

**The impact of structured self-monitoring of blood glucose on glycaemic variability in non-insulin treated type 2 diabetes: the SMBG study, a 12-month randomised controlled trial**

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## **Abstract**

**Background & Aims:** There is inconsistent evidence supporting the self-monitoring of blood glucose (SMBG) in people with non-insulin treated type 2 diabetes (T2D). Structured SMBG protocols have a greater impact on glycaemic control than unstructured SMBG and may improve measures of glycaemic variability (GV), though few previous studies have reported on specific GV outcomes. Our aim was to determine the impact of structured SMBG on simple measures of GV in people with T2D.

**Methods:** Participants undertook structured SMBG over 12 months, with HbA<sub>1c</sub> recorded at baseline and at 3-monthly follow-up. For each participant, the mean blood glucose (MBG), fasting blood glucose (FBG), standard deviation BG (SD-BG), coefficient of variation of BG (CV-BG), mean absolute glucose change (MAG) and HbA<sub>1c</sub> were determined for each 3-month period. Responders were participants with an improvement in HbA<sub>1c</sub> of  $\geq 5$  mmol/mol (0.5%) over 12 months.

**Results:** Data from two hundred and thirty-one participants were included for analysis. Participants had a baseline median [interquartile range] HbA<sub>1c</sub> 68.0 [61.5–75.5] mmol/mol (8.4%). Participants demonstrated significant improvements in the MBG (-1.25 mmol/L), FBG (-0.97 mmol/L), SD-BG (-0.44 mmol/L), CV-BG (-1.43%), MAG (-0.97 mmol/L), and HbA<sub>1c</sub> (-7.0 mmol/mol) (all  $p < 0.001$ ) at 12 months compared to these measures collected within the first 3 months of SMBG. Responders had a significantly higher baseline median [interquartile range] HbA<sub>1c</sub> of 70.0 [63.0–78.0] mmol/mol compared to 61.0 [56.5–66.0] mmol/mol in non-responders ( $P < 0.001$ ).

**Conclusions:** Structured SMBG improved all the observed measures of GV. These results support the use of structured SMBG in people with non-insulin treated T2D.

**Keywords:** Structured SMBG, glycaemic variability, type 2 diabetes, insulin-naïve

### **Study Highlights**

- This secondary analysis of the SMBG study explores the impact of structured self-monitoring of blood glucose on measures of glycaemic variability and glycaemic control in people with insulin-naïve type 2 diabetes.
- Significant improvements were observed in several measures of glycaemic variability and glycaemic control over the 12-month study period.
- Participants with poorer baseline glycaemic control were more likely to achieve a significant clinical response with structured self-monitoring of blood glucose.
- These data add significantly to the existing literature about the impact of structured self-monitoring of blood glucose on glycaemic variability and glycaemic control in people with type 2 diabetes.

## **Introduction**

The self-monitoring of blood glucose (SMBG) remains essential for people treated with insulin for type 1 diabetes (T1D) or type 2 diabetes (T2D). In addition to identifying hypo- and hyper-glycaemia, SMBG is associated with improved glycaemic control in T1D and insulin-treated T2D [1-3]. In people with non-insulin treated T2D the benefits associated with SMBG are less clear, in part because trials employ variable SMBG interventions with different study populations [4-16]. However, trials using structured SMBG more consistently demonstrate significantly greater improvements in glycaemic control than unstructured SMBG [4,6-7,9-10,12-13]. Recently, the SMBG study reported that non-insulin treated T2D participants using a structured SMBG protocol [17] demonstrated a reduction in HbA<sub>1c</sub> of 8.9 mmol/mol (0.8%) more than control participants over 12 months [18]. Nevertheless, there remains considerable debate surrounding the recommendation for the use of SMBG in people with non-insulin treated T2D.

Utilising SMBG has been observed to improve measures of glycaemic variability (GV), which broadly reflects the number and extent of a person's blood glucose excursions [19]. There are numerous measures employed to quantify glycaemic control including the mean blood glucose (MBG), fasting blood glucose (FBG) and HbA<sub>1c</sub> and several measures of GV including the standard deviation of blood glucose (SD-BG), coefficient of variation of blood glucose (CV-BG), post-prandial glucose (PPG), mean absolute glucose change (MAG), the mean amplitude of glycaemic excursion (MAGE) and continuous overall net glycaemic action (CONGA) [19,20]. Many of these measures of GV have been shown to correlate with hypoglycaemia frequency [21], incidence of cardiovascular complications [22-23] and mortality [24]. Some studies have demonstrated improvements in some measures of

glycaemic control and GV with SMBG in non-insulin treated T2D, including MBG, FBG, PPG, MAGE [6,13]. However, measures of GV are infrequently reported from SMBG trials in people with non-insulin treated T2D, and further analyses are needed.

This manuscript aims to determine the impact of structured SMBG on blood glucose control and GV in people with non-insulin treated T2D participating in the SMBG study. As a result of the significant impact of structured SMBG on HbA<sub>1c</sub> previously described in this cohort [18], we hypothesise that the use of structured SMBG will result in improvements in both GV and blood glucose control.

## **Methods**

### **Study design**

Details of the study protocol have been previously published [17]. The SMBG study was a 12-month open-label, multi-centre RCT conducted between December 2012 and September 2016 across 16 different sites in England and Wales, UK. Participants were randomised to a structured SMBG protocol with or without additional nurse-led telecare support, or to a control group receiving their usual diabetes care excluding the use of SMBG. Participants were followed for 12 months after randomisation and blood glucose data were collected (in the SMBG groups only) with HbA<sub>1c</sub> measurements every 3 months.

Following randomisation, participants in the groups using structured SMBG were provided with standardised SMBG education. This included technical training and education to recognise patterns of dysglycaemia, and participants were provided with standardised algorithms on how to adjust their lifestyle and/or medications in response to any patterns identified. Participants measured the FBG and blood glucose 2 hours after breakfast, then

before and 2 hours after their main meal 2 days each week. Throughout the week prior to each 3-monthly clinical review, participants were asked to check their blood glucose before and 2 hours after each of their 3 main meals and before bedtime to provide a 7-point profile, on 3 days including one day during the weekend.

### **Participants**

Participants were aged 18-80 years, with a diagnosis of T2D for at least 12 months. Baseline HbA<sub>1c</sub> was 58-119 mmol/mol (7.5–13.0%), and participants were insulin-naïve. Key exclusion criteria included diabetes other than T2D, pregnancy, severe chronic liver disease or end-stage renal disease, severe vision loss in both eyes and participants who used SMBG as part of their routine care.

### **Outcomes**

The primary outcome of the study was to determine whether structured SMBG resulted in a significant improvement in HbA<sub>1c</sub>, and whether additional nurse-led telecare had an additional impact on HbA<sub>1c</sub>. Secondary outcomes included HbA<sub>1c</sub> at 3, 6 and 9 months, serum cholesterol at 3, 6, 9 and 12 months, body mass index (BMI), waist circumference, medication use, acceptability of SMBG and quality of life measures [17].

### **Study Approval**

The South East Wales Research Ethics Committee (Panel C) gave ethical approval for the study to take place (Ref. 10/WSE03/50). The trial was registered with the UK Clinical Research Network (UKCRN 12038) and ISRCTN register (ISRCTN21390608).

## **Statistical analysis**

No comparison is made with the control group as no blood glucose data were collected by these participants. Blood glucose data for participants with or without telecare support were pooled as previous analyses demonstrated no significant differences in changes in glycaemic control between these two groups [18]. Participants with blood glucose data for at least 3 months were included for analysis, and blood glucose data were compared in 3-monthly intervals.

Continuous data following a normal distribution are presented by the mean (and standard deviation - SD), and data which did not have a normal distribution are presented by the median [and interquartile range - IQR]. Normality was checked using the Kolmogorov-Smirnov test and visualised using Q-Q plots. Non-normally distributed data were tested for statistical significance using either a Kruskal-Wallis test, Mann-Whitney U test or Wilcoxon signed-rank test as appropriate.

Responders were defined *a priori* as participants with a clinically meaningful improvement in HbA<sub>1c</sub> of at least 5 mmol/mol (0.5%) over 12 months.

## **Results**

### **Participant characteristics**

Of the 295 participants randomised to undertake structured SMBG, 231 participants (78.3%) had complete data for at least 3 months and were included for analysis. The median [IQR] age was 63.9 [56.3-68.1] years and 131 (56.7%) participants were male. One hundred and sixty-four (70.7%) participants had previously used SMBG in an unstructured way and 149 (64.5%) participants had a diagnosis of diabetes for more than five years. Prior to starting

SMBG, these participants had a baseline median [IQR] HbA<sub>1c</sub> of 68.0 [61.5–75.5] mmol/mol (8.4%).

### **The impact of structured self-monitoring blood glucose on glycaemic variability**

There were significant improvements in each of the observed measures of blood glucose control (Table 1). Participants demonstrated significant improvements in measures of glycaemic control [MBG (-1.25 mmol/L), FBG (-0.97 mmol/L) HbA<sub>1c</sub> (-7.0 mmol/mol (-0.7%)] and GV [SD-BG (-0.44 mmol/L), CV-BG (-1.43%), MAG (-0.97 mmol/L)] (all P <0.001) at 12 months compared with the first 3 months. These data are presented in Fig. 1.

There were no significant differences found at any point in follow-up for the MBG, FBG, SD-BG, CV-BG, MAG or HbA<sub>1c</sub> between participants who did and did not receive additional telecare in the SMBG study. These data are presented in supplementary Table S1.

### **Factors which predict glycaemic response to self-monitoring blood glucose**

Of the 231 participants, 203 had sufficient data to determine responder status. One hundred and fifty-six participants (76.8%) were responders, and 47 participants (23.2%) were non-responders. Responders and non-responders had no significant differences in age, gender, previous use of SMBG or the proportion of participants diagnosed with diabetes for over five years. At the baseline visit responders demonstrated a significantly higher HbA<sub>1c</sub> than non-responders, with a median [IQR] HbA<sub>1c</sub> of 70.0 [63.0-78.0] mmol/mol compared to 61.0 [56.5–66.0] mmol/mol in non-responders (P <0.001). These data are presented in Table 2.

### **Differences in glycaemic response between responder and non-responders**

There were no significant differences between responders and non-responders in any measure of glycaemic control or GV during the first 3 months using structured SMBG. At 12 months, responders had significantly improved measures of glycaemic control [MBG (-0.69 mmol/L,



P=0.001), FBG (-1.07 mmol/L, P=0.006), HbA<sub>1c</sub> (-11.00 mmol/mol, P <0.001)], and significantly improved measures of GV [SD-BG (-0.29 mmol/L, P=0.021), MAG (-0.58 mmol/L, P = 0.004)] than non-responders. Responders demonstrated improvements in all observed measures of blood glucose control [MBG (-1.42 mmol/L), FBG (-1.00 mmol/L), HbA<sub>1c</sub> (-8.00 mmol/mol (0.7%))] and GV [SD-BG (-0.45 mmol/L), CV-BG (-1.40%), MAG (-1.11 mmol/L)] at 12 months compared to 3 months (all P <0.001). In non-responders after 12 months there was a small reduction in the MAG (-0.30 mmol/L, P = 0.001), with no significant change in the MBG, FBG, SD-BG, CV-BG or HbA<sub>1c</sub> compared to the first 3 months. Data are presented by visit in Table 3 and Fig. 2.

## **Discussion**

Previously published results from this RCT showed that the use of structured SMBG in participants with non-insulin treated T2D improved HbA<sub>1c</sub> by 8.9 mmol/mol (0.8%) more than control participants over 12 months, and additional nurse-led telecare did not confer additional benefit in terms of the HbA<sub>1c</sub> [18]. Further analyses of the blood glucose data from the SMBG study are presented in this manuscript, demonstrating statistically and clinically significant improvements in blood glucose control including the MBG and FBG, in addition to several measures of GV including the SD-BG, CV-BG and MAG.

The findings presented in this manuscript are consistent with other studies exploring the use of SMBG under similar conditions. Khamseh and colleagues found that structured SMBG in participants with HbA<sub>1c</sub> greater than 64 mmol/mol (8.0%) significantly improved the HbA<sub>1c</sub> MBG and FBG by 19 mmol/mol (1.8%), 0.6 mmol/L and 1.1 mmol/L, respectively [6]. One meta-analysis observed a non-significant improvement in FBG of 0.23 mmol/L over 12 months [16]. However, Polonsky and colleagues [13] previously noted statistically significant

improvements in both glycaemic control and GV defined by the MAGE in participants with both insulin-treated and non-insulin treated T2D with structured SMBG. Clinical trials and meta-analyses continue to report inconsistent differences in responses associated with participants' glycaemic control when comparing structured and unstructured SMBG [5-6,8-9,13-14,16], thus complicating trial data interpretation. To date, GV is infrequently reported in SMBG trials, which is the purpose of this analysis. These data add significantly to the existing literature exploring the impact of structured SMBG on GV in this cohort, reporting multiple and unique measures of GV compared with previously published studies.

The data presented in this manuscript identified baseline glycaemic control as the only predictive variable of a clinically relevant response to structured SMBG in this cohort. To our knowledge, this is the first study to report this variable as a predictive factor for clinical response to structured SMBG. This was somewhat expected, as participants with poorer glycaemic control are more likely to benefit from any diabetes-related clinical intervention [25]. These findings challenge current NICE guidance [26] on the use of SMBG in people with non-insulin treated T2D as recommendations do not suggest accounting for a person's glycaemic control when considering SMBG use, however our data suggest the use of structured SMBG in people with sub-optimally controlled T2D not using insulin may improve glycaemic control and GV. This intervention would complement other aspects of diabetes management and support changes in their lifestyle and/or diabetes medication [27].

Further to the findings presented in this manuscript and previously published SMBG study outcomes [18], cost analyses of structured SMBG in this cohort would be important. Indeed, previous analyses in this area have provided mixed outcomes. One American-based study found that the projected 40-year economic outcome for people with T2D utilising 1 or 3

SMBG measurements daily resulted in a cost of only \$7858 and \$6601 per quality-adjusted life year (QALY) gained, respectively [28]. However, a primary care study taken in the United Kingdom found that the minimum extra cost associated with SMBG use in people with T2D using oral agents was £84 per patient per year and was not associated with improved quality of life [29]. Additionally, a cost analysis of SMBG use in people with non-insulin treated T2D reported a significant cost of \$113,643 (~£82,000) per QALY [30]. Accordingly, both studies concluded that SMBG is not cost-effective in people with non-insulin treated T2D [29, 30]. However, outcomes derived from these analyses utilised older trials which did not apply structured SMBG protocols, and we plan to undertake an analysis to determine whether structured intervention would be cost-effective in our cohort of patients. Certainly, glycaemic outcomes from studies utilising structured SMBG compared with unstructured SMBG observe greater improvements in glycaemic control [31], and newer cost analyses including a comparison of structured versus unstructured trials would be useful.

Several reasons for the improvement in both blood glucose control and GV may explain the benefits observed with structured SMBG. Firstly, participants would understand their blood glucose control and therefore be able to adjust their diet and/or lifestyle. Unfortunately, data related to potential changes made in diet and/or physical activity were not collected as part of the SMBG study. However, one study exploring continuous glucose monitoring (CGM) use in people with poorly-controlled non-insulin treated T2D found that CGM use was associated with reduced calorie intake and increased physical activity [32]. Secondly, paired blood glucose testing allows the physician to adjust diabetes medication to treat patterns of dysglycaemia. Some trials exploring SMBG in people with non-insulin treated T2D found significant changes in diabetes medication prescription [4,7,10,13], whilst others have not [5,11,15]. Further analyses of data collected in the SMBG study are needed to explain our

results, but a previous analysis shows a significantly greater proportion of people undertaking structured SMBG had an increase in the mean number of diabetes medications prescribed over the period of the study compared to the control group (47.8% vs 27.6%, respectively) [18].

## **Conclusion**

Structured SMBG utilising paired blood glucose testing to identify patterns of dysglycaemia is associated with significant improvements in blood glucose control and GV. Whilst NICE do not currently recommend the routine use of structured SMBG in this cohort, the results presented in this manuscript support its use. However, further work exploring the economic impact of such interventions in this cohort of patients is required.

## **Funding Statement**

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## **Disclosure of Interests**

The authors have no disclosures of interest to declare.

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**Table 1: Changes in glycaemic variability at each study visit**

	<b>0-3 months</b> (n = 224)	<b>3-6 months</b> (n = 209)	<b>P</b> <b>value</b>	<b>6-9 months</b> (n = 200)	<b>P</b> <b>value</b>	<b>9-12 months</b> (n = 195)	<b>P</b> <b>value</b>
<b>MBG</b> (mmol/L)	9.67 [8.66-11.16]	8.96 [8.03–10.21]	<0.001	8.66 [7.68–9.82] <sup>a</sup>	<0.001	8.42 [7.51–9.56] <sup>e</sup>	<0.001
<b>FBG</b> (mmol/L)	8.98 [7.75–10.42]	8.53 [7.58–9.90]	<0.001	8.28 [7.20–9.37] <sup>b</sup>	<0.001	8.01 [7.15–9.27] <sup>g</sup>	<0.001
<b>HbA<sub>1c</sub></b> (mmol/mol)	62.0 [55.0–69.0]	59.0 [53.0–65.0]	<0.001	57.5 [50.0–65.0] <sup>d</sup>	<0.001	55.0 [49.0–62.0] <sup>f</sup>	<0.001
<b>SD-BG</b> (mmol/L)	2.48 [2.04-2.97]	2.24 [1.85–2.66]	<0.001	2.08 [1.66–2.56] <sup>b</sup>	<0.001	2.04 [1.60–2.48] <sup>g</sup>	<0.001
<b>CV-BG</b> (%)	24.78 [21.34–28.58]	23.86 [21.15–27.95]	NS	23.63 [20.38–27.27] <sup>d</sup>	<0.05	23.35 [19.90–27.21] <sup>h</sup>	<0.001
<b>MAG</b> (mmol/L)	1.83 [0.98–2.68]	1.28 [0.57–2.14]	<0.001	1.19 [0.38–1.91] <sup>b</sup>	<0.001	0.86 [0.17–1.77] <sup>g</sup>	<0.001

**Table 1:** Changes in glycaemic control and GV at each study visit, presented in 3-monthly intervals. The number of participants with complete data at each visit are denoted by n. Data were not normally distributed and are presented as the median [IQR]. P-values compare to data collected at 0-3 months. For data at 6-9 months compared to 3-6 months: <sup>a</sup>P <0.001, <sup>b</sup>P <0.01, <sup>c</sup>P <0.05, <sup>d</sup>P = NS. For data at 9-12 months compared to 3-6 months: <sup>e</sup>P <0.001, <sup>f</sup>P <0.01, <sup>g</sup>P <0.05, <sup>h</sup>P = NS.

**Table 2: Participant characteristics by responder status**

	<b>Non-responders (n=47)</b>	<b>Responders (n=156)</b>	<b>Significance</b>
<b>Age*</b> <b>(years)</b>	64.5 [60.0–68.6]	64.0 [55.7–68.3]	NS
<b>Male</b>	27 (57.4%)	90 (57.7%)	NS
<b>Used SMBG previously</b>	37 (78.7%)	105 (67.3%)	NS
<b>Duration of diabetes &gt;5years</b>	34 (72.3%)	97 (62.2%)	NS
<b>HbA<sub>1c</sub>* (mmol/mol)</b>	61.0 [56.5–66.0]	70.0 [63.0–78.0]	P <0.001

**Table 2:** Characteristics of responders and non-responders at the baseline visit. \*Data were not normally distributed and are presented as median [IQR]. Mann-Whitney U test was used for analysis. Data otherwise presented as the number (%) of participants in that group. Chi-squared test was used to analyse the statistical significance of categorical variables: gender, previous use of SMBG, duration of diabetes. NS = non-significant.

**Table 3: Comparison of glycaemic variability between responders and non-responders at each study visit**

	0-3 months		3-6 months		6-9 months		9-12 months	
	R	NR	R	NR	R	NR	R	NR
	(n = 150)	(n = 47)	(n = 151)	(n = 46)	(n = 149)	(n = 46)	(n = 149)	(n = 46)
<b>MBG</b>	9.68	9.26	8.92	8.99	8.55	9.13	8.26	8.95
(mmol/L)	[8.64–11.15]	[8.51–10.08]	[7.93–10.24]	[8.20–9.62]	[7.48–9.75]	[8.13–10.43] <sup>a</sup>	[7.35–9.34]	[8.09–10.32] <sup>b</sup>
<b>FBG</b>	8.87	8.41	8.54	8.33	8.13	8.69	7.87	8.94
(mmol/L)	[7.75–10.43]	[7.30–9.72]	[7.62–9.84]	[7.40–9.48]	[7.17–9.30]	[7.49–9.44]	[6.97–9.05]	[7.53–9.87] <sup>c</sup>
<b>SD-BG</b>	2.45	2.25	2.21	2.20	2.02	2.24	2.00	2.29
(mmol/L)	[2.05–2.98]	[1.94–2.77]	[1.84–2.61]	[1.85–2.70]	[1.61–2.51]	[1.74–2.86] <sup>a</sup>	[1.57–2.39]	[1.67–2.70] <sup>a</sup>
<b>CV-BG</b>	24.76	25.05	23.82	23.86	22.66	24.62	23.36	23.31
(%)	[21.35–28.83]	[21.51–28.09]	[20.90–27.60]	[21.41–28.51]	[20.16–27.17]	[21.80–28.56]	[19.87–27.04]	[20.26–27.60]
<b>MAG</b>	1.85	1.62	1.28	1.59	0.92	1.51	0.74	1.32
(mmol/L)	[0.90–2.64]	[1.17–2.76]	[0.57–2.03]	[0.73–2.24]	[0.35–1.86]	[0.90–2.23] <sup>a</sup>	[0.11–1.62]	[0.61–2.19] <sup>a</sup>
<b>HbA<sub>1c</sub></b>	61.0	62.0	57.0	62.0	54.0	62.50	53.0	64.00
(mmol/mol)	[54.0–68.0]	[56.0–68.5]	[52.0–64.0]	[56.0–65.0] <sup>a</sup>	[49.0–63.0]	[57.0–69.0] <sup>c</sup>	[48.0–59.0]	[57.3–71.8] <sup>c</sup>

**Table 3:** R = Responders, NR = Non-responders. Data were not normally distributed and are presented as the median [IQR]. Kruskal-Wallis test was used for statistical analysis. The number of participants with complete data at each visit are denoted by n. Statistical significance between responders and non-responders at the same visit are indicated by <sup>a</sup>P <0.05, <sup>b</sup>P <0.01, <sup>c</sup>P <0.001.

Figure 1: Changes in glycaemic control and glycaemic variability at each study visit

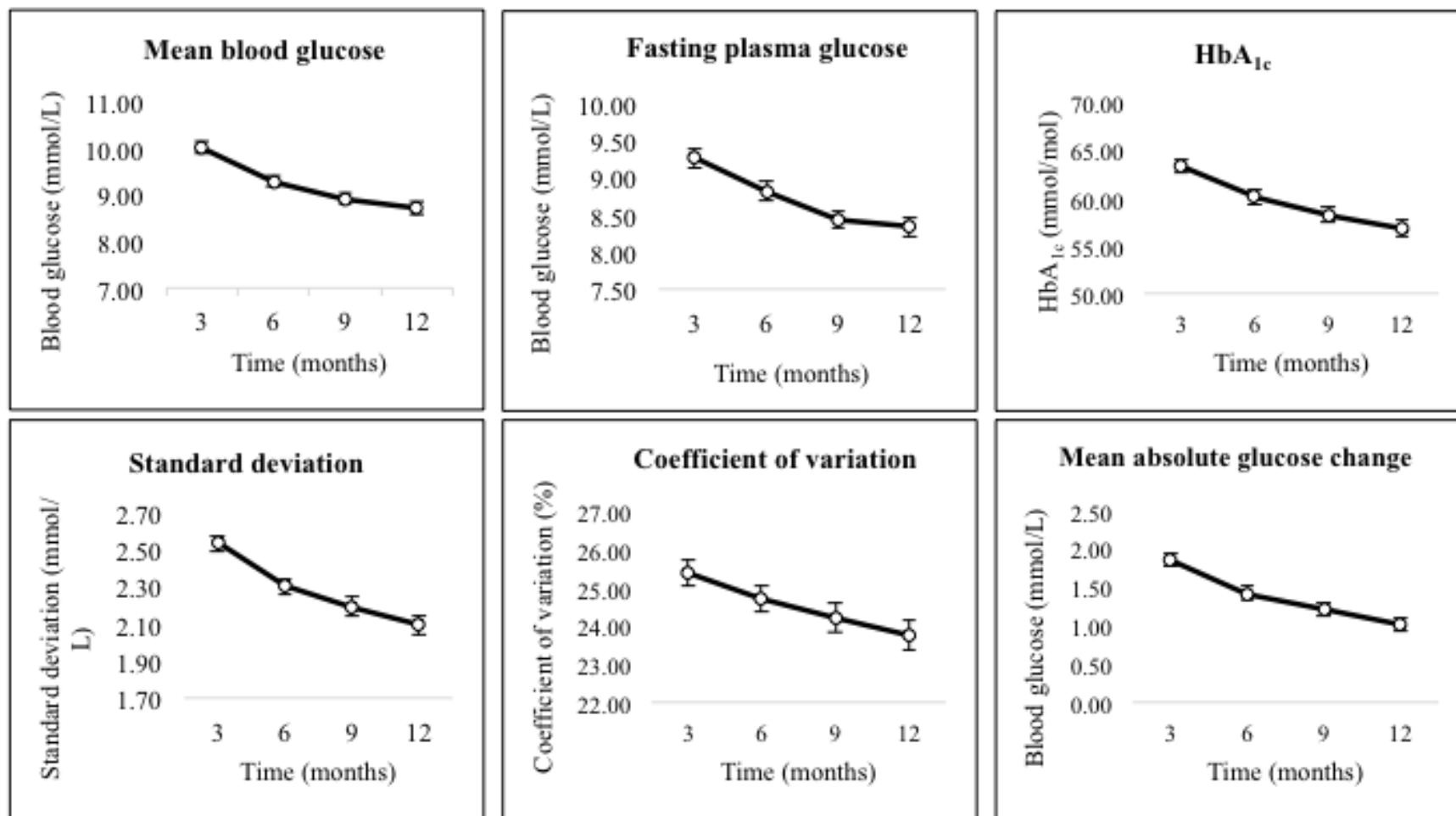
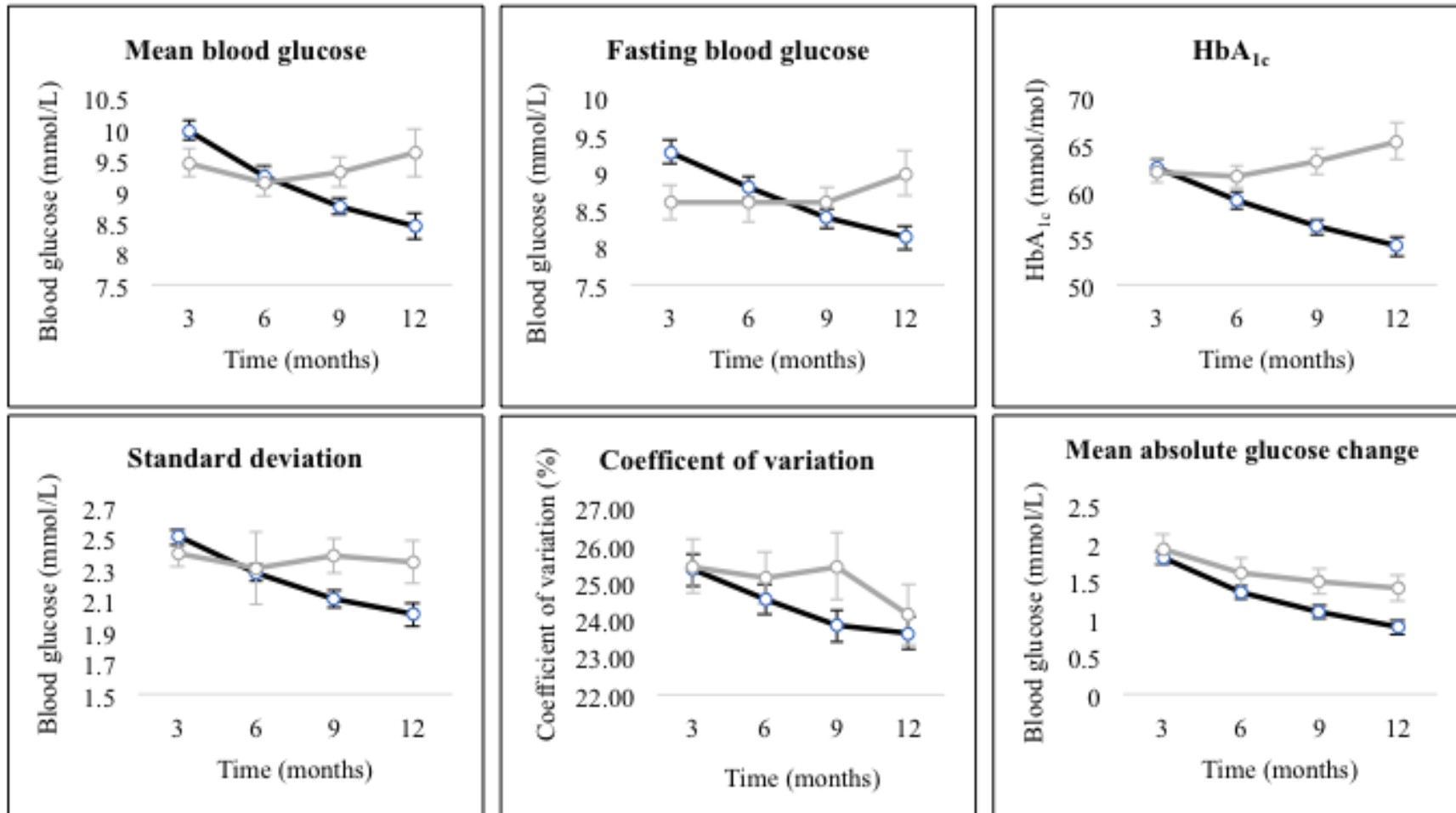


Figure 2: Comparison of glycaemic control and glycaemic variability between responders and non-responders at each study visit



## **Figure Legends**

### **Figure 1**

#### **Figure 1: Changes in glycaemic control and glycaemic variability at each study visit**

Figure 1: Line graphs illustrate the changes in the MBG, FBG, HbA1c, SD-BG, CV-BG and MAG and over 12 months. Data are presented as the mean, and the error bars represent the standard error of the mean (SEM).

### **Figure 2**

#### **Figure 2: Comparison of glycaemic control and glycaemic variability between responders and non-responders at each study visit**

Figure 2: Line graphs illustrate how glycaemic control and GV changed over the study between responders and non-responders. The black line represents responders, and the grey line represents the non-responders. Data are presented as the mean, and error bars represent the SEM.

**Supplementary Table S1: Glycaemic variability by study group over 12 months**

	0-3 months		3-6 months		6-9 months		9-12 months	
	No Telecare (n=110)	Telecare (n=114)	No Telecare (n=101)	Telecare (n=108)	No Telecare (n=94)	Telecare (n=106)	No Telecare (n=91)	Telecare (n=104)
<b>MBG</b> (mmol/L)	9.73 [8.56–11.36]	9.38 [8.70–10.20]	8.96 [8.06–10.36]	8.95 [8.03–9.94]	8.93 [7.57–10.08]	8.60 [7.73–9.69]	8.37 [7.44–9.66]	8.47 [7.59–9.55]
<b>FBG</b> (mmol/L)	8.87 [7.64–10.17]	9.08 [7.83–10.47]	8.44 [7.42–9.78]	8.56 [7.69–9.96]	8.21 [7.11–9.55]	8.37 [7.37–9.30]	7.93 [7.18–9.19]	8.04 [7.11–9.29]
<b>SD-BG</b> (mmol/L)	2.40 [1.96–2.95]	2.56 [2.12–3.00]	2.27 [1.85–2.69]	2.24 [1.85–2.60]	2.10 [1.60–2.64]	2.08 [1.69–2.52]	1.98 [1.51–2.50]	2.12 [1.66–2.46]
<b>CV-BG</b> (%)	24.15 [21.12–28.03]	25.57 [22.00–28.93]	23.84 [20.79–28.17]	23.88 [21.43–27.54]	22.58 [19.80–27.60]	23.69 [20.92–27.23]	23.00 [19.48–27.22]	23.70 [20.27–27.19]
<b>MAG</b> (mmol/L)	1.72 [0.92–2.90]	1.88 [0.98–2.62]	1.55 [0.68–2.37]	1.12 [0.46–1.96]	1.27 [0.52–1.95]	1.02 [0.37–1.87]	1.01 [0.20–1.84]	0.84 [0.16–1.68]
<b>HbA<sub>1c</sub></b> (mmol/mol)	61.5 [55.0–69.0]	62.00 [56.0–69.0]	60.0 [53.0–65.0]	58.0 [53.0–64.0]	57.0 [50.0–64.0]	58.0 [51.0–66.0]	54.0 [48.5–62.0]	56.0 [50.0–62.0]

**Table S1:** Data were not normally distributed and are presented as median [IQR]. Mann-Whitney U test was used for data analysis. The number of participants with complete data at each visit are denoted by n. There were no statistically significant differences in the observed measures of glycaemic control or GV between group receiving or not receiving telecare support at any point during the study.