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Behavioral resurgence in individuals varying in depression, anxiety, and autism-associated tendencies

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ABSTRACT

Resurgence is the reappearance of a previously reinforced, but then extinguished behavior, when an alternative behavior that has been reinforced to replace it is also extinguished. This phenomenon has been suggested as important in the re-occurrence of many clinical problems, but little is known currently about the relationship between this process and different psychopathological traits. This experiment addressed this gap by comparing the levels of resurgent behavior in participants scoring lower or higher on depression-, anxiety-, and autism-related characteristics. Sixty participants completed an experimental task of three phases. In the first, they were presented with a concurrent RR-5 ext schedule, in the second with a conc ext RR-5 schedule (each lasting 6min), and finally with a conc ext ext schedule (lasting 2 min). Following this, all participants completed the Beck Depression Inventory, State-Trait Anxiety Inventory, and Autism Quotient, questionnaires provided. Participants showed a resurgence of responding at test from the response extinguished in Phase 2 that was greater for those with lower levels of depression, but high levels of anxiety. These findings are discussed in terms of their implications for understanding individual differences in terms of psychiatric symptomatology, for their treatment, and in terms of theoretical predictions derived for the various psychopathologies.

1. Introduction

Developing potential models for understanding why many clinically-relevant and problematic behaviors re-emerge into behavior, sometimes after they have been removed through treatments like exposure therapies, is a key focus of translational research (Doughty and Oken, 2008; Reed and Clark, 2010; Zhao et al., 2007). There are many situations where apparently extinguished behaviors re-emerge or re-occur, which have been the subject of extensive reviews (Bouton, 2004; Bouton et al., 2012; Lattal and Wacker, 2015). Particular situations that have been well studied include: 'renewal', 'reinstatement', and 'spontaneous recovery' (see Bouton, 2004; Lattal and Wacker, 2015): 'renewal' refers to when changing contexts following extinction produces a reoccurrence of the behaviour (http://learnmem.cshlp.org/content/11/5/485.full Bouton and Bolles, 1979); 'reinstatement' refers to the reoccurrence of extinguished behaviour after exposure to the reinforcer alone (http://learnmem.cshlp.org/content/11/5/485.full Pavlov, 1927; http://learnmem.cshlp.org/content/11/5/485.full Rescorla and Heth, 1975), and 'spontaneous recovery' refers to the reoccurrence of an extinguished response after a delay (Pavlov, 1927). All of these situations have clinical relevance (Bouton et al., 2012; Lattal and Wacker, 2015).

A further situation in which behavior reoccurs after extinction is when an alternative behavior, that has been reinforced to replace it, is also extinguished. This phenomenon is known as 'resurgence' (Cleland et al., 2001), and has been observed in nonhumans (Bachá-Méndez et al., 2007; Reed and Morgan, 2007), and humans (Bruzek et al., 2009; Dixon and Hayes, 1998; McHugh et al., 2012; Reed and Clark, 2010). Importantly, although there is some research regarding resurgence (see Doughty and Oken, 2008; Kestner and Peterson, 2017, Lattal et al., 2017, for reviews), it is relatively understudied compared to 'reinstatement' and 'spontaneous recovery' (Lerman and Iwata, 1996). This presents a significant gap in the knowledge-base, as many behaviors can resurge, and the issue can become clinically-problematic if these behaviors are harmful to the individual, or to others around them (Kestner and Peterson, 2017; Lattal and Wacker, 2015).

Experimental work strengthens these concerns about the clinical importance of resurgent behaviors (Kestner and Peterson, 2017). For example, Podlesnik et al. (2006; see also Zhao et al., 2007) trained rats to self-administer alcohol, delivery of which was then discontinued, and was replaced by a non-drug reinforcement for an alternative behavior. When the non-drug reinforcement was stopped, the original alcohol self-administration behavior resurged despite alcohol not being available

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as a reinforcement. Podlesnik et al. (2006) argued that these resurgence findings suggested this form of control over behavior underlies human drug-seeking.

Undesirable re-emergent behaviors are also not just limited to those that are drug induced. Lieving, Hagopian, Long, and O'Connor (2004) tested participants with histories of problem behaviors, and noted that, when one of these behaviors (e.g., disruptiveness) had been reinforced and then extinguished, it would resurge when other problem behaviors (e.g., aggression) were themselves extinguished. Lieving et al. (2004) highlighted resurgence as an important limitation for treatment for undesirable behavior, often overriding therapeutic efforts to remove problem behaviors. The importance of resurgence as a potential limitation for clinical treatments has been noted in many contexts (see Kestner and Peterson, 2017; Smith et al., 2017, for discussions).

However, before a successful therapeutic approach to combat the effects of resurgence can be developed, the prevalence of this behavioral mechanism needs examination within the context of a broader range of individual variations in psychiatric symptomatology. The moderation and/or mediation of treatment efficacy by individual variations in symptomatology is increasingly recognized as an important contributor to outcome effectiveness across a range of therapeutic situations (Craske et al., 2014; Khan et al., 2013; Schneider et al., 2015). Resurgence has been suggested as important to facets of a number of psychological problems; in particular, it has been related to depression (Wenzlaff et al., 1988), anxiety (Smith et al., 2017), and aspects of developmental disorders (Volkert et al., 2009; Wacker et al., 2013). Unfortunately, the lack of basic research into resurgence within this context is a hindrance to furthering development of understanding of the relationship of resurgence to many such disorders and their treatments. Given this gap in knowledge, the current experiment aimed to investigate whether varying levels of exhibited sub-clinical depression, anxiety, and autistic characteristics has an impact on the degree of behavioral resurgence expressed.

Depression and anxiety appear appropriate to study as a starting point in this line of investigation, firstly due to their relative prevalence in clinical context, and secondly, as there are some theoretical reasons to suspect an impact on resurgence in those exhibiting these traits. It might be predicted that resurgence would not be seen to such an extent in those with higher levels of depression. From nonclinical laboratory studies, it is known that individuals who previously have received higher reinforcement rates are more susceptible to resurgence (Smith et al., 2017). If depressed individuals are not as sensitive to reinforcement, due to their anhedonic tendencies (Alloy et al., 2016), this may reduce the size of any resurgence effect, and/or make them less sensitive to the fact that contingencies have altered.

In terms of anxiety, there are several possible theoretical predictions. It has been suggested that anxious individuals display reduced levels of reward sensitivity, leading to lower rates of responing (Torrubia et al., 2001). Given this theoretical suggestion, less resurgence would be predicted for those with higher anxiety, as it is for those with depression. However, those showing greater anxiety also have been suggested to show increased behavioral inhibition (Gray, 1987), which would also reduce rates of response, but may not lead to a differential perception of reinforcement rates, and no differential resurgence.

With respect to Autism Spectrum Disorder (ASD), the association of this disorder with high levels of behavior problems (Reed, 2015b) warrants investigation in the context of resurgence (Lieving et al., 2004). Moreover, there have been two reports concerning resurgence using children with ASD (Reed and Clark, 2010; Volkert et al., 2009), both of which demonstrated the existence of the phenomenon in this population. However, neither study (Reed and Clark, 2010; Volkert et al., 2009) compared levels of resurgence in their sample of children with ASD to a comparison group without the disorder.

Given the need of the study, and the above theoretical reasons for focusing on these disorders, the participants in this study completed a three-phase experiment. In the first phase, participants received reinforcement (points) for responding to one colored stimulus, by clicking the mouse on a random ratio (RR) 5 schedule, whilst responding to another key was not reinforced (i.e., an extinction schedule). In the second phase, the contingencies were reversed; so that responding was reinforced for the colored stimulus that initially received no reinforcement, whilst no reinforcement could be gained by responding to the colored stimulus that was previously reinforced. In the final phase, both responses were placed into extinction. The purpose of this final phase was to examine the extent to which the behavior, conditioned in the first phase, resurged into the behavioral repertoire of the participants. It was assumed that resurgence would be reduced for individuals with higher levels of depression, but would be present (and perhaps higher) for those with higher levels of ASD traits. The prediction for anxiety is clear cut, and depends on whether reinforcement sensitivity or behavioral inhibition dominate the response pattern of those with anxiety. Such information may begin to develop an understanding of how various psychopathologies and behavioral resurgence interact, which has relevance to models of disorders and treatment (Bouton et al., 2012; Kestner and Peterson, 2017; Lattal and Wacker, 2015).

2. Methods

2.1. Participants

Sixty participants (24 male, 36 female) were recruited for this study; their ages ranging between 20 – 45 years (mean = 24.17, SD = 6.68). All participants were undergraduate students, who responded to advertisements regarding the study, and their participation was voluntary, and no payment or course credit were received for participation. None of the participants reported that they had a history of any psychiatric disorders. The research was approved by the University's Department of Psychology Ethics Committee.

3. Materials

3.1. Beck's depression inventory

(BDI; Beck et al., 1961) is a 21-item questionnaire that assesses the clinical symptoms of depression through asking about feelings over the past week. Responses are given on a four-point scale, and range from 0 to 63: 0–9 = low depression; 10–18 = mild; 19–29 = moderate; and 30+ = severe depression. The internal reliability of the scale is 0.93, and it correlates well with the Hamilton Depression Rating Scale (0.71).

3.2. Spielberger trait anxiety inventory

(STAI; Spielberger et al., 1983) rates the affective, cognitive, and physiological manifestations of anxiety in terms of long-standing patterns (trait anxiety). There are 20 statements, and the participant responds to each on a four-point Likert scale. The total score for each scale ranges from 20 to 80, with a higher score being indicative of a greater level of anxiety. The internal reliability of the scale is 0.93. The scale correlates with the Taylor Manifest Anxiety Scale (0.73), and Cattell and Scheier's Anxiety Scale Questionnaire (0.85).

3.3. Autistic spectrum quotient questionnaire

(AQ; Baron-Cohen et al., 2001) measures the level of autistic traits that an individual lacking an ASD diagnosis may possess. This questionnaire consists of 50 questions, and the person is required to state how much each statement best describes them by ticking one out of four options, with a score of 32 generally being suggested as indicating Asperger's syndrome or high functioning autism. The internal consistency of the scale is 0.82, there are no data available on concurrent validity, but the test-retest correlation was 0.70.

3.4. Experimental task

The program was run on a Viglen Omnino HW191D PC. Two, 6 cm × 6 cm colored boxes (red and green) were presented, equidistant from one another, in the top third of the screen. Directly below each box, a points-counter was displayed that showed the number of points obtained for responding to that color.

3.5. Procedure

The study took place in a quiet room, which was free from distraction. All participants were seated facing a computer. Before training, participants were presented with the following written instructions, and were required to follow them for the remainder of the study:

"*Remember, your task is to score as many points as possible by clicking in the coloured boxes using your mouse.*"

After this, all participants were exposed to a 6 min concurrent RR-5, ext schedule. In this phase, responses (using the mouse to click in the box) in one colored box resulted in the delivery of points (60) to the points counter below that box, which served as the potential reinforcer in this study. Responses to the other box never received points as a reinforcer. All participants started with 40 points in each box, and each response to a box resulted in the reduction of 1 point from this total. This procedure was adopted as a number of studies have shown that it allows human behavior in these types of schedule procedures to more closely resemble that of nonhumans (Raia et al., 2000; Reed, 2015a). Responses to the computer screen outside the area of the boxes has no programmed consequences. The color (red or green) and side (left or right) of the box that received reinforcement was randomly determined for each participant.

Following this phase, there was a 30s period during which the screen went blank, and then the boxes appeared again, as they had in the first phase. Training continued as described above, except the box that received reinforcement was reversed from Phase 1. That is, a conc ext, RR-5 schedule was in place. The purpose of this phase was to put the initial mouse clicking behavior to the first colored stimulus conditioned in Phase 1, into extinction, while reinforcing an alternative behavior (clicking the mouse into the second box) in its place. This phase lasted for a further 6 min.

After Phase 2, there was a 30s period during which the screen was black, and then the boxes reappeared as they had in the previous two phase. In this final phase, all participants were exposed to a 2 min conc ext, ext schedule. This time, their total number of points to both boxes would decrease by 1 every time they clicked the mouse in either box, but they received no reinforcement. The point of this procedure was to test how much, if at all, the extinguished point scoring behavior exhibited in Phase 1 resurged.

4. Results

Fig. 1 shows the group-mean number of responses to each component during the first two phases, for the component that was initially reinforced then non-reinforced (R–N), and for the component initially non-reinforced then reinforced (N-R). The right panel shows the ratio of responding in the final extinction phase of these two responses relative to their levels in Phase 2.

Inspection of these data in the left panel shows, for Phases 1 and 2, that responding was greater to the component being reinforced, and increased for the N-R response, but decreased for the R–N response across the two phases. A two-factor repeated-measures analysis of variance (ANOVA) with phase and component as factors, was conducted on these data. The results of the ANOVA, along with the effect size and 95% confidence limits, and the relevant Bayes factor, are reported below. This analysis revealed a significant main effect of phase, $F\,(1,59) = 68.54$, $p < .001$; $\eta^2_p = .537$ [95%CI = .355-.665]; $p(H_1/D) = .999$, not of component, $F\,(1,59) = 2.38$, $p = .128$; $\eta^2_p = .039$ [.000-.169]; $p(H_0/D) = .703$, but there was a significant interaction between the factors, $F\,(1,59) = 30.07$, $p < .001$; $\eta^2_p = .338$ [.149-.490]; $p(H_1/D) = .999$ Simple effect analyses revealed that there was a significant difference between the components in Phase 1, $F\,(1,59) = 11.55$, $p < .001$; $\eta^2_p = .237$ [.028-.327]; $p(H_1/D) = .965$, and in Phase 2, $F\,(1,59) = 18.98$, $p < .001$; $\eta^2_p = .537$ [.075-.406]; $p(H_1/D) = .998$.

The degree to which behavior resurges after reinforcement of an alternative response, which is subsequently extinguished, can be assessed by examining the relative rate of responding in the final extinction phase for the target response (that reinforced in Phase 1) and the non-target response (that not reinforced in Phase 1). The effect of the extinction schedules on responding in the final component is shown in the right panel of Fig. 1 by the ratio of responding during this phase to the

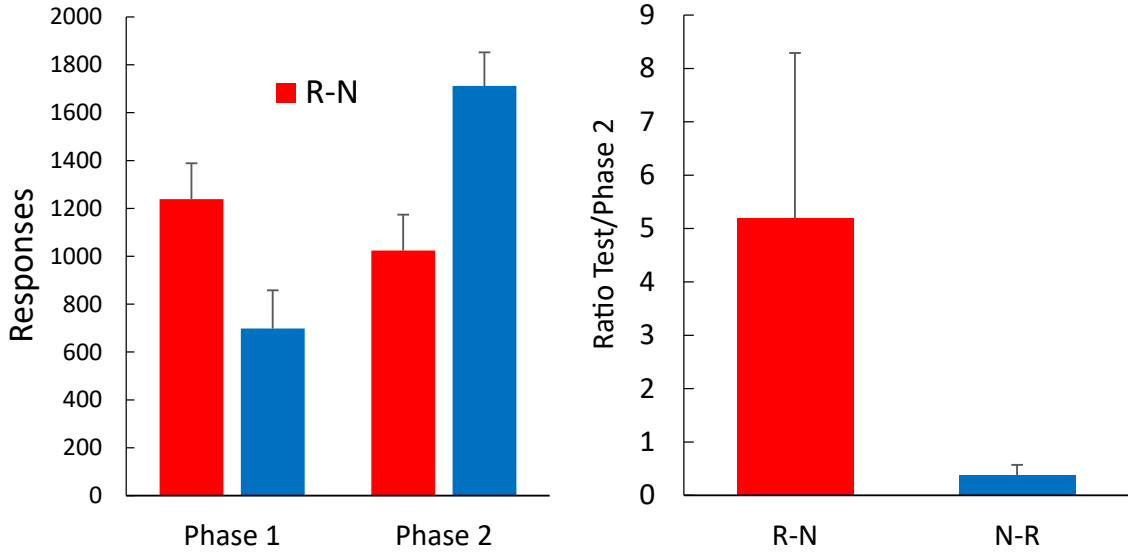

Fig. 1. Left panel = group-mean number of responses to each component during the first two phases, for the component that was initially reinforced then non-reinforced (R–N), and for the component initially non-reinforced then reinforced (N–R). Right panel = ratio of responding in the final extinction phase of these two responses relative to their levels in Phase 2.

responding associated with that component in Phase 2 (resurgence ratio). These data show much greater responding (resurgence) in the extinction phase for the component extinguished in Phase 2, than for the component reinforced in Phase 2, t (57) = 3.13, $p < .01$, $d = .41$; $p(H_1/D) = .936$.

Fig. 2 shows the data for the number of responses made in each phase, for the two responses, for the sample split into lower- and higher-scores for depression (BDI), anxiety (STAI-T), and autism (AQ), separately. Both the lower-scoring depression group ($N = 34$, *mean* = 2.82 ± 2.75; range 0–8), and the higher-scoring depression group ($N = 26$, *mean* = 15.92 ± 7.16; range 10–37), responded more in the reinforced than in the non-reinforced component of Phase 1, and showed a reversal of this pattern in Phase 2. The lower-scoring depression group responded more than the higher-scoring group, with this difference between the depression groups being larger in Phase 1. A three-factor mixed-model ANOVA (component x phase x depression) conducted on these data revealed significant main effects of depression, F (1,58) = 4.67, $p < .05$; $\eta^2_p = .074$ [.000-.223]; $p(H_1/D) = .567$, and phase, F (1,58) = 78.54, $p < .001$; $\eta^2_p = .575$ [.398-.683]; $p(H_1/D) = .999$, and significant interactions between component and phase, F (1,58) = 28.01, $p < .001$; $\eta^2_p = .326$ [.137-.481]; $p(H_1/D) = .999$, and phase and depression, F (1,58) = 5.89, $p < .05$; $\eta^2_p = .018$ [.002-.246]; $p(H_1/D) = .705$, but no other main effects or interactions were reliable, largest $p = .13$, smallest Bayes, $p(H_o/D) = .745$.

Both the lower-scoring anxiety group ($N = 26$, *mean* = 32.08 ± 38.95; range 20–41), and the higher-scoring anxiety group ($N = 34$, *mean* = 49.06 ± 7.00; range 42–69), responded more in the reinforced than in the non-reinforced component of Phase 1, and showed a reversal of this pattern in Phase 2, the difference between the components being greater for the lower-anxiety group. The lower-scoring anxiety group responded more than the higher-scoring group. A three-factor mixed-model ANOVA (component x phase x anxiety) conducted on these data revealed significant main effects of anxiety, F (1,58) = 15.15, $p < .001$; $\eta^2_p = .207$ [.051-.372]; $p(H_1/D) = .993$, and phase, F (1,58) = 66.17, $p < .001$; $\eta^2_p = .529$ [.348-.650]; $p(H_1/D) = .999$, and significant interactions between component and phase, F (1,58) = 33.47, $p < .001$; $\eta^2_p = .366$ [.173-.516]; $p(H_1/D) = .999$, and component and anxiety, F (1,58) = 4.89, $p < .05$; $\eta^2_p = .078$ [.000-.227]; $p(H_1/D) = .594$, but no other main effects or interactions were reliable, largest $p = .10$, smallest Bayes, $p(H_o/D) = .570$.

Both the lower-scoring autism group ($N = 28$, *mean* = 12.64 ± 3.41; range 7–17), and the higher autism group ($N = 32$, *mean* = 22.56 ± 5.11; range 18–36), responded more in the reinforced than in the non-reinforced component of Phase 1, and showed a reversal of this pattern in Phase 2. A three-factor mixed-model ANOVA (component x phase x autism) conducted on these data revealed significant main effects phase, F (1,58) = 69.09, $p < .001$; $\eta^2_p = .544$ [.361-.659]; $p(H_1/D) = .999$, and significant interactions between component and phase, F (1,58) = 30.68, $p < .001$; $\eta^2_p = .346$ [.154-.499]; $p(H_1/D) = .999$, but no other main effects or interactions were reliable, largest $p = .10$, smallest Bayes, $p(H_o/D) = .565$.

Fig. 3 shows the resurgence ratios for the sample divided by mean splits into lower and higher scoring groups in terms of depression (BDI), anxiety (STAI-T), and autism (AQ). The lower depression group had a higher ratio for the R–N component compared to the higher-scoring depression group, but there was little difference in terms of the R–N ratio. A two-factor mixed-model ANOVA, with component (R–N x N-R) as a within-subject factor, and group (lower versus higher) as a between-subject factor, revealed a significant main effect of component, F (1,56) = 8.91, $p < .01$; $\eta^2_p = .137$ [.015-.302]; $p(H_1/D) = .915$, not of group, F (1,56) = 2.84, $p = .098$; $\eta^2_p = .048$ [.000-.188]; $p(H_0/D) = .638$, but a significant interaction between the factors, F (1,56) = 3.94, $p < .05$; $\eta^2_p = .066$ [.000-.213]; $p(H_1/D) = .626$. Simple effect analyses revealed that there was a significant difference between the groups for the R–N component, F (1,56) = 6.07, $p < .05$; $\eta^2_p = .098$ [.026-.256]; $p(H_1/D) = .735$, but not for the N-R component, $F < 1$; $\eta^2_p = .004$ [.000-.088]; $p(H_0/D) = .609$.

The lower-scoring anxiety group had a lower ratio for the R–N component than the higher-scoring anxiety group, there was little difference in the N-R ratio. A two-factor mixed-model ANOVA (component x group) revealed significant main effects of component, F (1,56) = 8.90, $p < .01$; $\eta^2_p = .137$ [.015-.302]; $p(H_1/D) = .912$, no main effect of group, F (1,56) = 3.51, $p = .066$; $\eta^2_p = .059$ [.000-.204]; $p(H_0/D) = .554$, but a significant interaction, F (1,56) = 3.94, $p < .05$; $\eta^2_p = .097$ [.000-.214]; $p(H_1/D) = .559$. Simple effect analyses revealed that there was a significant difference between the groups for the R–N component, F (1,56) = 20.80, $p < .001$, $\eta^2_p = .271$ [.091-.435]; $p(H_1/D) = .881$, but not for the N-R component, $F < 1$, $\eta^2_p = .002$ [.000-.069]; $p(H_0/D) = .865$.

The lower-scoring autism group had a numerically higher R–N ratio than the higher autism group, but there was little difference between the groups in the N-R ratio. A two-factor mixed-model ANOVA (component x group) conducted on these data revealed significant main effects of component, F (1,56) = 10.19, $p < .01$, $\eta^2_p = .154$ [.022-.320]; $p(H_1/D) = .951$, but no main effect of group, F (1,56) = 2.00, $p = .163$; $\eta^2_p = .035$ [.000-.165]; $p(H_0/D) = .730$, or interaction, F (1,56) = 1.80, $p = .186$; $\eta^2_p = .031$ [.000-.159]; $p(H_0/D) = .752$.

5. Discussion

The current experiment is the first to examine the impact of individual differences on resurgence, and, in particular, whether different levels of depression, anxiety, and autism traits have differential effects on resurgence. This information will start to develop knowledge of the relationship between psychopathology and behavioral resurgence, which has been implicated as important to models of disorders and treatment (Bouton et al., 2012; Kestner and Peterson, 2017; Lattal and Wacker, 2015). The current results suggest that there are significant relationships

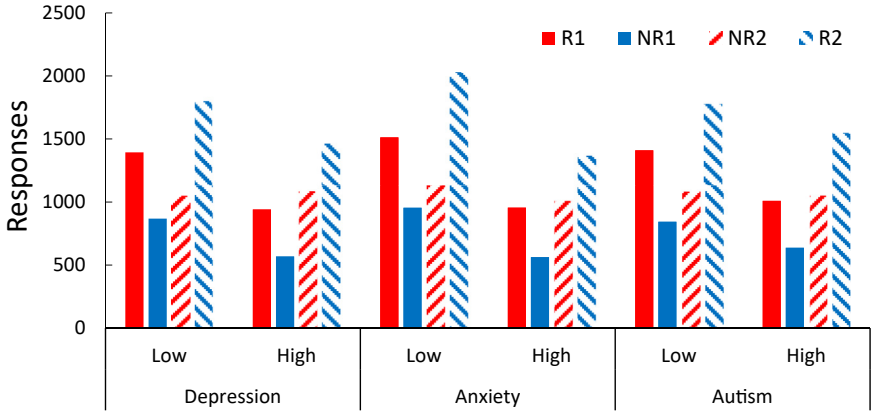

Fig. 2. Responses in each phase (1 and 2), for reinforced (R) and non-reinforced (NR) responses, for the sample split into lower and higher scoring traits for depression, anxiety, and autism.

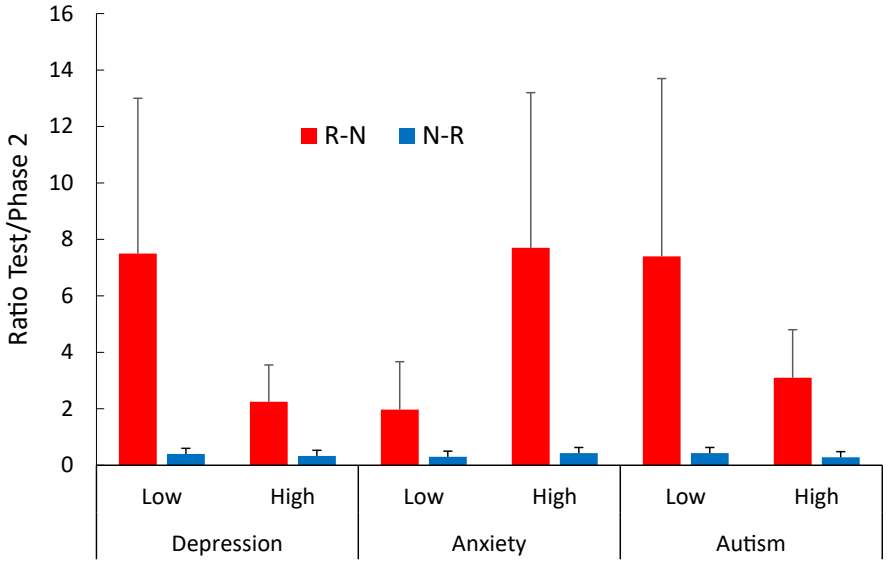

Fig. 3. Resurgence ratios for the sample divided by mean splits into lower and higher scoring groups in terms of depression (BDI), anxiety (STAI-T), and autism (AQ).

between individual differences in clinical symptoms and levels of re-emergent behavior; with lower levels of depression, and (to some extent) autism, traits being associated with greater resurgence, and higher levels of anxiety being linked to greater resurgence. The first of these outcomes is in line with theoretical predictions derived for depression, but the finding for anxiety and autism appear at odds with theoretical, and empirical, suggestions for those respective disorders.

That higher levels of depression lead to lower levels of resurgence may reflect the relative impairment in sensitivity to contingency change for those with higher levels of depression (Alloy et al., 2016). This result was in line with the prediction based on theoretical assumptions regarding depression. It may reflect the suggestion, and previous findings, that those who have experienced higher rates of reinforcement previously are more susceptible to resurgence effects (Smith et al., 2017). If it assumed that those with higher levels of depression *perceive* that reinforcement rates are actually lower, at any given rate of reinforcement, due to their anhedonic tendencies (Alloy et al., 2016), then depressed individuals would be *experiencing* lower levels of reinforcement than non-depressed individuals. The differences in the rates of response across lower- and higher-soring depression groups would support this view, and lead to the prediction that the lower-depressed group would display more resurgence than the higher-depressed group. The current results would support this interpretation. This finding, although apparently helpful in that unwanted behaviors are less likely to resurge in depressed individuals, also means that it may be problematic to recover previously extinguished positive behaviors in a depressed population. It is also less likely that these positive behaviors will spontaneously recover in depressed individuals. A suggestion made by Ferster (1973), in his analysis of depression.

The findings with respect to anxiety are more difficult to place within any extant context, and do not support the theoretical suggestions postulated in the Introduction based on anxiety-provoked reductions in reward sensitivity. It is generally assumed that those showing greater levels of anxiety would display reduced levels of reward sensitivity (Torrubia et al., 2001), and, as a consequence in the current context, should show less resurgence (Smith et al., 2017). That the difference between the reinforced and non-reinforced components in the initial two phases of this study was greater for the lower-anxiety group, than for the higher-anxiety group, tends to support this view. On the basis of the argument made for depression it would be predicted that lower levels of resurgence would be seen for the higher-anxiety group. However, this was not the case in the final test phase; with greater resurgence being noted in the group with greater levels of anxiety.

It may be that observed reduced levels of responding in the higher-anxiety group may have been due to increased levels of behavioral inhibition (Gray, 1987), rather than a tendency toward anhedonia. If so, then the above argument would not necessarily follow – a greater difference between the reinforced and nonreinforced components in the first two phases, may not translate into greater resurgence in the test phase, as there would be no difference in the *perceived* rates of reinforcement between lower and higher anxiety scoring subjects. These views will need to be teased apart in further studies, although the empirical findings suggest that resurgence of unwanted behaviors, reduced through exposure-therapies, may be more likely in those with higher levels of anxiety.

With respect to autism, although the current results were not statistically reliable, it is worth highlighting one difference between these findings and two previous reports. Reed and Clark (2010) and Volkert et al. (2009) used children with ASD as participants, and reported the presence of resurgent behavior. These previous findings bring up two issues; firstly, in this current study, adults were assessed for their autistic traits, rather than children. It must be pointed out that there are few, if any experiments, comparing the levels of resurgence experienced by children and adults. Even though some research has suggested that childhood behavior is often a good predictor of adult behavior (Stevenson and Goodman, 2001; Klinteberg et al., 1990), it is conceivable that the amount of re-emergent behavior exhibited by children with ASD may be different to that of an adult with sub-clinical autism traits. However, it might be noted that neither Reed and Clark (2010), nor Volkert et al. (2009), reported results from groups without ASD, so it is hard to tell whether re-emergent behavior would have been different in the latter type of individual.

There are limitations to the current study. The method of determining of whether a subject was considered as having high or low scores on any of the given dimension was based on the mean of all of the collected data. Although a wide range of scores were obtained for anxiety, the same cannot be said for depression or autism. This means that the higher-scoring groups for these dimensions were not particularly high in terms of clinically-relevant symptoms.

A suggestion for future research, and a potential solution to the above shortcoming would be to study behavioral resurgence levels in people who have a clinical diagnosis of depression, anxiety, or autism. This would serve to validate the suggestions made on the basis of the current model populations. Future studies could also separately assess reward

sensitivity and inhibition across populations, perhaps using the Behavioral Activation and Behavioral Inhibition Scales, to tease apart some of the theoretical suggestions made on the bases of the current findings. Finally, recent studies of resurgence (e.g., Sweeney and Shahan, 2016) have suggested that an untrained control response during testing cannot distinguish between true resurgence of the first-reinforced behavior and a more general increase in behavior, and additional controls may be used to address this issue in subsequent studies.

Nevertheless, the current findings have several potential applied implications. They suggest that resurgence may be weaker in depressed individuals, meaning that therapies that target a reduction in depression may not expect this to automatically lead to a re-emergence of previous positive behaviors exhibited by the individual, and this is less likely as the depression becomes more severe. These previously emitted behaviors may have to be re-established directly through reinforcement. In terms of anxiety, exposure therapy may need to be more prolonged with those with higher levels of anxiety – perhaps involving significant overtraining to prevent resurgence of unwanted behaviors following therapy. Of course, such speculation will need to be validated on clinical samples, as noted above.

In summary, the current findings begin to fill a gap in the knowledge base regarding the impact of psychometrically-defined levels of psychiatric symptoms on resurgence. The findings established that there are relationships between clinical symptoms and resurgence for depression and anxiety; with lower-levels of depression, and greater-levels of anxiety being associated with greater resurgence. These findings could be taken as the bases for further exploration and, potentially, development of better clinical interventions.

Declarations

Author contribution statement

Phil Reed: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

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The authors declare no conflict of interest.

Additional information

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