate was then used as a predictor of the dependent variables, using both groups combined in order to have a wide range of diprate.

A within subjects design was employed to assess patients with sleep apnoea before and after CPAP. The independent variable was CPAP status and the dependent variables of interest were frequency of nightmares, dreams, mean emotional tone of dreams and scores on anxiety and depression measures. Change in dip rate was assessed as a predictor of change in dependent measures.

Chapter 8

Study 2: Apnoea and nightmares

<u>Results</u>

Definition of nightmares and unpleasant dreams

In this study nightmares were defined as dreams on the logs that were rated very unpleasant or moderately unpleasant and that woke up the sleeper, unpleasant dreams were defined as dreams that were rated very unpleasant or moderately unpleasant, irrespective of whether they were the cause of the sleeper waking. We included the moderately unpleasant category due to the small number of dreams rated as very unpleasant, indeed neither group recorded at least one very unpleasant dream every night. Also, study 1 showed that between subjects correlations with personality variables, and within subjects correlations with pre and post-sleep mood did not differ between the use of the very unpleasant criterion alone, or the use of the criterion of moderately or very unpleasant.

Table 50 - Number of dreams rated as moderately, or very unpleasant

	Moderately unpleasant	Very unpleasant	
Apnoea group (n = 26)		9	
Sleepy/snoring control	13	8	
group $(n = 23)$			

Descriptive statistics

Table 51 shows the means and standard deviations (SD) for dream and mood

variables in patients with sleep apnoea and sleepy/snoring controls.

Table 51 - Means and standard deviations for dream and mood variables in patients

with apnoea and sleepy/snoring controls

	Apnoea group	Apnoea	Sleepy/	Sleepy/	Test of difference ¹
	means (SD)	group range	snoring control group	snoring control	z (p)
			means (SD)	group range	
Body Mass	36.20 (5.90)	28.10-48	33.02 (7.78)	24.17-52.42	t = -1.06(0.11)
Index					
Dip rate	30.95(20.71)	7.40-	4.05 (2.88)	(0.60-9.40)	z = -5.67* (<.001)
		75.80			
Dream frequency	6.5 (3.52)	0-10	5.35 (3.45)	0-10	z = -1.44 (0.15)
per 10 days					
Nightmare	0.85 (1.24)	0-6	0.78 (1.28)	0-5	z=-0.78 (0.44)
frequency per 10					
days					
Bad dream	0.23 (0.51)	0-2	0.13 (0.34)	0-1	z =-0.62 (0.53)
frequency per 10					
days					
Unpleasant	1.11 (1.63)	0-8	0.91 (1.13)	0-5	z = -0.63 (0.53)
dream frequency					
per 10 days					
Dream emotional	3.64 (0.66)	1.90-5	4.21 (1.19)	2-6.25	t = 2.04 (0.05)*
tone					
Epworth	14.73 (5.39)	2-24	10.48 (4.61)	0-18	t = -2.85* (0.01)
sleepiness scale					
Anxiety	4.93 (1.64)	2.28-9	4.66 (1.44)	1.53-7.33	t = -0.59 (0.56)
Depression	4.65 (2.03)	0-9	4.49 (1.30)	1.65-6.67	t = -0.30(0.77)

¹ Tests of difference were either t-tests or chi squared depending on whether the variables were normally distributed.

* p<0.05. All values are two tailed.

Inspection of table 51 shows that the frequency of dreams, nightmares and unpleasant dreams is slightly greater in the sleep apnoea group compared to sleepy/snoring controls, however, none of these differences are statistically significant. Dip rate is significantly higher in the apnoea group. BMI is not significantly different in either group.

The mean dream emotional tone in the sleep apnoea group (3.64) is significantly more negative than in the sleepy/snoring control group (4.21). The mean emotional tone in the apnoea group represents the point between 'slightly unpleasant' and 'neither pleasant or unpleasant', whereas the mean dream tone in the sleepy/snoring control group represents the point between 'neither pleasant or unpleasant' and 'slightly pleasant'. Therefore, although the patients with sleep apnoea syndrome do not have more nightmares or unpleasant dreams, overall their dreams are significantly more negatively toned than for sleepy/snoring controls.

The mean scores for anxiety and depression represents approximately the midpoint of the VAS line between relaxed /anxious and happy/ depressed (the line was 9cm in length). Therefore, the mean mood reported by both sleepy/snoring controls and apnoea patients tends to be in the neutral range with a slight tendency to the negative direction, with mean anxiety and depression slightly higher in the apnoea group. As expected, Epworth sleepiness scale is significantly higher in the group with sleep apnoea syndrome.

Frequency of unpleasant dreams and nightmares

61% of those with sleep apnoea syndrome had at least one unpleasant dream and 23.1% had two unpleasant dreams or more during the 10-day study period. 43.5% of sleepy/snoring controls had at least one unpleasant dream and 30.43% had two or more. When prorated to 1 month, sleep apnoea patients reported a mean of 2.58 nightmares and 3.38 unpleasant dreams. When prorated to one month sleepy/snoring controls report 2.37 nightmares and 2.77 unpleasant dreams. Thus, while not differing significantly from each other, both groups reported more than one nightmare a month and so would be considered by some researchers to be frequent nightmare sufferers (Belicki & Belicki, 1986; Berquier & Ashton, 1992; Hersen, 1971; Kales et al, 1980; Levin, 1989).

Distribution of variables.

Dream variables were assessed for normality. Shapiro-Wilk's test of normality demonstrated that dream frequency (S-W statistic = 0.87; p<0.01), nightmare frequency (0.68; p<0.01), unpleasant dream frequency (0.68; p<0.01) and diprate (0.77; p =0<0.001) are not normally distributed. Dream emotional tone (0.961; p=0.18), mean anxiety (0.998; p=0.90), mean depression (0.981; p=0.63) and ESS were normally distributed (0.977; p=0.52).

Dream frequency.

A spearman's rho correlation was conducted in order to determine if there was a relationship between the number of dreams recalled and severity of sleep apnoea. No significant relationship was found between dip rate and dream recall (r = 0.21; p = 0.16), therefore, dream frequency does not increase with severity of sleep apnoea.

Relationships between nightmares / unpleasant dreams /dream emotional tone and apnoea severity and Epworth Sleepiness Scale

Spearman's rho correlations were conducted for each group in order to examine the relationships between frequency measures and emotional tone with a measure of objective apnoea severity (dip rate) and subjective severity of sleepiness (ESS). Correlations were also conducted with BMI in order to assess any confounding effects of this variable. In order to reduce the probabilities if type 1 errors as a result of multiple comparison the level of significance was set at p=<0.01.

Table 52 - Spearman's rho correlations (p) of frequency measures of unpleasant dream types and emotional tone (ET) with body mass index (BMI), dip rate, Epworth Sleepiness Scale (ESS), for the sleep apnoea group and sleepy/snoring controls.

BMI	Dip rate	ESS
-0.06 (0.80)	-0.27 (0.22)	0.32 (0.14)
0.21 (0.34)	-0.02 (0.93)	0.51 (0.13)
0.03 (0.89)	-0.20 (0.37)	0.47 (0.02)
-0.14 (0.54)	-0.07 (0.77)	-0.61 (0.02)
0.20 (0.33)	-0.08 (0.70)	0.14 (0.54)
-0.08 (0.70)	-0.25 (0.23)	0.02 (0.93)
0.12(0.55)	-0.14 (0.52)	0.05 (0.82)
-0.20 (0.33)	-0.13 (0.56)	
	-0.06 (0.80) 0.21 (0.34) 0.03 (0.89) -0.14 (0.54) 0.20 (0.33) -0.08 (0.70) 0.12(0.55)	-0.06 (0.80) -0.27 (0.22) 0.21 (0.34) -0.02 (0.93) 0.03 (0.89) -0.20 (0.37) -0.14 (0.54) -0.07 (0.77) 0.20 (0.33) -0.08 (0.70) -0.08 (0.70) -0.25 (0.23)

Note: BMI = body mass index; ESS= Epworth Sleepiness Scale; NM freq = nightmare frequency; BD freq = bad dream frequency; UD freq =unpleasant dream frequency; ET = dream emotional tone.

* p = or < .01 (in order to reduce the probabilities of type 1 error as a result of multiple comparisons the level of

significance was set at p = <0.01). All values are two tailed.

Table 52 shows that there are no significant relationships between nightmare-type variables and BMI or dip rate in either group. However, there is a trend for positive correlations between mean dream emotional tone and unpleasant dream frequency with ESS in the sleepy/snoring control group (p = 0.02).

Given these findings for the two groups, and in order to further assess the effect of dip rate on nightmare-type variables the two groups were combined in order to utilize the variation in dip rate within the sleepy/snoring control group as part of the investigation of the apnoea group. Note that there is no difference in BMI between the two groups. Table 53 shows that even when the two groups are combined there are still no significant relationships between unpleasant dream / nightmare measures and apnoea severity, as assessed by diprate.

Table 53 - Spearman's correlations (p) of nightmare/unpleasant dream frequency measures and dream emotional tone with diprate for all participants combined

**************************************	Diprate
Nightmares	-0.11 (0.48)
Bad dreams	-0.11 (0.48)
Unpleasant dreams	-0.12 (0.43)
Emotional tone	-0.11 (0.46)

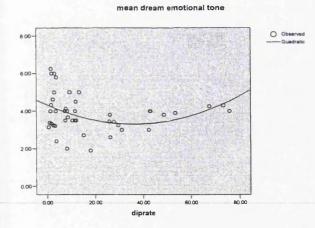
Given this lack of a simple relationship between nightmare-type variables and apnoea severity the hypothesised quadratic relationship was investigated next, using all participants combined.

Quadratic relationships between nightmare frequency and apnoea variables

The relationship between nightmares and dip rate was further investigated using a quadratic regression curve in order to test for the hypothesised inverted U shape function. However, there was no pronounced U shape curve and the model was not statistically significant (F(2,43) = 0.67; P = 0.514, $R^2 = 0.03$), with severity of apnoea only explaining 3% of the variance in nightmare frequency (see appendix 22), therefore, the hypothesis was not supported.

A regression curve (quadratic) was also conducted in order to explore the possibility of an inverted U shaped curve between emotional tone and dip rate (all participants were used). The graph is shown in Fig 5.

Figure 5 - Graph showing the quadratic curve between mean emotional tone and unpleasant dream frequency for all participants



This graph shows a U shaped curve when dip rate is the independent variable and emotional tone is the dependent variable. The regression shows that this model very nearly reaches statistical significance (F (2,14) = 3.13; p = 0.054). According to the

 R^2 value ($R^2 = 0.13$) dip rate accounts for 13% of the variance in emotional tone. However, the graph also shows that there is a greater range in mean emotional tone values for lower dip rate scores, and as severity of the apnoea increases the variance in mean emotional tone decreases. For example, for dip rate values over 40 the variance in emotional tone is small and mean emotional tone of dream seems to cluster between 4 and 5. This suggests that individuals with severe apnoea are having dreams, which are more neutrally toned. Thus, the dreams of people with more severe apnoea, contrary to the initial hypothesis, seem to be more neutrally toned than those with less severe apnoea or no apnoea.

In order to assess these differences in variance in emotional tone, severity of apnoea was divided into three groups defined as the highest, middle and lowest thirds of the dip rate distribution. A one-way ANOVA was conducted, and Levene's test for homogeneity of variance demonstrated a statistically significant difference in variance between groups (4.38; p=0.02). The difference in emotional tone between the three diprate groups was not significant (F (2,42) = 2.68; p =0.08). Therefore, as severity of the apnoea increases the variance in emotional tone decreases significantly, although the mean of emotional tone does not change significantly.

In order to determine whether this difference in variance is confounded by BMI, the variance of BMI at levels of apnoea severity was assessed. Levene's test found no significance difference in variance of BMI between the groups of increasing apnoea severity (1.70; p= 0.19). Thus, the difference in variance of emotional tone can be attributed to the presence and severity of apnoea rather than to any confound with BMI.

Relationships between nightmares/unpleasant dreams and mood

Participants were asked to rate their levels of anxiety and depression every morning for the ten-day study period. As demonstrated in Table 51 mean anxiety and depression did not differ between the apnoea group and sleepy/snoring controls. However, an investigation is needed of whether there are relationships between the mood variables and the frequency variables and emotional tone within the sleep apnoea and sleepy/snoring control groups. Table 54 shows the Spearman's rho correlations of frequency measures and dream emotional tone (ET) with anxiety and depression, for the sleep apnoea and sleepy/snoring control groups separately. Again because a number of correlations were conducted the stricter significance threshold of was used. Table 54 - Spearman's rho correlations (p) of frequency measures of unpleasant

dream types and emotional tone with anxiety and depression, for the sleep apnoea

group and sleepy/snoring controls

	Depression	Anxiety
NM freq (sleepy/snoring control)	0.34	0.39
	(0.12)	(0.07)
BD freq (sleepy/snoring control)	0.03	0.18
	(0.89)	(0.43)
UD freq (sleepy/snoring control)	0.29	0.36
	(0.20)	(0.10)
ET (sleepy/snoring control)	-0.29	-0.30
	(0.29)	(0.10)
NM freq (apnoea)	0.19	0.14
	(0.36)	(0.51)
BD freq (apnoea)	0.36	0.29
	(0.07)	(0.16)
UD freq (apnoea)	0.26	0.18
	(0.22)	(0.39)
ET (apnoea)	-0.29	-0.30
	(0.16)	(0.15)

Note: NM freq = nightmare frequency, BD freq = Bad dream frequency, UD freq = unpleasant dream

frequency; ET= emotional tone.

Table 54 shows that none of the nightmare-type or emotional tone variables are

significantly related to anxiety or depression the following day in either the apnoea or

sleepy/snoring control group.

This raises the question, however, whether state anxiety or state depression are related to the nightmare-type variables longitudinally. This is now investigated.

Within subjects correlations between state anxiety and state depression and nightmare-type variables

Each participant recorded the emotional tone of a dream, if they had one, on each of the 10 mornings, recorded whether the dream woke them up, and then rated their level of anxiety and of depression.

Within subject correlations were computed between the two mood variables and the dream variables:

- 1. Presence or absence of a dream
- 2. Emotional tone of the dream
- 3. Presence or absence of a nightmare, defined with waking criterion
- 4. Presence or absence of an unpleasant dream, i.e. nightmare defined without the waking criterion

As many of the participants' 10 values of anxiety or of depression were not normally distributed, Kendall's co-efficient of concordance was used as the measure of correlation. Within-subject (WS) correlations with emotional tone of the dream were only used in the data analysis if at least 5 dreams had occurred. For the presence/absence of a type of dream/nightmare measures most participants gave a full data set for all 10 nights, participants who provided fewer than 5 data points for a within-subject correlation were omitted from the data analysis for that correlation.

Table 55 - Within subject correlations with anxiety, for the apnoea group and the sleepy/snoring control group. Data presented for each group's within subjects correlations are mean, median, SD, maximum and minimum, number of correlations calculated (n), and number of correlations that were significant (# sig.)

	Apnoea participants							Sleepy/snoring control participants							
Correlate	Mean	Median	SD	Range	n	# sig	Mean	Median	SD	Range	n	# sig	Z	р	
Presence of a	10	14	.26	4630	8	0	.15	.22	.35	7273	14	2	1.84	.065	
dream															
Dream tone	38	36	.33	8119	16	4	52	59	.20	8119	12	3	1.25	n.s.	
Presence of a	.35	.42	.30	3982	15	1	.34	.40	.16	.0448	6	0	.234	n.s.	
nightmare															
Presence of	.33	.43	.37	3982	16	2	.35	.38	.25	.0482	7	1	.434	n.s.	
an unpleasant															
dream															

Table 55 shows that apnoea participants and sleepy/snoring controls have moderately sized correlations between morning anxiety and presence of a nightmare, presence of an unpleasant dream, and dream emotional tone. For the dream tone variable the number of significant within subject correlations is above that expected by chance. The two groups do not differ in the size of these correlations. However presence of a dream has a small negative effect on anxiety for the apnoea participants, but a small positive effect on anxiety for the sleepy/snoring controls. A comparison with Table 54 shows that the between and within-subject correlations of anxiety with nightmare and unpleasant dream frequency are of similar size for the sleepy/snoring control group, but WS correlations are larger for the apnoea group. WS correlations of anxiety with dream emotional tone are larger than the between-subjects correlations of mean anxiety and mean dream emotional tone for both groups.

Table 56 shows that apnoea participants and sleepy/snoring controls have moderately sized correlations between morning depression and presence of a nightmare, presence of an unpleasant dream, and dream emotional tone. The two groups do not differ in the size of these correlations. For the dream tone variable the number of significant within subject correlations is above that expected by chance. However, presence of a dream has no effect on depression for either the apnoea participants or the sleepy/snoring controls.

Table 56 - Within subject correlations with depression, for the apnoea group and the sleepy/snoring control group. Data presented for each group's within subjects correlations are mean, median, SD, maximum and minimum, number of correlations calculated, and number of correlations that were significant.

	Apnoea participants							Sleepy/snoring control participants							
larrelate	Mean	Median	SD	Range	N	# sig	Mean	Median	SD	Range	n	# sig.	Z	р	
nsence of a	.06	.06	.32	4645	8	0	.09	.22	.39	7263	14	1	0.21	n.s.	
ham tone	37	39	.30	7314	14	4	50	48	.22	8112	12	4	1.03	n.s.	
insence of a	.27	.32	.36	2773	14	1	.23	.21	.36	3076	6	1	0.37	n.s.	
nsence of nunpleasant	.25	.28	.39	4176	15	2	.33	.40	.33	3076	7	1	0.32	n.s.	

A comparison with Table 54 shows that the between and within-subject correlations of depression with nightmare/unpleasant dream frequency do not differ consistently. WS correlations of depression with dream emotional tone are larger than the betweensubjects correlations of mean anxiety and mean dream emotional tone for both groups.

CAR

Within subject correlations for effects of morning mood on dreams the next night showed no pattern of significant results, there is thus no association between morning mood and dream variables of the following night.

Comparisons pre and post CPAP

10 patients with sleep apnoea syndrome agreed to take part in a follow up study after a minimum of 3 months successful treatment with CPAP. Table 57 shows the descriptive statistics for demographic variables, dream and well-being variables before and after CPAP therapy.

Table 57 - Descriptive statistics for demographic variables, dream and well-being variables before and after CPAP therapy

	Pre CPAP	Post CPAP	Tests of difference ¹
Dream frequency	7.7 (3.30)	4.7 (3.12)	z = -2.25; p = 0.02*
Nightmare frequency	0.80 (0.63)	1.25 (0.50)	z = -1.34; p = 0.18
Unpleasant dream	1.0 (0.82)	1.6 (0.89)	z = -0.58; p = 0.56
frequency			
Emotional tone of the	3.55 (0.60)	3.99 (0.89)	t = -1.33; p = 0.21
dream			
Anxiety	4.22 (1.21)	2.98 (1.35)	t = 1.72; p = 0.14
Depression	4.15 (1.38)	2.71 (1.06)	t = 2.17; p = 0.07
Body mass index	37.30 (6.71)	37.15 (7.45)	t = 1.43; p = 0.195
Systolic blood pressure	154.60 (21.80)	150.25 (26.71)	t =0.96; p = 0.37
Diastolic blood pressure	90.1 (8.22)	85.12 (10.02)	t = 1.77; p = 0.12
Diprate	37.73 (20.32)	0.54 (0.60)	t = 7.44; p = 0.001
Epworth sleepiness scale	15.10 (3.54)	8.11 (4.81)	t = 4.08; p = 0.004).

Note: 1 tests of difference were either chi squared or t-tests depending on whether the variables were

normally distributed.

* significant at the 0.01 level.

Table 57 confirms the relative absence of apnoeas with CPAP. There are no significant differences before and after CPAP in BMI, systolic and disystolic blood pressure, but subjective sleepiness as measured by the ESS is significantly lower. There are trends towards patients reporting less anxiety and depression when established on CPAP. Contrary to what would be expected with more consolidated sleep, dream recall is significantly lower with CPAP, and there is a slight increase in the frequency of nightmares and unpleasant dreams, but mean emotional tone of dreams changes very little. Levene's test for equality of variance found that there was no significant difference in the variance of emotional tone before and after CPAP therapy (F= 0.20; p=0.899).

Chapter 9

Study 2: Apnoea and nightmares

Discussion

Waking life stress is known to cause negatively toned dreams, and these in turn affect daytime mood (Schredl, 1999). This is stated in the continuity hypothesis of dreaming (Cartwright & Lamberg, 1992), which argues that dreams reflect the waking life concerns of the dreamer. Research reviewed in the introduction to this chapter has supported the assertion that patients with sleep apnoea syndrome have increased waking stress as a result of chronic sleep fragmentation leading to daytime sleepiness and possible mood impairments. Study two assessed between subjects and within subjects correlations between nightmares/unpleasant dreams and mood in subjects with sleep apnoea and BMI matched sleepy/snoring controls, and also assessed the relationship between diprate and dream/nightmare/unpleasant dream frequency using all participants in the analysis.

Prevalance of nightmares

This study found no difference in the frequency of nightmares or unpleasant dreams between participants with sleep apnoea syndrome (SAS) or sleepy/snoring controls. However, interestingly there appears to be an elevated frequency of nightmares in both groups compared to general population norms.

When prorated to 1 month and 1 year respectively, sleep apnoea patients reported a mean of 3.38 and 40.51 unpleasant dreams and sleepy/snoring controls reported a mean of 2.77 and 33.21 unpleasant dreams. Thus, clearly both groups reported more than one nightmare a month and as such, would be considered by some researchers to be frequent nightmare sufferers (Belicki & Belicki, 1986; Berquier & Ashton, 1992; Hersen, 1971; Kales et al, 1980; Levin, 1989). It was found that 61% of those with sleep apnoea syndrome had at least one unpleasant dream and 23.1% had two unpleasant dreams or more during the 10-day study period. 43.5% of sleepy/snoring controls had at least one unpleasant dream and 30.43% had two or more. This is well in excess of population estimates. For example, Hartmann (1984) reported that the average number of nightmares experienced by adults is estimated to be one or two per year. The American Sleep Disorders Association (1990) estimated that 5% of the adult population experience at least one nightmare per month, and Hublin et al (1999) found that 3% of adults had nightmares weekly, whereas 10% had nightmares monthly. Also, a recent study by Spoormaker et al (in press) found that 2.2% of the Dutch population suffered 'much' or 'very much' from nightmares, whereas 7% suffered a 'little'. Thus, it seems that the prevalence of nightmares found in this study in both the sleep apnoea and sleepy/snoring control group (57.6% and 39% respectively for the 10 day study period) is well in excess of that in the general population.

Support for this study's finding of elevated nightmare frequency in the sleepy/snoring control group

It is interesting to note the elevated prevalence of nightmares in the sleepy/snoring control condition compared to population norms. This finding is in line

with Thoman et al (1997), who found that female subjects who reported more snoring also had more nightmares. Furthermore, DeGroen et al (1993) found that snoring was strongly associated with the occurrence of anxiety dreams. It may be that this population have lower well-being compared to the general population and so experience more unpleasant dreams. For example, Aikens, Wallace & Mandelson (1999) found that primary snorers showed elevated scores on the MMPI scales compared to population norms. They suggested that primary snorers might be located along a general continuum between normalcy and sleep apnoea in terms of symptoms and complications, a suggestion supported by Thoman et al's (1997) finding that sleep is more fragmented in snorers than non-snorers, and by the relationship between chronic nightmares and fragmented REM sleep (Hartmann, 1984).

Support from this study for previous studies with SAS and NMS

The findings that patients in the sleep apnoea group report at least 2 waking criterion nightmares a month and 3.38 unpleasant dreams a month supports findings from previous studies. For instance, Guilleminault et al (1996) assessed children with Sleep Disordered Breathing, including those with sleep apnoea and Upper Airway Resistance Syndrome (UARS) and found they had significantly more nightmares/ night terrors and sleep walking than would be expected in the general population. Owen et al. (1997) also reported that unrecalled nightmares were significantly increased in the sleep apnoea group in comparison to population norms. MacFarlane and Wilson (2006) found that 69% of patients with sleep apnoea reported nightmares 'sometimes', although only one patient reported nightmares 'often' and only one 'always'. However, it is not clear what time period 'sometimes' represents in this study, in order to meaningfully compare MacFarlane and Wilson's findings to that of

the current study or to population norms. However, the current study did find that 61% of patients with sleep apnoea syndrome had at least one unpleasant dream in the 10-day reporting period, which is comparable to the MacFarlane and Wilson finding of 69% experiencing nightmares 'sometimes'. This comparison is problematic, however, in that MacFarlane and Wilson (2006) used a retrospective measure of nightmare frequency, which has been shown to produce a lower estimate of nightmare frequency (Wood & Bootzin, 1990; Zadra & Donderi, 2000; Salvio et al, 1992).

Well-being measures in sleep apnoea group and sleepy/snoring controls

It was hypothesised that participants in the sleep apnoea group would have poorer scores on the well-being measures compared to sleepy/snoring controls, however, this hypothesis was not supported. Patients with untreated sleep apnoea and sleepy/snoring controls reported mean levels of anxiety and depression that were in the neutral range on the VAS rating scales of relaxed/anxious and happy/depressed.

Thus, importantly, this study found that mood is not significantly lower in sleep apnoea patients compared to BMI matched sleepy sleepy/snoring controls. Both the sleepy/snoring control and apnoea group are liable to have health problems due to their elevated BMI (such as hypertension), however, the additional effect of having sleep apnoea does not seem to cause a further significant reduction in mood. Moreover, it may be that levels of anxiety and depression are increased in the apnoea and sleepy/snoring control group relative to the general population. This explanation may account for the elevated prevalence of nightmares in both groups. The introduction to this chapter reviewed much evidence suggesting that mood was impaired in patients with sleep apnoea. However, many of these studies used healthy

control comparison groups. For example, a number of studies have suggested that patients with sleep apnoea have elevated levels of depression in comparison to healthy controls (Aiken et al, 1998; Cheshire et al, 1992; Derderian et al, 1988; Engleman et al, 1994; Guilleminault et al, 1977; Kales et al, 1985; Millman et al, 1989; Reynolds et al, 1984). However, healthy control groups may not be the best comparison group to use because they do not take account of the confounding health problems in a high BMI group. For instance, Bardwell et al (1999) suggest that depression and total mood disturbance are related more to age, weight and/or hypertension than they are to sleep apnoea.

Furthermore, symptoms of anxiety and depression are recognised in other respiratory diseases such as asthma, chronic obstructive pulmonary disease and sarcoidosis (Rimington et al, 2001; van Manen et al, 2002; Chang et al, 2001). Thoman et al (1997) also suggests that snorers have elevated health concerns. They found that the 37 snorers in their study reported a number of health problems including heart trouble (n=9), high blood pressure (n=11) and arthritis (n=24). Other studies have also suggested that daytime mood may be impaired in snorers (without sleep apnoea) in comparison to healthy non-snoring controls. Vandeputte and Weerd (2003) demonstrated that 31% of patients who snored had some form of depression (i.e. mild or above using the Beck Depression Inventory), and Mendelson et al (1992) found that primary snorers are just as likely to have been treated for depression in the past as patients with obstructive sleep apnoea.

Importantly, all patients who completed questionnaires in the current study had been referred to the sleep clinic for suspected sleep apnoea and the majority were reporting daytime sleepiness and/or snoring. Thus, even those in the sleepy/snoring control group had a complaint that warranted screening for sleep apnoea. Moreover, patients had not yet received a diagnosis for sleep apnoea when they completed the sleep and mood diaries, which may have created anxiety and uncertainty in both groups. The sleepy/snoring control group reported a mean Epworth Sleepiness Score of 10.48, which is only slightly less than the criterion for moderately pathological sleepiness (ESS<11 is normal, ESS 11-15 = moderate sleepiness).

However, in contrast to the current study, Aikens et al (1999) found that approximately twice as many sleep apnoea patients than primary snorers fell into the disturbed range for the Depression scale (49% versus 25%) and the Hysteria scale (35% versus 16%). Furthermore, Watson et al (1987) correlated the BDI and sleep apnoea syndrome and found a significant correlation between depressive symptomatology and the frequency of apnoeas-hypopneas per hour of sleep. In contrast, dip rate was not correlated with anxiety and depression in the current study. However, the current study did not use standardised trait measures of well-being.

Within subjects correlations

Within-subjects correlations between the presence of an unpleasant dream or nightmare with anxiety and depression the following day were no different in the sleepy/snoring control group compared to the apnoea group. Therefore, although it may be expected that the presence of an unpleasant dream would have more of a detrimental effect on patients with sleep apnoea than sleepy/snoring controls, this was not the case. In study 1 the WS correlations anxiety with presence/absence of a nightmare or unpleasant dream were both .24, and the WS correlations of depression with presence/absence of a nightmare or unpleasant dream were .26 and .28. In the current study, apnoea and sleepy/snoring control groups did not differ in size of WS correlations, these being .33 - .35 for anxiety, and .23 - .33 for depression. As these are Kendall's correlations, the effects are of moderate size. The main important conclusion, however, is that the individuals undergoing investigation for apnoea had a greater anxiety response to nightmares and unpleasant dreams than did the participants in study 1.

The apnoea and sleepy/snoring control participants differed in their betweensubjects correlations of mean anxiety and mean depression with number of nightmares and number of unpleasant dreams: apnoea group had Spearman correlations of .14 - .26 and sleepy/snoring controls had correlations of .29 - .39. In contrast, Study 1 had these between-subject correlations of .41 to .50. It is not clear why for this one class of correlation, the apnoea group had such low co-efficients.

Nightmares, dream emotional tone and severity of sleep apnoea

Our initial analysis of the results followed previous work in comparing an apnoea group with sleepy/snoring controls. In accordance with previous research there was not a higher number of nightmares in the apnoea group. There was, however, significantly more negative emotional tone for the apnoea group. This finding is in line with some previous research. For example, Santamaria et al (1998) found that the dreams of patients with untreated sleep apnoea were predominately negatively toned. However, as with work on nightmare frequency, treating apneoic patients as a group can result in no difference from controls. For example, Schredl's (1998) content analysis of the dreams of patients with sleep apnoea did not reveal a preponderance of negative emotions compared to healthy controls.

The lack of conclusiveness in the literature about negative dreams and apnoea had resulted in the hypothesis that there would be an inverted U shaped curve between nightmare frequency and severity of apnoea (using a 5% Sp0₂ dip rate measure, and combining the high diprate apnoea group with the lower diprate sleepy/snoring control group). Thus, patients with moderate sleep apnoea would show a higher frequency of nightmares/unpleasant dreams as a result of increased waking stress, but nightmares/unpleasant dreams would be lower in patients with severe apnoea, as despite increased waking stress these patients would not have sufficient periods of consolidated sleep to support dreams or nightmares. However, the quadratic regression curve between dip rate and nightmare frequency in the current study was not statistically significant, but the quadratic curve between mean dream emotional tone and dip rate was very nearly significant (p = 0.054). This graph showed that the dreams of patients with mild/moderately apnoea were more negative than the dreams of sleepy/snoring controls and severely apnoeic patients, as hypothesised, but, their dreams were not unpleasant, instead being around the neutrally toned mark.

Rather than differences in mean emotional tone, what was striking about the graph was that as diprate increased, there was a reduction in variation of dream emotional tone. When dip rate was separated into three group of increasing severity, Levene's test for homogeneity of variance confirmed that the difference in variance was significantly different. Thus, patients with severe apnoea are having fewer positively charged and fewer negatively charged dreams. An explanation of this could be that the sleep of patients with higher diprate is so fragmented that it interferes with the process of dreaming, thus not allowing dreams plots and dream emotion to develop. This is in line with Schredl et al's (1999) finding that high Respiratory Disturbance Index (RDI) was related to more realistic, less bizarre, less intense dreams with lower negative emotions. These findings are in line with the finding that as apnoea severity increases dreams become more neutrally toned (i.e. less negative, bizarre and intense). Of relevance to this is Merritt et al's (1994) demonstration that positive emotions were most likely to appear in the first quartile of the dream and negative emotions in the last two quartiles. They note that while 42% of reports containing emotions began with a positive emotion, only 24% ended with one. Furthermore, 58% of dreams began with a negative emotion and 76% of the final emotions were negative, suggesting that dream emotions tend to go 'from bad to worse' (Merritt et al. 1994, p56). Therefore, it could be sleep is so fragmented in patients with severe sleep apnoea that there is not a sufficient amount of consolidated sleep for dreams to go 'from bad to worse' and as a result dreams remain predominantly neutrally toned.

These findings suggest that sleep fragmentation actually interferes with the process of dreaming rather than with the recall of a dream. Stepanski et al (1984) reported that patients with sleep apnoea spent only 10.3% of their total sleep time in REM sleep compared to healthy controls that spent 17.4% of their sleep time in REM sleep. Thus, it can be proposed that the greater the severity of apnoea the less time is spent in REM sleep and the less time there is for dream plots to develop. The results therefore suggest that it is those patients with milder apnoea who report more

unpleasant dreams, and that the current finding that the apnoea group as a whole had more negative mean dream emotional tone than the sleepy/snoring controls was because of the patients with less severe apnoea.

Dream frequency and apnoea.

This study found a trend towards higher dream recall in the sleep apnoea group (p = 0.15 for a 2-tailed test). Dip rate, however, was not significantly associated with dream recall frequency. These are in line with previous findings. For example, Schredl et al (1998) found that neither the Respiratory Disturbance Index (RDI) or maximal decrease of blood oxygen saturation as severity measures were significantly correlated with dream recall frequency. Furthermore, although dream recall frequency has been found to be elevated in patients with sleep apnoea in comparison to healthy controls (Gross and Lavie, 1994; Schredl et al, 1998, 1999), Schredl et al (1998) argued that the heightened dream frequency in their sample was best explained by the lower emotional balance in the evening present in the patient group. They argue that this supports the salience hypothesis of dream recall (Cohen & MacNeilage, 1974) in that the more negative the presleep mood the more negative the dream affect and the higher the probability of dream recall. They conclude that heightened dream recall in patients with sleep apnoea can be partly explained by the fractionated sleep profile (frequent arousals) and by daytime stress.

Dream emotional tone and subjective sleepiness

There was a trend for mean dream emotional tone (and unpleasant dream frequency) to be positively related to subjective sleepiness (ESS) in the sleepy/snoring

control group but not in the sleep apnoea group (p = 0.02). This can be accounted for as follows.

In the sleepy/snoring control group as subjective sleepiness increases so does unpleasant dream frequency, and dream tone becomes more negative. This may be due to stress-related aspects of sleepiness. However, a significant correlation would not be expected in the apnoea group as the sleepier people in the apnoea group have a higher diprate, and thus have more neutrally toned dreams irrespective of stress. There is no association of dream emotional tone and dip rate in the sleepy/snoring control group, possibly because of the low range of dip rate scores in the sleepy/snoring control group (0.60 –9.40, as compared to the apnoea group's diprate range of 7.40-30.95). It may be that ESS is more informative of the effect of sleep disruption for the sleepy/snoring control group, as opposed to the apnoea group, where previous research indicates that the correlation between sleep AHI and RDI indices and the ESS is weak (Chervin & Aldrich, 1999; Chervin, Aldrich, Pickett & Guilleminault, 1997; Engleman et al. 1997; Kingshott et al, 1995, 1998).

Mood before and after CPAP

Mean levels of anxiety and depression decreased following CPAP but the differences were not significant. There was no decrease in BMI or blood pressure after CPAP. These findings accord with previous studies. For example, Derderian et al (1988) reported that patients with severe sleep apnoea (AHI = 40.7) showed significant improvements in POMS depression, fatigue and total mood disturbance two months after treatment with CPAP. Ramos-Platon and Sierra (1992) found a decrease in scores on the MMPI, particularly on the depression scale, one year following treatment with CPAP. Engleman et al (1994) found a reduction in anxiety

and depression using the Hospital Anxiety and Depression Scale (HADS) with CPAP. Sanchez et al (2001) also assessed the effectiveness of CPAP treatment on levels of depression, state anxiety and trait anxiety. They assessed 51 patients with sleep apnoea (mean AHI = 61.32) before, after 1 month and 3 months of CPAP treatment. They found that CPAP therapy produced a decrease in depression scores 1 month and 3 months following CPAP. Trait anxiety was decreased 1 and 3 months after treatment and a decrease in state anxiety was found at 3 months after treatment, suggesting that decreases in depressive symptoms occur earlier than decreases in anxiety. Indeed, the current study found that depressive symptoms were reduced more than those of state anxiety after a minimum of three months on CPAP.

However, Borak et al (1996) found no significant improvement in emotional status at 3 months or one year after CPAP. This is possibly due to the severity of the apnoea in their study, the mean AHI in the Borak et al study was 67, as against 38.05 (N= 19) in the current study. The mean AHI in the Bearpark et al (1987) and Engleman et al (1994) studies was 30 and 28 respectively which is similar to our sample and these authors also found improvements in mood following successful CPAP therapy. Therefore, perhaps improvements in mood are less evident in patients with very severe apnoea following CPAP therapy. It may be that mood impairments in patients with very severe apnoea may be in part a result of dysfunction in the prefrontal cortex (PFC), manifested behaviourally by so-called 'executive dysfunction' (Beebe & Gozal. 2002). One symptom of weakness in executive functioning is difficulties with the self-regulation of affect (e.g. irritability and affective lability). Dahl et al (1996a,b) have used a PFC model to account for the association betweens sleep disruption and emotional disturbances in children, which is consistent with poor behavioural inhibition, and attenuated self-regulation of affect and arousal state in some patients with apnoea. Indeed, some studies have concluded that patients whose sleep has been normalised by CPAP may continue to show deficits on executive functioning tests (Bearpark et al, 1987; Bebard et al, 1993; Feuerstein et al, 1997; Naegele et al, 1995).

CPAP and the frequency of dreams and nightmares

This study found a significant reduction in dream recall following treatment with CPAP therapy. This had also been found by Santamaria et al (1998), and is in line with the Functional State-Shift Model (Koukkou & Lehmann, 1983), where the number of nocturnal awakenings/arousals becomes less, resulting in a lower state of brain arousal than when untreated, and hence a decrease in dream recall. This finding is also in line with the arousal-retrieval model of dream recall (Koulack and Goodenough, 1976), which holds that increased awakenings result in increased dream recall. However, mood measures showed a tendency to improve with CPAP treatment, and therefore the effect of a more negative presleep mood causing increased dream recall pre-CPAP cannot be excluded.

When on CPAP therapy, patients showed a slight increase in the frequency of nightmares and unpleasant dreams, but mean emotional tone showed a slight tendency to become more positive (although these differences were not significant). This suggests that there may be more range in the emotional tone of dreams following CPAP. However, Levene's test for equality of variance found that there was no significant difference in the variance of emotional tone before and after a minimum of three months of CPAP therapy. As with the results of the current study, Gross and Lavie (1994) reported that dream reports from untreated nights were more negatively toned than dreams after nights with CPAP. Although Schredl (1998) argues that the Gross and Lavie finding may have been an artefact, in that patients underwent a night without their CPAP machines which may have triggered anxieties about the possible effects of recurring apnoeas, that objection does not apply to the current study.

Limitations of study 2

The sleepy/snoring control group was used rather than healthy controls as it enabled control over such confounding factors as BMI, sleepiness, hypertension, and recruitment from hospital. It was intended to recruit, from a slimming organisation, a second control group with matched BMI but who were not complaining of sleepiness; however, this was not possible.

A further limitation of this research was that standardised measures of wellbeing, which could have been compared with population norms, were not included. Instead, this study used VAS state measures of anxiety and depression, in order to minimise what was required of the participants. However, study one found mean VAS ratings were significantly correlated with SCL- anxiety and depression as well as with state and trait anxiety, although such correlations may have been increased if the VAS had assessed maximum or average daily anxiety or depression, as in Cellucci and Lawrence (1979), rather than the morning state.

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The follow up study assessing dreams after CPAP consisted of only ten patients. This was in part due to some of the baseline apnoea group patients not being treated with CPAP, or having very poor compliance with CPAP was poor and so they could not be included in the study. Research with a larger sample size is needed to explore the effects of CPAP on variance of dream emotional tone.

Summary of novel findings

This study found that, as diprate increased there was a significant reduction in the variation of dream emotional tone, however, there was no significant difference in mean emotional tone. Thus, it seems that as sleep apnoea increases in severity dreams become more neutrally toned.

Summary

This study has found that two groups with various aspects of chronic poor psychophysiological well-being have more nightmares / unpleasant dreams than the normally healthy population, and that the apnoeic group has a more negative mean emotional dream tone than the sleepy/snoring group. Study 1 had found relationships between nightmare / unpleasant dream frequency and poor psychological well-being, including life-event concerns and traumas. Study 3 now follows on from these with the aim of assessing the effect of frequent waking life stresses, as assessed by a posttraumatic stress scale, on dream emotion and nightmare / unpleasant dream frequency.

Chapter 10

Study 3: Nightmares in Fire-fighters

Introduction

Nightmares in Emergency Service Personnel

This chapter looks at the impact of exposure to traumatic incidents on the frequency of nightmares by assessing nightmares and their correlates in one group of emergency service personnel. To data, there have been no systematic studies of the frequency of nightmares in this population. It is hypothesized that nightmare prevalence will be elevated in this population and that the frequency of nightmares will have significant relationships with measures of well-being. Study one looked at the impact of life events on nightmare frequency and found that life events had stronger correlates with nightmare distress rather than the prevalence of nightmares. Yet it could be argued that the life events listed in study one are not sufficiently traumatic or frequent enough to highlight a relationship with the frequency of nightmares. Study two looked at repeated failures of breathing, finding a high incidence of nightmares for the group but no linear relationship between nightmares and frequency of apnoeic episodes within the group. Yes, of course, participants had no real knowledge of the severity of their condition. Study 3 aims to assess emergency service personnel as this population are repeated exposed to traumatic incidents.

Prevalence of traumatic stress in the general population

Although most people experience a certain amount of stress and daily hassles, serious traumatic events are rare and unexpected and, thus, not a common occurrence in most people's lives. The following studies highlight the prevalence of traumatic stress in the general population. Norris (1992) conducted a study of 1000 people (age range 18-60+) from four Southern US cities. She found that 69% experienced at least one major traumatic stressor over their lifetime and that 21% reported experiencing at least one traumatic event in the past year. In a survey of 1000 people in Winnipeg, Canada, Stein and colleagues (1997) found that 74% of women and 81% of men reported at least one traumatic event, and 46% of women and 55% of men reported experiencing more than one event. In the largest prevalence study of trauma, Kessler et al (1995) surveyed a representative national sample of 5877 persons (2812 men and 3065 women) in the United States. This study assessed 12 categories of trauma (rape, molestation, physical attack, combat, shock (trauma to significant other), threat with weapon, accident, disaster, witness, neglect, physical abuse and other trauma. They found that the majority of people had experienced at least one event. Approximately one quarter of the sample reported experiencing only one major trauma. 15% of men and 14% of women reported experiencing two, 10% of men and 5% of women and experienced three and 10% of women and 6% of men had experienced four major traumatic events. Therefore, over 35% of the men and 25% of the women reported more than one major traumatic event in their lives so far.

Trauma and sleep

There is little doubt that sleep is severely disrupted following exposure to a traumatic event. For example, Kato et al (1996) interviewed people three and eight

weeks after the Hashin earthquake in Japan in 1995. Sleep disturbance was the most frequently reported symptom (63% reported sleep disturbance at 3 months and 46% at 8 weeks). North et al (1999) found that 70% of survivors of the Oklahoma City bombings suffered sleep disturbances 6 months after the event and more than 50% had nightmares. Schuster et al (2001) used randomised digit dialling in the United States within 1 week of the September 11 terrorist attack. They questioned 560 adults about their own reactions and the reaction of their children (n = 170). They reported that 11% of adults and 10% of children had significant difficult falling asleep or staying asleep since the attacks. However, for most people the sleep disturbances following trauma are transient (Lavie, 2001) but for others the sleep disturbance becomes a persistent problem and typically forms part of the Acute Stress Disorder (ASD) and post traumatic stress disorder (PTSD) diagnoses (as discussed in the general introduction, chapter 1).

Dreaming, nightmares and trauma

This section commences with an account of the effects of being a victim of trauma on dreams. The last paragraph of the section concerns those who witness the trauma of others. It is acknowledged that this distinction is, in reality, blurred, and that the witnessing of trauma can itself be traumatic.

A number of researchers have examined the effects of trauma on dreams and nightmare. Hartmann (2001) states that dream content is altered after a trauma but that in the majority of people as the trauma resolves 'more and more usual' dream material is introduced along with the direct or metaphorical representations of the trauma (p2). He states that within time dreams will eventually return to their normal pattern. Thus, it seems that there is a gradual integration and making of 'connections' between the traumatic event with other events in the person's life. Hartmann (1973, 1991b) postulates that the function of dreaming is to make connections between recent material and older memories and this process can be reflected by changes in dream/nightmare content after a trauma. He suggests that the PTSD nightmare involves an absence or 'failure' or this connecting' or 'absorbing' process.

Hartmann (1996) suggested that emotions are central to prominent dream imagery and that the dream images create a picture context for, or 'contextualise' the dominant emotional concern of the dreamer.

Nightmares have been reported to occur in a large percentage of the population following an intensely frightening or highly emotional experience (Hefez et al, 1981; Ross et al, 1989). For example, Wood et al (1989) conducted a systematic evaluation of the effect of an earthquake (7.1 on the Richter scale) on the frequency of nightmares in a sample of students living in the San Francisco area where the hurricane struck, compared to control subjects from an unaffected university. A 3week prospective measure of nightmare frequency was obtained. It was found that nightmares were most frequent among subjects close to the earthquake. For example, over the 3 week period approximately 40% of the San Jose State students and Stanford students reported one or more nightmares about an earthquake, as compared with only 5% of the control students from Arizona. This finding supports the view that the experience of a potentially traumatic experience can result in more frequent nightmares, particularly about the event itself. However, the authors did not find that the nightmares about the event were significantly more intense than those not about the event. One study found that the dreams of Palestinian children living in Gaza under violent conditions contained more aggression, persecution and negative emotions than in the dreams of children living in a more peaceful area in Galilee (Punamaki, 1997, 1998). Similar results on Palestinian children were found by Valli et al (2006).

For those who witness events, even on the television, there can be an effect on dreams. Hartmann and Baslile (2003) examined a series of twenty dreams - the last ten dreams recorded before 9/11/01 and the first ten dreams recorded after 9/11/01 - from sixteen individuals in the United States who regularly record all their dreams. Blind scoring demonstrated that dreams after 9/11/01 had more intense imagery, did not contain significantly more content related to the attacks. Nielsen et al (2006) obtained retrospective estimates of nightmare frequency for 23,990 respondents to an Internet questionnaire. An increase in nightmare frequency was observed post-September 11 only for male respondents - particularly for 10- to 29-year-olds. This increase was sustained 2 years later. These effects were maintained when dream recall was held constant. The authors state that the nightmares of males may be differentially sensitive to traumatic events for which victims and/or perpetrators are primarily male.

Emergency services: a 'high risk' population?

Evidence suggests that there is a relationship between exposure to traumatic events and physical and psychological well-being (Everly & Lating, 1995; James, 1988; Tedeschi, Park & Calhoun, 1998). However, research has tended to focus on direct victims of trauma and only recently has widened to include the potentially adverse effects of traumatic incidents on those who aid the victims (Grevin, 1996; Mitchell & Dyregrov, 1993). Due to the high level of exposure to traumatic incidents, it has been demonstrated that a range of professionals, including police, ambulance and fire personnel, can become secondary victims of trauma, exhibiting symptoms akin to direct victims (Genest, Levine, Ramsden & Swanson, 1990). One reason why emergency services may not have been investigated as extensively as 'primary victims' of disasters (Clohessy & Ehlers, 1999) could be due to the perception that victims are 'helpless and resourceless', whereas helpers are 'strong and resourceful' (Short, 1979). However, emotional distress is recognised to be widely prevalent among emergency care workers (Mock et al, 1999).

There is little published research as to the actual prevalence of traumatic stressors experienced by emergency service personnel, however, it is undoubtedly far in excess of that experienced by the general population. Emergency workers are required to cope with extraordinary and persistent occupational demands that are cumulative. These include threats to their own and co-workers safety, dangerous fire suppression, injuries and deaths, injuries and deaths of children or infants, gruesome victims incidents, body handling, completed suicides and mass casualty accidents (Beaton and Murphy, 1995; Corneil, 1995). Beaton et al (1995) found in a survey of self-reports of 2,000 professional fire-fighters and paramedics that approximately 90% were confronted with at least one distressing mission including dead, dying or severely injured persons within the past year. Clohessy and Elhers' (1999) sample of 56 ambulance workers reported that incidents involving children were the most stressful (32%), followed by organisational managerial problems (29%), dealing with distressed relatives (23%), dealing with dead or dying patients (16%) and shift work (14%). Encountering personal threats and dealing with uncooperative and abusive

patients have been identified as factors that contribute to the stress of emergency medical service personnel. Little research has examined the degree to which such factors affect the stress and anxiety levels of emergency medical services personnel and even less is know about their anxiety level at work.

The National Commission on Fire Prevention and Control has declared that fire fighting is the most dangerous occupation in the United States (Hildebrand, 1984a). Everyday approximately 280 fire fighters are killed or injured. Every year, over 650 are forced to retire due to occupational illness, including psychological stress (Hildebrand, 1984b).

Limitation in examining distress in emergency services workers

There are a number of inherent methodological and theoretical difficulties in studying emergency service workers. They are undoubtedly a self-selected occupational group and may not be representative of the general population. It could be that they are more resilient to dealing with stress and trauma as they selected themselves for the profession. However, Moran and Britton (1994) concluded from their sample of emergency workers that they were no 'hardier than most....(nor possessed any) particular coping styles' and that the coping and personality measures used in their study were not statistically significant predictors of their reactions to traumatic incidents. However, some researchers claim that coping resources of emergency service workers are based on experience and years of service. For example, Hytten and Hasle's (1989) findings suggest that experienced or 'seasoned' emergency workers possess more effective cognitive and behavioural coping strategies. Moreover, those who choose not to take part in research studies may be suffering from more psychological distress, particularly in light of the fact that avoidance is one of the characteristics of PTSD. Alternatively, it could be that those choosing not to respond are those who are not experiencing any significant problems and therefore see no value in completing research questionnaires, thus inflating the apparent level of psychopathology of the group.

Increased occupational stress

Several studies have demonstrated high levels of occupational stress in emergency service workers (Cydilka et al, 1980; Hammer et al, 1990). Furthermore, work stress and low social support have been associated with high levels of depression and anxiety among those employed in the health care services (Bennett et al 2001;Tennant, 2004). Clohessy and Ehlers (1999) found that 29% of ambulance workers reported that organisational/ managerial problems were among the most distressing aspects of their work. Violant and Aron (1993) found that organisational stressors, mediated by job satisfaction and organisational goal orientation, increased psychological distress over six times more than inherent police stressors in a sample of American police officers.

Bennett et al (2004) found that 'caseness' on the Posttraumatic Diagnostic Scale (Foa, 1995) was predicted by the degree of stress experienced as a result of organisational factors, whereas, incident factors were not predictive of 'caseness'. These authors also found that organisational stress contributed more to levels of anxiety and depression than the stress associated with incidents. These findings suggest that organisational factors need to be considered when planning to minimise distress amongst ambulance personnel. However, this study does not provide details on the specific issues that may need to be addressed in preventative strategies. Furthermore, the study is cross-sectional and as such it is difficult to determine the direction of the relationship between the dependent and independent variables. It may be that depressed or anxious workers may have distorted perceptions of the level of work stress and as such inflate the association between these variables. Therefore, these studies seem to suggest that occupational factors lead to elevated levels of stress in emergency service workers and in some incidents can lead to increased levels of psychopathology.

Well-being in emergency service workers

The stress associated with emergency responses may also contribute to more general problems of depression or anxiety. Bennet et al (2001) found that 10% of their 617 sample of British Ambulance workers reported 'probable' levels of clinical depression and 22% reported 'probable' levels of anxiety based on the HADS scores. Regression analysis showed that the strongest predictor of anxiety was background stressors such as work-home conflict, tension with colleagues, and the unpredictable nature of the work. Incidents with children were the only incident stressors to independently predict anxiety scores. Together, it was found that these variables accounted for 38 percent of the variance in anxiety scores, and a similar set of predictors accounted for 31% of the variance in depression scores.

Alexander and Klein (2001) in a sample of 110 Scottish ambulance workers, found that 32% met the GHQ screening criteria for 'caseness'. Mock et al (1999) assessed anxiety levels in emergency medical service providers in order to determine the effects of experienced violence during a shift and the effects of different shift schedules. In a three month prospective observational study, 23 emergency medical technicians (EMTs) and 40 paramedics were observed. It was found that paramedics had lower anxiety scores than EMT's and that years of experience decreased anxiety scores. They found that there was no significant difference in anxiety state scores (using the Spielberger State-Trait Inventory) between those EMS providers who had encountered violence during the preceding 12 hours and those who had not. Furthermore, they conclude that although the working environment of EMS providers contains many stressors, this population were no more anxious than the general working public. Furthermore, they found that there was no difference in anxiety levels in providers who worked 12 and 24 hour shifts.

Prevalence of PTSD

The majority of studies assessing psychopathology in the emergency services have focused on the examination of post traumatic stress disorder symptoms. As emergency service personnel encounter potentially traumatic situations with some regularity, this may make them increasingly prone to develop PTSD, as research generally suggests that repeated exposure to potentially traumatic incidents has a sensitising effect, increasing vulnerability to the condition (Breslau, Chilcoat, Kessler & Davis, 1999; Dougall, Herberman, Delahanty, Inslicht & Baum, 2000). Indeed, studies of emergency staff responding to disasters report PTSD prevalence rates of between 10% and 17% (Anderson, Christensen and Petersen, 1991; Durham, McCammon and Allison, 1985; McCammon, Durham, Allison & Williamson, 1988; Weiss et al, 1995).

PTSD in ambulance workers

Rentoul and Ravenscroft (1993) studied the London Ambulance Service and found that 15% of frontline staff could be given a full diagnosis of PTSD. Of their sample, 53% met the criteria for 'recent mental disturbance'. Thompson and Suzuki (1991) assessed 40 experienced ambulance workers selected randomly from the London Ambulance service, using the Impact of Events Scale (IES; Horowitz et al, 1979) and the General Health Questionnaire (GHQ; Goldberg & Hiller, 1979). They found high scores on the intrusion scale of the IES and 60% showed signs of 'probable psychological distress' (as identified by the GHQ). Bennett et al (2004) assessed the prevalence of PTSD in a sample of 617 British Ambulance workers using the Post-traumatic Diagnostic Scale (PDS; Foa, 1995). They found that the overall prevalence of PTSD in this sample was 22%. This rate is similar to the average rate of 21% reported in other studies (Grevin, 1996; Clohessy and Ehlers, 1999). Bennet et al report that just under half their participants reported currently having 'troubling memories of events that have occurred in work', while nearly two-thirds reported having had such memories at some time in the past. They found that a high percentage of paramedics rather than Emergency Medical Technicians reported having troubling memories of the past. It was reasoned that as paramedics are more senior than emergency medical technicians this difference might reflect length of time working in emergency setting rather than any inherent difference between the two groups. Regression analysis showed PDS scores to be independently associated with the stress resulting from organisational factors, the frequency with which traumatic incidents were encountered, length of service and dissociation at the time of the incident. These variables accounted for 48% of the variance in PDS scores. These authors also found a gender difference in prevalence of PTSD. 32 women (35%) and 261 (51%)

completed the PDS, indicating experiencing recurrent memories of an event (or events) for at least one month: The percentage of the overall sample to achieve the criteria for a diagnosis of PTSD was 15 percent among the female and 23 percent among male ambulance personnel. This difference was found to be statistically significant ($X^2 = 4.67$; p<0.05). Thus, this study suggests that women report significantly lower levels of PTSD and also anxiety than men, although this finding is tentative due to the small number of women in the sample. However, this finding certainly requires more investigation as it is in contrast to the trends found in the general population (Kessler et al, 1995). It is also possible that women had better access to, or better utilised social support or other coping mechanisms in comparison to men, and it may also be that women who select themselves for the ambulance service are hardier than those in the general population.

PTSD in mountain rescuers

Mountain guides constitute a high risk population in terms of the development of primary and secondary PTSD symptoms because of adverse mountain conditions, sudden changes in weather conditions, avalanches, rock or ice falls and crevasses, which are a risk to themselves and their clients. Furthermore, many mountain guides are members of mountain rescue services and as such often risk their lives in search and rescue operations or the recovery of bodies. Sommer et al (2004) assessed the prevalence of PTSD symptoms in Swiss mountain guides. 552 participants completed the Posttraumatic Stress Diagnostic Scale (PDS), the General Health Questionnaire and the Sense of Coherence Scale (SOC-29). These authors found that although Swiss mountain guides had been exposed to many traumatic situations, each involving threat to life, serious injury, or death, the prevalence rate of PTSD was very low (2.7%), and subsyndromal PTSD was established in just 1.5% of participants. Furthermore, they found that the mountain guides who were involved in mountain rescue teams did not differ significantly from those who were only involved as expedition leaders or guides, but they did show significantly lower GHQ total scores and higher SOC scores than non-rescuers. According to the GHQ-28, approximately 75% of the sample showed no signs of mental disorder, while less than 25% were identified as showing such signs. The low prevalence of PTSD in this population is not consistent with the findings from other high-risk populations. The authors highlight that studies relating to PTSD in different types of rescue workers, of similar age and with a comparable duration of professional experience, report prevalence rates of more than 18% (Wagner et al, 1998; Clohessy and Ehlers, 1999, as described above). The findings of this study are important as they suggest the existence of protective factors in this population. For example, the authors suggest that the development of PTSD may depend on the subjective appraisal of the traumatic situation. Ballenger et al (2000) have highlighted the high correlation between the level of danger perceived by the individual exposed to the trauma and the likelihood of developing PTSD. This suggests that a high tolerance of the mountain guides towards objectively dangerous situations may be the reason for their low rate of PTSD symptoms. Although the gender bias in this study may be a factor, it is unlikely that this can account for the low levels of PTSD since comparable high-risk groups with higher prevalence rates also show a predominance of male participants (Corneil et al, 1999; Wagner et al, 1998; Gershon, Lin & Li 2002). This study also suggests that SOC cannot be seen as a protective factor against PTSD but rather as a psychological marker for the degree of health and/or disease.

Bennett et al (2004) found that the severity of PTSD was also predicted by the frequency of incident-related stress and length of service. This suggests that risk of developing PTSD increases over time rather than lessening of impact as individuals habituate the their experiences 'on the road'.

PTSD in Fire fighters

Bryant and Harvey (1995) reported that 37% of their fire-fighters suffered from PTSD symptoms. McFarlane (1989) found prevalence rates of 32%, 27% and 30% in a sample of fire fighters 4, 11 and 29 months after an Australian bush fire. About 20% of these fire fighters had perceived the situation as life threatening, 41% had to protect themselves from the fire, 23% suffered property damage and 25% were injured (McFarlane, 1987). These authors found a tendency of fire fighters to avoid thoughts and feelings associated with a traumatic event. However, in this study many of the fire fighters were personally affected by the disaster in that they were victims, not only helpers. Hence, it can be argued that this study doesn't reflect prevalence rates of PTSD arising just from helping other people in an emergency.

Wagner et al (1998) investigated the prevalence of PTSD and co-morbid symptoms among professional fire fighters. They examined a sample of 402 German fire fighters (mean age = 39.68; SD = 9.68); their average job experience was 15.80 years (SD=9.66). Participants were administered the General Health Questionnaire (Goldberg and Hiller, 1979), the PTSD symptom scale (Foa et al, 1995) and a number of subscales measuring 'openness', substance misuse and bodily complaints. They found that a prevalence rate of PTSD symptoms of 18.2%. This prevalence is lower than in the McFarlane study, however, the latter study involved fire fighters who shared first hand the experience of a severe traumatic event. Approximately 27% of participants had a mental disorder according to the GHQ. An analysis of the comorbidity of PTSD with other psychiatric dysfunctions showed that 39.7% of fire-fighters with PTSD symptoms (N=23) suffered from depressive mood, 60.3% (N=35) displayed social dysfunctions and 19.0% (N=11) were substance abusers. These authors also reported that in comparison to the general population fire fighters with PTSD symptoms showed a significantly higher level of substance abuse and bodily complaints, especially cardiovascular symptoms, tension and pain. Regression analysis revealed that job experience and the number of distressing missions during the last month were significant predictors of the extent of traumatic stress in fire fighters.

Haslam et al (2003) assessed 31 staff from a Fire and Rescue Service using the Post-traumatic Diagnostic Scale (PDS: Foa, 1995) and in depth interviews concerning incidents and symptoms of PTSD. All were in or had been in active service. They found that the most common event to cause distress in fire fighters was child fatalities (31%), serious accidents (28%), followed by life-threatening illness in the fire-fighters own family and 'other' (e.g. death within their family). Two participants met the DSM-IV criteria for PTSD. However, these events were related to life threatening illnesses in their family and other traumatic incidents rather than as a direct experience of their work. A number reported that they had experienced one or more of the symptoms associated with the symptoms of PTSD. The most common symptoms were feeling emotionally upset when reminded of the traumatic event (55%), having upsetting thoughts or images about the event (32%), and having trouble falling asleep or staying asleep (23%). Of the 22 that reported one or more PTSD symptom, 13 had experienced the symptoms for more than 3 months, 3 for between 1 and 3 months, and 6 had experienced the symptoms for less than once a month. Half of those who reported one or more symptoms said that these symptoms had not interfered with other areas of their life. Of those who reported some interference, the most common area was 'general satisfaction with life' (55%); this was followed by 'relationships with your family' (45%) and 'overall level of functioning in all areas of your life' (36%). The qualitative data yielded by this study found that the most common psychological response (65%) cited after an incident was rumination, and specifically worrying about similar results happening to them or their families. Rumination has been previously related to PTSD symptom severity (Ehlers, Mayou & Bryant, 1998). The second most common response was trouble sleeping. One third of participants stated that they had tried to avoid thoughts or feelings associated with the incidents and many stated that if they did not do this that they would be unable to do their job. Flashbacks were cited by over one-quarter of the participants. Some were associated with feelings of helplessness, other were associated with incidents involving horrific injuries and were most common among road traffic accidents. Hypersensitivity to smell was also reported, the most commonly reported being the smell of burnt flesh. Such symptoms of hyper-arousal are characteristic of PTSD (Helzer et al, 1987). The fire fighters in this study stated that feelings of helplessness lead to beliefs that they should have done more, which causes incidents to be perceived as traumatic. Such feelings of guilt and helplessness are also characteristic of PTSD (Briere, 1998).

Conclusions on PTSD in emergency service workers

Rates of PTSD in ambulance service workers are generally high and research indicates that they vary between 20 and 21 percent (Grevin, 1996; Clohessy and Ehlers, 1999). The rate of PTSD is estimated to be around 17 and 18 percent among fire-fighters (Wagner et al, 1998; Perkonigg et al, 2000). This difference is probably a result of the fact that ambulance service workers respond to more emergency calls than the police and fire service combined (James and Wright, 1991) and, therefore, may suffer from greater psychological distress than these other groups (Marmar et al, 1996). However, the rate of PTSD is clearly elevated in these populations in comparison to prevalence rates of between one and two percent in community based samples (Perkonigg et al 2000). Persons with persistent PTSD symptoms often suffer from poor job satisfaction, absenteeism from work, or early retirement (Hall et al, 1979). 'Subthreshold' PTSD has also been associated with levels of social and work morbidity comparable to full PTSD (Zlotnick et al, 2002).

The large fluctuation in prevalence rates of PTSD symptoms may be the result of variations in the types of research participants, sample size, and selection of PTSD measures. For example, researchers who studied mixed groups of emergency responders reported lower rates of PTSD (e.g. 6%, Andersen, Chhristensen & Petersen, 1991; Epstein, Fullerton & Ursano, 1988; 9%, Marmar, Weiss, Metzler, Ronfeldt & Foreman, 1996; 14-17%, McCammon, Durham, Allison & Williamson, 1998) than researchers who have examined rates of PTSD in large samples of firefighters only (e.g. 18.5%, Al-Naser & Everly, 1999; 37% Bryant & Harvey, 1995; 17-22%, Corneil, Beaton, Merphy et al, 1999). However, each of these studies (Al-Naser & Everly, 1999; Bryant & Harvey, 1995; Corneil, Beaton, Murphy et al, 1999) suffers from the same flaw in that PTSD symptoms were assessed with the Impact of Events Scale (IES; Horowitz, Wilner & Alvarez, 1979) which is an incomplete measure of PTSD symptoms (compared to DSM-IV criteria) that does not assess all of the Cluster C symptoms of avoidance or any of the Cluster D symptoms of hyperaroual. Furthermore, as Del-Ben et al (2006) highlight, many researchers have evaluated firefighters and other respondents with regard to a single recent traumatic event, whereas it may be the cumulative impact of multiple events over years of fire fighting that is more critical to the development of PTSD.

Neuroticism

Yehuda and McFarlane (1995) argued that there was a demonstrated role of vulnerability factors in the development of PTSD. Psychiatric factors such as a history of major depression or psychological dimensions such as neuroticism may place some individuals at greater risk for the development of PTSD following exposure to a traumatic event. Bromet et al (1998) found that several specific types of trauma, along with history of affective disorders in women and history of anxiety disorders and parental history of mental disorders in men, were significantly associated with PTSD in the National Comorbidity (NCS; Kesller et al, 1995) study data.

McFarlane (1988) found elevated neuroticism was associated with PTSD in fire-fighters exposed to a natural disaster. Furthermore, neuroticism has been associated with PTSD in other groups of trauma victims such as Vietnam veterans with combat related PTSD (Talbert et al 1993), young urban adults (Breslau et al, 1991), road traffic accident victims (Holeva & Tarrier, 2001) and in burn survivors (Fauerbach et al, 2000).

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Cox et al (2004) investigated the relative importance of neuroticism and selfcriticism in the only survey of PTSD in a nationally representative sample. Regression analyses found that elevated levels of neuroticism were significantly associated with PTSD among men and women who had experienced one or more traumatic events. After controlling for types of trauma experienced and other previously identified factors (e.g. psychosocial factors such as a non-confiding relationship with mother; Bromet et al, 1998), neuroticism remained significantly associated with PTSD in males and females. Thus, this study suggests that neuroticism is a promising candidate as a potential psychological determinant in PTSD. However, there are a number of limitations of this study. First the study is cross-sectional and because all the variables were assessed simultaneously the alternative explanation that elevated neuroticism is a consequence of severe and persistent PTSD cannot be excluded.

Dissociation

Dissociation at the time of any traumatic event may contribute to the development of PTSD as it may inhibit the cognitive processes required to integrate cognitive and emotional responses to the trauma into general memory systems in order to make them less 'traumatic' (Horowitz, 1979; Brewin & Holmes, 2003). Weiss et al (1995) demonstrated the role of dissociation in predicting post-traumatic symptoms in emergency service personnel. Marmar et al (1999) found that dissociation around the time of the trauma was strongly predictive of symptomatic response and long-term distress in emergency service personnel involved in the collapse of a freeway as a result of the Loma Prieta earthquake in California. Hodgins, Creamer and Bell (2001) found trait dissociation to be associated with PTSD

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symptoms in junior police officers. Bennet et al (2004) administered the Cognitive Appraisal Questionnaire (Bennett, Conway & Clatworthy, 2004) to their sample of 617 British Ambulance workers. This includes a measure of dissocative symptoms at the time of a work-related traumatic event. They found that the degree of dissociation at the time of the index incident was associated with frequency of encountering potentially traumatic incidents and organisational stressors and the degree of PTSD symptoms experienced as a result of both. This suggests that the more ambulance personnel experienced a variety of stressors, the more likely they were to engage in dissociation when facing a particularly threatening situation. Thus, this suggests that emergency service workers using dissociation as a coping mechanism may be at more risk from developing symptoms of PTSD.

Shift work

It has been suggested that long working hours and 24-hour shift are a major contributing factor to emergency medical service workers burnout (Fontanarosa, 1990). As shift work is a source of stress it is necessary to briefly consider the effects of shift work.

Individuals who regularly work atypical hours are at greater risk for physical and psychological impairment or disease than typical day workers (e.g. Costa, 1996; Costa et al, 2000). This risk is believed to be a result of the physical and psychological stress that develops from work schedule-related disruptions of their biological functions, sleep, and social and family life. Furthermore, this risk is increased by extended hours of work beyond the standard 40-hour week (Costa et al, 2000). Shift work has been associated with adverse health and well-being outcomes in some individuals. In fact, it is well accepted that shift work has a negative impact on health, as it interferes with physical, mental and social well-being (World Health Organisation, 1948). It has been found to perturb psychophysical homeostasis (circadian rhythms, eating and sleeping hours), decrease vigilance and performance (errors and accidents), impair family and social relationships and is also a recognised risk factor for gastrointestinal, psychoneurotic and cardiovascular disease (Costa, 1996). However, Costa (2003) highlights that tolerance to shift work is an extremely complex phenomenon. He argues that 'tolerance' goes beyond that of 'adjustment' which is mainly a biological response in terms of circadian rhythms and performance efficiency which includes physical and mental signs of decreased health and wellbeing, and, in particular, chronic fatigue, digestive, sleep and mental disorders (Andlauer & Reinberg, 1979; Harma, 1993). In an extensive review of the literature, Harrington (1978) estimated that 20-30% of workers dislike shift work and suffer from serious troubles, mainly due to circadian disruptions and severe sleep problems that cause them to quit. However, 10% do not complain at all during their working life.

Sleep and shift work

Sleep is the primary function that is most disrupted by shift work. Both the quality and quantity of shiftworker's sleep has been found to suffer (Costa, 1996). This enviably results in fatigue (Luna, French & Mitcha, 1997; Tepas & Carvalhais, 1990). Furthermore, severe sleep disturbances over time can result in the development of chronic fatigue, anxiety, nervousness and depression (Costa et al, 2000). Kaliterna et al (1995) found that after 3 years follow up on young shift workers rigidity of sleeping habits was the only variable that consistently correlated with poor health. Good sleep hygiene is one of the most important factors in counteracting stress and improving tolerance to shift work. Costa et al (2003) argue that the more a worker is able to adopt proper sleeping regimes and avoid external sleep disturbances, the more they are able to compensate for the sleep disruption as a result of working irregular hours (Akerstedt, 1996). Some studies have also shown that shift workers who usually take naps show a better tolerance to shift work (Costa et al, 1995).

Health and shift work

Gastrointestinal disorders are the most prevalent health complaint associated with shift work (e.g. Vener et al, 1989; Angersbach et al, 1980), although it appears that night work and not just shiftwork appears to be the critical factor in the development of gastrointestinal disease (Angersbach, 1980). In a review of 36 epidemiological studies covering 50 years of data and 98, 000 workers, Costa (1996) found that disorders of the digestive tract were 2-5 times more common among shift workers who experienced nightwork than among day workers and shift workers who did not work at night. Tucker et al (2000) also reported that the development of digestive problems was associated with working longer shifts (i.e. 12 hours vs 8 hours) and with relatively early changeovers (i.e. 6 am vs 7am).

In a meta-analysis of the epidemiological literature on shift work and heart disease, Boggild and Knutsson (1999) reported that shiftworkers have a 40% greater risk for cardiovascular mortality or morbidity than day workers. It has been suggested that the risk factors associated with cardiovascular disease are consistent with many of the problems associated with shiftwork, such as gastrointestinal problems, sleeping dysfunction, smoking and poor working conditions. Furthermore, shift work can also act as a stressor, increasing the stress response over time and resulting in increased blood pressure, heart rate, cholesterol and alterations in glucose and lipid metabolism (Costa, 1996).

Psychological effects of shift work

Although physical disorders in shift workers (e.g. gastrointestinal complaints) have received the most attention, the psychological and emotional distress that accompanies shift work is increasingly being recognised (e.g. Barton et al, 1993; Williamson, Gower and Clarke, 1994). Although some studies have found that the magnitude of the effects is sometimes low (Barton, 1994; Tucker, Barton and Folkard, 1996) Costa et al (1996) have suggested that psychological distress often accompanying shiftwork from its onset may be the primary factor that provokes many (varying between 20-50% in different studies) to leave shift work.

Individual differences and adaptation to shiftwork

Folkard, Monk and Lobban (1979) suggested that people have different circadian types and that this may explain why people adapt better than others to shift work.

Personality factors may also play a role. For example, Introverts have been shown to have an earlier circadian phase (i.e. they are more morning orientated) than extroverts (Vidacek et al, 1987). Circadian adjustment to shift schedules also seems to occur faster in extroverts than introverts (Colquohoun & Condon, 1980). Researchers have reported a relationship between neuroticism and shiftwork tolerance across several studies in that shift workers who are very neurotic are less tolerant to shift work (e.g. Iskra-Golec, Marek & Noworol, 1995; Costa et al, 1995). However, Kaliterna et al (1995) found a significant correlation between neuroticism and poor health after 1 year, but not after 3 years. Similar negative results have also been reported in other longitudinal studies (Wynne et al, 1986; Bohle & Tilley, 1989). This may suggests that neuroticism may increase with exposure to shift work and, hence, it acts more as an outcome or strain measure than a moderator variable (Bohle & Tilley, 1989).

Social functioning and shiftwork

Shift work has been demonstrated to impair family relationships. Shift workers report that family and social difficulties are their prominent concern rather biological factors. Individuals engaged in shift work, especially night work, are often out of phase with society and as a result they can face greater difficulties as most family and social activities are arranged during daylight hours. Shift work can also interfere with family relations. Time pressure can be a constant problem for those with a high family burden and can lead to strain in marital relationships and have adverse consequences for parenting and children's education, etc.

Working hours

Spurgeon et al's (1997) review found no clear evidence to confirm or disconfirm health and safety related problems of long working hours, however, some studies have suggested that the longer the working period (beyond 9 hours) the higher the fatigue, risk of accident and health troubles (Rosa, 1995; Colquhoun et al, 1996). Many studies have highlighted the negative effects on fatigue, performance efficiency, sleep, family and leisure times of 12 hour shifts in comparison to 8 hour

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shifts (Rosa, 1995, Fischer et al, 2000). However, other studies have not found any detrimental effects on performance, sleep (Axelsson et al, 1998), health (Smith et al, 1998a), safety (Laundry & Lees, 1991) or quality of life (Kogi et al 1989).

Therefore, this evidence suggests that shiftwork can act as a stress factor or a trigger when it is matched with conflicts such as endogenous rhythms, demanding work conditions and interference in family and social life.

The above review has shown that experiencing waking traumatic episodes has a negative effect on psychological well-being, that emergency service workers in general have a high incidence of PTSD and other psychopathologies, and have the further stress of doing shift-work. This chapter will now conclude with a review of literature on sleep and dreams in the target population of this study, and a list of the aims of the study.

Sleep and dreams in fire fighters

Haslam et al (2003) found that one third of their 31 fire service personnel reported experiencing problems sleeping after an incident. Some of the respondents reported bad dreams and nightmares. However, the exact proportion experiencing unpleasant dreams was not reported. The authors found that such dreams were related to incidents involving serious injuries; others were concerned with feelings of failure and feeling trapped. They report an extract from a 53 year-old leading fire-fighter talking about a recurrent dream after an incident. 'this lump on the road burning that turned out to be a young boy... I think what makes me feel bad about it is because I didn't even realise it was a person. There was this thing on the road, and I didn't recognise it as being a person.'

Fullerton et al (1992) reported that many fire-fighters had difficulty sleeping and that several experienced nightmares. However, no specific information was reported on prevalence. Mills and Mills (2005) looked at the symptoms of PTSD in emergency medical residents (EMR) who routinely deal with death and dying. Four groups of residents were assessed; a) those incoming to be first year EMR (MS4), b) interns finishing the first year of training (R1), C) residents finishing the second year of training (R2), d) residents graduating from the programme (R3). The authors found that 30% of residents reported symptoms associated with PTSD. Furthermore, they found that there was a positive correlation of PTSD symptoms with increased level of EMR training, with the R3 group reporting the most symptoms. Table 58 illustrates data on the percentage and number of participants experiencing sleep difficulties and dream disturbances. Table 58 – Data from Mills and Mills (2005) showing percentage (and number) of participants in 4 years of residence training who experience sleep difficulties and dream disturbances.

	Difficulty sleeping % (n/total)	Dream disturbances %
		(n/total)
MS 4	0 (0/16)	0 (0/16)
R1	21.43 (3/14)	14.29 (2/14)
R2	15.38 (2/13)	30.77 (4/13)
R3	43.75 (7/16)	25 (4/16)

Table 58 shows that sleep difficulties and dream disturbances increase with length of EMR training. However, the authors do not specifically comment on the nature or frequency of these dreams.

Although there have been no controlled systematic studies investigating sleep and dreams of emergency service personnel these preliminary data suggest that sleep disturbances may be more frequent than in the general population and that these sleep disturbances may worsen with increased exposure to traumatic work related incidents. It is also unclear as to whether the nightmares reported are idiopathic nightmares, nightmares in response to the traumatic events, or repetitive posttraumatic nightmares.

Aims of study 3:

1. To assess the prevalence of nightmares in a population exposed to frequent trauma. It is hypothesised that nightmare frequency will be elevated in this population as a result of the traumas experienced.

2. Given that the rate of PTSD is elevated, to determine if the frequency of nightmares can be predicted by PTSD scores.

3. To determine if neuroticism, depression and anxiety predict the frequency of nightmares in a population where PTSD would be the expected predictor (notwithstanding the fact that PTSD and anxiety are highly correlated).

4. To assess trait nightmare distress in this population and its correlates with well-being measures.

Chapter 11

Study 3: Nightmares in Fire-fighters

Method

Participants

57 front-line fire-fighters were recruited from three local fire stations (female = 1, male = 56; mean age = 37.64; SD = 7.48; range 22-53). Their mean number of years in the service was 10.38 (SD = 7.65; range =1-30).

Participants were reassured that all information would be treated with the utmost confidentiality and sensitivity.

Materials

Post-traumatic Diagnostic Scale (PDS: Foa, 1995).

The 17-item (part 3) PDS sub-scale used in this study, measures reexperiencing traumatic memories, attempts at avoidance, and arousal both at the time of re-experiences and more generally. The frequency of each symptom is rated on a four-point scale, ranging from 0 ('not at all or only one time') to 3 ('5 or more times a week/almost always'). The PDS is based on the DSM-IV (American Psychiatric Association, 1994) diagnostic criteria and provides both a severity score and a diagnostic categorisation for PTSD based on the pattern of sub-scores. A diagnosis of PTSD requires the existence of symptoms for a period of one or more months. Participants are asked to identify a key work-related incident that resulted in troubling memories and to report the frequency of symptoms in relation to the incident. The PDS demonstrates good positive psychometric characteristics, including high internal consistency (alpha = 0.92 for the 17 items), good test-retest reliability (k = 0.74), and good sensitivity and specificity with respect to PTSD diagnosis (0.82 and 0.77 respectively). The PDS had achieved an 86% concordance with diagnoses made with the Structured Clinical Interview for DSM-IV Disorders (Foa, 1995) and correlates strongly with other indicators of PTSD-type symptoms (Coffey, Dansky, Falsetti, Saladin & Brady, 1998). The PDS has not been normed on the general population; however, Foa (1995) reports PDS data for a group of 248 individuals, sampled from treatment and research centres that have high numbers of PTSD sufferers. It has been argued that because this instrument is criterion based (i.e. evaluates whether a client meets or does not meet diagnostic criteria for PTSD), general population norms are not required for its function. Therefore, the PDS does not yield standardised T scores, but rather defines PTSD symptom severity as 'mild', 'moderate', 'moderate to severe' or 'severe'. The cut-off scores used to determine severity were derived from a sample of 376 women with sexual or physical assault histories. However, Foa (1995) cautions that these are only rough estimates of PTSD severity, probably because the severity of assault related posttraumatic stress in her female assault victim sample may or may not compare to those of females victims of other types of trauma or of males with a trauma history of any type. However, the PDS is the only published test that yields a reliable and meaningful DSM-IV PTSD diagnosis, although it should be coupled with a formal diagnostic interview before a specific diagnosis can be made (Briere, 1997a).

One of the 17 questions on the PDS asks whether participants have 'bad dreams or nightmares about the traumatic event'. Responses to this item will be used as a separate variable in study three, and PDS scores when correlated with nightmare variables will therefore have this item omitted.

Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983).

Anxiety and depression were assessed using the 14-item Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). Participants are required to underline one of four statements that relate most specifically to them and how they have been feeling in the last week. Each statement has a rating of 0 to 4 and higher scores indicate higher levels of anxiety and/or depression. The HADS is a reliable screening instrument for clinically significant anxiety and depression. It has good reliability and validity and is widely used in clinical settings (Johnston et al, 2000). Crawford, Crombie and Taylor (2001) administered the HADS to a non-clinical sample, broadly representative of the general adult UK population and found a mean Anxiety score of 6.14 (3.76) and a mean Depression score of 3.68 (3.07). The test authors of the HADS recommended cut-off scores that are indicative of clinical levels of depression and anxiety (Snaith & Zigmond, 1994). A score of 8 or above is considered to be indicative of 'possible' clinical levels of each emotion. A score of 11 or above is considered indicative of a 'probable' diagnosis of depression or anxiety. However, while the HADS is a screening measure of anxiety and depression, it does not necessarily mean that elevated scores reflect clinical levels of depression and anxiety.

The Eysenck Personality Questionnaire-Revised Short Scale (EPQ-R Short Scale: EPO-RS, Eysenck et al, 1985)

This self-report questionnaire was described in Chapter 3, which is the method section for study 1. For this study only the neuroticism scale is used.

Dream/Sleep Questionnaire

Participants were required to make a retrospective estimate of the frequency of their dream recall and the recall of nightmares and repetitive nightmares (appendix 23). Nightmares were defined as 'a very disturbing dream that you can recall clearly'.

Participants were asked:-

1. How many dreams do you have each week?

2. If you do not have one dream per week how many dreams do you have per month?'

3. How many nightmares (if any) do you have a month?

4. If you have nightmare less than once a month, how many do you have a year?

5. Are nightmares a problem for you?

6. Do you ever have the same nightmare being repeated?

7. If so, how often do you have such a repetitive nightmare?

Participants were required to write their own responses for most of the questions above and were not constrained by any pre-determined response format. However, questions 5 and 6 were responded to on a yes/no format.

Participants were asked to rate the emotional tone of their average dream on a 7-point dream hedonic tone scale (Foulkes et al, 1966) ranging from 1 (very unpleasant) to 7 (very pleasant). The question asked:-

8. On average, how would you rate the overall mood of your dreams?

Participants were asked to provide their own responses to the following questions about their sleep on an average night on a rest day.

9. On average, what time do you turn the light out to go to sleep?10. How long does it normally take you to fall asleep?

11. What time do you normally wake up?

12. Between falling asleep and waking up, how much of the time as you usually awake during the night?

Participants were asked to rate their overall sleep quality on a 5-point likert scale ranging from 1 (very poor) to 5 (very good).

Procedure

Station officers of three local fire stations were contacted and informed about the aims and objectives of the study. Questionnaire packs containing a letter inviting participants to take part, consent forms, the questionnaire measures described above and a stamped addressed envelope were distributed to fire-fighters by their station officers. Participants were informed that the study aimed to assess sleep quality, dreams and mood. Contact details for the researchers were also provided in case of questions. If participants were happy to take part they were required to complete the questionnaires and return them in the stamped addressed envelope provided. Participants were reassured that consent forms would be stored separately from their questionnaires when received.

Design

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This is a correlational study in which regression analyses were used to determine factors predicting PDS score, nightmare frequency, and presence of repetitive nightmares and of trauma dreams/nightmares.

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Chapter 12

Study 3: Nightmares in Fire-fighters

Results

This study assessed nightmare frequency and its correlates in emergency service workers. A retrospective measure was used in this study in order to increase compliance. As studies 1 and 2 confirmed that unpleasant dream frequency, defined without the waking criterion, is the better index of psychopathology, the following definition of a nightmare was used; 'a very disturbing dream that can be clearly recalled'. Table 59 shows the mean scores and range for dream frequency variables and well-being measures in this population.

Table 59 – Table 59 shows the mean frequency of dreams and nightmares per month, mean emotional tone of the dreams, and poor well-being and sleep variables.

· · · · · · · · · · · · · · · · · · ·	Mean (standard deviation)	Range
Dreams per month	29.58 (11.44)	0-57
Nightmares per month	1.19 (2.28)	0-10
Dream emotional tone	4.23 (0.82)	3-7
Neuroticism	4.03 (3.06)	0-11
PDS: severity of symptoms	7.37 (7.73)	0-31
PDS: Total positive symptoms	5.33 (4.54)	0-17
Anxiety	5.93 (3.90)	0-19
Depression	4.23 (3.95)	0-15
Sleep quality	3.58 (1.05)	1-5
Sleep latency	19.48 (16.98)	1.5 - 90
Sleep efficiency	87.29% (11.14)	49.21 - 99.64%

Note: ¹PDS= post traumatic diagnostic scale.

Table 59 shows that this sample of fire-fighters experience on average one nightmare per month. In this study very unpleasant dreams were labelled as 1 on the hedonic scale and very pleasant dreams were labelled as 7. Participants rated the emotional tone of their average dream as 4.23, which is in the neutral range ('neither pleasant nor unpleasant'). Mean sleep quality was 3.58 suggesting that on average participants rated their sleep between 'neither good nor poor' and 'rather good'. On average participants took about 20 minutes to fall asleep on a rest day and their average sleep efficiency was fairly good (87.29%). In Eysenck and Eysenck (1991), the normative sample means for neuroticism at ages 31-40 are 5.75 (3.46) for males and 5.50 (2.92) for females. The mean of this predominantly male sample was 4.03(3.06) which is less than for the general population. Crawford, Crombie and Taylor (2001) administered the HADS to a non-clinical sample, broadly representative of the general adult UK population, and found a mean Anxiety score of 6.14 (3.76) and a mean Depression score of 3.68 (3.07). Thus, the mean anxiety score presented in table 59 is again lower than the general population, whereas the depression score for the sample is slightly above that of the general population. The mean PDS severity score of 7 is low as this scale ranges from 0-51. The total number of symptoms that could be endorsed on the PDS is 17 and so a mean score of 5.33 is also low.

Nightmare frequency

63.16% (n =36) reported no nightmares per month;
10.53% (n=6) reported one nightmare per month;
17.54% (n = 10) reported two nightmares per month; and
8.77% (n = 5) reported more than 3 per month.

Therefore, 36.84% participants reported experiencing at least one nightmare per

month

Nightmare distress

Only one participant stated that nightmares were a problem for them.

Repetitive nightmares

25.9% (n= 15) of fire-fighters report the occurrence of repetitive nightmares. Participants were asked how often they experienced repetitive nightmares, however, of the 15 reporting them, only 9 answered this question. The mean frequency of repetitive nightmares based on these 9 participants was 11.28 per year (SD = 10.01, range = 1.50-24).

Of those reporting at least one nightmare a month, 57.1% (n = 12) report the occurrence of repetitive nightmares.

Trauma nightmares

One of the questions on the PDS scale was 'having bad dreams or nightmares about the traumatic event'. Participants were required to respond 'not at all', 'once in a while', 'half the time' and 'almost always'.

68.4% (n=39) of participants responded 'not at all';
26.3% (n=15) responded 'once in a while'; and
5.3% (n=3) responded 'half the time'.

Anxiety and depression

A score of 8 or above is considered indicative of 'possible' clinical levels of depression and anxiety. 27.6% (n =16) reported 'possible' clinical levels of anxiety and 15.5% (n = 9) reported 'possible' clinical levels of depression. A score of 11 or above is considered indicative of a 'probable' diagnosis of depression or anxiety. 12.1% (n = 7) and 10.3% (n= 6) of participants reported these levels of anxiety and depression respectively.

<u>PTSD</u>

24.6% (n=14) of participants met the criteria for 'caseness' on the PDS scale (Foa, 1995). Five were considered to have PTSD with mild severity, six with moderate severity and three with moderate to severe severity.

Self-reported sleep quality

Table 59 shows that mean sleep quality was rated as average to rather good. Table 60 provides a breakdown of how participants rated their sleep.

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Table 60 – Frequency of self-reported quality of sleep categories

Sleep quality	Frequency	
Very poor	1.7% (n =1)	
Rather poor	17.5% (n=10)	
Neither good nor poor	20.7% (n = 12)	
Rather good	39.7% (n= 23)	
Very good	19.0% (n = 11)	

Inspection of the table suggests that the majority of participants reported good sleep quality but 19.2% (n = 11) reported poor sleep.

Sleep latency

55.2% (n = 32) reported an average sleep latency of 15 minutes or less;
32.8% (n = 19) reported an average sleep latency of 16 - 30 minutes;
8.6% (n = 5) reported a sleep latency of 31 - 60 minutes; and
1 participant (1.7%) reported a sleep latency of over an hour.

Sleep efficiency

Number of hours of actual sleep was calculated by subtracting the time spent awake during the night and sleep latency from time spent in bed. Sleep efficiency was calculated by dividing the number of hours slept by the number of hour spent in bed x100.

73.7% (n = 42) participants had a sleep efficiency of over 85%;
14.0% (n = 8) had a sleep efficiency of between 16 - 30 minutes,
7.0% (n = 4) had sleep efficiency between 65-75%
5.3% (n = 3) had a sleep efficiency of less than 65%.

Distribution of variables

Shapiro-Wilk tests were conducted to assess normality of distribution. The results are shown in Table 61.

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	Shapiro-Wilk statistic
Nightmare frequency	0.55 (p<0.01)
Dream emotional tone	0.70 (p<0.01)
¹ PDS: Severity of symptoms	0.80 (p<0.01)
PDS: Total positive symptoms	0.91 (p<0.01)
Neuroticism	0.92 (p<0.01)
Depression	0.90 (p<0.01)
Anxiety	0.94 (p<0.01)
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Table 61 - Shapiro-Wilk statistics for dream variables and well-being measures

Note: ¹PDS: Post traumatic diagnostic scale.

Table 61 shows that all the Shapiro-Wilk tests were significant, thus none of the variables are normal distributed and, therefore, non-parametric statistics will be used. Intercorrelations between the well-being variables are shown in Table 62.

Table 62 - Intercorrelations between measures of well-being (Spearman's rho)

	Depression	Anxiety	Neuroticism	PDS:SS	¹ PDS:TPS
Depression	-	0.67* (p<.01)	0.45* (p<.01)	0.53* (p<.01)	0.57 (p<.01)
Anxiety	0.67* (p<.01)	-	0.67* (p<.01)	0.67* (p<.01)	0.76 (p<.01)
Neurotism	0.45* (p<.01)	0.67* (p<.01)	-	0.43* (p<.01)	0.45 (p<.01)
¹ PDS: SS	0.53* (p<.01)	0.67* (p<.01)	0.43 * (p<.01)	-	0.58 (p<.01)
¹ PDS: TPS	0.57 (p<.01)	0.76 (p<.01)	0.45 (p<.01)	0.58 (p<.01)	-

Note: PDS:SS = Post traumatic diagnostic scale: severity of scores; PDS:TPS; posttraumatic

diagnostic scale: Total positive symptoms

* p<.01

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Nightmares and well-being

Kendall's tau_b correlations were conducted to assess the relationship between nightmare frequency, repetitive nightmares, traumatic nightmares and dream emotional tone with PDS, neuroticism, anxiety and depression. Relationship with age and years in job were also assessed. The number of nightmares per year variables was used in the following analysis in order to include more subjects.

Table 63 - Kendall's tau_b correlation coefficients (p values) between dream variables, age, number of years in the fire service and measures of well-being.

<u> </u>	Age	Years in	PDS:SS ¹	PDS:TPS ¹	Neuroticism	Anxiety	Depression
		post					
Nightmare	-0.11	-0.00	0.21	0.29*	0.26*	0.20	0.18
frequency	(p=.26)	(p=.99)	(p=.03)	(p<0.01)	(p=.01)	(p=.04)	(p=.08)
Repetitive	-0.88	-0.10	0.26	0.28*	0.30*	0.11	0.06
nightmares	(p=.05)	(p=.34)	(p=.02)	(p=.01)	(p=.01)	(p=.32)	(p=.60)
Trauma	-0.03	0.04	0.34*	0.32*	0.26	0.26	0.22
nightmares	(p=.77)	(p=.71)	(p=.00)	(p<0.01)	(p=.02)	(p=.02)	(p=.06)
Emotional tone	-0.07	-0.01	-0.08	-0.15	-0.18	-0.15	-0.09
	(p=.54)	(p=.94)	(p=.46)	(p=.17)	(p=.11)	(p=.17)	(p=.40)

Note PDS:SS = Post traumatic diagnostic scale: severity of scores; PDS:TPS: Posttraumatic diagnostic scale: Total

positive symptoms.

¹ scored here with nightmare question removed

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Table 63 shows that age and number of years in the service is not significantly related to any of the unpleasant dream variables or the dream emotional tone.

It can be observed that nightmare frequency and the presence/absence of repetitive nightmares is significantly positively correlated with the number of total positive symptoms on the PDS (with the nightmare question on this scale having been omitted) and neuroticism but not with anxiety and depression. The presence or absence of trauma dreams was significantly related to the number of total positive symptoms and the measure of symptom severity on the post traumatic diagnostic scale, however, anxiety and neuroticism just missed statistical significance (p=0.02). Mean dream emotional tone was not significantly related to any of the well-being measures. Thus, it seems that it is the frequency of these extreme dream types rather than mean dream emotional tone that has the strongest relationships with measures of well-being.

Sleep quality

Kendall's tau_b correlations were conducted to determine the relationship between sleep quality and well-being and dream variables. These are shown in Table 64.

Table 64 – Kendall's tau_b correlations between sleep quality; sleep efficiency, sleep

latency and well-being and dream variables.

Sleep quality	Sleep latency	Sleep efficiency
0.00 (0.96)	-0.15 (0.11)	0.02 (0.86)
-0.09 (0.38)	-0.14 (0.15)	-0.03 (0.74)
-0.08 (0.50)	0.03 (0.79)	-0.03 (0.71)
-0.18 (0.08)	-0.00 (0.98)	-0.10 (0.31)
-0.13 (0.21)	0.02 (0.84)	-0.09 (0.34)
-0.21 (0.04) ¹	-0.04 (0.69)1	-0.02 (0.86) ¹
-0.26* (0.01)1	-0.02 (0.83) ¹	-0.07 (0.48) ¹
-0.10 (0.33)	-0.04 (0.70)	-0.13 (0.19)
-0.06 (0.63)	-0.16 (0.16)	0.02 (0.86)
-0.23 (0.06)	0.10 (0.40)	-0.25 (0.02)
	$\begin{array}{c} 0.00 \ (0.96) \\ \hline 0.09 \ (0.38) \\ \hline -0.08 \ (0.50) \\ \hline -0.18 \ (0.08) \\ \hline -0.13 \ (0.21) \\ \hline -0.21 \ (0.04)^{1} \\ \hline -0.26^{*} \ (0.01)^{1} \\ \hline -0.10 \ (0.33) \\ \hline -0.06 \ (0.63) \end{array}$	$0.00 (0.96)$ $-0.15 (0.11)$ $-0.09 (0.38)$ $-0.14 (0.15)$ $-0.08 (0.50)$ $0.03 (0.79)$ $-0.18 (0.08)$ $-0.00 (0.98)$ $-0.13 (0.21)$ $0.02 (0.84)$ $-0.21 (0.04)^1$ $-0.04 (0.69)^1$ $-0.26^* (0.01)^1$ $-0.02 (0.83)^1$ $-0.10 (0.33)$ $-0.16 (0.16)$

Note: PDS = Post traumatic diagnostic scale.

¹scored here with sleep quality question removed

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Table 64 shows that sleep quality is significantly negatively related to the number of total positive symptoms on the PDS (with the sleep quality item omitted), suggesting that those reporting more symptoms of posttraumatic stress rate their sleep quality as worse. The inverse relationship between sleep efficiency and the presence of traumatic nightmares very nearly reaches statistical significance (p=0.02).

Factors predicting repetitive nightmares

The above tables show that the presence of repetitive nightmares is related to the total number of positive symptoms on the posttraumatic diagnostic scale and neuroticism.. As these variables are non-normally distributed and repetitive nightmares is a dichotomous variable a logistical regression was conducted. All factors that correlated with repetitive nightmares were entered as predictors. The symptom severity score on the posttraumatic diagnostic scale was also entered as a predictor (as it was very nearly significantly related to repetitive nightmares (p = 0.02)). Anxiety was also entered as a predictor as it had a correlation of 0.11 and is correlated with other nightmare frequency variables.

Pallant (2005) advises that multicollinearity can be a problem when using logistical regression and that this exists when the independent variables are highly correlated (r = 0.9 and above). Table 62 shows that the highest correlation between predictor variables was r = 0.76 (between anxiety and total number of positive symptoms on the PDS scale).

The Omnibus tests of model coefficients or 'goodness of fit' was significant (x^2 (4)= 17.49; p < 0.01) suggesting that this model is a better predictor than the statistical estimation using no predictors (73.7%). Cox and Snell and Nagelkerke R square tests suggests that between 26% and 39% of the variance in the presence or absence of repetitive nightmares are explained by the predictors. The model correctly classifies 75% of overall cases into either having or not having repetitive nightmares. The model had a negative predictive value of 90.5% and a positive predictive value of 55.5%. This means that the model was able to correctly identify 90% of the participants that did not have repetitive nightmares and 55% of those who did have repetitive nightmares. Table 65 below demonstrates that number of total positive symptoms on the PDS, neuroticism and anxiety all contribute significantly to the predictive ability of the model.

Table 65 - Wald test statistic and probability for the prediction of the presence of repetitive nightmares

Predictors	Wald test
PDS: Symptom severity	0.82 (0.97)
PDS: Total Positive Symptoms	4.19* (0.04)
Anxiety	5.81* (0.02)
Neuroticism	4.25* (0.04)

Note: PDS = Post traumatic diagnostic scale

* p = < .05.

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Factors predicting traumatic nightmares

Tables 63 and 64 show that both PDS variables are significantly correlated with the presence or absence of traumatic nightmares. Anxiety, neuroticism and sleep efficiency were related to presence/absence of traumatic nightmares although these just missed significance (p = 0.02). Therefore, these factors were entered as predictors.

Omnibus tests of model coefficients or 'goodness of fit' was significant (x^2 (4)= 14.45 p = 0.01) suggesting that this model is a better predictor than the statistical estimation using no predictors (73.7%). Cox and Snell and Nagelkerke R square tests suggests that between 22% and 33% of the variance in the presence or absence of traumatic nightmares are explained by these predictors. The model correctly classifies 80.7% of overall cases into either having or not having trauma nightmares. The model had a negative predictive value of 95% and a positive predictive value of 75%. This means that the model was able to correctly identify 95% of the participants that did not have trauma nightmares and 75% of those who did have trauma nightmares. The table below demonstrates that sleep efficiency is the only variable that significantly independently contributes to the predictive ability of the model.

Table 66 - Wald test statistic and probability for predictors of the presence of traumatic nightmares/bad dreams

Predictors	Wald test	
PDS: Symptom severity	0.10 (0.75)	
PDS: Total Positive Symptoms	0.13 (0.72)	
Anxiety	0.18 (0.67)	
Neuroticism	0.20 (0.66)	
Sleep efficiency	4.84* (0.03)	

Note: PDS = Post traumatic diagnostic scale

* p<0.05

A logistical regression could not be conducted on the frequency of nightmares as reducing this variable to a dichotomous one resulted in all correlations (as shown in tables 63 and 64) becoming insignificant. Therefore, partial correlations were conducted between nightmare frequency and measures of well-being, controlling for the effects of age, sleep quality, sleep latency, sleep efficiency and mean emotional tone of dreams. These are shown in Table 67 below. Table 67 - Kendall's tau-b partial correlation coefficients between number of nightmares per year with well-being before and after age, sleep quality, sleep efficiency, sleep latency and emotional tone are partialled out

Detween toth method	Neuroticism	PDS: SS	PDS:TPS	Depression	Anxiety
Nightmare frequency	0.26*	0.21	0.29*	0.18	0.20
	(<0.01)	(<0.05)	(p<0.01)	(>0.05)	(<0.05)
Age partialled out	0.26*	0.22	0.30*	0.17	0.21
	(<0.01)	(<0.05)	(<0.01)	(>0.05)	(<0.05)
Sleep efficiency partialled out	0.26*	0.21	0.28	0.17	0.19
	(<0.01)	(<0.05)	(<0.01)	(>0.05)	(<0.05)
Emotional tone partialled out	0.23*	0.20	0.27*	0.16	0.17
	(<0.01)	(<0.05)	(<0.01)	(>0.05)	(>0.05)
Anxiety partialled out	0.19	0.11	0.52*	-	0.09
	(<0.05)	(>0.05)	(<0.01)		(>0.05)
Depression partialled out	0.12	0.06	0.41*	0.03	-
	(>0.05)	(>0.05)	(<0.01)	(>0.05)	
PDS:SS partialled out	0.20	st-delts a b	0.21	0.09	0.09
	(<0.05)		(<0.05)	(>0.05)	(>0.05)
PDS:TPS partialled out	0.16	-0.04		0.06	0.03
	(>0.05)	(>0.05)		(>0.05)	(>0.05)
Neuroticism partialled out	-	0.12	0.20	0.08	0.10
		(>0.05)	(<0.05)	(>0.05)	(>0.05)

Note: PDS:SS = Post traumatic diagnostic scale: symptom severity; PSD: TPS = Post traumatic diagnostic scale: Total positive symptoms.

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Table 67 shows that nightmare frequency has moderately sized significant correlations with neuroticism and total positive symptoms on the PDS. The correlation between nightmare frequency and neuroticism became insignificant when controlling for anxiety, depression and PDS measures. However, the correlation between total number of positive symptoms on the PDS and nightmare frequency remains when anxiety and depression are partialled out. Thus, the table suggests that total positive symptoms on the PDS seems to be the main correlate of nightmare frequency.

Factors predicting PTSD 'caseness'

A regression analysis was also conducted to determine the extent to which nightmare frequency per year, neuroticism, anxiety, depression and sleep quality predicted caseness on the PDS scale.

The Omnibus tests of model coefficients or 'goodness of fit' was significant (x^2 (4)= 27.51; p <0.001) suggesting that this model is a better predictor than the statistical estimation using no predictors (75.4%). Cox and Snell and Nagelkerke R square tests suggests that between 38% and 57% of the variance in PTSD are explained by these predictors. The model correctly classifies 93% of overall cases into either having or not having PTSD. The model had a negative predictive value of 100% and a positive predictive value of 71%. This means that the model was able to correctly identify 100% of the participants that did not have PTSD and 71.4% of those who did have PTSD. The table below demonstrates that anxiety is the only variable that significantly independently contributes to the predictive ability of the model. However, the Wald test for nightmare frequency as a predictor is relatively high.

Table 68 -	Wald test	t statistic and	l probabilit	y of predict	tors for the	presence of PTSD
				/ - r		1

Predictors	Wald test	
Nightmare frequency	2.39 (0.12)	
Anxiety	5.54* (0.02)	
Depression	0.05 (0.82)	
Neuroticism	1.66 (0.20)	
Sleep quality	0.21 (0.64)	·

^{*} p<0.05

Regressions were also conducted where nightmare frequency was replaced as a

predictor by repetitive nightmares and secondly by trauma nightmares, however,

number of nightmares per year was able to account for more of the variance in PTSD

'caseness'.

Chapter 13

Study 3: Nightmares in Fire-fighters Discussion

Rescue workers are exposed to both the stress of events and the stress of their role as help provider (Raphael, 1986). Due to the high level of exposure to traumatic incidents, it has been demonstrated that a range of professionals, including police, ambulance and fire personnel, can become secondary victims of trauma, exhibiting symptoms akin to direct victims (Genest, Levine, Ramsden & Swanson, 1990; Raphael & Meldrum, 1993). This study, therefore, aimed to assess the frequency and distress of nightmares and their correlates in a non-treatment seeking sample of firefighters.

Depression and anxiety

In comparison to the means in the non-clinical adult sample of Crawford, Crombie and Taylor (2001), the mean anxiety and depression of the sample of firefighters is not elevated compared to the general population. These results are in accordance with Mock et al (1999), who concluded that although the working environment of emergence medical service providers contains many stressors, this population is not on average more anxious than the general working public. However, 12.1% of the sample had 'probable' clinical levels of anxiety and 10.3% had 'probable' clinical levels of depression based on the HADS scores. Bennett et al (2004) found that 10% of their 617 sample of British Ambulance workers reported 'probable' levels of clinical depression and 22% reported probable levels of anxiety based on the HADS scores. Hence, both studies report similar prevalence rates for depression in emergency service workers, however, Bennett et al (2004) report a higher prevalence rate for anxiety. This may be because ambulance workers respond to more emergency calls than the police and fire service combined (James & Wright, 1991), and therefore may suffer from greater psychological distress than these other groups (Marmar et al, 1996).

Post traumatic stress disorder: Prevalence

This study found that 24.6% (n=14) of the sample met the criteria for PTSD. Five participants were classified as having PTSD of mild severity; six had PTSD of moderate severity, and three of moderate to severe severity. General population studies demonstrate lifetime prevalence rates of at least 8 to 9% for PTSD (Breslau, Davis, Andreski & Peterson, 1992; Kessler, Sonnege, Bromet, Hughes & Nelon, 1995). Therefore, this study confirms the elevated levels of PTSD in this population.

These findings are in line with Wagner et al (1998), who investigated the prevalence of PTSD among 402 professional fire-fighters using the PSD symptom scale (Foa et al, 1993). They found a prevalence rate of PTSD symptoms of 18.2%.

McFarlane (1989) found prevalence rates of 32%, 27% and 30% in a sample of fire-fighters 4, 11 and 29 months after an Australian bush fire. These prevalence rates are higher than found in this study and also in Wagner et al (1998). This may be due to the fact that In the McFarlane (1989) study many of the fire-fighters were personally affected by the disaster in that they were victims, not only helpers. Hence, it can be argued that this study does not reflect prevalence rates of PTSD arising just from helping other people in an emergency.

PTSD and years in the service

The current study found no association between PTSD measures and number of years in the service. These findings are in line with Beaton, Murphy, Johnson, Pike and Corneil (1999) who found, in a sample of 220 fire-fighters, no relationship between number of years in the service, nor past half a years exposure to traumatic incidents, and self-reported post traumatic stress symptomatology. These findings contradict those of Hytten and Hasle (1989) who reported that 'seasoned' volunteer and professional fire-fighters with more years experience were evidently 'better able to cope' with a multicasualty, multifatality apartment fire, based on self-reported post trauma symptomatology. The latter investigators argued that years of service, training and exposure to previous traumatic incidents made it easier for these 'seasoned' volunteer fire-fighters to cope with this apartment fire. However, other authors have reported a positive relationship between number of years in the service and rates of PTSD (Corneil, 1995). Moran and Britton (1994) also reported that length of emergency service as a volunteer was positively related with both the chronicity of their adverse emotional reaction to their recollected 'worst' (duty-related) traumatic incident. However, both the aforementioned studies consisted partially and entirely of volunteers who probably possessed neither the degree of preparatory training nor the frequency of traumatic incidences as professional fire-fighters. Study three and the study of Beaton et al (1999) only used professional fire-fighters.

PTSD and well-being

A logistical regression analysis using PTSD as the dependent variable (i.e. those identified as 'cases' on the PDS; Foa, 1995) found that anxiety, depression,

neuroticism, sleep quality and nightmare frequency accounted for 38% and 57% of the variance in PTSD. However, the regression found that anxiety was the only variable that significantly independently contributed to the predictive ability of the model.

The finding that neuroticism did not significantly contribute to PTSD 'caseness' is not in line with the notion that this factor may place some individuals at greater risk for the development of PTSD following exposure to a traumatic event (Yehuda & McFarlane 1995). For example, McFarlane (1988) found elevated neuroticism was associated with PTSD in fire-fighters exposed to a natural disaster. Furthermore, neuroticism has been associated with PTSD in other groups of trauma victims such as Vietnam veterans with combat related PTSD (Talbert et al 1993), young urban adults (Breslau et al 1991), road traffic accident victims (Holeva & Tarrier, 2001) and in burn survivors (Fauerbach et al, 2000). In a nationally representative sample Cox et al (2004) also found that elevated levels of neuroticism were significantly associated with PTSD among men and women who had experienced one or more traumatic events. It is not clear why neuroticism was not related to PTSD 'caseness' in this sample. However, it may be that reducing PTSD scores to a dichotomous variable causes a loss of data.

Nightmare prevalence

It was hypothesised that nightmare prevalence would be elevated in this population compared to estimates from the general population. Participants reported a mean of 1.19 nightmares a month, and 39.7% participants report experiencing at least one nightmare per month. In contrast, the American Sleep Disorders Association (1990) estimated that 5% of the adult population experience at least one nightmare per month, and Hublin et al (1999) found that 3% had nightmares weekly, whereas 10% had nightmares monthly. Thus, the prevalence of nightmares in this population is in excess of general population estimates, thus supporting the hypothesis.

The current study demonstrated that 25.9% of fire-fighters report the occurrence of repetitive nightmares. Of those reporting at least one nightmare a month 57.1% reported the occurrence of repetitive nightmares. These findings are higher than in Feldman and Hersen (1967). They found for participants who seldom report nightmares the incidence of recurring nightmares was 3%, which increases to 37% in a group of participants reporting that they sometimes-experienced nightmares. This heightened prevalence of repetitive nightmares may reflect the elevated PTSD in the sample.

The question concerning nightmares on the PDS questionnaire was analysed separately. The question asked whether participants were' having bad dreams or nightmares about the traumatic event'. 26.3% of participants reported that they were.

Nightmares and well-being

This study assessed correlations between the frequency of nightmares, repetitive nightmares and trauma nightmares. All types of nightmares were significantly positively related to the number of positive symptoms on the PDS scale and trauma nightmares were also significantly related to the severity of post traumatic symptoms. Both nightmares and repetitive nightmares had significant positive correlations with neuroticism. Anxiety and sleep efficiency were correlated with traumatic nightmares although theses both just missed statistical significance (p =0.02).

Nightmare frequency had moderately sized significant correlations with neuroticism and total positive symptoms on the PDS. PDS total positive symptoms was the largest correlate and all other correlates became insignificant when it was partialled out.

A regression analysis in which trauma nightmares was the dependent variable found that anxiety, positive symptoms of PDS scale, PDS severity, neuroticism and sleep efficiency explained 22-33% of the variance. However, sleep efficiency was the only variable that significantly independently contributes to the predictive ability of the model.

A regression analysis with repetitive nightmares as the dependent variable found that anxiety, positive symptoms of PDS scale; PDS severity and neuroticism explained 26-39% of the variance. However, in contrast to traumatic nightmares, anxiety, neuroticism and the number of positive symptoms on the PDS scale all contribute significantly to the predictive ability of the model.

Low sleep efficiency thus predicts trauma nightmares more than it predicts repetitive nightmares. This may be because these trauma nightmares, which are characteristic of PTSD, have been found to occur earlier in the night than idiopathic nightmares and are associated with gross body movements (van der Kolk et al, 1984; Hartmann, 1996) and nocturnal awakenings (Germain & Nielsen, 2003). Thus, it could be that the repetitive nightmares found in this sample are more like the idiopathic nightmares assessed in study one.

Nightmare distress

One of the aims of this study was to assess the prevalence of nightmare distress in this non-treatment seeking group, and the correlates of nightmare distress. Nightmare distress was assessed by one question asking participants if nightmares were a problem for them (question taken from Wood & Bootzin, 1990). However, and surprisingly, only one participant (1.75% of the sample) reported that nightmares were a concern for them. This is in contrast to Wood and Bootzin (1990), where, in a sample of undergraduates, 19.5% reported having a current problem with nightmares. Klink and Quan (1987) asking the same question found that 7.1% of the general population reported such a problem. There may be several explanations for the low incidence of nightmare distress in this population.

The low nightmare distress could be due to the sample being almost entirely male. However, Wood and Bootzin (1990) found that 15.7% of men reported a current problem with nightmares. It could also be suggested that fire-fighters are less likely to admit to being distressed by their nightmares due to the image of the job and their role as 'rescuer'. In fact the minimisation of reporting symptoms has been noted repeatedly in studies of emergency service personnel (Pole et al, 2001; Wagner, Heinrichs & Ehlert, 1998). However, this explanation is doubtful in that the firefighters did report that they have nightmares and also reported symptoms of distress on well-being measures.

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The low prevalence of nightmare distress may be due to the sample having low scores on neuroticism and anxiety, scores that are in fact lower than for the general population. It may also be that emergency workers are a self-selected resilient occupational group and may not be representative of the general population in terms of their personalities nor their coping styles (Mitchell & Bray, 1990). For example, some studies have found that experienced emergency service workers have better coping skills in term of dealing with trauma (Moran & Britton, 1994; Corneil, 1995). This hypothesis is in line with Picchioni et al (2002), who found that when daily stressors, life stressors, social support and coping where used to predict nightmare frequency in a multiple regression, coping did not contribute any unique variance to the overall model. However, when nightmare distress was used as the criterion variable, coping was found to contribute a significant amount of unique variance to the overall model. Hence, it may be that a population with more refined coping skills for dealing with trauma are less distressed by nightmares, even though their experience of traumas is causing more nightmares to occur. It may also be that firefighters experience a good degree of social support from their colleagues, in fact many studies have commented on the strong degree of camaraderie between colleagues, and this may reduce how distressed they become to aversive stimuli (including nightmares). Similarly, Picchioni et al (2002) suggested that a high amount of social support could produce a reduction in nightmare distress.

Personality variables may also explain the low prevalence of nightmare distress. For example, there is evidence that emergency-service and military personnel often function with self-images characterised by high levels of perceived competence and self-efficacy (Solomon, 1989). Furthermore, Short (1979) suggests that helpers are perceived and may perceive themselves as 'strong and resourceful'. This is in contrast to the 'vulnerable, open and sensitive' frequent nightmare sufferers described by Hartmann (1984). It may also be, as concluded by Hartmann (1996), that patients with repetitive posttraumatic nightmares have different personalities characteristics from those who develop ordinary nightmares. He states that the patients in his study with PTSD 'definitely did not have the very thin boundaries found in those with life long nightmares' (Hartmann, 1996, p 112). It may thus be thick-boundariness that results in the very low level of nightmare distress.

This finding of low nightmare distress in a population with an elevated frequency of nightmares is intriguing. Krakow et al (2002) studied nightmare frequency and nightmare distress in sexual assault survivors with PTSD, and found a high association between nightmare distress and nightmare frequency. However, unlike the current study these participants were recruited specifically to treat their complaint of bad dreams, and Krakow et al suggest that their correlation between nightmare frequency and distress may not be generalisable to other types of chronic nightmare sufferers. Thus, it may be that correlations between nightmare distress and frequency differ according to the population studied.

Summary of novel findings

This study has found that PTSD score is related to nightmare frequency in a sample of emergency workers who have experienced various traumatic scenes over a long period of time. Possibly due to their self-selection as a group, and their social support, they do not report distress about their nightmares, which are more frequent than in the general population. Sleep efficiency was related to the incidence of trauma nightmares.

Limitations of this study

1. This study relied on the use of retrospective reports in order to increase compliance. There may be a problem with this in that retrospective reports have been found to underestimate the frequency of nightmares (as reviewed in the Introduction, chapter 1). However, to mitigate this problem it can firstly be noted that Schreuder et al (1998) investigated PTSD patients and reported a strong correlation between retrospective nightmare frequency and serial reports of nightmares collected for 28 days, and, secondly, that in a design using non-parametric correlations a linear reduction of scores does not affect the inferential results.

2. It would have been interesting to obtain some brief measure of dream content in order to fully appreciate the nature of the nightmare, as was done by Wood et al (1992) for the San Francisco earthquake.

3. The frequency of traumatic dreams rather than just their occurrence would have been a more sensitive measure.

Study 4 builds on this study by having a population that is also high in PTSD but that is far less self-selecting, in that they have had traumatic brain injury. It also reprises a theme from study 2, in that complete cessation of dreaming is investigated as an interfering factor in the production of nightmares.

Chapter 14

Study 4: Nightmares and traumatic brain injury Introduction

Study 3 involved the relationships between numerous traumas, of various intensities, sleep and dreams/nightmares. Study 4 involves the relationship of a single trauma, head injury, to sleep and dreams/nightmares. As in study 3 there are mediating variables between the trauma, sleep and dreams, such as anxiety and depression, as well as the memory of the trauma. There are novel factors for this study, in that there can have been amnesia from the trauma, and in that the brain injury might affect dream production.

Head and brain injury

Head injury can cause damage to the brain because of contusion, brain laceration, intracranial haematoma, or contrecoup injury. Diffuse closed head injury often results in the shearing of nerve fibres, often in the frontal and temporal lobes (Ommaya and Gennarelli, 1974). Secondary damage can also occur because of intracranial hypertension, hypoxia, anaemia, metabolic anomalies, hydrocephalus, and subarachnoid haemorrhage (Jennett and Teasdale, 1981).

Mild traumatic brain injury (TBI) involves a loss of consciousness for up to 30 minutes and a posttraumatic amnesia (PTA) of less than 24 hours (American Congress of Rehabilitation Medicine, 1993) and /or a Glasgow Coma Score (GCS) of 13-15 on

admission to hospital (Russell & Smith, 1961). Severe TBI involves a period of coma of at least 6 hours and/or a period of PTA of at least one day (McMillian and Greenwood, 2003).

Prevalence of TBI

Jennett and MacMillian (1981) have estimated that the annual number of hospital admissions in Britain involving head injury is between 250 and 300 per 100,000 of the population. It is suggested that, of these, 75% are classified as mild head injury (MHI; Kraus & Nourjah, 1988), whereas only 8% are classified as severe (Artiola et al. 1980). Jennett and Frankowski (1990) have estimated that in industrialised countries the main cause of all head injuries are road traffic accidents (RTA; approx 45%), falls (approx 30%), occupational accidents (approx 10%), recreational accidents (10%) and assaults (approx 5%).

In the United States, more than 80,000 individuals sustain permanent disability from TBI each year. TBI affects people of all ages and is the leading cause of longterm disability among young adults. The impact of TBI can be widespread, extending from cognitive, behavioural, and physical impairments to functional domains such as work, interpersonal relationships and leisure activities (Mailhan et al 2005).

Emotionality

Survivors of TBI are at risk of a range of neuropsychiatric and behavioural disorders; emotional disturbances such as anxiety and depression are particularly common. Following a TBI an individual is required to adjust to the new situation and

to come to terms with losses and limitations. They may experience a sense of being out of control and feel insecure about the future.

Depression

Depression has been reported in 10% to 75% of TBI patients. A number of recent studies have reported rates between 30% and 60% (Busch, 1998; Hibbard et al 1998; Satz et al 1998; Bowen et al, 1999; Douglas, 2000; Curran, 2000). Fleminger et al (2003) state that the prevalence of depression after TBI is in the order of 20-40% in the first year, and that about 50% of people suffer from depression at some point after their injury. Jorge et al (1993b) examined 66 consecutively admitted TBI patients to a trauma centre. These patients underwent psychiatric interview one month after injury and were reassessed at 3, 6 and 12 months. At one month 26% were diagnosed with major depression using the Hamilton Rating scales, and 3% were diagnosed with minor depression. It was found that at 3, 6 and 12 months the rate of depression remained relatively constant, being 22.2%, 23.2% and 18.6% respectively. Of those not diagnosed as depressed at 1 month, 10% developed depression at 3 months. New cases were further diagnosed at the rate of 15% at 6 months, and 12% at 12 months. Similar findings have been reported by Kersel et al (2001) and Bowen et al (1999). Thus, an important conclusion to be drawn from such studies is that depression is no more common in the acute phase than in the latter stages of recovery during the first vear after TBI.

However, a higher prevalence of depression has been reported in studies that recruited TBI patients at varying times post injury. Kreutzer et al (2001) examined 722 patients with mild to severe brain injuries. Average time post injury was 2.5 years, with a range of 3 months to 9 years. The authors found that 42% met the DSM- 1V criteria for a major depressive episode. These findings are in line with Hibbard et al (1998) and Hoofien et al (2001). The former studies diagnosed 48% with major depression post-TBI. The sample consisted of 100 TBI patients of varying levels of severity averaging 7.6 years post injury. Hoofien et al (2001) found that 45% of their 76 patients with severe TBI, and on average 14.1 years post-injury, were suffering from depression, as indicated by an elevated score on the Symptom Checklist 90-Revised. Generally such studies show that the prevalence of depression with TBI patients increases with time after the first year post injury.

Irritability and anger

Severe (Brooks et al. 1987) and mild (Haboubi et al. 2001; Debs et al. 1999) brain injury frequently lead to problems of anger and irritability. This may be the result of organic damage, abnormal electrical discharge or an emotional response to disabilities or indeed a learnt behavioural pattern for expressing needs (Alderman, 2003).

Quality of life (QoL)

There is substantial evidence that severe and moderate TBI (and often even mild TBI) affects several statuses that are central to most people's definition of quality of life. In a review article Dijkers (2004) stated that reduced QoL after TBI was common. In a fairly representative brain injured sample, Smith, Magil-Evans and Britnell (1998) reported QoL measures that were comparable to ratings of a different study consisting of spinal cord injured patients (Fuhrer et al, 1992); both had lower overall ratings than a study with non-injured adults (Willer et al. 1994). Interestingly, Talbot and Giroux (2000) found that patients with minor head injuries tend to under-estimate their perceived QoL when compared to close relative's ratings, whereas more severely injured people overestimate their rating. This finding may relate to the difference in levels of awareness often evident in the two groups, in that less severely injured people have a more accurate level of insight into their difficulties.

Post Traumatic Stress Disorder (PTSD)

The psychological impact of the actual trauma, as opposed to the brain injury, has received relatively little empirical attention. It is well accepted in the general literature that PTSD symptoms are most likely following the experience of intense fear and helplessness during a traumatic event, particularly where there is a threat of death or serious injury to self or others (Diagnostic Statistical Manual IV, American Psychiatric Association, 1994). Some have argued that PTSD cannot develop after TBI because the coma and organic amnesia associated with TBI prevent the experience of the trauma and as a result anxiety based on re-experience cannot develop (Bontke et al, 1996; Sbordone, 1992). However, recently there has been mounting evidence that PTSD can occur in both minor and severe head injury, although their mechanisms may differ (Bryant, 2001; McMillian, 1996).

PTSD and Mild TBI

Sbordone and Liter (1995) found no evidence of PTSD in a post-concussional symptom group. However, this study can be criticised on the grounds that it was retrospective and non-blind. Furthermore, no standardised measures of PTSD were used and there were a large proportion of males in their sample. Mayou et al. (1993) studied 188 RTA survivors and reported that none of the sample that displayed PTSD had sustained loss of consciousness. However, the generalisability of this study can be questioned because individuals who reported loss of consciousness for over 15 minutes were excluded. Moreover, the study did not specify how TBI was operationalised, and reliance on medical notes may have resulted in a loose definition of TBI.

Several controlled studies, though, have demonstrated that PTSD can occur following mild TBI despite a loss of consciousness during the event (e.g., Black and Bryant, 2000; Bryant and Harvey, 1995, 1999; Harvey and Bryant, 1998). Reports of PTSD have also been demonstrated in more uncontrolled studies. For example, Grigsby and Kaye (1993) reported PTSD in one third of patients who had whiplash injury or an undefined range of severity of TBI. Hickling et al (1998) found that 36% of 107 RTA survivors with mild TBI developed PTSD; 16 of their sample had lost consciousness and 9 of these had PTSD symptoms. Various single case reports of PTSD following minor TBI have also been reported (Horton, 1993; Layton et al. 1995; McGrath, 1997; Silver et al. 1997).

PTSD and severe TBI

The possibility of PTSD developing following a severe TBI is particularly controversial because the injury involves an extended period of PTA. In many cases these individuals suffer very significant periods of retrograde and anterograde amnesia, such that they do not recall any episodes of the traumatic experience.

To date there have been no controlled studies assessing PTSD in individuals with severe brain injury although there has been a number of uncontrolled studies suggesting that it can occur (e.g., Hibbard et al, 1998; McMillian, 1996; Ohry et al, 1996; Williams et al, 2002). However, Warden et al (1997) report 47 military personnel with severe TBI, none of whom had PTSD according to DSM-III-R criteria, although 14% had avoidance and arousal symptoms. A number of single case studies of PTSD following severe TBI (with significant periods of amnesia) have been reported (Bryant, 1996; King, 1997; Layton et al, 1995; McNeil and Greenwood, 1996; Silver et al. 1997; Williams et al 2002). Many of these case studies reported intrusive imagery or flashbacks in which the content was thematically related to the trauma sustained.

Bryant et al (2000a) reported a 6-month follow up of 96 patients. They found that 27% fulfilled the diagnostic criteria for PTSD using an interview schedule based on DSM-III criteria, but intrusive memories were reported in only 19.2% of TBI patients with PTSD. This contrasts with the rate of intrusive memories in PTSD after assault (93%) (Fleiss et al. 1981), terrorist activity (85%) (Loughrey et al 1993) and RTAs (65%) (Blanchard et al. 1994). This suggests that the profile of PTSD in TBI is distinct from other PTSD populations. They found that the symptoms with the highest positive predictive power of PTSD in TBI patients were intrusive memories, nightmares and emotional reactivity. Again these findings contrast with those of previous reports in which the symptoms of trauma reexperiencing had only moderate positive predictive power (Foa et al, 1995).

Thus, it seems that PTSD can develop after minor or severe TBI, despite loss of consciousness and post traumatic amnesia.

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Diagnostic problems

There are a number of problems in diagnosing PTSD after TBI. There is considerable overlap between ASD/PTSD and TBI in terms of dissociative (Alexander, 1995; Grisby, 1986; Sivec and Lynn, 1995), reexperiencing, arousal and neurological (Bryant, 2001) and post-concussive (Bohnen and Jolles, 1992) symptoms. The problem in differentiating these TBI symptoms and PTSD are exacerbated by the absence of reliable means to differentiate between organically and emotionally mediated symptoms. A further problem in diagnosing PTSD in TBI patients is the tendency of TBI patients to underestimate their symptoms (Prigitano et al 1990). Finally, it has been demonstrated that litigation is associated with higher levels of symptom reporting following TBI (Lees-Haley and Brown, 1993).

Mechanisms underlying PTSD after TBI

There are several proposed mechanisms by which people may develop PTSD following TBI. It could be that memory for events shortly before the traumatic event may become the source of memory intrusions, and islands of memory during post-traumatic amnesia may also form a substrate for intrusive experiences (McMillian, 1996). However, Williams et al (2002) found that having a memory for the event was not necessarily associated with PTSD, whereas Gil et al (2005) found that memory of a traumatic event was a strong predictor and potential risk factor for the subsequent development of PTSD. These authors state that the relative risk for PTSD among the participants with memory of the traumatic event, although 6% of the patients without memory for the traumatic event had PTSD, suggesting that PTSD can develop in the absence of memory of the traumatic event. However, as the authors acknowledge, it

may be that false memory of the traumatic event can increase the risk for PTSD, or alternatively, that a lack of confidence in one's own memory can serve as a protective factor against PTSD.

Benefits of memory disruption

Gil et al (2005) cite findings from Lavie and Kaminer (2001) who found in a study of Holocaust survivors that decreased dream recall is correlated with better adjustment to the trauma, and hence may serve a defensive function. It has also been proposed that 'deliberate' disruption of memory of the traumatic event may prove therapeutically beneficial. The assertion was addressed in a double-blind study that examined the severity of acute PTSD symptoms among 18 subjects who were given 40 mg of propranolol 6 hours after trauma in comparison to the severity of symptoms among 23 participants who received a placebo (Pitman et al 2002). The results demonstrated that participants in the experimental group tended to exhibit lower levels of PTSD symptoms 10 days after the traumatic event. Thus, these findings suggest that lack of memory of the traumatic event may protect against the development of PTSD but also that the pharmacologically induced disruption of the consolidation of traumatic memories can be therapeutically beneficial for trauma survivors.

Implicit memory

Brewin et al (1996) described a dual representational theory of PTSD, which recognises that memories may be encoded and stored at an implicit level. There is evidence that individuals with PTSD selectively respond to subliminal threat stimuli (Harvey, Bryant and Rapee, 1996). These patterns suggest that implicit encoding of trauma related material occurs in PTSD. Brewin's theory proposes that traumatic experiences can be encoded and stored as verbally accessible memories (VAMs) or as situationally accessible memories (SAMs). The latter may be experienced as flashbacks, or somatic sensations that are reminiscent of the traumatic experience. Thus, the theory suggests that during periods of impaired consciousness associated with TBI, individuals may encode features of their traumatic experience implicitly (for a review see Schacter, Chiu and Ochsner, 1993). Therefore, even when areas of the brain that store declarative memories are disrupted, implicit memories may be accessed during exposure to situations similar to those in which the original trauma occurred. There is evidence that the main reexperiencing symptoms that occur following severe TBI are psychological distress or physiological reactivity in response to reminders of the trauma (Bryant, Marosszeky, Crooks, Baguley and Gurka, 2001), rather than declarative memories of the traumatic events (Bryant et al. 2000). Thus, this theory can account for the development of PTSD following severe TBI.

Fear Conditioning

It has been argued that the physiological arousal of PTSD reflects extreme fear conditioning and that this ongoing arousal results in associations between trauma reminders and anxiety responses (Kolb, 1987), and is mediated in limbic structures (van der Kolk, 1996). Therefore, this view allows for the possibility that people who lose consciousness as a result of a TBI may still experience fear conditioning to the traumatic event. There is much evidence that PTSD subjects display marked physiological reactivity to trauma-specific stimuli (Orr and Kaloupek, 1997)..

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Memory reconstruction after TBI

PTSD patients may feel emotional pain and feel as though they need to create a memory for the event. Thus, reported memories may be consistent with the patient's emotional state but consist of a reconstruction of events containing information given to them by others as well as their own imaginings (Bryant et al. 1998; Bryant et al 2000; McMillian, 1996). For example, Harvey and Bryant (2001) found that 40% of mild TBI patients who suffered from significant memory loss in the first months after their trauma reported that they had full recall for the event when they were re-assessed 2 years later. Bryant (1996) reported a series of case studies that illustrates the reconstructive nature of trauma memories following severe TBI. Other case reports describe severe TBI patients developing images of the event based on police reports, dreams and other secondary sources (Bryant, 1996; McMillian, 1991, 1996).

Summary of relationships between PTSD and TBI

Although it has been argued that the loss of memory may protect against the development of PTSD (Sbordone and Leiter, 1995), this assertion has not received much support, although some authors propose that intrusive re-experiencing may be less frequent, less vivid and less severe in individuals with a brain injury (McMillian, 1996; Turnbull et al 2001). However, the literature (as briefly reviewed above) suggests that PTSD occurs in a wide range of severity of TBI and that no clear relationship has been demonstrated between severity of PTSD and severity of TBI (Feinstein et al, 2002). Severity of PTSD may instead be related to recall of the trauma, new learning, executive ability and coping strategies (McMillian et al, 2003) and insight (Evans et al, 2002), such that those with less insight report less intense symptoms.

Excessive daytime time sleepiness following head injury

Daytime sleepiness is one of the most important symptoms of TBI (Beetar et al. 1996). Beetar et al (1996) reported that 17% of their 202 TBI group reported excessive daytime somnolence during outpatient's appointments. Castriotta and Lai (2001) studied 10 patients with TBI who complained of excessive daytime sleepiness and all but one were classified as having EDS. In contrast, the prevalence of excessive daytime sleepiness in the general population is generally considered to be 0.5% - 5% (Benbadis et al. 1999), although reports from questionnaires vary from 0.3% to 13.3% (D'Alessandro et al. 1995).

Guilleminault et al (2000) assessed 184 patients with a history of head trauma who were complaining of excessive daytime sleepiness. On the MSLT it was found that mean sleep onset time was less than 5 minutes in 28% of the subject and less than 10 minutes in 82% of the subjects. A MSLT of less than 5 minutes is considered pathological and normal volunteers tend to score within the range of 10 to 20 minutes.

Sleep disorders after TBI

The prevalence of sleep disorders following TBI is between 50-73% (Castriotta et al. 2001; Cazalis et al. 2001 Clinchot et al. 1998). These include narcolepsy (Lankford et al, 1994), apnoea (Guilleminault et al, 1983, 2000; Masel et al, 2001) and periodic limb movement disorder (Masel et al, 2001), each occurring with prevalencies well in excess of the general population.

Sleep disturbances

Daytime sleepiness and sleep disturbance are among the most universal symptoms of TBI (Beetar et al. 996; Castriotta and Lai, 2001; Parsons and ver Beek, 1982; Deb, Lyons and Koutzoukis, 1998). TBI may result in damage to sleep regulating centres, including the reticular activating system and/or its connections, and the suprachiasmatic nuclei of the anterior hypothalamus, which regulate sleep timing (Evans, 2002; Boivin et al. 2003; Quinto et al. 2000). Furthermore, the experience of trauma and/or having to adjust to ongoing disability may cause mood changes, which may themselves result in sleep problems (Thaxon et al. 2002). Such sleep disturbance may then affect recovery from a head injury, quality of life and general health (Rao et al. 2002).

The prevalence of sleep disturbances following recent traumatic brain injury has been reported to range from 36% (McLean et al. 1984) to as high as 70% (Keshavan et al. 1981). Several polysomnographic studies that have been carried out in patients after traumatic brain injury have indicated various sleep-wake pattern disturbances (Appenzeller and Fisher, 1968; Feinberg et al, 1969;Markland and Dyken, 1976).

Objective sleep changes

Leonard and Pennigstroff (1970) found an increase in stage 2 sleep, a decrease in stages 3-4 and no change in the amount of REM sleep. Harada et al (1976), however, found a decrease in REM activity and an increase in spindles and K complexes in stage 2 sleep. Other studies have demonstrated a decrease in the amount of both rapid eye movement (REM) sleep and slow wave sleep and an increase in the number of awakenings from sleep in patients with TBI (George et al, 1981; Prigatano et al, 1982; Ron et al, 1980). Recovery can occur but may be subject to psychosocial influences (Fichtenberg et al, 2000). For example, Cohen et al (1992) found disorders of initiating and maintaining sleep (DIMS) were common following recent TBI, but in a group of discharged patients 29.5 months after injury, 52% complained of excessive daytime somnolence whereas only 8% reported DIMS.

Dagan et al (1991) suggested that patients may suffer from insomnia at the acute phase, however, some may go on to develop obsessive thoughts about their sleep, leading them to continue believing they are suffering from a sleep disturbance, which may, in fact have objectively improved. However, Kaufman et al (2000) highlight the difficulty in discriminating between the organic and psychological effects of a head injury. They argue that there data suggested insomniac pattern caused by mental stress rather than brain injury, and propose that the fear of going to sleep, fearful awakenings from sleep and frightening dreams, resemble findings observed in patients with PTSD. However, Perlis (1997) found that individuals with mild traumatic brain injury (MTBI) and post-concussive syndrome reported significantly more problems with sleep compared to orthopaedic controls, particularly in relation to initiating and maintaining sleep. This suggests that factors other than the psychological trauma may play a role in this sleep disturbance.

Parcell et al (2006) assessed the incidence of self-reported sleep disturbances in a community based cohort of patients with blunt TBI relative to that of a group of age and sex-matched healthy controls. A community-based sample was used as patients in hospitals or institutionalised settings tend to have sleep times and daily routines imposed on them as well as factors such as noise, room sharing and discomfort. 63 participants with TBI were consecutively recruited after discharge from rehabilitation and there were 63 controls from the general community. Participants were required to keep a sleep and wake diary for 1 week detailing bed times, sleep onset, frequency and duration of night time awakenings, time of morning awakening, number and duration of daytime naps, sleep efficiency, caffeine and alcohol consumption. They also completed the Epworth Sleepiness Scale (ESS) and a general sleep questionnaire noting whether their sleep had changed and the nature of the change since their TBI or in the past 3 months in the case of the control group. The Hospital Anxiety and Depression scales were also administered. The TBI group had a mean GCS of 9.58 and mean PTA of 19.84 days, with a range of 0 to 112 days. According to the PTA duration, 13% (n = 8) of the TBI sample had mild injuries (PTA < 24hrs), 21% (n=13) had moderate injuries (PTA 1-7days), 44% (n=27) had severe injuries (PTA 8-28 days), and 22% (n=14) had very severe injuries (PTA > 4weeks). Table 69 shows the comparisons of frequency of reported sleep changes for both groups using chi squared analysis.

Table 69 – Data from Parcell et al (2006) showing for a community based cohort of patients with blunt TBI the changes to their sleep since the TBI, and, in the control group, changes over the past 3 months

Reported change to sleep	TBI (%)	Control (%)	X ²	Р
	N=63	(n=63)		
Sleep has changed	80	23	40.20	<0.01
More night-time awakenings	46	5	27.03	< 0.01
Longer sleep onset latency	44	5	25.46	<0.01
Deeper sleep	21	10	3.01	0.08
Lighter sleep	25	3	11.82	<0.01
Bedtime earlier	34	5	16.81	<0.01
Bedtime later	18	3	6.97	<0.01
Bed wetting	2	0	1.01	0.32

Table 69 demonstrates that the TBI group were more likely to report changes in their sleep than controls, with the most frequently reported changes being increased nighttime awakenings and more difficulty falling asleep. 52% of the TBI patients reported a change in bedtime (earlier or later after the injury) compared with only 8% of controls. The TBI group reported poorer sleep quality and greater daytime sleepiness (as assessed by the ESS) than the control group, although these differences were not statistically significant. Scores for anxiety were similar for the TBI group and the control group. In the TBI group, higher levels of anxiety were associated with increased time post injury and milder injury according to GCS. This may be a result of increased insight as post injury time elapses and to the higher demands placed on TBI individuals as they attempt to return to pre-injury activities.

The TBI group scored significantly higher for depression relative to the control group. In the control group 98% scored in the normative range for depression and 2% had mild depression, however, in the TBI group, 62% scored in the normative range, 27% had mild depression and 5% had severe depression. No association was found between depression and time post injury or depression and injury severity using the GCS. On the sleep diary weekday and weekends were analysed separately. The TBI group reported significantly longer daytime naps; longer sleep onset latencies, later wakeup times and poorer sleep efficiency. On weekends the TBI group reported significantly earlier bedtimes, longer sleep onset latencies and poorer sleep efficiency than controls. Further analysis revealed that these differences were not due to employment status. The authors found that increased time since injury was associated with higher self-reported daytime sleepiness and an increase in reported number of night time awakenings. Higher scores for anxiety on the HADS were associated with increased daytime sleepiness, more reported night time awakenings, reports of sleep changes and poorer sleep quality. Higher scores for depression were also associated with reports of sleep change and worse sleep quality as well as an increase in nighttime awakenings and bedwetting.

Insomnia after TBI may be caused by depression. Fichtenberg et al. (2000) examined 91 consecutive post acute TBI patients. The Pittsburgh Sleep Quality Index (PSQI: Buysse et al, 1989) was used as a measure of sleep quality and the BDI as a measure of depression. They concluded that in the post acute phase, depression and milder brain injuries (as measured by the GCS) demonstrated the strongest relationships with insomnia. They found no relationship between the diagnosis of insomnia and age, gender and years of education, months post-injury or litigation status, and pain had only a small association with insomnia.

The above review, showing that TBI is associated with various stresses and deficiencies in well-being, combined with the review in chapter 1 showing associations between poor well-being and nightmares, leads to our first hypothesis for study 4:

There will be a higher incidence of nightmares in people who have had Traumatic Brain Injuries compared to the general population.

There is, however, a caveat to this hypothesis that will now be described. This is that some individuals with brain injury lose the ability to have, or to recall, dreams. A subset of our sample of TBI patients would thus be expected to have no nightmares because the injury had caused them to stop having dreams. This is similar to the second study of this thesis, involving apnoea, where the disorder was hypothesised to be stressful, and thus to cause nightmares, but was also hypothesised in some patients to decrease the production or recall of dreams, and hence to lead to a lack of nightmares. The changes that brain injury causes to dreaming will now be reviewed.

Changes in dreaming after neurological disease

In 1883 Charcot published the first case demonstrating that dreaming could be altered by neurological disease. Charcot reported that his patient Monsieur X had lost the ability to conjure up any visual mental images although his abstract memory was completely preserved. Monsieur X claimed that he had 'absolutely lost this power of mental vision', furthermore, he also reported a corresponding deficit within his dreams, claiming that he dreamt only of speech (Charcot, 1883/1889, p.158). Solms (1997) argues that the clinical evidence of Charcot's case suggested a vascular process in the distribution of the posterior cerebral arteries. A similar case was later reported by Wilbrand (1887): Autopsy revealed that his patient Fraulein G had a bilateral infarction in the occipito-temporal region (Wilbrand, 1892). His patient reported that prior to her illness she had dreamt frequently, whereas now she hardly dreamed at all. Hence, this syndrome became known as the 'Charcot-Wilbrand syndrome'. Critchley (1953) defined this syndrome as a condition 'whereby a patient loses the power to conjure up visual images or memories, and furthermore, ceases to dream during his sleeping hours' (p. 311). The fundamental symptom was thought to be deficient revisualization or 'visual irreminiscence' (Nielsen, 1946), accompanied by an alteration in the visual component of dreaming, prosopagnosia, and topographical agnosia or amnesia.

Charcot and Wilbrand's cases: two variants of dream abnormality

Solms (1997) highlights important differences between Charcot and Wilbrand's classic cases. He points out that on careful inspection of the original case reports it is evident that Wilbrand's case suffered an abnormality of visual recognition rather than a defective visual imagery and, hence, lacked the cardinal symptom of the Charcot-Wilbrand syndrome - visual irreminiscence. Charcot's case ceased to dream in visual images but continued to dream in words, whereas Wilbrand's patient hardly dreamt at all. Thus, the former patient appeared to lose the visual aspect of his dreams, whereas the latter lost the ability to dream at all for some period of time, or dreamt much less frequently. Solms argues that Charcot's variant of the syndrome may be an incomplete form of Wilbrand's variant. The latter assumption would imply that the lesion sites of the two variants of the Charcot-Wilbrand syndrome should overlap as should the clinical presentations of the patients.

Solms (1997): The neuropsychology of dreams

Mark Solms interviewed 361 consecutive neurological patients about changes in the nature and frequency of their dreaming since the onset of their illness. From these clinco-anatomical studies and review of the previous literature Solms (1997) postulated that there were 4 major disorders of dreaming after head injury. These being, cessation of visual dream imagery (reported by 1.1% of the effective sample). global cessation of dreaming (34.9%), confusion between dreams and reality (5.3%) and recurring nightmares (7.9%). Solms also found that patients reported a number of other subtle changes in their dreams; some were qualitatively similar to the gross changes described above, however, these were not considered to be clinical disorders of dreaming, although evidence suggested that some of these were sub-clinical forms of the major disorders. For instance, some patients reported reduced vivacity of visual dream imagery (a sub-clinical variant of cessation of visual dream imagery), reduced frequency of dreaming and reduced narrative complexity and emotional intensity of dreaming (possible sub-clinical variants of global cessation of dreaming), increased vivacity of dreaming and increased frequency of dreaming (possible sub-clinical variants of the confusion between dreams and reality) and, finally, some patients reported increased frequency of (nonrecurring) nightmares (a possible sub-clinical variant of recurring nightmares). Completely normal dreaming was only reported by 8.6% of the cerebrally impaired patients.

Solms found that all four of the major disorders of dreaming correlated with specific sites or types of lesion, however, this was not the case for the sub-clinical changes in dreaming, thus, he points out that these sub-clinical types are of scientific interest but are of limited diagnostic value.

Solms used a control group of 29 non-head injured individuals who had undergone similar trauma and hospital procedures as the study group. None of the clinical disorders of dreaming were reported in the control group with the exception of global cessation of dreaming, which was reported by one of the control participants (a case of hysteria). Of the controls, 34.5% reported no changes in their dreams since the onset of their physical symptoms.

Anatomical correlates of global cessation of dreaming

In his review of all the previously published cases of global cessation of dreaming with focal damage, Solms (2002) states that the lesions fell into two anatomical subgroups. The majority of these had parietal lobe lesions, or space occupying lesions in close proximity to the parietal lobe. The critical area seems to be the inferior parietal lobule. It has been suggested that lesions to Brodmann's areas 39 and 40 are the most restricted damage sufficient to produce the syndrome (Hobson et al. 2003). There appears to be no lateral bias between the hemispheres.

The second group had deep bifrontal lesions, more specifically the lesion was situated in the white matter immediately anterior to the frontal horns of the lateral ventricles; in these cases the damage was invariably bilateral. This observation is supported by Frank (1946), who found that loss of dreaming was a common result of

prefrontal leukotomy. Anatomical evidence has suggested that the white matter implicated in this syndrome is composed essentially of the fibre tracts connecting ventral midbrain nuclei with the limbic system (e.g. cingulated gyrus and nucleus accumbens) and frontal cortex. Bilateral damage to the white matter in the vicinity of the frontal horns of the lateral ventricles was found to be the most restricted site causing the syndrome.

This pattern of effects of the two types of lesions led Solms to his neurocognitive theory of dreaming, in which he proposed a 'backward projection' mechanism of dream formation, whereby abstract thinking in anterior areas of the brain is converted into concrete perceptions in posterior areas.

Imaging studies of REM sleep

Functional imaging studies have been remarkably complementary to the lesion data (Braun et al 1997, 1998; Heiss et al 1985; Madsen et al, 1991; Maquet et al, 1996; Nofzinger et al, 1997). Imaging studies have demonstrated specific activation of limbic and paralimbic regions of the forebrain in REM sleep compared to waking (Braun et al. 1997; 1998; Maquet et al. 1996; Nofzinger et al. 1997). Such findings, Hobson et al (2003) argue, imply that dream emotions may be the primary shaper of dream plots rather than following secondary to the dream plot. Nofzinger et al. (1997) concluded from their PET study that an important function of REM sleep is the integration of neocortical function with basal forebrain and hypothalamic motivational and reward mechanisms. PET studies have also demonstrated significant deactivation of a large area of the dorsolateral prefrontal cortex in REM (Braun et al. 1997; Maquet et al. 1996). Single photon emission computer tomography (SPECT) and fMRI have demonstrated a similar decrease in cerebral blood flow to frontal areas during REM (Lovblad et al. 1999; Madsen et al. 1991a). However, these findings need to be replicated. The latter observations are of interest in terms of the cognitive deficits in memory, orientation and self-reflective awareness during dreaming. These findings suggest that the forebrain activation and synthesis processes underlying waking and dreaming are very different. Thus, the combination of the preferential activation of subcortical and cortical limbic structures which mediate emotion and the relative inactivation of the lateral prefrontal cortex involved in directive thought paints an overall impression of REM sleep (and REM dreaming) as an emotion driven state with deficits in orientation, memory, volition and directive thought.

Global cessation of dreaming: Posterior variant

The finding that the parieto-temporal-occipital junction is involved in dreaming is not new (Doricchi & Violani, 1992; Greenberg & Farah, 1986). Murri et al (1985) assessed dream recall in 19 patients with unilateral hemispheric lesions of a vascular or neoplastic nature. Dream recall was investigated using both morning recall diaries and REM awakenings. They reported that the site of the lesion appeared to be the only factor significantly influencing dream recall. Patients unable to report dreaming on provoked awakenings often had lesions in the temporo-parieto-occipital region. Furthermore, Cathala et al (1983) using the REM awakening method in patients with unilateral or bilateral lesions in the frontal or parietal regions, also reported a reduction in dream activity mainly in patients with parietal lobe lesions.

Of the 361 patients interviewed by Solms, 34.9% reported that they had stopped dreaming completely since the onset of their illness. Of the 132 patients who reported changes in their dreaming, Solms (1997) found 112 patients with forebrain lesions who lost dreaming for varying periods of time or permanently. Looking only at the non-dreaming patients with localized lesions he found that 47 of these patients had focal lesions in one or both parietal lobes. Solms found that 45 of the 47 nondreaming patients with definite parietal lesions had unilateral lesions, and the laterality of these lesions was equally distributed between the two hemispheres. Thus, this suggests that global cessation of dreaming arises with equal frequency with left and right posterior cortical lesions. Hence, this is in agreement with the assertions of Doricchi and Violani (1992) that simple right-dominant versus left-dominant theories of dreaming are ''too generic and potentially misleading'. Also, as Solms points out, findings on image generation have also suggested that 'both hemispheres can generate images' (Kosslyn, 1994, p.319). This finding led Solms (1997) to the hypothesis that the cortical network for spatial representations, located primarily in the parietal lobes (Roberston, 1998), is essential for dreaming.

Deficits following left parietal lobe lesion

The left parietal lobe lesion associated with loss of dreaming was also found to be associated with left - right confusion and finger agnosia (2 of the components believed to make up Gerstmann syndrome). Thus, it is reasoned that a disorder in the function of this part of the brain results in the inability to represent perceptual information symbolically. Primary perceptual abilities appear to remain intact but patients lose the ability to extract higher-order abstraction from perceptual information in all modalities. Thus, they are not able to conceptualise concrete information and perform symbolic operations on it. Therefore, this suggests that abstraction, conceptualisation and symbolization are essential component functions in the process of dreaming. This ability to derive abstract concepts from spatially organised heteromodal information was termed 'quasi-spatial synthesis' (Luria, 1973, 1980). Solms (2002) details psychoanalytic observations of a patient with a focal lesion in the left parietal lobe (more specifically the lesion was localised to the supramarginal gyrus, but extending anteriorly to include the primary sensorimotor cortex). The patient, Mr. L, was found to demonstrate a real absence of constructive thoughts or even simply of active associations. He appeared to lack the capacity for elaboration or abstraction at any level. Although his 'ego activities' were judged to be rational and appropriate, they were extremely impoverished, restricted and concrete. He was 'mentally blank, save for these fragmentary islands which he tried desperately to join together, usually without success' (Solms, 2002, p.123). Mr. L also reported global cessation of dreaming. When Mr. L began regaining explicit continuous memories of his life and showed evidence of spontaneous thought, so he too began to dream again. This was very important to Mr. L as he felt it was 'a vital sign of inner life and of psychological recovery' (Solms, 2002, p.128). Luria's (1972) famous case Comrade Zasetsky, who temporarily ceased to dream, also suffered a similar wound to the left parieto-occipital region.

Deficits following right parietal lobe lesion

Solms found that loss of dreaming caused by right inferior parietal lobe lesions was also accompanied by deficits of visuospatial short-term memory performance. Again, these patients do not present with primary perceptual difficulties, but rather they cannot hold perceptual information in consciousness, in simultaneous visuospatial patterns, and this applies to both externally and internally generated information. Solms concludes that 'dreaming and visuospatial working memory share the same elementary component function that is contributed by this part of the brain' (Solms, 2002, p.47)

These findings suggest that both symbolic quasi-spatial functions and concrete spatial functions are fundamental to the process of dreaming (Solms, 1997; 2000), which is also supported by evidence from a neuroimaging study that showed activation of the right inferior parietal cortex during REM sleep (Maquet et al. 1996)

Some patients with parietal damage continue to dream

Solms also found that a large number of his patients with parietal lobe damage continued to dream. It was found that the dreaming and non-dreaming patients with parietal damage could not be distinguished on the basis of pathology type. However, Solms noted that the mean number of weeks elapsed since the last neurological insult was somewhat lower for the non-dreaming patients than for the dreaming patients (non-dreaming group, mean tine since neurological insult = 30.8 weeks, dreaming group, mean time = 97.2 weeks). Hence, this suggests that some of the dreaming subjects with parietal lesions may have been non-dreamers at an earlier stage in their illness. In order to analysis the issue of recovery from cessation of dreaming Solms followed up 13 of these non-dreaming cases. 10 of these started to dream again within 1 year of the last neurological insult. However, he states that there are at least some patients who do not recovery dreaming and that these tend to have extensive right hemisphere damage.

Cessation of dreaming: anterior variant

Of the patients with localising lesions reporting cessation of dreaming, Solms (1997) reported that nine of these had bifrontal lesions in the white matter inferior to the frontal horns of the lateral ventricles. This area provides a crucial link between limbic structures and frontal cortex. Solms found that in the bifrontal cases, global cessation of dreaming correlated significantly with advnamia (lack of spontaneous motivational impetus), disinhibition and perseveration (these symptoms are classically associated with frontal lobe dysfunction). Solms found that advnamia statistically discriminated between dreamers and non-dreamers, and argues that the frontal lobe syndrome in non-dreaming patients is thus of the mediobasal variety. Solms reasoned that the fundamental component factor which is disordered by damage to this part of the brain is spontaneous motivation, and thus dreaming is a meaningful psychological event. Interestingly, the precise lesion that causes this type of loss of dreaming was the same as that targeted in the modified prefrontal leukotomy procedure used as a treatment for severe mental illness (Panksepp, 1985), and loss of dreaming was one effect of that procedure (Frank, 1946, 1950; Jus et al. 1973). In contrast, Doricchi and Volani (1992) reported that the preservation of dreaming with frontal lobe lesions was their 'most robust and consistent conclusion' (Doricchi & Volani, 1992; p118). Solms explains this apparent contradiction, arguing that authors reporting a low incidence of loss of dreaming among frontal cases studied only patients with unilateral and/or convexity lesions, whereas those reporting a high incidence of loss of dreaming among frontal cases studied only patients with bilateral, white matter lesions. Indeed, Solms reported a number of patients with bifrontal lesions who reported preservation of dreaming, however, the majority of these patients had lesions in the cortical convexity of the frontal lobes.

Why is the ventromesial quadrant of the frontal lobes so important to dreaming?

Solms (1997, 2003) argues that the ventromesial quadrant of the frontal lobes contains substantial numbers of fibres connecting frontal and limbic structures with dopaminergic cells in the ventral tegmentum area (VTA). These circuits arise from a group of cells in the VTA, where the source cells for the mesocortical and mesolimbic dopamine systems are situated. They ascend through the forebrain bundles of the lateral hypothalamus via basal forebrain areas and they terminate in the amygdala, anterior cingulated gyrus, and frontal cortex. It has been suggested that this circuit instigates goal seeking behaviours and appetitive interaction with the world (Panksepp, 1985; 1998b), and it is frequently described as the 'seeking' or 'wanting' command system of the brain (Panksepp, 1998a). It is considered to be the primary site of action of many stimulants (e.g. amphetamine and cocaine, see Role and Kelly, 1991), with overactivity thought to result in the positive symptoms of schizophrenia (Bird, 1990) and also excessive, usually vivid, dreaming and nightmares (Nausieda et al. 1982; Scharf et al. 1978) in the absence of any effect on the intensity, frequency and duration of REM sleep (Hartmann et al. 1980). Furthermore, transaction or chemical inhibition of this circuit reduces the positive symptoms of schizophrenia (Breggin, 1980; Panksepp, 1985), some formal features of which have been equated with dreaming (Hobson, 1992, 1988b). Indeed, drugs that block this circuit (e.g., haloperidol) inhibit excessive and usually vivid dreaming (Sacks, 1985, 1990, 1991). Therefore, Solms argues that the mesocortical-mesolimbic dopamine system plays a causal role in the generation of dreams. This also accords with Braun et al's (1997)

neuroimaging study reporting increased activation of the caudal orbital frontal area during REM sleep in comparison to NREM sleep.

Is cessation or reduction of dreaming a memory disorder?

It is reasonable to ask whether patients reporting cessation of dreaming have actually stopped dreaming as a result of their neurological illness or whether they just cease to be able to remember their dreams. Hence, is cessation of dreaming a disorder of dreaming or a disorder of memory? In order to answer this question researchers have used two different methods. Firstly, the REM awakening technique has been used as it is generally accepted that most people experience dreams during REM sleep. It is argued that this technique minimises the potential for forgetting as even the most severe amnesiacs are able to recall what happened a few seconds ago. The second technique requires subjecting non-dreamers to formal neuropsychological tests in order to obtain objective scores of their current memory function, on the assumption that if these patients are forgetting their dreams they are also likely to forget other experiences.

Jus et al. (1973) awakened 9 amnesic patients who reported cessation of dreaming from REM defined sleep during two nights in a sleep laboratory. Of the 66 REM awakenings only two elicited the report of a dream. This incidence of 3% is in sharp contrast to the 80% normally obtained. Murri et al. (1985) subjected 12 participants who had been unable to recall any dreams over a 10-day observation period, together with seven patients who had been able to recall dreams over the observation period, to sleep laboratory investigation. (Note that the non-dreamers did not report global cessation of dreaming, they just had not recalled any dreams for 10

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days.) It was found that all seven of the dreamers frequently reported dreams on REM awakenings (75% of REM awakenings elicited dream reports), whereas only three of the 12 non-recallers reported dreams (17% of awakenings elicited dream reports). However, there were no differences between the groups in sleep profiles or in amount of REM sleep. Other studies have confirmed the subjective reports of dream cessation following neurological damage (Michel & Sieroff, 1981; Schanfald, Pearlman, and Greenberg, 1985). Thus, overall it seems that neurological patients' reports of nondreaming can be confirmed by REM sleep awakenings: this suggests a disorder of dreaming and not a disorder of memory.

Studies assessing waking memory function of dreamers and non-dreamers have, however, been inconsistent. For example, Muri et al. (1984) found no relationship between dreaming and memory, yet Cathala et al. (1983) found strong positive correlations between tests of visual and verbal memory and frequency and informational richness of dream recall, and Arena et al (1984) found that the absence of dreaming was related to poor long-term verbal recall. Furthermore, Talland (1965) reported that 10 out of 20 patients with Korsakoff amnesia stated that they never dreamed and 8 reported that they only ever dreamed rarely. These assertions were also verified in a sleep laboratory. Greenberg et al. (1968) obtained only 1 dream report from 7 Korsakoff annesics over several nights in a sleep laboratory with a total of 34 REM awakening (3% recall rate). He states that most of the truly amnesic patients in his series stated that they were unsure of whether or not they were dreaming. Hence, these and other studies add support to the notion that global cessation of dreaming may be an artefact of general amnesia. However, Solms' (1997) review of the previous literature found that in 79% of cases, global cessation of dreaming was not

associated with general amnesia. Thus, it seems that many amnesiacs do not dream, but very few non-dreamers are actually amnesic. Bischof and Bassetti (2004) reported the case of a 73 old woman reporting a total dream loss after acute, bilateral occipital artery infarction (including the right inferior lingual gyrus), which lasted over three months. They found that a detailed neuropsychological assessment revealed normal results on tests of alertness and vigilance, verbal and non-verbal learning, and shortterm as well as long-term memory. The patient was also awakened five minutes after polysomnographic onset of the first REM period and 10, 15 and 20 minutes after the following REM periods. No dream was reported after these awakenings.

The literature can be summarised as supportive of Solms' conclusions that 'although some cases with loss of dreaming may be dreaming but then forgetting their dream, in the majority of cases the dream disorder occurs independently of amnesia' (p. 36), and that 'subjective cessation of dreaming cannot be dismissed as an artefact of recent memory disorder as some authors have suggested' (p 160).

In his own cohort of patients demonstrating cessation of dreaming, Solms showed that neither long-term memory disorder nor audioverbal short-term memory disorder were able to significantly discriminate between dreaming and non dreaming patients. Solms also noted that most of his truly amnesic patients were unsure as to whether they were still dreaming or not, as opposed to being sure they were not dreaming. (A note should be made, however, that Solms did in fact find that dreamers and non-dreamers were significantly different in relation to visuospatial short-term memory as measured by the immediate recall trial of the Complex Figure Test. However, when patients were separated according to lesion site it was found that this difference only applied to patients with parietal lesions and not to patients with bifrontal lesions.) It will thus be important in study 4 to obtain various memory test scores for non-dreaming and dreaming TBI patients.

Is cessation of dreaming a language disorder?

Many authors have suggested that global cessation of dreaming may be the consequence of an aphasic disorder (Broughton, 1982; Doricchi & Violani, 1992; Epstein & Simmons, 1983; Foulkes, 1978; Jacobson, 1973). Some authors have claimed that the language disorder prevents patients from reporting their dreams, whereas others argued that the language disorder makes it impossible for these patients to generate dreams (Moss, 1972). The latter view means that the loss of ability to generate language necessarily results in the loss of ability to generate dreams. This idea is consistent with the idea that the dream-generation module is localized to the left hemisphere (Greenberg and Farah, 1986). However, the problem with such an argument is that only 52% of the non-dreamers reported in the literature were aphasic (Solms, 1997). Furthermore, in Solms' own study he found the incidence of aphasia among his non-dreaming patients was 25%. Indeed, Schanfald et al (1985) investigated the relationship between aphasia and language and concluded that aphasiacs are able to generate dreams. Moreover, Solms showed that aphasia also occurred in 50% of cases of non-visual dreaming, hence, when postulating a relationship between dreaming and language, this latter association must also be explained.

Doricchi and Violani (1992) proposed possible linguistic-semantic disorders in patients who report cessation of dreaming. In this account they suggest that such disorders may not necessarily produce manifest aphasia but may nevertheless prevent dreaming. Muller (1992) states that this explanation is unsatisfactory as it can explain

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'all disconfirmatory presentations by postulating ad hoc connections with language'. Finally, on this, Solms (1997) argues that if cessation of dreaming is caused by an aphasic disorder then it should never occur after right unilateral damage. Yet such cases of dream cessation with right unilateral damage have been reported (Humphrey & Zangwill, 1951; Gloning & Sternbach, 1953; Johann et al. 1957), indicating that cessation of dreaming can occur for reasons unconnected with aphasia.

Normal dreaming after brain injury

Solms (1997) found that unchanged dreaming correlated significantly with lesions to the dorsolateral frontal lobes. Thus, he suggested that the dorsolateral frontal region did not play a significant role in the normal dream process. In line with this, several H₂¹⁵ O PET studies have found significant deactivation of the dorsolateral frontal cortex during both REM (Braun et al. 1997; Maquet et al. 1997) and NREM (Anderson et al. 1998; Braun et al. 1997). Furthermore, a single photon emission computed tomography (SPECT) study found decreased blood flow to the frontal areas during REM sleep (Madsen et al 1991). However, these accounts of decreased frontal area activity and dreaming are contradicted by Heiss et al's (1984) finding of increased superior frontal activity during dreams, as compared to dreamless sleep, and Nofzinger et al's (1997) finding of no significant decrease of glucose uptake in the dorsolateral convexity when comparing REM sleep and waking.

So far the relationships between brain lesions and alterations or cessation of dreaming have been addressed. However, a confound in this relationship will now be described, that of sleep quality, as this will have to be taken account of in study 4.

Sleep quality and dreaming

Solms (1997) conducted a preliminary assessment of sleep quality following head injury. He asked his 361 patients whether or not their sleep had been affected by their illness. Responses were classified under four heading; sleep was considered to be (a) better, (b) disrupted, (c) unaffected since the onset of illness, or patients were (d) unsure of whether or not there had been any change in the quality of sleep. The results are shown in Table 70.

Table 70 – Data from Solms (1997) categorising dreams as either present or absent as a function of disruption of sleep since onset of illness

Dreams	Sleep since onset of illness		
	Disrupted	Not disrupted ^a	
Absent	49	52	
Present	59	127	

^a i.e. better or unaffected

Solms (1997) found that the non-dreaming patients experienced significantly more disturbed sleep than the dreaming patients. He interpreted this as providing support for the sleep-protection theory of dreaming. This thesis will not be following that line of argument, as there are alternative explanations of these data that do not impact on this or any other proposed function of sleep. But study 4 will be designed to take account of the sleep quality confound indicated by these data.

Aims of study 4

The first aim of this study is to assess the prevalence of idiopathic nightmares, repetitive nightmares and dream cessation in a population with traumatic brain injury.

As stated above, the first hypothesis is:

There will be a higher incidence of nightmares in people who have had Traumatic Brain Injuries compared to the general population.

The second hypothesis follows from the work of Solms reviewed above:

There will be a higher incidence of complete absence of dreaming in people who have had Traumatic Brain Injuries compared to the general population.

The second aim of this study is to investigate predictors of whether a TBI patient will have global cessation of dreaming, increased nightmares, repetitive nightmares, or dream normally. These predictors include measures of functional brain injury, anxiety, depression, pain and time since injury.

Methodological note on investigating hypothesis 3:

The work of Solms that has been reviewed above is highly important for the issue of what forebrain structures support dreaming, and hence what changes to dreaming may occur after head injury. However, it is evident that Solms' (1997) methodology is problematic as it relied upon patients reporting whether or not their dreams have changed subsequent to neurological damage. This method is problematic as people's beliefs about the content of their dreams are influenced by a number of factors apart from the actual dream content (Beaulieu-Prévost and Zadra, 2005a).

Furthermore, participants with neurological conditions may have difficulty recalling the frequency and nature of their dreams before the onset of their illness or injury. Indeed, Solms (1997) acknowledges that 'many patients had great difficulty framing definite responses to some of the...questions, which pertained to the more elusive aspects of dreaming' (pp. 84). For example, patients had difficultly assessing the narrative complexity, emotional intensity and frequency of their current dreams. Dream and nightmare frequency will thus be assessed with a short simple questionnaire, referring only to their current frequency of dreams and nightmares. Data from this questionnaire will be compared with normative data from other studies as opposed to patients' own assessment of premorbid dreaming.

Chapter 15

Study 4: Nightmares and traumatic brain injury

Method

Participants

51 participants who had suffered a head injury (M=35, F=16; mean age = 39.69, SD = 13.7, range = 20-77) were recruited from referrals to a head trauma clinic at the University of Wales Swansea for neuropsychological examination. 78% (n=41) of participants had sustained head injuries in a road traffic collision, 5.8% (n = 3) during an assault, 7.7% (n= 4) from a fall, and 7.6% (n=2) from a blow to the head. They were considered, by referring clinicians, to have suffered a moderate or severe head injury, based on levels of consciousness (recorded by Glasgow Come Scale, GCS). The mean GCS was 10.91 (SD = 4.24; range 3-15). The median length of posttraumatic amnesia (PTA) was category 4 (i.e., less than 2 weeks) and the interquartile range was categories 2 - 5 (less than one hour to more than 2 weeks). Mean time since injury was 49.35 months (SD=36.13, range = 9-177 months). CT brain scans were not available but the majority of participants were considered by referring clinicians to show neuropsychological signs of frontal dysfunction, against a background of diffuse cerebral injury.

Participants were given an information sheet regarding the study and its procedures. Informed consent was obtained from all participants (see appendix 24 for example of consent form). The study, its procedures and participant documents were

approved by the Ethics Committee at the University of Wales Swansea and, due to most participants being NHS patients, by the Local Research Ethics Committee (covering Neath, Port Talbot and Swansea) (appendix 25).

Materials

Injury Severity

The Glasgow Coma Scale (GCS) and duration of Post Traumatic Amnesia (PTA) were used as indices of injury severity. The GCS is a measure of depth of coma and is assessed during admission to hospital, lower scores indicate greater severity of injury. This information was obtained from patient's medical notes.

The duration of PTA is the time from injury to the start of continuous memories. It is the sum of any time spent in coma or in confusion when behaviour superficially seems to be normal but when individuals lack the capacity to remember ongoing events (Greenwood, 1997). This information was also obtained from patient's medical notes.

Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983)

This scale was described fully in Chapter 11, in the Method for Study 3. In study 4 anxiety was assessed using the 7 items forming the anxiety scale of the Hospital Anxiety and Depression Scale. A score of 8 or above is considered to be indicative of 'possible' clinical levels of anxiety, and a score of 11 or above is considered indicative of a 'probable' diagnosis anxiety. The anxiety scale of the HADS is particularly suited for this study as this scale tends to be less confounded by somatic symptoms, which are associated with medical conditions such as Traumatic Brain Injury.

The Beck Depression Inventory for Primary Care (BDI-PC-7).

Depression was measured using the BDI-PC. This is a 7-item self-report instrument specifically designed for medical patients in order to minimise the overlap between symptoms of their medical illness and symptoms of depression. For example, the somatic symptoms of depression, that is, tiredness or fatigue, as found in the 13item Beck Depression Inventory (Beck and Beck, 1972), may not be indicative of depression per se in patients with cardiovascular disease, but may be caused by a decreased circulation of oxygen. Also, some questions on the 21-item BDI-II, for example, loss of energy and difficulty concentrating, might overlap with the symptoms of a head injury. In order to overcome such problems Beck and colleagues derived a set of non-somatic items from the BDI-II (Beck-II; Beck, Steer and Brown, 1996) in order to specifically screen for depression in primary care patients. Sadness and Loss of Pleasure (anhedonia) was included because one of these two symptoms must be present for the establishment of a DSM-IV major depression disorder (MDD). Suicidal thoughts or wishes were included as an important clinical indicator of suicidal risk in depressed patients. Pessimism, Past Failure, Self-Dislike, and Self-Criticalness were chosen because the symptoms loaded saliently on the Cognitive dimension of the BDI-II presented in the Manual for the Beck Depression Inventory II (Beck et al. 1996) for 500 psychiatric outpatients.

Weiss (1996) found that the BD-PC 7 items were independent of the context of the other 14 BDI-II items and that it could function as a stand-alone instrument. The 1-week test-retest reliability of the BDI-PC for the 26 psychiatric outpatients who were re-administered the BDI-II by Beck et al. (1996) was 0.82, p>0.001. However, as withother measures high BDI-PC scores only indicate that a detailed psychiatric evaluaton may be warranted.

In this study the BDI-PC was used instead of the depression scale for the HADS as it has been found to be approximately twice as positively associated with the diagnosis of major depressive disorders as the HADS depression scale (Beck, Steer, Ball, Ciervo & Kabat, 1997). Good internal consistency has been demonstrated for the BDI-PC (0.86). Cicchetti (1994) described alpha coefficients within the mid .80s ashaving 'good' reliability for clinical purposes.

Steer, Thomas, Cavalieri, Leonard and Beck (1999) found that a BDI-PC cut off sccre of 4 and above had a 97% sensitivity and 99% specificity rates, respectively, for identifying internal medicine outpatients diagnosed with and with major depressive disorder. However, Beck et al (1997) found that a cut off score of 6 and above yielded maximum clinical efficiency with family practice outpatients of whom 39% had no medical disorder. Thus, Steer et al suggest that if a physician wants to minimise the possibility of identifying a patient as depressed then the cut off score should be raised to 5 or 6 and above.

The BDI-PC 7 is scored by summing all of the highest ratings for each of its sevenitems. Each item is rated on a 4-point scale ranging from 0 to 3. The maximum score 21. In order to address the minimum DSM-IV requirements for the duration of major depression disorders symptoms, respondents are asked to describe themselves for the 'past two weeks, including today'.

Pittsburgh Sleep Quality Index (PSQI; Buysse et al, 1989)

The PSQI is an 18 item self-report questionnaire, which examines sleep quality and sleep disturbances for the previous month. Participants are required to provide information such as usual bed time, usual time of getting up, time taken to fall asleep and the hours of actual sleep per night. They are required to complete a 4-point scale for a variety of different sleep factors. The questionnaire yields seven component scores and one global sleep quality score. The seven components consist of subjective sleep quality, sleep onset latency, total amount of sleep, sleep efficiency, specific sleep disturbances, use of sleep medication and daytime dysfunction. An overall score of 21 on the PSQ indicates the worst sleep quality. The PSQI was developed to measure 'sleep quality' but has been shown to be particularly sensitive to the presence of insomnia and to effectively discriminate between 'good' and 'poor' sleepers (Buysse et al, 1989).

The PSQI has been demonstrated to be a valid screening tool for assessing sleep difficulties among patients with traumatic brain injury (TBI; Fichtenberg et al, 2001). Fichtenberg et al (2001) demonstrated that when a sample of TBI patients were divided into insomnia and non-insomnia groups based on clinical interviews using DSM-IV criteria, those with insomnia had considerably higher PSQI scores than those without (mean PSQI was 13.9 (SD, 3.3) and 3.5 (SD, 2.3) respectively). The PSQI demonstrated impressive sensitivity and specificity to insomnia with the overlap between insomniacs and non-insomniacs consisting of only one value; a global score of 8.

Insomnia was correctly classified in 96% of the total sample using a cutting score of 8 on the PSIQ and this increased to 98% using a cutting score of 9. Thus, the

Global score alone seems to be an effective insomnia screen for a post acute TBI population.

Furthermore, PSQI derived calculations of sleep onset, latency and sleep duration were found to correlate relatively strongly for sleep onset and modestly for sleep duration and efficiency as measured by patient sleep diaries.

Pain and hypersomnia

The PSQI contains an item assessing the frequency with which pain disrupts sleep and two items assessing daytime sleepiness. Fichtenberg et al (2001) examined the concurrent validity of individual PSQI items relating to pain and hypersomnia with 47 TBI patients. In addition to the PSQI they administered the Multidimensional Pain Inventory (MPI), (Kerns, Turk & Rudy, 1985) and the Epworth Sleepiness Scale (ESS; Johns, 1992). It was found that the PSQI symptomatic scorers obtained significantly higher pain intensity scores on the MPI relative to asymptomatic scorers (mean = 34.3; SD =18.2 and mean =14.3; SD 10.1). On the PSQI measure of daytime somnolence symptomatic scorers obtained significantly higher levels of daytime sleepiness on the EES compared with asymptomatic scorers (M= 11.4 (SD= 5.8) and M= 4.7 (SD= 3.4) respectively; t=3.4; p<0.01).

On the basis of such research the PSQI was believed to be particularly appropriate for use in this study population. Furthermore, this and the other measures were appropriate as they are relatively brief and understandable for individuals with cognitive impairments.

Dream questionnaire.

Participants used a tick box format to comment on the frequency of dreams, nightmares and night terrors (appendix 26). Five responses were possible ranging from; at least once a week to never. The categories 'less than once a year' and 'never' were used in order to be sure that those reporting non-dreaming do not just dream very rarely. Participants used a 'yes/no' format to answer items on nightmare distress and repetitive nightmares. Nightmares were defined as 'a vivid dream that is frightening or disturbing, the events of which you can remember clearly and in detail on awakening'. A night terror was defined as 'a sudden awakening in fear, possibly accompanied by a scream, but where you do not remember any content'.

Neuropsychological tests

A battery of neuropsychological tests was administered to all participants. Patients were administered the Wechsler Adult Intelligence Scale (3rd edition) (WAIS III; Wechsler, 1997) and the Wechsler Memory Scale (WMS III; Wechsler, 1997), the Hayling and Brixton Tests (Burgess & Shalice, 1997) and two tests (Zoo Map and Key Search) from the Behavioural Assessment of Dysexecutive Syndrome battery (BADS;Wilson et al, 1996). The reliability, validity and normative data for these batteries are reported in their technical manuals.

The following subtests of the WMS-III were used:

Logical Memory

<u>Part I:</u> Two short stories are presented orally. The second story is presented twice. The participant is then asked to recall the stories from memory.

<u>Part II:</u> The participant is required to recall both the stories presented in logical memory part 1 after a delay of 25-35 minutes.

Faces:

<u>Part I:</u> A series of 24 faces is presented visually and the participant is told to remember each face. The participant is then presented with a series of 48 faces and asked to identify the faces they were asked to remember.

<u>Part II:</u> After 25-35 minutes the participant is again presented with 48 faces and asked to identify the faces they were initially shown.

Verbal Paired Associates:

<u>Part I:</u> This is an orally presented task which requires the participant to learn novel word associations. After eight word pairs are read, the first word of each pair is given, and the participant is required to provide the corresponding word. There are four trials in different orders.

<u>Part II:</u> After a delay of 25-35 minutes the participant is orally presented with the first word of each pair learned in the immediate condition and asked to provide the corresponding word.

Family pictures:

<u>Part I:</u> A family photograph and series of scenes are presented. The participant is then asked to recall information such as who was in each scene, what they were doing and where they were.

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<u>Part II:</u> The delayed condition is presented 25-35 minutes after the first; in this condition participants recall the same information as previously without the scenes being shown again.

Visual Reproduction

<u>Part I:</u> A series of 5 designs are shown; one at a time for 10 seconds each. After each design is presented the participant is required to draw each one from memory.

<u>Part II</u>: 25-35 minutes after the initial condition participants are asked to draw the designs learned in part one in any order.

Spatial Span: A series of spatial patterns is visually presented on a three dimensional board. In the first task, the examiner points to a series of blocks at a rate of one block per second, and the participant then points to the same blocks in the same order. For the second task, the examiner points to a series of blocks, and the participant points to the same blocks but in the reverse order.

<u>Letter - Number Sequencing</u>: A string of alternating letters and numbers is presented, and the participant is asked to repeat the string by putting the numbers together in ascending order. The length of the string is gradually increased.

Subtests of the Wais-III (UK) used in study 4

<u>Picture completion:</u> The participant is presented with a series of pictures each of which has some important part that is missing and that the participant must identify.

<u>Vocabulary:</u> Participants are presented with a series of words that they are asked to define.

Similarities: Participants are presented with pairs of words and the participant is required to explain the similarities between these words.

<u>Digit-symbol-Coding</u>: The subject is shown a series of numbers, each of which is paired with its own corresponding test symbol. The subject is then shown the above series of numbers with empty boxes beneath them and they are required to use the key to copy the symbol corresponding to the number into the empty box.

<u>Block design</u>: Participants are required to reproduce a set of modelled or printed two-dimensional geometric patterns using two-colour cubes.

<u>Arithmetic:</u> Participants are orally presented with a series of arithmetic problems to solve.

<u>Matrix Reasoning:</u> A series of incomplete matrices of patterns that the participant completes by pointing to or saying the number of the correct response from five possible choices.

<u>Digit span:</u> The participant is orally presented with a series of number sequences and is asked to repeat them verbatim for Digits Forwards and in reverse for Digits Backwards.

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<u>Information</u>: Participants are asked a series of questions that tap into their general knowledge of common events, objects, places and people.

<u>Picture Arrangement:</u> A set of pictures is presented to the participant in a mixed-up order and they are required to rearrange the pictures so that they are in the correct sequence.

<u>Comprehension</u>: Participants are required to answer a series of questions that tap into their understanding of social rules and concepts or solutions to everyday problems.

<u>Symbol search:</u> Participants are presented with a series of paired groups, each pair consisting of a target group and a search group and they are required to mark the appropriate box to indicate whether the target symbol appears in the search group.

Tests of executive function

The Hayling and Brixton tests (Burgess & Shallice, 1997) and the Zoo Map and Key Search sub tests from the BADS (Wilson et al. 1996) are all specialised tests of executive functioning. The Hayling test measures initiation speed and response suppression, while the Brixton test measures rule detection. Key search assesses ability to plan a strategy to solve a problem (finding a key in a field). The score is based on a number of criteria, including whether the rater believes the strategy to be systematic, efficient and likely to be effective. A penalty is imposed for lack of speed. The Zoo Map assesses ability to independently formulate and implement a plan (high demand condition) and to follow a pre-formulated plan (low demand condition). It involves plotting or following a route through a map that does not contravene a set of rules. The score is based on successful implementation of the plan. Penalties are imposed for rule breaks and lack of speed. Impaired performance on the Hayling and Brixton are determined by using a scaled score of 2 (abnormal), as recommended in the manual. A similar classification of patients based on the Zoo Map and key search tests is not possible as the manual only provides an overall profile score for the whole battery on a percentile basis.

Post traumatic Stress Disorder

A clinician using a semi-structured interview based on DSM-IV criterion assessed the presence of PTSD.

Procedure

Participants attending a head trauma clinic at the University of Wales Swansea were invited to take part in the research. It was emphasized to patients both verbally and in written information sheets that the research was entirely separate from their ongoing care at the head trauma clinic. The objectives of the study were explained to participants as well as what would be required of them. It was also stressed that any data collected about them would be entirely confidential and that the final analysis would not identify them.

Participants were administered the neuropsychological tests described above in addition to the Pittsburgh Sleep Quality Index, the HADS and the dream questionnaire. The presence of PTSD was assessed during a clinical interview with a clinician.

Design

This is a between groups design where assignment to groups is based upon whether individuals have ceased to dream, have frequent nightmares, or report dreams but do not have frequent nightmares. Analyses of variance are to be used to compare non-dreamers, dreamers without frequent nightmares and frequent nightmare sufferers on severity of injury, IQ measures, memory tests, mood measures and sleep quality. Regression analysis will be used to determine factors predicting nightmares and sleep quality.

Chapter 16

Study 4: Nightmares and traumatic brain injury

Results

Descriptive statistics

Study four assessed brain injury in a sample of participants who had suffered a moderate to severe brain injury. The median length of post traumatic amnesia (PTA) was 4 (less than 2 weeks) and the inter-quartile range was 2-5 (less than one hour to more than 2 weeks). The following table presents the descriptive statistics for this sample.

Table 71 - Mean (standard deviation) and range of injury severity, time since injury, sleep, mood, memory and IQ variables for all participants.

Mean (SD)	Range
10.91 (4.24)	3-15
49.35 (36.13)	9-177
2.94 (1.62)	1-5
2.38 (1.55)	1-5
2.04 (1.47)	1-5
8.29 (5.22)	2-21
1.67 (0.76)	0-3
10.46 (4.98)	0-21
7.15 (4.73)	0-21
1.04 (1.17)	0-3
90.39 (14.16)	66-119
89.38 (12.48)	65-122
78.57 (16.12)	53-109
87.53 (15.65)	50-120
79.00 (16.12)	50-112
82.18 (18.81)	46-117
	10.91 (4.24) 49.35 (36.13) 2.94 (1.62) 2.38 (1.55) 2.04 (1.47) 8.29 (5.22) 1.67 (0.76) 10.46 (4.98) 7.15 (4.73) 1.04 (1.17) 90.39 (14.16) 89.38 (12.48) 78.57 (16.12) 87.53 (15.65) 79.00 (16.12)

The mean Glasgow Coma Scale (GCS) score reflects that most participants had suffered a severe brain injury. The mean time of assessment since injury was 49.35 months. The mean dream frequency category for this sample suggests that on average participants reported one dream a year or less. On average participants reported one nightmare a year or less. The average frequency of night terrors reported was less than one a year. The Pittsburgh Global Sleep Quality Index (GSQI) score is the sum of the seven subscales assessing sleep (sleep quality and sleep efficiency being two of these subscales). A GSQI score of more than five indicates that a person is a poor sleeper (Buysse et al. 1989) and, thus, the mean score here of 8.29 indicates that the average sleep quality of the sample is poor. On average participants report feeling sleepy in the daytime once or twice each week.

Crombie and Taylor (2001) administered the HADS to a non-clinical sample, broadly representative of the general adult UK population, and found a mean Anxiety score of 6.14 (3.76). Thus, the mean anxiety score for this sample of 10.46 (SD= 4.95, range 0-21) is in excess of population norms. A score of 8 or above on the HADS anxiety scale is considered indicative of 'possible' clinical levels of anxiety and a score of 11 or above is considered indicative of a 'probable' diagnosis of anxiety. The mean score is therefore in 'possible' range and thus, clearly anxiety is elevated in this population. Similarly, the mean score on the BDI-PC indicates that the level of depression is elevated in this population. A cut off score of 6 on this scale has been suggested as a cautious cut off point to indicate probable depression on the BDI-PC (Steer et al, 1999). On average participants report experiencing pain less than once a week. Mean scores for verbal IQ and performance IQ are in the low range of average. Memory scores are below average.

Injury severity

The Glasgow Coma Scale and length of posttraumatic amnesia were used as indices of injury severity.

3.8% (n=2) had no PTA

7.7% (n= 4) had less than one hour PTA

13.5% (n = 7) had a PTA of less than 24 hours

9.6% (n = 5) had a PTA of less than 1 week

23.1% (n=12) had a PTA of less than 2 weeks

38.5% (n=20) had a PTA of over 2 weeks

Length of PTA was not available for two participants.

Nightmare prevalence

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The percentage of participants within each reporting category for nightmares were:

48.1% (n= 25) report 'never' having nightmares

9.6% (n=5) report having nightmares 'less than once a year'

11.5% (n=6) report having nightmares 'once a year'.

17.3% (n = 9) report having nightmares 'once a month'

13.5% (n = 7) report having nightmares 'once a week'.

Thus, 30.8% (n =16) report experiencing at least one nightmare per month.

Repetitive nightmares (RNMs)

23.1% (n= 12) reported that they have recently had the same nightmare more than once, whereas 76.9% (n=40) did not. Of those reporting at least one nightmare a month (n =16), 68.8% (n = 11) reported the occurrence of repetitive nightmares.

Nightmare distress

19.2 (n= 10) of the whole sample report that 'having nightmares is a problem' for them, whereas, 80.8% (n= 42) report that nightmares are not a problem. Of those who report monthly nightmares (n =16), 50% state that 'having nightmares is a problem' for them.

Prevalence of night terrors

59.6% (n=31) report 'never' experiencing night terrors
9.6% (n = 5) report night terrors 'less than once a year'
13.5% (n = 7) report night terrors 'once a year'
23.1% (n=12) report night terrors 'once a month'
23.1% (n=12) report night terrors 'once a week'.
Thus, 46.2% (n =24) report at least one night terror a month.

Dream frequency

34.6% (n=18) report that they 'never' dream
5.8% (n = 3) report dreaming 'less than once a year'
13.5% (n=7) report dreaming 'at least once a year'
23.1% (n=12) report dreaming 'at least once a month'
23.1% (n=12) report dreaming 'at least once a week'

Sleep quality

23.1% (n= 12) of participants rated their sleep quality as very good
42.3% (n=22) rated their sleep as rather good
25% (n=13) rated their sleep as fairly bad

9.6% (n= 5) rated their sleep as very bad.

Thus, over half of the sample (65.4%) reported good sleep. However, 32.6% report less than satisfactory sleep quality.

Sleep efficiency

48.1% (n= 25) report a sleep efficiency of over 85%
28.8% (n= 15) report a sleep efficiency of between 75-84%
17.3% (n= 9) report a sleep efficiency of between 65-74%
17.3% (n=9) report a sleep efficiency of less than 65%.

Pittsburgh Global Sleep Quality Index (GSQI)

This is the sum of the seven subscales assessing sleep (sleep quality and sleep efficiency being two of these subscales). A GSQI score of more than five indicates that a person is a poor sleeper (Buysse et al. 1989). 65.4% (N = 34) had a GSQI of over 5 and 34.6% (n=18) had a score of 5 or less.

<u>Insomnia</u>

Insomnia is correctly classified in 96% and 98% of cases using cut off scores of 8 and 9 respectively on the Pittsburgh Global Sleep Quality Index (Fichtenberg et al. 2001).

When a GPSQI score of above 9 was used as a cut off point for the presence of insomnia in this study, 34.6% (n=18) were classified as having insomnia. This suggests indicates sleep disturbance is fairly high in people with brain injury.

Depression

A cut off score of 6 and above was used to indicate probable depression on the BDI-

PC (Steer et al, 1999). According to this conservative estimate, 61.5% (n = 32) of this

sample reported 'probable' levels of depression.

Table 72 shows Shapiro-Wilk test statistics to check for normal distribution of

variables.

	Shapiro-Wilk statistic (p)
Post traumatic amnesia	0.81*(0.01)
Glasgow Coma Scale	0.91 (0.18)
Time since injury (months)	0.88 (0.07)
Performance IQ	0.91 (0.20)
Verbal IQ	0.93 (0.37)
Auditory immediate memory	0.96 (0.70)
Auditory delayed memory	0.95 (0.56)
Visual immediate memory	0.96 (0.71)
Visual delayed memory	0.95 (0.54)
Global Sleep Quality Index	0.91 (0.21)
Anxiety	0.95 (0.59)
Depression	0.99 (1.00)
Pain	0.78* (<0.001)
Hayling A	0.75* (<0.01)
Hayling B	0.66* (<0.01)
Hayling C	0.80* (0.01)
Zoo map	0.86* (0.04)
Nightmare frequency	0.67* (<0.001)
Dream frequency	0.79* (<0.01)
Repetitive nightmares	0.53* (0<0.001)
*n<0.05	

*p<0.05

Table 72 shows that the dream variables, measures of executive functioning, pain and

PTA are non-normally distributed and so non-parametric statistics will be used with

these variables.

Table 73 – Table 73 shows means (standard deviation) of injury severity, time since injury, dream variables, memory, IQ, executive functioning, sleep quality, anxiety and depression of participants reporting no dreams, those reporting dreams but not frequent nightmares, and those reporting frequent nightmares (at least one a month). The table also shows tests of difference for each variable across the three groups.

Mean (standard deviation)	Non-dreamers (n=18)	Dreamers (n = 19)	Frequent Nightmares (n =15)	Test of difference ¹
Post traumatic amnesia	4.41 (1.00)	4.00 (1.18)	2.80 (0.94)	x ² (2) ⁼ 14.51 (p=0.01)*
Glasgow Coma Scale	8.06 (3.94)	10.67 (4.15)	14.43 (1.02)	F (2,42)= 13.08* (p=0.001)
Time since injury (months)	52.89 (49.61)	45.33 (21.73)	49.93 (32.61)	F(2,48)= 1.93 (p=0.82)
Performance IQ	89.12 (14.91)	88.31 (10.38)	91.14 (12.59)	F(2,47)=0.20 (p=0.81)
Verbal IQ	93.41 (17.27)	86.78 (12.42)	91.36 (11.85)	F(2,46)=1.00 (p=0.37)
Auditory immediate memory	86.35 (19.23)	87.31 (14.09)	89.38 (13.54)	F(2,48)=0.14 (p=0.87)
Auditory delayed memory	79.12 (19.89)	83.68 (18.33)	84.00 (19.08)	F(2,48)=0.34 (p=0.72)
Visual immediate memory	74.70 (15.24)	80.47 (16.32)	80.85 (17.26)	F(2,48)= 0.74 (p=0.48)
Visual delayed memory	74.05 (14.87)	82.89 (16.66)	79.77 (17.19)	F(2,48)= 1.35 (p=0.27)
Global Sleep Quality Index	6.22 (4.75)	7.95 (5.06)	11.20 (4.92)	F(2,48)= 4.27* (p =0.02)
Anxiety	7.94 (4.89)	10.42 (4.62)	13.53 (3.94)	F (2,49) = 6.20* (p=0.01)
Depression	5.89 (3.72)	7.42 (4.90)	8.33 (5.50)	F (2,49)= 1.14 (p=0.33)
Pain	0.61 (0.98)	1.10 (1.19)	1.47 (1.24)	x ² (2) ⁼ 4.91 (p=0.09)
Hayling A	4.93 (1.39)	4.92 (1.24)	5.54 (0.93)	x ² (2) ⁼ 2.89 (p=0.23)
Hayling B	5.13 (1.35)	5.08 (1.08)	6.00 (0.89)	x ² (2) ⁼ 4.79 (p=0.09)
Hayling C	5.73 (2.05)	4.33 (2.57)	6.09 (1.81)	x ² (2) ⁼ 2.73 (p=0.25)
Zoo map	2.53 (1.30)	2.47 (1.12)	3.36 (0.92)	x ² (2) ⁼ 4.53 (p=0.10)

¹Chi square tests were used for variables which were shown in table 72 to be non-normally distributed

and one-way analysis of variance was used where variables were normally distributed.

* p<0.05

Table 73 shows that length of PTA is significantly lower in the group reporting frequent nightmares compared to those reporting dreams. Subsequent Mann Whitney tests also shows that length of PTA is significantly lower in the nightmare group compared to non-dreamers (z = -3.59; p<0.001). There is no significant difference between dreamers and non-dreamers in length of PTA (z = -1.21; p =0.23), however, the above table shows a trend for non-dreamers to have a longer length of PTA.

A similar pattern is found with GCS (where high scores indicate milder injury). Tukey HSD paired comparisons revealed that GCS is significantly higher in the nightmare group than in the non-dreaming group (z = -6.37, p<0.0001). The nightmare group also have a significantly higher GCS than dreamers (z = 3.76; p = 0.01). Again, although there is a trend for GCS to be lower in the non-dreaming group compared to the dreaming group, this is not statistically significant (z = -2.60; p = 0.10).

These findings, therefore, suggest that frequent nightmares are associated with milder head injuries compared to more severe head injury, and there is a trend for nondreaming to be associated with more severe injuries.

Table 73 shows that time since injury is not significantly different in the three groups. There is also no significant difference in verbal and performance IQ between the three groups. It was hypothesized that memory would not be significantly lower in nondreamers compared to dreamers, which is confirmed in the above table. Importantly in terms of dream recall there were no significant differences in delay visual or verbal memory between these groups. There were no significant differences in tests of frontal functioning (Hayling and Zoo map) between the three groups. Table 73 also shows that participants in the frequent nightmare group report the worst sleep quality and non-dreamers report the best sleep quality. Tukey HSD paired comparisons revealed that sleep quality is rated as significantly poorer in the nightmare group compared to the non-dreaming group (z = 4.98; p = 0.01). However, sleep quality is not significantly different between the nightmare group and dreamers group (z = 3.25; p = 0.14), nor between the dreaming and non-dreaming groups (z = 1.72; p = 0.54).

Depression was not significantly different across the three groups. Tukey HSD paired comparisons revealed that anxiety is significantly higher in the nightmare group compared to non-dreamers (z = -5.59; p < 0.01). The table shows that there was a trend for anxiety to be higher in the nightmare group compared to the dreaming group (z = 3.11; p = 0.13). No significant difference was found between anxiety in the non-dreaming group and the dreaming group (z = -2.48; p = 0.23)

<u>PTSD</u>

11.54% (n = 6) were considered to have PTSD in their clinical interview with a neuropsychologist. The following table (Table 3) shows the number of participants with PTSD in each group.

Table 74: Table 74 shows the number of participants in each of the three groups,

categorised by presence/absence of PTSD

Non-dreaming group	Dreaming group	Nightmare group
1	1	4
17	18	11
	Non-dreaming group 1 17	1 1

Table 74 shows that the frequency of PTSD is higher in the nightmare group.

However, a chi squared analysis shows that this association is not significant ($x^2 =$

4.48; p = 0.11)

<u>Insomnia</u>

A chi squared analysis was also conducted to determine if the frequency of insomnia differed in the three groups. Table 75 shows the contingency table for this analysis.

Table 75 – Number of participants in each of the three groups, categorised by presence/absence of insomnia

	Non-dreaming group	Dreaming group	Nightmare group
Participants with	3	8	10
insomnia	· ·- ····		
Participants without	15	11	5
insomnia			

Chi squared analysis showed a significant association $(x^2 (2) = 8.53; p = 0.01)$ suggesting that the incidence of insomnia is higher in the nightmare group compared to the non-dreaming group.

Logistical Regression

The above section reported that sleep quality and anxiety are worse in the nightmare group than the other groups, but that head injury is less severe.

A logistic regression was conducted to determine what percentage of the variance in nightmare frequency do these predictors (PTA, anxiety and sleep quality) account for. Depression was included as a predictor in order to control for the influence of this variable.

The regression was run firstly including subjects who report no dreams and then when excluding them from the analysis.

Predictors of monthly nightmares

The regression examines the extent to which anxiety, depression, PTA and sleep quality (GSQI) predict the presence or absence of monthly nightmares. The Omnibus tests of model coefficients or 'goodness of fit' was significant (x^2 (4)= 25.62; p < 0.001) suggesting that this model (with anxiety, depression, sleep quality and PTA as predictors) is a better predictor than the statistical estimation using no predictors (70%). Cox and Snell and Nagelkerke R square tests suggests that between 40% and 57% of the variance in the presence or absence of monthly nightmares are explained by these predictors. The model correctly classifies 78% of overall cases into either having or not having at least one nightmare per month. The model had a negative predictive value of 85.7% and a positive predictive value of 64.28%. This suggests that the model was able to correctly identify 85.7% of the participants that did not have monthly nightmares and 64.28% of those who did have monthly nightmares. The table below demonstrates that PTA and anxiety contribute significantly to the predictive ability of the model, whereas depression does not. Sleep quality is close to significance (p = 0.09).

Table 76 - Wald statistic (and probability value) for predictors of the

presence/absence of monthly nightmares

Predictors	Wald test
Post traumatic amnesia	5.72* (0.02)
Anxiety	4.99* (0.03)
Depression	1.87 (0.17)
Global sleep quality Index ¹	2.88 (0.09)

¹ with the nightmare question omitted

* p<0.05

This regression analysis was re-run with non-dreamers excluded in order to determine how this affected the findings.

The Omnibus tests of model coefficients or 'goodness of fit' was significant (x^2 (4)= 16.61; p < 0.01) suggesting that this model (with anxiety, depression, sleep quality and PTA as predictors) is a better predictor than the statistical estimation using no predictors (54.5%). Cox and Snell and Nagelkerke R square tests suggests that between 39% and 53% of the variance in the presence or absence of monthly nightmares are explained by these predictors. The model correctly classifies 73% of overall cases into either having or not having at least one nightmare per month. The model had a negative predictive value of 83.3% and a positive predictive value of 75%. This suggests that the model was able to correctly identify 83.3% of the participants that did not have monthly nightmares and 75% of those who did have monthly nightmares. The table below demonstrates that PTA and anxiety continue to contribute significantly to the predictive ability of the model, whereas depression and sleep quality do not.

Table 77 - Wald statistic and probability for prediction of monthly nightmares with non-dreamers excluded

Predictors	Wald test
Post traumatic amnesia	5.30* (0.02)
Anxiety	4.05* (0.04)
Depression	1.58 (0.21)
Global sleep quality Index ¹	0.94 (0.33)

with nightmare question omitted

* p<0.05

These analyses show that when non-dreamers are excluded the model explains a little less of the variance in monthly nightmares but generally the differences between the two models is small. In both analyses anxiety and injury severity (PTA) are significant independent predictors of monthly nightmares.

Predictors of repetitive nightmares

A logistical regression was then conducted to determine the extent to which anxiety, depression, PTA and sleep quality (PGSQI) predict the presence or absence of repetitive nightmares. The Omnibus tests of model coefficients or 'goodness of fit' was significant (x^2 (4)= 20.59; p < 0.001), meaning that this model (with anxiety, depression, sleep quality and PTA as predictors) is a better predictor than the statistical estimation using no predictors (76%). Cox and Snell and Nagelkerke R squared tests show that between 34% and 50% of the variance in the presence or absence of repetitive nightmares are explained by these predictors. The model correctly classifies 82% of overall cases into either having or not having repetitive nightmares. The model had a negative predictive value 94.7% and a positive predictive value of 71.43%. This means that the model was able to correctly identify 94.7% of the participants that did not have repetitive nightmares and 71.43% of those who did have repetitive nightmares. The table below demonstrates that PTA, depression and anxiety contribute significantly to the predictive ability of the model. PTA is close to significance (p = 0.06).

Table 78 - Wald statistic and probability for predictors of repetitive nightmares

Predictors	Wald test
Post traumatic amnesia	3.43 (0.06)
Anxiety	5.34* (0.02)
Depression	4.08* (0.04)
Global Sleep quality Index ¹	2.21 (0.14)

with nightmare question omitted

*p<0.05

This regression analysis was re-run with non-dreamers excluded in order to determine how this affected the findings. The Omnibus tests of model coefficients or 'goodness of fit' was significant (x^2 (4)= 14.17; p < 0.01), meaning that this model (with anxiety, depression, sleep quality and PTA as predictors) is a better predictor than the statistical estimation using no predictors (63.6%). Cox and Snell and Nagelkerke R squared tests show that between 35% and 48% of the variance in the presence or absence of repetitive nightmares are explained by these predictors. The model correctly classifies 72.7% of overall cases into either having or not having repetitive nightmares. The model had a negative predictive value of 85.7% and a positive predictive value of 66.7%. This means that the model was able to correctly identify 72.7% of the participants that did not have repetitive nightmares and 66.7% of those who did have repetitive nightmares. The table below demonstrates that anxiety contributes significantly to the predictive ability of the model, whereas PTA and depression are close to significance (both are p =0.058). Again these above two regressions are generally similar whether non-dreamers are excluded or included. However, the first model finds that depression is a significant single predictor of repetitive nightmares, which is not the case when non-dreamers are excluded.

Table 78 - Wald statistic and probability for predictors of repetitive nightmares with non-dreamers excluded

Predictors	Wald test
Post traumatic amnesia	3.60 (0.06)
Anxiety	4.50* (0.03)
Depression	3.59 (0.06)
Global Sleep quality Index ¹	0.55 (0.46)

with nightmare question

*p<0.05

Dream frequency and injury severity

Tests of difference found that length of PTA was shorter in the nightmare and dreaming groups compared to the non-dreaming group. This suggested that frequency of all dreams might be correlated with injury severity; with dream frequency becoming less as injury severity increases. Table 79 assesses the Kendall tau_b correlation between PTA and dream frequency and the magnitude of this correlation after controlling for memory indices, IQ, mood and sleep quality. Table 79 - Kendall tau_b correlation between PTA and dream frequency, and the partial correlation when controlling for memory indices, IQ, mood and sleep quality.

Dream frequency and post traumatic amnesia	-0.38 *(p<0.01)
Controlling for immediate auditory memory	-0.38 *(p<0.01)
Controlling for delayed auditory memory	-0.38 *(p<0.01)
Controlling for immediate visual memory	-0.41 *(p<0.01)
Controlling for auditory delayed memory	-0.41 *(p<0.01)
Controlling for verbal IQ	-0.37 *(p<0.01)
Controlling for Performance IQ	-0.40 *(p<0.01)
Controlling for depression	-0.38 *(p<0.01)
Controlling for anxiety	-0.35 *(p<0.01)
Controlling for Sleep quality	-0.34 *(p<0.01)

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Table 79 shows that there is a significant correlation between PTA and retrospective dream recall frequency, suggesting that as injury severity increases dream frequency decreases. Furthermore, this correlation remains significant after controlling for memory, IQ, mood and sleep quality, and so the decrease in dream frequency with increased severity of injury is not a result of deficits in memory, IQ, mood or sleep quality.

Sleep quality and memory in all participants

Tests of difference in table 73 showed that individuals with frequent nightmares reported worse sleep quality than those in the dreaming group without frequent nightmares and in the non-dreaming group. Thus, the following analysis, in Table 80, explored relationships of sleep quality with severity variables, IQ, memory and well being measures. In addition to sleep quality, this analysis also assessed relationship of daytime dysfunction.

Table 80 - Kendall tau_b correlations of sleep quality and daytime sleepiness with severity of injury, IQ, memory indices and mood.

	Sleep quality (GSQI)	Daytime Sleepiness
Post traumatic amnesia	-0.28* (0.01)	-0.14 (0.28)
Glasgow coma scale	0.26 (0.02)	0.08 (0.52)
Time since injury	-0.12 (0.23)	-0.09 (0.41)
Visual IQ	0.02 (0.90)	-0.20 (0.07)
Performance IQ	-0.05 (0.64)	-0.21 (0.06)
Immediate auditory memory	-0.20 (0.05)	-0.34* (<0.01)
Immediate visual memory	-0.13 (0.21)	-0.34* (<0.01)
Delayed auditory memory	-0.21 (0.04)	-0.37* (<0.001)
Delayed visual memory	-0.08 (0.43)	-0.43* (<0.001)
Depression	0.34* (<0.001)	0.40* (<0.001)
Anxiety	0.22 (0.03)	0.30* (0.01)

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of

significance was set at p = <0.01). All values are two tailed.

Table 80 shows that poorer sleep quality is significantly related to milder head injuries (as measured by PTA). Interestingly, this table shows that daytime dysfunction (which is a subscale of the Pittsburgh sleep quality questionnaire measuring daytime arousal) is significantly negatively related to all memory indices. This suggests that daytime arousal significantly impairs memory. Furthermore, both sleep quality and daytime dysfunctions are positively correlated with depression. This also suggests that poor sleep quality and reduced daytime arousal are related to increased negative waking mood.

Partial correlations were conducted in order to determine if relationships with daytime sleepiness remained significantly related to memory variables after controlling for anxiety, depression. These are shown in Table 81.

Table 81 - Kendall partial tau_b correlations between daytime sleepiness and verbal immediate and delayed memory and visual immediate and delayed memory before and after controlling for performance IQ, verbal IQ, anxiety, depression and post traumatic amnesia.

	Immediate	Delayed	Immediate	Visual
	auditory	auditory	visual	delayed
Daytime sleepiness with	-0.34*	-0.37*	-0.34*	-0.43*
	(p<0.001)	(p<0.001)	(p<0.001)	(p<0.001)
Performance IQ controlled for	-0.29*	-0.32*	-0.30*	-0.39*
	(p<0.001)	(p<0.001)	(p<0.001)	(p<0.001)
Verbal IQ controlled for	-0.28*	-0.32*	-0.30*	-0.39*
	(p<0.001)	(p<0.001)	(p<0.001)	(p<0.001)
Anxiety controlled for	-0.33*	-0.36*	-0.35*	-0.43*
	(p<0.001)	(p<0.001)	(p<0.001)	(p<0.001)
Depression controlled for	-0.31*	-0.34*	-0.33*	-0.42*
	(p<0.001)	(p<0.001)	(p<0.001)	(p<0.001)
Post traumatic amnesia controlled for	-0.35*	-0.39*	-0.40*	-0.49*
	(p<0.001)	(p<0.001)	(p<0.001)	(p<0.001)

* p = or < .01 (in order to reduce the probabilities of type 1 errors as a result of multiple comparisons the level of significance was set at p = <0.01). All values are two tailed.

Table 81 suggests that the negative relationships of daytime dysfunction with measures of immediate and delayed memory are very robust and remain relatively unchanged after controlling for IQ, injury severity and well-being measures.

Chapter 17

Study 4: Nightmares and traumatic brain injury

Discussion

One of the hypotheses of this study was that there would be a higher incidence of nightmares in a population with brain injury compared to the general population. The hypothesis was based on the evidence reviewed concerning the waking stressors and deficiencies in well-being often found in this populations. This chapter will thus begin by discussing the findings regarding well-being deficiencies in the sample studied.

Prevalence of depression

This study found that at an average of 4 years and 11 months post injury, the prevalence of 'probable' depression reported in this study was 61.5% (n=32). This is a high 'probable' prevalence, particularly as a stricter cut off point was adopted in this study following the advice of Steer et al (1999) to minimise the possibility of falsely identifying a patient as depressed. However, it is important to note that a high BDI-PC score only indicates that a detailed psychiatric evaluation may be warranted and so this prevalence rate is only a rough approximation.

High rates of depression have been reported in patients with traumatic brain injury. A number of recent studies have reported rates between 30% and 60% (Busch, 1998; Hibbard et al, 1998; Satz et al, 1998; Bowen et al, 1999; Douglas, 2000; Curran, 2000). Kreutzer et al (2001) found a prevalence rate of 42% in patients with mild to severe brain injuries, an average of 2.5 years post injury. Hibbard et al (1998) assessed 100 patients with traumatic brain injury (TBI) 7.6 years post injury and found a prevalence rate of 48% with major depression. Hoofien et al (2001) found a 45% prevalence for depression (on the SCL-90-R) in their 76 patients with severe TBI at an average 14.1 years post-injury. The mean time since injury in this sample is 4 years and 11 months. However, it is not uncommon for patients with a brain injury to report a high rate of depressive symptoms even though they may not be clinically depressed.

Prevalence of anxiety

73.1% (n =38) of the sample scored 8 or above on the HADS anxiety scale indicating 'possible' clinical anxiety and 53.8% score 11 or above, indicating 'probable' clinical levels of anxiety.

Post Traumatic Stress Disorder: PTSD

Some researchers have argued that PTSD cannot develop after TBI because the coma and organic amnesia associated with TBI prevent the experience of the trauma, and as a result anxiety based on re-experience cannot develop (Bontke et al, 1996; Sbordone, 1992). However, based on interviews with a clinical neuropsychologist, this study reports a prevalence rate of 11.54% (n = 6) for PTSD. It is worth noting that three of the patients diagnosed with PTSD had a PTA duration of less than 24 hours, one of less than a week, one more than two weeks, and one patient had no evidence of PTA. Therefore, this suggest that PTSD can develop after a traumatic head injury with extended periods of PTA. This prevalence rate is in line with the finding of Harvey and Bryant (1998). They recorded a 15% prevalence of acute stress disorder in 48 cases of mild TBI. Williams et al (2002) found a PTSD prevalence of 18% in a representative post-acute community sample of 66 patients with severe TBI. Hibbard et al (1998) examined general psychopathology in a retrospective study on 100 people with TBI (61% had a severe brain injury) and found that PTSD was reported in 19% (2% of these had received their diagnosis before their brain injury).

Of the 6 patients diagnosed with PTSD in the current sample, 4 reported that they had frequent nightmares, and Chi squared analysis revealed a trend for PTSD to occur more frequently in the nightmare group compared to the non-nightmare groups, and when compared specifically to the dreaming group. This offers some support for the findings of Bryant et al (2000a), suggesting that nightmares, emotional reactivity and intrusive memories had the highest positive predictive power of PTSD. This is in contrast to the findings of previous reports in which the symptoms of trauma reexperiencing had only moderate positive predictive power (Foa et al, 1995). However, their sample was not fully representative of severe TBI as participants were not selected at random and survivors with low GCS were excluded.

Sleep Quality

Global sleep quality

Buysse et al (1989) suggested that a score of over 5 on the global scale of the Pittsburgh Sleep Quality Index indicates that a person is a poor sleeper. Of the sample in study four, 65.4% (N = 34) had a GPSQI of over 5. Fichtenberg et al (2001) found that insomnia was correctly classified in 96% and 98% of their sample using a cut of

score of 8 and 9 respectively on the Pittsburgh Global Sleep Quality Index. Using this criterion 34.6% (n=18) of the sample in study four were classified as having insomnia. In contrast, in the United States, approximately 10% of the population suffer from insomnia (Hammond, 1984; Bixler et al,1979; Institute of Medicine, 1979; Karacan et al, 1983; Mellinger et al, 1985; Ford & Kamerow, 1989; Gallup Organisation, 1991). Therefore, the prevalence of 'probable' insomnia found in study four is well in excess of the general population.

As described in the introduction to this chapter the prevalence of sleep disturbances following recent traumatic brain injury has been reported to range from 36% (McLean et al, 1984) to as high as 70% (Keshavan et al, 1981) and a number of studies have suggested that sleep disturbances are among the most universal symptoms of TBI (Beetar et al, 1996; Castriotta & Lai, 2001; Parsons & ver Beek, 1982; Deb, Lyons & Koutzoukis, 1998). The 34.6% prevalence rate for insomnia found in this study is very similar to the 30% prevalence rate found by Mann, Fichtenberg and Putnam (1997) in a prospective study of 50 TBI patients. These findings are also in line with other research demonstrating that sleep complaints are common in patients who have suffered moderate or severe head injuries even years after the trauma (Prigatano et al, 1980; Guilleminault et al, 1983).

The current study also found that sleep quality was significantly related to injury severity (as measured by PTA). It was found that poorer sleep quality was found in those with milder injuries. These findings are in accordance with Clinchot et al (1998). These authors found that individuals with a GCS of less than or equal to 7 (more severe injuries) were less likely to have sleep problems than those with a GCS of over 7 (p = 0.034). Poor sleep quality was significantly related to depression (and anxiety was very close to statistical significance (p = 0.03). This is in line with Parcell et al (2006), who also found that higher scores for anxiety and depression were associated with increased reporting of nighttime awakenings, reports of sleep changes and poorer sleep quality.

Daytime sleepiness/arousal subscale of the PSQI

Fichtenberg et al (2001) examined the concurrent validity of the PSQI item relating to hypersomnia with 47 TBI patients. It was found that the PSQI symptomatic scorers obtained significantly higher levels of daytime sleepiness on the Epworth Sleepiness Scale (ESS; Johns, 1992) compared with asymptomatic scorers (M= 11.4; SD, 5.8 and M= 4.7; SD, 3.4; t=3.4; p<0.01). This suggests that the PSQI scale of daytime sleepiness is a valid measure of daytime sleepiness.

57.7% of participants in study 4 reported problems with daytime sleepiness/arousal at least once or twice a week. Only 3.8% of this sample reported that daytime sleepiness/arousal was not a problem for them in the last month. In contrast, the prevalence of daytime sleepiness in the general population has been estimated at 0.5% and 5% (Benbadis et al, 1999), although reports from questionnaires vary from 0.3% to 13.3% (D'Alessandro et al, 1995). Thus, clearly the estimate of 57.7% from study four is well in excess of these population norms. This finding is in accordance with the observation that daytime sleepiness is among the most universal symptom of TBI (Beetar et al, 1996).

Daytime sleepiness/arousal and memory deficits

There were significant negative correlations between daytime sleepiness / arousal with immediate and delayed visual and verbal memory. These correlations were robust and remained relatively unchanged after controlling for length of PTA, verbal and performance IQ, anxiety and depression (these findings have important clinical implications as they suggest that improving daytime arousal may improve memory).

Prevalence of nightmares

The above findings are of generally increased rates of psychopathology in this population, which is the basis for the hypothesis that they would have a high incidence of nightmares.

Study four indeed found that 30.8% (n =16) of participants reported experiencing at least one nightmare per month, and 13.5% report at least one nightmare per week. In contrast, the American Sleep Disorders Association (1990) estimated that 5% of the adult population experience at least one nightmare per month. Hublin et al (1999) found that 3% had nightmares weekly, whereas 10% had nightmares monthly. Therefore, the prevalence of nightmares in this sample of patients with a brain injury is well in excess of the general population, thus, confirming the hypothesis. In contrast to these findings, Solms (1997) found that only 14.9% of his effective sample reported an increased frequency of non-recurring nightmares. This may be a result of the different samples used. This sample here consisted primarily of patients who had sustained a traumatic brain injury in a road traffic accident, whereas, Solms assessed a large cross-section of the clinical neurological population representing a wide range of neurological conditions (e.g., benign cyst, cerebrovascular disease, idiopathic hydrocephalus, neoplasm, infection, idiopathic epilepsy). Thus, it is possible that the high percentage of nightmares found in study four is specific to a population who sustained a brain injury as a result of trauma.

Solms did, however, note that the prevalence of frequent nightmares was slightly higher in the cerebrally impaired group than in his control group and that it did correlate significantly with limbic system involvement. Solms argues that the clinical usefulness of non-recurring nightmares is questionable, especially in light of the fact that one control patient also reported it. However, although non-recurring nightmares may not be useful in terms of indicating underlying anatomical damage they may be clinically useful as indicators of mood disturbances.

The significance of frequent nightmares.

This study found that individuals with frequent nightmares (at least one a month) had higher levels of waking anxiety than those without frequent nightmares, and those who report non-dreaming. Furthermore, those with frequent nightmares also report lower sleep quality than those without nightmares, and non-dreamers. A regression analysis found that anxiety, sleep quality, post traumatic amnesia and depression accounted for 40-57% of the variance of monthly nightmares. Anxiety and

posttraumatic amnesia were found to contribute significantly to the predictive ability of this model. These findings concerning the relationship with anxiety and nightmares are in line with research using other populations, as reviewed in the first chapter.

Interestingly, this study found that frequent nightmares were significantly associated with milder head injuries (as measured by GCS and duration of PTA). One explanation for this is that as the severity of the injury increases dream recall decreases and, hence, the frequency of nightmares decreases.

Cessation of dreaming

Study four found that 34.6% of patients report that they never dream. In contrast, a large representative survey found that 6.1% of the general population reported that they never dream (Borbely 1984), and Pagel (2003) found that, in a sleep laboratory population, 6.5% reported that they did not dream. Thus, the incidence of non-dreaming found in this study is far in excess of general population and sleep disordered populations.

The prevalence rate of non-dreaming found in this study is remarkably similar to the 34.9% reported by Solms (1997) in his sample of 391 neurological patients. Solms (1997) also assessed a control group referred to him for neuropsychological assessment in which cerebral illness was initially suspected but ultimately excluded. Only one patient in the control group reported cessation of dreaming, and this was a case of hysteria. Solms (1997) found that cessation of dreaming in his patients with focal lesions fell into two anatomical subgroups. The majority of these had parietal lobe lesions, or space occupying lesions in close proximity to the parietal lobe, and a much smaller group that had deep bifrontal lesions, more specifically the lesion was situated in the white matter immediately anterior to the frontal horns of the lateral ventricles; in these cases the damage was invariably bilateral. Functional imaging studies have been remarkably complementary to the lesion data (Braun et al, 1997, 1998; Heiss et al, 1985; Madsen et al, 1991; Maquet et al, 1996; Nofzinger et al, 1997).

However, the current study used a sample of patients typically with diffuse axonal injuries and, hence, anatomical correlates of non- dreaming cannot be determined. However, this study included clinical measures sensitive to partial lobe functioning (Block design) and frontal 'executive functioning' (Hayling and Zoo map). None of these measures were significantly different in the group reporting dreaming compared to non-dreamers. However, our group of non-dreamers would likely have consisted of both patients with parietal and deep bifrontal lesions and so it is perhaps not surprising that differences were not found on these clinical measures.

This study found that the cessation of dream recall was independent of IQ or memory. However, dreamers had significantly lower anxiety and higher sleep quality than dreamers. It could be speculated that non-dreamers have less anxiety because they have more severe injuries and possibly less insight. For example, Talbot and Giroux (2000) found that patients with minor head injuries tend to under-estimate their perceived quality of life when compared to close relative's ratings, whereas more severely injured people overestimate their rating. This finding may relate to the difference in levels of awareness often evident in the two groups. Less severely injured people have a more accurate level of insight into their difficulties.

Non-dreaming and sleep quality

This study found that sleep quality was significantly poorer in dreamers compared to those who report they never dream. This is in line with Partridge (1950) who suggested that cessation of dreaming in leukotomised patients was the result of the unusual depth of sleep that prefrontal leucotomy was supposed to produce. However, these finding contradict those of Solms (1997). He found that non-dreaming patients experienced significantly more disturbed sleep than the dreaming patients. He argued that his finding provided support for Freud's (1900/1953) notion that dreams protect sleep. One possible reason for these differences between these studies is the different neurological populations (as described above) and secondly the different methods used. Solms methods were exclusively reliant upon clinical methods of investigation: He asked patients whether or not their sleep had been affected by their illness. Responses were classified under four heading: sleep was considered to be (a) better, (b) disrupted, (c) unaffected since the onset of illness, or patients were (d) unsure of whether or not there had been any change in the quality of sleep. However, the current study used a standardised measure of sleep quality that had been validated in a population with traumatic brain injury (Fichtenberg et al, 2001), and which did not require thinking back to before the trauma.

Dream frequency and severity of injury

Dream recall frequency was generally low in this sample, but was significantly negatively correlated to severity of injury (as measured by PTA). Thus, as the injury becomes more severe patients report fewer dreams. This correlation remained significant after controlling for immediate and delayed memory, IQ, anxiety, depression and sleep quality. Therefore, it can only be speculated that perhaps more severe injuries cause damage to the lesion sites that Solms' suggests and are necessary to support dreaming (e.g. deep bifrontal lesions).

Is cessation of dreaming a disorder of memory in this study?

It has been proposed that cessation of dreaming following neurological conditions may be a disorder of memory rather than dreaming. This study conducted neuropsychological tests to obtain objective scores of current memory functioning on the assumption that if patients forget their dreams they are also likely to forget other experiences. The results of this study demonstrate that there is no significant difference between immediate verbal and visual memory or delayed visual and visual memory between those reporting dreams and those reporting that they never dream.

These findings are in line with Murri et al (1984), and also with Solms (1997), who found that long-term memory disorder (both anterograde and retrograde) did not distinguish significantly between dreamers and non-dreamers. Also, Bischof and Bassetti's (2004) case study of a 73 old woman reporting cessation of dreaming showed that she had scores on short-term and long-term memory that were in the normal range. The findings from this study are also in line with REM awakenings studies confirming the absence of dreams in those who report cessation of dreaming within the neurological populations (Jus et al, 1973; Michel & Sieroff, 1981; Schanfald, Pearlman & Greenberg, 1985; Bischof & Bassetti 2004). However, the findings from study four are in contrast to Cathala et al (1983), who found strong positive correlations between tests of visual and verbal memory with the frequency and informational richness of dream recall. They also contradict the findings of Arena et al (1984), who reported that the absence of dreaming was related to poor long-term verbal recall (and also, in some cases, aphasia).

These discrepancies could be the result of methodological differences. Solms (1997) highlights that in the clinical literature patients are described as non-dreamers if they themselves report a total cessation of dreaming (as in study 4), whereas, experimental studies (e.g. Arena et al, 1984, and Cathala et al, 1983) classify patients as non-dreamers if they do not report specific dreams during a time-limited experiment (morning recall diaries). Given the low mean rate of dream recall found in this study, experimental definitions of non-dreaming are likely to be inaccurate. When assessing dream frequency this study included the category of dreaming less than once in year in order to distinguish between those who never dreams from those who rarely dream.

This study, therefore, suggests that non-dreaming is a primary phenomenon in this sample, independent of any disturbance of memory, although it is acknowledged that individuals in some studies may have dream cessation due to a memory disorder.

Dreaming and Wais - IQ scores

There are no differences in verbal or performance IQ scores between dreamers and non-dreamers, and so IQ differences cannot account for dream cessation in this study.

Summary/ new findings

This sample of people with traumatic brain injury had a far higher prevalence of frequent nightmares, and reported absence of dreaming, in comparison with the normal population. This is the first study to systematically assess the prevalence of idiopathic nightmares and their relationships with mood and with a standardised measure of sleep quality. It was found that individuals with frequent nightmares had higher levels of waking anxiety and lower sleep quality than those without frequent nightmares. This was the first study to find that individuals with complete lack of dreaming had more severe injuries (as measured by PTA) than those who were reporting dreaming, but that the lack of dreaming was not associated with deficits in IQ scores. The finding that lack of dreaming was not associated with deficits in memory scores confirms previous research. It is therefore possible, as suggested in the work of Solms, that these individuals have had such a severe trauma that dream production has halted, which has the benefit that nightmares cannot occur. They also had the better sleep quality.

Limitations of study four

One of the main limitations of study four is that all patients had diffuse damage as a result of their injury, and therefore conclusions about what locations of the brain subserve dreaming could not be made. This study also could have benefited from using objective measures of sleep quality and sleep architecture, to determine how accurate patients' assessments of sleep quality was, and also to ensure that sleep architecture was normal. This was not feasible in this particular sample as most lived a considerable distance away.

Chapter 18

A case study of the REM sleep of a patient with cessation of dreaming

Study four demonstrated the existence of a negative correlation between the severity of brain injury and frequency of dreaming. Furthermore, the study found a remarkably similar prevalence of non-dreaming to that of Solms (1997). As focal data in study four is lacking it is not possible to speculate about any focal damage that may have led to cessation of dreaming, and hence about the neuroanatomical structures underlying dreaming. However, there is the possibility that this cessation was the result of disruption of normal sleep architecture, as was investigated for sleep apnoea in study 2. The aim of this chapter is thus to present a case study showing the REM sleep profile of an individual who, following a traumatic brain injury reported cessation of dreaming. This will determine if sleep architecture is disrupted, or if the patient continues to show normal REM sleep, the latter possibility being an example of Solms' (2000) contention that REM sleep and dreaming are dissociable states and, that the termination of dreaming can occur when the REM sleep cycle is spared (Benson and Greenberg, 1969; Brown, 1972; Efron, 1968; Jus et al. 1973; Kerr et al 1978; Michel and Sieroff, 1981).

It should be noted that assessing the normality of sleep architecture, and specifically of REM sleep, does not mean that it is assumed here that dreaming is associated solely with REM sleep. It is acknowledged that complex mentation can be obtained from 43% to 50% of NREM awakenings (Foulkes, 1962; Nielsen, 1999; 2000), although REM dreams are more frequent, longer, more bizarre, more visual, more animated and more emotional than NREM reports (Domhoff 1996; Merritt et al 1994; Hobson 1988b; Hobson & Stickgold, 1994a; LaBerge, 1990). Rather, the aim here is to see if the sleep architecture of this patient with cessation of dreaming is greatly different from normality, as this would provide a simpler explanation of the cessation of dreaming than having to invoke a Solms' neurological explanation.

Method

This chapter focuses on a patient who reported a cessation of dreaming following a brain injury. The patient was referred to the researcher by a neuropsychologist who was aware of the researcher's interests. The patient reported to the clinician, during a neuropsychological assessment that he had stopped dreaming following his head injury. The study was approved by the local ethical committee. The patient (AJ) was a 43 year old male who suffered a small bifrontal contusion

following an unprovoked assault. On admission to hospital the patient had a Glasgow Coma Score of 11/15 and duration of PTA was 2.5 weeks.

Measures

<u>REMview</u>. - The REMview (nightcap) was used to record two consecutive nights of sleep. The REMview is a reliable and compact bedside computerised unit that records eyelid and body movements and, based on an algorithmic combination of these two measures, can determine sleep latency and the architecture of REM and non-REM sleep stages (Stickgold et al. 1994). The REMview is worn on the head overnight, and stores raw data which is then downloaded to a PC for analysis. The algorithm software program is in agreement with 85.6% of EEG-determined REM / NREM sleep states on a minute by minute basis, and sleep onset and REM sleep were both predicted within one minute of EEG determined onset (Mamelak and Hobson, 1989).

A dream diary was also kept each morning for 10 days. This consisted of only one question 'did you dream last night?'

The WAIS and the faces (visual memory) and logical memory (verbal memory) subtests of the WMS-III were obtained from the patient's neuropsychological assessment (these measured are described in detail in chapter 15). The neuropsychological assessment was carried out 5 months post injury and the patient's REM sleep and dream recall was assessed 12 months post injury, hence, it is likely that the patients cognitive capacity had increased during this time.

Procedure

The patient was asked detailed questions to ensure that he had not experienced any dream mentation since his injury. AJ wore the REMview monitor for 2 nights whilst keeping the dream diary.

Results

Table 82 shows the neuropsychological test scores for verbal and performance IQ and verbal (indexed by the immediate and delayed conditions of the logical memory subtest) and visual memory (indexed using the immediate and delayed conditions of the faces subtest).

VIQ	67
PIQ	80
Logical memory: Immediate condition	4
Logical memory: Delayed condition	1
Faces Immediate condition	7
Faces Delayed condition	8

The above table shows that the patient's verbal and performance IQ scores and verbal memory scores (as indexed by the logical memory story recall test) are in the impaired range.

However, AJ's memory scores on the visual memory test (faces immediate and delayed) are in the average range (i.e. the average range = 7-8). Thus, it could be argued that visual memory was adequate to recall as least some dreams.

The following table shows the average percentage of REM and NREM sleep across 2

nights' REMview recordings.

Table 83 – REMview assessed % REM, % NREM and total sleep time for AJ.

	AJ
% REM	17.45 %
% NREM	82 %
Total sleep time (mins)	456 mins

The table above demonstrates that AJ continues to show REM sleep despite the reported absence of dreaming. Pages 455 and 456 of this chapter show a summary of AJ's REMview output for the two nights. The REM view uses the following algorithm; periods of head movements are classed as the person being awake, periods of head movements and no eye movements are classed as non-REM sleep and periods of eye movements without head movements as classed as REM sleep (as indexed by the black bars on the sleep state hypnogram).

Discussion

These results indicate that the cessation of dreaming is not due to the disruption of the REM sleep cycle. This suggests that the findings of study four that dream recall decreases as severity of injury increases is not a result of the disruption of the REM sleep cycle. This is in line with previous research demonstrating cessation of dreaming together with an intact REM cycle.

Sleep State	PEB		0 Head Move	Eyelid Move 22:55 End:18/03/2005 08:25 Duration:450 min 39 min	Sessio	Name: . Print Date: . Medications:
 				At least one session for each flagged night must be marked as "accepted" (+).84 min Total REM periods:93 Total Awakenings:1 Sleep Period Time:410 min Total Sleep Time: 410 min	Session 13 of 16	
				I Sleep Period Time:410 min Total Sleep Time: 410 min	Page 1 of 1	454

Session 12 of 16	Print Date: Medications:			453
			Session 12 of 16	Page 1 of 1
	Eyelid Move 22:08	End:17/03/2005 07:17 Duration:549 min 46 min At least one session		wakenings:3 Sleep Period Time:502 min Total Sleep Time: 498 min
	Head Move			
		مساوية والمساوية المساوية والمساوية والمساوية والمساوية		
	-			
	PEB			
	Sleep State			

Full Study

Page 2

Chapter 19

General conclusions

Four main studies were conducted with samples having the following characteristics: people with frequent nightmares; b) people with sleep apnoea; c) fire-fighters and d) people with traumatic brain injury. For brevity, in this section, nightmare means waking criterion nightmares and non-waking criterion nightmares (i.e. unpleasant dreams, irrespective of whether they wake the sleeper).

Major Conclusions

- Nightmare frequency was significantly elevated in patients with sleep apnoea, traumatic brain injury and in fire fighters compared to the general population.
- Nightmare distress did not render nightmare frequency psychopathology correlations negligible in study 1. In study 3, only one fire-fighter reported having a problem with nightmares. Nightmare frequency is thus not an artifact of nightmare distress in these two studies. These findings support the intuitive hypothesis that poor waking well-being is associated with increased frequency of nightmares and that the is not solely a matter of how distressed one is by ones nightmares.
- Within subjects correlations of pre-sleep mood with the presence/absence of a nightmare that night were small in comparison to between subjects analyses.

Nightmares are thus more likely to be caused by general trait or medium-term poor well being rather than by acute poor well-being.

- None of the individual difference variables assessed in this study predispose individuals to have nightmares under conditions of high anxiety or high depression. This was despite the individual difference variables in many cases having significant between subjects correlations with nightmare frequency.
- Ratings of PTSD correlated with nightmare frequency in emergency service workers who have undergone repeated exposure to trauma. PTSD was more common in patients with traumatic brain injury and frequent nightmares than those without nightmares.
- Despite sleep fragmentation there was no indication of cessation of dreaming in the patients with apnoea, but approximately one third of the participants with brain injury had complete cessation of dreaming. For the individuals with brain injury, cessation of dreaming was significantly associated with severity of injury and not with tests of memory, IQ or frontal functioning.
- Severity of apnoea was associated with more neutrally toned dreams rather than more negatively toned dreams. Hence, this study has shown that both head injury and apnoea can affect the process of dreaming.

Other conclusions

 Correlations between nightmare frequency and measures of psychopathology, including anxiety and depression, were similar to those found in the more commonly used student samples.

- Contrary to Beaulieu-Prévost & Zadra (in press), correlations of individual difference variables with prospective dream frequency were significantly higher than with retrospective dream frequency. However, correlations of individual differences with prospective nightmare frequency were smaller than with retrospective nightmare frequency. This may question the accuracy of some individual's retrospective assessment of their nightmare frequency, in that individuals high on psychopathology and other individual difference variables appear to be overestimating their nightmare frequency retrospectively in comparison to low psychopathology individuals.
- In line with Zadra and Donderi (2000) and Blagrove et al (2004) it was found that logs produce a higher estimation of nightmare frequency compared to retrospective measures.
- Blagrove et al (2004) and Blagrove and Haywood (2006) had found that using the waking criterion in defining nightmares did not increase between subjects correlations between nightmare frequency and anxiety. The findings here confirm but also delineate the results of those papers as follows: Using a stricter definition of nightmares, by defining nightmares as having to wake up the sleeper, made little difference to the between subjects correlations with individual difference variables or well-being variables. However, using the criterion that the dream has to be very unpleasant as opposed to just moderately unpleasant to be classed as a nightmare, did cause an increase in between subjects correlations, in comparison to the criterion that the dream was at least moderately unpleasant. The former finding confirms the results of Blagrove et al in questioning the need for the

waking criterion, but the latter finding delineates that conclusion by showing that the definition of nightmares in terms of intensity is important. Given that the current thesis results confirmed Zadra et al's (2006) finding that nightmares that cause waking are more intense in terms of fear than bad dreams, which do not cause the sleeper to wake. It therefore follows that Zadra and Donderi's (2000) results in favour of the use of the waking criterion may be due to a confound with nightmare intensity.

Using either type of stricter definition of nightmares, either by defining
nightmares as having to wake up the sleeper, or as having an emotional tone of
very unpleasant, made little difference to within-subjects correlations with state
pre-sleep mood.

Applied implications of these results for the scientific study of dreaming and nightmares

A higher frequency of nightmares than in the general population has here been found in people with apnoea, with snoring and excessive daytime sleepiness, and with traumatic brain injury. It was also found in fire-fighters. This thesis and previous research suggests that the presence of frequent nightmares can be indicative of underlying psychopathology and thus potentially beneficial to clinicians. Furthermore, these are important findings from the standpoint of advising members of such populations about their likelihood of having nightmares. As imagery rehearsal treatment for nightmares has been validated for traumatized populations (e.g., Krakow et al, 1995), it should therefore be offered to people with traumatic brain injury and people in the emergency services. Work is needed, however, to find out if the therapy works for people with apnoea, given the obvious closeness between precipitating cause and effect in this case.

Theoretical implications of these results for the scientific study of dreaming and nightmares, and suggestions for further studies

This thesis confirmed the results of Zadra et al (2006), that nightmares defined with the waking criterion have more negative emotions than bad dreams. There are two possible explanations for this association. a) Instances of negative emotion in a dream cause an immediate waking, or b) the general higher intensity of nightmares causes awakenings, which are not necessarily at times of high emotional intensity within the nightmare. This distinction is similar to that make in lucid dreaming research, of whether becoming lucid is due to a general brain activation during a dream, or due to specific events, such as seeing an incongruity. The results of Blagrove et al (2006) support explanation a), in that people ascribe their awakening to the events or emotions of the nightmare. But the following study is still required to confirm this:

a) The Fosse method of line-by-line rating of emotion s in dreams should be done for a sample of nightmares that caused awakening, so as to confirm that, just preceding the point of waking, a high rating was given to a negative emotion. The null hypothesis is that highy intense emotions are present, but not immediately prior to waking.

Two further experiments would then add to this detailed exploration of emotions within nightmares:

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- b) Just as Blagrove and Haywood (2006) asked participants to rate how sure they were that a dream /nightmare had awoken them, confidence in ratings of unpleasantness need to be assessed as a first step to assessing the validity of unpleasantness ratings.
- c) Overall ratings from memory of pleasantness or unpleasantness waking events and experiences are highly dependent on the emotions present at the end of the experience and the highest rating of events during the experience. Such memory biases need to be assessed for dreams, as if present they would add to error variance in the overall rating of pleasantness/unpleasantness of a dream.

One of the major hopes of this thesis, that individual difference variables would be found that predisposed individuals to nightmares when state anxiety or depression was high, was not realised. Furthermore, although there was a wide range of within subjects correlations between mood and the presence of nightmares, the mean of these correlations was small. Two experiments are now proposed for the study of the interaction between predisposing traits and state anxiety and depression.

- d) longer diary study, assessing within-subject correlations over one month or more. This may produce a better estimate of the within subjects correlations, which may this have a higher between subjects correlation with the individual difference variables.
- e) It may be that the latter suggestion will fail to find predisposing traits, and that the failure to find them in this thesis accurately reflects a real lack of such predisposing factors. An alternative more complicated longitudinal analysis is

thus needed that allows for the mood on a day to affect not just the following night, but other nights as well.

Implications for theories of nightmare formation: The results here mirror many previous papers on the relationship between nightmares and waking life, in that the association is small but significant. That there is an association supports the notion of dreams being meaningful, and, given the review in chapter 1 on work concerning the memory consolidation function of REM sleep, this supports the notion that nightmares may be involved in the processing of emotionally traumatic information. These differences between patient / emergency service groups and the general population, and significant between subjects correlations, therefore support the proposal of Hartmann (1995, 1998) that nightmares are involved in a therapeutic connecting of emotional memories. However, for the between and within subjects correlations to be so moderately sized does mean that nightmares can occur with little obvious relationship with poor well being. There is thus still support from these findings for Revonsuo's (2000) proposal that we are all predisposed to have nightmares, which would thus occur almost whatever our degree of waking life poor well being, or, in extremis, the view of Hobson et al (2000) that dreaming is greatly disconnected from waking life, in terms of its content, neurochemistry, and logic.

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PSYCHOLOGY DEPARTMENT

ETHICS COMMITTEE

Memo

To:	Samantha Fisher
Copy:	Dr. Mark Blagrove
From:	Professor David Clark, Chair of Department Ethics Committee
Date:	14 th January 2003
Re:	Well-being and personality correlates of frequent nightmare sufferers

Members of the departmental Ethics Committee have now reviewed the above study and agree that it raises no substantive ethical issues, provided the information obtained from the questionnaires is kept absolutely confidential and that no personally identifiable information is entered on computer. You may therefore proceed with your study. A copy of this memo together with a copy of your Ethics Application Form and Risk Assessment Form should be included in your PhD thesis. A copy of this memo has also been passed to your project supervisor.

Appendix 2: Life events scale used in study 1

Please tick in the appropriate column to indicate whether the following events have affected you during the last year.

	YES	NO
You have had a serious illness, injury or accident		
You have cared for someone important to you who has had a serious illness, injury or accident		
You have had a separation or divorce		
You have had a serious problem in a relationship or with family members	Letters	
You have moved house	1	
You have had a serious problems at work		
You have had minor problems at work		
You have had serious financial problems		
You have been the victim of a serious crime		
You have been the victim of a minor crime		
You have had legal difficulties		
You have had a serious disappointment		
You have had continuous worry or stress or major trouble lasting some months	1.11	
There has been a death of a close family member or friend		

Appendix 3: Childhood items

The following statements are concerned with childhood. Please circle the appropriate response according to your experiences.

As a child did you ever feel unloved or unwanted?	Not a	t all	mildly	some	times	Very much
As a child were you ever unreasonably punished?	Not a	t all	mildly	some	times	Very much
Did you experience any types of abuse?	Not a	t all	mildly	some	times	Very much
How happy was your childhood? Very hap	ору	happy	fair	unhappy	Ve	ry unhappy

Appendix 4: retrospective dream questionnaire used in study 1

What is your age?

Are you male or female? M F

- How many dreams do you have each week?
- If you usually do not have one per week, how many do you have per month?_____
- A night terror is a sudden awakening in fear, but where you do not remember a dream.
- How often, if at all, do you experience night terrors?
- A nightmare is a very disturbing dream that you can recall clearly.
- If you have nightmares less than once a month, how many do you have per year?_____
- Now, please think about whether you are ever woken up by the emotions or events in your nightmares.

Please state in what percentage of your nightmares does the emotion or the events of the nightmare wake you up? (Please circle one of the following).

 0%
 20%
 40%
 60%
 80%
 100%

 none of them
 all of them
 all of them

- How long have you been experiencing nightmares?
- Do you ever have the same nightmare being repeated? YES/NO
- How often do you have such a repetitive nightmare?_____
- Are you taking any prescribed medicines that affect or may affect your mood or sleep?_____
- If so, please list them here
- Have you been diagnosed as having any medical disorder of sleep?
- If so, please state any medical diagnosis here_

Please complete this section after answerine the above questions. Please could you indicate how depressed or happy you feel, and how anxious or relaxed you feel? It may help you to look at the two sets of words under each line; they are related to the four main emotions. Please put a cross on the line according to how you feel. of words under each line; they are related to the four main emotions. Please put a cross on the line according to how you feel. very quite neither RELAXED e.g. you may feel series, calm. e.g. you may feel series, calm. e.g. you may feel tenss, nervous, jineary.	1. Did the emotion or events of the dream wake you up? Yes/No 2. How would you rate the overall mood of the dream. very Moderately Slightly Moderately Very (Circle one of the following numbers). upleased upleased upleased mixed pleased pleased	Other comments:-		Please complete the remainder of this page in the morning. If you recall more than one dream, please and the second dream in the space provided overleaf. Please write your dream report below. Please indicate in the right-hand column the appearance of any emotions. Use numbers from 1 to 3 to indicate the intensity of the emotions, where 1 is low, 2 is medium, and 3 is high. Explain 'other' emotions underneath.	e.g. you may feel serene, calm, untroubled, peaceful, composed. jittery, shaky, uneasy.	Please complete this section before you go to sleep each night. Please could you indicate how depressed or happy you feel, and how annous or relaxed you feel? It may help you to look at the two sets of words under each line, they are related to the four main emotions. Please put a cross on the line according to how you feel. of words under each line, they are related to the four main emotions. Please put a cross on the line according to how you feel. very quite very RELAXED ANXIOUS
very quite HAPPY- e.g. You may feel, cheerful, elated playful, light-bearest, joyful.	 Please tick whether you agree, nartly agree, or disagree with the follow 4. After awakening, I felt relieved that it was only a dream. 5. I am very preoccupied with the dream or with working out what the dream is about? 			Day 1 Date:	e.g. You may feel, cheerful,elated, playful, light-hearted, joyful.	ery quite
neither quite very — DEPRESSED e.g. you may feel sad, dejected, lonely, devorbeared, discouraged, glocury.	Please tick whether you agree, partly agree, or disagree with the following statements concerning your dream: 4. After awakening, I felt relieved that it was only a dream. disagree partly agree agree 5. I am very preoccupied with the dream or with working out what the disagree partly agree agree			Other Surprise Love/erotic Jaame Sadness Anxiety/feat Anset Anget	e.g. you may reet sad, dejected, ioneiy, downhearted, discouraged, gloomy.	neither quite very DEPRESSED

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Instructions to participants.

Each night and each morning for 14 days we would like you to quickly assess how depressed or happy and how anxious or calm you feel on the 2 scales provided and answer a few additional questions. For the rating scales you will be presented with a line running between relaxed and anxious and one between happy and depressed. You will be asked to put a cross on each line according to how you feel. For example, if you felt quite relaxed you would place the cross as shown below.

very	quite	neither	quite	very
RELAXED	X	10.000		ANXIOUS

We would also like you to record your dreams for 14 days. Please do not skip days at all and please complete the diary for 14 nights in a row. Thus, each day you will be asked to provide a written account in the dream diary provided of all the dreams you can recall from the previous night. This should take only about 5-10 mins each day.

When you are recalling each dream we would like you to indicate any emotions or moods that are present and rate on a scale of 1 to 3 how intensely the particular emotion or mood was experienced (where 1 is low, 2 is medium and 3 is high). Please write the content of the dream in the main box of the dream diary. In the right-hand column you will see listed 7 emotions. Please put a number in the appropriate column (1,2 and 3) to indicate the appearance of this emotion and its intensity. If you wish to report the experience of any emotions that are not present in the list please indicate this in the column marked 'other' or in the space for comments below. Please report the experience of any emotion; regardless of its duration or how related it seems to the dream content. For example,

"In my first dream I recall from last night I remember I was sleeping in my bedroom when H came in. I was irritated because I couldn't sleep and considered getting up (IRRATATION, 1). Then I flew out the door on a cookie plate or something. I flew around near the house. O and H were watching and they envied me' (JOY/ELATION, 3)."

After <u>each</u> dream report we would like you to answer the questions provided on the dream diary sheets.

N.B. We are interested in ALL your dreams not just your nightmares so please record as much information as possible regardless of how uneventful and unimportant it may seem. Please try and provide as honest a report as possible and not just the dramatic dream elements. We understand that scenes of a personal or embarrassing nature are a common feature of everyone's dreams. Therefore, all dream reports will be treated with the utmost confidentiality and sensitivity. Any information you provide will be available only to the two researchers undertaking this study.

After the two-week recording period please return the dream diaries to us in the return envelope.

If you have any problems or worries with the study please contact us at any time on the following details:-

Mark Blagrove 01792 295586 or 07976136193 Samantha Fisher 01792 205678 ext 4616 or 01685 376980 Email <u>160133@swan.ac.uk</u>

Summary

Before bed, fill in the 2 scales indicating your mood that day by putting a cross on each line according to how you feel. The following morning record the date, then the dream content including any emotions/moods present and their intensities by giving them a rating of either 1, 2 or 3. Finally, please answer the questions on the bottom of the diary page.

	APS	CEQ	STB	EPQ-	EPQ-	GHQ-	BQ-	STAI	STAI	GSI	PST	PSD	LE	ChE
12	age			N	L	12	18	-S	-T			1.1		
APS	-	0.39*	0.42*	0.57*	-0.07	0.24	0.40	0.37*	0.46	0.42	0.37	0.32	0.21	0.06
	AF CO	(0.01)	(<.01)	(<.01)	(0.57)	(0.05)	(0.01)	(0.05)	(0.01)	(<.01)	(<.01)	(0.01)	(0.08)	(0.62)
CEQ	0.39	-	0.43	0.17	-0.05	0.25	0.64	0.16	0.31	0.35	0.36	0.22	0.28	0.26
	(<.01)		(<.01)	(0.16)	(0.71)	(0.04)	(<.01)	(0.40)	(0.02)	(<.01)	(<.01)	(0.07)	(0.02)	(0.04)
STB	0.42	0.43	-	0.53	-0.23	0.51	0.51	0.64	0.66	0.53	0.54	0.43	0.34	0.07
	(<.01)	(<.01)		(<.01)	(0.06)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.57)
EPQ-	0.57	0.17	0.53	-	-0.07	0.39	0.28	0.72	0.68	0.64	0.60	0.55	0.29	0.08
N	(<.01)	(0.16)	(<.01)		(0.55)	(<.01)	(0.02)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.53)
EPQ-	-0.07	-0.05	-0.23	-0.07		-0.02	-0.02	-0.01	0.05	0.15	0.15	0.17	0.02	0.08
L	(0.57)	(0.71)	(0.06)	(0.55)		(0.87)	(0.89)	(0.94)	(0.73)	(0.22)	(0.21)	(0.18)	(0.02)	(0.51)
GHQ	0.24	0.25	0.51	0.39	-0.02		0.40	0.57	0.55	0.54	0.53	0.59	0.46	0.25
-12	(0.47)	(0.04)	(<.01)	(<.01)	(0.86)		(<.01)	(0.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.04)
BQ-	0.40	0.64	0.51	0.28	-0.02	0.40	-	0.43	0.40	0.54	0.54	0.44	0.40	0.22
18	(<.01)	(<.01)	(<.01)	(0.02)	(0.89)	(<.01)		(0.02)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(0.07)
STAI	0.37	0.16	0.64	0.72	-0.01	0.57	0.43	-	0.87	0.87	0.86	0.63	0.48	0.19
-S	(0.05)	(0.40)	(<.01)	(<.01)	(0.94)	(<.01)	(0.02)		(<.01)	(<.01)	(<.01)	(<.01)	(0.01)	(0.33)
STAI	0.46	0.31	0.66	0.68	0.05	0.55	0.40	0.87	-	0.67	0.62	0.65	0.44	0.31
-T	(<.01)	(0.25)	(<.01)	(<.01)	(0.73)	(<.01)	(<.01)	(<.01)		(<.01)	(<.01)	(<.01)	(<.01)	(0.03)
GSI	0.42	0.35	0.53	0.64	0.15	0.54	0.54	0.87	0.67		0.91	0.75	0.49	0.18
	(0.01)	(<.01)	(<.01)	(<.01)	(0.22)	(<.01)	(<.01)	(<.01)	(<.01)		(<.01)	(<.01)	(<.01)	(1.15)
PST	0.37	0.36	0.54	0.60	0.15	0.53	0.54	0.86	0.62	0.91	-	0.64	0.51	0.16
16	(<.01)	(0.03)	(<.01)	(<.01)	(0.21)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)		(<.01)	(<.01)	(0.20)
PSDI	0.32	0.22	0.43	0.55	0.17	0.59	0.44	0.63	0.65	0.75	0.64	-	0.42	0.33
	(0.01)	(0.07)	(<.01)	(<.01)	(0.18)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)	(<.01)		(<.01)	(0.01)
LE	0.21	0.28	0.34	0.29	0.02	0.46	0.40	0.49	0.44	0.49	0.51	0.42	-	0.12
100	(0.08)	(0.02)	(<.01)	(0.02)	(0.90)	(<.01)	(<.01)	(0.01)	(<.01)	(<.01)	(<.01)	(<.01)		(0.34)
Child	0.06	0.26	0.07	0.08	0.08	0.25	0.22	0.20	0.31	0.18	0.16	0.33	0.12	-
	(0.62)	(0.04)	(0.57)	(0.53)	(0.51)	(0.04)	(0.07)	(0.33)	(0.03)	(0.15)	(0.20)	(0.01)	(0.34)	

Appendix Table 7: Intercorrelations between variables well-being and individual difference variables.

APS = Arousal predisposition scale; CEQ = creative experience questionnaire; STB; STA-borderline personality scale; EPQ-N =PQ-R Neuroticism scale; EPQ-L =EPQ-R Lie scale; GHQ-12 = General health questionnaire -12; BQ-18 = Boundary Questionnaire-18; STAI-S = state anxiety ; STAI-T =; trait anxiety; GSI =SCL-90-R, Global symptom index; PST = SCL-90-R, Positive symptom total; PSDI = SCL-90-R, Positive symptom distress index; LE =sum of life event items; Child = Sum of childhood items

* p < 0.01

Appendix Table 8 - Spearman's rho correlations between frequency estimates and age.

Age and frequency of dream types	Spearman's rho coefficient
Retrospective dream frequency	r = -0.08 (0.52)
Retrospective unpleasant dream frequency	r = -0.11 (0.37)
Retrospective nightmare frequency	r = -0.07 (0.55)
Retrospective bad dream frequency	r = -0.26 (0.03)
Log dream frequency	r = 0.21 (0.11)
Log unpleasant dream frequency	r = 0.10 (0.44)
Log nightmare frequency	r = 0.21 (0.12)
Log bad dream frequency	r = -0.18 (0.20)
Log extreme nightmares	r = 0.19 (0.16)
Log extreme unpleasant dreams	r = 0.11 (0.41)
Log extreme bad dreams	r = 0.29 (0.21)

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Appendix Table 9 - mean	<u>s (standard d</u>	eviations) and	Wilcoxon test statistics for						
dream type frequencies between males and females.									
			,						

Dream type estimations	Mean (SD) per month -male	Mean (SD) per month -female	Mann Whitney test statistic
Retrospective dream frequency	32.41 (39.91)	31.89 (20.19)	z =-1.10 ;p = 0.28
Retrospective nightmare frequency	3.09 (3.70)	2.72 (3.17)	z = -0.58; p = 0.56
Retrospective unpleasant dream frequency	6.50 (7.13)	4.81 (4.72)	z =-0.08; p = 0.93
Log dream frequency	9.50 (4.85)	12.86 (4.62)	z = -2.32; p = 0.02
Log nightmare frequency	2.42 (3.17)	2.54 (2.38)	z = -0.58; p = 0.56
Log unpleasant dream frequency	3.33 (3.92)	4.02 (2.82)	z = -1.11; p = 0.26
Log extreme nightmare frequency	1.67 (0.58)	1.52 (1.35)	z = -0.64; p = 0.52
Log extreme unpleasant dream frequency	2.08 (2.71)	2.07 (0.58)	z = -0.85; p = 0.39
Log extreme bad dream frequency	1.67 (0.58)	1.23 (0.57)	z = -1.46; p = 0.14

Appendix Table 10 – Sex differences in personality and psychopathology/well-being measures

Group Statistics

nightmare distress male female 13 33.6154 11.83595 3.28270 arousal scale male 14 35.5556 8.57743 1.16724 arousal scale male 14 32.7143 10.94341 2.92475 creative experiences male 13 9.1538 4.12000 1.14268 quest female 54 10.1296 4.53101 6.1659 borderline personality male 14 6.6429 4.44811 1.18881 female 54 7.9074 2.96692 .40375 Eysenck Neuroticism male 14 4.2143 2.96592 .79268 General Health male 14 5.0714 4.81127 1.28587 Questionnaire female 54 3.9074 2.66542 .38272 General Health male 14 5.0714 4.81127 1.28587 Questionnaire female 53 34.6038 10.09063 1.38605 State Anxiety				 .		Std. Error
female 54 35.5556 8.57743 1.16724 arousal scale male 14 32.7143 10.94341 2.92475 creative experiences male 13 9.1538 4.12000 1.14268 quest female 54 10.1296 4.53101 6.6159 borderline personality male 14 6.6429 4.44811 1.18881 female 54 8.4444 4.14213 5.6567 Eysenck Neuroticism male 14 6.67143 4.19576 1.12136 Scale female 54 7.9074 2.96692 .79268 female 54 3.9074 2.66542 .36272 General Health male 14 4.2143 2.96592 .79268 female 54 3.9074 2.66542 .36272 General Health male 14 34.9266 10.9063 1.38605 Questionnaire female 54 3.5926 3.99301 .54338		gender	N	Mean	Std. Deviation	Mean
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Instruct Instruct Instruct Instruct Instruct female 54 37.3148 9.1252 1.24155 creative experiences male 13 9.1538 4.12000 1.14268 quest female 54 10.1296 4.53101 61659 borderline personality male 14 6.6429 4.44811 1.18881 female 54 8.4444 4.14213 .56367 Eysenck Neuroticism male 14 6.7143 4.19576 1.12136 Scale female 54 7.9074 2.96692 .40375 Eysenck Lie Scale male 14 4.2143 2.96592 .79268 General Health male 14 3.9074 2.66542 .36272 Quest female 54 3.5926 3.99301 .54338 Hartmann Boundary male 14 34.9286 10.68042 2.85446 Quest female 21 47.3810 15.32	· · · · · · · · · · · · · · · · · · ·		54			1.16724
creative experiences quest male 13 9.1538 4.12000 1.14268 quest female 54 10.1296 4.53101 .61659 borderline personality male 14 6.6429 4.44811 1.18881 female 54 8.4444 4.14213 .56367 Eysenck Neuroticism male 14 6.7143 4.19576 1.12136 Scale female 54 7.9074 2.96692 .40375 Eysenck Lie Scale male 14 4.2143 2.96592 .79268 female 54 3.9074 2.66542 .36272 General Health male 14 5.0714 4.81127 1.28587 Questionnaire female 54 3.5926 3.99301 .54338 Hartmann Boundary male 14 34.9286 10.068042 2.85446 Quest female 21 47.3810 15.32800 3.34484 Trait Anxiety male 13	arousal scale		14	32.7143	10.94341	2.92475
quest female 54 10.1296 4.53101 6.61659 borderline personality male 14 6.6429 4.44811 1.18881 female 54 8.4444 4.14213 .56367 Eysenck Neuroticism male 14 6.7143 4.19576 1.12136 Scale female 54 7.9074 2.96692 .40375 Eysenck Lie Scale male 14 4.2143 2.96592 .79268 female 54 3.9074 2.66542 .36272 General Health male 14 5.0714 4.81127 1.28587 Questionnaire female 54 3.5926 3.99301 .54338 Hartmann Boundary male 14 34.9286 10.068042 2.85446 Quest female 21 47.3810 15.32800 3.34464 Trait Anxiety male 13 46.0769 13.44409 3.72872 female 54 1.9000 13.31007 </td <td></td> <td>female</td> <td>54</td> <td>37.3148</td> <td>9.12352</td> <td>1.24155</td>		female	54	37.3148	9.12352	1.24155
borderline personality male female 14 6.6429 4.44811 1.18881 female 54 8.4444 4.14213 .56367 Eysenck Neuroticism male 14 6.7143 4.19576 1.12136 Scale female 54 7.9074 2.96692 .40375 Eysenck Lie Scale male 14 4.2143 2.96592 .79268 General Health male 14 5.0714 4.81127 1.28587 Questionnaire female 54 3.9074 2.66542 .36272 General Health male 14 5.0714 4.81127 1.28587 Questionnaire female 53 34.6038 10.09063 1.38605 State Anxiety male 8 49.5000 16.14223 5.70714 female 21 47.3810 15.32800 3.34484 Trait Anxiety male 13 46.0769 13.44409 3.72872 female 54 1.0663	•	male	13	9.1538	4.12000	1.14268
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female 54 3.9074 2.66542 3.6272 General Health male 14 5.0714 4.81127 1.28587 Questionnaire female 54 3.5926 3.99301 .54338 Hartmann Boundary male 14 34.9286 10.68042 2.85446 Quest female 53 34.6038 10.09063 1.38605 State Anxiety male 8 49.5000 16.14223 5.70714 female 21 47.3810 15.32800 3.34484 Trait Anxiety male 13 46.0769 13.44409 3.72872 female 39 51.0000 13.31007 2.13132 SCL-Depression male 13 1.5638 1.22012 .33840 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.680 .74881 .10190 P	Scale	female	[′] 54	7.9074	2.96692	.40375
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Hartmann Boundary male 14 34.9286 10.68042 2.85446 Quest female 53 34.6038 10.09063 1.38605 State Anxiety male 8 49.5000 16.14223 5.70714 female 21 47.3810 15.32800 3.34484 Trait Anxiety male 13 46.0769 13.44409 3.72872 female 39 51.0000 13.31007 2.13132 SCL-Depression male 13 1.5638 1.22012 .33840 female 54 1.4063 .92285 .12558 SCL-Depression male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 1.9077 .76894 .21327 <	General Health	male	14	5.0714	4.81127	1.28587
Quest female 53 34.6038 10.09063 1.38605 State Anxiety male 8 49.5000 16.14223 5.70714 female 21 47.3810 15.32800 3.34484 Trait Anxiety male 13 46.0769 13.4409 3.72872 female 39 51.0000 13.31007 2.13132 SCL-Depression male 13 1.5638 1.22012 .33840 female 54 1.4063 .92285 .12558 SCL-Anxiety male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 48.6667 20.52702 2.79337 Positive Symptom	Questionnaire	female	54	3.5926	3.99301	.54338
State Anxiety male 8 49.5000 16.14223 5.70714 female 21 47.3810 15.32800 3.34484 Trait Anxiety male 13 46.0769 13.4409 3.72872 female 39 51.0000 13.31007 2.13132 SCL-Depression male 13 1.5638 1.22012 .33840 female 54 1.4063 .92285 .12558 SCL-Depression male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 <	Hartmann Boundary	male	14	34.9286	10.68042	2.85446
female 21 47.3810 15.32800 3.34484 Trait Anxiety male 13 46.0769 13.44409 3.72872 female 39 51.0000 13.31007 2.13132 SCL-Depression male 13 1.5638 1.22012 .33840 female 54 1.4063 .92285 .12558 SCL-Anxiety male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 sum of 4 childhood items	Quest	female	53	34.6038	10.09063	1.38605
Trait Anxiety male 13 46.0769 13.44409 3.72872 female 39 51.0000 13.31007 2.13132 SCL-Depression male 13 1.5638 1.22012 .33840 female 54 1.4063 .92285 .12558 SCL-Anxiety male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14	State Anxiety	male	8	49.5000	16.14223	5.70714
female 39 51.0000 13.31007 2.13132 SCL-Depression male 13 1.5638 1.22012 .33840 female 54 1.4063 .92285 .12558 SCL-Anxiety male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 1.9077 .76894 .21327 Positive Symptom Total male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.0741 3.27495 .87527 sum of 4 childhood items male 14 3.4286 3.08132 .82352		female	21	47.3810	15.32800	3.34484
SCL-Depression male female 13 1.5638 1.22012 .33840 SCL-Anxiety male 54 1.4063 .92285 .12558 SCL-Anxiety male 13 1.1615 .95703 .26543 SCL-Anxiety male 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 48.6667 20.52702 2.79337 Positive Symptom Total male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 sum of 4 childhood items male 14 3.4286 3.08132 .82352	Trait Anxiety	male	13	46.0769	13.44409	3.72872
female 54 1.4063 .92285 .12558 SCL-Anxiety male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 1.9077 .76894 .21327 Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 sum of 4 childhood items male 14 3.4286 3.08132 .82352		female	39	51.0000	13.31007	2.13132
SCL-Anxiety male 13 1.1615 .95703 .26543 female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 .11800 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 48.6667 20.52702 2.79337 Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 sum of 4 childhood items male 14 3.4286 3.08132 .82352	SCL-Depression	male	13	1.5638	1.22012	.33840
female 54 .9611 .94578 .12870 Global Severity Idex male 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 48.6667 20.52702 2.79337 Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 sum of 4 childhood items male 14 3.4286 3.08132 .82352		female	54	1.4063	.92285	.12558
Global Severity Idex male 13 1.2608 .84667 .23483 female 13 1.2608 .84667 .23483 female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 48.6667 20.52702 2.79337 Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 sum of 4 childhood items male 14 3.4286 3.08132 .82352	SCL-Anxiety	male	13	1.1615	.95703	.26543
female 54 1.1680 .74881 .10190 Positive Symptom Total male 13 48.3846 24.98179 6.92870 female 54 48.6667 20.52702 2.79337 Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 female 54 3.0741 2.27249 .30925 sum of 4 childhood items male 14 3.4286 3.08132 .82352		female	54	.9611	.94578	.12870
Positive Symptom Total male female 13 48.3846 24.98179 6.92870 Positive Symptom female 54 48.6667 20.52702 2.79337 Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 sum of 4 childhood items male 14 3.4286 3.08132 .82352	Global Severity Idex	male	13	1.2608	.84667	.23483
female 54 48.6667 20.52702 2.79337 Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 female 54 3.0741 2.27249 .30925 sum of 4 childhood items male 14 3.4286 3.08132 .82352		female	54	1.1680	.74881	.10190
Positive Symptom male 13 1.9077 .76894 .21327 Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 female 54 3.0741 2.27249 .30925 sum of 4 childhood items male 14 3.4286 3.08132 .82352	Positive Symptom Total	male	13	48.3846	24.98179	6.92870
Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 female 54 3.0741 2.27249 .30925 sum of 4 childhood items male 14 3.4286 3.08132 .82352		female	54	48.6667	20.52702	2.79337
Distress Index female 54 1.9078 .57488 .07823 lifeevent14 male 14 3.5714 3.27495 .87527 female 54 3.0741 2.27249 .30925 sum of 4 childhood items male 14 3.4286 3.08132 .82352	Positive Symptom	male				
lifeevent14 male 14 3.5714 3.27495 .87527 female 54 3.0741 2.27249 .30925 sum of 4 childhood items male 14 3.4286 3.08132 .82352	Distress Index	female	54		.57488	.07823
female 54 3.0741 2.27249 .30925 sum of 4 childhood items male 14 3.4286 3.08132 .82352	lifeevent14	male	14	3.5714		
sum of 4 childhood items male 14 3.4286 3.08132 .82352		female	54			
	sum of 4 childhood items	male	14			
		female	54	4,1296	3.33674	.45407

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differences in personality and well-being.

Independent Samples Test

		Levene's	Test for Variances			t-test for	Equality of	Means		
		-quality of	Vanances					NICALIS	95% Co	
							Mean	Std. Error	Interva Differ	l of the rence
	E li inter	F	Sig.	t	df	Sig. (2-tailed)		Difference	Lower	Upper
nightmare distress	Equal variance assumed	4.250	.043	678	65	.500	-1.94017	2.86250	7.65697	3.77663
	Equal variance not assumed			557	15.171	.586	-1.94017	3.48405	-9.35895	5.47860
arousal scale	Equal variance assumed	.514	.476	-1.613	66	.112	-4.60053	2.85203	0.29479	1.09374
	Equal variance not assumed			-1.448	17.964	.165	-4.60053	3.17736	1.27687	2.07581
creative experiences quest	Equal variance assumed	.448	.506	709	65	.481	97578	1.37723	-3.72631	1.77474
	Equal variance not assumed			752	19.629	.461	97578	1.29842	-3.68754	1.73597
borderline personality	Equal variance assumed	.555	.459	-1.429	66	.158	-1.80159	1.26088	4.31901	.71584
	Equal variance not assumed			-1.369	19.263	.187	-1.80159	1.31567	4.55277	.94960
Eysenck Neuroticism Scale	assumed	5.322	.024	-1.226	66	.225	-1.19312	.97350	-3.13678	.75054
	Equal variance not assumed			-1.001	16.521	.331	-1.19312	1.19183	-3.71324	1.32699
Eysenck Lie Scale	Equal variance assumed	.126	.724	.375	66	.709	.30688	.81793	1.32617	1.93993
	Equal variance not assumed			.352	18.812	.729	.30688	.87172	-1.51890	2.13265
General Health Questionnaire	Equal variance assumed	1.957	.167	1.183	66	.241	1.47884	1.24970	-1.01628	3.97395
	Equal variance not assumed			1.059	17.917	.304	1.47884	1.39596	1.45495	4.41262
Hartmann Boundary Quest	Equal variance assumed	.021	.885	.106	65	.916	.32480	3.06844	-5.80329	6.45289
	Equal variance not assumed			.102	19.581	.920	.32480	3.17319	6.30344	6.95304
State Anxiety	Equal variance assumed	.006	.938	.328	27	.745	2.11905	6.45780	1.13126	5.36936
	Equal variance not assumed			.320	12.134	.754	2.11905	6.61509	2.27640	6.51449
Trait Anxiety	Equal variance assumed	.042	.839	-1.152	50	.255	-4.92308	4.27297	3.50560	3.65944
	Equal variance not assumed			-1.146	20.433	.265	-4.92308	4.29486	3.86984	4.02369
SCL-Depression	Equal variance assumed	3.199	.078	.518	65	.606	.15755	.30415	44988	.76498
	Equal variance not assumed			.436	15.467	.669	.15755	.36095	60978	.92488
SCL-Anxiety	Equal variance assumed	.109	.743	.684	65	.496	.20043	.29283	38439	.78525
	Equal variance not assumed			.679	18.080	.505	.20043	.29499	41913	.81998
Global Severity Idex	Equal variance assumed	.563	.456	.391	65	.697	.09281	.23721	38093	.56654
	Equal variance not assumed			.363	16.810	.721	.09281	.25598	44773	.63335
Positive Symptom To	assumed	.723	.398	043	65	.966	28205	6.61719	3.49750	2.93340
	Equal variance not assumed			038	16.121	.970	28205	7.47060	6.10932	5.54521
Positive Symptom Distress Index	Equal variance assumed	3.226	.077	.000	65	1.000	00009	.19010	37974	.37957
	Equal variance not assumed			.000	15.384	1.000	00009	.22716	48322	.48305
lifeevent14	Equal variance assumed	6.867	.011	.663	66	.510	.49735	.75035	1.00078	1.99548
	Equal variance not assumed			.536	16.386	.599	.49735	.92829	1.46678	2.46149
sum of 4 childhood ite	Equal variance assumed	.114	.737	711	66	.480	70106	.98611	2.66989	1.26777
	Equal variance not assumed			745	21.616	.464	70106	.94040	-2.65335	1.25123
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personality and psychopathology/well being measures,

Correlations

nightmare distress	Pearson Correlation	age 046
mynunare usuess		046 .714
	Sig. (2-tailed)	
	N Decrease Correlation	66
arousal scale	Pearson Correlation	126
	Sig. (2-tailed)	.311
	N	67
creative experiences	Pearson Correlation	228
quest	Sig. (2-tailed)	.065
	N	66
borderline personality	Pearson Correlation	216
	Sig. (2-tailed)	.079
	N	67
Eysenck Neuroticism	Pearson Correlation	059
Scale	Sig. (2-tailed)	.637
	N	67
General Health	Pearson Correlation	082
Questionnaire	Sig. (2-tailed)	.509
	N	.509 67
Hartmann Boundary	Pearson Correlation	353**
Quest		353 .004
	Sig. (2-tailed) N	
		66
State Anxiety	Pearson Correlation	061
	Sig. (2-tailed)	.754
	N	29
Trait Anxiety	Pearson Correlation	082
	Sig. (2-tailed)	.566
	<u>N</u>	51
SCL-Depression	Pearson Correlation	091
	Sig. (2-tailed)	.466
	<u>N</u>	66
SCL-Anxiety	Pearson Correlation	046
	Sig. (2-tailed)	.714
	N	66
Global Severity Idex	Pearson Correlation	081
	Sig. (2-tailed)	.515
	N	66
Positive Symptom Total	Pearson Correlation	153
	Sig. (2-tailed)	.219
	N	66
Positive Symptom	Pearson Correlation	.035
Distress Index	Sig. (2-tailed)	.780
	N	66
lifeevent14	Pearson Correlation	320**
	Sig. (2-tailed)	.008
}	N	67
sum of 4 childhood items	Pearson Correlation	.002
Sum of 4 Ginundou items	Sig. (2-tailed)	
	•••	.985
L	N	67

**. Correlation is significant at the 0.01 level (2-tailed).

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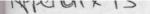
Sig. (2-tailed) N .260 .990 .226 N 48 41 48 reral Health Pearson Correlation .216 .097 070 estionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 st Sig. (2-tailed) .617 .442 .258 N 47 40 47 Ite Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22							
ghtmares each year Pearson Correlation Sig. (2-tailed) 208 229 151 Im Pearson Correlation Sig. (2-tailed) .156 .151 .304 Im Pearson Correlation Sig. (2-tailed) .315 .325 .442 N 48 41 48 mare distress Pearson Correlation Sig. (2-tailed) .189 .253 .297* N 48 41 48 mare distress Pearson Correlation Sig. (2-tailed) .015 .003 .031 N 48 41 48 .201 .040 .040 N 48 41 48 .041 .040 .041 .201 N 48 41 48 .042 .014 .201 Itrine personality Pearson Correlation .042 .014 .201 N 48 41 48 .035 .755 N 48 41 48 .046 .036 .046 Itrine personalit		5-0-	5-7-1-1		ws nights of unpld and presleep		
Sig. (2-tailed) .156 .151 .304 N 48 41 48 Im Pearson Correlation 148 158 113 Sig. (2-tailed) .315 .325 .442 N 48 41 48 mare distress Pearson Correlation 189 253 297* Sig. (2-tailed) .198 .110 .040 N 48 41 48 aal scale Pearson Correlation 015 .003 .031 Sig. (2-tailed) .920 .984 .835 . N 48 41 48 . eal scale Pearson Correlation .042 014 2011 at scale Pearson Correlation .042 014 2011 at scale Pearson Correlation .044 030 046 M 48 41 48							
N 48 41 48 Im Pearson Correlation Sig. (2-tailed) 148 158 113 M 48 41 48 mare distress Pearson Correlation Sig. (2-tailed) 189 253 297* Sig. (2-tailed) .198 .110 .040 N 48 41 48 mare distress Pearson Correlation 015 .003 .031 Sig. (2-tailed) .920 .984 .835 N 48 41 48 twe experiences Pearson Correlation .042 .014 201 A 8 .11 48 .48 .44 twe experiences Pearson Correlation .024 .030 046 Sig. (2-tailed) .777 .933 .170 N 48 41 48 twe experiences Pearson Correlation .044 .41 sig. (2-tailed) .764 .497 .807	191					Concerning and an and a second second second	ightmares each year
Im Pearson Correlation Sig. (2-tailed) 148 158 113 mare distress Pearson Correlation 189 .325 .442 mare distress Pearson Correlation 189 253 .297* Sig. (2-tailed) .198 .110 .040 N 48 41 48 sal scale Pearson Correlation 015 .003 .031 Sig. (2-tailed) .920 .984 .835 N 48 41 48 twe experiences Pearson Correlation .042 .014 .201 sig. (2-tailed) .777 .933 .170 N M 48 41 48 entime personality Pearson Correlation .044 .109 .036 Sig. (2-tailed) .764 .497 .807 .850 N 48 41 48 .41 48 enck Neuroticism Pearson Correlation .166 .002 .178	.232						
Sig. (2-tailed) .315 .325 .442 N 48 41 48 mare distress Pearson Correlation 189 253 297* Sig. (2-tailed) 198 .110 .040 N 48 41 48 sal scale Pearson Correlation 015 .003 .031 Sig. (2-tailed) .920 .984 .835 N 48 41 48 experiences Pearson Correlation .042 .014 .201 sig. (2-tailed) .777 .933 .170 .046 sig. (2-tailed) .777 .933 .70 .046 sig. (2-tailed) .764 .030 .046 sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Neuroticism Pearson Correlation .046 .002 .178 Sig. (2-tailed) .764 .497 .807 N 48	41						
N 48 41 48 mare distress Pearson Correlation Sig. (2-tailed) -189 253 297* Sig. (2-tailed) 198 110 0.40 N 48 41 48 sal scale Pearson Correlation Sig. (2-tailed) .920 .984 .835 N 48 41 48	115				the second s		Nm
mare distress Pearson Correlation Sig. (2-tailed) 189 253 297* N 48 41 48 val scale Pearson Correlation Sig. (2-tailed) .901 .003 .031 N 48 41 48 Atticle Pearson Correlation Sig. (2-tailed) .920 .984 .835 N 48 41 48 Inve experiences Pearson Correlation Sig. (2-tailed) .777 .933 .170 Attime personality Pearson Correlation Sig. (2-tailed) .024 014 201 Attime personality Pearson Correlation Sig. (2-tailed) .024 030 .046 Sig. (2-tailed) .873 .850 .755	.474					Sig. (2-tailed)	
Sig. (2-tailed) .198 .110 .040 N 48 41 48 sal scale Pearson Correlation .015 .003 .031 Sig. (2-tailed) .920 .984 .835 N 48 41 48 twe experiences Pearson Correlation .042 .014 201 d Sig. (2-tailed) .777 .933 .170 N 48 41 48 etrline personality Pearson Correlation .024 .030 .046 N 48 41 48	41						
N 48 41 48 sal scale Pearson Correlation Sig. (2-tailed) 015 .003 .031 Sig. (2-tailed) .920 .984 .835 N 48 41 48 file Pearson Correlation .042 014 201 st Sig. (2-tailed) .777 .933 .170 N 48 41 48 terline personality Pearson Correlation .024 030 046 Sig. (2-tailed) .873 .850 .755 N M 48 41 48 enck Neuroticism Pearson Correlation .044 109 036 Ite Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 sig. (2-tailed) .260 .990 .226	323*		297*				mare distress
sal scale Pearson Correlation Sig. (2-tailed) N 015 .003 .031 N 48 41 48 five experiences Pearson Correlation Sig. (2-tailed) .777 .933 .170 N 48 41 48 experiences Pearson Correlation Sig. (2-tailed) .777 .933 .170 N 48 41 48 48 infine personality Pearson Correlation N .024 .030 .046 Sig. (2-tailed) .873 .850 .755 N 48 41 48 enck Neuroticism Pearson Correlation .044 Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation Sig. (2-tailed) N 48 41 48 mans boundary <t< td=""><td>.039</td><td></td><td></td><td></td><td></td><td>Sig. (2-tailed)</td><td></td></t<>	.039					Sig. (2-tailed)	
Sig. (2-tailed) .920 .984 .835 N 48 41 48 experiences Pearson Correlation .042 014 201 sig. (2-tailed) .777 .933 .170 N 48 41 48 ethine personality Pearson Correlation .024 030 046 Sig. (2-tailed) .873 .850 .755 N nok Neuroticism Pearson Correlation 044 109 036 nok Neuroticism Pearson Correlation 044 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .216 .990 .226 N 48 41 48 teral Health Pearson Correlation .216 .097 .070 stionnaire Sig. (2-tailed) .617 .442 .258 N 41 48 41	41				48		
N 48 41 48 ilve experiences Pearson Correlation .042 014 201 st Sig. (2-tailed) .777 .933 .170 N 48 41 48 ierline personality Pearson Correlation .024 030 046 Sig. (2-tailed) .873 .850 .755 N 48 41 48 enck Neuroticism Pearson Correlation 044 109 036 ile Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 enack Lie Scale Pearson Correlation .216 .097 070 stionnaire Sig. (2-tailed) .617 .442 .258 N 48 41 48	044			.003	015	Pearson Correlation	salscale
Inverse Pearson Correlation .042 .014 .201 d Sig. (2-tailed) .777 .933 .170 N 48 41 48 terline personality Pearson Correlation .024 .030 .046 Sig. (2-tailed) .873 .850 .755 N 48 41 48 anck Neuroticism Pearson Correlation .044 .109 .036 ie Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 enck Lie Scale Pearson Correlation .216 .097 .070 sig. (2-tailed) .141 .545 .634 .070 sig. (2-tailed) .617 .442 .258 N 47 40 47	.786		.835	.984	.920	Sig. (2-tailed)	
d Sig. (2-tailed) N .777 .933 .170 N 48 41 48 terline personality Pearson Correlation Sig. (2-tailed) .024 030 046 Sig. (2-tailed) .873 .850 .755 N 48 41 48 enck Neuroticism Pearson Correlation 044 109 036 ie Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 meral Health Pearson Correlation .216 .097 070 stionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 41 Imann Boundary Pearson Correlation .075 125 168 Sig. (2-tailed) .617	41		48	41	48		
N 48 41 48 endine personality Pearson Correlation Sig. (2-tailed) .024 030 046 N 48 41 48 endk Neuroticism Pearson Correlation .044 109 036 N 48 41 48 endk Neuroticism Pearson Correlation 044 109 036 Ite Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 enck Lie Scale Pearson Correlation .216 .097 .070 stig. (2-tailed) .141 .545 .634 mann Boundary Pearson Correlation .075 .125 .168 Sig. (2-tailed) .617 .442 .258 N 47 40 47	266		201	014	.042	Pearson Correlation	tive experiences
entime personality Pearson Correlation Sig. (2-tailed) .024 030 046 N 48 41 48 enck Neuroticism Pearson Correlation 044 109 036 le Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Neuroticism Pearson Correlation .066 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 enck Lie Scale Pearson Correlation .260 .990 .226 N 48 41 48 44 enck Lie Scale Pearson Correlation .216 .097 .070 sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 .168 Sig. (2-tailed) .617 .442 .258 N 47 40 47	.092		.170	.933	.777	Sig. (2-tailed)	st
Sig. (2-tailed) .873 .850 .755 N 48 41 48 enck Neuroticism Pearson Correlation 044 109 036 le Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 sig. (2-tailed) .260 .990 .226 N 48 41 48 enck Lie Scale Pearson Correlation .216 .097 070 sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 sig. (2-tailed) .617 .442 .258	41		48	41	48		
N 48 41 48 enck Neuroticism Pearson Correlation 044 109 036 ie Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 eneral Health Pearson Correlation .216 .097 070 stionnaire Sig. (2-tailed) .141 .545 .634 ntmann Boundary Pearson Correlation .075 125 168 Sig. (2-tailed) .617 .442 .258 N 47 40 47 tanxiety Pearson	197		046	030	.024	Pearson Correlation	terline personality
enck Neuroticism Pearson Correlation 044 109 036 ie Sig. (2-tailed) .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 enck Lie Scale Pearson Correlation .216 .097 .070 Sig. (2-tailed) .141 .545 .634 neral Health Pearson Correlation .216 .097 070 stionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 Sig. (2-tailed) .617 .442 .258 N 47 40 47 te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .519 .543	.217		.755	.850	.873	Sig. (2-tailed)	
Image Sig. (2-tailed) N .764 .497 .807 N 48 41 48 enck Lie Scale Pearson Correlation Sig. (2-tailed) .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 heral Health Pearson Correlation .216 .097 .070 stionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 Sig. (2-tailed) .617 .442 .258 N 47 40 47 te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N N 22 19 .22 .419 .229 Sig. (2-tailed) .519 .543 .138 .081 N 40 .044	41		48	41	48	N	
N 48 41 48 enck Lie Scale Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 reral Health Pearson Correlation .216 .097 070 stionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 Sig. (2-tailed) .617 .442 .258 N 47 40 47 te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 .22 tt Anxiety Pearson Correlation .105 108 239 Sig. (2-tailed) .519 .543 .138 .081 N 40 .34 40 .081 N <td>212</td> <td></td> <td>036</td> <td>109</td> <td>044</td> <td>Pearson Correlation</td> <td>enck Neuroticism</td>	212		036	109	044	Pearson Correlation	enck Neuroticism
Pearson Correlation .166 .002 .178 Sig. (2-tailed) .260 .990 .226 N 48 41 48 heral Health Pearson Correlation .216 .097 070 estionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 Imann Boundary Pearson Correlation .075 125 168 stimann Boundary Pearson Correlation .075 125 168 stimann Boundary Pearson Correlation .075 125 168 stig. (2-tailed) .617 .442 .258 N 47 40 47 te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 t Anxiety Pearson Correlation N	.184		.807	.497	.764	Sig. (2-tailed)	le
Sig. (2-tailed) N .260 .990 .226 N 48 41 48 heral Health Pearson Correlation .216 .097 070 estionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 sig. (2-tailed) .617 .442 .258 N 47 40 47 ite Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22 it Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 .04 hal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590 .590	41		48	41	48	N	
N 48 41 48 meral Health Pearson Correlation .216 .097 070 stionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 sti Sig. (2-tailed) .617 .442 .258 N 47 40 47 te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22 tt Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138	.351*		.178	.002	.166	Pearson Correlation	enck Lie Scale
neral Health Pearson Correlation .216 .097 070 estionnaire Sig. (2-tailed) .141 .545 .634 N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 st Sig. (2-tailed) .617 .442 .258 N 47 40 47 te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22 It Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 .138 N 40 34 40 .081 Sig. (2-tailed) .519 .543 .138 N 40 34 40	.025		.226	.990	.260	Sig. (2-tailed)	
stionnaire Sig. (2-tailed) N .141 .545 .634 tmann Boundary Pearson Correlation .075 125 168 st Sig. (2-tailed) .617 .442 .258 N 47 40 47 ite Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22 it Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .519 .543 .138 .081 N 40 34 40 .081 .081	41		48	41	48	N	
N 48 41 48 tmann Boundary Pearson Correlation .075 125 168 st Sig. (2-tailed) .617 .442 .258 N 47 40 47 ite Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .662 .162 .756 N 22 19 22 it Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 thankiety Pearson Correlation .043 .081 Sig. (2-tailed) .519 .543 .138 N 40 34 40 thal Severity Idex Pearson Correlation .044 128 .081 Sig. (2-tailed) .770 .432 .590 .590	099	46	070	.097	.216	Pearson Correlation	
Image:	.537		.634	.545	.141	Sig. (2-tailed)	estionnaire
Sig. (2-tailed) .617 .442 .258 N 47 40 47 te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22 It Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590 .590	41		48	41	48	N	
N 47 40 47 Ite Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22 t Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590 .590	289		168	125	.075	Pearson Correlation	tmann Boundary
te Anxiety Pearson Correlation .039 334 .070 Sig. (2-tailed) .862 .162 .756 N 22 19 22 t Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590 .590	.071		.258	.442	.617	Sig. (2-tailed)	est
Sig. (2-tailed) .862 .162 .756 N 22 19 22 It Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 thal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590 .590	40		47	40	47	N	
N 22 19 22 it Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590 .500	113		.070	334	.039	Pearson Correlation	te Anxiety
It Anxiety Pearson Correlation 105 108 239 Sig. (2-tailed) .519 .543 .138 N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590	.645		.756	.162	.862	Sig. (2-tailed)	
Sig. (2-tailed) .519 .543 .138 N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590	19		22	19	22	N	
N 40 34 40 bal Severity Idex Pearson Correlation .044 128 081 Sig. (2-tailed) .770 .432 .590	253		239	108	105	Pearson Correlation	it Anxiety
bal Severity IdexPearson Correlation.044128081Sig. (2-tailed).770.432.590	.149	1	.138	.543	.519	Sig. (2-tailed)	
bal Severity IdexPearson Correlation.044128081Sig. (2-tailed).770.432.590	34	1	40	34		N	
Sig. (2-tailed) .770 .432 .590	112					Pearson Correlation	bal Severity Idex
	.492					Sig. (2-tailed)	
	40	6. 5					
sitive Symptom Total Pearson Correlation .120096110	128					Pearson Correlation	sitive Symptom Total
Sig. (2-tailed) .420 .554 .462	.430	-					
N 47 40 47	40						
sitive Symptom Pearson Correlation026167032	049						sitive Symptom
tress Index Sig. (2-tailed) .861 .302 .831	.764						
N 47 40 47	40						

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		Fisher's r of ws nights of unpld and presleep anxiety	FrRnprean	FrRupoa	FrRnpoa
of 4 childhood items	Pearson Correlation	.026	002	193	139
	Sig. (2-tailed)	.863	.992	.188	.385
	N	48	41	48	41
wents	Pearson Correlation	.106	.016	026	162
	Sig. (2-tailed)	.475	.923	.859	.310
	N	48	41	48	41

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		FrRuprede	FrRnprede	FrRupod	FrRnpod
nightmares each year	Pearson Correlation	.085	.196	.031	.012
ingrianaree each year	Sig. (2-tailed)	.565	.219	.836	.938
	N	48	41	48	41
Nm	Pearson Correlation	.045	.169	032	.137
	Sig. (2-tailed)	.760	.291	.832	.395
	N	48	41	48	41
tmare distress	Pearson Correlation	143	052	301*	401*
	Sig. (2-tailed)	.333	.746	.038	.009
	N	48	41	48	41
usal scale	Pearson Correlation	.162	.372*	.203	.174
Bar Source	Sig. (2-tailed)	.272	.012	.167	.277
	N	48	41	48	41
ative experiences	Pearson Correlation	.189	.244	061	009
st	Sig. (2-tailed)	.199	.124	.682	.954
	N	48	41	48	41
derline personality	Pearson Correlation	.118	.192	.021	.111
conne personanty	Sig. (2-tailed)	.425	.230	.885	.490
	N	48	41	48	.430
enck Neuroticism	Pearson Correlation	017	.067	.041	083
ale	Sig. (2-tailed)	.906	.007	.783	.607
	N	48	41	48	.007
senck Lie Scale	Pearson Correlation	-,136	201	037	033
SEICK LIE OLAIE	Sig. (2-tailed)	.357	.201	.804	.836
	N	48	41	48	.030
neral Health	Pearson Correlation	.246	.310*	037	084
estionnaire					
	Sig. (2-tailed) N	.092	.048	.801	.600
trans Deupdop	Pearson Correlation				41
itmann Boundary iest		.024	033	007	061
(J)	Sig. (2-tailed)	.873	.839	.964	.708
the Amiliante	N Rearran Correlation	47	40	47	40
ate Anxiety	Pearson Correlation	.075	110	.211	161
	Sig. (2-tailed)	.741	.654	.346	.510
i Anuintu	N Decrean Correlation	22	19	22	19
ait Anxiety	Pearson Correlation	040	.046	041	090
	Sig. (2-tailed)	.806	.795	.800	.614
1.10 11.11	N	40	34	40	34
lobal Severity Idex	Pearson Correlation	.098	.113	.020	041
	Sig. (2-tailed)	.513	.488	.894	.803
sitive Symptom Total	N Deserve Correlation	47	40	47	40
	Pearson Correlation	.119	.070	.137	007
	Sig. (2-tailed)	.426	.670	.359	.966
	N	47	40	47	40
sitive Symptom	Pearson Correlation	.009	.057	120	112
stress Index	Sig. (2-tailed)	.951	.729	.423	.492
	N	47	40	47	40



		FrRuprede	FrRnprede	FrRupod	FrRnpod
of 4 childhood items	Pearson Correlation	039	104	140	148
	Sig. (2-tailed)	.790	.518	.342	.356
	N	48	41	48	41
events	Pearson Correlation	.126	.184	.109	096
	Sig. (2-tailed)	.393	.250	.462	.552
	N	48	41	48	41



ghtmares each year					
ghtmares each year				121	
ghtmares each year					20
ghtmares each year		FrRuFprean	FrRnFprean	FrRuFpoa	FrRnFpoa
	Pearson Correlation	249	242	185	238
	Sig. (2-tailed)	.095	.127	.209	.133
	N	46	41	48	41
Nm	Pearson Correlation	237	181	132	149
	Sig. (2-tailed)	.113	.257	.370	.351
	N	46	41	48	41
mare distress	Pearson Correlation	381**	323*	328*	335*
	Sig. (2-tailed)	.009	.039	.023	.032
	N	46	41	48	41
sal scale	Pearson Correlation	184	101	013	040
	Sig. (2-tailed)	.222	.532	.932	.803
	N	46	41	48	41
tive experiences	Pearson Correlation	150	096	226	244
st	Sig. (2-tailed)	.319	.551	.122	.124
	N	46	41	48	41
erline personality	Pearson Correlation	129	101	112	207
	Sig. (2-tailed)	.393	.528	.447	.194
	N	46	41	48	41
enck Neuroticism	Pearson Correlation	171	168	102	215
e	Sig. (2-tailed)	.257	.293	.490	.176
	N	46	41	48	41
enck Lie Scale	Pearson Correlation	.180	.032	.226	.330*
	Sig. (2-tailed)	.232	.842	.123	.035
	N	46	41	48	41
eral Health	Pearson Correlation	.048	.037	.025	079
stionnaire	Sig. (2-tailed)	.750	.819	.866	.624
	N	46	41	48	41
mann Boundary	Pearson Correlation	099	192	138	249
st	Sig. (2-tailed)	.519	.234	.356	.121
	N	45	40	47	40
e Anxiety	Pearson Correlation	246	332	.014	148
	Sig. (2-tailed)	.283	.165	.950	.545
	N	21	19	22	19
Anxiety	Pearson Correlation	349*	193	280	280
	Sig. (2-tailed)	.032	.275	.080	.108
	N	38	34	40	34
ubal Severity Idex	Pearson Correlation	119	108	065	093
	Sig. (2-tailed)	.436	.507	.664	.569
	N	45	40	47	40
sitive Symptom Total	Pearson Correlation	079	145	033	107
	Sig. (2-tailed)	.604	.372	.825	.511
	N	45	40	47	40
itive Symptom	Pearson Correlation	178	161	069	017
- /	Sig. (2-tailed)	.241	.322	.644	.918
ress Index	Old. (Z-lalleu)	/ 40 1		11444	910



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		FrRuFprean	FrRnFprean	FrRuFpoa	FrRnFpoa
of 4 childhood items	Pearson Correlation	164	024	213	149
	Sig. (2-tailed)	.277	.881	.146	.353
	Ν	46	41	48	41
wents	Pearson Correlation	.014	.001	.060	136
	Sig. (2-tailed)	.927	.995	.685	.396
	N	46	41	48	41

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		FrRuFprede	FrRnFprede	FrRuFpod	FrRnFpod
ightmares each year	Pearson Correlation	.095	.204	011	059
	Sig. (2-tailed)	.524	.202	.940	.714
	N	47	41	48	41
Nm	Pearson Correlation	.051	.224	074	.063
	Sig. (2-tailed)	.731	.159	.618	.694
	Ν	47	41	48	41
tmare distress	Pearson Correlation	092	.051	251	308
	Sig. (2-tailed)	.539	.752	.085	.050
	N	47	41	48	41
salscale	Pearson Correlation	.106	.217	.272	.190
	Sig. (2-tailed)	.477	.174	.061	.234
	N	47	41	48	41
tive experiences	Pearson Correlation	.150	.274	.095	.151
st	Sig. (2-tailed)	.316	.083	.520	.345
	N	47	41	48	41
erline personality	Pearson Correlation	.118	.311*	.021	.118
	Sig. (2-tailed)	.431	.048	.885	.461
	Ν	47	41	48	41
senck Neuroticism ale	Pearson Correlation	009	.158	.063	050
	Sig. (2-tailed)	.952	.325	.672	.758
	Ν	47	41	48	41
enck Lie Scale	Pearson Correlation	107	244	063	047
	Sig. (2-tailed)	.472	.124	.671	.770
	Ν	47	41	48	41
eral Health	Pearson Correlation	.259	.324*	.009	047
stionnaire	Sig. (2-tailed)	.079	.039	.954	.771
	Ν	47	41	48	41
tmann Boundary	Pearson Correlation	.047	.094	.104	.059
est	Sig. (2-tailed)	.755	.564	.487	.719
	N	46	40	47	40
e Anxiety	Pearson Correlation	.172	.079	.233	078
	Sig. (2-tailed)	.443	.748	.296	.750
	Ν	22	19	22	19
ait Anxiety	Pearson Correlation	053	.123	060	088
	Sig. (2-tailed)	.748	.489	.713	.620
	N	39	34	40	34
obal Severity Idex	Pearson Correlation	.204	.297	.058	.008
	Sig. (2-tailed)	.173	.063	.700	.963
	N	46	40	47	40
sitive Symptom Total	Pearson Correlation	.153	.196	.146	.051
	Sig. (2-tailed)	.312	.226	.328	.753
	N	46	40	47	40
itive Symptom	Pearson Correlation	.122	.245	054	054
ress Index	Sig. (2-tailed)	.421	.128	.720	.739
	Ν	46	40	47	40

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		FrRuFprede	FrRnFprede	FrRuFpod	FrRnFpod
of 4 childhood items	Pearson Correlation	002	022	068	024
	Sig. (2-tailed)	.991	.889	.645	.883
	N	47	41	48	41
wents	Pearson Correlation	.167	.290	.236	.026
	Sig. (2-tailed)	.262	.066	.107	.870
	N	47	41	48	41



			行政主任法		
		1.			
		FrXRuprean	FrXRnprean	FrRXupoa	FrRXnpoa
nightmares each year	Pearson Correlation	190	246	244	238
	Sig. (2-tailed)	.222	.137	.115	.151
	N	43	38	43	38
Nm	Pearson Correlation	223	260	168	171
	Sig. (2-tailed)	.151	.114	.280	.305
	N	43	38	43	38
Itmare distress	Pearson Correlation	142	251	355*	338
	Sig. (2-tailed)	.364	.128	.019	.038
	N	43	38	43	38
usal scale	Pearson Correlation	091	112	104	130
	Sig. (2-tailed)	.560	.505	.505	.436
	N	43	38	43	38
ative experiences	Pearson Correlation	035	023	287	254
est	Sig. (2-tailed)	.823	.892	.062	.124
	N	43	38	43	38
derline personality	Pearson Correlation	039	130	265	295
	Sig. (2-tailed)	.802	.437	.086	.072
	N	43	38	43	38
enck Neuroticism	Pearson Correlation	085	282	175	276
ale	Sig. (2-tailed)	.590	.086	.262	.093
	N	43	38	43	38
senck Lie Scale	Pearson Correlation	.067	.068	.301	.388
	Sig. (2-tailed)	.667	.685	.050	.016
	N	43	38	43	38
neral Health	Pearson Correlation	.095	.191	065	071
estionnaire	Sig. (2-tailed)	.545	.252	.681	.672
	N	43	38	43	38
tmann Boundary	Pearson Correlation	064	026	268	266
est	Sig. (2-tailed)	.689	.880	.086	.112
	N	42	37	42	37
te Anxiety	Pearson Correlation	170	512*	017	259
	Sig. (2-tailed)	.487	.036	.946	.315
· · · ·	N	19	17	19	17
it Anxiety	Pearson Correlation	118	311	285	338
	Sig. (2-tailed)	.499	.088	.097	.063
	N	35	31	35	31
bal Severity Idex	Pearson Correlation	.005	089	128	140
	Sig. (2-tailed)	.977	.600	.418	.407
sitive Symptom Total	N Decrean Correlation	42	37	42	37
	Pearson Correlation	.087	026	134	181
	Sig. (2-tailed)	.582	.880	.397	.285
itivo Europtore	N Decrean Correlation	42	37	42	37
itive Symptom tress Index	Pearson Correlation	061	135	016	009
	Sig. (2-tailed)	.699	.427	.919	.958
	N	42	37	42	37

		FrXRuprean	FrXRnprean	FrRXupoa	FrRXnpoa
rof 4 childhood items events	Pearson Correlation	.116	.111	187	116
	Sig. (2-tailed)	.459	.508	.230	.487
	N	43	38	43	38
	Pearson Correlation	.142	.062	.047	044
	Sig. (2-tailed)	.364	.712	.764	.795
	Ν	43	38	43	38



					13.3
	Deersen Correlation	FrRXuprede	FrRXnprede	FrXRuFprean	FrXRnFprean
nightmares each year	Pearson Correlation	.067	.115	204	265
	Sig. (2-tailed)	.668	.492	.190	.108
- Man	N Pearson Correlation	43	38	43	38
e Nm		066	.003	259	288
	Sig. (2-tailed)	.674	.988	.093	.080
the set of the set	N Pearson Correlation	43	38	43	38
htmare distress		109	114	207	348*
	Sig. (2-tailed)	.488	.494	.184	.032
	N	43	38	43	38
usal scale	Pearson Correlation	.152	.179	175	225
	Sig. (2-tailed)	.330	.281	.261	.175
	N	43	-38	43	38
ative experiences	Pearson Correlation	.134	.201	105	134
est	Sig. (2-tailed)	.391	.226	.501	.421
	N	43	38	43	38
derline personality	Pearson Correlation	.134	.194	047	220
	Sig. (2-tailed)	.390	.244	.764	.184
	N	43	38	43	38
senck Neuroticism ale	Pearson Correlation	.048	026	115	357*
	Sig. (2-tailed)	.762	.879	.461	.028
	N	43	38	43	38
senck Lie Scale	Pearson Correlation	226	228	.084	.122
	Sig. (2-tailed)	.146	.169	.592	.465
	N	43	38	43	38
eneral Health	Pearson Correlation	.097	.164	.064	.111
uestionnaire	Sig. (2-tailed)	.537	.325	.683	.506
and the second	N	43	38	43	38
atmann Boundary	Pearson Correlation	045	.019	109	141
uest	Sig. (2-tailed)	.775	.911	.491	.406
	N	42	37	42	37
ate Anxiety	Pearson Correlation	206	285	200	499*
	Sig. (2-tailed)	.396	.267	.411	.041
	N	19	17	19	17
ait Anxiety	Pearson Correlation	091	179	164	386*
	Sig. (2-tailed)	.605	.334	.348	.032
	N	35	31	35	31
lobal Severity Idex	Pearson Correlation	.123	.141	.011	116
	Sig. (2-tailed)	.437	.405	.945	.495
	Ν	42	37	42	37
sitive Symptom Total	Pearson Correlation	.035	.025	.044	086
	Sig. (2-tailed)	.826	.882	.783	.612
	N	42	37	42	37
ositive Symptom	Pearson Correlation	.064	.085	051	178
istress Index	Sig. (2-tailed)	.687	.619	.746	.291
	N	42	37	42	37
		42	51	42	57

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Correlations

				1111	
		FrRXuprede	FrRXnprede	FrXRuFprean	FrXRnFprean
of 4 childhood items	Pearson Correlation	046	050	.078	.086
	Sig. (2-tailed)	.768	.765	.617	.606
	N	43	38	43	38
wents	Pearson Correlation	.097	.163	.123	.020
	Sig. (2-tailed)	.536	.329	.433	.904
	N	43	38	43	38

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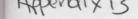
THENDIX 13

			1.2.62.63.22	1221-12-53	
		1.1.1	11111		Section 2.
				2. 1. 1. 1.	1
		FrRXuFpoa	FrRXnFpoa	FrRXuFprede	FrRXnFprede
ightmares each year	Pearson Correlation	287	277	.065	.146
	Sig. (2-tailed)	.062	.092	.678	.380
	N	43	38	43	38
Nm	Pearson Correlation	218	215	071	.099
	Sig. (2-tailed)	.161	.195	.651	.556
	N	43	38	43	38
mare distress	Pearson Correlation	379*	353*	064	092
	Sig. (2-tailed)	.012	.030	.683	.584
	N	43	38	43	38
sal scale	Pearson Correlation	095	144	.113	.129
	Sig. (2-tailed)	.543	.388	.472	.439
	N	43	38	43	38
tive experiences	Pearson Correlation	287	249	.111	.169
st	Sig. (2-tailed)	.062	.132	.478	.312
	N	43	38	43	38
erline personality	Pearson Correlation	294	340*	.135	.190
	Sig. (2-tailed)	.056	.037	.389	.253
	N	43	38	43	38
enck Neuroticism	Pearson Correlation	202	332*	.074	.021
le	Sig. (2-tailed)	.194	.042	.639	.899
	N	43	38	43	38
enck Lie Scale	Pearson Correlation	.269	.401*	175	171
	Sig. (2-tailed)	.081	.012	.261	.305
	Ν	43	38	43	38
eral Health	Pearson Correlation	046	063	.085	.205
stionnaire	Sig. (2-tailed)	.769	.706	.586	.218
	Ν	43	38	43	38
mann Boundary	Pearson Correlation	220	251	001	.055
est	Sig. (2-tailed)	.161	.134	.997	.745
	N	42	37	42	37
re Anxiety	Pearson Correlation	086	299	088	117
	Sig. (2-tailed)	.728	.244	.721	.655
	N	19	17	19	17
tAnxiety	Pearson Correlation	328	375*	081	174
	Sig. (2-tailed)	.055	.038	.643	.350
	N	35	31	35	31
bal Severity Idex	Pearson Correlation	115	134	.195	.222
	Sig. (2-tailed)	.468	.430	.217	.186
	N	42	37	42	37
tive Symptom Total	Pearson Correlation	109	151	.063	.076
	Sig. (2-tailed)	.492	.372	.692	.654
	N N	42	37	42	37
tive Symptom	Pearson Correlation	003	023	.154	.172
ress Index	Sig. (2-tailed)	.983	.892	.329	.309
	N	42	37	42	37



		FrRXuFpoa	FrRXnFpoa	FrRXuFprede	FrRXnFprede
of 4 childhood items	Pearson Correlation	199	103	.003	044
	Sig. (2-tailed)	.202	.537	.984	.792
	N	43	38	43	38
wents	Pearson Correlation	.102	029	.110	.134
	Sig. (2-tailed)	.516	.864	.482	.422
	N	43	38	43	38

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	and the second second				
		FrRXupod	FrRXnpod	FrRXuFpod	FrRXnFpod
hightmares each year	Pearson Correlation	188	211	221	241
	Sig. (2-tailed)	.228	.204	.155	.145
	N	43	38	43	38
Nm	Pearson Correlation	203	186	258	122
	Sig. (2-tailed)	.193	.265	.094	.466
	N	43	38	43	38
tmare distress	Pearson Correlation	459**	486**	373*	386*
	Sig. (2-tailed)	.002	.002	.014	.017
	N	43	38	43	38
usal scale	Pearson Correlation	.101	030	.131	.012
	Sig. (2-tailed)	.517	.860	.401	.943
	N	43	38	43	38
ative experiences	Pearson Correlation	076	.091	.042	.222
st	Sig. (2-tailed)	.629	.589	.788	.180
	Ν	43	38	43	38
derline personality	Pearson Correlation	046	.042	071	.018
	Sig. (2-tailed)	.770	.803	.652	.916
	N	43	38	43	38
enck Neuroticism	Pearson Correlation	079	250	079	194
ale	Sig. (2-tailed)	.612	.131	.613	.242
	N	43	38	43	38
senck Lie Scale	Pearson Correlation	111	.036	059	.005
	Sig. (2-tailed)	.479	.832	.707	.974
	N	43	38	43	38
neral Health	Pearson Correlation	052	.051	007	.009
estionnaire	Sig. (2-tailed)	.739	.760	.967	.957
	N	43	38	43	38
rtmann Boundary	Pearson Correlation	158	068	033	.030
est	Sig. (2-tailed)	.317	.689	.836	.859
	N	42	37	42	37
ate Anxiety	Pearson Correlation	176	357	120	319
	Sig. (2-tailed)	.472	.160	.625	.213
	N	19	17	19	17
ait Anxiety	Pearson Correlation	169	167	176	218
	Sig. (2-tailed)	.331	.370	.312	.239
	Ν	35	31	35	31
bal Severity Idex	Pearson Correlation	044	105	.022	.005
	Sig. (2-tailed)	.782	.535	.891	.975
	N	42	37	42	37
sitive Symptom Total	Pearson Correlation	008	051	.084	.027
	Sig. (2-tailed)	.958	.764	.596	.874
	N	42	37	42	37
sitive Symptom	Pearson Correlation	102	049	040	065
stress Index	Sig. (2-tailed)	.521	.772	.802	.702
	Ν	42	37	42	37

		FrRXupod	FrRXnpod	FrRXuFpod	FrRXnFpod
of 4 childhood items	Pearson Correlation	163	.044	060	.089
	Sig. (2-tailed)	.295	.792	.701	.596
	N	43	38	43	38
wents	Pearson Correlation	.056	.037	.170	.123
	Sig. (2-tailed)	.719	.827	.276	.463
	Ν	43	38	43	38

Correlation is significant at the 0.05 level (2-tailed).

Correlation is significant at the 0.01 level (2-tailed).

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		Fisher's r of ws nights of unpld and presleep	FrRnprean	FrRupoa	FrRnpoa
hightmares each year	Pearson Correlation	anxiety -,422*	421*	345	290
ignimates each year	Sig. (2-tailed)	.040	.040	.099	.170
	N	.040	.040	.099	.170
Nm	Pearson Correlation	082	175	129	113
NUT	Sig. (2-tailed)	.705	.413	129	.599
	N	.703	.413	.340	.399
tmare distress	Pearson Correlation	335	364	403	250
	Sig. (2-tailed)	.109	.080	.051	.239
	N	24	.000	.001	.233
sal scale	Pearson Correlation	- 043	019	120	094
isal Scale	Sig. (2-tailed)	.843	.931	.577	.661
	N	24	24	.577	.001
ative experiences	Pearson Correlation	.145	.098	422*	369
st	Sig. (2-tailed)	.498	.649	.040	.076
	N	24	24	24	24
derline personality	Pearson Correlation	.074	.023	421*	375
termine percontainty	Sig. (2-tailed)	.731	.914	.040	.071
	N	24	24	24	24
enck Neuroticism	Pearson Correlation	180	116	430*	378
ale	Sig. (2-tailed)	.400	.591	.036	.069
	N	24	24	24	24
senck Lie Scale	Pearson Correlation	038	159	.521**	.586**
	Sig. (2-tailed)	.861	.458	.009	.003
	N	24	24	24	24
neral Health	Pearson Correlation	.243	.132	128	100
estionnaire	Sig. (2-tailed)	.253	.538	.551	.642
	N	24	24	24	24
atmann Boundary	Pearson Correlation	.106	023	380	344
Jest	Sig. (2-tailed)	.621	.914	.067	.100
	N	24	24	24	24
ate Anxiety	Pearson Correlation	091	322	270	157
	Sig. (2-tailed)	.790	.334	.422	.646
	N	11	11	11	11
ait Anxiety	Pearson Correlation	038	016	440*	351
	Sig. (2-tailed)	.872	.943	.046	.119
	Ν	21	21	21	21
obal Severity Idex	Pearson Correlation	054	090	277	203
	Sig. (2-tailed)	.800	.677	.190	.342
	N	24	24	24	24
sitive Symptom Total	Pearson Correlation	.021	031	401	327
	Sig. (2-tailed)	.922	.885	.052	.119
	Ν	24	24	24	24
sitive Symptom	Pearson Correlation	066	114	069	.021
stress Index	Sig. (2-tailed)	.760	.597	.749	.923
	Ν	24	24	24	24

		Fisher's r of ws nights of unpld and presleep anxiety	FrRnprean	FrRupoa	FrRnpoa
nof 4 childhood items	Pearson Correlation	.075	.049	137	058
	Sig. (2-tailed)	.727	.821	.524	.788
	N	24	24	24	24
events	Pearson Correlation	.170	.194	031	068
	Sig. (2-tailed)	.428	.364	.885	.753
	Ν	24	24	24	24

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		FrRuprede	FrRnprede	FrRupod	FrRnpod
nightmares each year	Pearson Correlation	090	.024	118	090
	Sig. (2-tailed)	.675	.910	.584	.675
	N	24	24	24	24
Nm	Pearson Correlation	.080	.083	.082	.271
	Sig. (2-tailed)	.709	.700	.704	.199
	N	24	24	24	24
tmare distress	Pearson Correlation	240	138	475*	324
	Sig. (2-tailed)	.258	.521	.019	.123
	N	24	24	24	24
usal scale	Pearson Correlation	.082	.345	.061	.138
	Sig. (2-tailed)	.702	.099	.778	.519
	N	24	24	24	24
ative experiences	Pearson Correlation	.205	.230	034	.125
st	Sig. (2-tailed)	.337	.279	.875	.561
	Ν	24	24	24	24
derline personality	Pearson Correlation	.168	.098	036	.120
	Sig. (2-tailed)	.433	.649	.868	.577
	N	24	24	24	24
enck Neuroticism	Pearson Correlation	207	115	346	226
ale	Sig. (2-tailed)	.333	.592	.098	.288
	N	24	24	24	24
enck Lie Scale	Pearson Correlation	228	235	137	171
	Sig. (2-tailed)	.284	.269	.523	.425
	Ν	24	24	24	24
neral Health	Pearson Correlation	.312	.338	150	093
estionnaire	Sig. (2-tailed)	.138	.107	.484	.666
	Ν	24	24	24	24
tmann Boundary	Pearson Correlation	.074	020	166	049
est	Sig. (2-tailed)	.731	.925	.438	.819
	N	24	24	24	24
te Anxiety	Pearson Correlation	.038	.052	294	110
	Sig. (2-tailed)	.911	.880	.381	.748
	Ν	11	11	11	11
it Anxiety	Pearson Correlation	064	.034	334	159
	Sig. (2-tailed)	.782	.883	.139	.492
	N	21	21	21	21
bal Severity Idex	Pearson Correlation	.010	.044	266	114
	Sig. (2-tailed)	.964	.837	.208	.595
	N	24	24	24	24
sitive Symptom Total	Pearson Correlation	.054	.037	325	175
	Sig. (2-tailed)	.804	.865	.121	.414
	N	24	24	24	24
sitive Symptom	Pearson Correlation	013	.012	212	061
tress Index	Sig. (2-tailed)	.953	.954	.321	.776
	N N	24	24	24	24

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		FrRuprede	FrRnprede	FrRupod	FrRnpod
of 4 childhood items	Pearson Correlation	031	007	196	023
	Sig. (2-tailed)	.884	.974	.358	.915
	N	24	24	24	24
events	Pearson Correlation	.254	.273	.209	.159
	Sig. (2-tailed)	.230	.196	.327	.458
	N	24	24	24	24

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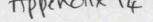
		ErDuEproop	FrRnFprean	FrRuFpoa	FrRnFpoa
nightmares each year	Pearson Correlation	FrRuFprean 298	329	337	283
ingritinales each year	Sig. (2-tailed)	.168	.116	.107	.180
	N	23	24	24	24
•Nm	Pearson Correlation	079	110	146	127
SINIT	Sig. (2-tailed)	.722	.609	.497	.556
	N	23	24	24	24
htmare distress	Pearson Correlation	417*	285	402	237
	Sig. (2-tailed)	.048	.177	.052	.265
	N	23	24	24	24
usal scale	Pearson Correlation	214	046	119	085
abai Sourc	Sig. (2-tailed)	.326	.831	.580	.693
	N	23	24	24	24
ative experiences	Pearson Correlation	131	.023	400	348
est	Sig. (2-tailed)	.552	.916	.053	.096
	N	23	24	24	24
derline personality	Pearson Correlation	090	004	471*	417*
denine percentanty	Sig. (2-tailed)	.684	.986	.020	.043
	N	23	24	24	24
senck Neuroticism	Pearson Correlation	224	091	462*	403
ale	Sig. (2-tailed)	.303	.672	.023	.051
	N	23	24	24	24
senck Lie Scale	Pearson Correlation	.079	134	.539**	.609**
SCHORE LIG COULD	Sig. (2-tailed)	.721	.532	.007	.002
	N	23	24	24	24
eneral Health	Pearson Correlation	.070	.096	128	103
lestionnaire	Sig. (2-tailed)	.752	.655	.550	.632
	N	23	24	24	24
atmann Boundary	Pearson Correlation	117	064	341	310
uest	Sig. (2-tailed)	.594	.768	.103	.140
	N	23	24	24	24
ate Anxiety	Pearson Correlation	376	092	265	160
	Sig. (2-tailed)	.284	.787	.430	.639
	N	10	11	11	11
ait Anxiety	Pearson Correlation	238	019	451*	362
	Sig. (2-tailed)	.312	.936	.040	.107
	N	20	21	21	21
obal Severity Idex	Pearson Correlation	151	.008	248	177
	Sig. (2-tailed)	.491	.972	.243	.408
	N	23	24	24	24
sitive Symptom Total	Pearson Correlation	099	030	370	290
	Sig. (2-tailed)	.655	.890	.075	.169
	N	23	24	24	24
ositive Symptom	Pearson Correlation	188	033	038	.052
stress Index	Sig. (2-tailed)	.391	.877	.860	.811
	N	23	24	24	24

		FrRuFprean	FrRnFprean	FrRuFpoa	FrRnFpoa
m of 4 childhood items	Pearson Correlation	229	.009	159	069
	Sig. (2-tailed)	.294	.967	.459	.749
	Ν	23	24	24	24
events	Pearson Correlation	.166	.290	.035	019
	Sig. (2-tailed)	.448	.170	.872	.930
	Ν	23	24	24	24



		FrRuFprede	FrRnFprede	FrRuFpod	FrRnFpod
nightmares each year	Pearson Correlation	.054	.098	040	064
	Sig. (2-tailed)	.802	.647	.851	.765
	N	24	24	24	24
e Nm	Pearson Correlation	.170	.175	.037	.236
	Sig. (2-tailed)	.428	.413	.862	.268
	N	24	24	24	24
htmare distress	Pearson Correlation	066	009	455*	325
	Sig. (2-tailed)	.761	.967	.026	.122
	N	24	24	24	24
ousal scale	Pearson Correlation	.053	.111	.192	.171
	Sig. (2-tailed)	.805	.606	.369	.424
	N	24	24	24	24
ative experiences	Pearson Correlation	.073	.157	.009	.177
lest	Sig. (2-tailed)	.734	.465	.968	.409
	N	24	24	24	24
iderline personality	Pearson Correlation	.170	.210	240	024
	Sig. (2-tailed)	.427	.325	.260	.911
	N	24	24	24	24
senck Neuroticism	Pearson Correlation	107	027	372	249
ale	Sig. (2-tailed)	.618	.899	.074	.240
	N	24	24	24	24
senck Lie Scale	Pearson Correlation	147	243	050	138
	Sig. (2-tailed)	.494	.253	.817	.521
	N	24	24	24	24
eneral Health	Pearson Correlation	.295	.333	156	180
estionnaire	Sig. (2-tailed)	.161	.112	.467	.401
	N	24	24	24	24
artmann Boundary	Pearson Correlation	.087	.109	164	046
Jest	Sig. (2-tailed)	.687	.612	.443	.831
	N	24	24	24	24
ate Anxiety	Pearson Correlation	.307	.354	028	.030
	Sig. (2-tailed)	.358	.286	.936	.931
	Ν	11	11	11	11
ait Anxiety	Pearson Correlation	015	.106	359	206
	Sig. (2-tailed)	.947	.649	.110	.371
	Ν	21	21	21	21
obal Severity Idex	Pearson Correlation	.206	.266	217	103
	Sig. (2-tailed)	.335	.210	.310	.633
	N	24	24	24	24
sitive Symptom Total	Pearson Correlation	.159	.190	271	135
	Sig. (2-tailed)	.457	.374	.200	.531
	Ν	24	24	24	24
sitive Symptom	Pearson Correlation	.188	.229	166	061
stress Index	Sig. (2-tailed)	.378	.281	.438	.775
	N	24	24	24	24

		FrRuFprede	FrRnFprede	FrRuFpod	FrRnFpod
n of 4 childhood items	Pearson Correlation	.029	.123	299	094
	Sig. (2-tailed)	.892	.567	.157	.662
	Ν	24	24	24	24
events	Pearson Correlation	.341	.363	.322	.222
	Sig. (2-tailed)	.103	.081	.125	.298
	N	24	24	24	24



			4.5.4		
		FrXRuprean	FrXRnprean	FrRXupoa	FrRXnpoa
ightmares each year	Pearson Correlation	433*	438*	374	353
	Sig. (2-tailed)	.034	.032	.072	.090
	N	24	24	24	24
Nm	Pearson Correlation	267	208	263	232
	Sig. (2-tailed)	.207	.329	.214	.276
	N	24	24	24	24
tmare distress	Pearson Correlation	180	258	398	328
	Sig. (2-tailed)	.399	.224	.054	.117
and the second	N	24	24	24	24
isal scale	Pearson Correlation	185	062	175	130
	Sig. (2-tailed)	.387	.772	.415	.545
	N	24	24	24	24
ative experiences	Pearson Correlation	.037	.064	441*	416*
st	Sig. (2-tailed)	.862	.765	.031	.043
	Ν	24	24	24	24
terline personality	Pearson Correlation	.016	009	451*	404
	Sig. (2-tailed)	.940	.968	.027	.050
	N	24	24	24	24
enck Neuroticism	Pearson Correlation	207	256	416*	390
ale	Sig. (2-tailed)	.332	.227	.043	.059
	N	24	24	24	24
enck Lie Scale	Pearson Correlation	076	126	.601**	.568**
	Sig. (2-tailed)	.724	.557	.002	.004
	N	24	24	24	24
neral Health	Pearson Correlation	.099	.162	202	159
estionnaire	Sig. (2-tailed)	.644	.450	.343	.459
	N	24	24	24	24
tmann Boundary	Pearson Correlation	072	065	372	362
est	Sig. (2-tailed)	.737	.764	.074	.083
	N	24	24	24	24
te Anxiety	Pearson Correlation	494	613*	353	316
	Sig. (2-tailed)	.122	.045	.286	.343
	N	11	11	11	11
it Anxiety	Pearson Correlation	120	193	411	375
	Sig. (2-tailed)	.603	.402	.064	.093
	N	21	21	21	21
bal Severity Idex	Pearson Correlation	089	112	298	255
bar octonity idex	Sig. (2-tailed)	.678	.602	.157	.229
	N	24	24	24	24
sitive Symptom Total	Pearson Correlation	062	097	385	357
and Oymptom rotal	Sig. (2-tailed)	.773	.651	.063	.087
	N	and the second			
sitive Symptom	Pearson Correlation	043	24	24	24
tress Index			086	065	018
and though	Sig. (2-tailed)	.842	.688	.762	.935
	N	24	24	24	24

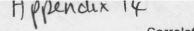
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		FrXRuprean	FrXRnprean	FrRXupoa	FrRXnpoa
of 4 childhood items	Pearson Correlation	.218	.199	192	111
	Sig. (2-tailed)	.307	.351	.368	.607
	N	24	24	24	24
events	Pearson Correlation	.174	.179	025	041
	Sig. (2-tailed)	.417	.403	.909	.851
	N	24	24	24	24

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				States and the states of the s	
		FrRXuprede	FrRXnprede	FrXRuFprean	FrXRnFprean
nightmares each year	Pearson Correlation	055	026	328	393
	Sig. (2-tailed)	.799	.906	.118	.057
and the second	N	24	24	24	24
• Nm	Pearson Correlation	001	.037	257	197
	Sig. (2-tailed)	.995	.863	.225	.356
	N	24	24	24	24
htmare distress	Pearson Correlation	056	096	098	235
	Sig. (2-tailed)	.794	.655	.648	.270
	N	24	24	24	24
usal scale	Pearson Correlation	.059	.110	247	111
	Sig. (2-tailed)	.786	.607	.245	.607
	N	24	24	24	24
ative experiences	Pearson Correlation	.192	.159	058	049
est	Sig. (2-tailed)	.370	.458	.788	.821
	N	24	24	24	24
iderline personality	Pearson Correlation	.175	.122	.050	088
	Sig. (2-tailed)	.414	.569	.815	.681
	N	24	24	24	24
senck Neuroticism	Pearson Correlation	240	283	188	287
ale	Sig. (2-tailed)	.259	.180	.379	.174
	N	24	24	24	24
senck Lie Scale	Pearson Correlation	278	306	014	064
	Sig. (2-tailed)	.189	.146	.948	.766
	N	24	24	24	24
eneral Health	Pearson Correlation	.152	.192	.089	.097
lestionnaire	Sig. (2-tailed)	.478	.368	.678	.651
	N	24	24	24	24
artmann Boundary	Pearson Correlation	002	008	081	158
uest	Sig. (2-tailed)	.994	.971	.708	.462
	Ν	24	24	24	24
ate Anxiety	Pearson Correlation	207	201	325	540
	Sig. (2-tailed)	.541	.554	.330	.087
	Ν	11	11	11	11
ait Anxiety	Pearson Correlation	120	167	110	255
	Sig. (2-tailed)	.603	.469	.636	.265
	N	21	21	21	21
obal Severity Idex	Pearson Correlation	.086	.092	.001	092
	Sig. (2-tailed)	.690	.670	.996	.669
	N	24	24	24	24
sitive Symptom Total	Pearson Correlation	016	040	035	149
	Sig. (2-tailed)	.941	.851	.870	.488
	N	24	24	24	24
sitive Symptom	Pearson Correlation	.079	.076	.058	053
stress Index	Sig. (2-tailed)	.715	.726	.789	.807
	N	24	24	24	24



		FrRXuprede	FrRXnprede	FrXRuFprean	FrXRnFprean
of 4 childhood items	Pearson Correlation	.137	.148	.167	.161
	Sig. (2-tailed)	.522	.491	.435	.453
	Ν	24	24	24	24
vents	Pearson Correlation	.368	.350	.236	.195
	Sig. (2-tailed)	.077	.094	.266	.360
	Ν	24	24	24	24

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Appendix 14

	Contractor of the Contractor				
		FrRXuFpoa	FrRXnFpoa	FrRXuFprede	FrRXnFprede
nightmares each year	Pearson Correlation	378	365	.027	.110
	Sig. (2-tailed)	.068	.079	.900	.610
	N	24	24	24	24
:Nm	Pearson Correlation	293	256	.043	.268
	Sig. (2-tailed)	.165	.227	.840	.205
	N	24	24	24	24
htmare distress	Pearson Correlation	376	313	.064	026
	Sig. (2-tailed)	.070	.137	.765	.904
	N	24	24	24	24
usal scale	Pearson Correlation	157	111	.010	.062
	Sig. (2-tailed)	.464	.605	.962	.772
	N	24	24	24	24
ative experiences	Pearson Correlation	427*	404	.086	.036
est	Sig. (2-tailed)	.037	.050	.688	.869
	N	24	24	24	24
derline personality	Pearson Correlation	493*	447*	.163	.098
	Sig. (2-tailed)	.014	.028	.446	.650
	N	24	24	24	24
senck Neuroticism	Pearson Correlation	453*	423*	181	230
ale	Sig. (2-tailed)	.026	.039	.397	.280
	N	24	24	24	24
senck Lie Scale	Pearson Correlation	.623**	.584**		232
	Sig. (2-tailed)	.001	.003	.408	.275
	N	24	24	24	24
eneral Health	Pearson Correlation	213	172	.160	.279
estionnaire	Sig. (2-tailed)	.317	.421	.456	.187
	N	24	24	24	24
rtmann Boundary	Pearson Correlation	349	344	.041	.020
lest	Sig. (2-tailed)	.095	.100	.851	.925
	N	24	24	24	24
ate Anxiety	Pearson Correlation	370	371	.032	.084
	Sig. (2-tailed)	.262	.261	.925	.806
	Ν	11	11	11	11
ait Anxiety	Pearson Correlation	428	400	079	142
	Sig. (2-tailed)	.053	.073	.734	.540
	Ν	21	21	21	21
obal Severity Idex	Pearson Correlation	272	239	.242	.229
	Sig. (2-tailed)	.199	.260	.255	.282
	N	24	24	24	24
sitive Symptom Total	Pearson Correlation	353	331	.082	.049
	Sig. (2-tailed)	.090	.114	.702	.819
	Ν	24	24	24	24
sitive Symptom	Pearson Correlation	039	004	.247	.211
stress Index	Sig. (2-tailed)	.858	.986	.245	.322
	N	24	24	24	24

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Correlations

		FrRXuFpoa	FrRXnFpoa	FrRXuFprede	FrRXnFprede
nof 4 childhood items	Pearson Correlation	205	120	.177	.129
	Sig. (2-tailed)	.337	.578	.408	.548
	N	24	24	24	24
events	Pearson Correlation	.010	018	.405*	.317
	Sig. (2-tailed)	.962	.934	.050	.131
	N	24	24	24	24

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		FrRXupod	FrRXnpod	FrRXuFpod	FrRXnFpod
nightmares each year	Pearson Correlation	111	249	058	232
	Sig. (2-tailed)	.605	.241	.788	.275
	N	24	24	24	24
e Nm	Pearson Correlation	071	190	129	031
	Sig. (2-tailed)	.740	.373	.548	.887
	N	24	24	24	24
htmare distress	Pearson Correlation	324	361	313	340
	Sig. (2-tailed)	.122	.083	.137	.105
	Ν	24	24	24	24
usal scale	Pearson Correlation	.128	.106	.198	.177
	Sig. (2-tailed)	.551	.621	.354	.408
	N	24	24	24	24
ative experiences	Pearson Correlation	.050	.182	.068	.206
est	Sig. (2-tailed)	.816	.394	.753	.335
	N	24	24	24	24
derline personality	Pearson Correlation	.086	.191	102	.004
	Sig. (2-tailed)	.689	.370	.636	.986
	N	24	24	24	24
senck Neuroticism	Pearson Correlation	270	279	320	290
ale	Sig. (2-tailed)	.203	.187	.128	.169
	N	24	24	24	24
senck Lie Scale	Pearson Correlation	027	038	.065	139
	Sig. (2-tailed)	.901	.859	.764	.516
	N	24	24	24	24
eneral Health	Pearson Correlation	189	008	253	246
lestionnaire	Sig. (2-tailed)	.376	.971	.232	.247
	N	24	24	24	24
artmann Boundary	Pearson Correlation	043	.062	047	.022
Jest	Sig. (2-tailed)	.843	.772	.828	.918
	N	24	24	24	24
ate Anxiety	Pearson Correlation	209	189	052	167
	Sig. (2-tailed)	.537	.577	.879	.624
	N	11	11	11	11
ait Anxiety	Pearson Correlation	149	044	207	218
	Sig. (2-tailed)	.519	.851	.369	.343
	N	21	21	21	21
lobal Severity Idex	Pearson Correlation	130	160	082	106
	Sig. (2-tailed)	.546	.457	.702	.622
	N	24	24	24	24
sitive Symptom Total	Pearson Correlation	210	137	146	174
, , , , , , , , , , , , , , , , , , , ,	Sig. (2-tailed)	.325	.524	.496	.416
	N	24	24	24	24
sitive Symptom	Pearson Correlation	022	.084	.030	033
stress Index	Sig. (2-tailed)	.919	.695	.890	.878
	N	24	24	24	24

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Correlations

	Annexes des 194 Annexes and 194				al de comé in
	1011 martine	FrRXupod	FrRXnpod	FrRXuFpod	FrRXnFpod
n of 4 childhood items	Pearson Correlation	110	.152	218	001
	Sig. (2-tailed)	.610	.478	.307	.996
	N	24	24	24	24
events	Pearson Correlation	.267	.226	.357	.287
	Sig. (2-tailed)	.207	.288	.087	.174
	Ν	24	24	24	24

Correlation is significant at the 0.05 level (2-tailed).

Correlation is significant at the 0.01 level (2-tailed).

relations

Difference between nightmares and	Wilcoxon test statistic
bad dreams	
Frequency of anxiety/fear	z = -2.99*(0.03)
Frequency of anger	z = -0.76 (0.44)
Frequency of shame	z = 0.12 (0.91)
Frequency of sad	z = -1.64 (0.10)
Frequency of joy	z = -0.76 (0.44)
Frequency of love	z = -1.25 (0.21)
Frequency of surprise	z = -1.82 (0.07)
Intensity of anxiety/fear when present	z = -3.75 (<.01)
Intensity of anger when present	z = -0.12 (0.99)
Intensity of shame when present	
Intensity of sad when present	z = -1.97 (0.05)
Intensity of joy when present	z = -0.95 (0.34)
Intensity of love when present	z = -1.27 (0.20)
Intensity of surprise when present	z = -0.97 (0.33)
Intensity of anxiety/fear per dream	z = -1.75 (0.08)
Intensity of anger per dream	z = -2.05 (0.04)
Intensity of shame per dream	z = -0.38 (0.70)
Intensity of sad per dream	z = 1.92 (0.05)
Intensity of joy per dream	z = -2.23 (0.03)
Intensity of love per dream	z = -0.45 (0.65)
Intensity of surprise per dream	z = -1.21 (0.22)

Appendix Table 15 - Difference between nightmares and bad dreams in frequency and intensity of emotions

Sheet for sherdy 2. CD Carmarthenshire NHS TRUST Prince Philip Hospital, Bryngwynmawr Dafen, Llanelli. SA14 8QF

Ysbyty Tywysog Philip, Bryngwynmawr Dafen, Llanelli. SA14 8QF

Telephone: 01554 756567 Fax: (01554) 772271 WHTN: 1824

DEPARTMENT OF RESPIRATORY MEDICINE

YMDDIRIEDOLAETH GIG Sir Gaerfyrddin

Our Ref/Ein Cyf:KL/ADBDirect Line:01554 783133Fax Line:01554 772271Email: alison.baker@carmarthen.wales.nhs.uk

Patient's Initials: Date: E No.

PATIENT INFORMATION SHEET: DREAM RECALL AND SLEEP APNOEA

Dear Sir/Madam

. . . /

We are researching quality and frequency of dreams in patients who may be suffering from obstructive sleep apnoea.

If you are willing please could you take home the enclosed sheets and report any dreams for the next ten days. Please bring these sheets back with you when you attend for your sleep study. We would like to repeat these questionnaires again for ten days only when on treatment should you require CPAP.

All information is kept strictly confidential and no individuals can be identified in any final analysis. You are free to withdraw at any time and this will not affect your ongoing treatment in any way.

For more information, please contact either the above number or Samantha Fisher on 01792 205678 Ext. 4616.

Thank you very much indeed for any help you can offer.

Yours sincerely

Dr Keir LEWIS Senior Lecturer and **Consultant in Respiratory Medicine**

Samantha FISHER Post-graduate Researcher Swansea University

Margret Price OBE, MA Diairmon/Codeirydd





Paul M. Barnett Chief Executive/Prif Weithredwr

Appendix 17: Patient consent form for study 2.

Version 01 08/03/05

Patient Consent Form

Title: Dream recall in patients with sleep apnoea before and after treatment with CPAP.

Centre number

Study number

Patient identification number for this trial.....

Name of researchers: Ms Samantha Fisher, Dr Keir Lewis, Robin Ghosal & Dr. MarkBlagrove.

Contact telephone number: University of Wales Swansea. Ms. Samantha Fisher- 01792 205678 (ext 4616) Dr. Mark Blagrove – 01792 295586 Dr Keir Lewis – 01554 756567

Please initial Box

- 1. I confirm that I have read and understood the information sheet dated.....) for the above study and have had the opportunity to ask questions.
- 2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.
- 3. I understand that the study is non therapeutic and for research purposes only.
- 4. I agree to take part in the above study.

 	-	 -

Date

Signature

Name of Person taking consent

Date

Signature

Researcher

Date

Signature

sleep aprova study

Hospital No.

Day 1

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very	moderately	slightly	neither or	slightly	moderately pleasant	very
unpleasant	unpleasant	unpleasant	mixed	pleasant		pleasant
1	2	3	4	5	6	7

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

Please could you indicate how depressed or happy you feel, and how anxious or relaxed you feel. It may help you to look at the two sets of words under each line; they are related to the four main emotions. Please put a cross on the line according to how you feel.

RELAXED	very	quite	never	quite	very ANXIOUS
e.g. you may feel untroubled, peace					e.g. you may feel tense, nervous jittery, shaky, uneasy
НАРРҮ	very	quite	never	quite	Very DEPRESSED
e.g. you may feel playful, light-hea	l, cheerful, el arted, joyful	ated,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Day 2

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very	moderately	slightly	neither or	slightly	moderately	very
unpleasant	unpleasant	unpleasant	mixed	pleasant	pleasant	pleasant
1	2	3	4	5	6	7

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

RELAXED	very	quite	never	quite	Very ANXIOUS
e.g. you may fee untroubled, peac					e.g. you may feel tense, nervous jittery, shaky, uneasy
НАРРҮ	very	quite	never	quite	DEPRESSED
e.g. you may fee playful, light-he		ed,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Day 3

Patient's initials:

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very unpleasant	moderately unpleasant	slightly unpleasant	neither or mixed	slightly pleasant	moderately pleasant	very pleasant	
1	2	3	4	5	6	7	

Hospital No._

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

Please could you indicate how depressed or happy you feel, and how anxious or relaxed you feel. It may help you to look at the two sets of words under each line; they are related to the four main emotions. Please put a cross on the line according to how you feel.

	very	quite	never	quite	very
RELAXED					ANXIOUS
e.g. you may feel untroubled, peace				1	e.g. you may feel tense, nervous jittery, shaky, uneasy
HAPPY	very	quite	never	quite	DEPRESSED
e.g. you may feel playful, light-hea	, cheerful, elate rted, joyful	d,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Day 4

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very unpleasant	moderately unpleasant	slightly unpleasant	neither or mixed		moderately pleasant	-
1	2	3	4	5	6	7

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

	very	quite	never	quite	very
RELAXED					ANXIOUS
e.g. you may feel	serene, calr	n			e.g. you may feel tense, nervous
untroubled, peace	eful, compos	sed			jittery, shaky, uneasy
	verv	quite	never	quite	very
HAPPY	vory	quite	never	quite	DEPRESSED
e.g. you may feel playful, light-hea		lated,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Hospital No.

Day 5

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very	moderately	slightly	neither or	slightly	moderately	very
unpleasant	unpleasant	unpleasant	mixed	pleasant	pleasant	pleasant
1	2	3	4	. 5	6	7

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

Please could you indicate how depressed or happy you feel, and how anxious or relaxed you feel. It may help you to look at the two sets of words under each line; they are related to the four main emotions. Please put a cross on the line according to how you feel.

RELAXED	very	quite	never	quite	very ANXIOUS
e.g. you may fee untroubled, peac					e.g. you may feel tense, nervous jittery, shaky, uneasy
HAPPY	very	quite	never	quite	VETY DEPRESSED
e.g. you may fee playful, light-hea		lated,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Day 6

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very	moderately	slightly	neither or	slightly	moderately	very
unpleasant	unpleasant	unpleasant	mixed	pleasant	pleasant	pleasant
1	2	3	4	5	6	7

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

RELAXED	very	quite	never	quite	ANXIOUS
e.g. you may fee untroubled, peac					e.g. you may feel tense, nervous jittery, shaky, uneasy
НАРРҮ	very	quite	never	quite	very DEPRESSED
e.g. you may fee playful, light-he		lated,			e.g. you may feel sad, dejected,

Hospital No.

Dav 7

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very unpleasant	moderately unpleasant	slightly unpleasant	neither or mixed	slightly pleasant	moderately pleasant	very pleasant	
1	2	3	4	5	6	7	

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

Please could you indicate how depressed or happy you feel, and how anxious or relaxed you feel. It may help you to look at the two sets of words under each line; they are related to the four main emotions. Please put a cross on the line according to how you feel.

RELAXED	very	quite	never	quite	very ANXIOUS
e.g. you may feel untroubled, peace					e.g. you may feel tense, nervous jittery, shaky, uneasy
HAPPY	very	quite	never	quite	very DEPRESSED
e.g. you may feel playful, light-hea	l, cheerful, e rted, joyful	lated,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Dav 8

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very	moderately	slightly	neither or	slightly	moderately	very	
unpleasant	unpleasant	unpleasant	mixed	pleasant	pleasant	pleasant	
1	2	3	4	.5	6	7	

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

	very	quite	never	quite	. very
RELAXED					ANXIOUS
e.g. you may fee untroubled, peac					e.g. you may feel tense, nervous jittery, shaky, uneasy
	very	quite	never	quite	very
HAPPY			-1		DEPRESSED
e.g. you may fee playful, light-he		ated,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Hospital No.

Day 9

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very	moderately	slightly	neither or	slightly	moderately	very
unpleasant	unpleasant	unpleasant	mixed	pleasant	pleasant	pleasant
1	2	3	4	5	6	7

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

Please could you indicate how depressed or happy you feel, and how anxious or relaxed you feel. It may help you to look at the two sets of words under each line; they are related to the four main emotions. Please put a cross on the line according to how you feel.

	very	quite	never	quite	very
RELAXED	a la company	n nd nashidili	he ward tree	and Diverse	ANXIOUS
e.g. you may feel untroubled, peace		d	nuve enclose et to us	ol e sieme	e.g. you may feel tense, nervous jittery, shaky, uneasy
НАРРҮ	very	quite	never	quite	very DEPRESSED
e.g. you may feel playful, light-hea	, cheerful, ela rted, joyful	ited,	o ogudeo. Hi y od send the	ord multiple it	e.g. you may feel sad, dejected, lonely, downhearted, gloomy

Day 10

1. Did you dream last night? Yes / No

2. How would you rate the overall mood of the dream or dreams?

very	moderately	slightly	neither or	slightly	moderately	very
unpleasant	unpleasant	unpleasant	mixed	pleasant	pleasant	pleasant
1	2	3	4	5	6	7

3. Did the unpleasant emotions or events in the dream wake you up? Yes / No

RELAXED	very	quite	never	quite	Very ANXIOUS
e.g. you may fea untroubled, pea					e.g. you may feel tense, nervous jittery, shaky, uneasy
НАРРҮ	very	quite	never	quite	very DEPRESSED
e.g. you may fe playful, light-he		lated,			e.g. you may feel sad, dejected, lonely, downhearted, gloomy

librurie it become at ions and a set

8/3/05 Version 1

Follow up letter to be sent 3 months after CPAP has started.

Dear

Thank you for taking part in the research project assessing sleep and dreaming. As you may recall it is our intention to continue this research by assessing any changes in sleep following treatment with continuous positive airway pressure (CPAP). I have enclosed a questionnaire and would be very grateful if you could complete the questions again daily for 14 days. I have enclosed a stamped addressed envelop so that the questionnaire can be returned to us.

May I take this opportunity to thank you for all your help with the project. A copy of the findings will be available in due course. If you would like us to send you a copy please complete the details below and send the slip back to us.

Thank you,

Yours sincerely,

Samantha Fisher

.....X...

Yes I would like a summary of the research findings.

Name :

Address:

PRIFYSGOL CYMRU ABERTAWE

Yr Adran Seicoleg Parc Singleton, Abertawe SA2 8PP



UNIVERSITY OF WALES SWANSEA

Department of Psychology Singleton Park, Swansea SA2 8PP

Appendix 20: Departmental enical approval.

Samantha Fisher Department of Psychology

05/05/2004

Dear Samantha,

I am writing to inform you that the Departmental Ethics Committee has considered your application "Dream recall and sleep apnoea", to be carried out in collaboration with Dr. Keir Lewis. I am happy to say that the application has been approved.

Good luck with the study.

Yours sincerely,

Professor David Clark Chair of Ethics Committee

> Pennaeth yr Adran · **Professor Rodger Ll. Wood** · Head of Department Tel 01792 295278 Fax 01792 295679 Email psychology@swansea.ac.uk

Hependax 20:

Ethical Committee application

Title of the investigation: Dream recall and Sleep Apnoea

<u>Names and statuses of investigators:</u> Dr. Keir Lewis (Senior Lecturer and Consultant in Respiratory Medicine) Dr. Mark Blagrove and Samantha Fisher.

Category of application:

Practical class - program of study - single experiment- grant application -other (please specify) routine - non-routine.

A 'single experiment' which is considered to be 'routine'.

Brief description of purposes:

A study conducted by Gross and Lavie (1994) investigated the impact of successful treatment of apnoeas on dreaming. Although they found that there was no significant systematic incorporation of the apnoea stimulus into the dream reports they reported that dreams after apnoeas were significantly more negative than after healthy sleep. This significant difference in 'emotional colouring' was found to be independent of dream length. Very little research has assessed the frequency of nightmares in sleep apnoea patients. We suggest that nightmare frequency may take a U shape function where patients with apnoea of 'moderate' severity are most at risk from nightmares. We suggest that patients with less severe apnoea may not experience sufficient distress for the occurrence of nightmares, whereas, the sleep of those with severe apnoea may be too fragmented to allow for the occurrence of REM nightmares. Furthermore, we are interested in the how levels of anxiety and depression are affected by CPAP treatment.

Methods:

Patients who attend Dr Lewis' clinic at Prince Phillip Hospital, Llanelli will be asked if they are willing to complete a dream diary for 10 days before and after treatment with continuous positive airway pressure (CPAP). Each morning patients will be asked to state if they had a dream or nightmare (no content is required) and how anxious or depressed they feel on two visual analogue scales.

<u>Ethical considerations:</u> Does the study involve any of the potentially controversial procedures listed below?

Administration of drugs - collection of body fluids or tissue – unpleasant stimulation or procedures-collection of confidential information – deprivation – active deception – withholding information – payment

Are any other potentially controversial procedures involved? Please specify? Indicate how it is intended to minimise any risk of harm or distress, which could arise from each identified procedure.

Patients will be informed that all information is kept strictly confidential and that no individual can be identified from the final analysis.

It will be made clear to patients that their names may be stored on a computer whilst the study is in progress but will be destroyed afterwards. Data will be registered under the data protection act. It will be emphasised to patients that they do not have to take part in the study and can refuse to do so if they wish without giving a reason. Patients will be informed of their right to withdraw from the study at any time and that this will not affect their ongoing treatment in any way.

Informed consent: Please state how informed consent will be obtained. Attach copies of the consent form to be used plus any participant information sheets.

A consent form and patient information form will be issued to all participants. which must be signed before the study commences (see appendices). Participants will be told exactly what they are required to do and informed that they may withdraw from the study at any time. Again they will be assured full confidentiality.

This study raises to ethical issues that I can see

1 ml 4/5/04

Committee + R+D approval.



Canolfan Gwasanaethau Busnes Business Services Centre

Ms Samantha Fisher Department of Psychology University of Wales Swansea Singleton Park Swansea SA2 8PP

15 April 2005

Dear Ms Fisher,

Full title of study: Dream frequency in patients with sleep apnoea before and after treatment with CPAP REC reference no: 05/WMW01/20

Thank you for your letter of 12 April 2005, responding to the Committee's request for further information on the above research [and submitting revised documentation].

The further information has been considered on behalf of the Committee by the Chairman.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation [as revised].

The favourable opinion applies to the following research site:

Site: Carmarthenshire NHS Trust Principal Investigator: Ms Samantha Fisher, PhD Student

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

- Application form
- CV
- Protocol, version 1, 8/3/05
- Invitation letter to sleepy control participants, version 2, 11/4/05
- Follow-up letter, version 1, 8/3/05
- Letter to GP, version 1, 8/3/05
- Patient Consent Form, version 2, 11/4/05
- Patient Consent Form (control group 1), version 2, 11/4/05
- Patient Consent Form (control group 2), version 2, 11/4/05

SOPs version 1.0 dated February 2004Canolfan Gwasanaethau BusnesSL15Favourable opinion following costsyde Batter further information

NHS WALES GIG CYMRU GJA O' V Caerfyrddin Sir Gaerfyrddin, SA31 3YH Ffôn: 01267 225225 WHTN: 1820 Ffacs: 01267 225060 DX QW3DYF 121550 Business Services Centre St David's Hospital Carmarthen Carmarthenshire, SA31 3YH Telephone: 01267 225225 WHTN: 1820 Fax: 01267 225060 DX QW3DYF 121550

rhan o Bwrdd lechyd Lleol Powys / part of Powys Local Health Board

- Patient Information Sheet, version 2, 11/4/05
- Patient Information Sheet (control group 1), version 2, 11/4/05
- Patient Information Sheet (control group 2), version 2, 11/4/05
- Questionnaire, version 2, 11/4/05

Management approval

The study may not commence until final management approval has been confirmed by the organisation hosting the research.

All researchers and research collaborators who will be participating in the research must obtain management approval from the relevant host organisation before commencing any research procedures. Where a substantive contract is not held with the host organisation, it may be necessary for an honorary contract to be issued before approval for the research can be given.

Notification of other bodies

We shall notify the research sponsor [the host organisation] that the study has a favourable ethical opinion.

Statement of compliance (from 1 May 2004)

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

REC reference number: 05/WMW01/20

Please quote this number on all correspondence

Yours sincerely,

Dr Mark Turtle Chairman

Enclosures Standard approval conditions [SL-AC1 or SL-AC2]

Copy:

Host organisation

SOPs version 1.0 dated February 2004 SL15 Favourable opinion following consideration of further information



Carmarihenshire NHS TRUST

YMDDIRIEDOLAETH GIG Sir Gaerfyrddin

West Wales General Hospital Carmarthen SA31 2AF

Ysbyty Cyffredinol Gorllewin Cymru Caerfyrddin SA31 2AF

Telephone: 01267 235151 WHTN: 1827

RESEARCH & DEVELOPMENT OFFICE

Director: Dr Imroz Salam R&D Officer: Ingaret Eden, ext 2149 Secretary: Alison May, ext 2149

email: ingaret.eden@carmarthen.wales.nhs.uk

24 August 2005

Samantha Fisher Postgraduate Researcher Psychology Department Swansea University Singleton Park Singleton SWASEA

Dear Samantha

Project title: Nightmare frequency in patients with Sleep Apnoea before and after treatment with CPAP R&D Reference: 2005.010

We write to confirm that the above project has received approval from Carmarthenshire NHS Trust to proceed and we enclose your application form duly signed by Dr Imroz Salam, director of R&D. Trust approval is subject to the project receiving approval from the Local Research Ethics Committee, and we acknowledge receipt of their letter of favourable opinion dated 29 March 2005 reference 05/WMW01/20.

Under Research Governance, and as lead researcher, you are required to:

- Adhere to the protocol approved by the LREC and inform the R&D Office of any changes (including changes to the end date of the project) and any changes you refer to the Local Research Ethics Committee
- 2. Inform the R&D Office of any adverse events that may occur, whilst also reporting these through the proper channels in the Trust.
- 3. Complete any interim and final reports requested by the R&D office. These are required every six months, or at the end of the project if sooner.
- 4. Comply with the Research Governance Framework and co-operate with any audit inspection of the project files. We enclose an audit checklist which you may find useful.
- 5. Ensure that your research complies with the Data Protection Act 1998. The Trust Data Protection Officer and the Caldicott Guardian have approved the project.

Please note that R&D approval from this Trust does not give approval for the research to take place at any other Trust: if you wish to extend the project to another Trust, local R&D approval must be sought and the local research ethics committee contacted for advice.

We enclose a summary of your responsibilities as lead researcher under the Research Governance Framework.

With all good wishes for your project.

Yours sincerely

Ingaret Eden **R&D** Officer

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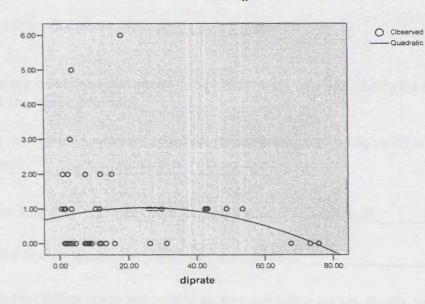




Paul M. Barnett Chief Executive/Prif Weithredwr

The Trust welcomes correspondence in Welsh or English Y mae'r Ymddiriedolaeth yn croesawu gohebiaeth yn Gymraeg neu yn Saesneg

Appendix 22 - Graph showing the quadratic regression curve between dip rate measure and nightmare frequency for all participants



nmfreqpre

Appendix 23: Retrospective dream questionnaire used in study 3.

Gender	Male / Female Age
Job titl	e
Numbe	er of years in current postyears
	llowing questions ask about your sleep on an average night (for a rest day), please r in the space provided
•	On average (on a rest day), what time do you usually turn the lights off in order to go to sleep?
•	How long does it normally take you to fall asleep (on a rest day)?
	What time do you normally wake up (on a rest day)?
•	Between falling asleep and waking up, how much time are you usually awake during the night? hours and minutes
•	How is your sleep normally (on a rest day)? Please circle on of the following responses.
1.	very poor
2.	rather poor
3.	neither poor nor good
4.	rather good
5.	very good
	How many dreams do you have each week?
•	If you do not have one dream per week how many do you have a month
•	On average how would you rate the overall mood of your dreams? Please circle on of the following responces
	very moderately slightly neither or slightly moderately very unpleasant unpleasant mixed pleasant pleasant pleasant pleasant 1 2 3 4 5 6 7

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Yr Adran Seicoleg Parc Singleton, Abertawe SA2 8PP

UNIVERSITY OF WALES SWANSEA

Department of Psychology Singleton Park, Swansea SA2 8PP

Version 03 17/03/04

Patient Consent Form

Centre number Study number Patient identification number for this trial.....

Name of researchers: Ms Samantha Fisher, Dr. Mark Blagrove and Professor Rodger Wood.

Contact telephone number: University of Wales Swansea. Ms. Samantha Fisher- 01792 205678 (ext 4616) Dr. Mark Blagrove – 01792 295586 Professor Rodger Wood – 01792 295778

- 1. I confirm that I have read and understood the information sheet dated.....) for the above study and have had the opportunity to ask questions.
- 2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.
- 3. I understand that sections of any of my medical notes may be Looked at by the three names researchers; Professor Rodger Wood, Dr. Mark Blagrove and Ms. Samantha Fisher. I give permission for these individuals to have access to my records.
- 4. I understand that the study is non therapeutic and for research purposes only.
- 5. I agree to take part in the above study.

Name of Patient	Date	Signature
Name of Person taking consen	t Date	

Researcher

Date

Signature

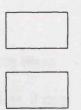
Pennaeth yr Adran · Professor Rodger Ll. Wood · Head of Department Tel 01792 295278 Fax 01792 295679 Email psychology@swansea.ac.uk

Please initial Box



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Canolfan Gwasanaethau Busnes Business Services Centre

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Dr M Blagrove, Professor R Wood & Ms S Fisher University of Wales Swansea Department of Psychology Singleton Park SWANSEA SA2 8PP

LOCAL RESEARCH ETHICS COMMITTEE

Miss Lawmary Champion Protocol No.: <u>Please always Ouote</u>

(01792) 458066 Ext.7416 Direct Line 01792 607416

19 March 2004

Dear Dr Blagrove, Professor Wood and Ms Fisher

2004. 021 : Sleep quality and dreaming after head injury

Thank you for your undated letter received on 25th February 2004, enclosing Amendments requested by the Committee. The following documents have now been approved and registered: Application Form Version 2 dated 4/03/04; Patient Information Form for the control group Version 3 dated 17/3/04; Patient Information Sheet for those who have suffered brain injury Version dated 17.3.04; Consent Form Control Group Version 3 dated 17.3.04 and Consent (Head Injury) Form Version 3 dated 17.3.04. This Study has now been approved.

Please quote our Reference Number in all future correspondence.

Please also note:

- 1 The enclosed document is confidential and not for publication
- 2 Any publication resulting from the Protocol must define how subjects were chosen and to what extent they were volunteers.
- 3 That the form of consent must be read and signed by each subject or, if oral consent has been approved by the Committee, that the consent of each subject must be appropriately recorded. In either case, forms and records must be kept for subsequent examination, if required, by the Committee
- 4 That changes to the Protocol as approved must be referred to the Committee
- 5 Ethical approval does not imply acceptance of materials and drug costs by the Authorities or provider units
- 6 Any untoward incident which occurs in connection with this Protocol must be reported back to the Chairman of the Committee **without delay**.

Yours sincerely

NICOLA JOHN - CONSULTANT IN PHARMACEUTICAL PUBLIC HEATH NATIONAL PUBLIC HEALTH SERVICE & SECRETARY OF THE LOCAL RESEARCH ETHICS COMMITTEE

cc Mr Dorian Edwards R & D Support Office

Canolfan Gwasanaethau Busnes 41 Stryd Fawr Abertawe, SA1 1LT Ffôn: 01792 458066 WHTN: 1780 Ffacs: 01792 607533 DX 121810, Abertawe 7

Business Services Centre 41 High Street Swansea, SA1 1LT Telephone: 01792 458066 WHTN: 1780 Fax: 01792 607533 DX 121810, Swansea 7



ť	ippendix 2	o Dream o Study 4	queshormanne	e for	
Date					
Please read the fo	llowing questions a	nd put a tick in the	appropriate box.		
• How often do	you wake up and re	emember a dream?			
at least once a week	a least once month	at least once a year	less than once a year	never	
 <u>A nightmare is a vivid dream that is frightening or disturbing, the events of which you can remember clearly and in detail on awakening.</u> <u>How often do you have a nightmare?</u> 					
at least once a week	a least once month	at least once a year	less than once	never	
			a year		
 Is having a r yes 	no	or concern for you?	2		
Have you recently had the same nightmare more than once?					
yes	no				
	s a sudden awakenin ot remember any dre		ccompanied by a screa	. <u>m. but</u>	
• How often of	do vou have night te	ntors?			
at least once a week	a least once month	at least once a year	less than once a year	never	
 Please could you state any medication you are taking below: 					