Do diabetes cognitions and poor emotional health predict adherence to diabetes self-care? A longitudinal test of the Common Sense Self-Regulation Model using Structural Equation Modelling

A thesis submitted to the University of Manchester for the degree of Doctor of Philosophy in the Faculty of Medical and Human Sciences

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Abbreviations

AIC	Akaike Information Criterion
ANCOVA	Analysis of Covariance
AMOS	Analysis of Moments Structures
B	Baseline
BMQ	Beliefs about Medicines Questionnaire
BMI	Body Mass Index
B-IPQ	Brief Illness Perceptions Questionnaire
CES-D	Centre for Epidemiological Studies Depression Scale
CS-SRM	Common Sense Self-Regulation Model
CFI	Comparative Fit Index
CI	Confidence Interval
CFA	Confirmatory Factor Analysis
CQ	Consequences
C	Cross-sectional
DF	Degrees of Freedom
DV	Dependent variable
DIRQ	Diabetes Illness Representations Questionnaire
eGFR	Estimated Glomerular Filtration Rate
FSN	Fail-safe N
GP	General Practitioner
GAD	Generalised anxiety disorder
HbA1c	Glycosylated haemoglobin
HR	Hazards ratio
HADS	Hospital Anxiety and Depression Scale
IP	Illness perception
IPQ	Illness Perception Questionnaire
IPQ-R	Illness Perception Questionnaire-Revised
ICD-10	International Classification of Disease
IQR	Interquartile range
KMO	Kaiser-Meyer-Olkin
MDD	Major depressive disorder
MD	Mean difference
MAR	Missing at random

DF_m	Model degrees of freedom
NICE	National Institute for Health and Care Excellence
NR	Non-responders or not reported
NS	Non-significant
NA	Not applicable
NMAR	Not missing at random
N	Number
k	Number of studies
OR	Odds ratio
PVD	Peripheral Vascular Disease
PMDI	Personal Model of Diabetes Interview
PCA	Principal Component Analysis
P	Prospective
RCT	Randomised controlled trials
RR	Relative risk
R&D	Research and Development
R	Responders
RMSEA	Root Mean Square Error of Approximation
SMBG	Self-monitoring of blood glucose
SF-STAI	Short Form Spielberger State Anxiety Inventory
SD	Standard Deviation
SRMR	Standardised Root Mean Square Residual
SPSS	Statistical Package for the Social Sciences
S	Step
SEM	Structural Equation Modelling
SDSCA	Summary of Diabetes Self-Care Activities Scale
Timeline A/C	Timeline acute/chronic
TC	Treatment control
T1	Type 1 diabetes
T2	Type 2 diabetes
VIF	Variance Inflation Statistic
Veg	Vegetables
WBQ	Well-being Questionnaire

The University of Manchester
Joanna Louise Hudson
Doctor of Philosophy
Do diabetes cognitions and poor emotional health predict adherence to diabetes self-care? A longitudinal test of the Common Sense Self-Regulation Model using Structural Equation Modelling
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Depression and anxiety are prevalent in adults with diabetes and are associated with poorer adherence to diabetes self-care. The Common Sense Self-Regulation Model (CS-SRM) hypothesises that both illness cognitions and emotions determine adherence to health behaviours but little is known about the causal pathways through which poor emotional health impairs adherence. Using the CS-SRM as a theoretical framework this thesis aimed to identify the pathways that exist between diabetes cognitions and poor emotional health and explored their combined and independent effects on diabetes self-care.

A systematic review with meta-analysis identified that all previous observational studies used cross-sectional designs to explore the relationships between diabetes cognitions and poor emotional health, excluding one. Randomised controlled trials showed that changes in diabetes cognitions were associated with changes in poor emotional health but these mechanisms of action were not confirmed by mediation analyses. Few observational studies have rigorously tested how diabetes cognitions and poor emotional health operate together to determine diabetes self-management behaviours.

This thesis used a longitudinal design to test whether: i) diabetes cognitions and poor emotional health have a longitudinal bi-directional relationship and ii) diabetes cognitions and poor emotional health have a combined (mediated) and independent (direct) longitudinal effect on diabetes self-care. Outpatients with Type 2 diabetes (N=261) were recruited at baseline and completed self-report measures of poor emotional health (Well-being Questionnaire), cognitions (Revised Illness Perception Questionnaire; Beliefs about Medicines Questionnaire) and diabetes self-care (Summary of Diabetes Self-Care Activities Scale) at baseline and six months follow-up. Hypothesised pathways were tested simultaneously using structural equation modelling.

Participants who were more anxious at baseline perceived diabetes to be unpredictable and were apprehensive about their medications at follow-up. These effects were not observed for depression. Baseline diabetes cognitions did not predict change in poor emotional health at follow-up. Equally, neither baseline depression or anxiety predicted change in diabetes self-care behaviours at six months (directly or indirectly via diabetes cognitions). Baseline personal control beliefs remained independent of poor emotional health: greater personal control beliefs were associated with reduced adherence to diabetes self-care.

A bi-directional relationship between diabetes cognitions and poor emotional health was absent in this sample. The main direction of effect was from anxiety only to diabetes cognitions. Depression and anxiety had no relationship with diabetes self-care. Whilst personal control remained independent of poor emotional health, mastery beliefs appear to be insufficient on their own to sustain adherence behaviours over time suggesting that interventions should also provide patients with action plans whilst managing outcome expectations. The unexpected findings for the relationship between poor emotional health, cognitions, and diabetes self-care may be because the sample did not include individuals with more severe depression or anxiety. This study needs to be replicated among people experiencing clinically significant levels of depression and anxiety in diabetes.

Declaration

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I dedicate this thesis to my family.

About the author

I studied Psychology and Cognitive Neuroscience at the University of Nottingham. It was there that I realised that the topics that most interested me were those that had a health and clinical psychology focus. Because of this I decided to volunteer weekly at a day centre that provided a supportive environment for individuals recovering from a severe mental illness. I also gained research experience by working under the supervision of two health psychologists as part of the Q-Active scheme at the Queen's Medical Centre, Nottingham. I helped to produce a literature review that explored the impact of workplace smoking bans in healthcare settings, to inform the design of a behaviour change policy to promote a smoke free hospital.

My enjoyable undergraduate experience encouraged me to undertake a Masters in Health Psychology at the University of Nottingham. This post-graduate degree allowed me to gain a more in depth knowledge of the theoretical, methodological and practical aspects of health psychology with regard to health policy, public health, health care, health behaviour and chronic illness. During my Masters I became heavily involved in the Student Welfare Group and implemented a number of health promotion campaigns targeted at improving the health and well-being of students. Campaigns addressed numerous issues including: sexual health, mental health, nutritional health, and physical health. This work gave me practical experience in translating theoretical concepts outlined in social cognition models into health promotion materials.

After my Masters I worked as a Psychological Well-being Practitioner for Nottingham City Primary Care Trust. I delivered individual and group based low intensity psychological interventions to individuals with mild to moderate anxiety and depression. I was uncomfortable in this role however when patients were referred to my caseload with a chronic physical long term condition. I was unsure how best to manage their emotional needs without hindering their physical health. Because of this unease and wanting to maintain my interest in health psychology I became involved in the long term condition management group within the trust. It was in this group that I began to work collaboratively with the community cardiac nurses. We set up a two-way direct referral strategy to allow patients to have better access to care for their physical health and vice versa. We also held bi-monthly psycho-education group meetings with both me and a cardiac nurse present. However, I still felt that health psychology had a lot more to give in terms of shaping the design of interventions to adequately manage poor emotional health in the context of a chronic physical long term condition. When the opportunity came about to explore these issues further by doing a PhD I could not resist it.

1 *General Introduction*

1.1 *Chapter overview*

How poor emotional health negatively impacts on diabetes health outcomes is unclear. This introductory chapter offers an overview of empirical research which has studied: i) the prevalence of poor emotional health in diabetes and its association with diabetes health outcomes, ii) the causal pathways through which poor emotional health and diabetes are associated, and iii) the effectiveness of interventions which have targeted the management of diabetes and/or poor emotional health. The chapter will then discuss the importance of health psychology theoretical models for exploring how poor emotional health operates in the context of physical long term conditions. Leventhal et al's (1980) Common Sense Self-Regulation Model (CS-SRM) will be described in detail and psychological interventions based on its principles will be discussed. Areas for theoretical and empirical testing will be identified, specifically the need to gain a greater understanding of the pathways through which poor emotional health and physical health interact.

1.2 *Prevalence and aetiology of diabetes mellitus*

Diabetes mellitus is a generic term for a group of conditions which share in common elevated blood glucose levels (Harris and Zimmet, 1985). Type 1 and Type 2 diabetes are the two most common forms of the condition, with a combined estimated prevalence of 4.6% in the UK (Diabetes UK, 2013). Globally an estimated 371 million people have diabetes, with higher prevalence rates in low and middle income countries (International Diabetes Federation, 2012).

Type 1 diabetes is caused by insufficient or non-existent production of insulin from beta cells in the pancreas. Insulin is a hormone which lowers the body's blood glucose levels. Type 1 diabetes typically occurs in children and young adults, as a result of an auto-immune response (International Diabetes Federation, 2011).

Type 2 diabetes is caused by the body's cells becoming insulin resistant and/or due to insufficient insulin production by the pancreas. Type 2 diabetes often occurs in mid to late adulthood. Biological risk factors for its development include: obesity, aging, ethnic origin (in particular black and minority ethnic groups) and having a family history of the disease (International Diabetes Federation, 2011).

1.3 *Management of diabetes mellitus*

Diabetes mellitus affects the homeostatic control of blood glucose levels, therefore individuals with diabetes must undertake conscious control of this process by engaging in self-management behaviours.

Glycosylated haemoglobin (HbA1c) is a measure of glucose concentration in red blood cells over the preceding 12 weeks (National Institute for Health and Clinical Excellence, 2008; NICE). In the UK individuals are encouraged to maintain their HbA1c levels between 6.5% (48mmol/mol) and 7.5% (58 mmol/mol; NICE, 2004; 2008).

To achieve HbA1c targets individuals with Type 1 diabetes are advised to regularly monitor their blood glucose levels using a portable self-monitoring device. The device provides a momentary blood glucose reading and is used to inform decisions about insulin dose administration, nutritional intake and exercise expenditure. The intensity of the treatment regimen depends upon the capabilities and preferences of the individual (NICE, 2004).

For Type 2 diabetes HbA1c targets can be achieved through dietary change and exercise alone. Some individuals may need to take oral and/or injectable medications. The intensity of the treatment regimen depends on the person's degree of insulin insensitivity and/or lack of insulin secretion and their personal capabilities and preferences. The degree of emphasis placed on self-monitoring of blood glucose in Type 2 diabetes therefore depends on a person's treatment regimen (NICE, 2008).

1.4 *Physical health complications and diabetes mellitus*

Diabetic ketoacidosis is an acute, but severe complication that occurs when blood glucose levels become extremely high, because of lack of insulin. It can induce a diabetic coma and in some cases cause death (International Diabetes Federation, 2011).

Individuals must also ensure that their blood glucose levels do not become too low to cause hypoglycaemia. A hypoglycaemic episode can cause acute negative health consequences including: slowed cognitive functioning, dizziness, nausea, and in severe cases loss of consciousness (International Diabetes Federation, 2011).

Chronic moderately elevated blood glucose levels can induce both macrovascular (e.g. coronary artery disease, peripheral arterial disease, and cerebrovascular incidents) and microvascular (e.g. neuropathy, retinopathy, and nephropathy) complications (International Diabetes Federation, 2011). Two large randomised controlled clinical trials demonstrated that tight blood glucose control through intensive behavioural self-management postpones the development of long term diabetes complications in Type 1 (Shamoon, et al., 1993) and Type 2 diabetes (Stratton, et al., 2000).

1.5 *Depression and anxiety in diabetes mellitus*

A meta-analysis found that individuals with diabetes were twice as likely to have depression compared with individuals without diabetes (OR= 2.0, 95% CI 1.8 to 2.2; Anderson, et al., 2001). Anderson, et al. (2000) defined depression as a concept ranging from major depressive disorder to subclinical depression; measured using either diagnostic clinical interviews or self-report questionnaires. The odds ratios were not confounded by the measures of depression used, gender, diabetes type, methodological design or sampling frame.

Similarly, a meta-analysis of 18 studies explored the prevalence of anxiety among adults with diabetes (Grigsby, et al., 2002). Anxiety was measured using either diagnostic clinical interviews or self-report measures. The prevalence rates (unadjusted for potential confounders) for the following were: generalised anxiety disorder, 14%, simple phobia, 22%, subclinical anxiety disorder not otherwise specified, 27% and anxiety related symptoms (measured using self-report measures), 40%. These rates are considerably higher than found in healthy adult populations. For example, in the general UK population, the prevalence of mixed anxiety and depressive disorder is 9% and the prevalence of generalised anxiety disorder is 4.4% (measured using diagnostic clinical interviews; McManus, et al., 2007). Equally, a review of twelve studies found that anxiety (measured using diagnostic interviews or self-report measures) was more likely to occur among an adult population with Type 1 or Type 2 diabetes compared with an adult population without diabetes (OR=1.25, 95% CI 1.10 to 1.39; Smith, et al., 2013).

1.6 *Poor emotional health and HbA1c*

The presence of depression and anxiety among patients with diabetes has been found to be associated with increased levels of HbA1c.

Depression

A systematic review with meta-analysis of 24 cross-sectional studies reported that depression was significantly associated with increased HbA1c levels (r= 0.17, 95% CI 0.13 to 0.21; Lustman, et al., 2000a). Reported effect sizes were similar for both Type 1 (r=0.19) and Type 2 diabetes (r=0.16). The relationship between depression and HbA1c was larger for those studies that measured depression using diagnostic clinical interviews (r=0.28) compared with studies that used standardised self-report measures (r=0.15). The impact of socio-demographic and clinical confounders on outcomes was not tested because of the methodological limitations of included studies.

Anxiety

Equally a meta-analysis of 11 cross-sectional studies reported that anxiety (measured using diagnostic clinical interviews) was associated with increased HbA1c levels (r=0.25, 95% CI 0.10 to 0.38; Anderson, et al., 2002). In contrast, self-report measures of anxiety had a non-statistically significant relationship with HbA1c (r=0.07; 95% CI 0.01 to 0.13). Of the eleven included studies, only one involved individuals with Type 2 diabetes, thus preventing comparisons of the impact of anxiety across diabetes type. This meta-analysis also was unable to adjust for confounding factors because of limitations of included studies.

1.7 *Poor emotional health and diabetes complications*

Depression

A meta-analysis of 27 cross sectional studies explored the relationship between depression and diabetes complications; depression was associated with a greater number or severity of diabetes complications (r=0.25, 95% CI 0.22 to 0.28; de Groot, et al., 2001). A similar effect size was reported for both Type 1 (r =0.21) and Type 2 (r=0.27) diabetes. The complications in which statistically significant relationships were found included: retinopathy, nephropathy, neuropathy, macrovascular complications, and sexual dysfunction. Effect sizes were fairly similar across all complications (r=0.17 to 0.32).

Anxiety

No meta-analysis has been conducted to test the relationship between anxiety and diabetes complications. On the whole studies have shown a relationship between increased levels of

anxiety (measured using either diagnostic clinical interviews or self-report scales) and an increased number and/or severity of diabetes complications (Collins, et al., 2009; Fisher, et al., 2008b; Gore, et al., 2005; Khuwaja, et al., 2010). However, Hermanns, et al. (2005) found no relationship. Differences may be because of methodological heterogeneity in the measurement of diabetes complications (e.g. patients self-report, extraction from medical notes, diagnostic clinical examination).

1.8 *The causal pathways between poor emotional health and diabetes mellitus*

Despite the above studies demonstrating a relationship between poor emotional health and the presence of diabetes and its associated complications the causal direction of these associations is unclear. Most studies researching these relationships have used cross-sectional designs. The onset of depression and anxiety and/or its exacerbation may occur as a consequence of having diabetes. Equally depression and anxiety may provoke the onset and/or exacerbation of diabetes.

1.8.1 *Poor emotional health predicts diabetes onset or vice versa?*

The directional relationship between the onset of depression and the onset of Type 2 diabetes has received the most research attention. A meta-analysis of 18 longitudinal studies examined the relationship between depression and Type 2 diabetes (Mezuk, et al., 2008). The largest effect was found in the direction of depression predicting the onset of Type 2 diabetes (RR 1.60, 95% CI 1.37 to 1.88). Equally, Type 2 diabetes predicted the onset of depression, yet only a modest relationship was reported (RR= 1.15, 95% CI 1.02 to 1.30). In this study, meta-analysed findings were not adjusted for confounders. Depression was measured using diagnostic clinical interview or self-report measures. Consistent with Mezuk et al (2008), Rotella and Manuucci's (2012) meta-analysis of 16 longitudinal studies found that Type 2 diabetes was associated with an increased chance of developing depression (OR=1.29, 95% CI; 1.18 to 1.40).

Fewer studies have tested the hypothesised bi-directional relationship between anxiety onset and Type 2 diabetes onset. Edwards and Mezuk (2012) tested whether generalised anxiety disorder, panic disorder, social phobia, and agoraphobia (measured using diagnostic clinical interviews) at baseline predicted Type 2 diabetes onset at 11 years follow-up on a cohort of 1920 individuals. Their findings found no evidence to support this

direction of effect whilst controlling for demographic variables (OR=1.28, 95% CI 0.75 to 2.18, p>.05). No longitudinal studies were found that tested whether Type 2 diabetes was associated with the incidence of anxiety.

No longitudinal studies have tested the bi-directional relationship between Type 1 diabetes onset and depression or anxiety onset using valid measures of poor emotional health.

Table 1.1 summarises the findings reviewed above, overall:

i) Evidence supports the bi-directional hypothesis between depression and Type 2 diabetes. This conclusion seems highly plausible because it is based on systematic reviews with meta-analysis.
ii) Few studies have tested the relationship between anxiety and Type 2 diabetes onset, thus conclusions about this relationship are limited.
iii) No longitudinal studies have explored the bi-directional relationship between Type 1 diabetes onset and poor emotional health onset.

Table 1.1: Summary of findings of the directional relationship between poor emotional health and diabetes onset

Direction of effect	Effect size estimates
Depression → Type 1 diabetes onset	No published evidence from longitudinal studies for valid measures of depression.
Anxiety → Type 1 diabetes onset	No published evidence from longitudinal studies for valid measures of anxiety.
Depression → Type 2 diabetes onset ++	Mezuk (2008): RR=1.60 (95% CI 1.37 to 1.88; k=13)
Anxiety → Type 2 diabetes onset +	Edwards (2012): OR=1.28 (95%, CI 0.75 to 2.18)
Type 1 diabetes →depression onset	No published evidence from longitudinal studies for valid measures of depression.
Type 1 diabetes → anxiety onset	No published evidence from longitudinal studies for valid measures of depression.
Type 2 diabetes →depression onset ++	Findings from two systematic reviews with meta-analysis: Mezuk (2008): RR=1.15 (95% CI 1.02 to 1.30; k=7) Rotella (2012): OR=1.29 (95% CI; 1.18 to 1.40; k=16)
Type 2 diabetes → anxiety onset	No published evidence from longitudinal studies for valid measures of depression.

Key: CI, confidence interval; k, number of studies included in meta-analysis; RR, Relative risk; OR, Odds ratio; ++, reported findings are based on systematic review(s), +, findings are based on a small number of individual studies. All studies had to use valid and reliable measures of depression and anxiety.

1.8.2 *Poor emotional health exacerbates diabetes or vice versa?*

Poor emotional health and HbA1c

Longitudinal studies examining the directional effect of depression on HbA1c at follow-up have found both statistically significant and non-significant results among adults with Type 2 diabetes (Aikens, 2012; Dirmaier, et al., 2010; Heckbert, et al., 2010; Richardson, et al., 2008). The mixed findings may be a consequence of study differences in the: i) time interval between the measurement of depression and the measurement of HbA1c as these ranged from 6 months to 5 years, ii) measures of depression used, iii) statistical control of potential confounding variables, and iv) baseline depression severity.

No studies have longitudinally tested the effects of baseline depression on HbA1c at follow-up among individuals with Type 1 diabetes. Similarly, no studies have explored the directional effect of anxiety on HbA1c in either Type 1 or 2 diabetes.

Studies examining the reverse directional pathway, the effect of HbA1c levels on the exacerbation and persistence of depression and anxiety are limited. Fisher, et al. (2008b) found a relationship between high levels of HbA1c at baseline and persistence of depression (continuous self-report measures) at 9 and 18 month follow-up in adults with Type 2 diabetes. HbA1c levels had no effect on the development of major depressive disorder, generalised anxiety disorder, panic disorder, and mixed depression and anxiety disorder when measured using diagnostic clinical interviews. Aikens, et al. (2009) found no effect of baseline HbA1c on depression (continuous self-report measure) at 6 months follow-up in adults with Type 2 diabetes.

Table 1.2 summarises reported effect sizes and levels of statistical significance for longitudinal studies of the hypothesised bi-directional relationship between poor emotional health and HbA1c. On balance it seems likely that the relationship between poor emotional health and HbA1c is bi-directional yet the factors that determine whether these relationships are observed needs to be systematically examined.

Table 1.2: Summary of findings of the directional relationship between poor emotional health and HbA1c

Direction of effect	Studies identified and their associated effect sizes and p values
Depression → HbA1c control in Type 1 and/or Type 2 diabetes +	Aikens (2012): β=0.07, p>0.05 (depression continuous variable, 6 months follow-up). Dirmaier (2010): Depressed participants were 2.67 times (OR) more likely to have HbA1c ≥7% at 12 months follow-up. Heckbert (2010): Mean difference in HbA1c between majorly depressed and non-depressed group= +10%, 95% CI -0.03 to 0.24, p>0.05 (5 year follow-up period). Richardson (2008): Mean difference in HbA1c between majorly depressed and non-depressed group +13%, 95% CI -0.03- to 0.22, p≤0.05 (4.1 year mean follow-up).
Anxiety → HbA1c control in Type 1 and/or Type 2 diabetes	No published evidence from longitudinal studies for valid measures of anxiety.
HbA1c control in Type 1 and/or Type 2 diabetes →exacerbates depression +	Aikens (2009): β=0.04, p≥0.05 (depression continuous variable, 6 months follow-up). Fisher (2008): β=0.11, p≤0.01 (depression continuous variable, persistence of depression at both 9 and 18 months follow-up). When depression was a categorical variable findings were NS.
HbA1c control in Type 1 and/or Type 2 diabetes →exacerbates anxiety +	Fisher (2008): NS effects on persistence of GAD, panic disorder and mixed anxiety and depression (categorical variables) at both 9 and 18 months follow-up.

Key: CI, Confidence interval; GAD, Generalised Anxiety Disorder; NS, Non-significant; OR, Odds ratio; ++, reported findings are based on systematic review(s), +, findings are based on a small number of individual studies. All studies had to use valid and reliable measures of depression and anxiety.

Poor emotional health and diabetes complications

Longitudinal studies have shown that depression influences the development and severity of microvascular (Gonzalez, et al., 2010; Sieu, et al., 2011; Williams, et al., 2010) and macrovascular complications (Lin, et al., 2010) in patients with Type 2 diabetes or a combined sample of Type 1 or 2 diabetes. This directional relationship has not been explored for anxiety.

Studies that have investigated the longitudinal impact of diabetes complications or associated procedures (e.g. angioplasty) on the presence/severity of depression or anxiety at follow-up have shown mixed statistically significant and non-significant findings (Fisher, et al., 2008b; Katon, et al., 2009a; Nefs, et al., 2012; Vileikyte, et al., 2009). Identified studies were methodologically heterogeneous in terms of: i) the measurement of diabetes complications and poor emotional health, ii) the time interval between baseline and follow-up assessments, and iii) the population studied (e.g. a Type 2 diabetes sample only or a combined sample of Type 1 and Type 2 diabetes).

Table 1.3 summarises the findings of longitudinal studies which have explored the relationship between poor emotional health and diabetes complications. Overall:

i) Studies provide support for the influence of poor emotional health on the development or exacerbation of diabetes complications.
ii) Evidence in support of the directional effect of diabetes complications on poor emotional health is mixed, likely because of methodological and statistical heterogeneity across the studies.
iii) Classification of studies according to their methodological and statistical procedures is needed to gain a precise understanding of the contexts in which directional relationships are observed.

Table 1.3: Summary of the findings of the directional relationship between poor emotional health and diabetes occurrence/exacerbation

Direction of effect	Studies identified and their associated effect sizes and p values
Depression → diabetes complications in Type 1 and/or Type 2 diabetes +	Gonzalez (2010): Increased chance of developing first ever foot ulcer, HR=1.68, 95% CI 1.20 to 2.35, p≤0.01(depression continuous variable, 18 month follow-up) Lin (2010): Increased risk of macrovascular complications in participants with MDD compared with those with no depression, HR=1.25, 95% CI 1.00 to 1.54, p≤0.05 (depression categorised from self-report measure, 5 year follow-up period) Sieu (2011): Increased risk of developing retinopathy, OR=1.03, 95% CI 1.00 to 1.05, p≤0.05 (depression continuous variable, 5 year follow-up period) Williams (2010): Increased risk of foot ulceration in participants with MDD compared with those with no depression, HR=2.00, 95% CI 1.25 to 3.25, p≤0.05 (depression categorised from self-report measure, 5 year follow-up period)
Diabetes complications in Type 1 and/or Type 2 diabetes →exacerbates depression +	Katon (2009a): Increased risk of MDD among patients who have had coronary procedures, OR=1.92, 95% CI 1.10 to 3.35, p≤0.05. Findings were NS for specific microvascular and macrovascular complications (depression categorised from self-report measure, 5 year follow-up period). Nefs (2012): Baseline cardiovascular, OR=1.10, 95% CI 0.80 to 1.51, p>0.05 and microvascular complications, OR=1.33, 95% CI 0.91 to 1.95, p>0.05 had NS effects on the presence of depression (depression categorised from self-report measure, 2.5 year follow-up)

Table 1.3: Poor emotional health predicts the occurrence/exacerbation of diabetes complications and vice versa, summary of findings continued

Direction of effect	Studies identified and their associated effect sizes and p values
Diabetes complications in Type 1 and/or Type 2 diabetes →exacerbates depression +	Fisher (2008): Number of diabetes complications had a NS effect on persistence of depression when measured by either self-report (β=0.06) or diagnostic clinical interview (β=0.00) over an 18 month period. Vileikyte (2009): baseline neuropathy disability score predicted increased depression: β=0.10, p≤0.05 (Depression continuous variable, 18 month follow-up).
Diabetes complications in Type 1 and/or Type 2 diabetes →exacerbates anxiety +	Fisher (2008): Number of diabetes complications had a statistically significant relationship with the persistence of GAD (β=0.13, p≤.01) and mixed anxiety and depression (β=0.11, p≤0.05) when measured using diagnostic clinical interviews over an 18 month follow-up period. The relationship for panic disorder was NS (β=0.04).

Key: CI, Confidence interval; GAD, Generalised Anxiety Disorder; HR, Hazards ratio; MDD, Major Depressive Disorder; NS, Non-significant; OR, Odds ratio; ++, reported findings are based on systematic review(s), +, findings are based on a small number of individual studies. All studies had to use valid and reliable measures of depression and anxiety.

1.9 *The mechanisms through which poor emotional health and diabetes mellitus interact*

The proposed mechanisms through which poor emotional health and diabetes interact involve both biological and behavioural pathways.

Studies of the hypothesised biological mechanisms have shown that insulin resistance can occur because of increased hypothalamic-pituitary-adrenal (HPA) activity, sympathetic nervous system activation, and cytokine secretion; these biological mechanisms are active in depressed and anxious populations (Rustad, et al., 2011). Equally, Type 2 diabetes can trigger increased pro-inflammatory cytokine secretion because of obesity and/or aging (Musselman, et al., 2003). Pro-inflammatory cytokines promote "sickness behaviour" a

response that has symptoms akin to depression for example loss of appetite, lethargy (Dantzer, 2001).

Research on the hypothesised behavioural pathway has shown a relationship between depression and anxiety and unhealthy behaviours (e.g. exercise, poor diet and smoking; Strine, et al., 2008). These lifestyle factors are known to contribute to the development of Type 2 diabetes and associated complications (International Diabetes Federation, 2011).

In adults with existing Type 1 and Type 2 diabetes studies have consistently shown a relationship between depression and decreased adherence to: medication regimens (Ciechanowski, et al., 2000; Gonzalez, et al., 2008b; Katon, et al., 2009b; Lin, et al., 2004), decreased physical exercise (Katon, et al., 2010; Lin, et al., 2004; Piette, et al., 2004), poor diet/weight control (Chiu, et al., 2010; Ciechanowski, et al., 2000; Gonzalez, et al., 2008b; Katon, et al., 2010; Lin, et al., 2004), decreased self-monitoring of blood glucose (Gonzalez, et al., 2008b), and missed health care appointments (Gonzalez, et al., 2008b).

The relationship between anxiety and behavioural adherence has received less research attention. Evidence suggests that diabetes specific anxieties including injection phobia and fear of hypoglycaemia are associated with decreased adherence to treatment regimens (Mollema, et al., 2001; Shiu and Wong, 2000; Zambanini, et al., 1999). In addition, studies have shown non-diabetes specific anxieties (measured using self-report) to impact negatively on behavioural adherence (Herzer and Hood, 2010; Zambanini, et al., 1999).

1.10 *An overview of treatment interventions targeting poor emotional health and/or glycaemic control*

A meta-analysis of 49 randomised controlled trials examined the impact of psychosocial interventions on both glycaemic control and poor emotional health in persons with Type 1 and Type 2 diabetes (Harkness, et al., 2010). types of interventions included in the meta-analysis targeted either: i) diabetes control via lifestyle management strategies, ii) poor emotional health via psychological therapies, or iii) used a combination of lifestyle and psychological management techniques. Across the included studies, poor emotional health was measured using self-report scales of depression, anxiety or negative affect.

Overall, interventions improved HbA1c (standardised mean difference = -0.29, 95% CI -0.37 to -0.21) and poor emotional health (standardised mean difference= -0.16, 95% CI -0.25 to -0.07). Findings did not differ according to how poor emotional health was measured or diabetes type. However, interventions did not improve both diabetes control and poor emotional health simultaneously (r=0.25, p=0.08). Indeed, treatment effects were reduced after removing an outlying study (Pearson r= 0.11, p=0.45).

Similarly, pharmacological treatments for depression in adults with Type 1 and Type 2 diabetes have improved depression in the intervention groups relative to the control group (Lustman, et al., 1997; Lustman, et al., 2000b). However a corresponding statistically significant difference in HbA1c between the intervention and control groups in each of the interventions was not found. No randomised controlled trials have explored the impact of pharmacological treatments for anxiety on HbA1c.

Taken together these findings demonstrate that the causal relationship between diabetes mellitus and poor emotional health is complex. Improvements in one condition have not demonstrated the anticipated improvements in the other. These findings may be a consequence of intervention studies having insufficient follow-up periods to allow beneficial effects to be observed. The mean follow-up period for the studies included in Harkness et al's (2010) review was 6.8 months. Likewise, the pharmacological interventions for depression had a follow-up period of 8 weeks only (Lustman et al., 1997; 2000b).

Also, the current hypothesised biological and behavioural relationships between poor emotional health and diabetes are reductionist. They do not precisely define the causal pathways through which poor emotional health and diabetes interact. Without this knowledge treatment interventions cannot be developed that are mutually beneficial to the management of both negative emotional states and diabetes. This thesis elaborates on the hypothesised behavioural pathway. Specifically, the route through which poor emotional health negatively impacts on adherence to diabetes self-care behaviours will be explored with the use of health psychology based theoretical concepts.

1.11 *Theoretical Models in Health Psychology*

Health psychology theoretical models provide an adequate foundation to explore factors that influence health behaviours among individuals with chronic illness. Social cognition

models are a group of theoretical frameworks which emphasise the importance of socially derived cognitions in determining health behaviours (Conner, 2005). Prominent social cognition models typically applied to promote and maintain health are summarised in Table 1.4.

Table 1.4: Summary of prominent social cognition models

Social cognition models	Cognitions included in model
Health Belief Model (Becker, et al., 1977)	Perceived health threat (susceptibility and severity) Perceived benefit and costs of health behaviour
Theory of Planned Behaviour (Ajzen, 1991)	Attitudes (outcome expectancies and perceived benefits of behaviour) Perceived behavioural control Subjective norms All cognitions combine to generate a behavioural intention which determines subsequent behaviour.
Protection Motivation Theory (Rogers, 1983)	Threat appraisal (susceptibility and severity of health threat) Coping appraisal (includes evaluation of personal capabilities and efficacy of the behaviour being evaluated) Threat and coping appraisals combine to produce 'protection motivations' to perform appropriate health behaviours
Social Cognitive Theory (Bandura, 1982)	Self-efficacy Outcome expectancies

The theoretical models summarised in Table 1.4 identify important determinants of health behaviours. However Leventhal et al's (1980) Common Sense Self-Regulation Model (CS-SRM) is a more suitable theoretical framework to apply to aid our understanding of the determinants of health behaviours in the context of chronic illness. This is because the CS-SRM:

i. Is concerned with determinants of health behaviours when a person is experiencing an illness episode (e.g. self-management).
ii. Is derived from empirical studies among patients with existing chronic conditions (Meyer, 1981 cited in Leventhal, et al. 1980 p 224; Nerenz, 1979 cited in Leventhal, et al. 1980 p229) promoting greater specificity for determinants of health behaviours among individuals with chronic illness.
iii. Acknowledges a person's emotional response to a health threat as an important determinant of health behaviour.
iv. Recognises the social context.

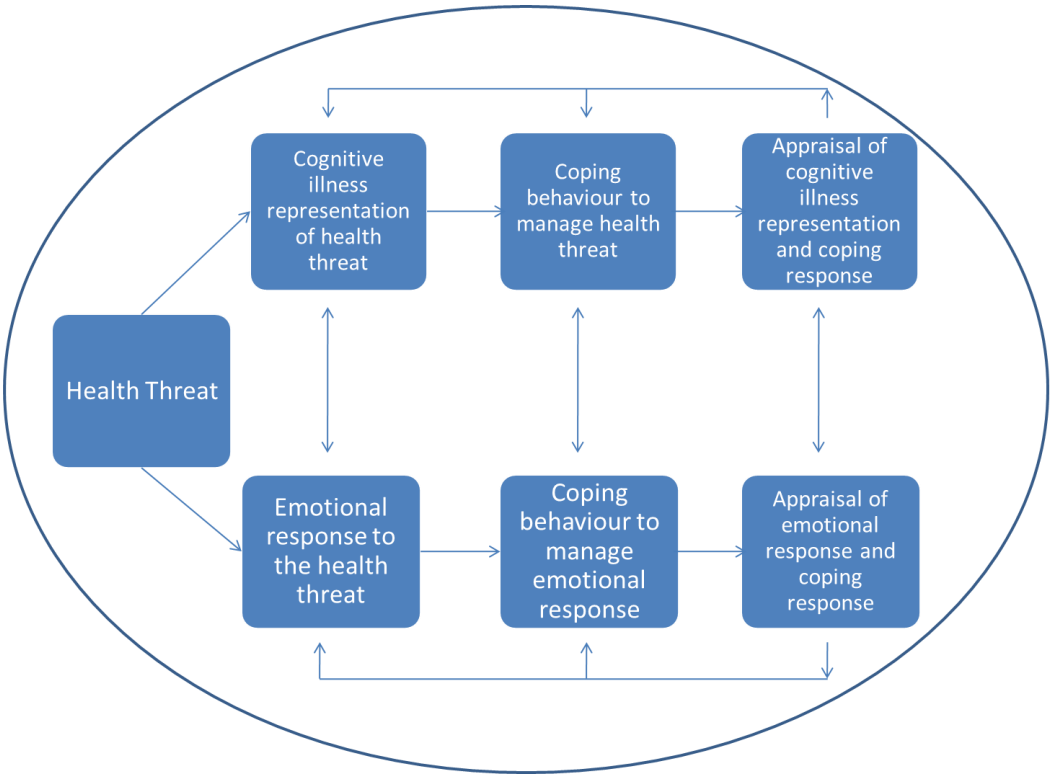
1.12 *Overview of Common Sense Self-Regulation Model (CS-SRM)*

The CS-SRM (Leventhal, et al., 1980; Leventhal, et al., 1984) suggests that when presented with a health threat two parallel partially independent information processing systems are activated: a cognitive processing system and an emotional processing system (Figure 1.1).

1.12.1 *Cognitive processing system*

The cognitive processing system consists of three stages: the development of a cognitive illness representation (illness cognitions) in response to the health threat, a coping response to manage the health threat, and an appraisal phase. The CS-SRM proposes that cognitive illness representations causally inform coping behaviours aimed at managing the health threat. Both cognitive illness representations and coping behaviours are continually appraised and updated for their adequacy. The processing of a health threat is therefore not unidirectional but dynamic.

Figure 1.1: The common sense self-regulation model (Leventhal et al., 1980; 1984)



Key: Ellipse represents social context

Cognitive illness representations of the health threat

Cognitive illness representations provide a framework to understand and interpret the health threat (Leventhal, et al., 1980; Leventhal, et al., 1984). There are a number of interacting domains which include: identity (knowledge of the symptoms associated with the health threat and the disease label attributed to it), cause (causal factors assigned to the health threat), consequences (impact of the health threat on a person's lifestyle), timeline (perceived duration of the health threat), and finally control (manageability of the health threat in terms of personal resources available).

Subsequent research has led to the generation of additional illness cognition domains (Moss-Morris, et al., 2002), these include: illness coherence (degree of illness understanding), treatment control (perceived efficacy of treatment), and timeline cyclical (variability of illness symptoms). The domains treatment control and timeline cyclical were generated in response to empirical work that had used the Illness Perception Questionnaire

(IPQ; Weinman, et al., 1996). The IPQ was a measure designed to quantitatively assess the five illness cognition domains outlined in the CS-SRM. However the IPQ had low internal consistencies when measuring control and timeline. Exploratory factor analyses revealed that these domains could be subdivided into perceptions of personal and treatment control, and perceptions of the health threat as either being acute/chronic in nature, or cyclical in course. The illness coherence domain was generated to add explanatory value in determining adherence to health behaviours. These findings resulted in an updated version of the IPQ named the Illness Perception Questionnaire-Revised (IPQ-R; Moss-Morris, et al., 2002).

In addition to cognitive illness representations, Horne et al. (1999) emphasise the importance of medications beliefs for determining medication adherence. Horne et al. (1999) created the Beliefs about Medicines Questionnaire (BMQ). It includes the domains: necessity (perceived requirement for medications), concerns (degree of worry about taking medication), harm (perceived detrimental effects of medication), and overuse (perceived excess use of medications).

The CS-SRM acknowledges the social context within which a person generates and refines their cognitive illness representations. It recognises the impact of the following on illness cognitions:

i. Cultural perspectives including the language used to communicate about illness.
ii. Social interactions with significant others (e.g. health practitioners, others with the illness).
iii. The illness experience whereby information from previous illness episodes are used to elaborate on the cognitive representation of the current health threat.

Indeed Leventhal, et al. (1984) emphasise the importance of identifying the unique social conditions which influence illness representation perspectives.

Coping response to the health threat

The CS-SRM proposes that cognitive illness representations provide a frame of reference to guide the selection of coping procedures to manage health threats (Leventhal, et al., 2010). Hagger and Orbell (2003) synthesised the theoretical claims of the CS-SRM by conducting a meta-analysis of 45 studies which quantitatively explored the relationship between illness cognitions and coping behaviours. The included studies provided data across 23 different health conditions of which the majority (20 out of the 23) were physical long-term conditions and the remaining three were acute illnesses. For the purposes of aggregating the data across included studies Hagger and Orbell (2003) categorised the measures of coping behaviours into the following domains: problem focussed coping (generic), problem focus coping (illness specific), avoidance, emotional expression, cognitive reframing, social support, and frequency of doctor visits. Only the illness cognition domains identity, timeline, consequences, and control (personal control and treatment control) were meta-analysed. The cause domain was deemed too heterogeneous to include in the quantitative synthesis.

Only the control domain demonstrated a relationship with both generic problem focussed health behaviours ($r=0.27$, $p\leq0.05$) and specific problem focussed health behaviours ($r=0.12$, $p\leq0.05$). Greater perceptions of control were associated with increased problem focussed behaviours. The remaining illness cognition domains were associated with emotion focussed coping strategies.

Hagger and Orbell's (2003) findings highlight that perceptions of control play an important role in driving problem focussed coping behaviours. The meta-analysis was largely based on cross-sectional studies (33 out of 45), preventing conclusions about the causal direction of the association between illness cognitions and health behaviours. In addition, the relative value of illness cognitions in relation to health behaviours may differ across individual long-term conditions because of the unique self-management challenges of each condition.

A number of studies have explored the relationship between illness cognitions and adherence to diabetes specific health behaviours, including diet, exercise, self-monitoring of blood glucose, and medication adherence (Barnes, et al., 2004; Broadbent, et al., 2011; Hampson, et al., 1990; Hampson, et al., 1995; Searle, et al., 2007). Consistent with the claims of the CS-SRM all studies demonstrated that illness cognitions were associated with

health behaviours; however there was considerable variation between the studies about which domains played a prominent role. These differences occurred across and within diabetes self-care behaviours (e.g. diet, exercise, medication adherence). Differences in findings may reflect variation in the measures of diabetes self-care behaviours, the populations studied, and the timeframe in which measures were administered.

McSharry et al. (2011) meta-analysed nine studies which tested the relationship between illness cognitions and HbA1c. HbA1c is partly determined by adherence to diabetes self-care and arguably is a proxy measure of adherence. Because HbA1c can be uniformly measured the relationships between illness cognitions and HbA1c can be more validly compared across studies. Using HbA1c as a proxy measure of adherence overcomes the challenges associated with heterogeneous measures of diabetes self-care. McSharry et al. (2011) found that the illness cognition domains: identity (r=0.14), consequences (r=0.14), timeline cyclical (r=0.26), and personal control (r=-0.12) had statistically significant associations with HbA1c. However all meta-analysed studies were cross-sectional preventing conclusions about direction of effects. In addition the extent to which HbA1c provides an accurate representation of adherence to health behaviours is limited. This may account for the small reported effect sizes. Variance in HbA1c is explained by factors other than adherence including: biological factors, treatment intensity, diabetes duration, and other health co-morbidities (Johnson, 1992).

Appraisal of the cognitive illness representations and coping response to the health threat

The CS-SRM views an individual as an active problem solver continually involved in the pursuit of maintaining good health. The cognitive processing system is therefore dynamic. Feedback on a person's cognitive illness representation and coping procedure for the health threat are continually evaluated.

1.12.2 ***Emotional Processing System***

The emotional processing arm of the model represents negative emotional responses to health threats, coping responses used to deal with emotions, and an appraisal phase of both the emotional response and the effectiveness of the strategy selected to deal with it. The CS-SRM is an elaboration on the parallel processing model (Leventhal, 1970), a theory developed to explain the role of fear and distress in the adoption of health behaviours.

The parallel process model (Leventhal, 1970) was developed to account for empirical findings which demonstrated no difference between high and low fear based messages in the uptake of health behaviours (findings of which are reviewed in Leventhal et al., 1980 and Leventhal, et al., 2003). It was anticipated that greater levels of fear content in messages would be better predictors of behavioural change because individuals would be motivated to offset the increased arousal provoked by fear. Some degree of fear (independent of level) was required to promote behavioural uptake, yet it was dependent on being combined with a specific action plan or information (e.g. effectiveness of treatment, susceptibility to negative health consequences). Neither fear nor specific action plans/information alone was sufficient to motivate behavioural change.

Although high fear messages did demonstrate marginal independent effects on health behaviours, this was time limited lasting a few hours. Indeed, fear on some occasions was detrimental and promoted avoidance behaviours, particularly when the recommended health behaviour was related to the detection of ill health (e.g. x-ray for the identification of cancer), rather than health promoting (e.g. stopping smoking). The parallel process model therefore emphasised the necessity of both emotional and cognitive processes in promoting health behaviours. This model recognised that strategies to manage the health threat are independent of strategies that manage fear/distress but the two processes interact with each other. The model therefore provided the foundations for the development of the CS-SRM.

Emotional responses to health threats

The parallel process model largely considered the role of fear and distress specifically. In contrast the CS-SRM considers a wider range of emotional reactions that can occur in response to health threats (Leventhal, et al., 1984). Cameron (2003) has outlined the common emotional responses that can occur, including: depression, anxiety, anger and in some cases positive emotions. Empirical research however has largely focussed on depression and anxiety which are present in other long term conditions in addition to diabetes including: coronary heart disease (Evans, et al., 2005; Todaro, et al., 2007), arthritis (Dickens, et al., 2003; Isik, et al., 2007), asthma (Weiser, 2007; Zielinski, et al., 2000), and cancer (Massie, 2004; Stark and House, 2000). The IPQ-R has a subscale which measures negative emotional responses provoked by illness and includes items pertinent to depression, anxiety, and anger.

Coping with emotional responses to the health threat

The type of coping behaviours initiated as a result of a person's emotional response to the health threat has received less research attention. This is perhaps a consequence of difficulties in the conceptualisation and measurement of behaviours that are specifically targeted at controlling a person's emotions. Cameron and Jago (2008) defined four types of coping procedures that may occur when an anxiety response is provoked by a health threat. The coping procedures outlined by Cameron and Jago (2008) are an adaptation of those originally conceptualised by Gross (1999) and include: altered attention-directed at either avoiding or focussing on the health threat, proactive behaviours-aimed at addressing the health threat, cognitive change-appraising and understanding the health threat, and response modulation-attempts at managing the experience of emotion (e.g. relaxation, expression, suppression).

Emotional coping responses consistent with those outlined by Cameron and Jago (2008) have been reported across a number of long term conditions including diabetes (Macrodimitris and Endler, 2001), coronary heart disease (Karademas, et al., 2011), cancer (Schlatter and Cameron, 2010), and arthritis (Affleck, et al., 1999) and occur alongside the experience of both anxiety and depression. However the majority of studies have focussed on emotional expression or suppression. Whether the coping behaviours outlined by Cameron and Jago (2008) occur in response to negative emotions or indeed provoke negative emotions remains unclear because the majority of studies conducted in this area are cross-sectional, with the exception of Schlatter and Cameron (2010). They found that emotional suppression was longitudinally associated with changes in mood states over a follow-up period of 84 days. Suppression of anxious emotions was associated with lower reports of anxiety at follow-up, yet no effects were observed for depression and anger (Schlatter and Cameron, 2010).

Appraisal of emotional response and coping efforts to control affect

Consistent with the cognitive processing system, the emotional processing system occurs in a continuous cycle of appraisal and reappraisal. The emotional response and related coping procedure are evaluated for their efficacy.

1.13 *The concrete and abstract illness experience – a hierarchical processing system*

It is important to note that the CS-SRM acknowledges that illness information is processed at both a concrete and abstract level. Concrete illness information is the somatic symptoms experienced by individuals. Abstract illness information is the conceptual illness cognition domains individuals use to appraise and understand their illness. Leventhal et al. (1980) describe how the concrete and abstract levels of illness information interact with each other. Somatic (concrete) symptoms encourage abstract illness cognition domains to be elaborated on. For example, the cognition domain identity will alter in response to somatic cues and the symptoms may be labelled as a physical health threat or an emotional response (consistent with James-Lange theory of emotion reviewed by Lang, 1994). Equally, abstract illness cognitions domains specifically identity promote a symptom matching search for somatic signs which correspond to a person's disease label for their health threat (Meyer, et al., 1985). Disparities between concrete and abstract illness representations may explain differences in adherence, whereby a person is only driven to behave in a health protective manner when they are experiencing concrete symptoms associated with the health threat.

1.14 *Interaction between the cognitive processing system and the emotional processing system*

The CS-SRM proposes that the cognitive and emotional processing systems are largely independent but with some degree of interaction with each other. The interplay between the two processing systems can occur between illness cognitions and emotional responses. The proposed mechanisms through which this occurs include: altered attention and information processing at both the abstract and concrete level.

1.14.1 *Altered attention*

Negative emotional responses to health threats can promote self-focussed attention to somatic symptoms (Cameron, 2003; Leventhal, et al., 1984). Once a somatic change is detected (accurate or not) it encourages a person to elaborate on their cognitive illness representation of the health threat. The cognitive change induced by a person's emotional response equally can reinforce the negative emotional response that elicited it. A person enters a vicious cycle of negative emotional responses driving illness cognitions and illness cognitions worsening emotional responses (Leventhal, et al., 1984).

Empirical studies support this hypothesis. Salovey and Birnhaum (1989) tested the impact of mood on symptom perception using mood induction techniques among individuals both with and without acute illness. Participants who had low mood induced via imagery reported an increased frequency of symptoms (e.g. pain) compared with participants whose mood was positively induced. The external validity of this study is limited however by its experimentally induced mood states.

Leventhal et al. (1996) performed two separate studies recruiting participants from primary care settings in study one and community settings in study two. Interviews were conducted with participants at baseline and six months follow-up to measure mood (depression and anxiety), self-reported physical symptoms, and demographic variables. In both studies increased baseline levels of depressive affect predicted increased physical symptom reporting at six months follow-up. An effect for baseline anxiety was also observed but only in the community sample, probably because the measure of anxiety differed across the two recruitment settings. Whilst this study provides support for the relationship between mood and symptom perception outside of experimental settings it remains equally plausible that somatic changes experienced and detected by participants are real because poor emotional health induced physical ill health via immunological pathways (de Ridder, et al., 2008).

Studies which measure both subjective symptom perceptions via self-report and objective measures of illness allow the altered attentional processes associated with mood to be more rigorously tested. Main et al. (2003) explored whether individuals with asthma and high in state negative affect attributed a higher number of symptoms to their asthma independent of objective measures of asthma symptoms. Over one week participants recorded daily their mood, self-perceived asthma symptoms, and peak flow (objective measure of asthma

symptoms). Multivariate regression analyses demonstrated that both participants daily negative affect scores and objective asthma symptoms remained as independent predictors of symptoms attributed to asthma, with the largest effect size occurring for negative affect. If low mood was having an effect via actual somatic change then it would not have demonstrated an independent relationship with self-reported symptoms relative to objective symptom measures.

1.14.2 *Information processing-abstract cognitive illness representations*

A person's cognitive representation and processing of the health threat can influence their emotional response (Leventhal et al., 1980; 1984). If a person appraises the health threat in a pessimistic manner then this will encourage an individual to feel more negative about their health and generate a negative emotional response. This proposed mechanism is consistent with cognitive theories of emotion and their associated empirical work (Beck, 1967; 1976; Beck, et al., 1979). Cognitive theories of emotion suggest that the appraisal of a situation and its associated thought content determines a person's emotion response.

Equally however emotions can bias cognitive appraisals of events (Cameron, 2003). Affective states influence information processing strategies by causing mood congruent memories, schemas, and thoughts to be accessed and elaborated on. For example an anxious individual may retrieve threatening health information and integrate this into their cognitive illness representation for their current health threat. This mechanism is consistent with both Forgas' (2000) explanation of the affect infusion model and Teasdale's (1983; 1988) differential activation hypothesis which argues that mood permeates thoughts by increasing access to mood consistent information.

Empirical evidence in the context of diabetes support the hypotheses that mood is determined by and/or determines cognitions. Lange and Piette (2005) cross-sectionally explored whether lower perceptions of personal control were associated with poorer mental health (self-report measure). They also tested the accuracy of participant's perceptions of control by studying its relationship with HbA1c. Participants with poorer mental health perceived their personal control over diabetes to be worse. Of note participants who demonstrated poorer mental health were more likely to inaccurately rate their diabetes control as poor when it was adequate compared with those participants who accurately rated their diabetes control as poor. Conversely participants with better mental health were

more likely to inaccurately rate their diabetes control as adequate when it was indeed poor compared with those participants who accurately rated their control as adequate.

Whilst the causal relationship between cognitions and mood cannot be determined by Lange and Piette's (2005) study because of its cross-sectional design their findings emphasise the potential for either mood and/or diabetes "misregulation" to occur (Detweiler-Bedell, et al., 2008). Dysfunctional beliefs and their associated maladaptive moods lead individuals to erroneously conclude that their diabetes management is poor, leading to feelings of helplessness and eventual withdrawal from diabetes self-care practices. Conversely, optimistic beliefs and their associated positive emotional states allow an individual to maintain a positive mood, yet efforts at managing their physical health become thwarted as their mood is biasing judgements of their diabetes control.

1.14.3 *Information processing-concrete illness experience*

An alternative but not mutually exclusive explanation for Lange and Piette's (2005) findings is that negative affective states and their associated physiological symptoms (e.g. tiredness, restlessness, sweating) are misinterpreted as being concrete somatic symptoms associated with diabetes (Cameron, 2003). Individuals use their affective states as heuristics to guide their interpretation of the physical health threat and erroneous conclusions may occur (Leventhal et al., 1980). Qualitative research in people with multi-morbid conditions have shown individuals report difficulties in appropriately labelling their somatic symptoms to the relevant health conditions (Bower, et al., 2012; Mc Sharry, et al., 2013).

1.15 *Optimising the management of both the health threat and negative emotional responses*

Detweiler-Bedell et al. (2008) acknowledge that the effective management of both the cognitive and emotional processing system can be difficult to achieve and can often result in self-regulation failure. They describe how the management of one processing system is often prioritised over the management of the other. Indeed Leventhal et al. (1980) highlighted that an individual has a limited number of resources available to them to effectively manage both processing systems. Qualitative research provides empirical support for this argument as individuals reported prioritising their health conditions (Bower, et al., 2012; Mc Sharry, et al., 2013).

Depletion in resources can be either behavioural or physical. If a person prioritises the management of the physical health threat then they have a limited number of behavioural resources available to effectively manage their emotions and vice versa. Equally, the somatic symptoms associated with the physical health threat may disrupt the physical resources a person has available to them to manage their emotions. A person suffering from painful diabetic neuropathy for example may not have the physical capabilities to participate in behaviours that can effectively manage their emotional health. Thus attempts to manage their emotions are thwarted because of symptomatic barriers. Similarly, the somatic symptoms associated with negative emotional responses to the health threat e.g. lack of energy, tiredness, change in appetite, deplete the physical resources available for the management of physical health (Detweiler-Bedell, et al., 2008; Leventhal, et al., 1984).

The acknowledgement of the potential for physical or emotional prioritisation to occur aids with the identification of strategies for intervention which efficiently use the limited resources an individual has available to them. Perhaps of more concern to the design of interventions is when the effective management of one processing system is antagonistic to the effective management of the other (e.g. "misregulation" occurs, Detweiler-Bedell et al., 2008).

The effective management of a person's emotional health can be counter-intuitive to a person's management of physical health via a number of pathways. Firstly, if a person's emotional health improves; they may become less vigilant to their physical health status because their attentional processes are less aroused. This may prevent the detection of somatic cues as indicators of worsening physical health status (Detweiler-Bedell et al., 2008). In addition a person may use their improved emotional health status as an indicator that their physical health has improved (Leventhal et al., 1980) and no longer perform the required self-management practices.

Secondly, if an individual adopts the coping mechanism of cognitive reappraisal to offset the negative emotional response provoked by the health threat an improvement in their emotional health may occur; but this may result in unrealistic perceptions of the health threat. Leventhal et al. (2001) highlight the importance of "hot cognitions" to motivate behavioural change. Hot cognitions are emotionally salient cognitive illness representations. For example the belief that "My diabetes is a chronic condition", is related to an interpretation of diabetes as a condition that continually needs managing. If the health

threat is appraised in a manner that does not warrant a coping procedure an individual may not adhere to their treatment regimens.

Equally, effective management of physical health can be counter-productive to the effective management of emotional health. For example, adhering to a strict treatment regimen may cause an individual to appraise their diabetes as having a large amount of consequences on their life. Such negative interpretations of diabetes may provoke a negative emotional response to diabetes.

1.16 *Interventions based on the CS-SRM*

The CS-SRM provides a useful theoretical framework to identify how physical and emotional health can be effectively managed through clinical intervention. Few intervention studies however have applied the theoretical principles of the CS-SRM. Of the interventions applying the concepts outlined in the CS-SRM, most have focused on the management of the physical health threat by addressing a participant's maladaptive illness cognitions.

Petrie et al. (2002) designed the first intervention based on the CS-SRM. It was delivered in a hospital to people who had had a myocardial infarction. The intervention informed patients about the symptoms associated with coronary events, challenged inaccurate beliefs about the duration, cause and anticipated consequences of coronary events and increased perceptions of control about implementing lifestyle management action plans and their efficacy. Immediately after the intervention, patients (n=31) reported lower timeline and consequences beliefs, lower levels of distress (2 item scale) and greater perceptions of control relative to the usual care group (n=34). No statistically significant differences occurred for the identity subscale and outcome data on cause was not reported. At three months follow-up the differences between the intervention group and control group only remained statistically significant for the illness cognition domains timeline and control. In terms of behavioural and physical functioning participants in the intervention group reported fewer coronary related symptoms and returned to work sooner than patients in the control group after controlling for demographic and clinical variables.

Since the publication of Petrie et al's (2002) study, subsequent interventions based on the CS-SRM aimed at managing the physical health threat have been conducted across a number of conditions including: acute coronary events (Broadbent, et al., 2009), psoriasis

(Fortune, et al., 2004), and asthma (Petrie, et al., 2012). These interventions were effective in altering participants' illness cognitions and associated improvements in behavioural and/or health outcomes; Fortune et al's (2004) findings were not statistically significant however, possibly because the study lacked statistical power. Differences across the studies with regards to which illness cognition domains changed in response to interventions were evident and may reflect the relative importance of specific illness cognitions domains across different long term conditions.

In people with diabetes, four studies have either explicitly targeted illness cognitions (Keogh, et al., 2011) or elements of the intervention indirectly addressed them (Davies, et al., 2008; French, et al., 2008; George, et al., 2008). Two of the interventions reported statistically significant changes in illness cognition domains and corresponding improvements diabetes self-care and/or HbA1c (Davies et al., 2008; Keogh et al., 2011). One intervention reported increased consequences beliefs in the intervention groups relative to the control group (French et al., 2008) but improvements in diabetes self-care were observed in the control group only. These improvements were not mediated by consequences beliefs. The final intervention reported no differences between the intervention and control group for illness cognition domains and clinical outcomes (George et al., 2008).

Intervention studies based on the CS-SRM which have aimed to improve physical health by targeting illness cognitions have shown promise in relation to improving behavioural adherence and health outcomes.

Fewer interventions based on the CS-SRM have addressed poor emotional health in the context of chronic illness. Cameron et al.(2007) used the CS-SRM as a framework to design an intervention to improve emotion regulation strategies among patients with recently diagnosed breast cancer. The intervention targeted patient's emotional arousal, expression of emotion, and cognitive appraisal of events. The intervention aimed to encourage benefit finding by altering participant perceptions of personal control and susceptibility to cancer progression.

Four months after the intervention participants in the intervention group (n=54) compared with participants in the usual care (n=44) or declined participation groups (n=56) had increased use of relaxation techniques, higher perceptions of personal control and coping efficacy beliefs and lower perceptions of susceptibility to subsequent cancer risks. The

intervention group also had lower levels of cancer worry, state anxiety, and increased emotional well-being compared with the usual care and declined participation groups.

At 12 months follow-up the intervention group also showed decreased use of emotional suppression compared with the other two comparator groups. Differences between the groups were not sustained at 6 months for risk perceptions, cancer worry, and coping efficacy. Cameron et al's (2007) findings emphasise the potential for interventions to address emotional responses to beneficially effect mood and illness cognitions.

While the CS-SRM acknowledges the importance of managing both physical and emotional health, interventions based on its principles have failed to address both. Indeed, Cameron et al's (2005) secondary analysis of Petrie et al's (2002) intervention highlighted the dangers of exclusively intervening to manage physical health among individuals high in trait negative affect. The cardiac rehabilitation benefits observed in the intervention group by Petrie et al (2002) applied only to patients low in trait negative affect. Those high in trait negative affect who received the intervention reported increased disability scores at three months follow-up and decreased dietary and exercise adherence behaviours at six months follow-up, compared with participants high in trait negative affect who had received usual care.

Consistent with the theoretical claims of the CS-SRM, Cameron et al. (2005) attribute these findings to the problem focussed nature of the intervention disrupting effective emotional health management in those participants with high trait negative affect. In contrast participants high in trait negative affect in the control group had the opportunity to attend to their emotional needs and prioritise accordingly. In addition, Cameron et al. (2005) suggest that the reassurance provided as part of challenging participant's illness representations may have led patients high in negative affect to feel as though their emotional response was an unnecessary reaction and caused emotional suppression. Finally, Cameron et al (2005) suggest that the increased attentional focus that occurs in anxious and depressed states may have resulted in patients high in negative affect detecting more somatic symptoms, which may have damaged their perceptions of recovery. Johnson and Leventhal (1974) demonstrated that patients who were encouraged to objectively appraise the somatic sensations of an uncomfortable procedure as being typical of that procedure rather than appraising it as a somatic cue to be feared were able to perform the

medical procedure (e.g. management of the health threat) with fewer interruptions, because their emotional response was being effectively managed.

While the CS-SRM recognises the potential for bi-directional relationships to occur between illness cognitions and emotional responses, studies based on the theoretical framework have largely ignored the importance of emotional responses in a person's management of their physical health. Because empirical studies have shown the importance of both illness cognitions and poor emotional health as predictors of diabetes self-management behaviours (when tested in independent statistical models) their combined effect on diabetes self-care behaviours needs exploring. This will help to identify clearer targets for therapeutic intervention.

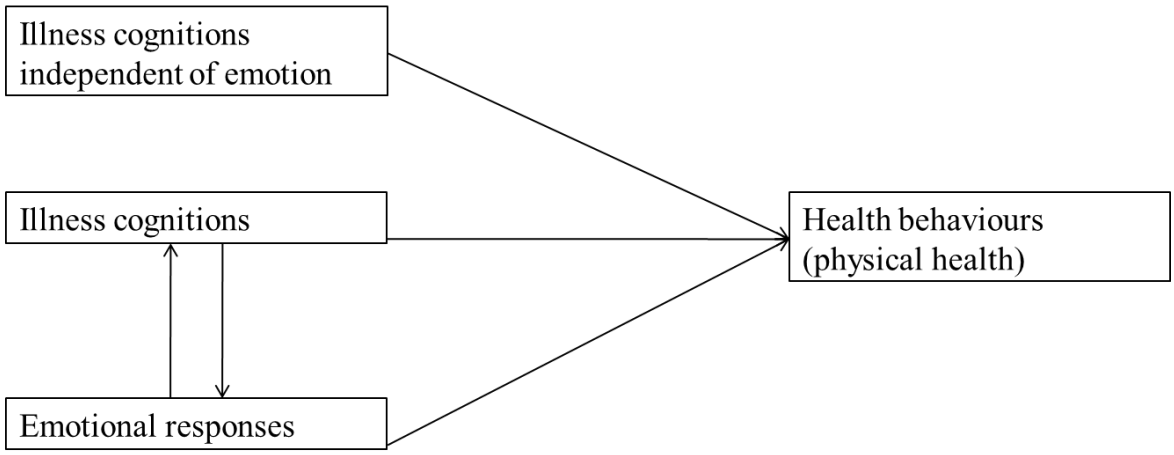
A number of questions remain unanswered when considering the types of relationships that exist between illness cognitions and poor emotional health and their relevance to self-care in the context of diabetes mellitus. These include:

i. What illness cognition domains are associated with poor emotional health in diabetes mellitus?

ii. Does a bi-directional relationship exist between diabetes cognitions and poor emotional health? If so, does one causal pathway have directional dominance and thus should become the focus of clinical intervention?

iii. How do diabetes cognitions and negative emotional responses interact to determine adherence to self-care behaviours? More specifically (See Figure 1.2):
    a. Is there evidence of cognitions mediating the effect of emotions on self-care?
    b. Do negative emotional responses mediate cognitions effect on self-care?
    c. Do some illness cognitions remain completely independent from negative emotional responses?

Figure 1.2: Hypothesised pathways of the causal relationship between illness cognitions, emotional health, and self-care behaviours



1.17 *Chapter summary*

- There is an increased prevalence of poor emotional health among individuals with diabetes relative to the general population.
- The presence of poor emotional health in diabetes is associated with poor health outcomes (elevated HbA1c, increased number/severity of diabetes complications).
- The limited numbers of longitudinal studies allude to a bi-directional relationship between diabetes onset/exacerbation and poor emotional health onset/exacerbation.
- Diabetes mellitus and poor emotional health are hypothesised to interact via both biological and behavioural pathways.
- Interventions which have addressed either condition alone have been effective in improving the targeted condition but a corresponding improvement in either diabetes mellitus or poor emotional health has not been observed.
- Findings from intervention studies suggest that the effective management of one condition may be counterproductive to the effective management of the other.

- The CS-SRM's acknowledgement of partially independent processing systems for the regulation of physical and emotional health provides an appropriate framework to explore how these management processes interact.
- The model proposes that poor emotional health can disrupt the management of physical health and vice versa via: i) altered attention and information processing strategies and ii) physical and/or behavioural resource depletion.
- Intervention studies based on the CS-SRM targeting illness cognitions have shown positive effects on health behaviours and/or health outcomes.
- Users of the CS-SRM have largely ignored the role of poor emotional health in the management of physical health.
- The pathways through which poor emotional health operates to negatively impact on the management of physical health needs further examination.

2 *Systematic review with meta-analysis*

2.1 *Introduction*

It is unclear how poor emotional health negatively impacts on diabetes health outcomes. The Common Sense Self-Regulation Model (CS-SRM) recognises that poor emotional health can affect a person's ability to behaviourally manage their physical health. One hypothesis provided by the CS-SRM for why this occurs is because of the relationships that exist between illness cognitions and poor emotional health. To elaborate a person's cynical appraisal of diabetes can provoke a negative emotional response. Once negative emotions are activated they can cause a person to elaborate further on their pessimistic diabetes illness representation. A person becomes locked into a cycle of negative appraisals driving negative emotions and vice versa, thus thwarting their efforts at engaging in problem focussed health behaviours.

2.1.1 *Review Aims/Objectives*

This chapter aims to systematically explore the nature of the association between diabetes cognitions and poor emotional health among adults with diabetes and their combined effect on adherence to diabetes self-care behaviours. This systematic review had three objectives:

i. To explore which diabetes cognitions are associated with poor emotional health.
ii. To establish whether intervention studies which targeted diabetes cognitions impacted on poor emotional health *and* vice versa.
iii. To examine the relative effects of diabetes cognitions and poor emotional health on diabetes self-care behaviours.

2.2 *Methodological considerations*

A systematic methodological approach to reviewing the literature was chosen to provide an unbiased and thorough account of the available literature (Centre for Reviews and Dissemination, 2009; Higgins and Green, 2008).

The first objective of the review was evaluated using meta-analytic techniques. This approach was adopted because of its ability to statistically combine correlation coefficients across included studies into a single summary statistic whilst evaluating its statistical significance.

The second review objective was narratively reviewed. The focus of this objective was not on establishing whether the intervention as a whole was effective in changing diabetes cognitions *or* poor emotional health but to identify the process through which this may have occurred (e.g. which diabetes cognitions changed and whether a corresponding change in emotional health was observed). A narrative analysis of these findings allows a thorough examination of plausible mechanisms of action. Popay (2004) recommends a narrative synthesis as a viable alternative to meta-analysis when the focus of the review is not effectiveness.

Similarly, the third objective of the review was evaluated using narrative synthesis. This approach was adopted because of i) multivariate relationships being evaluated (e.g. the relative effects of cognitions and poor emotional health on diabetes self-care behaviours) and ii) the diverse methods used to measure adherence to diabetes self-care behaviours (e.g. questionnaires, self-report diaries, pharmacy claims data). Such findings cannot be aggregated using conventional meta-analytic techniques.

2.3 *Methods*

2.3.1 *Eligibility Criteria*

Studies were included in the review if they:

i. Included individuals over the age of 18 with Type 1 or Type 2 diabetes mellitus defined by either self-report or clinical diagnosis. Studies including a subset of the relevant population were included if at least 80% of the sample had diabetes and reported findings in relation to diabetes outcomes separately.

ii. Used quantitative measures of illness cognition domains conceptually derived from the CS-SRM including the:

-Illness Perception Questionnaire (IPQ; Weinman, et al., 1996)

-Revised Illness Perception Questionnaire (IPQ-R; Moss-Morris et al., 2002)

-Personal Models of Diabetes Interview (PMDI; Hampson et al., 1990; 1995)

-Brief Illness Perception Questionnaire (BIPQ; Broadbent, et al., 2006)

-Diabetes Illness Representation Questionnaire (DIRQ; Skinner, et al., 2003)

Measures of medication beliefs were also included (e.g. Beliefs about Medicines Questionnaire, BMQ; Horne et al., 1999). Studies may have used either the full scale or specific subscales.

iii. Used a quantitative measure of emotional states: specifically depression, anxiety or negative well-being.

Self-report measures had to include items which measured symptoms outlined in either the:

a) Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 2000) criteria for major depressive disorder (e.g. loss of interest or pleasure, feelings of worthlessness), anxiety disorders (e.g. apprehension, tension) or mixed anxiety and depressive disorder (e.g. a combination of symptoms associated with both depression and anxiety).

OR

b) International Classification for Disease (ICD-10; World Health Organization, 2010) criteria for depressive episodes, anxiety disorders or mixed anxiety and depressive disorder.

Measures of psychological quality of life and diabetes specific distress were excluded from the review because they measure concepts relating to mental health needs and functioning (Bigelow, et al., 1991) which do not adhere to clinically endorsed symptom clusters.

iv. Examined the association between diabetes cognitions and emotional state(s) either cross-sectionally or longitudinally

OR

Allowed an assessment of whether changes in diabetes cognitions as a result of intervention were associated with a change in poor emotional health and vice versa.

v. Studies which examined the simultaneous (multivariate) effects of cognitions and poor emotional on diabetes self-care were included if they initially explored the associations between cognitions and emotions and had a quantitative measure of health behaviour.

   Health behaviours were defined as any behaviour which serves to promote, protect or redeem health (Steptoe, et al., 2010) in relation to the management of diabetes and its associated health complications. Relevant health behaviours included: medication adherence, self-monitoring of blood glucose, diet, exercise, abstinence from smoking, foot care, and appointment attendance. These health behaviours are in accordance with NICE (2004; 2008) guidelines for the effective management of diabetes.

vi. Were published in English, in peer reviewed journals.

Reviews or editorials were excluded from this review.

2.3.2 *Search Strategy*

Medline, Psycinfo, EMBASE, and CINAHL were searched from inception to 30th June 2010 with updated searches performed on the 8th February 2012 and 10th June 2013. A search strategy was developed combining MeSH terms and keyword searches relevant to: diabetes (e.g. 'diabetes mellitus', 'diabet$'), illness cognitions (e.g. 'illness cognition$', 'illness perception$'), and emotional states (e.g. 'depression', 'anxiety'). Please see methodological Appendix A for the search strategy. Included studies were citation and bibliography searched.

2.3.3 *Study Selection*

Titles and abstracts were screened against the inclusion criteria. After the initial screening phase, full texts were accessed and assessed for eligibility. Queries about the inclusion of specific studies were resolved among the supervisory team.

2.3.4 *Data extraction*

An electronic data extraction form was developed and piloted on four studies and modified in light of that experience. Data were extracted for: participant socio-demographic and clinical characteristics, study design, diabetes cognition, emotional state, and health behaviour measurement scales.

Depending on the review objective being addressed one or more of the following outcomes were extracted from each study:

i) Measures of association between cognitions and poor emotional health (bivariate and/or multivariate) with poor emotional health defined as three separate constructs: depression, anxiety, and negative well-being.
ii) Outcome scores for both cognitions and poor emotional health across intervention and control groups for studies of randomised controlled trials.
iii) Illness cognitions and emotional states relative contribution to health behaviours.

Where applicable factors controlled in multivariate analyses were extracted.

A reliability check on the data extraction process was performed by a blinded independent researcher (DK, PhD student) on 4 randomly selected studies. This revealed a 99% agreement rate on the 78 data points checked.

2.3.5 *Assessment of within study risk of bias*

Risk of bias was assessed using the Effective Public Health Practice Project Quality Assessment Tool (2009; 2010; Thomas, et al., 2004). This assessment tool provides explicit criteria to rate studies as strong, moderate or weak across six domains including: selection bias, study design, confounding variables, blinding, data collection methods, and degree of withdrawal/dropouts. Ratings are based on published information only. The rating given to each specific criterion was used to generate a global quality score for each study according to the assessment tool guidelines. A study was rated strong if it received no weak ratings, moderate if it received one weak rating, and weak if it received two or more weak ratings.

The quality appraisal tool was applied to studies meeting the inclusion criteria for each review objective separately. The study design quality criterion was evaluated in relation to types of analyses performed. For example if baseline cross-sectional analyses were extracted as part of a broader randomised controlled trial then the quality criteria was applied to the analytical design used as opposed to the broader study design. In addition for review objective one, the quality criteria confounders and blinding were not applied. It was inappropriate to apply the confounding criterion as meta-analysed findings were performed on the least statistically controlled analyses to allow greater comparisons across studies. This would have resulted in studies being unfairly weighted. Similarly the application of the blinding criterion was not appropriate because measures of association between cognitions and emotional states were largely based on observational study designs. The blinding criterion was not applied to review objective three also.

JH appraised risk of bias and any discrepancies were discussed by the supervisory team.

2.3.6 *Methods of analysis*

Where necessary illness cognition subscales had their content mapped onto the following conceptually distinct domains: identity, timeline acute/chronic, timeline cyclical, cause, consequences, personal control, treatment control, seriousness, illness coherence, concerns and necessity beliefs about medicines. The seriousness subscale from the personal models of diabetes interview (PMDI) was maintained as a standalone construct because Hampson et al. (1990) identified a high degree of multicollinearity between the consequences and timeline acute/chronic subscales of the PMDI, and therefore combined them into a single seriousness subscale.

Clark and Watson's (1991) tripartite model was used to conceptually distinguish between the emotional states depression, anxiety, and negative well-being. The tripartite model acknowledges the unique and shared features of depression and anxiety with the generation of the following three dimensions:

i. A negative affect/emotional distress dimension which represents the overlapping features of depression and anxiety. This conceptualisation was applied to categorise measures of negative well-being.
ii. A lack of positive affect dimension which represents the unique features of depression. This definition was applied to classify measures of depression.

iii. A physiological hyper-arousal dimension which represents the defining features of anxiety. This conceptualisation was used to group measures of anxiety. Anxiety measures could also include items associated with worry.

Meta-analysis

The first review objective was evaluated using meta-analytic techniques. Findings were meta-analysed (where applicable) to generate average weighted effect size r correlation coefficients for each cognition domains associative relationship with either: depression, anxiety or negative well-being. Summaries are provided as forest plots.

Each correlation coefficient was weighted by its sample size using Fisher's z statistic. This allowed an unbiased estimate of standard error which can occur in the context of correlations (Lipsey and Wilson, 2001). Findings were meta-analysed using Dersimonian and Laird's random effects model (Egger, et al., 2008) once Fisher's z statistic was back transformed to generate a mean effect size r. A random effects model was used based on the assumption that effect sizes varied between study populations. Heterogeneity was explored using Cochrane's Q statistic (Higgins and Green, 2008). The Q statistic provides a statistical test of whether observed differences across studies are greater than would be expected to occur by chance alone and are therefore not measuring the same concept (Higgins and Green, 2011). Factors that may contribute to heterogeneity may include: the population studied, measures used, and risk of bias. Publication bias was measured using Rosenthal's (1979) fail safe N (an estimate of the number of non-statistically significant studies required for a meta-analysed finding to become non-significant).

Meta-analysis was performed on the least statistically controlled statistic (e.g. bivariate correlation coefficients). If a study reported only multivariate standardised beta coefficients and no response from the author for additional information was gained the following transformation was applied to convert findings into usable r coefficients (Peterson and Brown, 2005):

$$r=\beta+0.05\lambda$$

whereby λ is equal to one when the beta coefficient is positive and zero when the beta coefficient is negative. This transformation can only be applied if the beta coefficient is in the range of ± 0.50

In addition if studies which addressed the first review objective also reported multivariate findings these findings were narratively reviewed. If studies reported the associations between cognitions and emotions at multiple time points, findings were grouped according to measurement timings (e.g. baseline, 3 months).

Narrative analysis

The second and third review objectives were explored using narrative analysis. For the second review objective findings were examined to see whether statistically significant changes in illness cognitions were associated with statistically significant changes in emotional states, and vice versa where applicable. Objective three analysed the independent contribution of cognitions and poor emotional health to health behaviours by interpreting the standardised beta coefficients or partial r and statistical significance of the coefficients. The effects of within study bias on the findings of the review were explored by grouping the findings of the included studies according to their risk of bias rating: strong, moderate or weak. These effects were statistically explored post-hoc where necessary.

2.4 *Results*

2.4.1 *Characteristics of included studies*

Twelve independent studies met the inclusion criteria for the review (See Figure 2.1 for a flow diagram of included studies; Davies, et al., 2008; Dimitraki and Karademas, 2013; Farmer, et al., 2009; French, et al., 2008; Hampson, et al., 2000; Keogh, et al., 2011; Khunti, et al., 2008; Khunti, et al., 2012; Luyckx, et al., 2010; Nsereko, et al., 2013; Paschalides, et al., 2004; Ponzo, et al., 2006; Rees, et al., 2012; Skinner, et al., 2011; Vileikyte, et al., 2005; Welschen, et al., 2012).

Ten of the twelve studies addressed review objective one (Dimitraki and Karadema, 2013; French et al., 2008; Hampson et al., 2000; Khunti et al., 2008; Luyckx et al., 2010; Nsereko et al., 2013; Paschalides et al., 2004; Ponzo et al., 2006; Rees et al., 2012; Skinner et al., 2011; Vileikyte et al., 2005). Four studies addressed review objective two (Davies et al., 2008; Farmer et al., 2009; French et al, 2008; Keogh et al., 2011; Khunti et al., 2012; Welschen et al., 2012), and two studies met the inclusion criteria for review objective three (Hampson et al., 2000; Nsereko et al., 2013).

The characteristics of included studies are summarised in Table 2.1. Across the twelve studies sample sizes ranged from 50 to 824 (median n=189) with a total sample size of n=3263. The mean age range across the twelve studies was 24 to 68 years (median=61 years), with all studies including a mix of male and female participants of largely even proportions.

Eight studies included individuals with Type 2 diabetes (Davies et al., 2008; Dimitraki and Karadema, 2013; Farmer et al., 2009; French et al., 2008; Khunti et al., 2008; 2012; Keogh et al., 2011; Nsereko et al., 2013; Paschalides et al., 2004; Ponzo et al., 2006; Skinner et al., 2011; Welschen et al., 2012), one with Type 1 diabetes (Luyckx et al., 2010), and three with a combined sample of Type 1 and Type 2 diabetes (Hampson et al., 2000; Rees et al., 2012; Vileikyte et al., 2005). Three studies recruited participants from primary care (Davies et al., 2008; Farmer et al., 2009; French et al., 2008; Khunti et al., 2008; 2012 Skinner et al., 2011; Welschen et al., 2012), seven from outpatient clinics (Dimitraki and Karadema, 2013; Hampson et al., 2000; Keogh et al., 2011; Nsereko et al., 2013; Paschalides et al., 2004; Rees et al., 2012; Vileikyte et al., 2005), one study recruited from a combined primary care/community sample (Ponzo et al., 2006), and one study sampled individuals from a national diabetes register (Luyckx et al., 2010).

Across all twelve studies diabetes cognitions were measured with either the IPQ (Paschalides et al., 2004), IPQ-R (Davies et al., 2008; Dimitraki and Karadema, 2013; Farmer et al., 2009; French et al., 2008; Khunti et al., 2008; 2012; Luyckx et al., 2010; Nsereko et al., 2013; Rees et al., 2012; Skinner et al., 2011; Vileikyte et al., 2005), Brief IPQ (Keogh et al., 2011; Welschen et al., 2012), PMDI (Hampson et al., 2000; Ponzo et al., 2006), DIRQ (Davies et al., 2008; Khunti et al., 2008; 2012; Skinner al., 2011) or BMQ (Farmer et al., 2009; French et al., 2008).

Measures of poor emotional health included the well-being questionnaire in either its full (Paschalides et al., 2004) or brief version (Farmer et al., 2009; French et al., 2008; Keogh et al., 2011), the Centre for Epidemiologic Studies Depression scale (CES-D; Hampson et al., 2000; Luyckx., 2010; Nsereko et al., 2013; Ponzo et al., 2006), the Hospital Anxiety and Depression Scale (HADS; Davies et al., 2008; Dimitraki and Karadema, 2013; Khunti et al., 2008; 2012; Rees et al., 2012; Skinner et al., 2011; Vileikyte et al., 2005), and the Short Form Spielberger State Anxiety Inventory (Welschen et al., 2012).

Figure 2.1: Flow diagram of included studies

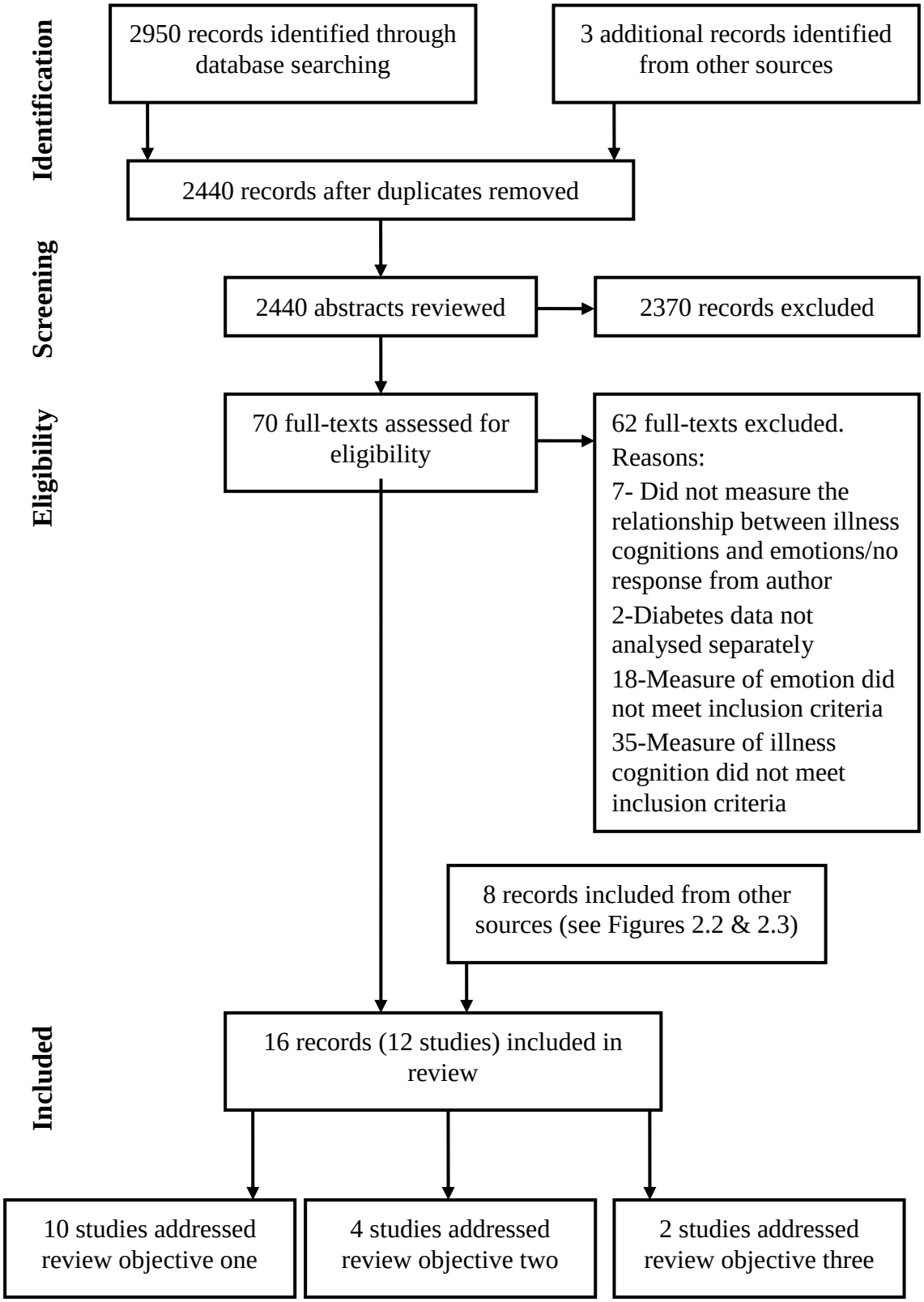


Figure 2.2: Flow diagram of included studies for updated search (8th February 2012)

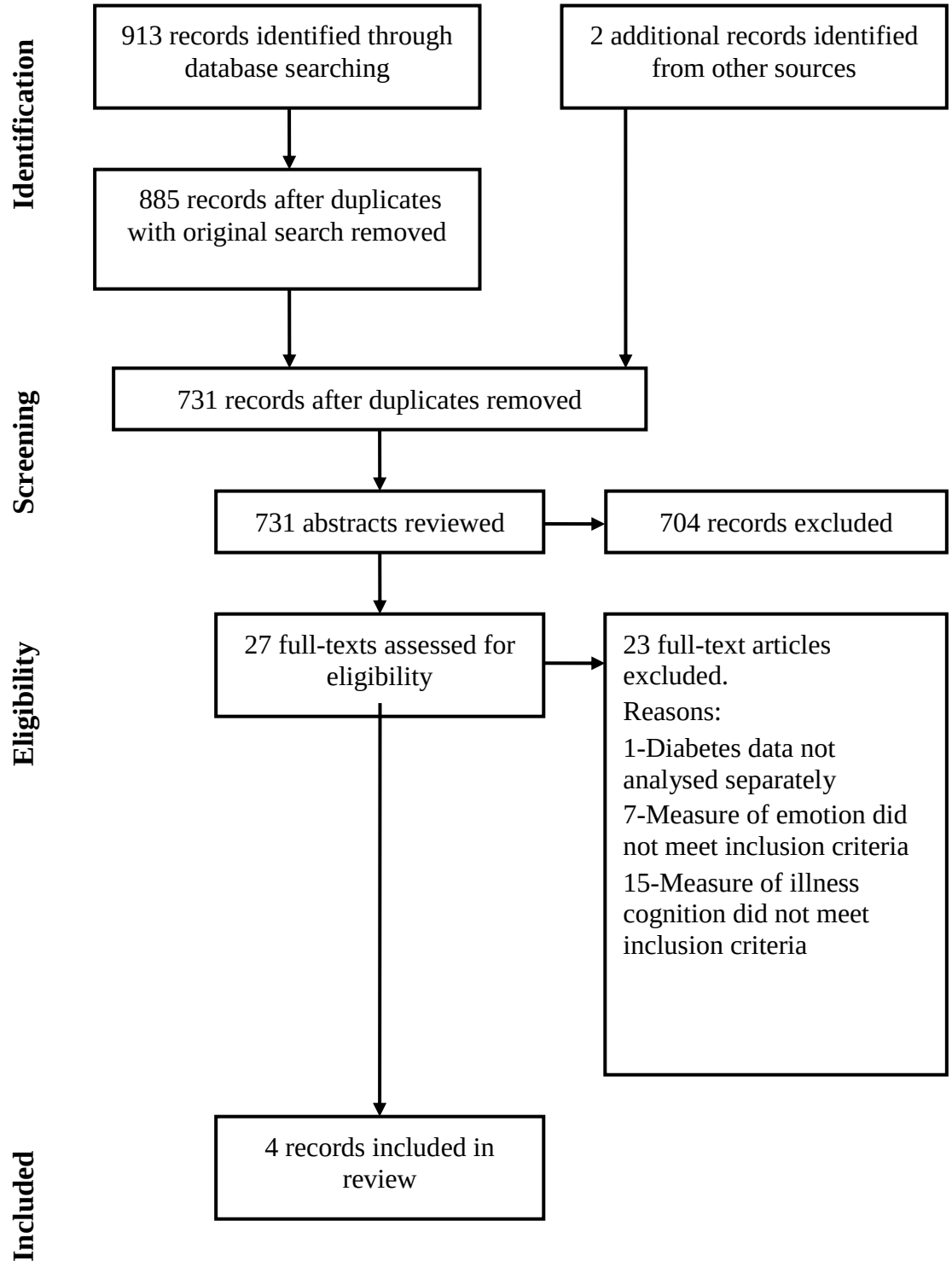


Figure 2.3: Flow diagram of included studies for updated search (10th June 2013)

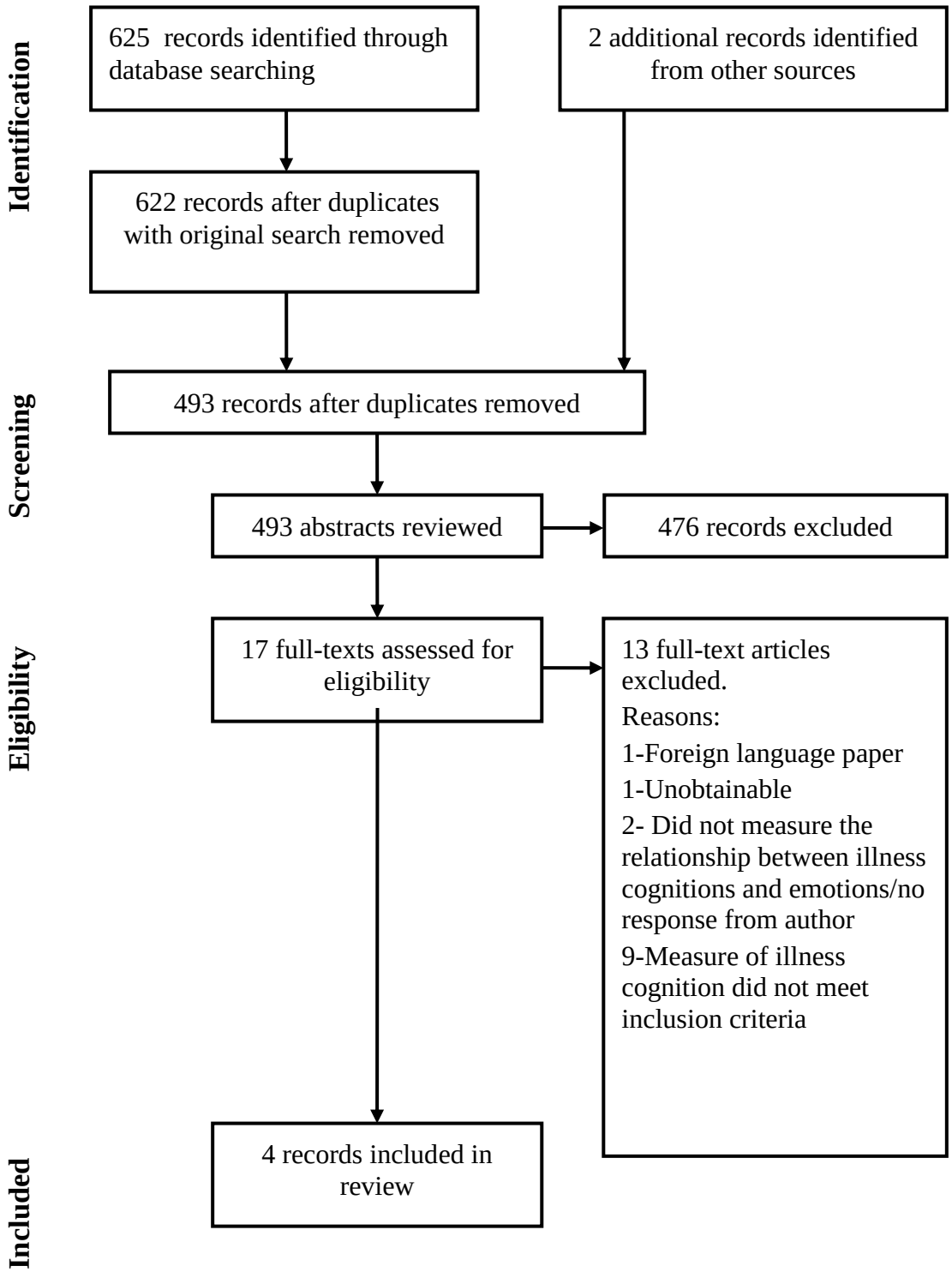


Table 2.1: Characteristics of included studies

Study/Diabetes type/Design	Mean age/ years	Baseline N (% male)	Source of sample	Measure of illness cognition (subscales)	Measure of emotion	Measure of diabetes self-care behaviours
Dimitraki et al., 2013, T2, C	65	84 (35)	Outpatient clinic, Greece	IPQ-R: Timeline acute/chronic Timeline cyclical Consequences	HADS: Depression subscale Anxiety subscale	NA
French et al., 2008; Farmer et al., 2009; DiGEM T2, RCT (Baseline, 12 months)	66	453(59)	Primary Care, England	IPQ-R: Identity Timeline acute/chronic Timeline cyclical Personal control Treatment control Illness coherence BMQ: Medication necessity Medication concerns	WBQ (12 item): Negative well-being subscale	NA
Hampson et al., 2000 T1 (20%) T2 (80%), P (Baseline, 3, 9 and 12 months)	62	111 (38)	2 Diabetologists, USA	Personal Models of Diabetes Interview: Seriousness Treatment effectiveness, (TC) Personal Control	CES-D	Kristal food habits questionnaire (high score=unhealthy eating patterns) 4 day food diary (% calories from saturated fat)

Table 2.1: continued

Study/Diabetes type/Design	Mean age/ years	Baseline N (% male)	Source of sample	Measure of illness cognition (subscales)	Measure of emotion	Measure of diabetes self-care behaviours
Keogh et al., 2011, T2, RCT	59	121(64)	Outpatient clinic, Ireland	Brief IPQ: Identity Timeline acute/chronic Consequences Personal control Treatment control Illness coherence	WBQ (12 item): Negative well-being subscale	NA
Khunti et al.,2008; 2012 Davies et al., 2008; Skinner et al., 2011, DESMOND T2, RCT	60	824 (55)	Primary Care, England & Scotland	IPQ-R: Illness coherence Timeline acute/chronic Personal control DIRQ: Seriousness Impact (CQ)	HADS: Depression subscale	NA
Luyckx et al., 2010 T1, C	24	194 (40)	Belgium Diabetes Registry, Belgium	IPQ-R: Personal Control Consequences	CES-D	NA

Table 2.1: continued

Study/Diabetes type/Design	Mean age/ years	Baseline N (% male)	Source of sample	Measure of illness cognition (subscales)	Measure of emotion	Measure of diabetes self-care behaviours
Nsereko et al., 2013, T2, C Questionnaires were translated into Kinyarwanda	52	86 (35)	Outpatient clinic, Rwanda	IPQ-R Identity Timeline acute/chronic Timeline cyclical Consequences Personal control Treatment control Illness coherence Cause (risk factors, 7 items)	CES-D	SDSCA (summed score for four subscales: diet, exercise, self-monitoring of blood glucose and foot care)
Paschalides et al., 2004 T2, C	61	184 (56)	Outpatient clinic, England	IPQ: Identity Timeline acute/chronic Consequences Personal Control	Well-being questionnaire: Depression subscale Anxiety subscale	NA
Ponzo et al., 2006, T2 C	68	50 (50)	Diabetes education centre or GP Canada (Italian ethnic group)	Personal models of diabetes questionnaire: Seriousness Treatment effectiveness (TC)	CES-D	NA

Table 2.1: Continued

Study/Diabetes type/Design	Mean age/ years	Baseline N (% male)	Source of sample	Measure of illness cognition (subscales)	Measure of emotion	Measure of diabetes self-care behaviours
Rees et al., 2012, T1(25%), T2 (75%)	60	400 (62)	Retinopathy clinics, Australia	IPQ-R: Identity Timeline acute/chronic Timeline cyclical Personal control Treatment control Illness coherence	HADS: Depression subscale Anxiety subscale	SDSCA
Vileikyte et al., 2005, T1 (28%) T2 (72%), C	62	494 (70)	Outpatient clinic, England & USA	IPQ-R: Treatment control Timeline cyclical	HADS: Depression subscale	NA

Table 2.1: Continued

Study/Diabetes type/Design	Mean age/ years	Baseline N (% male)	Source of sample	Measure of illness cognition (subscales)	Measure of emotion	Measure of diabetes self-care behaviours
Welschen et al., 2012, T2, RCT	59	262 (56)	Primary Care, Netherlands	Brief IPQ: Identity Timeline acute/chronic Consequences Personal control Treatment control Illness coherence	Short Form Spielberger State Anxiety Inventory(SF-STAI)	NA

Key: B-IPQ, Brief Illness Perception Questionnaire, BMQ, Beliefs about medicines questionnaire; CES-D, Centre for Epidemiologic Studies Depression Scale; CQ, Consequences; C, Cross-sectional; DIRQ, Diabetes Illness representations questionnaire; GP, General Practitioner, HADS, Hospital Anxiety and Depression Subscale; IPQ, Illness perception questionnaire, IPQ-R Illness perception questionnaire-revised; NA, Not Applicable; NR, Not reported; P, Prospective; PMDI, Personal models of Diabetes Interview; RCT, Randomised controlled trial; SDSCA, Summary of Diabetes Self-Care Activities Scale; T1, Type 1 diabetes; T2, Type 2 diabetes; TC, Treatment control; WBQ, Well-being questionnaire

2.4.2 *Review objective one: What is the nature of the association between diabetes cognitions and poor emotional health?*

Depression

Nine studies measured the association between diabetes cognitions and depression (Dimitraki and Karadema, 2013; Hampson et al., 2000; Khunti et al., 2008; Luyckx et al., 2010; Nsereko et al., 2013; Paschalides et al., 2004; Ponzo et al., 2006; Rees et al., 2012; Skinner et al., 2011; Vileikyte et al., 2005). Across the nine depression studies, all illness cognition domain concepts were meta-analysed excluding illness coherence[1] and risk factor causal beliefs because these concepts were measured only once across included studies. Findings of the relationship between depression and illness cognition domains are shown in Table 2.2. Depression was associated with increased perceived timeline cyclical ($r=0.25$, $p\leq0.01$), consequences ($r=0.41$; $p\leq0.01$), and seriousness beliefs ($r=0.38$; $p\leq0.01$) and lower perceived personal control ($r=-0.27$; $p\leq0.01$), and treatment control ($r=-0.10$; $p-\leq0.01$). The meta-analysed findings for identity and timeline acute/chronic beliefs were statistically non-significant. Forest plots for the meta-analysed findings can be found in Statistical Appendix A.

For the meta-analysed findings the Cochrane Q statistic indicated that the meta-analysed studies were from a homogenous population (e.g. the Q statistic was non-significant; See Table 2.2). Tests of publication bias demonstrated that a relatively large number of null findings would be required to make the statistically significant meta-analysed findings non-significant. All cognition domains had fail safe N (FSN) statistics ranging from 36 to 273. However for the treatment control subscale, only an additional two non-statistically significant studies would be required for the meta-analysed findings to become statistically non-significant.

Cross-sectional correlations between depression and the cognition domains illness coherence and risk factor causal attributions were reported in a single study by Nsereko et al. (2013). Both of these cognition domains had a statistically non-significant relationship with depression. No included studies explored the relationship between depression and the cognition domains: medication necessity and medication concerns.

[1] Khunti et al. (2008) measured the cross-sectional association between depression and illness coherence but the direction of effect reported in the text differed from that presented for the correlation coefficient and was therefore excluded from analyses.

Skinner et al., (2011) reported follow-up analyses on the cross-sectional data presented by Khunti et al., (2008). Cognition domains at four months were entered into a multivariate regression model as predictors of depression at 12 month follow-up. Four month depression scores, gender, age, and ethnicity were also entered as covariates into the regression model. Increased perceived consequences at four months predicted increased depression scores at 12 months follow-up (β=0.17, $p \leq 0.01$; n=374). Similarly increased illness coherence scores at four months predicted increased depression scores at 12 months follow-up (β=0.11, $p \leq 0.01$; n=374). No statistically significant effects were found for the cognition domains: timeline acute/chronic, personal control, and seriousness beliefs (See Table 2.3). .

Rees et al., (2012) tested the relative effects of cognitions on depression using multivariate quantile regression and thus were ineligible for meta-analysis. The cross-sectional relationships between depression and illness cognitions were explored in two subgroups: those with and without the diabetes complication retinopathy. Consistent with the meta-analysed findings for both subgroups a positive relationship was found between depression and the cognition domains: consequences and timeline cyclical. In contrast with the synthesised findings no relationships were found between depression and control beliefs (personal and treatment) in both subgroups. Consistent with Nsereko et al. (2013) no statistically significant relationships were found in both subgroups between depression and illness coherence. A statistically significant positive relationship was found between depression and identity beliefs in those without retinopathy, but this relationship was not present in participants with retinopathy. In contrast, among participants with retinopathy a statistically significant positive association was reported between timeline acute/chronic beliefs and depression. This effect was not found among participants without retinopathy (See Table 2.3).

Anxiety

Only two of the twelve identified studies performed correlations to explore the relationship between anxiety and illness cognition domains (Dimitraki and Karadema, 2013; Paschalides et al., 2004). Two illness cognition domains were measured across both studies and were meta-analysed (See Table 2.2). Meta-analysed findings showed a positive association between anxiety and timeline acute/chronic beliefs (r=0.20, $p \leq 0.01$) and consequences beliefs (r=0.44, $p \leq 0.01$). Forest plots of meta-analysed findings can be found in Statistical Appendix A. Dimitraki and Karadema (2013) also measured the association

between anxiety and timeline cyclical beliefs and reported a statistically significant positive association (r=0.31, p≤0.01). Equally, Paschalides et al. (2004) tested the relationship between anxiety and the cognition domains identity and personal control and found a statistically significant positive association for identity beliefs (r=0.51, p≤0.01) and a negative association for personal control (r=-0.20, p≤0.01) beliefs. No studies measured the relationship between anxiety and the cognition domains: causal attributions, treatment control, illness coherence, seriousness, medication necessity, and medication concerns.

Rees et al., (2012) performed two cross-sectional multivariate quantile regression analyses to explore the relative effects of diabetes cognitions as predictors of anxiety for individuals with and without diabetic retinopathy. Consistent with Paschalides et al. (2004) increased identity beliefs were associated with increased feelings of anxiety among participants with and without diabetic retinopathy. In agreement with above bivariate findings increased perceived consequences and timeline cyclical beliefs were associated with increased anxiety scores but only among participants with diabetic retinopathy. In addition participants with retinopathy showed statistically significant relationships between anxiety and illness coherence. A lower perceived illness coherence belief was associated with higher levels of anxiety. In contrast to the bivariate findings, Rees et al (2012) found no statistically significant relationships between anxiety, timeline acute/chronic, personal control, and treatment control beliefs in both subgroups (See Table 2.3).

Negative well-being

Only one study was identified which measured the relationship between cognitions and negative well-being (French et al., 2008). This study demonstrated that increased negative well-being scores were associated with greater identity (r=0.31, p≤0.01), timeline cyclical (r=0.29, p≤0.01), consequences (r=0.31, p≤0.01), and medication concern beliefs (r=0.21, p≤0.05) and lower perceptions of illness coherence (r=-0.13, p≤0.05). No statistically significant relationships were observed between negative well-being and timeline acute/chronic (r=0.00, p≥0.05), personal control (r=-0.02, p≥0.05), treatment control (r=-0.02, p≥05), and medication necessity beliefs (r=0.09, p≥0.05).

Risk of within study bias (review objective one)

Assessments of within study bias for review objective one are shown in Table 2.4. The quality criteria confounders and blinding were not applicable for the reasons described in Section 2.3.5. All studies received uniform ratings across the following quality criteria:

study design, data collection, and withdrawals and dropouts. Thus selection bias was the only quality criterion to demonstrate a sufficient amount of variance to distinguish between the studies. This criterion was adopted post-hoc as the global quality rating to rate studies as methodologically strong, moderate or weak. Using this approach eight studies received a moderate rating (Dimitraki and Karadema, 2013; Hampson et al., 2000; Khunti et al., 2008; Nsereko et al., 2013; Paschalides et al., 2004; Ponzo et al., 2006; Rees et al., 2012; Skinner et al., 2011; Vileikyte et al., 2005) and two studies received a weak rating (French et al., 2008; Luyckx et al., 2010).

Post-hoc sensitivity analyses: risk of bias across studies

Meta-analysed findings were repeated for depression and the cognition domains consequences and personal control by removing from the analyses studies that received a weak rating. No subgroup analyses were performed for the remaining meta-analysed cognition domains because all studies received a moderate quality rating. The relationship between consequences and depression for the moderately rated studies only (n=3) was r=0.42, p≤0.01 (95% CI 0.37 to 0.47) compared with r=0.32, p≤0.01 for the one study that received a weak rating. Thus the effect size was larger for studies that received a moderate quality rating.

Similarly the relationship between personal control and depression for the studies that received a moderate rating (n=4) was r=-0.29, p≤.01 (95% CI -0.39 to -0.19) compared with r=-0.18, p≤0.05, for the one study that received a weak rating. Thus, the effect size was larger for the studies that received a moderate quality rating.

Table 2.2: Association between cognitions and emotions from meta-analysed and individual studies

Illness cognition domain	Sample size	N included studies	mean r/individual study correlation coefficient	P	95% Confidence intervals Lower	Upper	Q statistic χ^2	DF	P	FSN
Meta-analysed findings										
Depression										
Identity	270	2	.22	>0.05	-.35	.67	1.00	1	.32	NA
Timeline acute/chronic	354	3	.06	>0.05	-.12	.23	2.31	2	.32	NA
Timeline cyclical	654	3	.25	≤0.01	.14	.35	2.09	2	.35	36
Consequences	1124	4	.41	≤0.01	.36	.45	2.61	3	.46	273
Personal control	1337	5	-.27	≤0.01	-.35	-.18	4.64	4	.33	146
Treatment control	731	4	-.10	≤0.01	-.17	-.02	1.18	3	.76	2
Seriousness	923	3	.38	≤0.01	.08	.61	2.73	2	.26	61
Findings from individual studies										
Depression										
Illness coherence	86	1	-.20	>0.05						
Cause (risk factors)	86	1	-.11	>0.05						
Meta-analysed findings										
Anxiety										
Timeline acute/chronic	268	2	.20	≤0.01	.08	.31	0.15	1	.70	6
Consequences	268	2	.44	≤0.01	.33	.53	0.01	1	.93	39

Table 2.2: Continued

Illness cognition domain	Sample size	N included studies	mean r/individual study correlation coefficient	P	95% Confidence intervals Lower	Upper	Q statistic χ^2	DF	P	FSN
Findings from individual studies										
Anxiety										
Identity	184	1	.51	≤0.01						
Timeline cyclical	84	1	.31	≤0.01						
Personal control	184	1	-.20	≤0.01						
Findings from individual studies										
Negative well-being										
Identity	394	1	.31	≤0.01						
Timeline acute/chronic	381	1	.00	>0.05						
Timeline cyclical	381	1	.29	≤0.01						
Consequences	378	1	.31	≤0.01						
Personal control	382	1	-.02	>0.05						
Treatment control	381	1	-.02	>0.05						
Illness coherence	377	1	-.13	≤0.05						
Medication necessity	97	1	.09	>0.05						
Medication concerns	92	1	.21	≤0.05						

Key: DF, Degrees of Freedom, N, Number; FSN, Fail-Safe N

Table 2.3: Multivariate associations between cognitions and emotions ineligible for meta-analysis

Study	Variables statistically controlled for	Relative effect of cognitions on depression	Relative effect of cognitions on anxiety
Rees et al., 2012	Quantile regression Simultaneous entry of: age, gender, diabetes duration, HbA1c, diabetes complications other than retinopathy	DV: Depression (retinopathy absent) Identity: median difference=0.52, p≤.01 Timeline acute/chronic: median difference=-0.14, p>.05 Timeline cyclical: median difference=0.29, p≤.01 Consequences: median difference= 0.18, p≤.01 Personal control: median difference=-0.21, p>.05 Treatment control: median difference=-0.20, p>.05 Illness coherence: median difference=-0.12, p>.05 DV: Depression (retinopathy present) Identity: median difference=0.34, p>.05 Timeline acute/chronic: median difference=0.14, p≤.05 Timeline cyclical: median difference=0.27, p≤.01 Consequences: median difference= 0.24, p≤.01 Personal control: median difference=-0.05, p>.05 Treatment control: median difference=-0.19, p>.05 Illness coherence: median difference=-0.19, p>.05	DV: Anxiety (retinopathy absent) Identity: median difference=0.74, p≤.01 Timeline acute/chronic: median difference=-0.04,p>.05 Timeline cyclical: median difference=0.34, p>.05 Consequences: median difference= 0.19, p>.05 Personal control: median difference=-0.37, p>.05 Treatment control: median difference=-0.05, p>.05 Illness coherence: median difference=-0.23, p>.05 DV: Anxiety (retinopathy present) Identity: median difference=0.71, p≤.01 Timeline acute/chronic: median difference=0.19, p>.05 Timeline cyclical: median difference=0.44, p≤.01 Consequences: median difference= 0.42, p≤.01 Personal control: median difference=-0.03, p>.05 Treatment control: median difference=-0.18, p>.05 Illness coherence: median difference=-0.31, p≤.01

Table 2.3: Continued

Study	Variables statistically controlled for	Relative effect of cognitions on depression	Relative effect of cognitions on anxiety
Skinner et al, 2011 (follow-up from Khunti et al., 2008; DESMOND)	Hierarchical regression S1: Gender, age, ethnicity, depression scores at four months S2: Illness cognitions	DV: Depression scores (12 months follow-up), N=374 Timeline acute/chronic: β=NR, p>.05 Consequences: β=.17, p≤.01 Personal control: β=NR, p>.05 Illness coherence: β=.11, p≤.01 Seriousness: β=NR, p>.05	Not applicable

Key: DV, Dependent variable; N, Number; NR, Not reported; NS, Non-significant; S, Step, TC, Treatment control

Table 2.4: Risk of bias assessment review objective one

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
Dimitraki et al., 2013	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Recruitment method other than random sampling/self-referral. Response rate: 68%	Cross-Sectional			Valid and reliable	Cross-sectional
French et al., 2008	Weak	Weak	NA	NA	Strong	Moderate
Weak	Sequential sampling. Response rate: 32% from initial sampling frame.	Cross-sectional			Valid and reliable	Cross-sectional
Hampson et al., 2000	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Recruitment method other than random sampling/self-referral. Response rate: not reported.	Cross-sectional			Valid and reliable.	Cross-sectional

Table 2.4: Continued

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
Khunti et al., 2008; Skinner et al., 2011	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Consecutive sampling at practice and patient level. Patient response rate=74%	Cross-sectional			Valid and reliable	Cross-sectional
Nsereko et al., 2013	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Recruitment method other than random sampling/self-referral. Response rate: Not reported.	Cross-sectional			Valid and reliable.	Cross-sectional
Luyckx et al., 2010	Weak	Weak	NA	NA	Strong	Moderate
Weak	Recruitment method other than random sampling/self-referral. Response rate:39%	Cross-sectional			Valid and reliable.	Cross-sectional
Paschalides *et al.*, 2004	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Consecutive sampling. Response rate: 84%	Cross-sectional			Valid and reliable.	Cross-sectional

Table 2.4: Continued

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
Ponzo et al., 2006	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Recruitment method other than random sampling/self-referral. Response rate: 96%	Cross-sectional			Valid and reliable.	Cross-sectional
Rees et al., 2012	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Recruitment method other than random sampling/self-referral. Response rate: Not reported	Cross-Sectional			Valid and reliable	Cross-sectional
Vileikyte *et al.,* 2005	Moderate	Weak	NA	NA	Strong	Moderate
Moderate	Consecutive sampling. Response rate= 95%	Cross-sectional			Valid and reliable.	Cross-sectional

2.4.3 *Review objective two: Impact of intervention studies on cognitions and poor emotional health*

Four intervention studies were identified that met the inclusion criteria (Davies et al., 2008; Farmer et al., 2009; French et al, 2008; Keogh et al., 2011; Khunti et al., 2012; Welschen et al., 2012). All four were psychological interventions which addressed beliefs about diabetes. Diabetes beliefs were either directly targeted by the intervention via thought challenge techniques (Keogh et al., 2011) or indirectly targeted via either behavioural experiments (Farmer et al., 2009; French et al., 2008) or education (Davies et al., 2008; Khunti et al., 2012; Welschen et al., 2012). No interventions were identified which addressed mood directly. Table 2.5 summarises each interventions content and findings and Table 2.6 provides a visual summary of the findings.

All interventions demonstrated statistically significant differences in diabetes cognitions for the intervention group(s) relative to the control group excluding Welschen et al., (2012) who found no differences in either cognitions (Brief IPQ subscales) or anxiety at four and twelve weeks follow-up.

Keogh et al's (2011) intervention reported statistically significant effects for five out of the seven illness cognition domains measured at six months follow-up. Specifically, compared with the control group participants in the intervention group reported lower identity perceptions and increased perceptions of personal control, treatment control, and illness coherence. Causal beliefs also differed with the intervention group reporting lower hereditary and increased lifestyle causal attributions. No statistically significant differences were observed for the domains timeline acute/chronic and consequences. With regards to changes in emotional health the intervention group reported lower levels of negative well-being at six months follow-up relative to the control group.

Similarly the DESMOND trial (Davies et al., 2008; Khunti et al., 2012) reported statistically significant results for four out of the five illness cognition domains measured at four, eight, twelve months, and three years follow-up. Compared with the control group, scores in the intervention group increased for the following cognition domains: timeline acute/chronic, personal control, illness coherence, and seriousness. No statistically significant differences were observed for the consequences domain. Decreased depression scores were observed in the intervention group compared with the control group at 12 months follow-up only.

Finally, the DiGEM trial (Farmer et al., 2009; French et al; 2008) reported a statistically significant effect for one out of the nine cognition domains measured in both the intervention groups relative to the control group. This effect was observed for the consequences subscale at 12 months follow-up, with greater perceived consequences being observed. No statistically significant differences between the intervention and control groups for the negative well-being subscale at 12 months follow-up were found.

Risk of within study bias (review objective two)

Table 2.7 presents the risk of bias assessment for each of the intervention studies. Two studies received a moderate global rating (Farmer et al., 2009; French et al., 2008; Keogh et al., 2011) whilst two studies received a strong global quality rating (Davies et al., 2008; Khunti et al., 2012; Welschen et al., 2012). Given the small number of intervention studies it was difficult to determine the impact of study quality on intervention findings.

Table 2.5: Findings from intervention studies which have targeted cognitions or poor emotional health

Study	Intervention	Intervention/ Control groups/ Baseline N	Statistical analysis	Cognition domain findings (reported for intervention group(s) relative to control	Emotion domain findings (reported for intervention group(s) relative to control
Davies et al., (2008); Khunti et al., 2012); DESMOND	6 hour group education programme within 12 weeks of diagnosis, focussed on behavioural change. Cognitions used as prompts to encourage learning. Social learning theory principles also used.	i) "Enhanced" usual care with equivalent contact time to intervention; n= 488 ii) Intervention; n=621	Generalised estimating equations (adjusted for baseline and cluster effects)	Timing of outcome measures: 4, 8, 12 months & 3 years (Findings reported for 12 months). Timeline A/C: MD=+0.69, p≤0.05 Consequences: NS Personal control: MD =+0.51, p≤.01 Illness coherence: MD =+1.22, p≤.01 Seriousness: MD=+0.85, p≤.01	Timing of outcome measures: 4, 8, 12 months (Findings reported for 12 months). Depression: MD = -0.50, p≤0.05 (statistically significant effects only observed at 12 months)

Table 2.5: Continued

Study	Intervention	Intervention/ Control groups/ Baseline N	Statistical analysis	Cognition domain findings (reported for intervention group(s) relative to control	Emotion domain findings (reported for intervention group(s) relative to control
French et al., (2008); Farmer et al., (2009); DiGEM	Use of SMBG to identify beliefs about diabetes. Elicited beliefs were used as a platform for diabetes self-care goal setting; specifically identity, personal control and treatment control. Duration: Twelve months with quarterly GP visits.	i) Usual care; n=152 (included goal setting) ii)SMBG ≤ intensive; n=150 (clinician interpretation) iii) SMBG > intensive; n=151 (patient interpretation)	ANCOVA (controlled for baseline scores) Cohen's d not adjusted for baseline scores, change scores only	Timing of outcome measures: 12 months Identity: NS Timeline acute/chronic: NS Timeline cyclical: NS Consequences: Increased for both SMBG intervention groups (p≤.01). Cohen's d: ≤ intensive =0.19; > intensive=0.36 Personal control: NS Treatment control: NS Illness coherence: NS BMQ-N: NS BMQ-C: NS	Negative well-being: NS

Table 2.5: Continued

Study	Intervention	Intervention/ Control groups/ Baseline N	Statistical analysis	Cognition domain findings (reported for intervention group(s) relative to control	Emotion domains findings (reported for intervention group(s) relative to control
Keogh et al., (2011)	3 intervention sessions with patient and family member over 3 weeks. Challenge erroneous illness cognitions & explore how cognitions determine diabetes self-care. Use of motivational interviewing.	i) Usual care; n=61 ii) Intervention; n=60	Regression analysis. Controlled for duration, medication type & baseline HbA1c .	Timing of outcomes measures: 6 months Identity: Lower, 3.48 vs 5.33, p≤0.01 Timeline acute/chronic: NS Consequences: NS Personal control: Higher, 7.02 vs 4.75, p≤0.01 Treatment control: Higher, 8.41 vs 6.64, p≤0.01 Illness coherence: Higher, 8.39 vs 6.28, p≤0.01 Hereditary causal attributions: Lower, 21.7% vs 39.3%, p≤0.01 Lifestyle causal attributions: Higher, 6.7% vs 1.6%., p≤0.01 NS differences for following causal attributions: weight, diet, exercise, physical, psychological unclear	Negative well-being: Lower; 2.48 vs 4.08, p≤0.01

Table 2.5: Continued

Study	Intervention	Intervention/ Control groups/ Baseline N	Statistical analysis	Cognition domain findings (reported for intervention group(s) relative to control	Emotion domains findings (reported for intervention group(s) relative to control
Welschen et al., 2012	Intervention delivered as part of usual care (~5mins). Educate patients about cardiovascular risks, including causes & consequences. Use of personalised risk estimates. Positive framing of messages for lifestyle change and strategies to implement change (personal control & treatment control). Response from patient and use of think aloud techniques.	i) Usual care; n=130 ii) Intervention; n=132	Linear regression. Identified differences between groups were adjusted at baseline.	Timing of outcomes measures: 2 weeks (immediately after usual care visits and 12 weeks) Identity: β=NR, NS Timeline acute/chronic: β=NR, NS Consequences: β=NR, NS Personal control: β=NR, NS Treatment control: β=NR, NS Illness coherence: β=NR, NS *Applies to both 2 & 12 week follow-up*	Short Form Spielberger State Anxiety Inventory 2 weeks: β=-0.09, p>.05 12 weeks: β=0.09, p>.05

Key: ANCOVA, Analysis of co-variance; BMQ-C, Beliefs about medicines-concerns subscale; BMQ-N, Beliefs about medicines-necessity subscale, MD, Mean difference; NR, Not reported; NS, Non-significant; SMBG, Self-monitoring of blood glucose; Timeline A/C, Timeline acute/chronic

Table 2.6: Summary of diabetes cognition and poor emotional health concepts that changed in response to intervention relative to the control group

Illness cognition domains												
Identity	Timeline acute/chronic	Timeline cyclical	Consequences	Seriousness	Personal control	Treatment control	Illness coherence	Medication necessity	Medication concerns	Hereditary (cause)	Lifestyle (cause)	Poor emotional health
Keogh et al., (2011)												
↓	NS	NA	NS	NA	↑	↑	↑	NA	NA	↓	↑	↓
Davies et al., (2008); Khunti et al., (2012); DESMOND												
NA	↑	NA	NS	↑	↑	NA	↑	NA	NA	NA	NA	↓
Farmer et al. (2009); French et al., (2008); DiGEM*												
NS	NS	NS	↑	NA	NS	NS	NS	NS	NS	NA	NA	NS
Welschen et al., (2012)												
NS	NS	NA	NS	NA	NS	NS	NS	NA	NA	NA	NA	NS

*Applies to both intervention groups (more intensive and less intensive self-monitoring of blood glucose intervention groups).Key: NA, Not applicable; NS, Non-significant, ↑, a statistically significant increase in the concept measured was observed in the intervention group relative to the control group, ↓ a statistically significant decrease in the concept measured was observed in the intervention group relative to the control group.

Table 2.7: Risk of bias assessment review objective two

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
Davies et al., 2008; Khunti et al., 2012; DESMOND	Moderate	Strong	Strong	Moderate	Strong	Moderate
Strong	See Table 2.4 (Khunti et al., 2008)	RCT; computer generated randomisation at the cluster level. Independent from research team.	Differences were reported between intervention and control groups at baseline. Statistical analyses controlled for baseline scores.	Outcome assessors not aware of intervention status however participants were.	Valid and reliable.	Response rate: 68.2%

Table 2.7: Continued

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
French et al., 2008; Farmer et al., 2009; DiGEM	Weak	Strong	Strong	Moderate	Strong	Moderate
Moderate	See Table 2.4.	RCT, process of randomisation described (minimisation procedure). Occurred independent of researchers (computer generated and allocated by study co-ordinator with no patient contact).	No reported differences between groups at baseline.	Outcome assessors not aware of intervention status however participants were.	See Table 2.4.	Response rate: 74.8%

Table 2.7: Continued

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
Keogh et al., 2011	Weak	Strong	Strong	Moderate	Strong	Moderate
Moderate						
	Recruitment method other than random sampling/self-referral. Response rate: 50%	RCT process of randomisation described as computer generated, independent from research team	No reported differences between groups at baseline.	Outcome assessors not aware of intervention status but participants were.	Valid and reliable.	Response rate: 71.0%
Welschen et al., 2012	Moderate	Strong	Strong	Moderate	Strong	Moderate
Strong						
	Recruitment method other than random sampling/self-referral. Response rate: 85%	RCT process of randomisation described as compute generated. Manager independent of the care team allocated patients to appropriate group	Identified differences between groups were adjusted at baseline.	Patients and clinicians were not blinded but principal investigator was.	Valid and reliable.	Response rate: 79%

2.4.4 *Review objective three: Relative effects of cognitions and poor emotional health on diabetes self-care behaviours*

Of the studies that measured the association between cognitions and poor emotional health, only two (Hampson et al., 2000; Nsereko et al., 2013) tested their relative effects on diabetes self-care behaviours (see Table 2.8).

Hampson et al., (2000) simultaneously entered the cognition domains (seriousness and treatment control) alongside depression (measured at nine months) into a linear regression model to predict dietary self-care behaviours at twelve months follow-up. Baseline scores on the dietary self-care behaviour scales(s), sex, age, co-morbidities, diabetes type, and intervention group were also entered as covariates.

When high fat eating patterns was the dependent variable increased perceptions of treatment control predicted improved eating patterns; no statistically significant effects were observed for seriousness beliefs or depression. When percentage of calories consumed from saturated fat was the dependent variable neither illness cognitions nor depression were statistically significant predictors.

Nsereko et al. (2013) tested two cross-sectional multivariate regression models (hierarchical and stepwise backwards elimination) to explore the relative effects of diabetes cognitions (identity, timeline acute/chronic, timeline cyclical, illness coherence and personal control) and depression on adherence to diabetes self-care behaviours (measured using a composite scale of diet, exercise, self-monitoring of blood glucose, and foot care). Age, gender and diabetes duration were also entered as covariates.

The hierarchical regression model found only depression to predict diabetes self-care behaviours: participants with higher levels of depression adhered less to their diabetes self-care routine. In contrast the backwards elimination model (which allows variables other than the most dominant predictor to remain in the model) found depression, personal control, and timeline cyclical beliefs to be statistically significant predictors of diabetes self-care. Participants, who were more depressed and perceived diabetes to be unpredictable adhered less to their treatment regimens. In contrast participants with greater believe in their personal capabilities to manage their diabetes adhered more to their recommended health behaviours.

Because depression and personal control were correlated, Nsereko et al (2013) tested whether personal control mediated the relationship between depression and diabetes self-care. No evidence was found to support this relationship (Nsereko et al., 2013; See Table 2.6).

Risk of within study bias (review objective three)

The Hampson et al. (2000) study received a weak global quality rating in contrast Nsereko et al's (2013) study received a moderate quality rating (See Table 2.9). This finding may account for study differences.

Table 2.8: Relative effects of cognitions and emotions on diabetes self-care behaviours

Study	Variables statistically controlled for	Relative effect of cognitions and emotions on diabetes self-care
Hampson et al., 2000	Hierarchical regression S1: Baseline DV S2: Sex, age, co-morbidity, diabetes type, experimental condition (entered only if correlated with DV) S3: 9 month measures of illness cognitions & depression	DV: Eating patterns (12 months; N=111) Depression: partial r=NS Seriousness: partial r=NS Treatment effectiveness (TC): partial r=-0.25, $p \leq 0.01$ DV: Calories from saturated fat (12 months; N=97) Depression: partial r=NS Seriousness: partial r=NS Treatment effectiveness (TC): partial r=NS Control: excluded from both regression models because of lack of reliability

Table 2.8: Continued

Study	Variables statistically controlled for	Relative effect of cognitions and emotions on diabetes self-care
Nsereko et al., 2013	Hierarchical and backwards elimination regression S1: Age, gender, diabetes duration. S2: Depression S3: Illness cognitions (those that correlated with health behaviours)	DV: Health behaviours (composite scale: diet, exercise, self-monitoring of blood glucose and foot care; N=86) *Hierarchical:* Depression: β= -0.48, p≤.01 Identity: β=0.13, p>.05 Timeline acute/chronic: β=0.15, p>.05 Timeline cyclical: β=-0.18, p>.05 Illness coherence: β=0.10, p>.05 Personal control: β=0.15, p>.05 *Backwards:* Depression: β= -0.48, p≤.01 Identity: β=NR, p>.05 Timeline acute/chronic: β=0.17, p>.05 Timeline cyclical: β= -0.23, p≤.05 Illness coherence: β=NR, p>.05 Personal control: β= 0.20, p≤.05 *Mediation analysis:* Depression → Personal control → Health behaviours (using Sobel's test of indirect effects) Effect size (unstandardised regression coefficient): 0.19, p>0.05

Key: DV, Dependent variable; NR, Not reported; NS, Non-significant; S, Step, TC, Treatment control

Table 2.9: Risk of bias assessment review objective three

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
Hampson et al., 2000	Moderate	Strong	Strong	NA	Weak	Weak
Weak	See Table 2.4	Study described as RCT. Process of randomisation not reported.	Controlled for ≥80% of variables reported to have an association with the dependent variable.		Not adequately described. Other reports have indicated validity and reliability of the Kristal food habits questionnaire; 4 day food record may produce non-valid findings.	Not adequately described in text.

Table 2.9: Continued

Study/Global rating	Selection bias	Study design	Confounders	Blinding	Data collection	Withdrawals and dropouts
Nsereko et al., 2013	Moderate	Weak	Strong	NA	Strong	Moderate
Moderate	See Table 2.4	Cross-sectional	Controlled for socio-demographic/ clinical characteristics		Valid and reliable.	Cross-sectional

2.5 *Discussion*

2.5.1 *Summary of evidence*

This systematic review with meta-analysis explored: i) the nature of the association between cognition domains and poor emotional health (depression, anxiety, and negative well-being), ii) whether interventions which targeted cognitions resulted in a corresponding change in negative emotional states or vice versa, and iii) the relative effect of cognitions and poor emotional health on diabetes self-care.

Cognition domains were associated with poor emotional health constructs in the theoretically expected direction. Optimistic illness cognitions correlated negatively with poor emotional health and similarly pessimistic cognitions correlated positively with poor emotional health. Meta-analysed findings showed that effect size estimates were larger when studies with a weak quality rating were excluded. The relationship between treatment control and depression may be susceptible to publication bias. Psychological intervention studies showed changes in diabetes cognitions and corresponding improvements in negative mood in two of the four intervention studies. The relative effects of cognitions and poor emotional health (specifically depression) on diabetes self-care was tested in only two studies. Mixed results were found. Depression predicted adherence to diabetes self-care in one study but not the other; however both studies identified specific diabetes cognitions as important predictors of diabetes self-care. The relationships identified between cognition domains and diabetes self-care differed across the types of health behaviour measures used.

2.5.2 *Strengths and Limitations*

This systematic review had a number of strengths. Following a thorough systematic search data were reliably extracted from studies as was evidenced from the validity of extraction checks. The review was limited to specific measures of illness cognitions which allowed greater comparisons across included studies and findings for the first review objective to be meta-analysed. Meta-analysis has greater statistical power to identify meaningful effects by combining findings across included studies appropriately weighted by their sample size (Lipsey and Wilson, 2001). Where studies reported beta coefficients these were transformed into correlation coefficients to allow their findings to be included in the review decreasing the chances of sampling error (Peterson and Brown, 2005). Risk of

within study bias was appraised with a quality assessment tool with published evidence of inter-rater reliability (Armijo-Olivo, et al., 2010) and content validity (Thomas, et al., 2004). The tool was applied to a broad range of research designs and acknowledged the importance of the reliability and validity of measurement instruments (Armijo-Olivo, et al., 2010).

There were number of limitations of this review. Only studies published in English peer reviewed journals were included, findings of the review are potentially exposed to publication bias. Indeed, the FSN statistic for the meta-analysed findings between treatment control and depression was low (FSN=2) and this relationship in particular should be interpreted with caution. However the association between illness cognitions and poor emotional health was often not the primary research question of included studies and in this sense, the effects of publication bias may be attenuated. In future, inclusion criteria for reviews of this kind should be extended to include foreign language papers and grey literature searches.

Whilst risk of bias was rigorously appraised using a validated tool, a global quality score was used. Global quality ratings are discouraged by the Cochrane Collaboration as they assume each quality criteria has equal weight when a total score is generated (Higgins and Green, 2008). Despite using an overall quality rating, each quality criteria was also reported separately to allow specific aspects of bias to be explored where necessary. In addition, whilst the quality assessment tool used in this review is more appropriate than tools used to assess conventional domains of bias associated with randomised controlled trials, its validity in appraising observational study designs is questionable. In the context of this review two items became redundant (confounding and blinding). More consideration is needed in the field of epidemiological research on how best to appraise study quality when findings are meta-analysed (Stroup, et al., 2000).

2.5.3 *Interpretation of findings*

Are diabetes cognitions associated with poor emotional health?

Review objective one's findings support the CS-SRM's claims that illness cognitions and emotional health interact with each other (Leventhal et al., 1980; 1984). When cognition domains had a statistically significant association with depression, anxiety or negative well-being the nature of the association was in the direction hypothesised by the CS-SRM;

negative cognitive appraisals were associated with poorer emotional health excluding illness coherence for one study. These findings are consistent with other psychological theories of mood disorders which emphasise the importance of negative thinking styles in the development and maintenance of depression and anxiety (Beck, et al., 1979; Mathews and MacLeod, 2005; Teasdale, 1983; 1988).

The statistically significant relationships identified for the pessimistic cognition domains: timeline cyclical, consequences, and seriousness beliefs can be considered most robust as these findings were consistently reported across the emotional states depression, anxiety, and negative well-being (excluding seriousness beliefs because this concept was studied in relation to depression only). In addition these relationships were largely based on meta-analysed findings. This shows that appraising diabetes as an unpredictable condition, that impairs functioning, has emotional relevance across all emotional health concepts included in this review. The importance of seriousness beliefs in relation to anxiety and negative well-being is still unknown. It is worth highlighting that the consequences domain only had a relationship with anxiety in Rees et al's (2012) study among participants with diabetic retinopathy. Thus the presence of diabetes complications may determine the extent to which relationships are observed between diabetes cognitions and poor emotional health.

The relevance of the two remaining pessimistic illness cognition domains (identity and timeline acute/chronic) to negative emotional states is less clear. Identity showed consistent statistically significant relationships with anxiety and negative well-being but not with depression. Likewise timeline acute/chronic beliefs showed a statistically significant relationship with anxiety but not with depression or negative well-being. Thus it is likely that specific diabetes cognition domains have greater salience for some emotional states but not others. This finding is consistent with the content specificity hypothesis which argues that emotional states can be distinguished from each other based on their cognitive content (Beck, et al., 1987).

The importance of optimistic diabetes cognition domains (personal control, treatment control, and illness coherence) to negative emotional states is also ambiguous. The negative correlation found between personal control and depression can be considered robust because it was based on meta-analysed findings. In addition this relationship was replicated for the emotional state anxiety but in a single study. However no statistically significant effect was observed between personal control and negative well-being and the

effect of personal control on both depression and anxiety was statistically non-significant for the study which tested these relationships using only multivariate regression. Personal control appears to have a salient relationship with depression and anxiety but its role may be limited when considered relative to other illness cognition domains. However other methodological differences may account for these effects other than statistical factors yet none were identified through the data extracted for this review.

Whilst meta-analysed findings demonstrated a statistically significant relationship between depression and treatment control the effect size was small (r=-0.10) with an indication that it may be subject to publication bias (FSN=2). In addition these effects were not found in Rees et al's (2012) multivariate regression model (irrespective of diabetic retinopathy status) when control beliefs were entered simultaneously alongside other illness cognition domains. Likewise, no relationships were observed between negative well-being and treatment control. On balance these findings indicate that treatment control is not a key cognitive domain in terms of its relationship with depression, anxiety, and negative well-being.

The relationship between illness coherence and negative emotional states had mixed statistically significant and non-significant findings both across and within emotional state concepts. In addition for one study higher illness coherence beliefs were associated with higher levels of depression which was at odds with the negative association reported by the remaining studies which found a statistically significant relationship. The role of this construct in relation to poor emotional health is unclear. For some it may have little salience, in others it may generate either a sense of empowerment or the reverse a sense of powerlessness because of the emotional burden associated with increased illness understanding. Indeed, individuals lower in illness coherence may be in denial. Avoidance coping behaviours are associated with improved emotional well-being in diabetes (Macrodimitris and Endler, 2001).

The relevance of medication beliefs to poor emotional health is unclear as these effects were only studied in relation to negative well-being and warrant further testing.

Do interventions which target illness cognitions improve poor emotional health and vice versa?

Findings for review objective two showed that when changes in cognition domains in the intervention group(s) were observed they were associated with either improvements in negative moods or stability in negative mood relative to the control group. Perceived personal control and illness coherence changed consistently across two out of the four intervention studies, both increased. In both of these intervention studies a corresponding improvement in mood was observed. These findings indicate the potential for these domains to be targeted by intervention to positively impact on negative mood states. However as identified in review objective one the association between illness coherence and poor emotional health requires further clarity.

In the DiGEM intervention (Farmer et al., 2009; French et al., 2008) consequences beliefs increased in both the intervention groups relative to the control group but no differences in participants' scores on negative well-being occurred. Review objective one showed robust associations between consequences beliefs and poor emotional health, thus the DiGEM findings are at odds with the observational studies. This may indicate either i) consequences beliefs have no causal effect on low mood or ii) other positive cognition domains (e.g. personal control) moderate the effects of negative diabetes beliefs on poor emotional health.

Conclusion one seems unlikely as Skinner et al., (2011) found that consequences beliefs were able to longitudinally predict depression. Conclusion two seems plausible because the two interventions in this review which showed improvements in low mood did so whilst allowing participant's to maintain pessimistic but accurate cognitive representations of their diabetes. To elaborate, increased timeline acute/chronic and seriousness beliefs were observed in the DESMOND intervention (Davies et al., 2008; Khunti et al., 2012) and increased lifestyle causal attributions (which could be interpreted as self-blame) were found by Keogh et al. (2011). Because each of the interventions also reported increased perceptions of personal control it seems likely that this domain prevented feelings of helplessness and thwarted the effects of the pessimistic diabetes cognitions on low mood.

Empirical support for positive illness cognitions moderating the impact of negative illness cognitions is provided by Skinner et al's (2011) cluster analysis. This analysis was performed on participants who were recruited into the DESMOND trial. Cluster analysis

allows the interaction between illness cognition domains to be statistically acknowledged and provides a more robust test of the CS-SRM by testing illness representation schemas rather than assuming cognition domains to be standalone constructs.

Skinner et al. (2011) identified four distinct groups of participants whose illness cognition domains correlated in different ways and labelled them: accepters, resisters, accepters resisting the consequences, and resisters accepting the consequences. The accepters group accepted the medical model of diabetes (high timeline acute/chronic and illness coherence beliefs), felt high in personal control, and had the lowest consequences beliefs relative to the remaining four clusters. Participants classified as accepters were found to have lower depression scores at eight months follow-up.

The pattern of relationships reported by Skinner et al (2011) for the accepters group provides support for the findings observed in the intervention studies. It appears that individuals are able to accept the "pessimistic" medical of model of diabetes whilst maintaining or indeed improving their emotional health if they perceive themselves to be high in personal control. Further research is needed to test whether positive cognitions can indeed moderate the impact of negative diabetes beliefs. It should be highlighted however that whilst the "accepters" group had beliefs consistent with the medical model they perceived diabetes to have fewer consequences on their life which may equally explain the positive effects on mood.

The conclusions for review objective two should be interpreted cautiously. All interventions which observed changes in mood were complex and included multiple "active ingredients" which may also plausibly explain the observed improvements or stability of mood e.g. social learning in the DESMOND intervention, goal setting in the DiGEM trial or social support in the intervention by Keogh et al (2011). Consistent with the Medical Research Council's (2000; 2008) framework for complex interventions studies need to map out the key causal mechanisms of action to allow targets for future intervention to be identified. Both the DESMOND and DiGEM trials did perform this process but only in relation to the primary outcome measure either: HbA1c or diabetes self-care.

What are the relative effects of cognitions and emotions on diabetes self-care?

This review identified a lack of research into the relative effects of illness cognitions and poor emotional health on diabetes self-care behaviours. Only two studies were identified which explored these relationships. Their findings were mixed.

One study showed that treatment control and not depression predicted improved adherence to dietary diabetes self-care behaviours. The authors attribute the inability of depression to predict dietary adherence to a lack of variance in participant's baseline depression scores. But these findings were not replicated when a different measure of dietary self-care adherence was used, thus poor health behaviour measurement scales may limit a study's ability to detect statistically significant effects.

The second study showed both depression and illness cognitions (personal control and timeline cyclical beliefs) to be important predictors of adherence to a general measure of diabetes self-care behaviours (cross-sectional analysis). But tests of indirect effects did not support the hypothesis for personal control mediating the relationship between depression and diabetes self-care. Yet the reverse indirect pathway was not explored (e.g. the potential for depression to mediate the relationship between personal control and diabetes self-care). In addition the study had a small sample size and thus may not have been sufficiently powered to detect effects. In sum, few studies have rigorously tested the potential for diabetes cognitions and poor emotional health to have a combined effect on diabetes self-care behaviours.

2.6 *Conclusion*

This review has identified a number of diabetes cognitions which have demonstrated a relationship with poor emotional health (depression, anxiety, and negative well-being). These may be important targets for clinical intervention using cognitive based approaches (e.g. cognitive reappraisal) to allow individuals with diabetes to experience positive changes in their mood. However the clinical utility of this proposed intervention strategy remains unclear because:

i. All studies (excluding Skinner et al., 2011) measured the association between cognitions and emotion cross-sectionally.

ii. Intervention studies which observed changes in cognition domains and improvements or no effect on mood did not explicitly test the causal pathways through which this occurred.
iii. Some cognition domains have been less researched and their importance for mood may have been overlooked.

The causal relationships that exist between cognitions and poor emotional health remain unanswered. Longitudinal studies of the relationship between poor emotional health and all cognitions domains of the CS-SRM are needed to tease apart the direction of effects that exist between them.

This review has also identified a lack of studies into the combined effect of cognitions and emotions on diabetes self-care behaviours. Longitudinal studies of the relationship between cognitions and emotions also need to incorporate into their statistical model diabetes self-care to test the direct and indirect effects of cognitions and emotions on adherence.

It remains unclear whether cognitive profiles which are adaptive for emotional health regulation are equally as adaptive for the management of diabetes. The intervention studies allude to the potential for this to occur with participants maintaining cognition profiles consistent with the medical model of diabetes (the types of cognitions needed to motivate adherence to diabetes self-care; Cameron and Moss-Morris, 2010) whilst also demonstrating improvements or stability in mood. Future research needs to explicitly acknowledge the role of emotions in the context of long term condition management and identify methods of intervening which are not antagonist to the management of a physical long term condition.

2.7 *Chapter summary*

2.7.1 *Statement of contribution*

What was already known?

- Diabetes illness and medication cognitions are associated with adherence to diabetes self-care behaviours.
- Depression and anxiety decrease adherence to diabetes self-management behaviours.
- The CS-SRM recognises that diabetes cognitions and poor emotional health have the potential to interact with each other.

What does this review add?

- Specific diabetes cognitions interact with poor emotional health with the most robust associations occurring between poor emotional health and timeline cyclical, personal control, seriousness, and consequences beliefs.
- The causal relationships that exists between diabetes cognitions and poor emotional health are unclear because studies have either:
    - **i.** Used cross-sectional analyses
      Or
    - **ii.** Not formally evaluated whether diabetes cognitions mediate an interventions effect on poor emotional health
- The relative effects of diabetes cognitions and poor emotional health on diabetes self-management behaviours is uncertain because of few studies testing these relationships simultaneously.

3 *General methodology*

3.1 *Introduction*

This chapter outlines the research hypotheses to emerge from the literature reviewed in chapters one and two and describes how these findings have informed the methodological design of the empirical research conducted for this thesis. An overview of the statistical approach of structural equation modelling is also provided to serve as a reference point for when analyses are presented in subsequent chapters.

3.2 *Research hypotheses*

The general introduction in chapter one showed that the majority of studies using the Common Sense-Self-Regulation Model (CS-SRM) as a theoretical framework have focussed on the relationships between illness cognitions and problem focussed health behaviours to the detriment of considering the importance of emotional responses. Chapter two systematically explored the relationships between illness cognitions, medication cognitions, poor emotional health, and problem focussed health behaviours in adults with diabetes.

The review identified only one longitudinal observational study of the relationship between diabetes cognitions and poor emotional health. This study was limited however because it only measured a small number of cognition domains and tested the causal effect of cognitions on poor emotional health only and not vice versa. This finding identified the need to conduct a longitudinal observational study to explore these pathways over time and to test the theoretical claims of the CS-SRM that cognitions and emotions have a bi-directional relationship.

The systematic review also identified only two studies which have tested the combined effect of cognitions and emotions on diabetes self-care behaviours. These studies were methodologically weak however. Whilst one study had a longitudinal design, it included a very specific measure of diabetes self-care (saturated fat eating patterns) and only two cognition domains. The second study was limited by its cross-sectional design. It did however begin to unpick the potential for cognitions to mediate the relationship between depression and diabetes self-care. These review findings established the need for a

longitudinal test of the combined (mediated) and independent (direct) effect of diabetes cognitions and poor emotional on diabetes self-care behaviours.

Based on the theoretical claims of the CS-SRM and previous empirical work reviewed in chapter one the following primary research hypotheses emerged:

Hypothesis one: Diabetes cognitions and poor emotional health have a longitudinal bi-directional relationship (cognitions ↔ emotions).

Hypothesis two: Diabetes cognitions and poor emotional health have a combined and independent longitudinal effect on diabetes self-care at follow-up, specifically:

i. Diabetes cognitions have a direct and indirect effect (via poor emotional health) on diabetes self-care (cognitions → diabetes self-care, *and* cognitions → emotions → diabetes self-care).
ii. Poor emotional health has a direct and indirect (via diabetes cognitions) on diabetes self-care (emotions → diabetes self-care, *and* emotions →cognitions → diabetes self-care).

Secondary research hypotheses

McSharry et al's (2011) review identified an absence of studies testing the longitudinal effect of illness cognitions on HbA1c. In addition whilst studies have explored longitudinally the effect of poor emotional health on HbA1c an examination of the degree to which these effects are replicable was needed. A secondary research hypothesis was therefore generated:

Secondary research hypothesis one: Baseline diabetes cognition domains and poor emotional health have a combined and independent longitudinal effect on HbA1c at follow-up, specifically:

i. Diabetes cognitions have a direct and indirect effect (via poor emotional health) on HbA1c (cognitions → HbA1c, *and* cognitions → emotions →HbA1c).
ii. Poor emotional health has a direct and indirect effect (via diabetes cognitions) on HbA1c (emotions → HbA1c, *and* emotions →cognitions → HbA1c).

3.3 *Design rationale*

3.3.1 *Longitudinal observational study*

A longitudinal study allows key directional pathways to be identified under the following conditions (Kenny, 1979):

i. Time precedence: for a variable to be considered a "causal agent" of another it must go before it in time.
ii. Relationship: two variables must be identified whereby the association between them is not independent, for example knowing the value of Variable X can inform the predicted value of Variable Y.
iii. Non-spuriousness: the relationship between the two variables is not caused by a third "other" variable common to both variable X and Y.

This design was therefore chosen and is consistent with the Medical Research Council's recommendations for an iterative and reflexive approach to identify key predictive pathways to inform the development of clinical interventions (Medical Research Council; 2000, 2008).

It must be acknowledged however that randomised controlled trials (RCTs) offer the most robust approach to establishing causality because they allow the explicit manipulation of the hypothesised causal variable in the intervention group whilst comparing its effects to the no intervention control group, controlling for confounding variables by randomisation (Bowling, 2002). However the focus of this thesis was on identifying predictive pathways of action to inform the design of subsequent RCTs. With this in mind the term "cause" may be used in this thesis when describing longitudinal relationships between variables yet it should be interpreted with caution.

Gollob and Reichardt (1987) emphasise the importance of identifying and implementing optimal time intervals for the hypothesised predictive variable to demonstrate its effects on the outcome variable. If inappropriate time intervals are applied it can have a substantial impact on effect sizes.

The CS-SRM acknowledges that a person's cognitive illness representation and emotional response to a health threat is dynamic. Thus change can occur at any time in response to factors specific to health or the broader social context.

A time-interval of six months was deemed an appropriate time interval to allow events in relation to a person's diabetes or external to diabetes (e.g. life stress/change in personal circumstances) to change. In addition a change in a person's glycosylated haemoglobin (HbA1c) is detectable at three monthly intervals and is a marker of diabetes control (NICE, 2008). Six months gives sufficient time for a person to experience a change in their overall diabetes control.

In addition, previous longitudinal studies exploring the relationship between illness cognitions and emotional health have demonstrated statistically significant effects over a six month time interval (Llewellyn, et al., 2007; Skinner, et al., 2000). However the temporal relationships between illness cognitions, emotional health, and health behaviours are not explicitly stated within the CS-SRM.

3.3.2 *Self-report questionnaire measures*

The relationship between cognitions, emotions, and diabetes self-care was quantitatively measured with validated self-report Likert scale questionnaires. This approach was taken to obtain effect size estimates of the relationships between variables and to identify key areas for future intervention.

3.4 *Statistical approach: Structural Equation Modelling (SEM)*

The statistical approach of structural equation modelling (SEM) was implemented to test the primary research hypotheses and secondary research hypothesis one. In brief SEM allows the strength and direction of multiple direct and indirect pathways to be tested simultaneously whilst also providing global estimates of the accuracy of the hypothesised model.

3.4.1 *What is SEM?*

Structural equation modelling (SEM) is a confirmatory hypothesis testing statistical approach (Byrne, 2010). It allows hypothesised theoretical models to be statistically

analysed by the generation and simultaneous testing of multiple pathways between variables important to the theoretical model under study (Schumacker and Lomax, 2004).

3.4.2 *Why use SEM?*

SEM has a number of advantages over traditional methods of testing relationships between variables (e.g. linear regression analysis, analysis of variance) including the:

i. Use of latent variables in the model.
ii. Statistical evaluation of the entire hypothesised theoretical model.
iii. Capacity to test the relative importance of multiple hypothesised direct and indirect relationships in a single analysis.

Modelling of latent variables

Unlike regression, when testing hypothesised directional relationships between variables SEM allows associations between latent variables to be included in the statistical model.

A latent variable can be used to represent a construct that is not directly observable for example, depression. They are generated by specifying the type of observed variables (directly measureable) the underlying construct is likely to influence responses to (Anderson and Gerbing, 1984; Hoyle and Smith, 1994; Kline, 2005; Tomarken and Waller, 2005). For example depression may be specified to drive responses to measures of sadness, concentration, self-worth etc.

Latent variables provide an estimate of the degree of measurement error in each of the observed variables specified to reflect the underlying theoretical construct (Hoyle and Smith, 1994). This measurement error is then statistically accounted for when estimates of an independent variable's effect on the dependent variable are calculated and permits a more precise effect size estimate (Kline, 2005).

Evaluation of the entire theoretical model

Statistical tests which assess the whole model are known as goodness to fit indices and are described in Table 3.1. If these statistics show that the hypothesised model provides a good

fit to the sample data then the relationships specified within the model (e.g. directional pathways) can be interpreted as theoretically meaningful (Hancock and Liu, 2012).

In contrast, regression analysis does not provide estimates which evaluate the whole model. Regression based R squared estimates and their associated ANOVA F statistic provide an indication of whether the amount of variance explained in the dependent variable is statistically significant yet deficiencies in the hypothesised model and whether its hypothesised relationships are valid go undetected (Bollen, 1989).

Testing the relative effects of multiple direct and indirect pathways between variables simultaneously

Testing multiple pathways simultaneously allows shared variance between the multiple predictors to be statistically controlled (Hayes, 2009; Preacher and Hayes, 2008). This permits an examination of whether hypothesised causal effects can occur independently from other direct and indirect effects specified within the model and identifies the most salient predictors.

3.4.3 ***How is SEM conducted?***

3.4.3.1 *The generation of measurement and structural models*

A two-step approach to structural equation modelling is encouraged (Anderson and Gerbing, 1988; Hoyle and Smith, 1994). Prior to exploring the hypothesised directional relationships between latent variables a statistical evaluation of their hypothesised latent structure using confirmatory factor analysis is advocated (Anderson and Gerbing, 1988; Hoyle and Smith, 1994). This permits problems in the measurement and specification of theoretical constructs to be identified and resolved in advance of testing directional relations between them.

The statistical evaluation of latent variables is referred to as a measurement model (Anderson and Gerbing, 1988; Hoyle and Smith, 1994; Kline, 2005). An example of a hypothetical measurement model is shown in Figure 3.1a for illustration. It consists of two latent variables X and Y. Latent variables in SEM diagrams are always represented by circles or ellipses. Latent variables within a measurement model are assumed to co-vary with each other. For example they are assumed to be associated but the direction of effect is not specified (Anderson and Gerbing, 1988; Kline, 2005). The covariance between the latent variables is represented in Figure 3.1a by a double headed arrow between the latent variables X and Y. Co-variance statistics represent the strength of association between two variables, a correlation coefficient is a standardised covariance statistic (Kline, 2005).

Each latent variable is specified to drive responses to a number of observed variables also referred to as indicators (Kline, 2005). Indicators can be item scores on a specific questionnaire subscale or whole subscale/scale scores from one or more questionnaires (Anderson and Gerbing, 1988; Floyd and Widaman, 1995).

The indicators in Figure 3.1a are represented by squares and labelled I1 to I6. Square symbols in a SEM diagram indicate that the variable is directly observed/measured. The directional arrow leading from the latent variable to the indicators demonstrates that the underlying theoretical construct (the latent variable) influences the responses given to the indicators. The extent to which the latent variable has an effect on each indicator is estimated using factor loadings. Factor loadings are typically estimated using regression

coefficients (Kline, 2005). This method of interpretation applies in the context of the current thesis.

Indicators with high factor loadings provide evidence of construct validity (Anderson and Gerbing, 1988; Hoyle and Smith, 1994; Little, et al., 2007). Generally factor loadings above 0.40 are considered meaningful indicators of the underlying theoretical construct (Floyd and Widaman, 1995; Hatcher, 1994).

Each indicator in the measurement model has an error term associated with it. Error terms estimate the amount of variance observed in the indicator that is not accounted for by the latent variable (Kline, 2005). There are two sources of measurement error: systematic and random (Kline, 2005; Thurston, 1947 cited in Coffman and MacCallum, 2005). Systematic error includes imprecision related to the specific indicator, for example less than perfect internal reliability, test-retest reliability (Kline, 2005). Random error includes inaccuracies which are not specifically associated with the indicator - latent variable relationship, for example the conditions in which measured variables were obtained, social desirability in responding etc. (Kline, 2005; Thurston, 1947 cited in Coffman and MacCallum, 2005).

Error terms are latent variables because the causes of the error have not been directly observed (Hoyle and Smith, 1994). The error terms in Figure 3.1a are shown by the circles labelled E1 to E6. The directional arrow leading from the error term to the indicator demonstrates that sources of error have an effect on the scores observed on the indicators. The directional effect of error terms on indicator variables are always scaled to equal one.

Once the hypothesised indicators of each latent variable have been specified by the researcher the statistical properties of the measurement model are tested using SEM software. If the outcomes of the statistical tests are satisfactory (e.g. well fitting) the hypothesised directional relationships between the latent variables are tested by generating a structural model (Anderson and Gerbing, 1988). Latent variables which were previously specified to co-vary in the measurement model are replaced with directional arrows in the structural model according to the hypothesised theory. As in Fig 3.1b variable X is now specified to have a direct effect on variable Y as indicated by the single headed arrow leading from X to Y. The estimate of the directional effect of variable X on Y is a

regression coefficient (Kline, 2005). If a structural model has more than one independent variable they are specified to co-vary with each other (Kline, 2005).

The move from a measurement model to a structural model also requires the addition of disturbance terms associated with each dependent variable in the model. Disturbance terms represent all remaining variance in the dependent variable(s) which is not accounted for by the independent variables in the model, for example omitted independent variables (Kline, 2005). As shown in Figure 3.1b disturbance terms are latent variables as they are not directly observed. The directional arrow leading from the disturbance term to the dependent variable indicates that they have an effect on the variance of the dependent variable. This is typically scaled to one.

The stages involved in statistically testing the measurement and structural model are identical and are outlined below. Once the stages have been completed for the measurement model the same series of steps are carried out to evaluate the structural model.

Figure 3.1: Diagrammatic representation of a measurement and structural model

Figure 3.1a: Measurement model

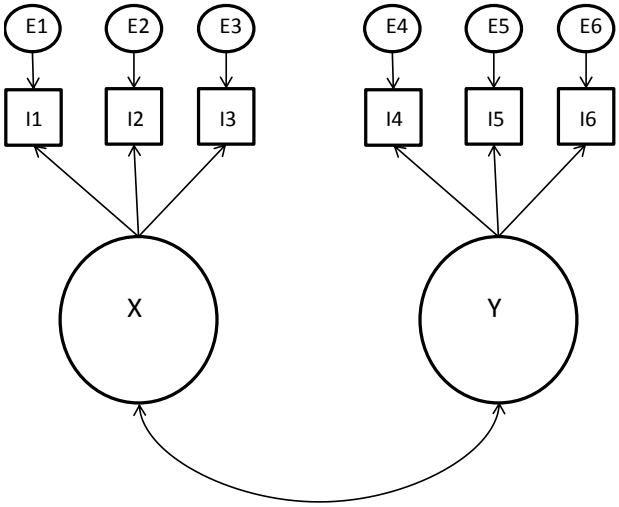
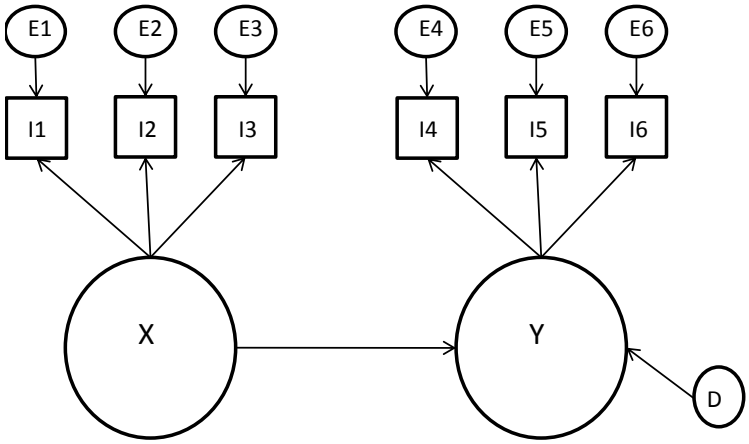


Figure 3.1b: Structural model



Key: Ellipses/circles represent latent variables; squares represent measured variables, X & Y represent hypothetical constructs; E, error term associated with indicator variable; I, Indicator variable/questionnaire item

3.4.3.2 *Stages involved in testing measurement and structural models*

Stage One: Ensure the model is identified

Meeting the Principles of Identification is a necessary condition for SEM (Kline, 2005; MacCallum, 1995; Schumacker and Lomax, 2004). An identified model permits unique statistical solutions to be obtained for the model parameters, elements within the model that require estimating (Byrne, 2010; Kline, 2005; MacCallum, 1995; Schumacker and Lomax, 2004).

For a model to be identified it must meet a number of statistical conditions. Establishing whether these conditions are met can be difficult to determine without the aid of computer software (MacCallum, 1995). A key condition fundamental to the statistical interpretation of SEM outputs is that the number of parameters specified to be estimated in the model does not exceed the number of observations available in the sampled data set (Kline, 2005; MacCallum, 1995). This is consistent with regression but this condition is always met because there is only ever one outcome variable in a regression model.

The number of statistical observations available in a sampled data set is represented by the number of variances and co-variances between observed/measured variables (Kline, 2005; Schumaker and Lomax, 2004). If a model had three observed variables the number of observations within the model equals six (e.g. three variances for each measured variable and three co-variances which exist between them).

If the number of parameters specified in the hypothesised model equals the amount of observations available in the sampled data set then the model is just-identified (Chou and Bentler, 1995; Kline, 2005; MacCallum, 1995). Unique parameter estimates can be obtained; however a global estimate of model fit cannot be estimated. This is because the relationships specified in the model duplicate those observed in the sampled data, and the model therefore is not summarising sampled relationships (Chou and Bentler, 1995; Kline, 2005; MacCallum, 1995). A typical regression analysis meets these principles.

When the number of observations exceeds the number of parameters to be estimated a model is over-identified. Over-identified models are preferred in SEM as they allow i) unique parameter estimates, and ii) an estimate of global fit which provide a robust test of

how well the relationships summarised in the model reflect those observed in the sampled data set (Chou and Bentler, 1995; Kline, 2005; MacCallum, 1995).

The higher the ratio of observations to number of parameters to be estimated the more stringent the test of the hypothesised theoretical model. This is because the model has had to satisfy a greater number of statistical conditions (Chou & Bentler, 1995; Kline, 2005; MacCallum, 1995). The parameter solutions obtained from over-identified models do not directly replicate the observations from the sample data as unique parameter estimates which satisfy all of the observations cannot be obtained. Instead parameter estimates provide the optimal solution for the model with the smallest possible residual difference between the relationships proposed by the model and those observed in the sample data (Chou & Bentler, 1995; Kline, 2005). When a model is over-identified it has positive model degrees of freedom (df_m; Kline, 2005).

Stage Two: Statistical evaluation of the entire model

To evaluate the entire theoretical model the *goodness of fit* indices are examined.

Goodness of fit indices statistically test for discrepancies between the relationships hypothesised in the model and those observed in the sample data (Boomsma, 2000; Byrne, 2010; Hoyle, 1995).

Numerous goodness of fit statistics are available. Each index has its own merits in terms of what it takes into consideration when evaluating a model including: sample size, statistical power, and model complexity (Boomsma, 2000). Given the relative strengths and weaknesses of differing fit indices more than one index is reported when evaluating models. The following fit indices should be reported as a minimum and are summarised in Table 3.1 (Boomsma, 2000; Kline, 2005; McDonald and Ho, 2002):

i. Model chi-square χ^2
ii. Root mean square error of approximation
iii. Comparative Fit Index
iv. Standardised root mean square residual

In addition when comparing competing alternative theoretical models comparative fit indices should be reported e.g. Aikaike's Information Criterion.

Table 3.1: Summary of Goodness of Fit Indices

Goodness of fit index	Description	Statistical interpretation	Advantages	Disadvantages
Model chi-square χ^2	Assumes no difference between the parameters estimated by the model and those observed in the sample data (Byrne, 2010; Hu and Bentler, 1995; Kline, 2005).	Requires a true null hypothesis Smaller χ^2 = better model fit	Most frequently reported in studies of SEM Useful for evaluating nested model comparisons (described later, see Chapter Seven, Section 7.3.1).	Unrealistic assumption that model will demonstrate perfect fit (Byrne, 2010) Larger effect sizes = larger χ^2 (Kline, 2005) Larger samples = greater power to detect differences. Small samples = magnified χ^2 (Byrne, 2010). Non-normal data = magnified χ^2 (West, et al., 1995)
Comparative Fit Index (CFI)	Tests difference in χ^2 between hypothesised and independence model (a model that assumes no relationships between variables in the population)	Values close to 0.95 indicate a good fitting model (Hu & Bentler, 1999).	Less sensitive to small sample sizes (Fan et al., 2009)	Sensitive to non-normality = moderately lower CFI estimates (Byrne, 2010). However less sensitive than χ^2 (Lei and Lomax, 2005)

Goodness of fit index	Description	Statistical interpretation	Advantages	Disadvantages
Root Mean Square Error of Approximation (RMSEA)	Compares model parameter estimates with those that are estimated to occur at the sampled population level. Provides 90% confidence intervals and an estimate of closeness of fit: PCLOSE	Requires a false null hypothesis Values ≤ 0.06 indicate good fit (Hu and Bentler, 1999). PCLOSE should exceed 0.50 to indicate close fit (Byrne, 2010). Values ≤0.10=adequate fit (MacCallum, et al., 1996)	Adjusts for model complexity Responsive to model misspecification (Hu and Bentler, 1998)	Small sample size can lead to rejection of true population models (Bentler and Yuan, 1999) however less sensitive to sample size than other estimates (Fan, et al., 1999). Sensitive to non-normality (Nevitt and Hancock, 2000).
Standardised Root Mean Square Residual (SRMR)	Estimates the mean residual difference between the hypothesised model and the observed sample data (sample based estimator).	Values ≤0.10 indicate good fit (Kline, 2005)	Sensitive to model mis-specification (Hu and Bentler, 1998) Less sensitive to the assumptions of multivariate normality (Iacobucci, 2010)	Does not adjust for model complexity (Hooper, et al., 2008) Elevated estimates in small samples (Hooper, et al., 2008).
Akaike Information Criterion (AIC)	Used in the selection of competing theoretically plausible models	The model with the smallest AIC value is preferred (Kline, 2005).	Adjusts for model complexity (Kline, 2005)	Does not take into account sample size (Byrne, 2010).

Stage Three: Model re-specification

If goodness of fit indices suggest that the model does not adequately explain the hypothesised relationships between variables then model re-specifications are applied. When model re-specification is implemented the confirmatory nature of SEM no longer applies and the approach moves into exploratory realms (Byrne, 2010; Kline, 2005).

Statistical criteria are used to identify areas for model modification. Nonetheless the model re-specifications implemented should be theoretically meaningful (Anderson and Gerbing, 1988; Byrne, 2010; Kline, 2005). Inspection of residual matrices, modification indices (outputs generated by SEM software), factor loadings, and co-variances can help determine areas for model re-specification. A detailed account of the process involved in identifying areas for model re-specification is not provided here. However, Anderson and Gerbing, (1988), Byrne (2010), and Kline (2005) provide an outline of the steps involved.

Stage four: Statistical evaluation of the parameters in the model

Once a model adequately fits the sample data then the directional relationships within the model can be statistically interpreted for example factor loadings within the measurement model, predictive pathways within the structural model, co-variances, and variances.

The application of SEM to longitudinal models is described in the statistical analysis section of Chapter Seven.

3.4.4 *Item parcelling*

An alternative approach to testing hypothesised structural relationships between variables is to generate item parcels. Parcels are generated by summing or taking the mean of a group of questionnaire items and these are used as latent variable indicators in place of individual item scores (Coffman and MacCallum, 2005; Kline, 2005). The approach is advantageous when estimating a complex model with a limited sample size as it is a method of reducing the number of parameters to be estimated in the model (Kline, 2005; Little, et al., 2002; West, et al., 1995). For example if a six item measure of depression was used these items could be summed to generate a single depression parcel. The latent variable depression would then be specified to have a single indicator consisting of the depression parcel as opposed to having six item level indicators.

When latent variables have only single indicators the model will fail to meet the principles of identification (see above, Section 3.4.3.2) and therefore cannot be statistically estimated (Coffman and MacCallum, 2005). If this occurs Bollen (1989) suggests calculating an estimate of the indicator/parcel's error term which can then be "fixed" in the model. By providing the SEM software with a "fixed" estimate of the error term it no longer has to account for its calculation and allows the reliable estimation of the remaining model parameters. An estimate of a single indicator latent variable's error term can be obtained using the formula below:

$$(1-\alpha) \times \text{variance}$$

Whereby α=Cronbach's alpha reliability coefficient of the items contained within the parcel, and variance is the estimated variance of the summed/averaged item scores used to generate the parcel. Whilst this approach results in less biased parameter estimates relative to when measured variables are used it only allows unique error variance to be accounted for and not random error (Coffman and MacCallum, 2005). See above Section 3.4.3.1 for a definition of the distinction between systematic and random error variance.

3.4.5 *Statistical assumptions of SEM*

The statistical assumptions of SEM are outlined below:

Adequate sample size and statistical power

Determining the necessary sample size in the context of SEM is challenging. There is no agreed methodological procedure for calculating sample sizes (Chan, et al., 2007; Quintana and Maxwell, 1999). Typically there are four approaches which can be taken: i) model based power calculations, ii) parameter based power calculations, iii) adequate ratio of participants to model parameters, and iv) use of findings from statistical simulation studies. Each of these approaches is briefly outlined below.

Model based power calculations

Model based power calculations allow estimates of the necessary sample size required to detect an acceptable level of overall model fit. MacCallum et al. (1996) outlines an approach which can be used for the RMSEA fit index. Consistent with standard power calculations a researcher specifies both the required level of alpha (p value) and statistical

power (beta) in addition to the hypothesised model's degrees of freedom and anticipated model fit value (effect size). This approach allows a study to be adequately powered to evaluate the overall theoretical model but it may not be sufficiently powered to detect whether pathways included in the model have statistically significant effects.

Parameter based power calculations

Parameter based power calculations allow sample size estimates to be generated which allow a study to be powered to statistically evaluate the contribution of a particular pathway in explaining variance in a specific dependent variable included in the model. This approach is consistent with regression based power calculations (Kline, 2005). However it may result in a study being underpowered to evaluate the entire theoretical model.

Participant to parameter ratio

Whilst a study may be sufficiently powered to detect a particular effect size (at either the model and/or parameter level) the precision of the statistical estimates (width of confidence intervals) may be compromised. Bentler and Chou (1987) in reviewing statistical simulation studies recommend that the ratio of participants to parameters be no less than 5:1 to allow reasonably reliable parameter estimates to be generated.

Statistical simulation studies

More generic methods for determining appropriate sample sizes in the context of SEM are based on findings from statistical simulation studies, which have manipulated a number of factors in addition to sample size including: number of indicators per latent variable, multivariate normality, and indicator factor loadings. Sample sizes within the region of 200 participants or more are generally agreed to be both sufficient and practical to provide robust estimates of both overall model fit and model parameters (Anderson and Gerbing, 1988; Chou and Bentler, 1995; Gerbing and Anderson, 1985; Quintana and Maxwell, 1999; Tanaka, 1987). Sample sizes in excess of 200 participants are also less likely to result in model estimation (convergence) failures (Anderson and Gerbing, 1984; 1988). Convergence failures occur when the necessary statistical algorithms cannot be satisfied and sampling errors along with other causes (e.g. latent variables with only two indicators,

low factor loadings) can contribute to their occurrence (Anderson and Gerbing, 1984; 1988).

Sample size calculations for this thesis are described in Chapter Four.

Absence of multicollinearity

Multicollinearity indicates the presence of strong correlations between two or more independent variables in a model. If variables are multicollinear it prevents the estimation of reliable parameter estimates (beta coefficients) and can impact on the amount of variance explained in the dependent variable (Field, 2009). Correlations greater than 0.80 are deemed to indicate the presence of multicollinearity. In addition co-linearity diagnostic statistics can be implemented to detect multicollinearity. Variance inflation factor (VIF) scores greater than 10 would indicate the presence of multicollinearity (Pallant, 2005). If variables are found to be highly correlated then one should be removed from the statistical model or the variables combined to generate a single variable (Kline, 2005).

Continuous data

SEM assumes that the sampled data are continuous (Floyd and Widaman, 1995), but research in the social sciences is often dominated by Likert response scales (Byrne, 2010). Bentler and Chou (1987) argue that it is not problematic to treat Likert scales as continuous when they are generated from scales with a minimum of four response categories and are normally distributed.

Multivariate normality

When a model has multiple variables testing the assumptions of multivariate normality can be challenging to explore, however examining tests of univariate normality can provide an adequate indication (Kline, 2005).

A visual inspection of a plotted histogram of participants scores on each variable should occur to ensure that it reflects an evenly distributed bell shaped curve (Altman and Bland, 1995). In addition statistical tests of normality should be performed e.g. skewness, kurtosis, and Kolmogorov-Smirnov tests.

A skew index of greater than ±2 on a rescaled standardised skew statistic indicates the presence of an asymmetric distribution and the variable is considered to be non-normal (Curran, et al., 1996; George and Malley, 2001).

A kurtosis index of greater than or equal to ±7 on a rescaled standardised index indicates that the distribution is kurtotic and therefore has absence of normality (West, et al., 1995).

A statistically significant Kolmogorov-Smirnov test indicates the absence of normality (Pallant, 2005).

One index capable of detecting multivariate kurtosis is Mardia's index (1970, 1974 cited in Byrne, 2010). A Mardia's index greater than 7.98 (Harlow, 1985 cited in Gao, et al., 2008) and a critical ratio score of greater than ±5 (Byrne, 2010) can be an indicator for the absence of multivariate kurtosis.

Methods used to handle multivariate non-normality

Bootstrapping is one approach recommended to address non-normally distributed relationships in the context of SEM (Byrne, 2010; Kline, 2005; West et al., 1995). In contrast to parametric statistics whereby the sample data are statistically compared against hypothesised normal distributions, bootstrapping enables statistical comparisons to occur against empirically defined distributions (Byrne, 2010; West et al., 1995).

Empirically defined distributions are generated by re-sampling from the existing sampled population. The technique involves taking a series of subsamples of the same size from the original data. A sampling with replacement strategy is implemented whereby once a case is randomly selected from the original sample it is immediately replaced to allow it to be re-sampled again (West et al., 1995).

For each subsample generated the necessary parameter estimates are calculated and these multiple parameter estimates are used to generate an empirically derived sampling distribution (West et al, 1995). The statistical technique of bootstrapping assumes that the empirically defined distribution generated from the multiple subsamples replicates the population distribution from which it was originally drawn (Kline, 2005; Mooney and Duval, 1993). Under these assumptions it permits a more valid evaluation of the statistical significance of parameter estimates. Robust standard errors and associated confidence

intervals are calculated from the empirically defined sampling distribution as opposed to the observed non-normal distribution whereby standard errors are typically underestimated resulting in an increased Type I error (Arbuckle, 2009; Byrne, 2010; West, et al., 1995).

In the context of SEM the effect of non-normally distributed data on the relationships specified within the model e.g. variances and co-variances can be addressed using bootstrapping. However the application of bootstrapping to the evaluation of model fit indices is limited by SEM software capabilities (Byrne, 2010; Kline, 2005). Model fit indices should therefore be reported which have less rigid assumptions with regards to multivariate normality.

Missing data

SEM techniques require a complete data set with no missing values (Byrne, 2010). The manner in which missing data are handled when using SEM can depend on the computational properties of the software program. Analysis of Moments Structures Version 18 (AMOS; Arbuckle, 2009) was used in this thesis.

AMOS handles missing data by performing either a complete case analysis (e.g. list wise deletion) or uses a statistically robust approach of direct maximum likelihood estimation (single imputation). Complete cases analysis allows greater confidence in the statistical estimates generated because they reflect "real" data points (Allison, 2009). It is also considered to generate relatively reliable estimates when applied in the context of regression based analyses under the assumptions of missing at random (MAR) and when the data loss pattern is not associated with the dependent variable (Field, 2009). However if there are a large amount of missing values occurring across variables to be included in the statistical model it can result in loss of statistical power (Allison, 2009). In addition list wise deletion should not be applied when the data are not missing at random (NMAR; Allison, 2009).

Direct maximum likelihood estimation methods makes the use of all available data, reduces loss of statistical power, and makes less rigid assumptions about the patterns of missing data (Allison, 2003; 2009). It is arguably the favoured approach to handling missing data as it has been shown to demonstrate relatively unbiased statistical estimates (Allison, 2009; Arbuckle, 2007).

For completeness, both approaches were used in this thesis based on the recommendations of Sterne et al. (2009).

3.5 *Summary*

This chapter provided:

- A description of the research hypotheses.
- A rationale for the use of a longitudinal observational study.
- An overview of the statistical approach of structural equation modelling in relation to its analytical strengths, methodological approach, and statistical assumptions.

4 *Procedural Methods*

4.1 *Participants*

Consecutive participants with Type 2 diabetes were recruited from Manchester Diabetes Outpatient Centre, UK from May 2011 to October 2011.

The inclusion/exclusion criteria outlined below were applied.

Inclusion Criteria

Participants were eligible for inclusion if they:

i. had a diagnosis of Type 2 diabetes mellitus

ii. were aged 18 years or over

iii. attended the Manchester Diabetes Centre outpatient clinic

Exclusion criteria

Participants were excluded from the study if they:

i. Suffered from a disability, physical frailty or cognitive impairment that was deemed inappropriate by the participant themselves, a carer or the participant's medical team for them to take part in the study.

ii. The participant suffered from a severe mental illness (including severe depression with a risk of suicide or psychosis).

Eligible participants were approached by a member of their medical team and asked whether they would be interested in participating in a questionnaire based study on emotional distress in diabetes.

Interested participants were introduced to the researcher (JH) and the nature of the study was fully explained in a private consultation room. Participants who remained interested in taking part in the study were given a minimum of 24 hours before giving their informed

consent. A maximum of two polite telephone calls were made to those participants if the questionnaire had not been returned at baseline.

Participants who returned the questionnaire at baseline and gave their consent to be contacted at follow-up were sent a second questionnaire six months after the date of their initial recruitment into the study via post. Two polite reminder phone calls were made if the questionnaire was not returned.

Ethical and research and development approval (R & D) was gained in March 2011 (North West 12 Research Ethics Committee Lancaster, 11/NW/0069 and Central Manchester University Hospital, PIN R01506).

4.2 *Sample size calculations*

Sample size calculations were based on there being a large enough sample to:

i) allow precise model parameter estimates (e.g. less susceptible to sampling variability with smaller standard errors; Gerbing and Anderson, 1985)
ii) prevent model convergence failures at the overall model level (Anderson and Gerbing, 1984)
iii) allow robust estimates of overall model fit (Anderson and Gerbing, 1984; Quintana and Maxwell, 1999)

Statistical simulation studies using SEMs of varying complexity have found sample sizes of 200 participants to be sufficient to allow stable parameter and model estimates (Anderson and Gerbing, 1988; Chou and Bentler, 1995; Gerbing and Anderson, 1984; Quintana and Maxwell, 1999; Tanaka, 1987). A sample of 260 participants was therefore recruited at baseline to allow for a 20% attrition rate at six months follow-up.

4.3 *Self-report questionnaire measures*

A copy of the self-report questionnaire pack can be found in Methodological Appendix B.

4.3.1 *Revised Illness Perception Questionnaire (IPQ-R)*

Description

The IPQ-R (Moss-Morris, et al., 2002) was used to measure participant's illness cognitions. The measure provides a quantitative assessment of the illness cognition domains outlined in the Common Sense Self-Regulation Model (CS-SRM).

The IPQ-R was developed across different health conditions including Type 2 diabetes and includes the following three parts:

Part I: Identity subscale (14 items)

Part II: Timeline acute/chronic, timeline cyclical, consequences, personal control, treatment control, illness coherence, and emotional responses subscales (38 items in total).

Part III: causal items (18 items) and three open ended response options.

Part I of the IPQ-R consists of a list of 14 symptoms with a dichotomous yes/no response format. Participants are required to indicate: i) whether they have experienced any of the list of symptoms since having their illness and ii) whether they believe that the symptom (if experienced) is related to their illness. A score of one is given to each yes response to the belief that the symptom is related to their illness and a score of zero to each no response. Items are summed to give a total identity subscale score.

The seven subscales in Part II of the IPQ-R are scored on a 5-point Likert scale. A numerical score is assigned to each response category ranging from one (strongly disagree) to five (strongly agree). Items from each subscale are summed once the appropriate questionnaire items have been reverse scored.

Part III of the questionnaire contains 18 items which measure perceived causal factors of illness and the items have the same scoring system as Part II of IPQ-R. The 18 items of the causal domain are analysed using principle components analysis (PCA) to identify a collection of causal beliefs pertinent to the illness under study which can then be used as illness specific causal subscales. The four causal subscales identified by Moss-Morris, et al. (2002) were: psychological attributions, risk factors (biological and behavioural),

immunity, and accident/chance. In addition participants are required to rank order their three most important perceived causes of their illness using a free-text response.

A high score on a subscale represents a stronger belief in the illness cognition domain being measured.

Reliability

The Cronbach's alpha coefficient provides a measure of internal consistency/reliability and measures whether the hypothesised subscale/questionnaire items are responded to in a homogenous manner to reflect a single underlying construct (Bowling, 2002; Hoyle and Smith, 1994). The Cronbach's alpha coefficients for the IPQ-R found by Moss-Morris et al. (2002) were within an acceptable to good range (α=0.67 to α=0.86); based on Kline's (2005) criteria ($\geq$0.90 excellent, between 0.80-0.89 good, and values within the range of 0.70 are adequate). However the Cronbach's alpha coefficient for the accident/chance subscale was low (α=0.23) and Moss-Morris et al. (2002) recommend treating the items which make up this subscale as standalone causal attributions.

The IPQ-R subscales have displayed adequate test-retest reliability (a test of the stability of a measure of time; Bowling, 2002). Among participants with rheumatoid arthritis correlations ranged from 0.35 to 0.82 for each subscale over a six month interval. The lowest Pearson correlation coefficient (r=0.35) was for the timeline cyclical domain, but this low correlation can be attributed to the nature of rheumatoid arthritis as a condition.

Validity

All items had factor loadings $\geq$0.50 onto their relevant subscales (factor loadings $\geq$0.40 are generally considered as a meaningful; Floyd and Widaman, 1995). The identity subscale was found to adequately discriminate between general symptoms reported by participants and those perceived to be associated with their condition (t (15.94), $p \leq 0.01$). Subscales demonstrated evidence of predictive validity in relation to a measure of adjustment (see Moss-Morris et al., 2002 for a detailed description).

Rationale for use

The IPQ-R was chosen to measure the illness cognition domains described in the CS-SRM. The IPQ-R and its previous version (IPQ; Weinman, et al., 1996) are widely used in applied health psychology and findings from this thesis will be comparable with similar studies (French and Weinman, 2008).

Other measures of illness cognitions are available in the context of diabetes. One measure is the diabetes illness representation questionnaire (DIRQ; Skinner, et al., 2003) yet this measure was developed for use specifically among adolescents with Type 1 diabetes and therefore was not appropriate for use in the current sample. An alternative measure is the Personal Models of Diabetes Interview (PMDI; Hampson, et al., 1990; Hampson, et al., 1995). The PMDI takes a long time to complete (approximately 40 minutes; Hampson et al, 1995) and it was not practical to use it in addition to the other self-report questionnaires included in this study.

4.3.2 *The Beliefs about Medicines Questionnaire (BMQ)*

Description

The BMQ (Horne, et al., 1999) was used to measure participant's thoughts about their diabetes mediations.

The questionnaire consists of two parts: BMQ-specific and BMQ-general. Each part can be administered separately. Only the BMQ-specific component was used here. The BMQ-specific has two subscales:

i. medication necessity: measures participant's beliefs about the need for medication to manage their health condition.
ii. medication concerns: measures participant's perceived negative impact of taking medications (e.g. side effects, intrusiveness).

Each subscale consists of five items scored on a 5-point Likert scale with responses ranging from strongly disagree (one) to strongly agree (five). Items from each subscale are summed after relevant items are reversed scored. High scores on each subscale represent stronger beliefs in the treatment cognition domain being measured.

The psychometric properties of the BMQ were evaluated on a large sample of individuals with a long term condition (n=524) of which 99 participants had diabetes mellitus.

Reliability

Both medication necessity and concerns subscales demonstrated evidence of internal consistency among individuals with diabetes providing Cronbach alpha coefficients of 0.74 and 0.80 respectively. In addition, the subscales demonstrated evidence of test-retest reliability over a period of two weeks with Spearman correlation coefficients of 0.77 (necessity) and 0.76 (concerns).

Validity

Both subscales demonstrated evidence of construct validity as items had factor loadings of ≥0.58 onto their respective factors and no evidence of item cross-loadings using principle components analysis (PCA).

Subscales displayed evidence of both convergent validity (high correlations with measures of a similar construct; Bowling, 2002) and discriminant validity (low correlations with measures of a dissimilar construct; Bowling, 2002) in the expected manner with appropriately selected outcomes (see Horne et al., 1999 for a detailed description).

Rationale for use

There are measures of diabetes specific treatment beliefs including the Insulin Treatment Appraisal Scale (Snoek, et al., 2007) and Barriers to Insulin Treatment Questionnaire (Petrak, et al., 2007). However these measures are not frequently used by health psychology researchers, limiting their comparability with other research. In addition the scales relate specifically to insulin which is not always used in the management of Type 2 diabetes.

4.3.3 *The Well-Being Questionnaire (WBQ)*

Description

The Well-Being Questionnaire (Bradley, 1994) was used to measure emotional health.

The scale was developed among individuals with Type 1 and Type 2 diabetes to measure depression (six items), anxiety (six items), energy (four items), and positive well-being (six items). Poor emotional health in the context of this thesis was therefore conceptualised as two distinct constructs: i) depression and ii) anxiety.

The depression and anxiety subscales of the questionnaire were adapted from Zung's Self Rating Depression (Zung, et al., 1965) and Anxiety Scales (Zung, 1974). Items relating to the somatic symptoms of depression and anxiety were excluded from these subscales because of their symptomatic overlap with features of diabetes. The subscales reflect the cognitive-affective elements of mood and are not used as a diagnostic tool for depression and anxiety.

The energy and positive well-being subscales were developed from a pool of items generated by diabetologists and psychologists.

Items for each subscale are scored on a four point Likert scale with scores ranging from zero to three, a score of zero being consistent with "not at all" and a score of three being consistent with "all the time".

Items reflect a participant's mood within the time period "the past few weeks". Items are summed for each subscale after appropriate items have been reversed scored. A high score demonstrates greater endorsement of the concept measured by the subscale.

Reliability

Among a UK population with Type 2 diabetes subscales demonstrated high internal consistencies with Cronbach's alpha scores ranging from 0.70 to 0.88 across each subscale. A Cronbach's alpha score for the energy subscale was not reported by Bradley (1994) for patients with Type 2 diabetes as this subscale emerged at a later stage of the psychometric testing of the questionnaire. However among patients with Type 1 diabetes a Cronbach's alpha of 0.64 was reported for energy.

No test-retest correlation coefficients were reported by Bradley (1994).

Validity

Items as a whole demonstrated acceptable factor loadings (≥0.40) on the constructs that emerged as a result of a PCA performed by Bradley (1994) among individuals with Type 2 diabetes. However, two items hypothesised to be related to depression and two items predicted to load onto anxiety failed to demonstrate factor loadings ≥0.40 onto their hypothesised construct. Specifically two depression items loaded onto the anxiety component and the two anxiety items loaded onto the positive well-being component.

The factor loadings for the energy subscale were reported by Bradley (1994) only for individuals with Type 1 diabetes. Items demonstrated factor loadings ≥0.67.

Convergent validity with other measures of depression and anxiety was not investigated by Bradley (1994); however Pincus et al. (1997) found the depression and anxiety subscales to correlate relatively highly with those of the Hospital Depression and Anxiety Scale (HADS; Zigmond and Snaith, 1983) in patients with rheumatoid arthritis. Specifically the Pearson correlation coefficients for depression and anxiety were 0.63 and 0.69 respectively.

Since the publication of the 22 item WBQ, a revised 12-item questionnaire has been developed. The 12 item WBQ was generated in response to concerns about: i) reverse scored positively phrased items loading onto the positive well-being factor, ii) some items not loading onto their hypothesised factors, and iii) some items having high cross-loadings (Bradley, 2000; Riazi, et al., 2006). The 12 item WBQ consists of three four item subscales: negative well-being (generated from 2 items from the depression subscale and 2 items from the anxiety subscale), positive well-being, and energy. This factor structure has also been supported in two other studies (Mitchell and Bradley, 2001; Pouwer, et al., 1999).

Rationale for use

The WBQ was chosen to measure emotional health for a number of reasons. It excludes somatic symptoms of depression and anxiety from their respective subscales and therefore prevents participants from wrongly scoring highly on the constructs as a result of their diabetes. This permits greater validity in the measurement of depression and anxiety in the context of a long term condition. The inclusion of both depression and anxiety subscales

allowed both of these emotional health concepts to be explored in relation to illness and medication cognitions, and diabetes self-care.

The HADS (Zigmond and Snaith, 1983) also excludes somatic features from its measure and is widely used among individuals with physical long term conditions (Bjelland, et al., 2002). However the WBQ was preferred because it was specifically developed among individuals with diabetes and its psychometric properties have been robustly tested. In addition, chapter two showed that this measure was commonly used by health psychology researchers, thus allowing the findings of this thesis to be compared with similar research.

4.3.4 *Summary of Diabetes Self-Care Activities Scale (SDSCA)*

Description

The summary of diabetes self-care activities scale (SDSCA; Toobert, et al., 2000) was used to measure diabetes self-management behaviours.

The SDSCA consists of 12 items within which are seven subscales which measure adherence to:

i. General diet e.g. following a healthy eating plan (2 items)
ii. Specific diet e.g. decreased saturated fat consumption and increased intake of fruit and vegetables (2 items)
iii. Exercise (2 items)
iv. Self-monitoring of blood glucose (2 items)
v. Foot care (2 items)
vi. Medication adherence (1 item)
vii. Smoking (1 item)

Questionnaire items reflect a person's degree of self-care over the preceding seven days. Items are responded to by participants indicating the number of days per week they performed the specific behaviour and therefore responses range from zero to seven days. The smoking item has a different scoring method as participants provide a dichotomous yes/no response to whether they have smoked over the last seven days.

Toobert et al. (2000) do not advise summing all the SDSCA items to generate a total diabetes self-care score because of the distinct nature of each of the diabetes self-care

behaviours. However, a total diabetes self-care score was generated for the structural equation models to retain model parsimony (see chapters six, seven, and eight).

Toobert et al's (2000) justification for not calculating a total SDSCA score was based on low inter-correlations between the SDSCA subscales. However, low inter-correlations may reflect the discriminant validity of each of the subscales, which are embedded within a higher order construct of global diabetes self-management. Indeed, each of the behaviours included in the SDSCA are important to the overall management of diabetes (NICE, 2008). It was therefore considered justified to sum item responses across all subscales of the SDSCA to generate a total subscale named: global diabetes self-care. However the smoking item was excluded from the analysis because of its dichotomous measurement.

The psychometric properties of the SDSCA were evaluated by Toobert et al. (2000) by synthesising the findings of seven studies which had used the SDSCA with a combined sample of n=1988. The findings reported below are therefore the summary statistics presented by Toobert et al. (2000) from across the seven studies.

Reliability

As a whole subscales demonstrated evidence of adequate internal reliability (inter-item correlations mean r=0.47 across each of the seven subscales). However the specific diet subscale performed poorly with internal reliability scores ranging from r=0.07 to r= 0.23 across the seven studies. Toobert et al (2000) suggests using the items from this subscale separately.

All subscales demonstrated adequate test-retest reliability over a period of three to four months across the seven subscales (mean r=0.40).

Validity

Subscales demonstrated adequate concurrent validity with the appropriate outcome measures e.g. other measures of health behaviours (see Toobert et al., 2000 for a detailed account).

Rationale for use

Whilst other self-report measures of diabetes self-care are available for example the Diabetes Regimen Adherence Questionnaire (Brownlee-Duffeck, et al., 1987), the Self-Care Inventory-Revised (Weinger, et al., 2005), and Orme and Binik's (1989) self-care measure, the SDSCA was chosen because of its frequency of use among participants with diabetes permitting findings from the current research to be compared with other similar studies.

Diabetes self-care behaviours can also be measured by self-report diaries/records (Howat, et al., 1994) and pharmacy claims data for both the degree of medication adherence (Cohen, et al., 2010) and extent of self-monitoring of blood glucose (Evans, et al., 1999). However diaries/records are labour intensive for participants and require a high degree of literacy and competency for them to be correctly completed (Vitolins, et al., 2000). Whilst pharmacy/electronic data provide a more accurate account of participants' levels of self-care such an approach was not feasible with the resources available in the context of this PhD.

4.3.5 ***Demographic and clinical characteristics***

To describe the sampled population demographic and clinical characteristics were obtained.

Age, gender, and ethnicity were self-reported by participants in the questionnaire pack.

The following clinical characteristics were extracted from participant's electronic medical records:

i. *Glycosylated haemoglobin (HbA1c):* a measure of blood glucose concentration in the red blood cells over the preceding 12 weeks (NICE, 2008). Individuals with diabetes are encouraged to maintain HbA1c levels around 48 mmol/mol (6.5%) to 58mmol/mol (7.5%). HbA1c was treated as a continuous variable rather than dichotomising it into a measure of good vs poor long term blood glucose control to retain statistical power.

ii. *Body Mass Index (BMI) and weight*: Insulin resistance is both caused and exacerbated by obesity (NICE, 2008). A BMI of 18-25 is considered normal, 25-30 overweight/obese, ≥30 severely obese (World Health Organisation, 2000).

iii. *Blood pressure and total cholesterol*: Elevated blood pressure and irregular cholesterol levels are associated with insulin resistance and increased risk of cardiovascular disease (DeMott, et al., 2008; NICE, 2008; 2011). If a person's blood pressure is in excess of 140/80 (systolic/diastolic) they are considered to be hypertensive and require clinical intervention (NICE, 2008). Individuals with Type 2 diabetes should achieve total cholesterol targets of less than 4.0 mmol/litre (NICE, 2008).

iv. *Estimated Glomerular Filtration Rate (eGFR):* provides an indication of renal functioning, an eGFR of ≥90/1.73m² represents normal renal function in the absence of other renal complications (NICE, 2008).

v. *Diabetes duration:* Number of years diagnosed with diabetes mellitus.

vi. *Diabetes Medication Regimen:* Participants prescribed diabetes medications were coded as either: i) diet and/or oral hypoglycaemic agents or ii) injectable medications (in isolation or in combination with oral hypoglycaemic agents). This coding strategy was implemented based on the advice of diabetologists at the Manchester Diabetes Centre. Additionally, previous studies have shown that negative perceptions about insulin therapy, thus emphasising an important distinction between the two treatment modalities (Larkin, et al., 2008; Polonsky, et al., 2005b).

vii. *Diabetes complications:* the criteria outlined in Table 4.1 were used to categorise diabetes complications. The criteria were chosen based on discussions with Diabetologists at the Manchester Diabetes Centre. Their definitions compare with the notation system used on electronic medical records at the outpatient clinic. Participants received a score of zero (no complication present) or one (one or more complications present) per complication category. A participant's number of diabetes complication score could range from zero to seven.

viii. *Other health co-morbidities:* Non-diabetes specific complications were extracted from participant's electronic medical records and coded according to their membership to the International Classification of Disease (ICD-10; World Health Organization, 2010) categories. Participants received a score of zero (no co-morbidities present) or one (co-morbidities present irrespective of frequency) per ICD-10 disease category. The ICD-10 category nine: diseases of the circulatory system was excluded from the count of other co-morbidities as these conditions were considered to be diabetes related and classified under either the cardio-vascular or cerebro-vascular diabetes complication category. Equally category 15 pregnancy was excluded because of its lack of applicability. The disease categories outlined below were combined to generate broader disease categories because of their lack of observed frequency among the sampled population.

- Category 16-conditions originating in the perinatal period and category 17 congenital malformations, deformations and chromosomal abnormalities.
- Category 18-symptoms, signs and abnormal clinical and laboratory findings, category 19-injury, poisoning and certain other consequences of external cause, and category 20-external causes of morbidity and mortality.

A participant's number of co-morbidities score could range from zero to seventeen.

Table 4.1: Criteria used to code diabetes complications

Complication categories	Identification criteria from electronic medical records
Retinopathy and eye complications	Evidence of diabetic retinopathy reported in problem lists
	Evidence of receiving eye treatment at Manchester Royal Eye Hospital due to retinopathy
	Maculopathy
	Cataract
	Partially sighted/blind (reported as being due to diabetes)
Nephropathy	Microalbuminuria reported in problem lists
	Proteinuria reported in problem lists
	Chronic renal failure reported in problem lists or eGFR ≤ 30
	Dialysis
	Renal transplant
Metabolic	Hypoglycaemia requiring 3rd party intervention reported in problem list and/or text of letters, defined as more than 2 episodes per year.
	Hyopglycaemic unawareness reported in problem list
Neuropathy	Peripheral neuropathy reported in problem list or vibration perception threshold ≥ 25
	Autonomic neuropathy
	Gastroparesis/diarrhoea
	Postural hypotension
	Charcot neuroarthropathy (Charcot's foot)
	Foot ulcer past
	Foot ulcer present
	Amputation-toe
	Amputation- foot
	Amputation-leg
	Amputation-bilateral
Cardiovascular	Angina
	Myocardial Infarction
	Acute coronary syndrome
	Evidence of bypass surgery
	Heart failure or ejection fraction $\leq 30\%$
	Evidence of pacemaker

Table 4.1: continued

Complication categories	Identification criteria from electronic medical records
Cerebrovascular	Stroke
	Transient Ischemic Attack
Peripheral Vascular Disease	Intermittent claudication
	Absent foot pulses
	Evidence of arterial surgery to leg for vascular disease/peripheral angioplasty
	Aortic aneurysm
	Erectile dysfunction

The clinical characteristics outlined above were extracted from participants' electronic medical records at baseline and six months follow-up (for those participants who returned the follow-up questionnaire).

Data in relation to diabetes complications, other health co-morbidities, and diabetes treatment regimen were obtained at baseline only. It was assumed that these characteristics would stay relatively stable over a six month period. In addition, the number of participants experiencing changes in their disease states would not have been large enough to perform meaningful statistical analyses.

Clinical characteristics were extracted from electronic medical records for the date closest in proximity to the time of baseline recruitment and mail out of the follow-up questionnaire.

At baseline clinical outcome measures (e.g. HbA1c) were extracted from medical notes for a period of up to six months before the date of recruitment into the study and up to a maximum of one month after the baseline recruitment date. Any clinical outcome measured after this time point was deemed to be representative of a participant's follow-up clinical outcome score. Clinical measures obtained greater than six months after the follow-up recruitment date were not extracted.

4.4 *Statistical analyses*

The statistical analyses were performed in a number of phases each of which are described in detail in the relevant results chapter but are briefly outlined below:

i. Chapter five presents the baseline univariate and bivariate analyses and six months follow-up univariate analyses only.
ii. Chapter six presents the measurement models/confirmatory factor analyses performed on the self-report measures of cognitions, emotions, and diabetes self-care.
iii. Chapters seven, eight, and nine present the structural equation models of the directional relationships that exist between cognitions, poor emotional health, and diabetes self-care *or* HbA1c

Statistical analyses were carried out using either the Statistical Package for the Social Sciences (IBM SPSS Statistics, Version 19) or Analysis of Moments Structures (AMOS; Arbuckle, 2009).

Before analysing the data it was screened to test the assumptions of normality, to detect outliers, and to check for missing values.

Testing the assumptions of normality

Histograms of the plotted distributions of participants' scores on self-report questionnaire subscales and continuous clinical/demographic data were visually inspected in addition to evaluating the statistical tests of normality (skew, kurtosis).

The Kolmogorov-Smirnov test was used to test for the assumptions of normality. All self-report subscales and continuous demographic and clinical data were non-normally distributed excluding age and global levels of diabetes self-care (at baseline and follow-up), weight and blood pressure (follow-up only), and perceived consequences (baseline only). A copy of the normality tests can be found in Statistical Appendix A (only baseline distribution plots are presented because distributions were consistent at baseline and follow-up, excluding those for weight, blood pressure, and consequences where both distributions are shown).

Methods applied to statistical analyses to handle non-normal data

Because the majority of variables had non-normal distributions for consistency non-parametric tests were applied to all variables when univariate and bivariate tests were performed.

For multivariate tests findings were bootstrapped with 1000 re-samples and 95% percentile confidence intervals (Hayes, 2009; Mooney and Duval, 1993). Whilst other methods of generating bootstrapped confidence intervals are available percentile based estimates are valued for their simplicity (Mooney and Duval, 1993). Limited differences have been observed between computationally intensive methods of confidence interval generation and the simple percentile method (Hancock and Liu, 2012).

Detecting outliers

A Mahalanobis distance analysis was performed to identify if multivariate outliers were present in the data. All baseline self-report scores of cognitions (9 variables), emotions (4 variables), and global diabetes self-care were included in the test. A separate Mahalanobis distance test was performed substituting global diabetes self-care with Hba1c. This separate analysis was performed to explore the multivariate outliers in relation to the secondary research hypothesis (see Chapter Three, Section 3.2). Follow-up measures were not included in the analysis in order to include the full baseline sample; and high correlations between baseline and follow-up scores minimised any additional benefit from including the latter.

Using a p value of 0.001 as recommended by Tabachnick and Fidell (1996) only one participant was identified as having a Mahalanobis distance score of greater than 36.123 (the criterion chi-squared value with 14 degrees of freedom). This was for the model that included global diabetes self-care behaviours. None were found when the model included HbA1c. Because only one outlier was found it was retained in subsequent models and no sensitivity analyses were performed because the removal of one case would have limited effects.

Missing value analysis

Missing value analyses were performed to explore the extent of missing data at: baseline, six months follow-up, and at both time points for self-report questionnaire measures and demographic/clinical data. A detailed account of these findings can be found in Statistical Appendix A, Tables A1 and A2.

Analyses were performed at both the item and the subscale level. Item level missing value analyses were necessary for the purposes of structural equation modelling when multiple indicator item level latent variables were specified. All other statistical analyses were tested using subscale level data.

Item level missing data

The percentage of participants who had one or more items missing per subscale at baseline ranged from 2.7% to 8.4 % with the exception of the uncontrollable cause subscale of the IPQ-R and global measure of diabetes self-care which had 13% and 11.9% missing data respectively. The percentage of participants at 6 months follow-up who had item level missing data ranged from 26.4% to 32.2% however 25.7% of the missing data was because of the non-return of the follow-up questionnaire.

The percentage of participants with valid item scores on a subscale at both time points ranged from 59.8% to 72.4% across each subscale.

Subscale level missing data

When the degree of item level missing data is ≤ 33% per subscale the authors of the IPQ-R (Moss-Morris et al., 2002) recommend the use of mean imputation using syntax codes provided on the IPQ-R website. For consistency this approach was also used with the BMQ subscales (Horne et al., 2002) and WBQ subscales (Bradley, 1994). Mean imputation was not used with the identity subscale of the IPQ-R as this approach is not recommended by Moss-Morris et al. (2002). In addition it was not used with subscales that had ≤2 items per subscale or with the global measure of diabetes self-care because a person's level of adherence to diet may differ greatly from that of medication adherence for example.

After mean imputation the degree of subscale level missing data at baseline was ≤ 5% across all subscales (excluding subscales which did not have the mean imputed). Similarly, at 6 months follow-up the degree of missing data ranged from 25.7 % to 29.1% (excluding subscales which did not have the mean imputed). Once more, 25.7% of this missing data was because of the non-return of the follow-up questionnaire.

The number of participants with a valid score on a subscale at both baseline and six months follow-up ranged from 60.9% to 73.6%.

Demographic/clinical missing data

The extent of demographic and clinic level missing data at baseline ranged from 0% to 21.5% excluding total cholesterol of which 33.7% was missing at baseline. At six months follow-up the degree of missing data ranged from 25.7% to 48.0% and the proportion of participants who had a valid score on demographic and clinical characteristics at both time points ranged from 35.2% to 74.3%. The variability in the proportion of missing data on demographic and clinical variables was because of the reliance on this data being available in electronic medical records within an acceptable timeframe.

Methods applied to handle missing data

List wise deletion was used for each univariate statistic, multivariate regression, and structural equation models. Pairwise deletion was applied to correlation analyses because of the high volume of variables included in these tests[2].

A sensitivity analysis was performed on the final structural equation models to determine whether results differed once missing data had been imputed using the direct maximum likelihood estimation method.

Identifying variables for testing using SEM

Because the systematic review in chapter two showed that the relationships between diabetes cognitions, poor emotional health, and diabetes self-care had not been extensively tested in the literature longitudinally it was not possible to a priori determine the key

[2] These methods were applied once mean imputation had been performed on the self-report questionnaires for all analyses excluding SEM.

variables for testing using SEM. Thus variables were screened for inclusion using bivariate and multivariate regression analyses (described in detail in Chapter Seven, Section 7.2.1). Regression analyses were used for this initial screening phase because performing these analyses using SEM would have been overly laborious.

4.5 *Chapter summary*

This chapter has outlined the methodological design of the empirical work of this thesis. In brief:

- A longitudinal observation study with a six month time interval was conducted.
- Outpatients with Type 2 diabetes were consecutively recruited.
- Participants completed the following self-report measures at baseline and six months follow-up: Revised Illness Perception Questionnaire, Beliefs about Medicines Questionnaire, Well-being Questionnaire, and Summary of Diabetes Self-Care Activities Scale.
- Clinical characteristics were extracted from medical records
- The statistical assumptions of SEM were explored and missing value analyses performed.

5 *Results: baseline and follow-up descriptive statistics*

This chapter presents the recruitment results, univariate, and bivariate descriptive statistics performed on socio-demographic, clinical, and self-report questionnaire subscales at baseline. It also presents univariate analyses for the six month follow-up data, as well as mean change over time for the self-report measures of cognitions, emotions, diabetes self-care and HbA1c.

5.1 *Statistical analyses*

Univariate statistics

Descriptive univariate analyses for continuous socio-demographic, clinical characteristics, and subscale scores on self-report questionnaires are presented as means with standard deviations and as medians with the 25^{th} and 75^{th} percentile scores because of the non-normally distributed data. In the text only means and standard deviations are reported to follow usual convention for these measures.

Categorical data are presented as frequencies and percentages.

Bivariate analyses

At baseline, mean differences between continuous variables were explored with the Mann-Whitney U test. When associations between continuous variables were examined Spearman's rho correlations were calculated. Pearson Chi Square (χ^2; continuity correction; Pallant, 2005) statistic was used to study the statistical difference between two categorical variables.

Given the exploratory nature of bivariate analyses it was deemed acceptable to assume statistical significance at the level of $p \leq 0.05$ despite numerous comparisons (Feise, 2002).

Principal component analysis

A Principal Component Analysis (PCA) was performed on the 18 causal belief items in accordance with the recommendations of Moss-Morris et al. (2002). The theoretical constructs identified and their associated items were used to generate causal belief

subscales specific to individuals with Type 2 diabetes. Because of the preliminary nature of these subscales they were only explored descriptively at baseline.

Change over time analysis

Change scores for each participant were calculated by subtracting baseline scores from follow-up scores on self-report measures of cognitions, emotions, and diabetes self-care and for the biological outcome HbA1c. Descriptive statistics were performed to explore the degree of inter-individual change.

5.2 *Results*

5.2.1 *Number of participants approached/included at each stage of the study*

Figure 5.1 shows a flow chart of participants at each stage of the study.

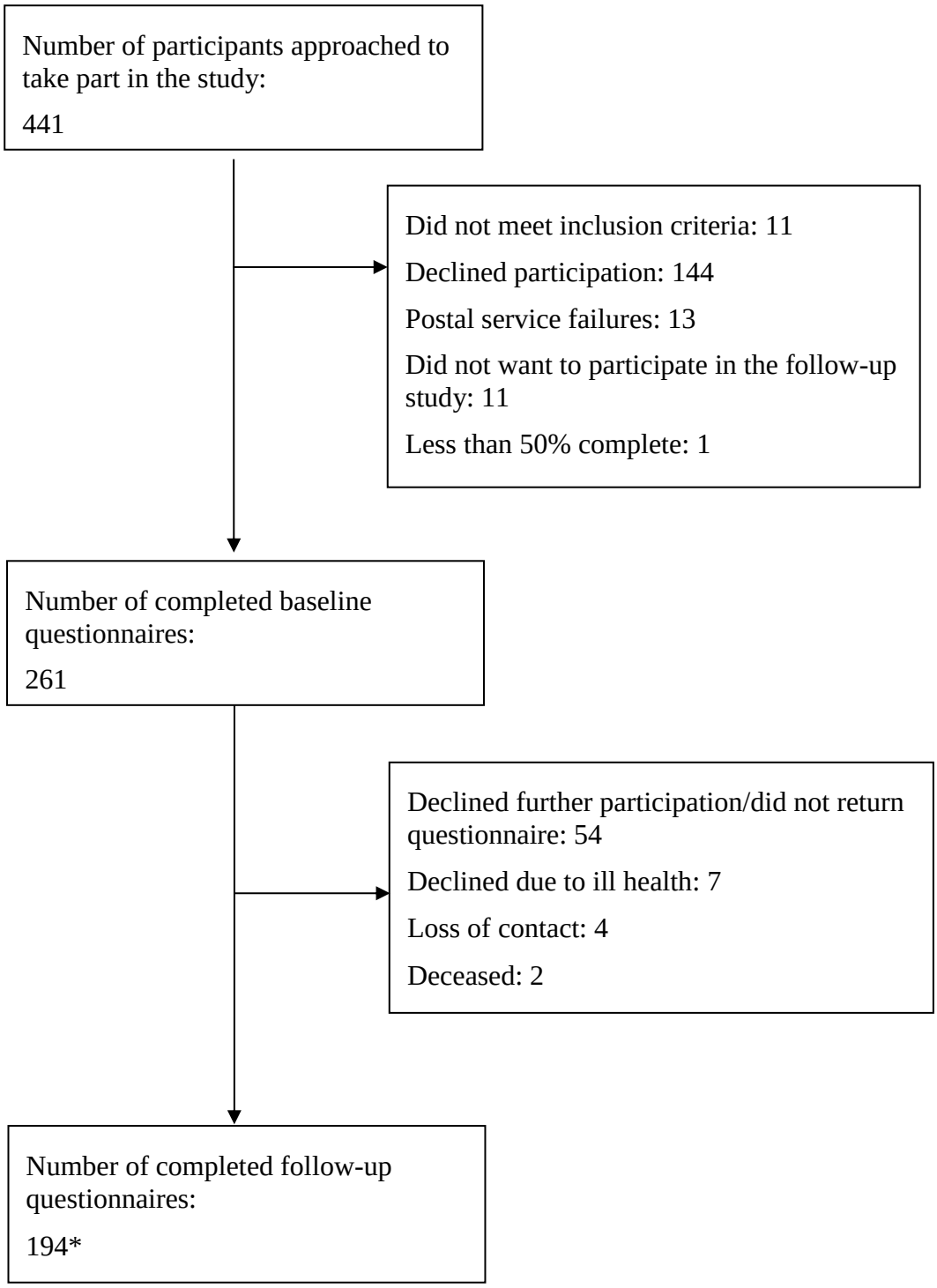
Of the 441 participants who had the information about the study explained to them by JH, 261 participants completed and fully consented into the study at baseline (59% response rate).

Before six months follow-up two participants died and of those remaining 194 out of 261 (74.3%) participants returned a second questionnaire. Reasons for non-completion at follow-up included: declined/questionnaire not returned (54 participants), participants felt too ill to take part (7 participants), and loss of contact (4 participants).

It was not possible to perform statistical analyses to compare demographic and clinical characteristics between those participants who agreed to take part in the study at baseline and those who refused because of ethical restrictions (e.g. this information could only be accessed once a participant had consented to take part in the study).

Participants who completed questionnaires at baseline and follow-up were compared with those participants who completed questionnaires at baseline only. These analyses are presented when six month follow-up analyses are reported.

Figure 5.1: Flow chart of participants recruited and retained at each stage of the study



*Based on number of returned questionnaires only. Missing value analyses for self-report measures and demographic/clinical data can be found in Statistical Appendix A Tables A1 and A2

5.3 *Univariate baseline demographic and clinical descriptive statistics*

Table 5.1 includes abbreviations used in all the tables presented in this section.

Table 5.1: Key to aid with the understanding of tables presented in chapter five

Abbreviation/Symbol	Definition
IQR	Inter-quartile range (reported for the 25th and 75th percentile).
N	Number
SD	Standard Deviation
*	$p \leq 0.05$
**	$p \leq 0.01$

Tables 5.2 and 5.3 display the descriptive statistics for demographic and clinical characteristics of participants who returned questionnaires at baseline.

At baseline 157 (60.2%) participants were male and the mean age of the sample was 62.3 years (SD 12.4). The majority of the sample were of a white ethnic background (n=170, 65.1%).

Participants mean diabetes duration was 14.4 years (SD 8.7). Nearly two thirds of the sample were on insulin/injection based treatment regimens either in isolation or in combination with oral hypoglycaemic agents (n=167, 64.0%).

Participant's mean HbA1c at baseline was 65.6 mmol/mol (SD 17.4; HbA1c as a percentage=8.2%) and was above the NICE (2008) recommended HbA1c target of 48 mmol/mol to 58 mmol/mol.

Participants mean body mass index (BMI) was in the severely obese category: 32.8 (SD 6.9) according to World Health Organisation (2010) guidelines. Mean blood pressure: 134.3/68.9 mmHg (SD 17.4/9.8) and mean total cholesterol: 4.0 mmol/litre(SD 1.1) were within an acceptable clinical range according to NICE (2008) guidelines. Participants mean level of renal function as approximated by their Estimated Glomerular Filtration Rate

(eGFR) demonstrated evidence of reduced kidney function: 61.4, 1.73m² (SD 24.7), relative to the guidelines provided by NICE (2008).

The mean number of diabetes complications per participant was 2.0 (SD 1.2), of which cardiovascular disease was the most common, occurring in 219 (83.9%) of participants at baseline. The mean number of other health complications was 1.5 (SD 1.3) of which problems with the endocrine system other than diabetes e.g. hyperthyroidism, were the most common, present in 98 (37.6%) of the participants at baseline.

Table 5.2: Demographic and clinical characteristics at baseline

Variable		Average/ Frequency	SD/IQR/%
Gender	Male	157	60.2
	Female	104	39.8
Age/years	mean	62.3	12.4
	median	62.0	55.0-72.0
Diabetes duration/years	mean	14.4	8.7
	median	13.4	8.0-19.0
Ethnicity	White	170	65.1
	Black	41	15.7
	Asian	43	16.5
	Mixed race	5	1.9
	Other/prefer not to say	2	0.77
Diabetes treatment regimen	Diet/oral hypoglycaemics	72	27.6
	Injections/Combination	167	64.0
	No access to medical records/missing data	22	8.4
Clinical outcomes			
HbA1c mmol/mol	mean	65.6	17.4
	median	61.7	54.1-75.5
Weight/Kg	mean	92.3	22.1
	median	89.2	75.7-108.1
BMI	mean	32.8	6.9
	median	31.9	27.8-37.0
Systolic/mmHg	mean	134.3	17.4
	median	134.0	123.8-142.0
Diastolic/mmHg	mean	68.9	9.8
	median	68.0	63.00-74.00
eGFR/1.73²	mean	61.4	24.7
	median	67.0	42.5-86.0
Total cholesterol/mmol/l	mean	4.0	1.10
	median	3.8	3.3-4.7

Table 5.3: Frequencies of diabetes complications and other co-morbidities extracted from electronic medical records at baseline (n=245)

Variable		Average/Frequency	SD/IQR/%
Number of complications	mean	2.0	1.2
	median	2.0	1.0-3.0
Neuropathy		95	36.4
Nephropathy		51	19.5
Retinopathy		74	28.4
Cardiovascular		219	83.9
Cerebrovascular		17	6.5
Peripheral vascular disease		40	15.3
Metabolic		1	0.38
Number of other co-morbidities	mean	1.5	1.3
	median	1.0	1.0-2.0
Endocrine		98	37.6
Respiratory		29	11.1
Musculoskeletal		59	22.6
Genitourinary		26	10.0
Other		97	37.2

5.4 *Principal components analyses on IPQ-R causal items*

Before reporting univariate and bivariate statistics for the self-report questionnaires a principal components analysis (PCA) with direct oblimin rotation (delta=0; Jennrich and Sampson, 1966) was performed using the 207 participants who provided complete data on the IPQ-R causal belief items retained for PCA at baseline. This allowed causal beliefs specific to Type 2 diabetes to be identified.

Items were obliquely rotated based on the theoretical assumption that the causal components generated would be correlated as was found by Moss-Morris et al. (2002). Direct oblimin extraction method was used following the recommendations of Field (2009).

Preliminary PCA analysis checks

The preliminary assumptions of PCA were explored including normality, adequate sample size, absence of multicollinearity, and item correlation coefficients ≥ ± 0.30 (Field, 2009; Hutcheson and Sofroniou, 1999). All assumptions were met, but causal beliefs relating to the hereditary nature of diabetes and ageing did not demonstrate correlation coefficients above ±0.30 with any other causal items. These two items were excluded from the PCA based on the recommendations of Hutchinson and Sofroniou (1999; See Statistical Appendix A, Table A3 for correlation matrix). A Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was computed for the remaining 16 causal items (a test of whether likely relationships exist between items to be factor analysed; Field, 2009). The overall KMO statistic was 0.81 exceeding the minimum threshold of 0.50. Inspection of item level KMO statistics also exceeded the 0.50 threshold.

Main analysis findings

Eigenvalues of greater than one were used to determine the number of components to extract. Consistent with Moss-Morris et al. (2002) four components had eigenvalues greater than one and were obliquely rotated.

The initial obliquely rotated solution demonstrated that item nine "my mental attitude" had factor loadings of greater than 0.40 on more than one component. This item was removed from the PCA to generate conceptually distinct components (Floyd and Widaman, 1995). Findings from the initial four component, 16 item obliquely rotated solution can be found in Statistical Appendix A, Tables A4 to A5. The preliminary assumptions of the PCA remained unchanged once item nine had been removed from the analysis.

The eigenvalues resulting from the PCA of the remaining 15 items were examined and are presented in Table 5.4. Four components demonstrated eigenvalues greater than one and cumulatively accounted for 60.0% of the total variance in the sample data. These four components were obliquely rotated.

After oblique rotation all items loaded exclusively onto one component with factor loadings ≥ 0.40. Item factor loadings onto the pattern matrix (whereby factor loadings are regression coefficients, and thus represent the independent contribution of an item to the component; Field, 2009) are presented in Table 5.5.

Six items loaded onto the first component labelled *uncontrollable cause,* items included: altered immunity, pollution in the environment, poor medical care in past, chance or bad luck, my personality, and germ or virus.

Two items demonstrated high factor loadings on the second component and was labelled *lifestyle;* items included diet or eating habits and my own behaviour.

The third component was labelled *risk factors* and included the following three items: smoking, alcohol, and accident or injury.

The final component generated was labelled *psychological attributions* and included four items: stress or worry, family problems or worries, my emotional state, and overwork.

Based on the recommendations of Pedhazur and Schmelkin (1991) component scores were allowed to correlate to reflect an assumption that the causal components are correlated. Two components demonstrated evidence of inter-correlations ≥0.30 (uncontrollable cause, and psychological attributions), thus justifying the use of oblique rotation (see Table 5.6).

The Cronbach alpha coefficients for each of the components were acceptable ranging from α=0.69 to α=0.81 across the four components. However examination of the *Cronbach's alpha if item deleted statistic* demonstrated that if item 16 "accident or injury" were deleted from the *risk factor* subscale the Cronbach's alpha coefficient would increase from 0.72 to 0.79. Given this finding it was removed from the subscale. The PCA was repeated on the remaining 14 items and findings were consistent with those presented in Tables 5.4 to 5.6. (See Statistical Appendix A, Tables A6 to A8). The size of the factor loadings differed marginally but all remained ≥0.40 onto their respective component.

Given that ageing and hereditary did not demonstrate correlations ≥0.30 with other causal IPQ-R items, yet both are known biological causes of diabetes (International Diabetes Federation, 2011), they were included as standalone causal items in subsequent analyses.

Table 5.4: Eigenvalues and variance explained for each component in the IPQ-R causal item PCA

Component	Eigenvalue	% of Variance	Cumulative %
1	**4.6**	**30.6**	**30.6**
2	**1.8**	**12.0**	**42.6**
3	**1.5**	**10.1**	**52.6**
4	**1.1**	**7.4**	**60.0**
5	0.9	6.2	66.3
6	0.9	6.0	72.2
7	0.7	4.8	77.0
8	0.7	4.4	81.3
9	0.6	3.7	85.0
10	0.5	3.6	88.6
11	0.5	3.1	91.6
12	0.4	2.9	94.51
13	0.3	2.2	96.7
14	0.3	1.8	98.5
15	0.2	1.5	100.0

Table 5.5: Principal components analysis pattern matrix of the 15 IPQ-R causal items.

	Components			
Items	I	II	III	IV
Uncontrollable cause (α=0.72)				
Altered immunity	**0.76**	.04	-0.02	0.11
Pollution in the environment	**0.65**	-0.15	-0.05	-0.13
Poor medical care in my past	**0.63**	0.13	0.02	-0.06
Chance or bad luck	**0.61**	-0.08	0.14	0.01
My personality	**0.51**	0.24	-0.20	-0.05
Germ or virus	**0.42**	-0.20	-0.18	-0.21
Lifestyle (α=0.69)				
Diet or eating habit	-0.04	**0.86**	0.01	0.04
My own behaviour	0.01	**0.85**	-0.02	-0.08
Risk factors (α=0.72)				
Smoking	-0.05	-0.07	**0.92**	0.04
Alcohol	-0.11	0.13	**0.88**	0.02
Accident or injury	0.33	-0.07	**0.52**	-0.13
Psychological Attributions (α=0.81)				
Stress or worry	-0.18	-0.08	0.04	**0.93**
Family problems or worries	-0.00	0.07	-0.10	**0.82**
My emotional state	0.27	0.15	0.01	**0.68**
Overwork	0.28	0.13	0.05	**0.52**

Table 5.6: Correlation matrix between identified IPQ-R causal components

Components	I	II	III
I. Uncontrollable cause			
II. Lifestyle	.04		
III. Risk factors	.20	.13	
IV. Psychological attributions	.43	.12	.27

5.5 *Baseline univariate statistics: diabetes cognitions, well-being subscales, and diabetes self-care*

Table 5.7 displays the baseline univariate descriptive statistics for participant's scores on the self-report questionnaires.

Participant's baseline mean illness and medication cognition scores were largely above the mid-point of the scale (3.0), demonstrating a stronger belief in the concept being measured. However, mean sub-scale scores for timeline cyclical, emotional representations, and medication concerns were just below the scale mid-point. For the identity subscale, participants identified a mean of 3.9 (SD 3.3) symptoms associated with their diabetes.

Baseline mean scores on the newly generated causal subscales demonstrated that the most strongly endorsed causal factors of diabetes were lifestyle and hereditary beliefs, with a mean of 3.2 (SD 1.1) and 3.4 (SD 1.4) respectively. A belief that diabetes was caused by risk factors (e.g. alcohol) was the least firmly held belief with a mean score of 2.0 (SD 1.0). These findings are consistent with participants free text responses on the causal subscale. A total of 76 participants (29.1%) ranked hereditary/genetics as the most important cause of their diabetes, 57 participants (21.8%) ranked diet as the primary cause, and 36 participants (13.8%) reported they believed diabetes was due to other causes not listed on the causal IPQ-R items. The most commonly cited other causes were being overweight and medications, with 10 (3.8%) participants each citing these as the main cause. A summary of the frequencies of participants ranked primary, secondary, and tertiary causes of diabetes are provided in Table 5.8.

At baseline participants' mean scores for symptoms of depression and anxiety on the WBQ were low, scoring 4.9 (SD 3.8) and 5.5 (SD 4.3) respectively out of a possible maximum score of 18 for each subscale (low scores indicate fewer depression and anxiety

symptoms). Participant's energy levels were at the scale mid-point with a mean score of 5.9 (SD 3.2) out of a potential maximum score of 12. Finally participants demonstrated relatively high levels of positive well-being scoring a mean of 11.2 (SD 4.8) out of possible score of 18.

On average participants' baseline levels of adherence to each diabetes self-care behaviour category and global diabetes self-care was often ≥ four days per week. Participants reported the highest levels of adherence to their medication regimen (mean=6.8 days, SD 1.1). However levels of adherence to exercise (mean=2.3 days, SD 2.3) and foot care (mean 3.6 days, SD 2.6) were lower than for the remaining health behaviours.

Table 5.7: Means and standard deviations of diabetes cognitions, well-being subscales and diabetes self-care behaviours

Variables		Average	SD/IQR
Revised Illness Perception Questionnaire			
Identity	mean	3.9	3.3
	median	3.0	1.0-6.0
Timeline acute/chronic	mean	4.2	0.7
	median	4.3	3.8-4.8
Timeline cyclical	mean	2.9	0.1
	median	3.0	2.0-3.5
Consequences	mean	3.3	0.8
	median	3.3	2.7-3.8
Personal control	mean	3.9	0.7
	median	4.0	3.5-4.5
Treatment control	mean	3.6	0.6
	median	3.6	3.2-4.0
Illness coherence	mean	3.6	0.9
	median	3.8	3.0-4.0
Emotional representations	mean	2.7	1.0
	median	2.6	2.0-3.5
Uncontrollable cause	mean	2.0	0.6
	median	2.0	1.5-2.5
Lifestyle	mean	3.2	1.1
	median	3.0	2.5-4.0
Risk factors	mean	2.0	1.0
	median	2.0	1.0-2.5
Psychological attributions	mean	2.4	0.1
	median	2.3	1.8-3.2
Hereditary	mean	3.4	1.4
	median	4.0	2.0-5.0
Ageing	mean	2.7	1.3
	median	3.0	1.0-4.0
Beliefs about Medicines Questionnaire			
Medication necessity	mean	4.1	0.8
	median	4.0	3.6-4.8
Medication concerns	mean	2.8	0.1
	median	2.8	2.2-3.6

Table 5.7: continued

Variables		Average	SD/IQR
Well-being questionnaire			
Depression	mean	4.9	3.8
	median	4.0	2.0-7.2
Anxiety	mean	5.5	4.3
	median	5.0	2.0-9.0
Energy	mean	5.9	3.2
	median	6.0	3.0-8.0
Positive well-being	mean	11.2	4.8
	median	12.0	7.5-15.0
Summary of Diabetes Self-are Activity Scale			
General diet	mean	4.8	2.1
	median	5.0	3.9-7.0
Specific diet (fruit & veg)	mean	4.5	2.3
	median	5.0	3.0-7.0
Specific diet (saturated fat)	mean	4.4	2.0
	median	5.0	3.0-6.0
Exercise	mean	2.3	2.3
	median	2.0	0.0-4.0
Self-monitoring of blood glucose	mean	4.5	2.6
	median	5.0	2.0-7.0
Foot care	mean	3.6	2.6
	median	3.5	1.0-6.0
Medication adherence	mean	6.8	1.1
	median	7.0	7.0-7.0
Global diabetes self-care	mean	4.2	1.2
	median	4.3	3.4-5.1

Table 5.8: Frequencies of participants ranked primary, secondary and tertiary causes of diabetes at baseline

Self-reported causes	Primary ranked cause		Secondary ranked cause		Tertiary ranked cause	
	Frequency	%	Frequency	%	Frequency	%
Baseline						
Stress	31	11.9	27	10.3	10	3.8
Hereditary	76	29.1	24	9.2	13	5.0
Diet	57	21.8	47	18.0	21	8.1
Other (IPQ-R listed causes)	35	13.4	71	27.2	90	34.5
Other (Non-IPQ-R listed causes)	36	13.8	21	8.1	17	6.5
Medication	10	3.8	1	0.4	1	0.4
Weight	10	3.8	13	5.0	4	1.5
Co-morbidity	7	2.7	2	0.8	3	1.2
Other	9	3.5	5	1.9	9	3.5
Valid N	235	90.0	190	72.8	151	57.9

5.6 *Bivariate baseline analyses*

5.6.1 *The relationship between demographic variables and diabetes cognitions, well-being subscales, and diabetes self-care*

Bivariate exploratory analyses were performed to examine the relationship between socio-demographic variables and cognitions, emotions, and diabetes self-care behaviours. Results are presented in Tables 5.9 to 5.16.

Gender

Female participants had statistically significant lower beliefs in treatment control, illness coherence, and believed that diabetes was less likely to be caused by risk factors compared with male participants. Female participants also demonstrated increased emotional representations of diabetes, medication concerns, and a greater belief in the hereditary nature of diabetes relative to males. In relation to the well-being questionnaire subscales female participants had increased scores on depression and anxiety and lower levels of energy and positive well-being compared with males. The only subscale that males scored higher on than females was self-reported levels of exercise. No other differences were observed (see Tables 5.9 and 5.10).

Ethnicity

Participants of a non-white ethnic background had statistically significant higher beliefs in identity and psychological causal attributions compared with participants of a white ethnic background. In addition, individuals of a non-white ethnic background had lower beliefs in the chronic nature of diabetes, perceptions of personal control, and were less likely to believe that diabetes was caused by either ageing or hereditary factors. No statistically significant differences were observed between participants of white and non-white ethnic background on all diabetes well-being questionnaire subscales. Participants of a white ethnic background reported a higher frequency of self-monitoring of blood glucose and higher levels of medication adherence compared with participants of a non-white ethnic background (See Tables 5.11 to 5.12).

Table 5.9: Relationship between gender and cognition domains

Variables		Male Average	SD/IQR	Female Average	SD/IQR	z [3]	p value
Identity	mean	3.7	3.2	4.3	3.5	-1.3	.19
	median	3.0	1.0-5.0	4.0	2.0-4.0		
Timeline acute/chronic	mean	4.2	0.7	4.2	0.8	0.0	.98
	median	4.3	3.8-4.8	4.5	3.9-4.8		
Timeline cyclical	mean	2.8	0.9	3.0	1.0	-1.3	.20
	median	2.8	2.0-3.5	3.0	2.1-3.8		
Consequences	mean	3.3	0.8	3.2	0.9	-0.2	.88
	median	3.3	2.7-3.8	3.2	2.7-4.0		
Personal control	mean	4.0	0.7	3.9	0.7	-0.9	.35
	median	4.0	3.5-4.5	3.9	3.3-4.3		
Treatment control	mean	3.6	0.6	3.4	0.6	-2.2	.03*
	median	3.6	3.2-4.0	3.5	3.0-3.8		
Illness coherence	mean	3.7	0.9	3.4	1.0	-2.0	.04*
	median	3.8	3.2-4.2	3.6	2.8-4.0		
Emotional representations	mean	2.5	1.0	3.0	1.0	-3.5	.00**
	median	2.3	1.9-3.2	3.0	2.2-3.8		
Medication necessity	mean	4.2	0.7	4.0	0.9	-0.9	.39
	median	4.2	3.8-4.8	4.0	3.6-4.8		
Medication concerns	mean	2.7	1.0	3.0	1.0	-2.6	.01**
	median	2.8	2.0-3.4	3.0	2.4-3.8		
Uncontrollable cause	mean	2.0	0.7	2.1	0.7	-1.0	.30
	median	2.0	1.5-2.5	2.0	1.7-2.5		
Lifestyle	mean	3.3	1.0	3.1	1.1	-1.1	.29
	median	3.5	2.5-4.0	3.0	2.4-4.0		
Risk factors	mean	2.1	1.1	1.8	0.9	-2.0	.05*
	median	2.0	1.0-3.0	2.0	1.0-2.0		
Psychological attributions	mean	2.4	1.0	2.6	1.0	-1.3	.18
	median	2.3	1.8-3.0	2.5	1.8-3.3		
Hereditary	mean	3.3	1.4	3.6	1.4	-1.9	.05*
	median	4.0	2.0-4.0	4.0	2.0-5.0		
Ageing	mean	2.7	1.3	2.7	1.3	-0.3	.78
	median	3.0	2.0-4.0	3.0	1.0-4.0		

[3] Mann-Whitney U Test non-parametric equivalent of the T test

Table 5.10: Relationship between gender and both the well-being subscales and diabetes self-care behaviours

Variables		Male Average	SD/IQR	Female Average	SD/IQR	z	p value
Depression	mean	4.4	3.5	5.6	4.0	-2.4	.02*
	median	4.0	1.2-7.0	5.0	2.0-8.9		
Anxiety	mean	4.8	3.9	6.5	4.7	-2.8	.00**
	median	4.0	1.0-8.0	6.0	2.3-10.0		
Energy	mean	6.4	3.0	5.1	3.3	-3.2	.00**
	median	6.0	4.0-8.0	5.0	3.0-8.0		
Positive well-being	mean	12.1	4.4	9.9	5.1	-3.4	.00**
	median	12.0	9.0-16.0	10.0	6.0-14.0		
General diet	mean	4.9	2.0	4.8	2.2	0.0	.99
	median	5.3	4.0-7.0	5.0	3.5-7.0		
Specific diet (fruit& veg)	mean	4.4	2.3	4.7	2.3	-1.0	.32
	median	5.0	3.0-7.0	5.0	3.0-7.0		
Specific diet (fat consumption)	mean	4.4	1.9	4.5	2.2	-1.1	.26
	median	4.0	3.0-6.0	5.0	3.0-6.0		
Exercise	mean	2.6	2.3	1.9	2.2	-2.3	.02*
	median	2.5	0.0-4.0	1.0	0.0-3.5		
Self-monitoring of blood glucose	mean	4.5	2.6	4.6	2.7	-0.4	.73
	median	5.0	2.5-7.0	5.5	2.0-7.0		
Foot care	mean	3.8	2.5	3.3	2.7	-1.4	.18
	median	3.5	1.5-6.0	3.5	0.5-6.0		
Medication adherence	mean	6.8	1.1	6.7	1.1	-0.3	.78
	median	7.0	7.0-7.0	7.0	7.0-7.0		
Global self-care	mean	4.3	1.3	4.1	1.1	-1.4	.17
	median	4.4	3.5-5.3	4.3	3.2-4.9		

Table 5.11: Relationship between ethnicity and cognition domains

Variables		White Average	SD/IQR	Non-White Average	SD/IQR	z	p value
Identity	mean	3.6	3.3	4.4	3.3	-1.9	.05*
	median	3.0	1.0-5.0	4.0	2.0-7.0		
Timeline acute/chronic	mean	4.3	0.7	4.0	0.8	-2.8	.01**
	median	4.5	3.8-4.8	4.2	3.7-4.7		
Timeline cyclical	mean	2.9	0.9	2.9	1.0	-0.7	.46
	median	3.0	2.0-3.5	3.0	2.3-3.8		
Consequences	mean	3.2	0.8	3.3	0.9	-0.7	.51
	median	3.2	2.7-3.8	3.3	2.7-4.0		
Personal control	mean	4.0	0.7	3.8	0.8	-2.4	.02*
	median	4.0	3.7-4.5	3.8	3.2-4.3		
Treatment control	mean	3.5	0.6	3.6	0.7	-0.5	.65
	median	3.6	3.2-4.0	3.6	3.2-4.0		
Illness coherence	mean	3.6	0.9	3.5	0.9	-0.5	.62
	median	3.8	3.0-4.0	3.6	3.0-4.1		
Emotional representations	mean	2.7	1.0	2.7	1.0	-0.4	.67
	median	2.5	2.0-3.5	2.7	2.0-3.5		
Medication necessity	mean	4.2	0.8	4.0	0.8	-1.4	.15
	median	4.2	3.8-4.8	4.0	3.6-4.6		
Medication concerns	mean	2.8	1.0	2.9	0.9	-0.8	.43
	median	2.8	2.0-3.6	3.0	2.2-3.4		
Uncontrollable cause	mean	2.1	0.7	2.0	0.7	-0.9	.40
	median	2.0	1.5-2.5	2.0	1.5-2.3		
Lifestyle	mean	3.2	1.1	3.1	1.1	-1.1	.29
	median	3.0	2.5-4.0	3.0	2.0-4.0		
Risk factors	mean	2.0	1.0	2.0	1.1	-0.1	.91
	median	2.0	1.0-2.5	2.0	1.0-2.5		
Psychological attributions	mean	2.3	1.0	2.6	1.0	-2.2	.03
	median	2.3	1.8-3.0	2.5	2.0-3.4		
Hereditary	mean	3.2	1.4	3.8	1.2	-3.4	.00**
	median	4.0	2.0-4.0	4.0	3.0-5.0		
Ageing	mean	2.8	1.3	2.5	1.2	-2.1	.03*
	median	3.0	2.0-4.0	2.0	1.0-4.0		

Table 5.12: Relationship between ethnicity and both the well-being subscales and diabetes self-care behaviours

Variables		White Average	SD/IQR	Non-White Average	SD/IQR	z	p value
Depression	mean	5.0	3.7	4.6	3.9	-1.1	.28
	median	5.0	2.0-8.0	4.0	1.5-7.0		
Anxiety	mean	5.5	4.4	5.5	4.1	-0.2	.82
	median	5.0	2.0-9.0	4.0	2.0-9.0		
Energy	mean	5.7	3.2	6.2	3.1	-1.1	.25
	median	6.0	3.0-8.00	6.0	4.0-8.0		
Positive well-being	mean	11.2	5.0	11.3	4.6	-0.1	.93
	median	12.0	8.0-15.0	12.0	7.0-15.0		
General diet	mean	5.0	2.0	4.6	2.2	-1.4	.15
	median	5.5	4.0-7.0	5.0	3.5-7.0		
Specific diet (fruit& veg)	mean	4.6	2.4	4.4	2.2	-0.9	.36
	median	5.0	3.0-7.0	5.0	3.0-7.0		
Specific diet (fat consumption)	mean	4.4	2.1	4.5	1.9	-0.3	.80
	median	5.0	3.0-6.0	5.0	3.00-6.0		
Exercise	mean	2.1	2.2	2.6	2.3	-1.4	.15
	median	1.5	0.0-3.5	2.0	0.0-4.5		
Self-monitoring of blood glucose	mean	4.9	2.5	3.9	2.7	-3.0	.00**
	median	6.0	3.0-7.0	4.0	1.1-7.0		
Foot care	mean	3.5	2.7	3.7	2.4	-0.4	.66
	median	3.5	1.0-6.6	3.5	2.0-6.0		
Medication adherence	mean	6.8	1.1	6.7	1.0	-2.2	.03*
	median	7.0	7.0-7.0	7.0	7.0-7.0		
Global self-care	mean	4.2	1.2	4.1	1.3	-0.8	.44
	median	4.4	3.5-5.2	4.2	3.2-5.1		

Age

Increased age was associated with increased scores on the following subscales: energy, positive well-being, and adherence to general diet, and medication regimens. In contrast, increased age correlated negatively with the following subscales: identity, consequences, emotional representations, medication concerns, psychological attributions, and a belief in the hereditary nature of diabetes (See tables 5.13 to 5.16).

5.6.2 *The relationship between diabetes complications/health co-morbidities and diabetes cognitions, well-being subscales, and diabetes self-care*

Spearman's correlations were performed between the number of diabetes complications and the number of other health co-morbidities and each cognition, WBQ, and SDSCA subscale (See Tables 5.13 to 5.16).

An increased number of diabetes complications correlated positively with the following subscales: energy, positive well-being, and foot care. In contrast, the number of diabetes complications correlated negatively with: personal control, emotional representations, medication concerns, psychological causal attributions, and lifestyle. An increased number of other co-morbidities correlated positively with only psychological causal attributions and anxiety.

Table 5.13: Correlation matrix of illness cognitions and continuous demographic/clinical variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Age	1.00																	
2. Duration	.45**																	
3. Weight	-.20**	-.11																
4. BMI	-.19**	-.11	.86**															
5. Systolic	.23**	-.01	.17*	.17*														
6. Diastolic	-.16*	-.35**	.29**	.17*	.49**													
7. HbA1c	-.28**	-.02	.16*	.13	-.01	.17*												
8. eGFR	-.61**	-.32**	.06	.06	-.23**	.00	.30**											
9. Total cholesterol	-.21**	-.25**	-.09	.00	.01	.21*	.20*	.17*										
10. N diabetes complications	.38**	.40**	-.06	-.14*	.14	-.04	-.09	-.49**	-.19*									
11. N co-morbidities	.07	.06	.06	.14*	-.02	-.05	-.05	-.08	.06	.03								
12. Identity	-.19**	-.15*	.10	.17*	-.02	.07	.25**	.22**	.07	-.13	-.01							
13. Timeline acute/chronic	.05	.19**	-.01	-.06	.00	-.10	.05	.04	-.09	.07	.09	.04						
14. Timeline cyclical	-.10	.03	-.06	.01	.03	.01	.23**	.16*	.09	.02	.10	.23**	.01					
15. Consequences	-.19**	.09	.02	.06	-.01	.00	.16*	.17*	.08	-.04	.01	.39**	.24**	.32**				
16. Personal control	-.11	-.02	.04	.02	.07	.13	-.14*	.11	-.01	-.13*	-.03	-.16*	.11	-.23**	-.02			
17. Treatment control	-.08	-.13	.07	.00	.08	.14	.04	.10	-.08	-.12	-.11	-.05	-.12	-.11	-.09	.42**		
18. Illness coherence	.01	.04	.01	-.08	-.01	.08	-.15*	-.01	-.14	.06	.04	-.27**	.13*	-.38**	-.20**	.44**	.23**	
19. Emotional representations	-.30**	-.19**	.04	.14*	.03	.14*	.28**	.22**	.22**	-.13*	.05	.49**	-.08	.33**	.48**	-.25**	-.13*	-.43**

Table 5.14: Correlation matrix of causal cognitions, medication cognitions and continuous demographic/clinical variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Age																		
2. Duration	.45**																	
3. Weight	-.20**	-.11																
4. BMI	-.19**	-.11	.86**															
5. Systolic	.23**	-.01	.17*	.17*														
6. Diastolic	-.16*	-.35**	.29**	.17*	.49**													
7. HbA1c	-.28**	-.02	.16*	.13	-.01	.17*												
8. eGFR	-.61**	-.32**	.06	.06	-.23**	.00	.30**											
9. Total cholesterol	-.21**	-.25**	-.09	.00	.01	.21*	.20*	.17*										
10. N diabetes complications	.38**	.40**	-.06	-.14*	.14	-.04	-.09	-.49**	-.19*									
11. N co-morbidities	.07	.06	.06	.14*	-.02	-.05	-.05	-.08	.06	.03								
12. Medication necessity	.08	.17**	.00	.01	.06	-.03	.06	-.01	-.18*	.11	.11							
13. Medication concerns	-.21**	-.15*	.06	.15*	-.03	.06	.24**	.15*	.24**	-.15*	.01	-.03						
14. Uncontrollable cause	.05	-.07	-.04	.04	.07	.07	.11	.04	.19*	.01	.07	-.01	.36**					
15. Risk factors	.01	-.19**	.10	.04	-.01	.07	-.02	.06	-.04	-.07	-.02	-.02	.16*	.25**				
16. Psychological attributions	-.13*	-.10	-.07	.04	-.05	.02	.23**	.17*	.20**	-.16*	.13*	.01	.46**	.50**	.28**			
17. Lifestyle	-.27**	-.11	.17*	.15*	.13	.18*	.11	.28**	.12	-.17**	-.08	-.08	.13*	.05	.16*	.18**		
18. Hereditary	-.20**	.00	-.06	.01	-.13	-.09	.08	.25**	.00	-.11	-.04	.21**	.06	.02	.08	.10	-.08	
19. Ageing	.11	-.05	.00	.05	.02	.00	-.08	-.01	-.12	.05	.04	-.02	.11	.35**	.23**	.23**	.12	.05

Table 5.15: Correlation matrix of well-being subscales and continuous demographic and clinical variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Age														
2. Duration	.45**													
3. Weight	-.20	-.11												
4. BMI	-.19	-.12	.86**											
5. Systolic	.23**	-.01	.17*	.18**										
6. Diastolic	-.16	-.35	.29**	.18**	.49**									
7. HbA1c	-.28	-.02	.16*	.13	-.01	.17*								
8. eGFR	-.61	-.32	.06	.06	-.23	.00	.30**							
9. Total cholesterol	-.21	-.25	-.09	.00	.01	.21*	.20*	.17*						
10. N diabetes complications	.38**	.41**	-.06	-.13	.15*	-.03	-.09	-.49	-.20					
11. N co-morbidities	.07	.06	.06	.13	-.02	-.05	-.05	-.08	.06	.03				
12. Depression	-.24	-.06	.08	.16*	-.07	.00	.17*	.20**	.12	-.25	.12			
13. Anxiety	-.30	-.12	.08	.14*	-.07	.01	.22**	.30**	.12	-.24	.15*	.71**		
14. Energy	.20**	.03	-.10	-.21	.07	.04	-.27	-.16	-.02	.17*	-.12	-.67	-.64	
15. Positive well-being	.26**	.08	-.02	-.14	.11	-.01	-.19	-.21	-.15	.24**	-.14	-.73	-.63	.70**

Table 5.16: Correlation matrix of diabetes self-care behaviour subscales and continuous demographic and clinical variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Age																		
2. Duration	.45**																	
3. Weight	-.20	-.11																
4. BMI	-.19	-.12	.86**															
5. Systolic	.23**	-.01	.17*	.18**														
6. Diastolic	-.16	-.35	.29**	.18**	.49**													
7. HbA1c	-.28	-.02	.16*	.13	-.01	.17*												
8. eGFR	-.61	-.32	.06	.06	-.23	.00	.30**											
9. Total cholesterol	-.21	-.25	-.09	.00	.01	.21*	.20*	.17*										
10. N diabetes complications	.38**	.41**	-.06	-.13	.15*	-.03	-.09	-.49	-.20									
11. N co-morbidities	.07	.06	.06	.13	-.02	-.05	-.05	-.08	.06	.03								
12. General diet	.18**	.09	-.17*	-.11	.04	-.10	-.25**	-.11	-.05	.04	.01							
13. Specific diet (fruit & veg)	.05	-.04	-.02	-.03	-.01	.02	-.01	.10	.02	-.05	.05	.33**						
14. Specific diet (saturated fat)	.06	.16*	-.06	-.10	.03	.01	-.10	.00	-.05	.11	.07	.21**	.12					
15. Exercise	-.06	-.05	-.11	-.19**	-.08	.00	-.08	.13	.01	-.12	-.06	.23**	.26**	.08				
14. SMBG	.06	.22**	.02	.02	.00	-.06	.00	-.10	.03	.03	.10	.16*	.03	.07	.05			
15. Foot care	.11	.19**	.00	-.05	.07	.06	-.01	-.13	.02	.22**	-.03	.23**	.22**	.14*	.15*	.16*		
16. Medication adherence	.14*	.15*	.04	.00	.06	.06	-.10	-.06	-.05	-.01	.03	.08	.09	.03	.10	.23*	.07	
17. Global diabetes self-care	.12	.20**	-.08	-.17*	.01	.00	-.10	-.09	.01	.11	.03	.57**	.47**	.32**	.55**	.52**	.65**	.22**

Key: SMBG, Self-monitoring of blood glucose; Veg, vegetables

Mann Whitney U tests were also performed to explore the relationship of each diabetes complication and other health co-morbidity separately with cognitions, well-being subscales, and diabetes self-care behaviours. These findings are reported in the text below and in Statistical Appendix A Tables A9 to A18.

Neuropathy

Participants with the diabetes complication neuropathy had more negative illness cognitions with lower perceptions of personal and treatment control. They also had a greater belief that diabetes was caused by ageing. No differences on diabetes well-being subscales were observed however lower levels of exercise and increased frequency of foot care were reported among participants with neuropathy relative to those without.

Nephropathy

Compared with participants without nephropathy those with the condition had decreased timeline cyclical, consequences, and medication concerns and attributed diabetes to being a hereditary condition. They also reported lower scores on depression and anxiety subscales and increased positive well-being. No differences were observed for the energy subscale or diabetes self-care behaviour subscales.

Retinopathy

Participants with retinopathy had decreased identity beliefs and were less likely to perceive that diabetes was caused by psychological factors compared with those participants without retinopathy. Those with retinopathy also reported lower scores on depression and anxiety subscales and increased scores on positive well-being and energy subscales. Higher levels of foot care and global levels of diabetes self-care adherence were observed among those participants with retinopathy relative to those participants without it.

Macrovascular disease

Participants with macro-vascular disease endorsed greater medication necessity beliefs compared with those without this complication. No statistically significant differences were observed for well-being questionnaire or diabetes self-care subscales.

Peripheral vascular disease (PVD)

Participants with PVD were less concerned about their medications compared with participants without PVD. No statistically significant differences were observed for well-being questionnaire subscales or diabetes self-care subscales.

Respiratory conditions

Participants with respiratory conditions reported elevated timeline acute/chronic, timeline cyclical, consequences, emotional representation beliefs, causal psychological attributions, and lower treatment control beliefs compared with those participants without respiratory conditions. They also reported increased scores on depression and anxiety subscales and lower levels of positive well-being compared with participants without respiratory conditions. No differences in levels of diabetes self-care were observed.

Musculoskeletal conditions

Individuals with musculoskeletal conditions had decreased treatment control beliefs and were more likely to attribute diabetes to lifestyle factors compared with those participants without this complication. No differences in well-being questionnaire or self-care subscales were observed.

Endocrine/Genitourinary/Other conditions

No statistically significant differences were observed between individuals with or without endocrine, genitourinary, and other health co-morbidities for cognition, well-being or health behaviour variables excluding genitourinary co-morbidities. Participants with genitourinary conditions reported higher levels of exercise compared with participants without these conditions.

5.6.3 *The relationship between diabetes health outcomes and other clinical characteristics and diabetes cognitions, well-being subscales, and diabetes self-care*

Diabetes medication regimen

Mann Whitney U tests were performed to examine the relationships between diabetes medication regimen and cognitions, well-being, and diabetes self-care behaviour subscales. These findings are reported in the text below and in Statistical Appendix A, Table A19.

Individuals treated with insulin/non-insulin injections or combination therapy (injections and oral hypoglycaemic medicines) had increased identity, timeline acute/chronic, and hereditary beliefs compared with participants treated with diet or oral hypoglycaemics. Participants on insulin therapy also had lower personal control beliefs and thought that diabetes was less likely to be caused by ageing or psychological factors.

Participants who managed their diabetes with diet only and/or oral medications reported increased levels of energy compared with those participants who managed their diabetes with injectable medications. No other statistically significant differences were observed on any of the other diabetes well-being questionnaire subscales.

Participants on injection based treatment regimens monitored their blood glucose levels more frequently and reported higher global levels of diabetes self-care adherence compared with those participants treated with diet alone or oral hypoglycaemics.

Continuous health outcomes (See Tables 5.13 to 5.16 above)

Weight and BMI

A participant's weight correlated positively with a belief that diabetes was caused by lifestyle factors and was associated with decreased baseline adherence to general diet. No other correlations between cognitions, well-being subscales or diabetes self-care were observed. Participants with a higher baseline BMI reported increased identity beliefs, emotional representations, medications concerns, and lifestyle causal attributions. They also reported higher depression and anxiety scores and lower adherence to exercise and global diabetes self-care.

Systolic and diastolic blood pressure

No associations were observed between systolic blood pressure and cognition, well-being or health behaviour variables. However diastolic blood pressure was associated with increased emotional representations and lifestyle causal attributions only.

HbA1c

Participants with increased baseline HbA1c (poorer diabetes control) demonstrated increased beliefs in identity, timeline cyclical, consequences, emotional representations, medication concerns, psychological causal attributions, and elevated levels of depression and anxiety. They also reported lower personal control over their diabetes, less illness coherence and decreased adherence to a healthy general diet.

eGFR

Individuals with high baseline eGFRs (indicating better renal function) had elevated scores on: identity, timeline cyclical, consequences, emotional representations, medication concerns, psychological causal attributions, lifestyle, and hereditary beliefs and increased levels of depression and anxiety. No relationships were observed with diabetes self-care.

Total cholesterol

Total cholesterol correlated only with cognition domains. Specifically, total cholesterol demonstrated a positive correlation with emotional representations, medication concerns, uncontrollable cause, and psychological attributions, and negatively with medication necessity beliefs.

Diabetes duration

Longer diabetes duration correlated positively with the following subscales: timeline acute/chronic, medication necessity, specific diet, self-monitoring of blood glucose, foot care, medication adherence, and global diabetes self-care. Increased diabetes duration also correlated negatively with: identity, emotional representations, medication concerns, and causal risk factors.

5.6.4 *Intra-relationships occurring between diabetes cognitions, well-being subscales, and diabetes self-care*

Diabetes cognitions

Spearman's rho correlation coefficients were calculated at baseline to examine how illness cognition domains relate to each other. These findings are presented in Table 5.17.

Diabetes cognition domains with pessimistic meanings about diabetes demonstrated positive correlations with each other specifically: identity, timeline cyclical, consequences, medication necessity, medication concerns, emotional representations, uncontrollable cause, and psychological attributions. However, medication necessity did not demonstrate a relationship with emotional representations or either of the causal subscales.

Additional correlations were found for consequences and medication necessity beliefs in that they both correlated positively with hereditary causal beliefs. Also, increased medication concerns were associated with greater beliefs in risk factors and lifestyle causes.

Excluding consequences and medication necessity beliefs all of the above pessimistic subscales demonstrated negative correlations with personal control and illness coherence; consequences did however correlate negatively with illness coherence only. Personal control and illness coherence were positively correlated and they both showed positive associations with treatment control. Treatment control did show some negative associations with pessimistic illness cognition domains, specifically: emotional representations, medication concerns, and uncontrollable cause. Both personal control and treatment control were positively correlated with lifestyle.

Timeline acute/chronic is also an illness cognition domain with negative health connotations, but it only demonstrated positive associations with consequences and medication necessity beliefs, and, unlike the other pessimistic illness cognitions correlated positively with illness coherence. It also demonstrated a negative correlation with lifestyle beliefs.

The newly generated causal variables from the PCA also demonstrated positive correlations with each other in particular: uncontrollable cause, psychological attributions,

risk factors, and ageing. Psychological attributions and risk factors also correlated positively with lifestyle beliefs.

Well-being questionnaire subscales

Correlations between WBQ subscales are presented in Table 5.18 and were performed to establish how well-being questionnaire constructs relate to each other. Depression and anxiety were positively correlated with each other and negatively associated with positive well-being and energy. Similarly, positive well-being and energy were positively correlated. All correlations between the well-being questionnaire subscales were large ranging from ± 0.63 to 0.73.

Diabetes self-care

Table 5.20 presents the correlations between the diabetes self-care behaviour subscales. The general diet and foot care subscales correlated with all other subscales of the SDSCA, excluding medication adherence. Adherence to a diet rich in fruit and vegetables correlated positively with adherence to exercise. Self-monitoring of blood glucose correlated positively with medication adherence. All subscales correlated positively with global levels of diabetes self-care. No other intra-relationships between SDSCA subscales were observed.

5.6.5 *Inter-relationships between cognitions, well-being subscales, and diabetes self-care*

Cognitions and well-being questionnaire subscales

Exploratory analyses were performed to examine the relationships between cognitions and well-being questionnaire subscales at baseline. Findings are presented in Tables 5.18 and 5.19.

Depression correlated positively with the domains: identity, timeline cyclical, consequences, emotional representations, medication concerns, uncontrollable cause, psychological attributions, and lifestyle. Depression also showed negative correlations with the subscales personal control, treatment control, and illness coherence. These relationships were largely the same for anxiety, except anxiety showed no statistically

significant association with lifestyle, and instead demonstrated positive correlations with medication necessity and hereditary beliefs.

The opposite relationships occurred for positive well-being and energy, specifically the subscales for identity, timeline cyclical, consequences, emotional representations, uncontrollable cause, psychological attributions, and hereditary beliefs all had negative correlations with positive well-being and energy. The subscales personal control, treatment control and illness coherence had positive correlations with both energy and positive well-being subscales. Medication necessity also had a negative correlation with energy only, and a causal belief that diabetes was caused by ageing correlated negatively with positive well-being.

Relationship between baseline diabetes self-care behaviours and diabetes cognitions and well-being subscales

Tables 5.20 to 5.22 present the correlation coefficients between the diabetes self-care subscales and the diabetes cognition and well-being subscales at baseline. Relationships for each self-care behaviour subscale are outlined below.

General diet

Participants who adhered more to their general diet had decreased scores on emotional representations and medication concerns, and lower beliefs that diabetes was caused by risk factors, and lifestyle. They also demonstrated increased positive well-being.

Specific diet (fruit and vegetables and saturated fat)

Participants who adhered more to a diet high in fruit and vegetables had increased identity perceptions. In contrast participants who indicated that they consumed a diet high in saturated fat believed that diabetes was caused by risk factors. No diabetes well-being questionnaire subscales demonstrated associations with specific diet subscales.

Exercise

Participants who displayed increased levels of exercise demonstrated increased treatment control beliefs, lower medication concerns, and decreased beliefs that diabetes was caused

by ageing. They also demonstrated lower scores on depression and increased levels of energy, and positive well-being.

Self-monitoring of blood glucose

Increased frequency of self-monitoring of blood glucose was associated with increased timeline acute/chronic, consequences, and medication necessity beliefs and decreased beliefs in causal risk factors and lower levels of energy.

Foot care

Participants who cared for their feet more had greater consequences and illness coherence beliefs and believed that diabetes was less likely to be caused by ageing or risk factors. Increased foot care was also associated with increased positive well-being.

Medication adherence

Beliefs about medication necessity, energy, and positive well-being were positively correlated with medication adherence whereas medication concerns, lifestyle, and psychological causal attributions were negatively correlated with medication adherence.

Global diabetes self-care

No diabetes well-being variables were associated with global diabetes self-care. However, increased timeline acute/chronic, consequences, illness coherence, and medication necessity beliefs were associated with increased global diabetes self-care. In contrast, medication concerns and causal beliefs: risk factors, psychological attributions, lifestyle. and aging were negatively correlated with global diabetes self-care.

Table 5.17: Intra-relationships between baseline diabetes cognition domains[4]

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Identity															
2. Timeline acute/chronic	.04														
3. Timeline cyclical	.23**	.01													
4. Consequences	.39**	.24**	.32**												
5. Personal control	-.16*	.11	-.23**	-.02											
6. Treatment control	-.05	-.12	-.11	-.09	.42**										
7. Illness coherence	-.27**	.13*	-.38**	-.20**	.44**	.23**									
8. Emotional representations	.49**	-.08	.33**	.48**	-.25**	-.13*	-.43**								
9. Medication necessity	.13*	.34**	.24**	.32**	.05	.00	-.10	.09							
10. Medication concerns	.40**	-.10	.33**	.35**	-.26**	-.23**	-.43**	.62**	-.03						
11. Uncontrollable cause	.22**	-.09	.22**	.21**	-.24**	-.17**	-.34**	.32**	-.01	.36**					
12. Risk factors	.08	-.06	-.09	.00	.07	.09	-.02	.10	-.02	.16*	.25**				
13. Psychological attributions	.35**	-.02	.32**	.27**	-.17**	-.11	-.28**	.44**	.01	.46**	.50**	.28**			
14. Lifestyle	.05	-.14*	.06	.04	.19**	.28**	-.04	.10	-.08	.13*	.05	.16*	.18**		
15. Hereditary	.10	.09	.12	.20**	.00	-.08	.01	.11	.21**	.06	.02	.08	.10	-.08	
16. Ageing	.03	-.03	.02	-.08	-.03	-.13*	-.02	.11	-.02	.11	.35**	.23**	.23**	.12	.05

[4] Correlations between the causal subscales differ from those reported between the generated components as part of the PCA (See section 5.4). This is because they are based on summed scores across the items.

Table 5.18: Correlation matrix of baseline cognitions and well-being subscales

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Depression											
2. Anxiety	.71**										
3. Energy	-.67**	-.64**									
4. Positive well-being	-.73**	-.63**	.70**								
5. Identity	.33**	.45**	-.45**	-.33**							
6. Timeline acute/chronic	-.03	.01	-.09	-.01	.04						
7. Timeline cyclical	.27**	.35**	-.36**	-.28**	.23**	.01					
8. Consequences	.33**	.37**	-.43**	-.35**	.39**	.24**	.32**				
9. Personal control	-.20**	-.18**	.22**	.17**	-.16*	.11	-.23**	-.02			
10. Treatment control	-.13*	-.14*	.21**	.18**	-.05	-.12	-.11	-.09	.42**		
11. Illness coherence	-.32**	-.26**	.36**	.30**	-.27**	.13*	-.38**	-.20**	.44**	.23**	
12. Emotional representations	.48**	.57**	-.54**	-.49**	.49**	-.08	.33**	.48**	-.25**	-.13*	-.43**

Table 5.19: Correlation matrix of baseline causal cognitions, medication cognitions, and well-being subscales

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Depression											
2. Anxiety	.71**										
3. Energy	-.67**	-.64**									
4. Positive well-being	-.73**	-.63**	.70**								
5. Medication necessity	.05	.13*	-.20**	-.11							
6. Medication concerns	.43**	.47**	-.44**	-.43**	-.03						
7. Uncontrollable cause	.28**	.20**	-.20**	-.28**	-.01	.36**					
8. Risk factors	.06	.00	-.03	-.09	-.02	.16*	.25**				
9. Psychological attributions	.36**	.39**	-.39**	-.39**	.01	.46**	.50**	.28**			
10. Lifestyle	.13*	.13	-.07	-.10	-.08	.13*	.05	.16*	.18**		
11. Hereditary	.02	.17**	-.13*	-.13*	.210**	.06	.02	.08	.10	-.08	
12. Ageing	.08	.07	-.11	-.19**	-.02	.11	.35**	.23**	.23**	.12	.05

Table 5.20: Correlation matrix of illness cognitions and diabetes self-care behaviours

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Identity															
2. Timeline acute/chronic	.04														
3. Timeline cyclical	.23**	.01													
4. Consequences	.39**	.24**	.32**												
5. Personal control	-.16*	.11	-.23**	-.02											
6. Treatment control	-.05	-.12	-.11	-.09	.42**										
7. Illness coherence	-.27**	.13*	-.38**	-.20**	.44**	.23**									
8. Emotional representations	.49**	-.08	.33**	.48**	-.25**	-.13*	-.43**								
9. General diet	.02	.12	-.06	.08	.04	.04	.11	-.15*							
10. Specific diet (fruit & veg)	.15*	.03	.00	.09	.05	.03	.12	.08	.33**						
11. Specific diet (saturated fat)	-.01	.07	.07	.09	-.06	-.03	.01	-.04	.21**	.12					
12. Exercise	-.01	-.10	-.09	-.11	.12	.19**	.16*	-.11	.23**	.26**	.08				
13. SMBG	.04	.18**	.06	.16*	-.04	-.02	-.11	.04	.16*	.03	.07	.05			
14. Foot care	.09	.02	.01	.16*	-.02	.04	.16*	-.01	.23**	.22**	.14*	.15*	.16*		
15. Medication adherence	-.08	.04	-.04	-.08	.07	.02	.04	-.04	.08	.09	.03	.10	.23**	.07	
16. Global diabetes self-care	.06	.14*	-.02	.13*	.05	.12	.17*	-.09	.57**	.47**	.32**	.55**	.52**	.65**	.22**

Key: SMBG, Self-monitoring of blood glucose; Veg, vegetables

Table 5.21: Correlation matrix of baseline causal cognitions, medication cognitions and diabetes self-care behaviours

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Medication necessity															
2. Medication concerns	-.03														
3. Uncontrollable cause	-.01	.36**													
4. Risk factors	-.02	.16*	.25**												
5. Psychological attributions	.01	.46**	.50**	.28**											
6. Lifestyle	-.08	.13*	.05	.16*	.18**										
7. Hereditary	.21**	.06	.02	.08	.10	-.08									
8. Ageing	-.02	.11	.35**	.23**	.23**	.12	.05								
9. General diet	.12	-.15*	-.03	-.16*	-.08	-.18**	.00	-.06							
10. Specific diet (fruit & veg)	.03	.02	-.03	-.08	.01	-.10	.08	-.01	.33**						
11. Specific diet (saturated fat)	.00	-.09	-.01	-.15*	.02	-.12	.06	-.03	.21**	.12					
12. Exercise	.02	-.17**	-.11	-.01	-.04	-.04	.06	-.13*	.23**	.26**	.08				
13. SMBG	.29**	-.07	.03	-.22**	-.09	-.06	-.08	-.01	.16*	.03	.07	.05			
14. Foot care	.05	-.12	-.05	-.20**	-.11	-.11	.01	-.18**	.23**	.22**	.14*	.15*	.16*		
15. Medication adherence	.14*	-.19**	-.06	-.06	-.14*	-.15*	-.02	.00	.08	.09	.03	.10	.23**	.07	
16. Global health behaviours	.19**	-.20**	-.11	-.26**	-.13*	-.16*	.05	-.17*	.57**	.47**	.32**	.55**	.52**	.65**	.22**

Key: SMBG, Self-monitoring of blood glucose; Veg, vegetables

Table 5.22: Correlation matrix of baseline well-being subscales and diabetes self-care behaviours

	1	2	3	4	5	6	7	8	9	10	11
1. General diet											
2. Specific diet (fruit & veg)	.33**										
3. Specific diet (saturated fat)	.21**	.12									
4. Exercise	.23**	.26**	.08								
5. SMBG	.16*	.03	.07	.05							
6. Foot care	.23**	.22**	.14*	.15*	.16*						
7. Medication adherence	.08	.09	.03	.10	.23**	.07					
8. Global health behaviours	.57**	.47**	.32**	.55**	.52**	.65**	.22**				
9. Depression	-.09	-.06	.06	-.20**	.09	-.08	-.05	-.11			
10. Anxiety	-.09	-.01	.04	-.09	.12	-.02	-.07	.00	.71**		
11. Energy	.12	.09	-.06	.33**	-.18**	.12	.06	.17*	-.67**	-.64**	
12. Positive well-being	.20**	.09	-.06	.25**	-.06	.21**	.08	.22**	-.73**	-.63**	.70**

Key: SMBG, Self-monitoring of blood glucose; Veg, vegetables

5.7 *Follow-up analyses*

Of the available pool of 259 eligible participants at six months follow-up completers and non-completers were compared in relation to their baseline demographic and clinical characteristics. Findings of these statistical tests can be found in Table 5.23.

There was a statistically significant relationship between ethnicity and responding at six months follow-up. Of those participants who completed follow-up a greater proportion of completers (72.2%) were of a white ethnic background; while only 43.1% of the non-completers were of white ethnicity.

Both weight and systolic blood pressure differed between completers and non-completers. Compared with non-completers (86.7kg, SD 23.8), completers had a higher mean weight (93.9 kg, SD 21.4). Similarly, compared with non-completers (130.7 mmHg, SD 14.3) completers had a higher mean systolic blood pressure (135.3 mmHg, SD 18.8).

No other statistically significant differences between completers and non-completers were observed.

5.7.1 *Follow-up univariate analyses*

Tables 5.24 to 5.26 present the univariate descriptive statistics for participants who completed follow-up questionnaires. The patterns of findings reported at baseline were replicated at follow-up.

Table 5.23: A comparison of completers and non-completers on demographic and clinical characteristics

Variables		Completers Average/N	SD/IQR/%	Non-completers Average/N	SD/IQR/%	Test statistic	P value
Continuous variables							
Age*	mean	62.8	11.9	60.6	13.7	z=-1.08	.28
	median	63.0	55.0-72.0	61.0	52.8-71.5		
Diabetes duration¶	mean	14.3	8.8	14.5	8.5	z=-0.43	.67
	median	13.0	8.3-19.0	14.0	8.0-22.0		
Hba1c/mmol/mol ≀	mean	65.6	16.7	65.8	19.8	z=-0.07	.95
	median	61.7	54.1-74.9	65.0	49.7-79.2		
Weight/kg§	mean	93.9	21.4	86.7	23.8	z=-2.22	.03*
	median	91.4	78.0-111.0	84.5	72.6-96.6		
BMI†	mean	33.3	6.8	31.4	7.0	z=-1.75	.08
	median	32.4	28.3-37.2	29.9	26.4-36.3		
Systolic/mmHg ‡	mean	135.3	18.2	130.7	14.3	z=-2.05	.04
	median	135.0	124.0-144.0	128.0	120.0-136.0		
Diastolic/mmHg **	mean	69.6	0.8	66.6	9.7	z=-1.32	.19
	median	69.0	63.0-74.5	66.0	63.0-73.0		
eGFR/1.73m² ¶¶	mean	61.2	24.9	62.6	24.2	z=-0.21	.83
	median	67.0	41.5-87.5	67.0	45.3-84.3		

Table 5.23: continued

Variables		Completers Average/N	SD/IQR/%	Non-completers Average/N	SD/IQR/%	Test statistic	p value
Total cholesterol/mmol/l ϥϥ	mean	4.0	1.1	4.2	1.2	z=-0.80	.44
	median	3.8	3.2-4.7	3.9	3.4-4.9		
Number of complications§§	mean	2.0	1.8	2.2	1.2	z=-0.91	.36
	median	2.0	1.0-3.0	2.0	1.0-3.0		
Number of co-morbidities††	mean	1.5	1.2	1.5	1.4	z=-0.08	.94
	median	1.0	1.0-2.0	1.5	0.0-3.0		
Categorical variables		N	%	N	%	Test statistic	p value
Gender	male	120	61.9	36	55.4	χ^2=0.60	.44
	female	74	38.1	29	44.6		
Ethnicity	White background	140	72.2	28	43.1	χ^2=16.80	.00**
	Non-White background	54	27.8	37	56.9		
Diabetes treatment regimen	Diet/Oral hypoglycaemics	53	29.3	19	33.9	χ^2=0.25	.62
	Injections/combination	128	70.7	37	66.1		

Key: Valid N among responders (R) and Non-responders (NR): *R=190, NR=62; ¶R=180, NR=54; ϥR=162, NR=53; §R=171, NR=48; † R=168, NR=48; ‡R=161, NR=47; ** R=161, NR=47; ¶¶R=153, NR=50; ϥϥR=130, NR=41; §§R=185, NR=58; †† R=185, NR=58

Table 5.24: Demographic and clinical characteristics of participants at 6 months follow-up

Variable		Average/ Frequency	SD/IQR/%
Gender	Male	120	61.9
	Female	74	38.1
Age/years	mean	62.8	11.9
	median	63.0	55.0-72.0
Diabetes duration/years	mean	14.3	8.8
	median	13.0	8.3-19.0
Ethnicity	White	140	72.2
	Black	25	12.9
	Asian	24	12.4
	Mixed race	4	2.1
	Other/prefer not to say	1	0.52
Diabetes treatment regimen	Diet/oral hypoglycaemics	53	27.3
	Injections/Combination	128	66.0
	No access to medical records/missing data	13	6.7
Clinical outcomes			
HbA1c mmol/mol	mean	65.6	16.7
	median	61.7	54.1-74.9
Weight/Kg	mean	93.9	21.3
	median	91.4	78.0-111.0
BMI	mean	33.3	6.8
	median	32.4	28.3-37.2
Systolic/mmHg	mean	135.3	18.2
	median	135.0	124.0-144.0
Diastolic/mmHg	mean	69.6	9.8
	median	69.0	63.0-74.5
eGFR/1.73²	mean	61.2	24.9
	median	67.0	41.5-87.5
Total cholesterol/mmol/l	mean	4.0	1.1
	median	3.8	3.2-4.7

Table 5.25: Frequencies of diabetes complications and other co-morbidities extracted from electronic medical records at baseline for those participants who returned a follow-up questionnaire

Variable		Average/Frequency	SD/IQR/%
Number of complications	mean	2.0	1.2
	median	2.0	1.0-3.0
Neuropathy		66	34.0
Nephropathy		38	19.6
Retinopathy		52	26.8
Cardio-vascular		164	84.5
Cerebrovascular		13	6.7
Peripheral vascular disease		33	17.0
Metabolic		1	0.52
Number of other co-morbidities	mean	1.5	1.2
	median	1.0	1.0-2.0
Endocrine		78	40.2
Respiratory		21	10.8
Musculoskeletal		40	20.6
Genitourinary		22	11.3
Other		73	37.6

Table 5.26: Means and standard deviations of diabetes cognitions, well-being subscales, and diabetes self-care behaviours at 6 months follow-up

Variables		Average	SD/IQR
Revised Illness Perception Questionnaire			
Identity	mean	3.8	3.2
	median	3.0	1.0-5.0
Timeline acute/chronic	mean	4.2	0.7
	median	4.5	3.8-4.8
Timeline cyclical	mean	2.9	1.0
	median	3.0	2.0-3.0
Consequences	mean	3.3	0.8
	median	3.3	2.7-3.8
Personal control	mean	4.0	0.7
	median	4.0	3.7-4.5
Treatment control	mean	3.6	0.6
	median	3.6	3.2-4.0
Illness coherence	mean	3.6	0.9
	median	3.8	3.1-4.2
Emotional representations	mean	2.7	1.0
	median	2.5	2.0-3.5
Uncontrollable cause	mean	2.0	0.7
	median	2.0	1.0-2.5
Lifestyle	mean	3.2	1.1
	median	3.0	2.5-4.0
Risk factors	mean	2.1	1.1
	median	2.0	1.0-3.0
Psychological attributions	mean	2.4	1.0
	median	2.3	1.8-3.0
Hereditary	mean	3.5	1.4
	median	4.0	2.0-5.0
Ageing	mean	2.8	1.3
	median	3.0	2.0-4.0
Beliefs about Medicines Questionnaire			
Medication necessity	mean	4.1	0.8
	median	4.2	3.8-4.8
Medication concerns	mean	2.8	1.0
	median	2.8	2.0-3.4

Table 5.26: continued

Variables		Average	SD/IQR
Well-being questionnaire			
Depression	mean	4.7	3.6
	median	4.0	2.0-7.0
Anxiety	mean	5.4	4.2
	median	4.0	2.0-8.0
Energy	mean	5.8	3.1
	median	6.0	3.8-8.0
Positive well-being	mean	11.3	4.8
	median	12.0	8.0-15.0
Summary of diabetes self-care activity scale			
General diet	mean	5.0	2.1
	median	5.5	4.0-7.0
Specific diet (fruit & veg)	mean	4.7	2.3
	median	5.0	3.0-7.0
Specific diet (saturated fat)	mean	4.5	2.0
	median	5.0	3.0-6.0
Exercise	mean	2.3	2.3
	median	2.0	0.0-3.8
Self-monitoring of blood glucose	mean	4.6	2.7
	median	5.5	2.0-7.0
Foot care	mean	3.7	2.6
	median	3.5	1.0-6.0
Medication adherence	mean	6.8	0.9
	median	7.0	7.0-7.0
Global diabetes self-care	mean	4.3	1.2
	median	4.3	3.5-5.3

5.8 *Individual change over time analysis*

Table 5.27 shows the descriptive statistics for degree of individual change over six months for self-report measures of cognitions, emotions, and diabetes self-care and the biological outcome measure HbA1c.

Diabetes cognitions remained stable over six months. All cognitions showed limited mean change over time which ranged from ± 0.0 to 0.3, excluding illness coherence which showed a mean decrease of 1.1 points on the IPQ-R. The narrow standard deviations (range ±0.7 to 1.3; excluding the identity subscale because of its different scoring system) indicate individual changes over time for cognitions had low variability.

Equally all well-being subscales showed limited mean change over time with changes ranging from ± 0.1 to 0.2 relative to the well-being questionnaire subscale range of zero to eighteen, excluding the energy subscale which ranged from zero to twelve. Similarly, the degree of variance for individual change over six months was narrow with standard deviation statistics ranging from ± 0.0 to 3.1.

Scores on the self-reported diabetes self-care behaviours could range from zero to seven but the degree of mean change over time was small ranging from ± 0.0 to 0.3 for each specific diabetes self-care subscale. The degree of mean change for the global measure of diabetes self-care was greater decreasing by 0.4 days over time. Likewise, standard deviation statistics showed that there was moderate variability in participant's diabetes self-care practices over time with standard deviation statistics ranging from ±1.1 to 2.1 across each diabetes self-care subscale and global measure of diabetes self-care adherence.

HbA1c can range from 20 mmol/mol to 128 mmol/mol. Mean change in this sample over six months was small, decreasing by 2.1 mmol/mol, equally HbA1c in this sample had fairly narrow variance over time, with a standard deviation of ±15.5.

Table 5.27: Means and standard deviations of changes in diabetes cognitions, well-being subscales, diabetes self-care behaviours, and HbA1c over 6 months

Variables	Possible range		Average	SD/IQR
Revised Illness Perception Questionnaire				
Identity	0 to 14	mean	0.1	3.1
		median	0.0	-2.0 to 2.0
Timeline acute/chronic	1 to 5	mean	-0.3	0.7
		median	0.0	-0.5 to 0.3
Timeline cyclical	1 to 5	mean	0.0	0.9
		median	0.0	-0.3 to 0.5
Consequences	1 to 5	mean	0.1	0.7
		median	0.2	-0.3 to 0.5
Personal control	1 to 5	mean	-0.1	0.7
		median	0.0	-3.3 to 0.5
Treatment control	1 to 5	mean	-0.1	0.6
		median	0.0	-0.4 to 0.2
Illness coherence	1 to 5	mean	-1.1	0.9
		median	0.0	-0.6 to 0.4
Emotional representations	1 to 5	mean	0.1	0.9
		median	0.0	-0.6 to 0.4
Uncontrollable cause	1 to 5	mean	0.1	0.7
		median	0.0	-0.3 to 0.5
Lifestyle	1 to 5	mean	0.0	0.1
		median	0.0	-0.5 to 0.5
Risk factors	1 to 5	mean	0.0	1.2
		median	0.0	-0.5 to 1.0
Psychological attributions	1 to 5	mean	0.1	0.9
		median	0.0	-0.3 to 0.5
Hereditary	1 to 5	mean	0.0	1.0
		median	0.0	0.0 to 0.0
Ageing	1 to 5	mean	0.0	1.3
		median	0.0	-1.0 to 1.0
Beliefs about Medicines Questionnaire				
Medication necessity	1 to 5	mean	0.0	0.7
		median	0.0	-0.4 to 0.4
Medication concerns	1 to 5	mean	0.2	0.8
		median	0.0	-0.4 to 0.6

Table 5.27: continued

Variables	Possible range		Average	SD/IQR
Well-being questionnaire				
Depression	0 to 18	mean	0.2	2.9
		median	0.0	-2.0 to 2.0
Anxiety	0 to 18	mean	0.1	3.1
		median	0.0	-2.0 to 2.0
Energy	0 to 12	mean	-0.1	2.6
		median	0.0	-1.0 to 1.0
Positive well-being	0 to 18	mean	-0.1	0.0
		median	0.0	-2.4 to 2.2
Summary of Diabetes Self-are Activity Scale				
General diet	0 to 7	mean	0.0	1.8
		median	0.0	-1.0 to 1.0
Specific diet (fruit & veg)	0 to 7	mean	-0.1	2.1
		median	0.0	-1.0 to 1.0
Specific diet (saturated fat)	0 to 7	mean	0.0	1.9
		median	0.0	-1.0 to 1.0
Exercise	0 to 7	mean	-0.1	2.1
		median	-0.0	-1.0 to 1.0
Self-monitoring of blood glucose	0 to 7	mean	-0.1	1.9
		median	0.0	-0.5 to 0.0
Foot care	0 to 7	mean	0.3	2.0
		median	0.0	-0.5 to 1.0
Medication adherence	0 to 7	mean	0.0	0.7
		median	0.0	0.0 to 0.0
Global diabetes self-care	0 to 7	mean	-0.4	1.1
		median	-0.5	-1.1 to 0.5
Biological outcomes				
|HbA1c mmol/mol	20 to 128	mean	-2.1	15.5
		median	1.3	-7.8 to 4.4

5.9 *Chapter summary*

- Baseline clinical characteristics showed that the sample had poorly controlled diabetes. Clinical outcomes were above NICE recommended targets and the frequency of diabetes complications was high.
- The PCA on the causal items of the IPQ-R identified four subscales: uncontrollable cause, lifestyle, risk factors, and psychological attributions.
- Participants demonstrated low levels of depression and anxiety.
- On whole, older participants, females, participants of non-white ethnicity, persons on injectable medications, and individuals with either neuropathy, respiratory or musculoskeletal conditions had a poorer cognitive (increased pessimistic beliefs), emotional, and behavioural response to diabetes with some exceptions.
- Pessimistic diabetes cognitions correlated positively with each other as did optimistic diabetes cognitions. Increased pessimistic diabetes cognitions often correlated negatively with optimistic diabetes cognitions.
- All WBQ subscales correlated with each other. Similarly, general diet and foot care had the most intra-correlations with other SDSCA subscales.
- Depression and anxiety largely correlated positively with pessimistic diabetes cognitions and negatively with optimistic diabetes cognitions.
- Adaptive cognitions were associated with increased adherence to diabetes self-care behaviours overall (e.g. greater medication necessity and illness coherence beliefs). Depression and anxiety did not correlate with diabetes self-care behaviours.
- Change over time analyses showed that the sample had relatively stable cognition, emotion, behavioural, and HbA1c profiles over time.

6 *Results: Measurement models*

Before testing the primary research hypotheses about the directional relationships that exist between cognitions, poor emotional health, and diabetes self-care using structural equation modelling, it was necessary to ensure that each measure was adequately defined. This process is consistent with the approach outlined in Chapter Three, Section 3.4.3.

6.1 *Statistical analysis*

Measurement models were generated for each self-report measure and tested using confirmatory factor analysis (CFA). These models provided an empirical test of whether the hypothesised factor structure of each measure was supported among outpatients with Type 2 diabetes. It also allowed discriminant validity (e.g. all correlations between subscales less than 0.85; Kline, 2005) and uni-dimensional measurement (e.g. all questionnaire items load onto a single construct; Anderson and Gerbing, 1988) to be tested. CFAs were therefore performed on the baseline data for the:

i. Revised Illness Perception Questionnaire (IPQ-R)
ii. Beliefs about Medicines Questionnaire (BMQ)
iii. Well-being questionnaire (WBQ)
iv. Summary of Diabetes Self-Care Activity questionnaire (SDSCA)

For the IPQ-R a CFA was carried out on all subscales excluding the causal items. Because of the dichotomous scoring of the identity subscale this was included in the CFA as a parcelled variable only (See Chapter Three, Section 3.4.4 for a description of item parcelling). Causal subscales were excluded because these subscales were identified in the present sample from a Principal Component Analysis and cannot be validated using the same sample.

Each of the IPQ-R items was specified to load onto their hypothesised subscale and each subscale was allowed to co-vary with each other. Similarly, the BMQ and WBQ item scores were specified to load onto their hypothesised construct and subscales were allowed to co-vary.

For the SDSCA a second order measurement model was generated to represent global diabetes self-care (See Figure 6.17). Second order (hierarchical) models are applied when it is hypothesised that a number of conceptually distinct concepts are related to each other

by a broader underlying concept (Chen, et al., 2005). In the present case it was hypothesised that each specific diabetes self-care behaviour construct (e.g. diet, exercise, self-monitoring of blood glucose, foot care, medication adherence; referred to as first order constructs) was a sub-component of a single over-arching diabetes self-management concept (second order construct). Second order models permit greater model parsimony by allowing multiple related constructs to be summarised by a single latent variable (Chen et al., 2005).

Goodness of fit indices and the questionnaire item factor loadings were used to evaluate each CFA. The model fit indices used were those described in Chapter Three (Table 3.1) and the criteria below were applied to make conclusions about the adequacy of each hypothesised model. Models were deemed to be well fitting if they had a:

i. Non-significant chi square statistic (a very stringent test which is often not met by models but is routinely reported; Kline, 2005)
ii. Root mean square error of approximation (RMSEA) values within the range of ≤0.10 (indicates adequate fit) and ≤0.06 to (indicates good fit).
iii. Comparative fit index (CFI) value close to or above 0.95
iv. Standardised root mean square residual (SRMR) values ≤0.10

The statistical significance of each factor loading was also evaluated at the level of $p \leq 0.05$. Evidence of items loading onto more than one factor was identified from modification indices. Modification indices highlight all plausible areas for model adaptation by providing statistical estimates of how much the model chi squared statistic would decrease (e.g. model fit improves) if a pathway which is not currently specified in the model were added to it (e.g. a questionnaire item loading onto an alternative factor; Byrne, 2010; Kline, 2005).

Measurement models were also generated for each questionnaire subscale singularly based on the recommendations of Hooper et al (2008). This allowed a more thorough examination of each subscale/latent variable factor structure and permitted problems with their conceptualisation to be more easily identified. Model re-specifications were applied where necessary.

Singular measurement models were not generated for the energy and positive well-being subscales of the WBQ. The energy subscale was not rigorously explored because its items

can be a reflection of both mood and symptoms associated with poor diabetes control and was therefore excluded from subsequent structural models. Equally, the positive well-being subscale was excluded because the focus of this thesis is on the role of poor emotional health in a person's management of their diabetes and not on the role of positive emotions.

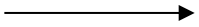
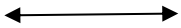
All measurement models did not meet the assumptions of multivariate normality (Mardia's index critical ratio score $\geq \pm 5$). Parameter estimates within each model were therefore bootstrapped. Bias corrected standard errors and p values for unstandardised model parameters were used to interpret their degree of statistical significance.

6.2 *Results*

The outcomes for each CFA are presented in Figures 6.1 to 6.18. Each figure presents the standardised factor loadings for questionnaire items onto their hypothesised construct and model fit indices. Although not explicitly indicated on the figures all factor loadings and latent variable correlations are statistically significant at the level of $p \leq 0.05$ unless otherwise stated. A full summary of the unstandardised and standardised factor loadings, and levels of statistical significance for each measurement model can be found in statistical Appendix B, Tables B1 to B18.

To avoid repetition notations used across each of the figures are provided in Table 6.1 as opposed to providing a key after each figure. Any additional terms are provided with the relevant figure.

Table 6.1: Key to aid with the interpretation of Figures 6.1 to 6.18

Abbreviation/Symbol	Definition
B_WBQ(N)	Used to represent questionnaire item numbers from the WBQ e.g. B_WBQ1
B_IP(N)	Used to represent questionnaire item numbers from the IPQ-R e.g. B_IP1
B_BMQ(N)	Used to represent questionnaire item numbers from the BMQ e.g. B_BMQ1
B_SDSCA (N)	Used to represent questionnaire item number from SDSCA e.g. B_SDSCA1
	Indicates the direction of effect
	Indicates that variables are assumed to co-vary but the direction of effect is not explicitly stated
	Squares indicate measured variables
	Ellipses indicate latent variables

6.2.1 *CFA of the IPQ-R and each of its individual subscales*

The initial CFA performed on the IPQ-R demonstrated poor to moderate fit on all model fit indices (χ^2=1441.8, $df_{m=}$675 ,p ≤.01; RMSEA=0.08, CFI=0.79, SRMR=0.09). Because of this finding the factor structure of each of the IPQ-R subscales were explored singularly to allow problems with the specification of the overall IPQ-R scale to be identified. The model fit indices for each of the IPQ-R subscales (excluding identity because this subscale was parcelled) are presented in Table 6.2.

Table 6.2: Model fit indices for each IPQ-R subscale

Model fit indices					
χ^2	DF	P	RMSEA	CFI	SRMR
Timeline acute/chronic (N=248)					
45.11	9	0.00	*0.13*	0.94	0.07
Timeline cyclical (N=251)					
7.27	2	0.03	0.10	0.99	0.03
Consequences (N=243)					
102.18	9	0.00	*0.21*	*0.75*	*0.11*
Personal control (N=239)					
57.37	9	0.00	*0.15*	*0.86*	0.08
Treatment control (N=249)					
16.03	5	0.01	0.09	*0.89*	0.05
Illness coherence (N=251)					
23.02	5	0.00	*0.12*	0.97	0.03
Emotional responses (N=251)					
168.62	9	0.00	*0.27*	*0.86*	0.08

All subscales excluding timeline cyclical demonstrated evidence of poor fit on one or more of the model fit indices. Those indices which indicated poor fit are in italics. The poor fit of each of the IPQ-R subscales (excluding timeline cyclical) identified the need to apply model re-specifications to the singular subscales first to allow these modifications to be applied to the full IPQ-R scale.

The statistical significance of factor loadings, modification indices, and correlations between residual error terms were used to identify areas for model modification. This is consistent with the approach described in Chapter Three, Section 3.4.3.2. The re-specified models and the original timeline cyclical model are presented in Figures to 6.1 to 6.7 with their associated model fit indices. Statistical Appendix B, Tables B1 to B7 presents a summary of all unstandardised and standardised statistical outputs for each IPQ-R subscale.

For the subscales timeline acute/chronic, personal control, and treatment control inspection of their residual error correlations demonstrated high correlations within the subgroups of positively scored (e.g. I have the power to influence my diabetes) and negatively (reverse) scored items (e.g. Nothing I do will affect my diabetes). This is a common psychometric phenomenon (Podsakoff, et al., 2003). Highly correlated residual error terms can be

indicative of a need to generate an additional factor which incorporates these items (Anderson and Gerbing, 1988). In response to these findings each of these subscales was specified to consist of two factors, factor one: positively scored items and factor two: reverse scored (negative) items. To enable all items originally hypothesised by Moss-Morris et al. (2002) to be retained in the model a second order latent variable was specified to drive responses to both the reverse scored and positively scored items (See Figures 6.2 to 6.4)[5]. This type of model re-specification is consistent with the recommendations of Gerbing and Anderson (1984). Item 20 was also removed from the treatment control subscale because of its non-statistically significant factor loading.

These model re-specifications demonstrated excellent fit to the sample data on all model fit indices for all subscales excluding the model chi squared statistic for personal control and emotional representations. In addition all factor loadings were statistically significant at the level of $p \leq 0.05$ with factor loadings ≥ 0.40, apart from the factor loading for item 18 onto the timeline acute/chronic subscale. It was however statistically significant (See Figure 6.2).

For the consequences subscale examination of the residual error matrices indicated high residual correlations between items nine, ten, and eleven. Examination of the conceptual content of these items indicated that their content may be interpreted in relation to social functioning/identity, for example item nine "My diabetes strongly affects the way others see me". Whereas items six to eight may reflect items more specific to health threats for example item six "My diabetes is a serious condition".

The clustering of items in relation to social functioning and health threat was explored through the generation of a second order model. This model specified the consequences subscale to represent a broad concept which influenced a person's perception of diabetes as a physical health threat and as a condition which impacted on their social functioning (See Figure 6.5).

The re-specified second order consequences model demonstrated excellent fit to the sample data once item seven was removed from the model "My diabetes has major consequences on my life". Item seven was removed because of a negative error variance

[5] When second order models are specified with only two first order indicators it is necessary to apply equality constraints. This allows the higher order part of the model to meet the principles of identification (Bentler, 2005; Byrne, 2010). Equality constraints were placed on the disturbance terms for the first order indicators. .

estimate, which caused an inadmissible solution. Negative error variances can occur when an indicator provides an almost perfect measure of the latent variable (Bollen, 1989). However because an estimate of the population is being made from the sample the software assumes that the solution is inadmissible because of sampling error (which is also plausible). All factor loadings were high and statistically significant for this re-specified model.

Similarly for the emotional representation subscale of the IPQ-R inspection of the residual error matrices demonstrated a high degree of residual error correlation between items 36, 37, and 38. The conceptual content of these items were explored and indicated that these items addressed content specific to the emotional health concept anxiety (e.g. Item 36 "My diabetes does not worry me"). In contrast the remaining three items content was related to the emotional states depression and anger (e.g. Item 33 "I get depressed when I think about my diabetes").

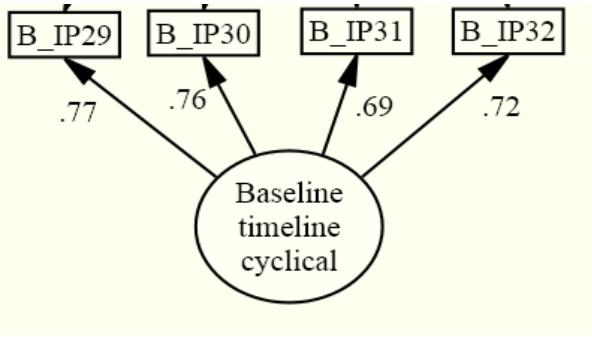
The grouping of the emotional representation items into emotional states associated with depression/anger and anxiety/worry was tested through the generation of a second order model whereby these specific emotional states were imbedded within an over-arching emotional representations concept (See Figure 6.6). The re-specified emotional representation subscale demonstrated good fit to the sample data on all model fit indices excluding the model chi-square fit index and standardised factor loadings were high and statistically significant.

For the illness coherence subscale, inspection of the modification indices highlighted that a substantial improvement in model fit would occur if the error terms of items 24 and 25 were allowed to correlate. The above findings can occur when questionnaire items are similarly worded (Byrne, 2010). Anderson and Gerbing (1988) caution against allowing residual error terms within time points of a measurement model to correlate as it brings into question the underlying meaning of the latent variable. Instead they encourage the removal of problematic item(s). For this reason item 24 was removed from the model because of its lower factor loading relative to item 25. Its removal resulted in a well-fitting model on all fit indices with high standardised and statistically significant factor loadings as shown in Figure 6.7.

Table 6.3: Baseline IPQ-R re-specified model fit indices (n=201)

χ^2	DF	P	RMSEA	CFI	SRMR
687.12	439	0.00	0.05	0.92	0.06

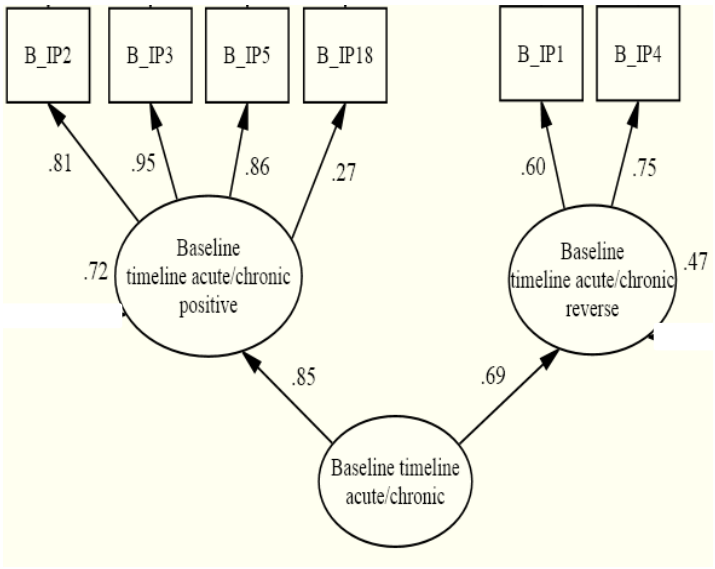
Figure 6.1: Baseline timeline cyclical measurement model



Model fit indices: Baseline timeline cyclical (n=251)

χ^2	DF	P	RMSEA	CFI	SRMR
7.27	2	0.03	0.10	0.99	0.03

Figure 6.2: Baseline timeline acute/chronic re-specified measurement model

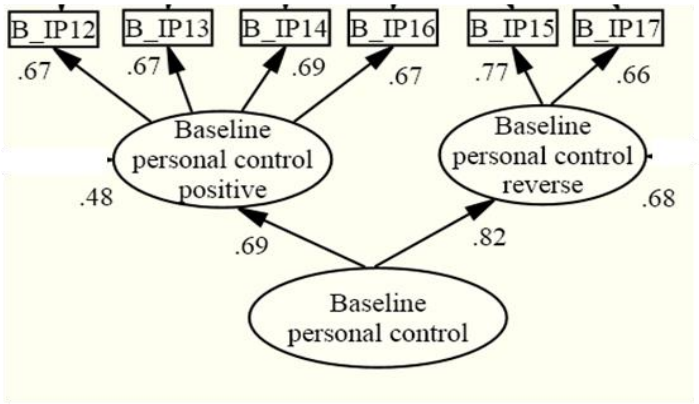


Key: Scores adjacent to the first order latent variables represent variance explained by the second order latent (R^2)

Model fit indices: Baseline timeline acute/chronic, re-specified (n=248)

χ^2	DF	P	RMSEA	CFI	SRMR
13.57	8	0.09	0.05	0.99	0.04

Figure 6.3 Baseline personal control re-specified measurement model

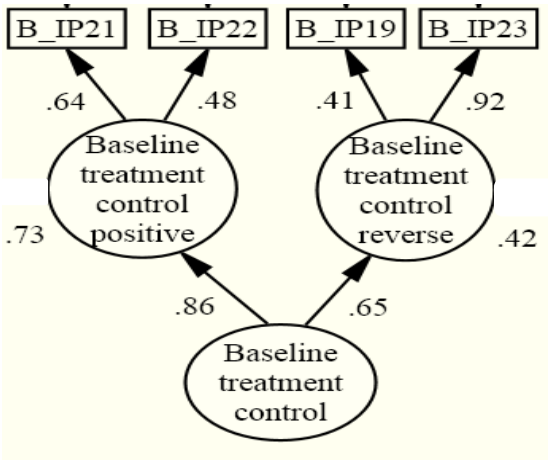


Key: Scores adjacent to the first order latent variables represent variance explained by the second order latent (R^2)

Model fit indices: Baseline personal control re-specified (n=249)

χ^2	DF	P	RMSEA	CFI	SRMR
17.16	8	0.03	0.07	0.97	0.04

Figure 6.4: Baseline treatment control re-specified measurement model

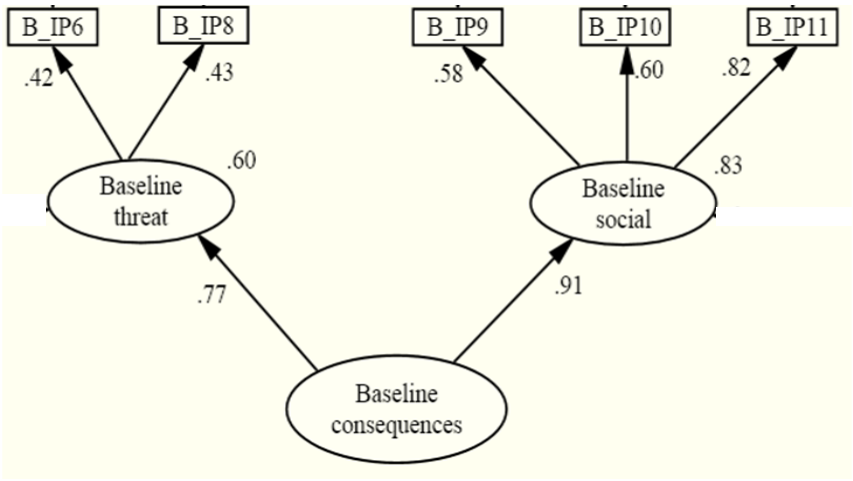


Key: Scores adjacent to the first order latent variables represent variance explained by the second order latent (R^2)

Model fit indices: Baseline treatment control re-specified (n=249)

χ^2	DF	P	RMSEA	CFI	SRMR
3.20	1	0.07	0.09	0.98	0.03

Figure 6.5: Baseline consequences re-specified measurement model (re-specified)

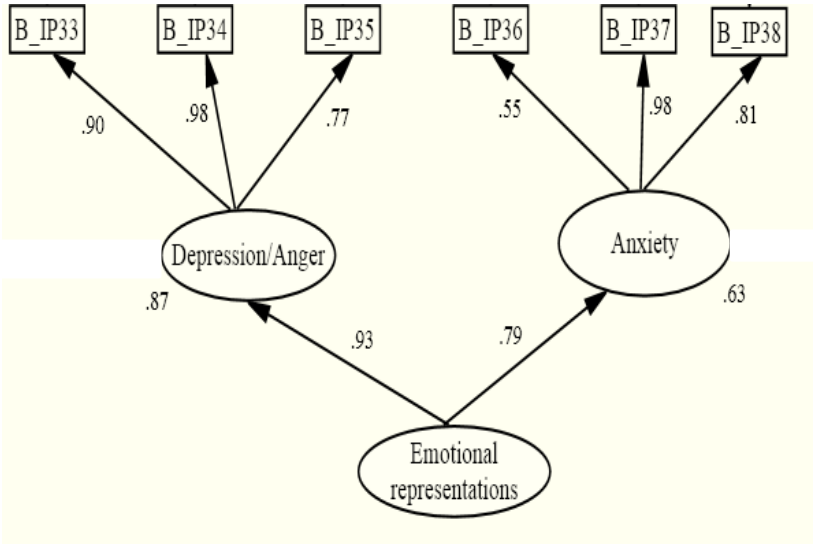


Key: Scores adjacent to the first order latent variables represent variance explained by the second order latent (R^2)

Model fit indices: Baseline consequences re-specified (n=243)

χ^2	DF	P	RMSEA	CFI	SRMR
5.73	4	0.22	0.04	0.99	0.03

Figure 6.6: Baseline emotional representations re-specified measurement model

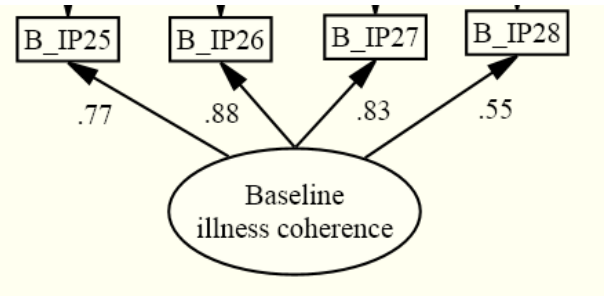


Key: Scores adjacent to the first order latent variables represent variance explained by the second order latent (R^2)

Model fit indices: Baseline emotional representations re-specified (N=251)

χ^2	DF	P	RMSEA	CFI	SRMR
21.78	8	0.01	0.08	0.99	0.04

Figure 6.7: Baseline illness coherence re-specified measurement model



Model fit indices: Baseline illness coherence re-specified (n=251)

χ^2	DF	P	RMSEA	CFI	SRMR
3.22	2	0.20	.05	1.00	.02

The good fit evidenced by all of the re-specified subscales of the IPQ-R allowed these model modifications to be applied to the full scale of the IPQ-R. Initially this resulted in a convergence (estimation) failure. Estimation failures can occur for a number of reasons including: small sample size, non-normality of the data, empirical under-identification (correlations between factors which are too large/small), model mis-specification, too few indicators per latent variable, or a combination of these factors (Anderson and Gerbing, 1988; Chen, et al., 2001; Kenny, 1979; Rindskopf, 1984).

Inspection of the statistical outputs indicated that estimation failures were associated with the treatment control subscale, most likely because this subscale consisted of only two indicators per latent variable at both the first order and second order level. Once the treatment control subscale was removed the full IPQ-R scale demonstrated excellent fit to sample data however there was a need to allow Item 6 of the consequences subscale "My diabetes is a serious condition" to cross-load onto the timeline acute/chronic positively worded subscale. The re-specified IPQ-R is shown in Figure 6.8 and its fit indices are presented in Table 6.3. All factor loadings were statistically significant (See Statistical Appendix B, Table B8 for a full tabular summary of the unstandardised and standardised factor loadings).

A correlation matrix of IPQ-R subscales is presented in Table 6.4. Subscales of the IPQ-R largely demonstrated statistically significant correlations with each other. However the timeline acute/chronic subscale did not correlate with any subscales of the IPQ-R. All

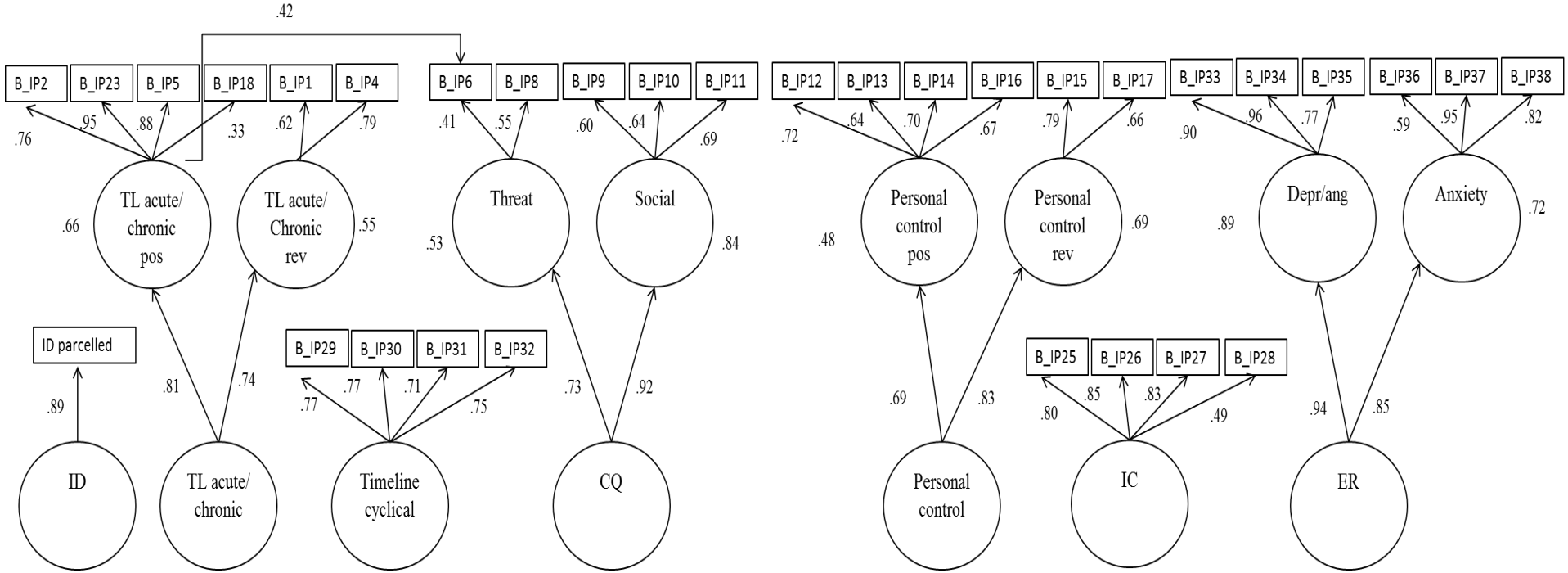
correlations were ≤0.85 indicating discriminant validity. Similarly examination of the modification indices did not demonstrate any item cross-loadings other than Item 6 of the consequences subscale loading onto the timeline acute/chronic subscale.

Table 6.4: Baseline IPQ-R re-specified correlation matrix

	1	2	3	4	5	6
1. Identity	1					
2. Timeline acute/chronic	.13	1				
3. Timeline cyclical	.34**	.10	1			
4. Consequences	.66**	.08	.55**	1		
5. Personal control	-.33**	-.06	-.44**	-.38**	1	
6. Illness coherence	-.35**	.08	-.51**	-.54**	.57**	1
7. Emotional representations	.61**	.04	.37**	.78**	-.39**	-.57**

Key: **p≤0.01 based on bootstrapped p values

Figure 6.8: Baseline IPQ-R re-specified measurement model



*Although not shown in the figure (for simplicity) all subscales of the IPQ-R were allowed to correlate with each other.

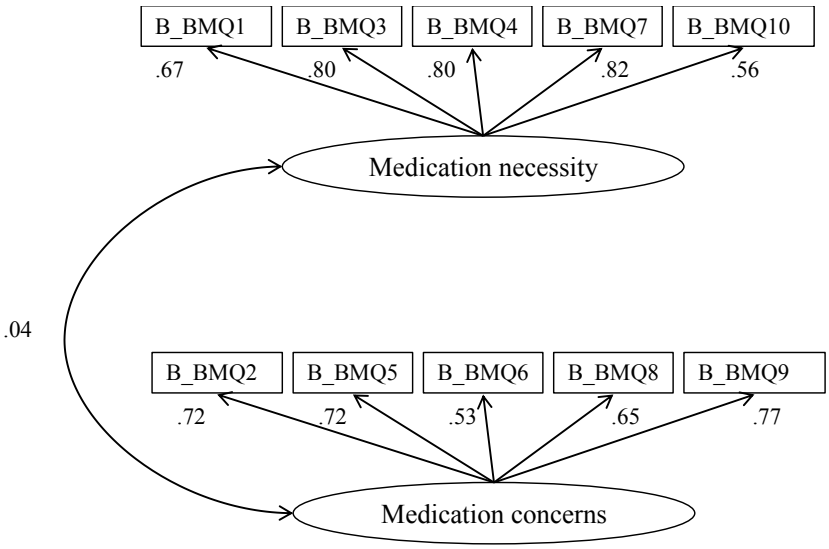
Key: CQ, Consequences; Depr/Ang, Depression/Anger, ER, Emotional representations; IC, Illness coherence; ID, Identity, Pos, Positive; Rev, reverse TL, Timeline; Scores adjacent to the first order latent variables represent variance explained by the second order latent (R^2).

6.2.2 *CFA on the whole BMQ and each of its individual subscales*

The measurement model for the whole BMQ is presented in Figure 6.9. This model demonstrated excellent fit on all model fit indices. All item factor loadings were ≥0.40 and were statistically significant at p≤0.01. The subscales had discriminant validity with a low non-statistically significant correlation between the factors of r=0.04. There was no indication of any item cross-loadings when modification indices were explored.

Similarly, the singular models of medication necessity and medication concerns demonstrated excellent fit on all fit indices with high and statistically significant factor loadings across all items (See Figures 6.10 and 6.11 and Statistical Appendix B, Tables B9 to B11 for a tabular summary of the statistical tests)

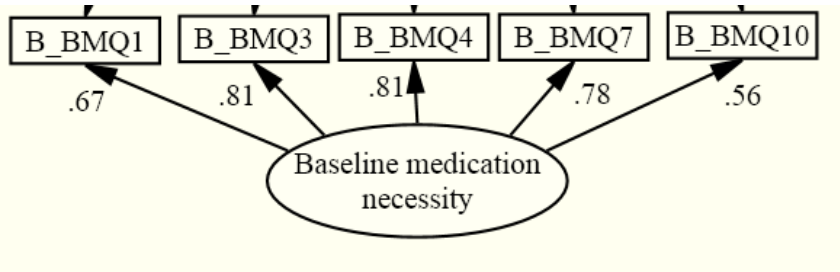
Figure 6.9: Baseline BMQ measurement model



Model fit indices: Baseline BMQ (n=240)

χ^2	DF	P	RMSEA	CFI	SRMR
41.68	34.00	.17	.03	.99	.05

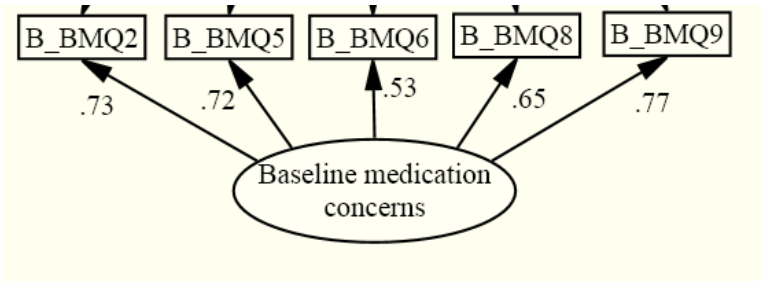
Figure 6.10: Baseline medication necessity measurement model



Model fit indices: Baseline medication necessity (n=244)

χ^2	DF	P	RMSEA	CFI	SRMR
9.60	5.00	.09	.06	.99	.02

Figure 6.11: Baseline medication concerns measurement model



Model fit indices: Baseline medication concerns (n=243)

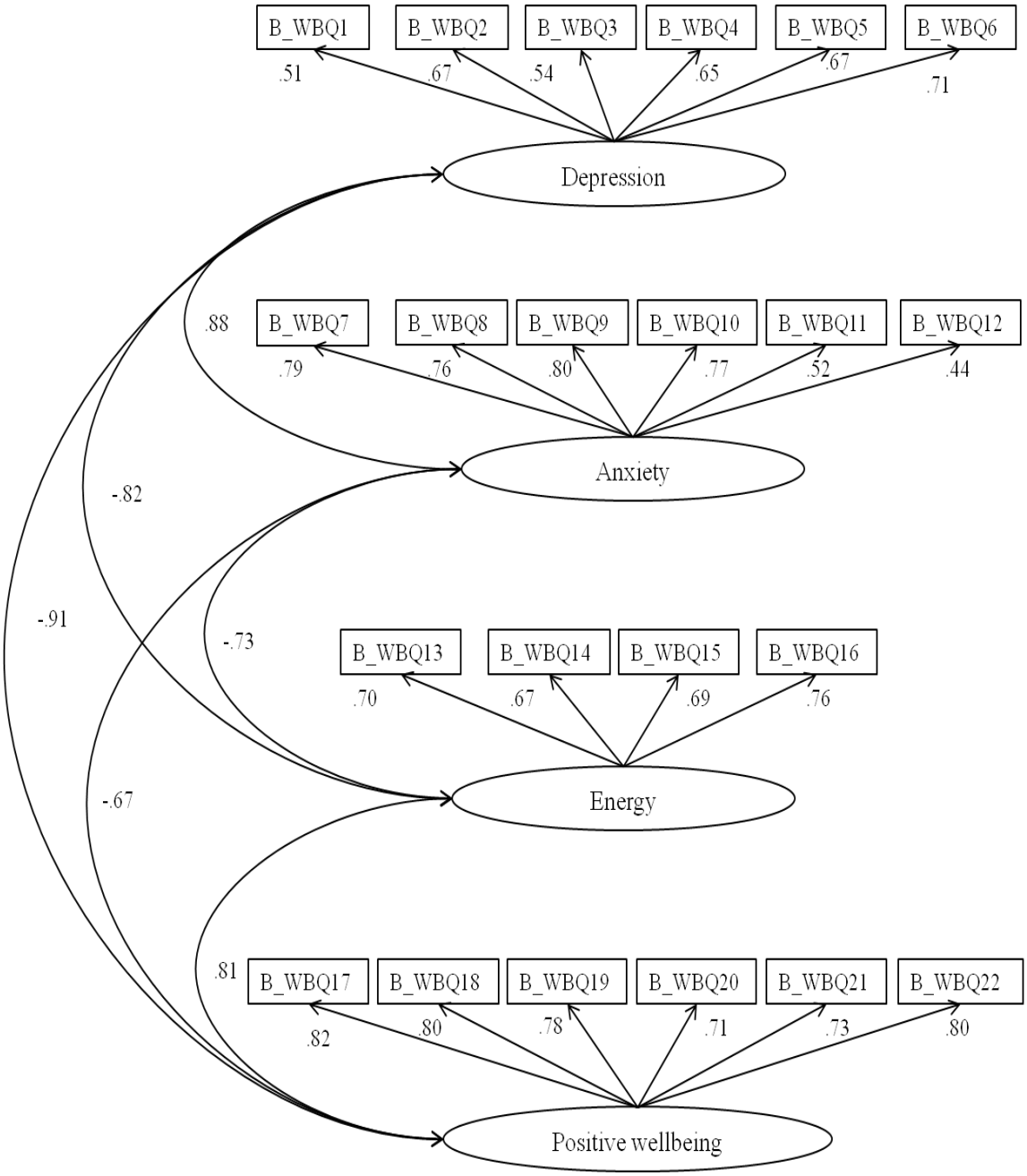
χ^2	DF	P	RMSEA	CFI	SRMR
7.25	5.00	.20	.04	.99	.02

6.2.3 *CFA on the whole WBQ and depression and anxiety singularly*

Figure 6.12 presents the CFA performed on all subscales of the WBQ simultaneously. The fit of this model was reasonable for the RMSEA and SRMR fit indices, but the model chi squared and CFI indicated evidence of poor fit. All questionnaire items had standardised factor loadings ≥0.40 and were statistically significant (See Figure 6.12 and Statistical Appendix B, Table B12).

Correlations between subscales indicated a lack of discriminant validity. Large statistically significant correlations occurred between depression and anxiety (r=0.88) and depression and positive well-being (r=-0.91). The correlations between the remaining subscales were also large ranging from ±0.67 to 0.82 (See Figure 6.12). Examination of the modification indices however did not indicate the presence of any substantial item cross-loadings between the subscales.

Figure 6.12: Baseline WBQ measurement model



Model fit indices: Baseline WBQ (n=237)

χ^2	DF	P	RMSEA	CFI	SRMR
656.99	203	0.00	0.10	0.84	0.08

Singular measurement models were generated for depression and anxiety given that only these subscales were to be used in subsequent structural models (See Figures 6.13 and 6.14 and Statistical Appendix B, Tables B13 and B14). The fit of the depression subscale was poor on all model fit indices excluding the SRMR; nevertheless its factor loadings were large and statistically significant (See Figure 6.13). In contrast the anxiety subscale demonstrated good levels of fit on the CFI and SRMR fit indices and acceptable levels of fit on the RMSEA index, but poor fit on the model chi-square statistic. All anxiety items factor loadings were high and statistically significant (See Figure 6.14).

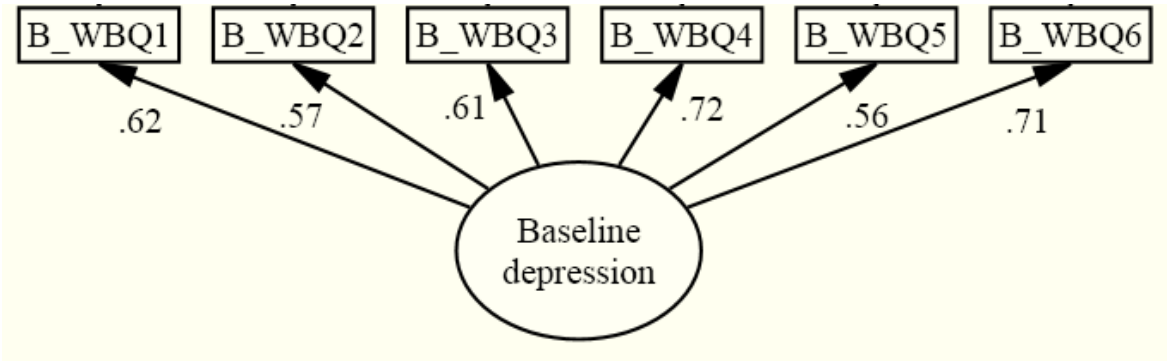
The original hypothesised specification of the anxiety subscale was retained given that its fit indices largely demonstrated that its specification was acceptable. However the modification indices and residual error correlations of the depression subscale were explored because of its poor fit. Assessment of the residual error matrices identified a large positive residual correlation between the two non-reverse scored depression items. As with some of the IPQ-R subscales to allow all the items originally hypothesised by Bradley (1994) to be retained in subsequent models of depression a second order depression factor was specified to drive responses to both the reverse scored and positively scored items (See Figure 6.15 and Statistical Appendix B, Table B15). This re-specified depression model demonstrated excellent fit to the sample data on all model fit indices with high statistically significant standardised factor loadings.

The poor fit of the depression subscale and the lack of discriminant validity between the depression and anxiety subscales found by the CFA of the whole WBQ identified the need to test an alternative measurement model for the conceptualisation of depression and anxiety. Testing competing theoretical models is encouraged in SEM (Tomarken and Waller, 2003).

Consistent with the work of Riazi et al. (2006) and as was discussed in chapter four, the two positively scored depression items (items two and five) and two of the positively scored anxiety items (items eight and nine) were combined to generate a negative well-being subscale (See Figure 6.16). This subscale demonstrated good fit to the sample data on all fit indices (excluding a slightly high RMSEA index of 0.09). All questionnaire items had high and statistically significant standardised factor loadings (See Figure 6.16 and Statistical Appendix B, Table B16).

When comparing theoretically alternative models an additional fit index is used known as the Aikaike Information Criterion (AIC). The model with the lowest AIC index is favoured and a difference of ≥10 between the two competing models is considered statistically meaningful (Burnham and Anderson, 2004). On this criteria the AIC index for the negative well-being subscale was lower (e.g. ≥ 10) than for both the depression (13.65 points lower) and anxiety subscale (31.53 points lower).

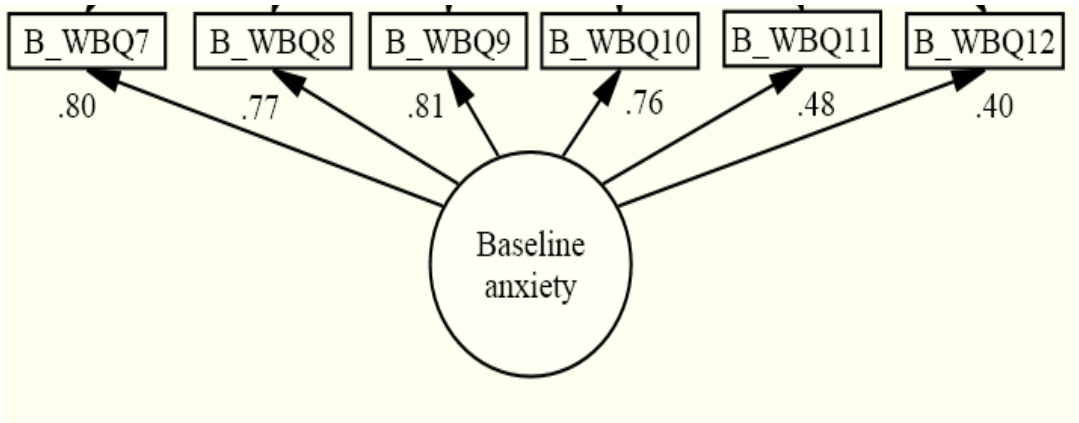
Figure 6.13: Baseline depression measurement model



Model fit indices: Baseline depression (n=248)

χ^2	DF	P	RMSEA	CFI	SRMR	AIC
64.31	9	*0.00*	*0.16*	*0.87*	0.07	88.31

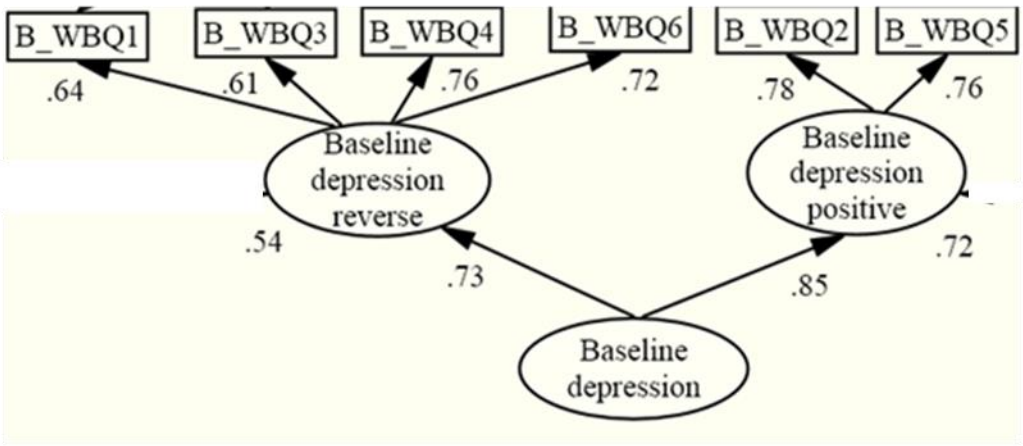
Figure 6.14: Baseline anxiety measurement model



Model fit indices: Baseline anxiety (n=249)

χ^2	DF	P	RMSEA	CFI	SRMR	AIC
29.79	9	0.00	0.10	0.96	0.05	53.79

Figure 6.15: Baseline depression re-specified measurement model

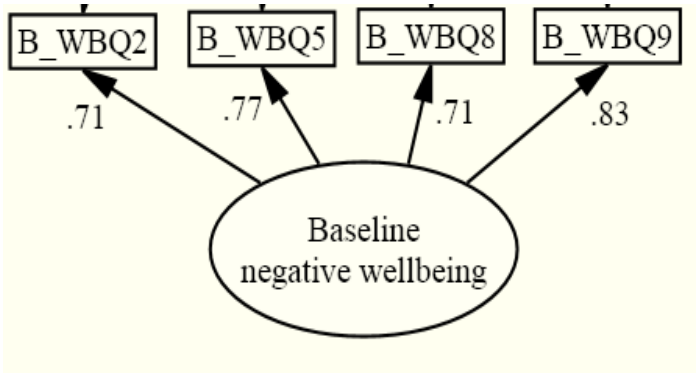


Key: scores adjacent to the first order latent variables represent variance explained by the second order latent (R^2)

Model fit indices: Baseline depression re-specified (n=248)

χ^2	DF	P	RMSEA	CFI	SRMR	AIC
9.91	8	0.27	0.03	1.00	0.03	35.91

Figure 6.16: Baseline negative well-being measurement model



Model fit indices: baseline negative well-being (n=252)

χ^2	DF	P	RMSEA	CFI	SRMR	AIC
6.26	2	0.04	0.09	0.99	0.02	22.26

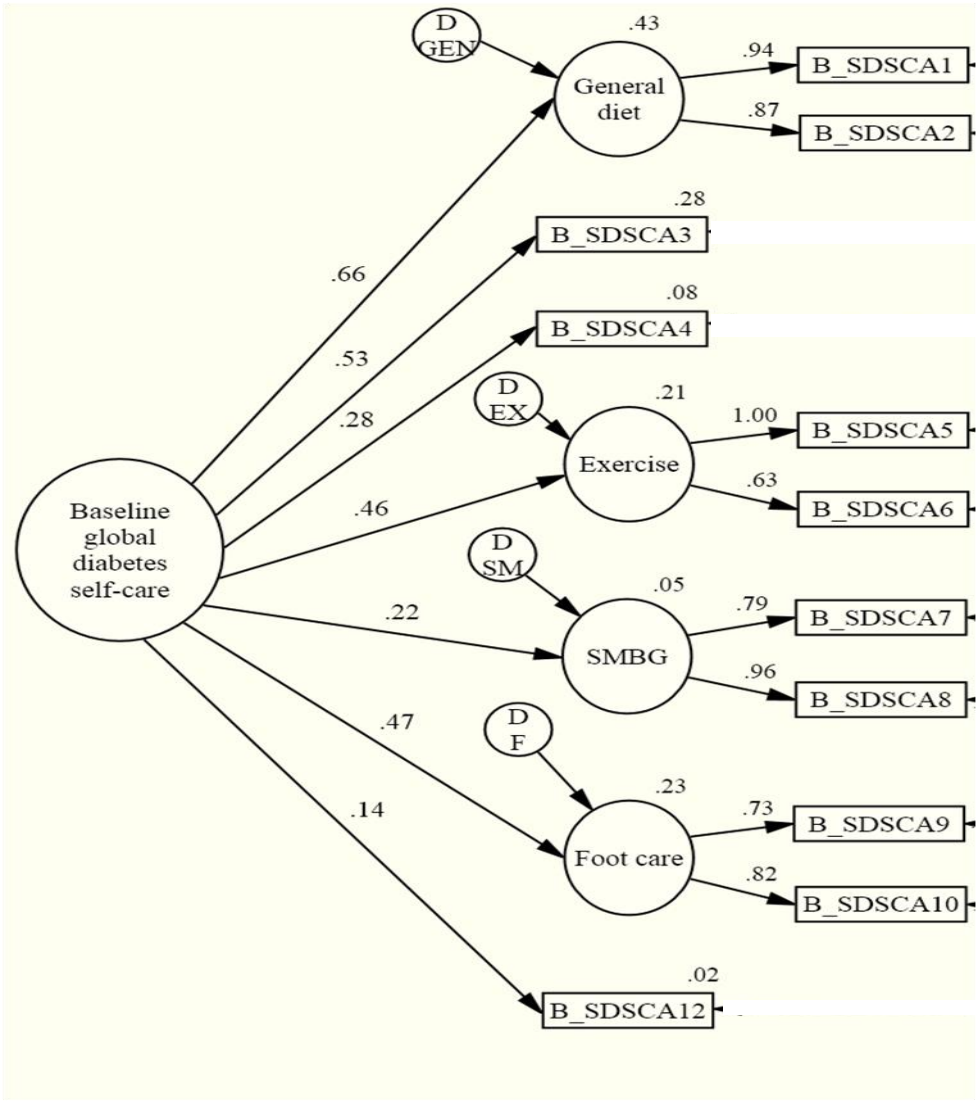
6.2.4 *CFA on the second order measurement model of the SDSCA*

The second order measurement model of the SDSCA hypothesised that each diabetes self-care component would be driven by an underlying global diabetes self-care construct as is shown in Figure 6.17. The global model of diabetes self-care was the conceptualisation used in all subsequent structural models and therefore evaluation of each SDSCA subscale was not explored singularly. In addition each SDSCA subscale could not be explored singularly as it would have resulted in each model being under-identified (See Chapter Three, Section 3.4.3.2).

The second order global diabetes self-care model demonstrated excellent fit to the sample data on all model fit indices with the exception of the model chi-squared statistic. All first order and second order factor loadings were statistically significant, excluding the factor loading for item 12 onto global diabetes self-care. This item measured participant's self-reported levels of adherence to their medication regimen. The majority of the factor loadings were large (≥0.40), but the first order construct self-monitoring of blood glucose had a lower factor loading (β=0.22) relative to the remaining first order constructs. Please see Statistical Appendix B, Table B17 for a tabular summary of the statistical outputs. For these reasons item 12 and the two self-monitoring items were removed as indicators of global diabetes self-care to maintain model parsimony consistent with the recommendations of Floyd and Widaman (1995).

The revised global diabetes self-care baseline measurement model is shown in Figure 6.18. All its fit indices were excellent and all standardised factor loadings ranged from 0.26 to 0.94 and were statistically significant. Whilst the factor loading of item 4 of the SDSCA (adherence to a diet low in saturated fat) was relatively low, the item was retained because this offered a greater degree of multi-dimensional measurement. In addition, it was deemed to be conceptually similar to the remaining SDSCA items that were retained for the revised model of global diabetes self-care. Examination of the modification indices demonstrated no evidence of item cross-loadings for the first order latent variables. Please see statistical Appendix B, Table B18 for a tabular summary of the statistical outputs.

Figure 6.17: Baseline global diabetes self-care measurement model

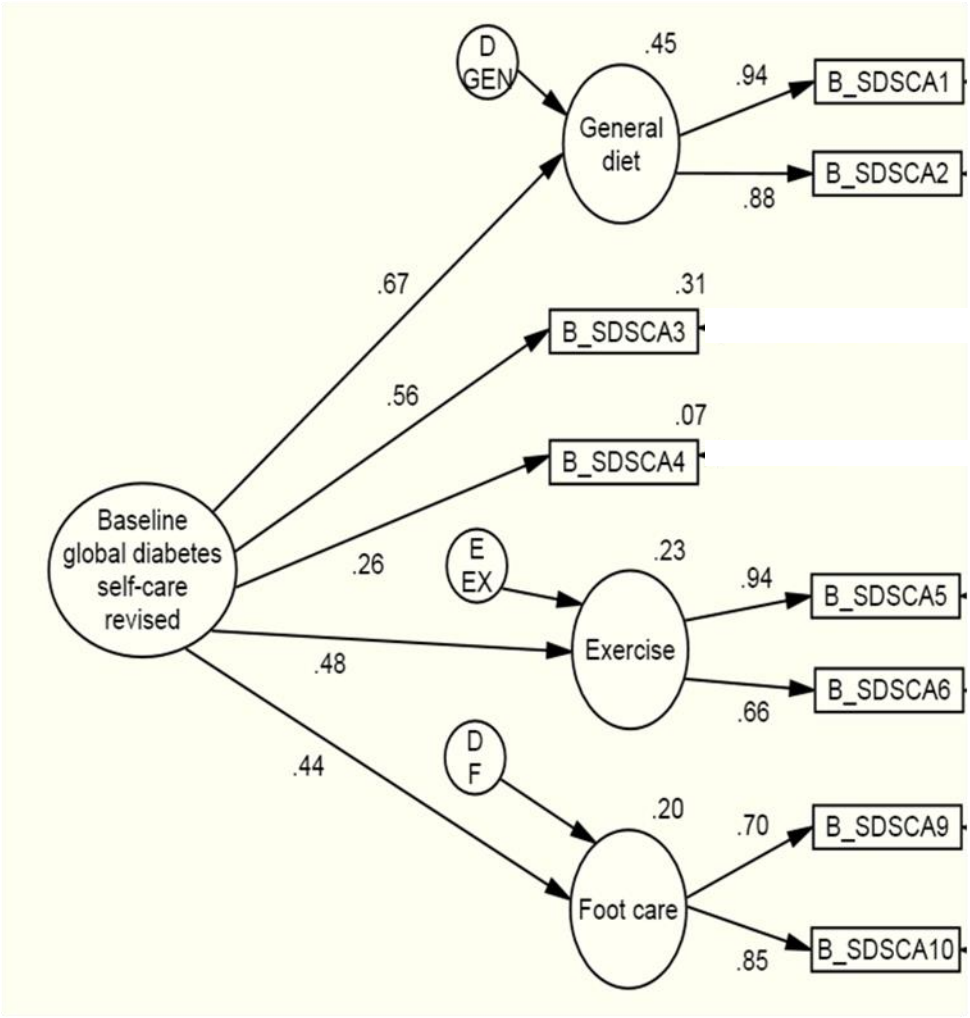


Key: first order latent variables include general diet, exercise, SMBG (self-monitoring blood glucose) and foot care; scores above first order latent variables or items represent variance explained by the second order factor (R^2); latent variables associated with the first order latent variables represent disturbance terms; D gen, disturbance general diet; D Ex, disturbance exercise; D SM, disturbance self-monitoring blood glucose; D F, disturbance foot care

Model fit indices: Baseline global diabetes self-care (n=230)

χ^2	DF	P	RMSEA	CFI	SRMR
60.25	40	0.02	0.05	0.97	0.06

Figure 6.18: Baseline global diabetes self-care re-specified measurement model



Key: First order latent variables include general diet, exercise, and foot care; scores above first order latent variables or items represent variance explained by the second order factor (R^2); latent variables associated with the first order latent variables represent disturbance terms; D gen, disturbance general diet; D Ex, disturbance exercise; D F, disturbance foot care

Model fit indices: Baseline global diabetes self-care re-specified (n=239)

χ^2	DF	P	RMSEA	CFI	SRMR
17.64	17	0.41	0.01	1.00	0.03

6.3 *Chapter summary*

This chapter tested measurement models for cognition, poor emotional health, and diabetes self-care. These analyses demonstrated that all cognition and diabetes self-care variables had good discriminant validity. However the poor emotional health subscales depression and anxiety demonstrated a high degree of overlap with a correlation of 0.88.

Each questionnaire subscale was also tested singularly through the generation of a series of separate measurement models. Some subscales required a degree of modification largely as a result of reverse and positively scored items clustering together or because of low item factor loadings. Once model modifications were applied all subscales demonstrated good fit to the sample data. The modified subscales are used in subsequent results chapters[6].

The high correlation between depression and anxiety prompted the testing of a theoretically competing concept of poor emotional health, defined as negative well-being. This competing conceptualisation of poor emotional health was well-fitting and was deemed to provide a superior representation of poor emotional health relative to depression and anxiety singularly. Given this finding structural models presented in the following chapters will contain poor emotional health constructs conceptualised in two different ways:

i. depression and anxiety defined as being conceptually distinct (as originally hypothesised by Bradley, 1994).
   AND
ii. depression and anxiety defined as being overlapping constructs as represented by the negative well-being subscale.

While depression and anxiety were highly correlated they were retained for testing because there was no evidence of item cross-loadings between each of the subscales (e.g. subscales were uni-dimensional) therefore each subscale was measuring a different yet overlapping concept. Chapters seven and eight present the analyses performed to establish the directional relationships that exist between cognitions, poor emotional health, and diabetes

[6] Because the conceptual content of the revised global diabetes self-care differed from the conceptualisation used in Chapter Five, Statistical Appendix A, Tables A20 to A27 present the baseline and follow-up univariate and bivariate statistics for the revised global diabetes self-care subscale. These tables also show baseline and follow-up univariate and bivariate statistics for poor emotional health when defined as negative well-being.

self-care. Chapter seven presents the findings for poor emotional health defined as two conceptually distinct constructs: depression and anxiety. Chapter eight presents the findings for the theoretically alternative conceptualisation of poor emotional health as negative well-being.

7 *Structural models of the simultaneous relationships between cognitions, depression, anxiety, and diabetes self-care*

7.1 *Chapter outline*

This chapter performs the necessary statistical analyses to address the primary research hypotheses. In this chapter poor emotional health is conceptualised as two distinct constructs: depression and anxiety as originally hypothesised by Bradley (1994). A reminder of the primary research hypotheses are provided below.

Hypothesis one: Diabetes cognitions and poor emotional health have a bi-directional relationship (cognitions ↔ emotions).

Hypothesis two: Diabetes cognitions and poor emotional health have a combined and independent effect on diabetes self-care, specifically:

iii. Diabetes cognitions have a direct and indirect effect (via poor emotional health) on diabetes self-care (cognitions →diabetes self-care *and* cognitions → emotions →diabetes self-care).
iv. Poor emotional health has a direct and indirect effect (via diabetes cognitions) on diabetes self-care (cognitions → diabetes self-care *and* emotions →cognitions → diabetes self-care).

7.2 *Statistical analyses*

To address the primary research hypotheses a number of sequential analyses were performed. All identified pathways were tested simultaneously in a final model.

7.2.1 *Identifying variables for testing using SEM*

As discussed in Chapter Four, Section 4.4 it was not possible to identify key diabetes cognition domains for testing in relation to both poor emotional health and diabetes self-care because few studies have tested these relationships longitudinally among adults with Type 2 diabetes (see Chapter Two). As a consequence it was necessary to empirically identify the most important relationships between diabetes cognitions, poor emotional health, and diabetes self-care. Therefore bivariate *and* multivariate regression analyses were used to identify longitudinal relationships between:

i. Diabetes cognitions and poor emotional health (research hypothesis one)
ii. Diabetes cognitions and diabetes self-care (research hypothesis two)
iii. Poor emotional health and diabetes self-care (research hypothesis two)

Both bivariate and multivariate regression analyses were performed and variables were retained for testing using SEM if they demonstrated a statistically significant relationship with the dependent variables at the level of $p \leq 0.10$ in either the bivariate or multivariate analysis, to prevent variables from being prematurely excluded in the initial stages. Bootstrapped bias corrected standard errors and associated p values were used because variables were non-normally distributed. No bootstrapping was applied when global diabetes self-care was the dependent variable because of its normal distribution.

For bivariate regression models each baseline independent variable was entered as a predictor of the dependent variable at follow-up, whilst statistically controlling for baseline scores on the dependent variable only (see Figure 7.1). This allowed an examination of the effect of each predictor variable on the dependent variable, independent of baseline dependent variable scores.

A series of separate bivariate regression models were therefore performed to explore the following relationships longitudinally:

i. The explanatory ability of baseline diabetes cognition's on poor emotional health at six months follow-up and vice versa (cognition → emotion *and* emotion → cognition).
ii. The explanatory ability of baseline diabetes cognition's on global diabetes self-care at six months follow-up and vice versa. (cognition → global diabetes self-care *and* global diabetes self-care → cognition). Whilst the effect of global diabetes self-care on diabetes cognitions was not a primary research hypothesis Tomarken and Waller (2003) recommend exploring equally plausible theoretical models.
iii. The explanatory ability of poor emotional health on global diabetes self-care at six months follow-up and vice versa (emotion → global diabetes self-care *and* global diabetes self-care → emotion). The effect of global diabetes self-care on poor emotional health was explored for the reasons described above (point ii).

For the multivariate regression analyses backwards elimination regression models were used. This type of regression model was adopted in preference to full or forwards entry

methods because it is the more inclusive approach to the identification of key variables when there is potential for a high degree of co-variance between predictors. Thus, variables other than the most dominant predictor are more likely to be identified. The following multivariate backwards elimination regression models were performed for each of the directional pathways of interest:

i. *Cognition → emotion (See Figure 7.2a):* All baseline subscales of the IPQ-R and BMQ (excluding the emotional representation subscale because of its conceptual overlap with the depression and anxiety subscale) were entered as simultaneous explanatory variables of both depression and anxiety at six months follow-up, whilst statistically controlling for baseline scores on the dependent variable.

ii. *Emotion →cognition (See Figure 7.2b):* All baseline subscales of the WBQ were entered as simultaneous explanatory variables of each diabetes cognition domain at six months follow-up, whilst statistically controlling for baseline scores on the dependent variable. All WBQ subscales were included as predictors to establish whether the depression and anxiety subscales demonstrated an effect on cognitions independent of positive mood and energy levels.

iii. *Cognitions + emotion →global diabetes self-care (See Figure 7.2c):* All baseline subscales of the IPQ-R (excluding emotional representation), BMQ, and WBQ were entered as simultaneous explanatory variables of global diabetes self-care at six months follow-up, whilst statistically controlling for baseline scores on global diabetes self-care.

Figure 7.1: Variables entered into the bivariate regression analyses

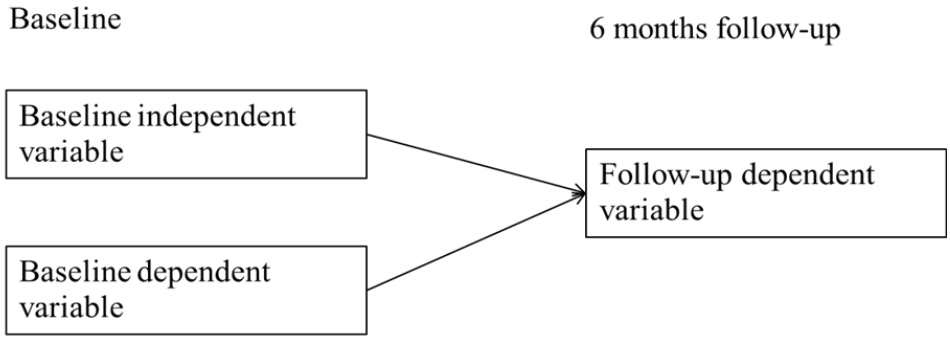


Figure 7.2: Summary of the independent and dependent variables analysed in the backwards elimination regression models

Figure 7.2a: Diabetes cognitions as predictors of poor emotional health

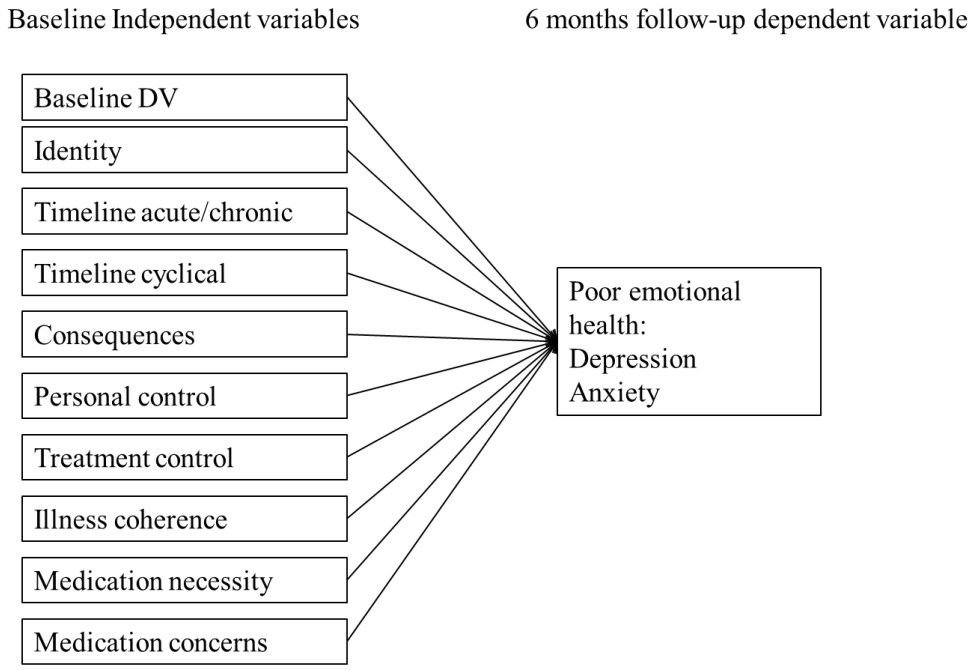


Figure 7.2b: Emotional health as predictors of diabetes cognition domains

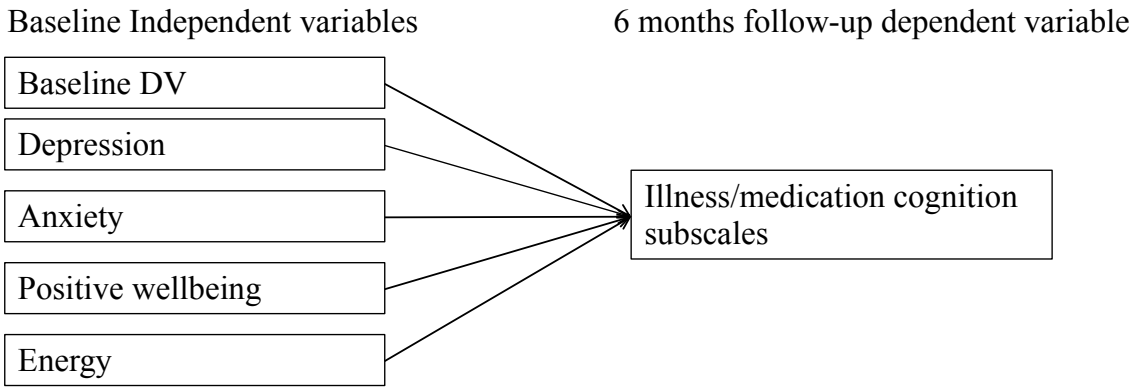
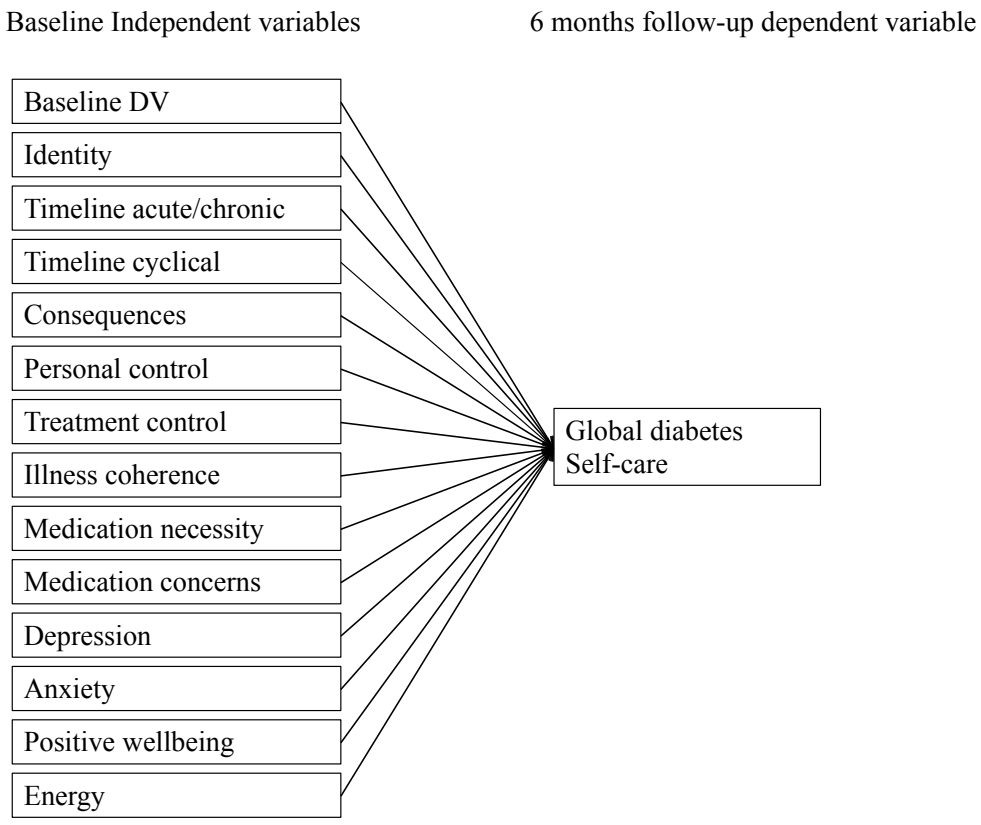


Figure 7.2c: Diabetes cognitions and emotional health as predictors of global diabetes self-care



Key: DV, Dependent variable

Whilst the measurement models presented in the previous chapter highlighted some problems with a number of subscales, these problems were not because of a lack of construct validity (e.g. all items loaded as hypothesised, excluding item 20 for the

treatment control subscale "My treatment will be effective in curing my diabetes."). In addition whilst the treatment control subscale was not included in the final CFA model of the full IPQ-R the treatment control subscale was still included as an independent variable in both the bivariate and multivariate regression models. This approach was taken: i) to allow all subscales using traditional regression based methods to be explored and ii) because it was hypothesised that the problems observed for the treatment control subscale were not because of a lack of validity but because the re-specified treatment control subscale was restricted to having two indicators per first and second order latent variable (See Chapter Six, Figure 6.4), which are known to be problematic in SEM particularly when estimating complex models.

In addition, whilst the CFA of the WBQ presented in the previous chapter indicated a large correlation between the depression and anxiety subscales, tests of multicollinearity for all linear regression models in the present chapter were within acceptable range (variance inflation factor scores were ≤ 10). Thus depression and anxiety were entered as two independent constructs for the relevant regression analyses.

In sum, it was felt appropriate to use regression models to identify longitudinal relationships between cognition, emotion, and diabetes self-care for more rigorous and simultaneous testing using SEM.

7.2.2 *Structural equation modelling*

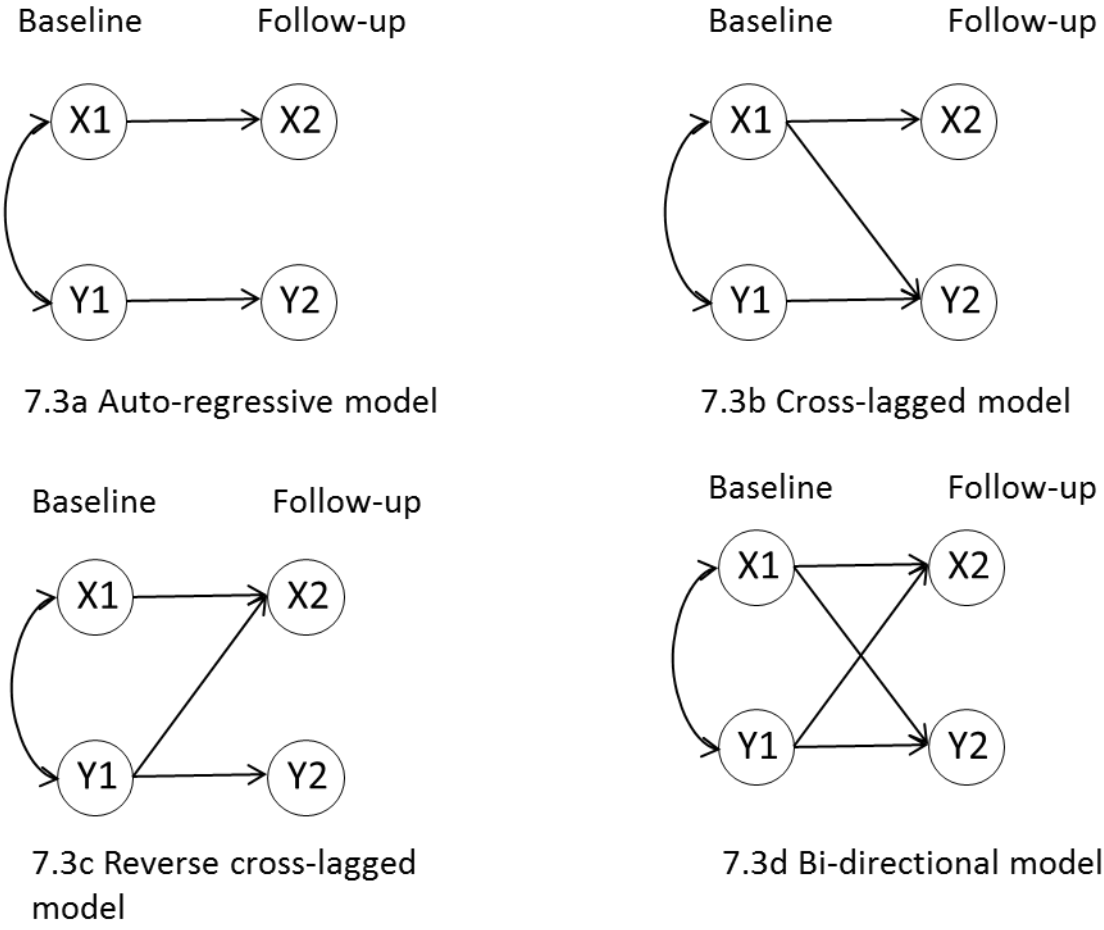
Associations identified from the regression analyses were then tested using SEM. Relationships between variables were initially tested in pairs. Paired models therefore consisted of a series of separate cognition-emotion, cognition-diabetes self-care, and emotion-diabetes self-care paired models. Each of these paired models consisted of the relevant latent variables consistent with the model specifications identified in chapter six.

7.2.3 *Nested cross-lagged analyses*

Nested cross-lagged analyses statistically test a series of separate a priori models. Each model contains the same variables but the specified directional relationships between them differ. This approach aids with the interpretation of the directional dominance of pathways between variables and encourages model parsimony (Cole and Maxwell, 2003; Martens and Haase, 2006; Selig and Little, 2012). Four theoretically plausible directional models

are evaluated and are shown in an abstract manner in Figure 7.3 below. For ease of presentation only hypothetical latent variables are depicted.

Figure 7.3: Nested model approach to testing directional relationships



The first theoretically plausible model tested is the auto-regressive model and is shown in Figure 7.3a. This model is the most parsimonious model and states that baseline variables only have a directional effect on themselves at follow-up and that independent variables co-vary with each other. This model serves as a baseline model to which subsequent directional pathways are sequentially added. Whether the addition of a pathway to the auto-regressive model makes a statistically significant improvement to model fit is evaluated using the chi-squared difference test. If its addition improves fit (e.g. there is a statistically significant decrease in the model chi squared statistic) it is retained, otherwise the most parsimonious model (e.g. fewer pathways) is favoured. This is conceptually similar to exploring the change in R^2 with hierarchical regression analysis.

Using this approach each of the competing directional models is tested relative to the auto-regressive model. For example Figure 7.3b depicts the directional effect of baseline variable x on variable y at follow-up and Figure 7.3c depicts the reverse directional effect of baseline variable y on variable x at follow-up. If either or both of these models provide a statistically significant improvement in model fit relative to the auto-regressive model they are retained and the fully cross-lagged model is tested (Figure 7.3d). The statistical fit of the cross-lagged model is compared with the best fitting model which preceded it (e.g. either model 7.3b or 7.3c); if no statistically significant differences are observed then the best fitting parsimonious model is retained.

For the cognition-emotion paired models the following theoretically plausible pathways were tested relative to the baseline auto-regressive model:

Model 1: cognition → emotion, a directional pathway was added to the baseline model leading from the cognition latent variable to the emotion latent variable only.

Model 2: emotion → cognition, a directional pathway was added to the baseline model leading from the emotion latent variable to the cognition latent variable only.

Model 3: fully cross-lagged, if either models one and/or two demonstrated superior fit to the baseline model then the fully cross-lagged (bi-directional) model was explored. The goodness of fit of this model was compared with the best fitting model which preceded it (e.g. either model one or two). The same nested model approach was applied to the cognition-diabetes self-care and emotion-diabetes self-care paired models.

7.2.4 *Tests of measurement stability*

Analyses were also performed to assess the robustness of the findings when the latent constructs were assumed to be fully stable over time. For this analysis the structural models were specified such that the factor loadings for each latent variable remained constant across time (equality constraints). If the model fit indices and path coefficients remain largely unchanged under these equality constraints this supports the conclusion that the findings are not a function of changes (actual or due to random error) in the constructs themselves (Cole and Maxwell, 2003; Little, et al., 2007).

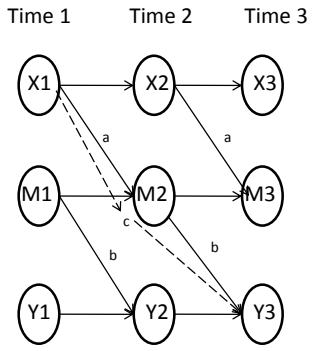
7.2.5 *Testing the simultaneous relationship between cognitions, poor emotional health, and diabetes self-care*

Directional pathways between variables identified from the paired structural models as making a statistically significant improvement to model fit were retained for testing in the final model. This model simultaneously evaluated the relationships between multiple cognition-emotion variables and global diabetes self-care. This model allowed hypotheses one and two to be evaluated simultaneously, providing a more robust test of the claims of the CS-SRM.

Having only two time points of data collection prevented the mediatory pathways specified in research hypothesis two from being fully tested longitudinally. Cole and Maxwell's (2003) recommendations for testing longitudinal mediation when limited to two time points were therefore adhered to.

These authors argue that under the assumptions of *stationarity* mediation can be tested longitudinally with two time points. Stationarity assumes that the relationships between variables remain unchanged over time. An example of this assumption is shown in Figure 7.4. When this holds true an estimate of an independent/mediator variable at time one on a dependent variable at time two (e.g. M1 to Y2) is deemed to be equivalent to the estimated effect of an independent/mediator variable at time two on a dependent variable at time three (e.g. M2 to Y3); thus the effect of the mediator on the dependent variable can be estimated longitudinally. This approach however assumes full mediation and path c cannot be estimated in an unbiased manner.

Figure 7.4: An example of longitudinal mediation with two time points



*For ease of presentation covariances, indicators, error terms, and disturbance terms have are not shown in the figure. Time 3 was not measured in this thesis.

Using these recommendations, longitudinal mediation was assumed to be present if the following two conditions were met:

Condition a: The independent variable at time one demonstrated a statistically significant relationship with the mediating variable at time two, whilst statistically controlling for the mediator at time one (e.g. path *a* of Figure 7.4 from time one to time two).

and

Condition b: The mediating variable at time one demonstrated a statistically significant relationship with the dependent variable at time two, whilst statistically controlling for the dependent variable at time one (e.g. path *b* of Figure 7.4 from time one to time two).

Variables meeting conditions *a* and *b* were identified from the separate cross-lagged paired models. Testing these relationships in the final model allowed multiple mediatory pathways to be evaluated simultaneously where applicable.

7.2.6 *Statistical control for confounding variables*

To establish whether the identified simultaneous relationships between cognitions, poor emotional health, and diabetes self-care occurred independently of a participant's number of diabetes complications (used as a marker of diabetes severity) the effect of this confounder was added to the final model. Number of diabetes complications was specified to have a direct effect on each of the dependent variables and to correlate with each of the independent variables. This approach is consistent with adding a covariate in a regression model (Little, et al., 2007).

7.2.7 *Sensitivity analyses*

Complete cases analyses were used for all SEM analyses. To establish whether this approach to handling missing data impacted on the findings of the final model a sensitivity analysis was performed. This analysis explored whether findings differed when direct maximum likelihood estimation of missing values was applied to the final model. Data were imputed for all participants who returned baseline questionnaires (n=261).

Statistical evaluation of structural models

The criteria applied to model fit indices (e.g. χ^2, RMSEA, CFI, SRMR) in chapter six were also applied in this chapter (See Chapter Six, Section 6.1).

All measurement and structural models did not meet the assumptions of multivariate normality (Mardia's index critical ratio score $\geq \pm 5$) and parameter estimates within each model were therefore bootstrapped. Bias corrected standard error and associated 95% confidence intervals for unstandardised model parameters were used to interpret their degree of statistical significance.

7.3 *Results: Cognition-depression, cognition-anxiety, and cognition + emotion-diabetes self-care regression models*

Tables 7.1 to 7.3 present a summary of the findings from the bivariate and multivariate regression analyses used to identify variables for further testing using SEM. To conserve space the full statistical outputs of each regression model are not presented here; instead Table 7.1 to 7.3 summarises the standardised beta coefficient and associated p values for:

i. Each cognition domains explanatory effect on depression and anxiety
ii. Depression and anxiety's explanatory effect on each cognition domain
iii. Cognition and emotion's (depression and anxiety) explanatory effect on global diabetes self-care
iv. Global diabetes self-care explanatory effect on cognition domains and emotion (depression and anxiety)

Variables in italics are those that had relationship at the level of $p \leq 0.10$.

In total 15 paired relationships were identified for further testing using SEM and Table 7.4 summarises each of the cognition-depression, cognition-anxiety, cognition-diabetes self-care, and emotion-diabetes self-care pairs identified.

Table 7.1: A summary of the findings from the bivariate and multivariate regression analyses used to identify cognition-depression pairs

Cognition →Depression	Depression →Cognition*
Bivariate findings	*Bivariate findings*
Identity: β=0.01, p=0.92	Identity: β=0.10, p=0.17
Timeline acute/chronic: β=-0.06, p=0.42	Timeline acute/chronic: β=0.01, p=0.84
Timeline cyclical: β=0.07, p=0.32	*Timeline cyclical: β=0.17, p=0.01*
Consequences: β=0.12, p=0.08	Consequences: β=-0.03, p=0.67
Personal control: β=0.06, p=0.36	Personal control: β=-0.05, p=0.53
Treatment control: β=-0.13, p=0.03	Treatment control: β=-0.04, p=0.50
Illness coherence: β=-0.05, p=0.50	*Illness coherence: β=-0.13, p=0.02*
Medication necessity: β=0.08, p=0.11	Medication necessity: β=0.06, p=0.34
Medication concerns: β=0.19, p=0.00	Medication concerns: β=0.09, p=0.18
Multivariate findings	*Multivariate findings*
Identity: β=-0.03, p=0.61	Identity: β=-0.03, p=0.76
Timeline acute/chronic: β=-0.10, p=0.17	Timeline acute/chronic: β=-0.06, p=0.47
Timeline cyclical: β=0.03, p=0.64	Timeline cyclical: β=0.02, p=0.83
Consequences: β=0.05, p=0.41	Consequences: β=-0.11, p=0.12
Personal control: β=0.18, p=0.01	Personal control: β=-0.12, p=0.24
Treatment control: β=-0.16, p=0.01	Treatment control: β=0.05, p=0.52
Illness coherence: β=0.04, p=0.67	*Illness coherence: β=-0.12, p=0.02*
Medication necessity: β=0.08, p=0.06	Medication necessity: β=-0.03, p=0.67
Medication concerns: β=0.24, p=0.00	Medication concerns: β=0.00, p=0.98

*Cognition domains were the outcome variables in the regression models. The beta coefficients and p values represent depression as a predictor of each cognition domain.

Table 7.2: A summary of the findings from the bivariate and multivariate regression analyses used to identify cognition-anxiety pairs

Cognition →Anxiety	Anxiety →Cognition*
Bivariate findings	*Bivariate findings*
Identity: β=-0.01, p=0.82	Identity: β=0.13, p=0.16
Timeline acute/chronic: β=-0.03, p=0.63	Timeline acute/chronic: β=0.07, p=0.33
Timeline cyclical: β=0.08, p=0.16	*Timeline cyclical: β=0.24, p=0.00*
Consequences: β=0.08, p=0.16	Consequences: β=0.04, p=0.60
Personal control: β=0.00, p=0.95	Personal control: β=-0.01, p=0.92
Treatment control: β=-0.08, p=0.12	Treatment control: β=-0.06, p=0.35
Illness coherence: β=-0.07, p=0.28	*Illness coherence: β=-0.12, p=0.03*
Medication necessity: β=0.11, p=0.03	*Medication necessity: β=0.12, p=0.07*
Medication concerns: β=0.12, p=0.09	*Medication concerns: β=0.20, p=0.00*
Multivariate findings	*Multivariate findings*
Identity: β=-0.03, p=0.66	Identity: β=0.05, p=0.60
Timeline acute/chronic: β=-0.05, p=0.46	Timeline acute/chronic: β=0.07, p=0.36
Timeline cyclical: β=0.05, p=0.40	*Timeline cyclical: β=0.24, p=0.00*
Consequences: β=0.03, p=0.65	Consequences: β=0.05, p=0.58
Personal control: β=0.04, p=0.51	Personal control: β=0.02, p=0.81
Treatment control: β=-0.05, p=0.41	Treatment control: β=-0.03, p=0.78
Illness coherence: β=-0.01, p=0.93	Illness coherence: β=-0.07, p=0.50
Medication necessity: β=0.09, p=0.03	Medication necessity: β=0.06, p=0.46
Medication concerns: β=0.13, p=0.09	*Medication concerns: β=0.20, p=0.00*

*Cognition domains were the outcome variables in the regression models. The beta coefficients and p values represent anxiety as a predictor of each cognition domain.

Table 7.3: A summary of the findings from the bivariate and multivariate regression analyses used to identify cognition-diabetes self-care and emotion-diabetes self-care pairs

Cognition + Emotion →Global diabetes self-care	Global diabetes self-care →cognition + emotion*
Bivariate findings	*Bivariate findings*
Identity: β=-0.01, p=0.87	Identity: β=0.02, p=0.74
Timeline acute/chronic: β=-0.07, p=0.18	Timeline acute/chronic: β=-0.02, p=0.74
Timeline cyclical: β=0.09, p=0.07	Timeline cyclical: β=0.05, p=0.40
Consequences: β=0.06, p=0.26	Consequences: β=0.08, p=0.28
Personal control: β=-0.15, p=0.01	Personal control: β=-0.01, p=0.89
Treatment control: β=-0.03, p=0.55	Treatment control: β=0.03, p=0.62
Illness coherence: β=-0.09, p=0.11	Illness coherence: β=0.04, p=0.59
Medication necessity: β=0.03, p=0.55	Medication necessity: β=0.00, p=0.93
Medication concerns: β=0.04, p=0.45	Medication concerns: β=0.01, p=0.93
Depression: β=0.00, p=0.98	*Depression: β=-0.10, p=0.06*
Anxiety: β=0.03, p=0.66	Anxiety: β=-0.04, p=0.45
Multivariate findings	*Multivariate findings*
Identity: β=-0.08, p=0.16	Not applicable
Timeline acute/chronic: β=-0.06, p=0.23	
Timeline cyclical: β=0.02, p=0.73	
Consequences: β=0.09, p=0.17	
Personal control: β=-0.22, p=0.00	
Treatment control: β=0.13, p=0.03	
Illness coherence: β=-0.06, p=0.44	
Medication necessity: β=0.01, p=0.89	
Medication concerns: β=-0.04, p=0.57	
Depression: β=-0.10, p=0.17	
Anxiety: β=0.10, p=0.11	

*Cognition and emotion domains were the outcome variables in the regression models. The beta coefficients and p values represent self-care as a predictor of each cognition and emotion domain

Table 7.4: Variable pairings identified for testing using SEM

Depression	Anxiety	Global diabetes self-care
Timeline cyclical	Timeline cyclical	Timeline cyclical
Consequences	Illness coherence	Personal control
Personal control	Medication necessity	Treatment control
Treatment control	Medication concerns	Depression
Illness coherence		
Medication necessity		
Medication concerns		

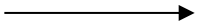
7.4 ***Results: Nested cross-lagged paired directional models***

Each of the fifteen paired associations presented in Table 7.4 were more rigorously evaluated using nested cross-lagged analyses (described above section 7.3.1) with cognition, emotion, and global diabetes self-care conceptualised as latent variables. The findings from each of the cross-lagged analyses are presented in the following order:

i. Cognition-depression paired models
ii. Cognition-anxiety paired models
iii. Cognition *and* emotion-diabetes self-care paired models

Model fit indices associated with each paired cross-lagged analyses are provided in Tables 7.6 to 7.8. Figures 7.5 to 7.8 present the standardised parameter estimates for the best fitting structural models (e.g. regression coefficients associated with each pathway). However in cases where the auto-regressive model was found to be the best fitting model parameter estimates are not presented diagrammatically as these relationships were of little theoretical interest. Statistical Appendix C, Tables C1 to C13 present a full tabular summary of all standardised and unstandardised parameter estimates for each of the best fitting models identified from the cross-lagged analyses (including those for the auto-regressive models). Table 7.5 below provides a key to aid with the interpretation of Figures 7.5 to 7.8.

Table 7.5: Key to aid with the interpretation of Figures 7.5 to 7.12

Abbreviation/Symbol	Definition
WBQ(N)	Used to represent questionnaire item numbers from the WBQ (e.g. WBQ1)
IP(N)	Used to represent questionnaire item numbers from the IPQ-R (e.g. IP1)
BMQ(N)	Used to represent questionnaire item numbers from the BMQ (e.g. BMQ1)
SC (N)	Used to represent questionnaire item number from SDSCA (e.g. SC1)
	Indicates the direction of effect
	Indicates that variables are assumed to co-vary but the direction of effect is not explicitly stated
	Squares indicate measured variables
	Ellipses/circles indicate latent variables
*	$p \leq 0.05$
**	$p \leq 0.01$

All numbers displayed on the figures excluding those adjacent to the latent variables at follow-up are standardised beta coefficients. The statistical significance of factor loadings is not shown in the figures, but all are statistically significant at $p \leq .05$ (based on bootstrapped p values) unless otherwise stated in the text. Numbers adjacent to the latent variables at follow-up represent the variance explained in the dependent variable (R^2).

7.4.1 *Cognition-emotion paired models: Depression*

A series of seven separate nested cross-lagged analyses were generated to explore the relationship between depression and the empirically identified cognition domains. However, the relationships between depression and treatment control could not be explored. Consistent with the findings reported in Chapter 6, Section 6.2.1 when the treatment control subscale was included in a model with other variables (in this case depression) it resulted in an estimation failure. The directional relationship between treatment control and depression could therefore not be explored as all plausible model re-specifications to the treatment control subscale had been applied in Chapter Six. The findings for the remaining six cross-lagged analyses are summarised below and in Table 7.6.

All theoretically plausible directional models of the relationship between depression and the cognition domains: consequences, personal control, illness coherence, medication necessity, and medication concerns had evidence of good fit on all model fit indices excluding the model chi-square statistic (See Table 7.6). However, the findings did not support either the directional effect of each of these five cognition domains on depression or vice versa (e.g. the chi-square change statistic was not statistically significant for either of the directions tested in each paired cross-lagged model). These findings resulted in the auto-regressive model being retained as the best fitting model of the relationships that exist between depression and consequences, personal control, illness coherence, medication necessity, and medication concerns. Statistical Appendix C, Tables C2 to C6 present the parameter estimates for each of the directional pathways specified in the auto-regressive models.

Likewise all theoretically plausible directional models of the relationship between timeline cyclical and depression demonstrated evidence of good fit, excluding the model chi-square statistic (See Table 7.6). The model exploring baseline timeline cyclical as a predictor of depression at six months follow-up did not provide any statistically significant improvement in model fit relative to the auto-regressive model ($\Delta\chi^2=0.00$, $p=0.99$; see Table 7.6). This pathway was not retained. However, the model exploring the reverse directional relationship with baseline depression as a predictor of timeline cyclical at six months follow-up demonstrated a statistically significant improvement in model fit relative to the auto-regressive model and therefore was retained in the model ($\Delta\chi^2=10.40$, $p=0.00$; See Table 7.6).

The above findings indicated a need to explore the fully cross-lagged model to test whether a model of the bi-directional relationship between depression and timeline cyclical improved the model fit further compared with a model consisting only of the directional effect of depression on timeline cyclical beliefs. The cross-lagged model did not statistically improve model fit ($\Delta\chi^2$=0.00, p=0.98; See Table 7.6). The model depicting depression as a predictor of timeline cyclical beliefs at six months follow-up was retained as the best fitting model. This model and its standardised regression coefficients are shown in Figure 7.5. All coefficients were statistically significant at the level of $p \leq 0.05$. A full statistical output of its standardised and unstandardised parameter estimates can be found in Statistical Appendix C, Table C1.

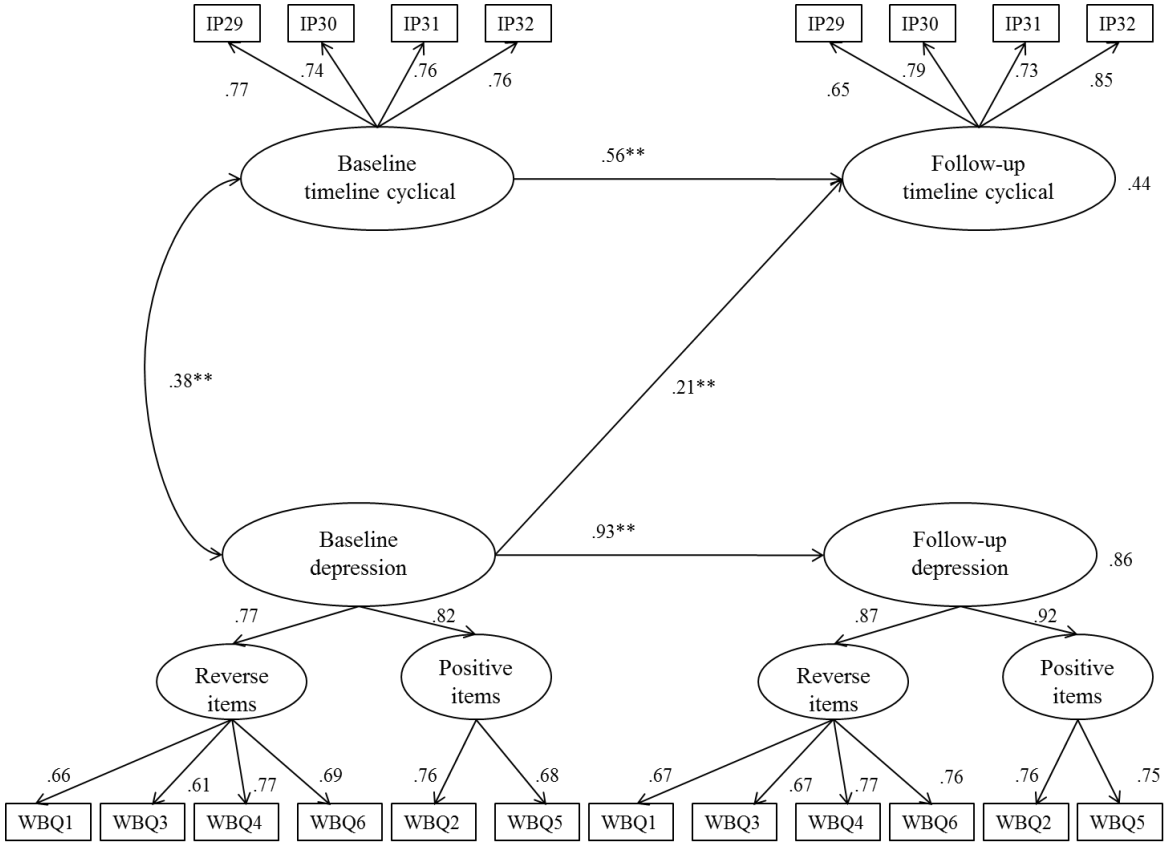
Table 7.6: Nested model analyses linking depression and diabetes cognition domains

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Depression and timeline cyclical (N=170) see Statistical Appendix C, Table C1 for model parameter estimates: depression →timeline cyclical									
Auto-regressive model	239.93	155	.00	.06	.94	.08	349.93		
Timeline cyclical to depression	239.93	154	.00	.06	.94	.08	351.93	.00	.99
Depression to timeline cyclical	234.50	154	.00	.06	.95	.07	346.50	5.43	.02
Cross-lagged	234.50	153	.00	.06	.95	.07	348.50	.00	.98
*$\Delta\chi^2$ between depression to timeline cyclical and cross lagged									
Depression and consequences (N=166) see Statistical Appendix C, Table C2 for model parameter estimates: auto-regressive model									
Auto-regressive model	285.90	191	.00	.06	.93	.06	409.90		
Consequences to depression	285.90	190	.00	.06	.93	.06	411.90	.00	.97
Depression to consequences	284.55	190	.00	.06	.93	.06	410.55	1.35	.25
Cross-lagged	278.89	189	.00	.05	.93	.06	406.89	NA	NA
Depression and personal control (N=164) see Statistical Appendix C, Table C3 for model parameter estimates: auto-regressive model									
Auto-regressive model	293.38	233	.00	.04	.96	.07	427.38		
Personal control to depression	292.47	232	.00	.04	.96	.07	428.47	.91	.34
Depression to personal control	292.66	232	.00	.04	.96	.07	428.66	.72	.40
Cross-lagged	292.03	231	.00	.04	.96	.07	430.03	NA	NA
Depression and illness coherence (N=174) see Statistical Appendix C, Table C4 for model parameter estimates: auto-regressive model									
Auto-regressive model	238.98	155	.00	.06	.95	.07	348.98		
Illness coherence to depression	238.90	154	.00	.06	.95	.07	350.90	.07	.79
Depression to illness coherence	236.28	154	.00	.06	.95	.06	348.28	2.70	.10
Cross-lagged	236.14	153	.00	.06	.95	.06	350.14	NA	NA

Table 7.6: Continued

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Depression and medication necessity (N=166) See Statistical Appendix C, Table C5 for model parameter estimates: Auto-regressive model									
Auto-regressive model	239.24	193	.01	.04	.97	.06	359.24		
Medication necessity to depression	238.99	192	.01	.04	.97	.06	360.99	.25	.62
Depression to medication necessity	237.15	192	.02	.04	.98	.05	359.15	2.09	.15
Cross-lagged	236.91	191	.01	.04	.97	.05	360.91	NA	NA
Depression and medication concerns (N=165) See Statistical Appendix C, Table C6 for model parameter estimates: Auto-regressive model									
Auto-regressive model	279.78	193	.00	.05	.95	.07	399.78		
Medication concerns to depression	279.56	192	.00	.05	.95	.07	401.56	.22	.64
Depression to medication concerns	276.92	192	.00	.05	.95	.06	398.92	2.86	.09
Cross-lagged	276.91	191	.00	.05	.95	.06	400.91	NA	NA

Figure 7.5: Depression's directional effect on timeline cyclical



7.4.2 *Cognition-emotion paired models: Anxiety*

Four separate nested cross-lagged analyses were generated to explore the directional relationship between anxiety and the empirically identified cognition variables. The findings from these analyses are presented in Table 7.7 and are summarised in the text below.

All models of the possible directional relationships between anxiety and both of the cognition domains illness coherence and medication necessity (evaluated in two separate nested model analyses) had evidence of good fit (excluding the model chi-square statistic). However the addition of pathways to the auto-regressive model leading from baseline cognition domains (either illness coherence or medication necessity) and the competing reverse directional pathway leading from baseline anxiety to either illness coherence or medication necessity did not make a statistically significant improvement in the model chi-square statistic relative to the auto-regressive model. Thus the auto-regressive model was retained as the best fitting model of the relationship between anxiety and illness coherence and anxiety and medication necessity beliefs (See Table 7.7 for the model fit indices and Statistical Appendix C, Tables C8 and C9 for a summary of the parameter estimates associated with both of these auto-regressive models).

All theoretically plausible models of the relationship between timeline cyclical and anxiety demonstrated evidence of good fit, excluding the model chi-square statistic (See Table 7.7). The model testing the directional effect of baseline timeline cyclical beliefs on anxiety at six months follow-up did not provide statistical evidence to retain this pathway. No statistically significant improvement in the chi-square statistic over and above that of the auto-regressive model was observed ($\Delta\chi^2$=0.47, p=0.49; See Table 7.7). The model testing the pathway leading from baseline anxiety to timeline cyclical at six months follow-up was upheld. This model demonstrated a statistically significant improvement in the model chi-square statistic relative to that of the auto-regressive model ($\Delta\chi^2$=10.50, $p\leq0.01$; See Table 7.7).

Given this finding, the fully cross-lagged (bi-directional model) was tested to explore whether a model which included both directional pathways provided an improvement in model fit compared with the model consisting only of the explanatory effect of anxiety on timeline cyclical. The findings did not provide statistical support for the fully cross-lagged model ($\Delta\chi^2$=0.52, p=0.47; See Table 7.7) and therefore the directional effect of anxiety on

timeline cyclical was retained as the best fitting model. The standardised regression coefficients for this model are presented in Figure 7.6 and a full tabular summary is presented in Statistical Appendix C, Table C7. All latent variable factor loadings were statistically significant at the level of $p \leq 0.05$.

The models for each of the possible relationships between medication concerns and anxiety were well fitting (See Table 7.7). No evidence was found to support the directional effect of baseline medication concerns on anxiety at six months follow-up. The addition of this pathway to the auto-regressive model did not make a statistically significant improvement to the chi-square fit index ($\Delta\chi^2=3.03$, $p=0.08$). The addition to the auto-regressive model of the directional effect of baseline anxiety on medication concerns at six months follow-up made a statistically significant improvement in the model chi-square statistic ($\Delta\chi^2=4.21$, $p \leq 0.05$). This finding prompted an examination of the fully cross-lagged (bi-directional) model which tested both directional pathways simultaneously. This model did not make a statistically significant improvement in model fit relative to the model consisting of only the explanatory effect of anxiety on medication concerns ($\Delta\chi^2=2.43$, $p=0.12$), and therefore the most parsimonious model was retained. Figure 7.7 depicts the standardised regression coefficients for the directional effect of baseline anxiety on medication concerns. A full tabular summary of the parameter estimates can be found in Statistical Appendix C, Table C10. All latent variable factor loadings were statistically significant.

Table 7.7: Nested model analyses linking anxiety and diabetes cognition domains

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Anxiety and timeline cyclical (N=171) See Statistical Appendix C, Table C7 for model parameter estimates: Anxiety → timeline cyclical									
Auto-regressive model	258.01	157	.00	.06	.94	.08	364.01		
Timeline cyclical to anxiety	257.54	156	.00	.06	.94	.08	365.54	.47	.49
Anxiety to timeline cyclical	247.51	156	.00	.06	.95	.07	355.51	10.50	.00
Cross-lagged *	247.00	155	.00	.06	.95	.07	357.00	.52	.47
*$\Delta\chi^2$ between anxiety to timeline cyclical and cross lagged									
Anxiety and illness coherence (N=174) See statistical Appendix C, Table C8 for model parameter estimates: Auto-regressive model									
Auto-regressive model	276.01	157	.00	.07	.94	.07	382.01		
Illness coherence to anxiety	275.95	156	.00	.07	.94	.07	383.95	.06	.80
Anxiety to illness coherence	273.05	156	.00	.07	.94	.06	381.05	2.96	.09
Cross-lagged	273.00	155	.00	.07	.94	.06	383.00	NA	NA
Anxiety and medication necessity (N=162) See Statistical Appendix C, Table C9 for model parameter estimates: Auto-regressive model									
Auto-regressive model	238.14	195	.02	.04	.98	.06	354.14		
Medication necessity to anxiety	237.88	194	.02	.04	.98	.06	355.88	.26	.61
Anxiety to medication necessity	237.73	194	.02	.04	.98	.06	355.73	.40	.53
Cross-lagged	237.47	193	.02	.04	.98	.06	357.47	NA	NA
Anxiety and medication concerns (N=162) See Statistical Appendix C, Table C10 for model parameter estimates: medication concerns → anxiety									
Auto-regressive model	337.35	195	.00	.07	.92	.08	453.35		
Medication concerns to anxiety	334.32	194	.00	.07	.92	.08	452.32	3.03	.08
Anxiety to medication concerns	333.14	194	.00	.07	.92	.08	451.14	4.21	.04
Cross-lagged*	330.70	193	.00	.07	.92	.07	450.70	2.43	.12
*$\Delta\chi^2$ between anxiety to medication concerns and cross-lagged									

Figure 7.6: Anxiety's directional effect on timeline cyclical

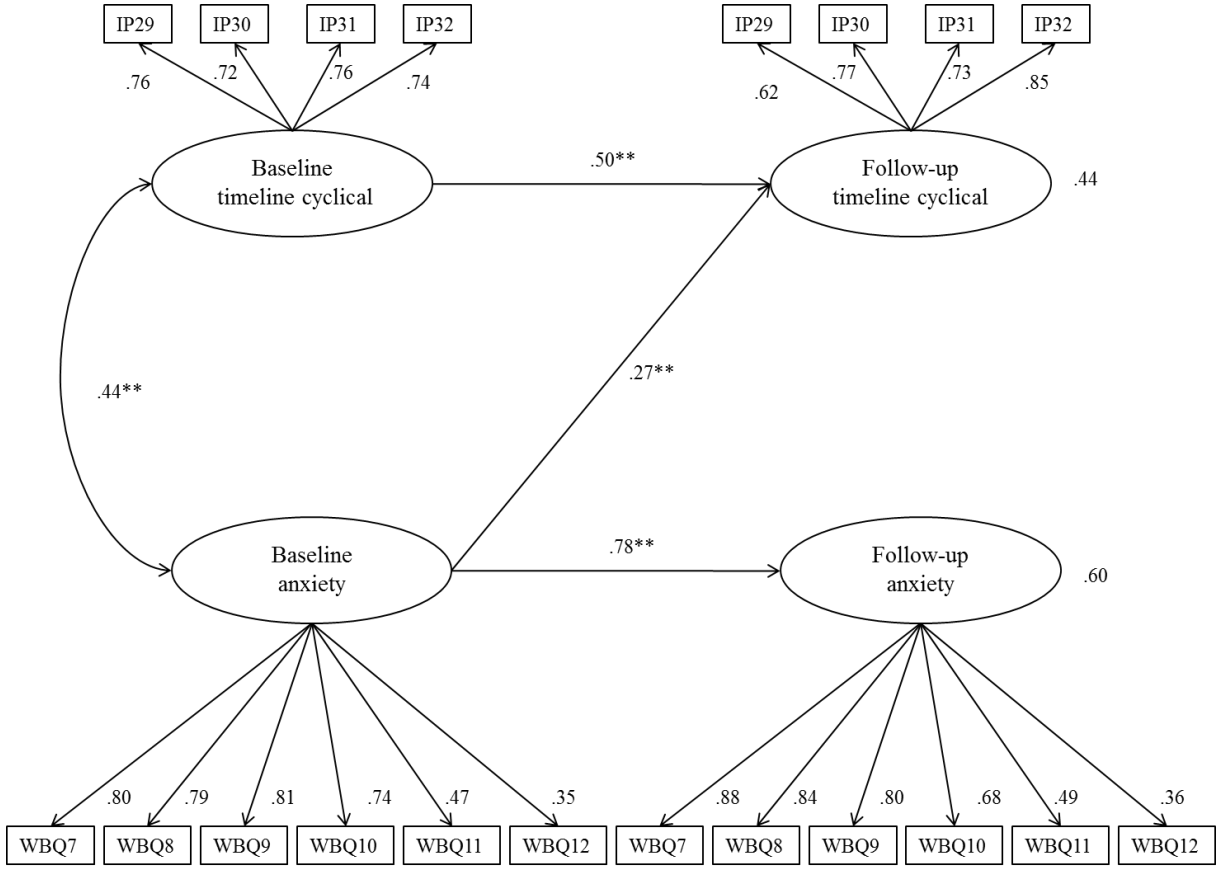
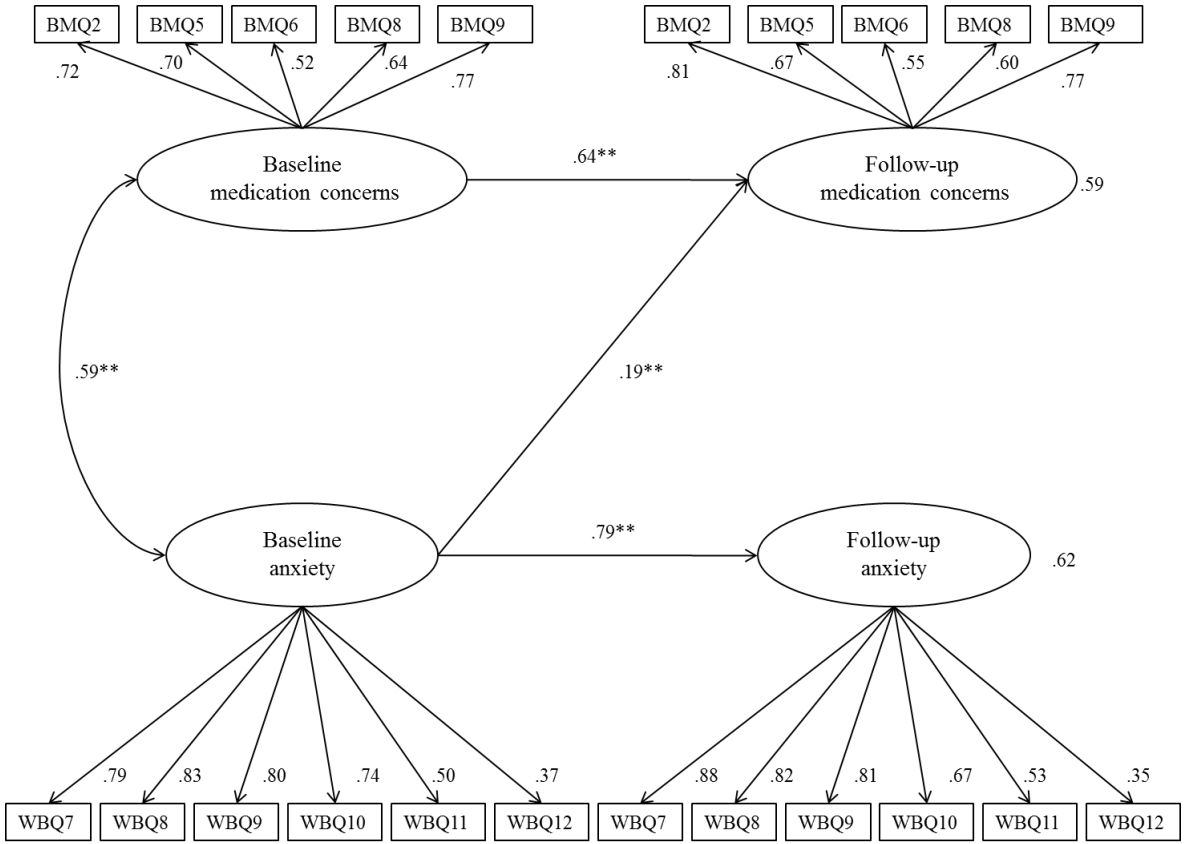


Figure 7.7: Anxiety's directional effect on medication concerns



7.4.3 *Global diabetes self-care paired models*

Four nested model analyses were performed to explore the relationships between the cognition and emotion concepts and global diabetes self-care identified from the preliminary screening phase. However the model testing the relationship between treatment control and global diabetes self-care resulted in an estimation failure, thus the relationship between these two variables could not be explored any further for the reasons described earlier.

Table 7.8 presents the model fit indices for the remaining three cognition and emotion variables. All theoretically plausible relationships between global diabetes self-care and both of the variables timeline cyclical and depression (evaluated in separate paired cross-lagged models) demonstrated evidence of good fit on all model fit indices excluding the model chi-square statistic. However for both timeline cyclical and depression the directional effect of either variable on global diabetes self-care at six months follow-up relative to the baseline auto-regressive model did not make a statistically significant improvement in model fit. The same was true for the models that explored the reverse directional pathway (e.g. the effect of baseline global diabetes self-care on either timeline cyclical or depression at six months follow-up). Given these findings the auto-regressive models were retained as the best fitting models for both timeline cyclical and depression. Statistical Appendix C, Tables C11 and C13 present a tabular summary of the directional parameters for each of these models.

All theoretically plausible models of the relationship between personal control and global diabetes self-care demonstrated good fit excluding the model chi-square statistic (See Table 7.8). The model examining the directional pathway leading from baseline personal control to global diabetes self-care at follow-up made a statistically significant improvement in model fit relative to the baseline auto-regressive model ($\Delta\chi^2$=9.68, $p\leq0.01$). Similarly for the reverse directional model, when a pathway was added to the auto-regressive model leading from baseline global diabetes self-care to personal control at follow-up a statistically significant different chi-square change statistic was found ($\Delta\chi^2$= 3.74, $p\leq0.05$).

The statistically significant pathways in both directions relative to the baseline auto-regressive model warranted the examination of the fully cross-lagged model whereby both directional pathways were tested simultaneously. The fit of the cross-lagged model was

compared with the model depicting baseline personal control as a predictor of global diabetes self-care at follow-up as this model had the most superior fit (e.g. a smaller chi-square relative to the effect of global diabetes self-care on personal control; See Table 7.8). The fully cross-lagged model did not make a statistically significant improvement in model fit relative to the explanatory effect of personal control on global diabetes self-care, therefore this model was retained and is shown in Figure 7.8. All latent variable factor loadings were statistically significant excluding item 4 of the summary of diabetes self-care activity scale at baseline. This item was retained in the model because its factor loading was statistically significant at follow-up. Statistical Appendix C, Table C12 presents a tabular summary of the parameter estimates associated with this model.

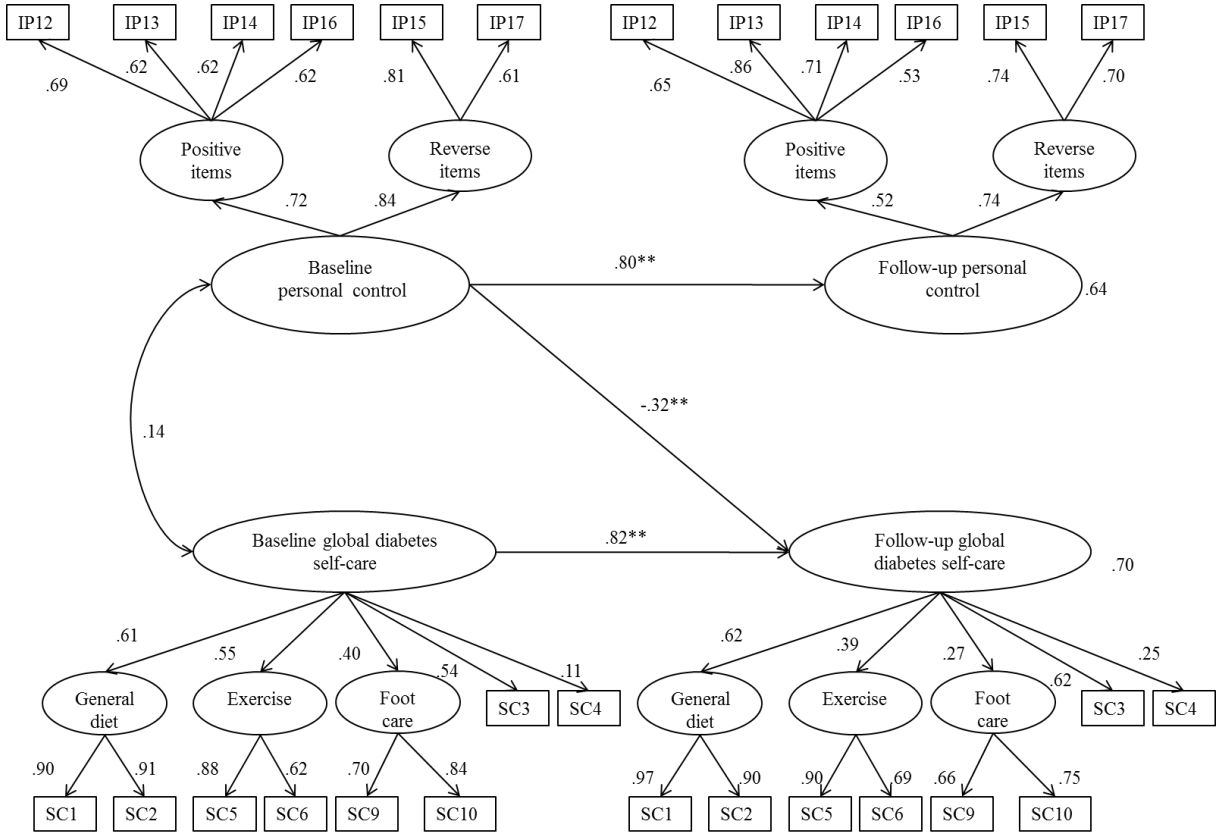
7.4.4 *Tests of measurement stability*

Analyses assuming stability of the constructs over time (factorial invariance) indicated that model fit indices and path coefficients remained largely unchanged when factor loadings were specified to be constant over time. When differences were observed in the model fit indices they occurred within the region of ±0.01 for all model fit indices excluding the model chi-square test statistic. Standardised beta coefficients for the directional pathways varied within the range of ±0.03. Greater changes in the model chi-square statistic can be anticipated as this fit index is sensitive to model degrees of freedom which substantially increase when factorial invariance is tested with equality constraints. These analyses indicate that the directional models identified in this chapter are not a function of variation over time in how latent factors psychometrically perform but reflect “true” change. The model fit indices and parameter estimates associated with these sensitivity analyses can be found in statistical Appendix C, Tables C14a to C14d.

Table 7.8: Nested model analyses linking cognitions and emotions with global diabetes self-care

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Timeline cyclical and global diabetes self-care (N=164) See Statistical Appendix C, Table C11 for model parameter estimates: auto-regressive model									
Auto-regressive model	330.39	228	.00	.05	.94	.07	474.39		
Timeline cyclical to global diabetes self-care	329.24	227	.00	.05	.94	.07	475.24	1.15	.28
Global diabetes self-care to timeline cyclical	330.39	227	.00	.05	.94	.07	476.39	.00	.98
Cross-lagged	329.24	226	.00	.05	.94	.07	477.24	NA	NA
Personal control and global diabetes self-care (N=153) See Statistical Appendix C, Table C12 for model parameter estimates: personal control → global diabetes self-care									
Auto-regressive model	465.50	322	.00	0.05	0.91	0.08	633.50		
Personal control to global diabetes self-care	455.82	321	.00	0.05	0.92	0.08	625.82	9.68	0.00
Global diabetes self-care to personal control	461.76	321	.00	0.05	0.92	0.08	631.76	3.74	0.05
Cross-lagged*	452.66	320	.00	0.05	0.92	0.08	624.66	3.16	0.08
*$\Delta\chi^2$ between personal control to global diabetes self-care and cross lagged									
Depression and global diabetes self-care (N=157) See Statistical Appendix C, Table C13 for model parameter estimates: auto-regressive model									
Auto-regressive model	422.97	322	.00	.05	.95	.07	590.97		
Depression to global diabetes self-care	422.40	321	.00	.05	.95	.07	592.40	.57	.45
Global diabetes self-care to depression	422.11	321	.00	.05	.95	.07	592.11	.86	.35
Cross-lagged	421.51	320	.00	.05	.95	.07	593.51	NA	NA

Figure 7.8: Personal control's directional effect on global diabetes self-care



7.4.5 *Summary of paired cross-lagged findings*

Figure 7.9a-c and the text below provide a summary of the directional pathways identified from the paired cross-lagged analyses for: depression and cognitions, anxiety and cognitions, and cognitions + emotions and global diabetes self-care. The following relationships were observed:

i. For depression only a directional effect on timeline cyclical beliefs at six months follow-up was observed. No directional effects were observed for the remaining cognition domains relative to their auto-regressive model (See Figure 7.9a).
ii. For anxiety cross-lagged analyses demonstrated that baseline anxiety had a directional effect on timeline cyclical and medication concerns at follow-up. No relationships in either direction were supported for the anxiety-illness coherence pairing or the anxiety-medication necessity pairing (See Figure 7.9b)
iii. Personal control was the only illness cognition domain to demonstrate a longitudinal relationship with global diabetes self-care at follow-up (personal control → global diabetes self-care). Neither timeline cyclical nor depression demonstrated a relationship with global diabetes self-care in either direction (See Figure 7.9c)
iv. Findings did not indicate the presence of longitudinal mediation occurring between cognitions, poor emotional health, and global diabetes self-care according to Cole and Maxwell's (2003) criteria.

Figure 7.9: Summary of findings from the paired directional models represented simultaneously

Figure 7.9a: Depression and diabetes cognitions

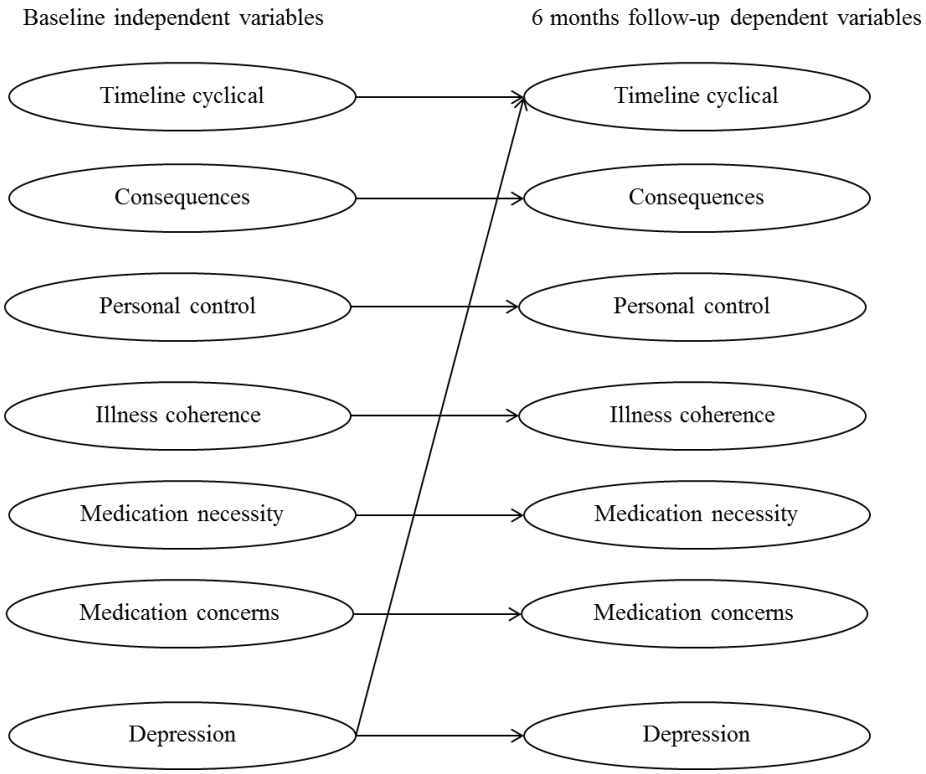


Figure 7.9b: Anxiety and diabetes cognitions

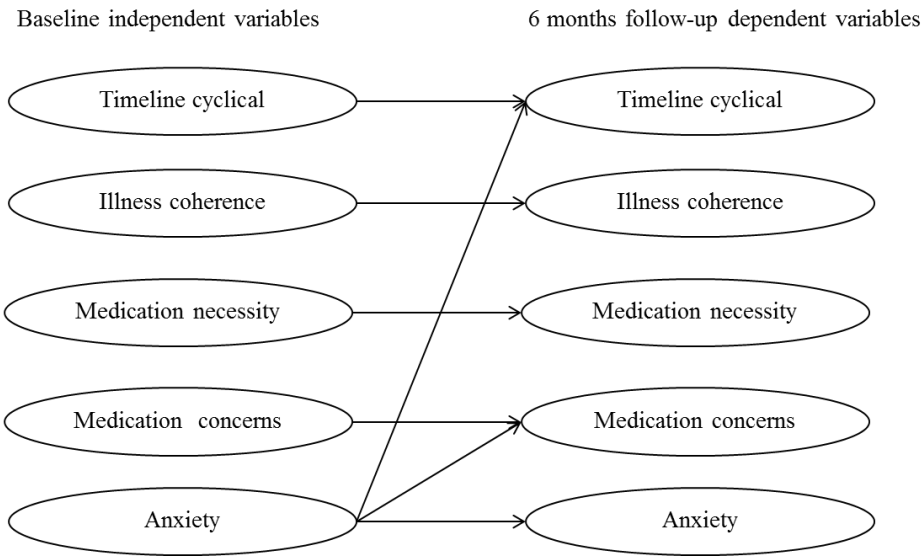
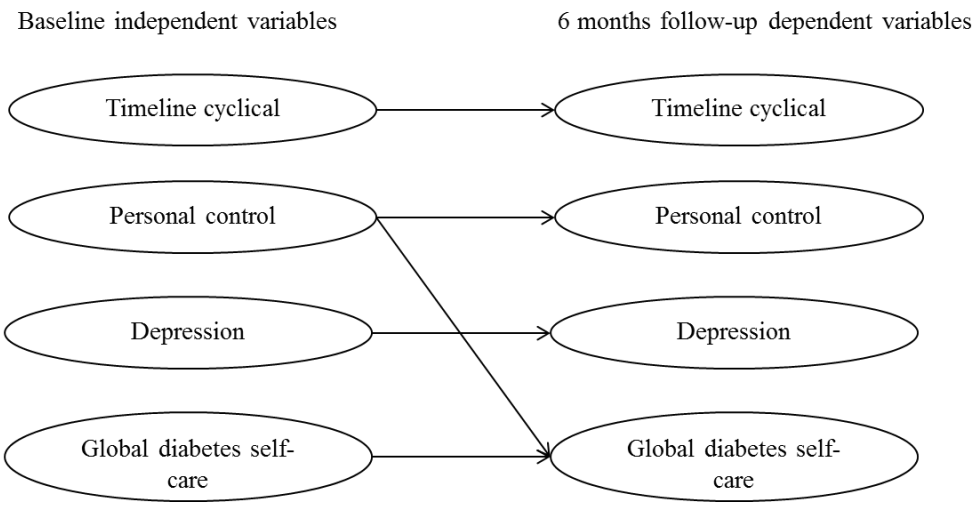


Figure 7.9c: Global diabetes self-care and diabetes cognitions + emotions



7.5 *Results of the simultaneous structural equation models: cognitions, poor emotional health, and global diabetes self-care*

The findings from the separate paired cross-lagged analyses identified the need to test a model which explored the simultaneous effect of depression and anxiety on timeline cyclical beliefs, anxiety's explanatory effect on medications concerns and the independent effect of personal control on global diabetes self-care. Testing these relationships simultaneously allowed the claims of the CS-SRM with regards to the partially independent cognitive and emotional processing systems to be more rigorously tested by evaluating all pathways in one statistical analysis.

It was not feasible to specify multiple indicator latent variables for each of the emotion and cognition variables at baseline and follow-up. This would have resulted in too many parameters being estimated relative to the number of participants who provided complete data on all cognition and poor emotional health items at baseline and six months follow-up. The item parcelling approach described in Chapter Three, Section 3.4.4 was therefore adopted to specify cognition and poor emotional health latent variables.

Item parcelling was not adopted for the global measure of diabetes self-care because of the multi-faceted nature of the construct which would not have been adequately represented using the item parcelling approach.

7.5.1 *Complete cases model: cognitions, poor emotional health, and global diabetes self-care*

The model testing the simultaneous relationships between depression, anxiety, diabetes cognitions, and global diabetes self-care demonstrated good fit to the sample data on all model fit indices excluding the model chi-square statistic (χ^2=388.9, df_m=267, p≤.01; RMSEA=.05, CFI=.94, SRMR=.07, AIC=556.9, N=159). However when the statistical significance of the parameter estimates (directional pathways) were explored, the explanatory effect of depression on timeline cyclical was statistically non-significant. To retain model parsimony this pathway was removed from the model. Parameter estimates associated with the originally specified model can be found in Statistical Appendix C, Table C15.

Figure 7.10 depicts the standardised parameter estimates for the re-specified model testing the simultaneous relationships between anxiety, cognitions, and global diabetes self-care only. The model provided good fit to the sample data excluding the model chi-square statistic (χ^2=301.0, df_m=225, p≤.01; RMSEA=0.05, CFI=0.95, SRMR=0.07, AIC=451.0; N=159). All directional pathways estimated within the model remained statistically significant specifically:

i. Increased baseline anxiety was associated with elevated scores on timeline cyclical beliefs (β=0.21, p≤0.01) and medication concerns (β=0.24, p≤0.01) at six months follow-up.
ii. Baseline anxiety and medication concerns explained 66% of the variance (R^2) in medication concerns at follow-up. Similarly, baseline anxiety and timeline cyclical explained 52% of the variance (R^2) in timeline cyclical at follow-up.
iii. Increased perceptions of personal control at baseline was associated with lower adherence to global diabetes self-care at six months follow-up (β=-0.25, p≤0.01).
iv. Baseline personal control and global diabetes self-care explained 70% of the variance (R^2) in global diabetes self-care at follow-up.
v. All baseline variables predicted themselves at follow-up with standardised beta coefficients for cognition domains ranging from 0.58 to 0.65, and the beta coefficients associated with anxiety and global diabetes self-care were 0.93 and 0.85 respectively.

vi. Correlations between baseline latent variables are depicted in Table 7.9. A summary of all unstandardised and standardised parameters estimated in the model can be found in Statistical Appendix C, Table C16.

Table 7.9: Correlations between baseline latent variables shown in Figure 7.10

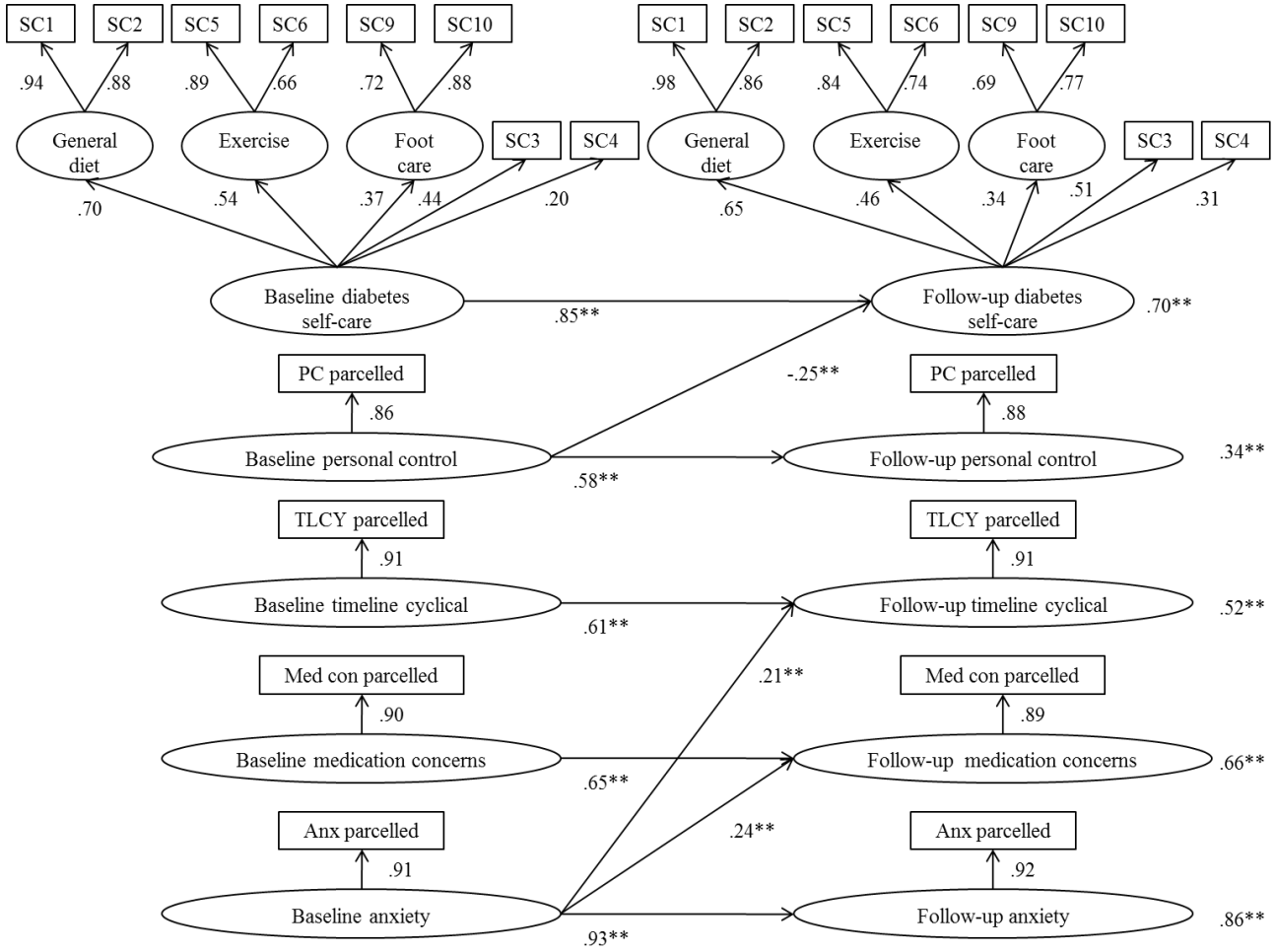
Variables	Global diabetes	Personal control	Timeline cyclical	Medication concerns
Personal control	0.18			
Timeline cyclical	0.04	-0.31**		
Medication concerns	-0.22	-0.33**	0.34**	
Anxiety	-0.02	-0.20*	0.43**	0.62**

Key: *p≤0.05. **p≤0.01 based on bootstrapped p values

Figure 7.10: Model of the simultaneous effect of anxiety, cognitions, and global diabetes self-care

Key: Anx, Anxiety; Med Con, Medication concerns; PC, Personal control; TLCY, Timeline cyclical and see Table 7.8.

For simplicity co-variances are not displayed in the figure but can be found in Table 7.9.



7.5.2 *Statistical control for confounders: clinical severity*

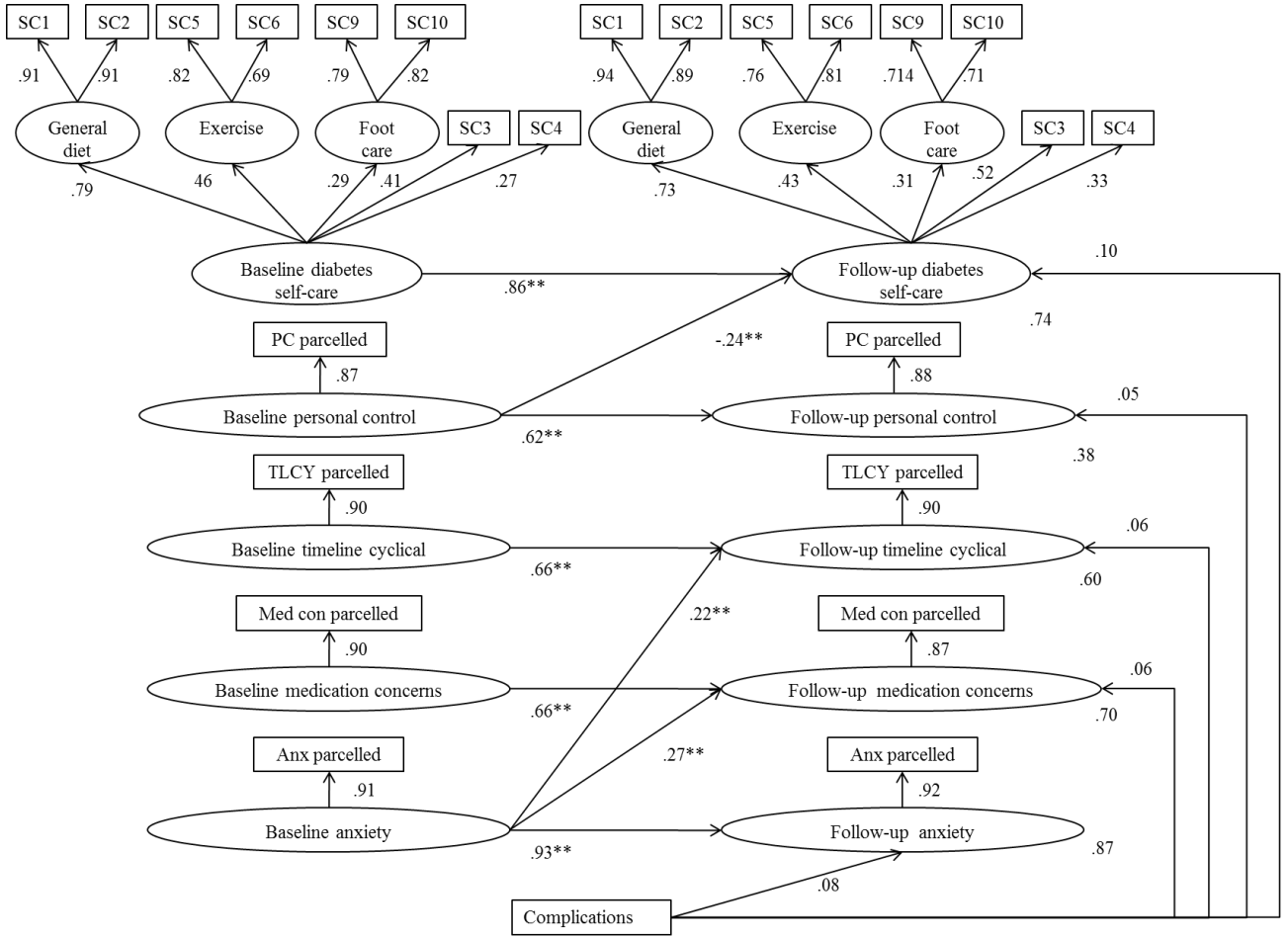
To establish whether the directional relationships identified between anxiety, cognitions, and global diabetes self-care might be accounted for by a person's level of diabetes severity, the total number of diabetes complications (as a marker of diabetes severity) was added to the model. Baseline number of diabetes complications was specified to have a directional effect on each of the dependent variables and to co-vary with each of the independent variables consistent with the recommendations of Little et al. (2007; see Figure 7.11).

Model fit became marginally worse when number of diabetes complications was added as an independent variable to the model (χ^2=333.1, df_m=240, $p \leq 0.01$; RMSEA=0.05, CFI=0.94, SRMR=0.07, AIC=503.1; N=152). This is because the model degrees of freedom increased. Number of diabetes complications only demonstrated a statistically significant relationship with baseline personal control; its relationship with the remaining variables was statistically non-significant thereby rejecting the notion that the effect of anxiety on cognition domains and the effect of personal control on diabetes self-care are spurious. More diabetes complications were however associated with decreased perceptions of personal control. A summary of parameter estimates associated with this model can be found in Statistical Appendix C, Table C17.

Figure 7.11 : Model of the simultaneous effect of anxiety, cognitions, and global diabetes self-care whilst statistically controlling for number of diabetes complications

Key: Anx, Anxiety; Med Con, Medication concerns; PC, Personal control; TLCY, Timeline cyclical and See Table 7.9.

For simplicity co-variances are not displayed in the figure (see Statistical Appendix C, Table C17).



7.5.3 *Sensitivity analyses: missing data*

To explore the impact of missing data on model fit and parameter estimates on the final model the statistical technique of imputation of missing values using direct maximum likelihood estimation was applied to provide a data set with complete cases for all participants who returned questionnaires at baseline (n=261).

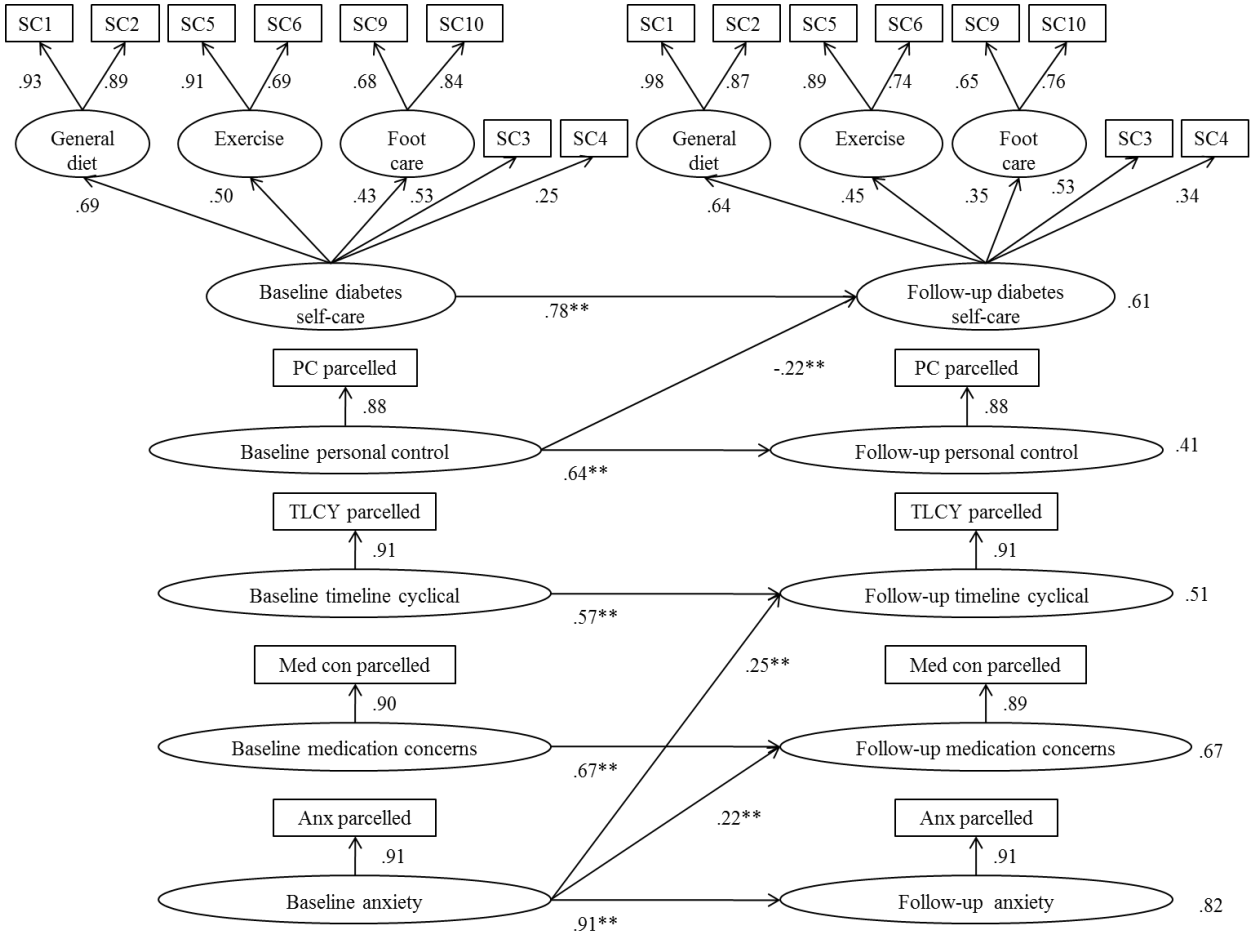
Consistent with the complete case analyses, all model fit indices demonstrated good fit to the sample data (χ^2=284.6, df_m =225, $p \leq 0.01$; RMSEA=0.03, CFI=0.97, SRMR=NA, AIC=482.6). Improvements in the fit indices of the model chi-squared statistic, RMSEA and CFI were observed, yet the AIC fit was poorer.

Similarly, effects size estimates for directional pathways were relatively the same with standardised beta coefficients ranging from ±0.02 to 0.06 compared with the complete cases list wise deletion models (see Figure 7.12). Levels of statistical significance for each of the pathways were unaltered for the direct maximum likelihood estimation model however this was based on non-bootstrapped standard errors and p values. A tabular summary of parameter estimates is provided in Statistical Appendix C, Table C18.

Figure 7.12: Model of the simultaneous effect of anxiety, cognitions, and global diabetes self-care with direct maximum likelihood estimation applied

Key: Anx, Anxiety; Med Con, Medication concerns; PC, Personal control; TLCY, Timeline cyclical and See Table 7.9.

For simplicity co-variances are not displayed in the figure (see Statistical Appendix C, Table C18).



7.6 *Chapter summary*

This chapter explored the simultaneous relationships that exist between cognitions, poor emotional health, and global diabetes self-care with poor emotional health defined as two conceptually distinct constructs: depression and anxiety.

After a series of incremental analyses the final complete cases model identified the following directional pathways:

i. Anxiety had a directional effect on medication concerns and timeline cyclical beliefs. People who were more anxious at baseline perceived diabetes to be an unpredictable condition and were more apprehensive about their diabetes medications at follow-up.
ii. Personal control predicted change in global diabetes self-care at follow-up. Participants who felt more in control of their diabetes at baseline adhered less to their diabetes self-care regimens at six months follow-up.

There was no evidence to support:

i. The directional effect of cognitions on poor emotional health
ii. The directional effect of depression on cognitions.
iii. A longitudinal relationship between poor emotional health and global diabetes self-care.

Research hypotheses one and two were thus partially supported. There was no evidence of a bi-directional relationship between cognitions and emotions. The main direction of effect was from poor emotional health (anxiety only) to cognitions.

Poor emotional health did not have a direct or indirect effect (mediated by diabetes cognitions) on diabetes self-care behaviours. Personal control remained independent from poor emotional health and predicted change in diabetes self-care behaviours.

8 *Structural models of the simultaneous relationships between cognitions, negative well-being, and diabetes self-care*

8.1 *Chapter outline*

This chapter reports the statistical analyses that address the primary research hypotheses with poor emotional health conceptualised as a single construct: negative well-being. All analytical procedures remain the same as those described in Chapter Seven, Section 7.2.

8.2 *Results: Cognition-negative well-being and cognition + negative well-being-diabetes self-care regression models*

Table 8.1 and 8.2 provide a summary of the findings from the bivariate and multivariate regression models used to identify potential relationships between diabetes cognitions, negative well-being, and global diabetes self-care. Space restrictions prevented the full statistical output for each regression model from being presented but Tables 8.1 and 8.2 present the standardised beta coefficient and associated p values for:

i. Each diabetes cognition domains explanatory effect on negative well-being
ii. Negative well-being's explanatory effect on each diabetes cognition domain
iii. Cognition and negative well-being's explanatory effect on global diabetes self-care
iv. Global diabetes self-care explanatory effect on negative well-being (the findings for diabetes cognitions are consistent with those found in Chapter 7, Table 7.3 and are therefore not duplicated here).

Variables in italics are those that showed an association at the level of $p \leq 0.10$.

Four cognition-negative well-being pairs were identified from the regression analyses for more rigorous testing using SEM, these included: timeline cyclical, personal control, illness coherence, and medication concerns. Negative well-being did not demonstrate a longitudinal relationship with global diabetes self-care when it was included as either a predictor or a dependent variable in the regression models. The cognition-global diabetes self-care pairs identified were consistent with those found in Chapter Seven. Thus the nested cross-lagged analyses exploring diabetes cognitions relationship with global diabetes self-care will not be duplicated here (See Chapter Seven, Section 7.4.3). However, the findings from these paired cross-lagged analyses were used in the final model that

tested the simultaneous relationships between negative well-being, cognitions, and global diabetes self-care.

Table 8.1: A summary of the findings from the bivariate and multivariate regression analyses used to identify cognition-negative well-being pairs

Cognition →Negative	Negative well-being →Cognition*
Bivariate findings	*Bivariate findings*
Identity: β=-0.01, p=0.88	Identity: β=0.10, p=0.23
Timeline acute/chronic: β=-0.06, p=0.31	Timeline acute/chronic: β=0.02, p=0.83
Timeline cyclical: β=0.08, p=0.15	*Timeline cyclical: β=0.23, p=0.00*
Consequences: β=0.08, p=0.17	Consequences: β=0.03, p=0.70
Personal control: β=0.05, p=0.33	Personal control: β=-0.02, p=0.84
Treatment control: β=-0.05, p=0.35	Treatment control: β=-0.05, p=0.42
Illness coherence: β=-0.04, p=0.54	*Illness coherence: β=-0.16, p=0.01*
Medication necessity: β=0.06, p=0.25	Medication necessity: β=0.09, p=0.13
Medication concerns: β=0.18, p=0.01	*Medication concerns: β=0.12, p=0.09*
Multivariate findings	*Multivariate findings*
Identity: β=0.00, p=0.99	Identity: β=0.03, p=0.76
Timeline acute/chronic: β=-0.06, p=0.31	Timeline acute/chronic: β=-0.02, p=0.84
Timeline cyclical: β=0.09, p=0.12	*Timeline cyclical: β=0.23, p=0.00*
Consequences: β=0.02, p=0.74	Consequences: β=-0.01, p=0.90
Personal control: β=0.11, p=0.08	Personal control: β=-0.02, p=0.84
Treatment control: β=-0.09, p=0.14	Treatment control: β=-0.01, p=0.93
Illness coherence: β=0.02, p=0.77	*Illness coherence: β=-0.16, p=0.01*
Medication necessity: β=0.05, p=0.29	Medication necessity: β=0.05, p=0.43
Medication concerns: β=0.17, p=0.02	*Medication concerns: β=0.14, p=0.04*

*Cognition domains were the outcome variables in the regression models. The beta coefficients and p values represent negative well-being as a predictor of each cognition domain.

Table 8.2: A summary of the findings from the bivariate and multivariate regression analyses used to identify cognition-diabetes self-care and negative well-being-diabetes self-care pairs.

Cognition + negative well-being →Global diabetes self-care	Global diabetes self-care →cognition + emotion*
Bivariate findings	*Bivariate findings*
Bivariate findings for the cognition domains are consistent with those reported in Chapter Seven, Table 7.1c	Bivariate findings for the cognition domains are consistent with those reported in Chapter Seven, Table 7.1c
Negative well-being: β=-0.01, p=0.84	Negative well-being: β=-0.03, p=0.56
Multivariate findings	*Multivariate findings*
Identity: β=-0.08, p=0.17	Not applicable
Timeline acute/chronic: β=-0.07, p=0.20	
Timeline cyclical: β=0.05, p=0.39	
Consequences: β=0.10, p=0.11	
Personal control: β=-0.22, p=0.00	
Treatment control: β=0.13, p=0.03	
Illness coherence: β=-0.03, p=0.72	
Medication necessity: β=0.03, p=0.61	
Medication concerns: β=-0.03, p=0.74	
Negative well-being: β=0.02, p=0.79	

*Cognition and emotion domains were the outcome variables in the regression models. The beta coefficients and p values represent diabetes self-care as a predictor of negative well-being.

8.3 *Results: Nested cross-lagged paired directional models*

Table 8.3 summarises the findings from the four separate nested cross-lagged analyses performed for negative well-being and the four empirically determined cognition domains. Figures 8.1 to 8.3 provide a summary of the standardised regression coefficients for the directional pathways of the best fitting model identified using the chi-squared difference test. A key to aid with the interpretation of the figures is provided in Table 8.4. If the nested model comparisons indicated that the auto-regressive model had the best fit then parameter estimates for these models were not presented diagrammatically because of their limited theoretical value. A tabular summary of unstandardised and standardised

variables estimated in the best fitting paired models (including the auto-regressive models) is provided in Statistical Appendix D, Tables D1 to D4.

8.3.1 *Negative well-being and timeline cyclical*

All theoretically plausible models of the directional relationships between timeline cyclical and negative well-being had evidence of good fit. Of note the directional model depicting negative well-being's explanatory effect on timeline cyclical beliefs and the fully cross-lagged (bi-directional) model had excellent fit as indicated by the non-statistically significant model chi-square statistic (See Table 8.3).

The model testing the directional pathway leading from timeline cyclical at baseline to negative well-being at six months follow-up did not provide support for this directional relationship. This model did not provide a better fit when compared with the auto-regressive model ($\Delta\chi^2$=0.08, p=0.78).

The findings did provide support for the directional effect of baseline negative well-being on timeline cyclical beliefs at six months follow-up. This model provided a statistically significant improvement in model fit compared with the auto-regressive model ($\Delta\chi^2$=7.83, $p \leq 0.01$).

The fully cross-lagged (bi-directional model) was therefore explored to examine whether the addition of the pathway leading from timeline cyclical to negative well-being provided further improvement in model fit. Evidence in support of this pathway was not found (See Table 8.3). The directional effect of negative well-being on timeline cyclical beliefs was retained as the best fitting model; its standardised regression coefficients are shown in Figure 8.1. A tabular summary of the parameter estimates associated with this model can be found in Statistical Appendix D, Table D1. All latent variable factor loadings were statistically significant.

8.3.2 *Negative well-being and personal control*

All theoretically plausible models of the relationship between negative well-being and personal control demonstrated excellent fit to the sample data as evidenced by all fit indices and the non-statistically significant chi-square statistic (See Table 8.3).

The auto-regressive baseline model was retained as the best fitting model. The addition of pathways to the baseline model leading from personal control to negative well-being ($\Delta\chi^2$=. 41, p=0.52) and vice versa ($\Delta\chi^2$=2.45, p=0.12) did not demonstrate a statistically significant improvement in model fit. As a result of these findings the fully cross-lagged model was not explored. Parameter estimates of the auto-regressive personal control – negative well-being model can be found in Statistical Appendix D, Table D2. All latent variable factor loadings were statistically significant.

8.3.3 *Negative well-being and illness coherence*

All models of probable relationships between illness coherence and negative well-being demonstrated evidence of good fit, excluding the model chi-squared statistic (See Table 8.3).

Findings did not support the directional effect of baseline illness coherence on negative well-being at six months follow-up. An elaboration on the auto-regressive baseline model by the addition of this pathway did not demonstrate a statistically significant improvement in model fit ($\Delta\chi^2$=0.06, p=0.81; See Table 8.3).

The results did support the directional effect of baseline negative well-being on illness coherence at six months follow-up ($\Delta\chi^2$= 5.18. p≤0.05). Given this finding the fully cross-lagged (bi-directional) model was explored to test whether the addition of the pathway leading from baseline illness coherence to negative well-being at follow-up could improve the model fit further. Findings did not support the addition of this pathway to the model ($\Delta\chi^2$=0.06, p=0.80) and therefore the explanatory effect of negative well-being on illness coherence was retained as the best fitting model (See Figure 8.2 and Statistical Appendix D, Table D3 for unstandardised and standardised model parameter estimates). All latent variable factor loadings for this model were statistically significant.

8.3.4 *Negative well-being and medication concerns*

All models of the plausible relationships that exist between medication concerns and negative well-being had good fit, excluding the model chi-squared statistic (See Table 8.3).

The directional effect of baseline medication concerns on negative well-being was not endorsed by the findings. The addition of this pathway to the auto-regressive model did not make a statistically significant improvement to the chi-square statistic ($\Delta\chi^2$=1.65, p=0.20).

The results did provide support for the reverse directional pathway. There was a statistically significant improvement in the chi-square statistic once the auto-regressive model was elaborated on to include the directional effect of baseline negative well-being on medication concerns at six months follow-up ($\Delta\chi^2$=4.17, p≤0.05).

Given this finding the fully cross-lagged (bi-directional) model was tested. This model did not provide a statistically significant improvement in model fit relative to the model which only included the directional effect of baseline negative well-being on medication concerns at six months follow-up ($\Delta\chi^2$=1.09, p=0.30) and therefore the most parsimonious model was retained.

Figure 8.3 shows directional effect of baseline negative well-being on medication concerns at six months follow-up. Statistical Appendix D, Table D4 presents a tabular summary of the parameters estimated in this model. All latent variable factor loadings were statistically significant.

8.3.5 *Tests of measurement stability*

Measurement stability was explored by specifying questionnaire item factor loadings at baseline and six months follow-up to be equal (equality constraints). Once these constraints were applied to the models their fit indices were compared with those reported in Table 8.3. Statistical Appendix D, Table D5a to D5c presents the model fit indices with equality constraints applied. These comparisons indicated that model fit was largely unchanged as were standardised path coefficients. These findings demonstrate that the constructs being measured remain stable over a six months interval, thus any modelled relationships reflect “true” relationships and not measurement instablilty.

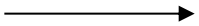
Table 8.3: Nested model analyses linking negative well-being and diabetes cognition domains

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Negative well-being and timeline cyclical (N=172) see Statistical Appendix D, Table D1 for model parameter estimates: negative well-being →timeline cyclical									
Auto-regressive	120.65	93	.03	.04	.98	.07	206.65		
Timeline cyclical to negative well-being	120.57	92	.02	.04	.98	.07	208.57	.08	.78
Negative well-being to timeline cyclical	112.82	92	.07	.04	.99	.05	200.82	7.83	.01
Cross-lagged*	112.73	91	.06	.04	.98	.05	202.73	.09	.76
*$\Delta\chi^2$ between negative well-being to timeline cyclical and cross lagged									
Negative well-being and personal control (N=165) see Statistical Appendix D, Table D2 for model parameter estimates: auto-regressive model									
Auto-regressive	175.92	155	.12	.03	.98	.07	285.92		
Personal control to negative well-being	175.51	154	.11	.03	.98	.07	287.51	.41	.52
Negative well-being to personal control	173.47	154	.14	.03	.98	.07	285.47	2.45	.12
Cross-lagged	173.25	153	.13	.03	.98	.07	287.25	NA	NA
Negative well-being and illness coherence (N=172) see Statistical Appendix D, Table D3 for model parameter estimates: negative well-being →illness coherence									
Auto-regressive	168.72	93	.00	.07	.95	.07	254.72		
Illness coherence to negative well-being	168.66	92	.00	.07	.95	.07	256.66	.06	.81
Negative well-being to illness coherence	163.54	92	.00	.07	.96	.05	251.54	5.18	.02
Cross-lagged*	163.48	91	.00	.07	.96	.05	253.48	.06	.80
*$\Delta\chi^2$ between negative well-being to illness coherence and cross lagged									

Table 8.3: continued

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Negative well-being and medication concerns (N=166) see Statistical Appendix D, Table D4 for model parameter estimates: negative well-being →medication concerns									
Auto-regressive	186.61	123	.00	.06	.96	.07	282.61		
Medication concerns to negative well-being	184.96	122	.00	.06	.96	.06	282.96	1.65	.20
Negative well-being to medication concerns	182.44	122	.00	.06	.96	.06	280.44	4.17	.04
Cross-lagged*	181.38	121	.00	.06	.96	.06	281.38	1.07	.30
*$\Delta\chi^2$ between negative well-being to medication concerns and cross-lagged									

Table 8.4: Key to aid with the interpretation of Figures 8.1 to 8.7

Abbreviation/Symbol	Definition
WBQ(N)	Used to represent questionnaire item numbers from the WBQ
IP(N)	Used to represent questionnaire item numbers from the IPQ-R
BMQ(N)	Used to represent questionnaire item numbers from the BMQ.
	Indicates the direction of effect
	Indicates that variables are assumed to co-vary but the direction of effect is not explicitly stated
	Squares indicate measured variables
	Ellipses indicate latent variables
*	$p \leq .05$
**	$p \leq .01$

All numbers displayed on the figures excluding those adjacent to the latent variables at follow-up are standardised beta coefficients. The statistical significance of factor loadings is not shown on the figures but all are statistically significant at $p \leq .05$ (based on bootstrapped p values) unless otherwise stated. Numbers adjacent to the latent variables at follow-up represent the variance explained in the dependent variable (R^2).

Figure 8.1: Negative well-being's directional effect on timeline cyclical

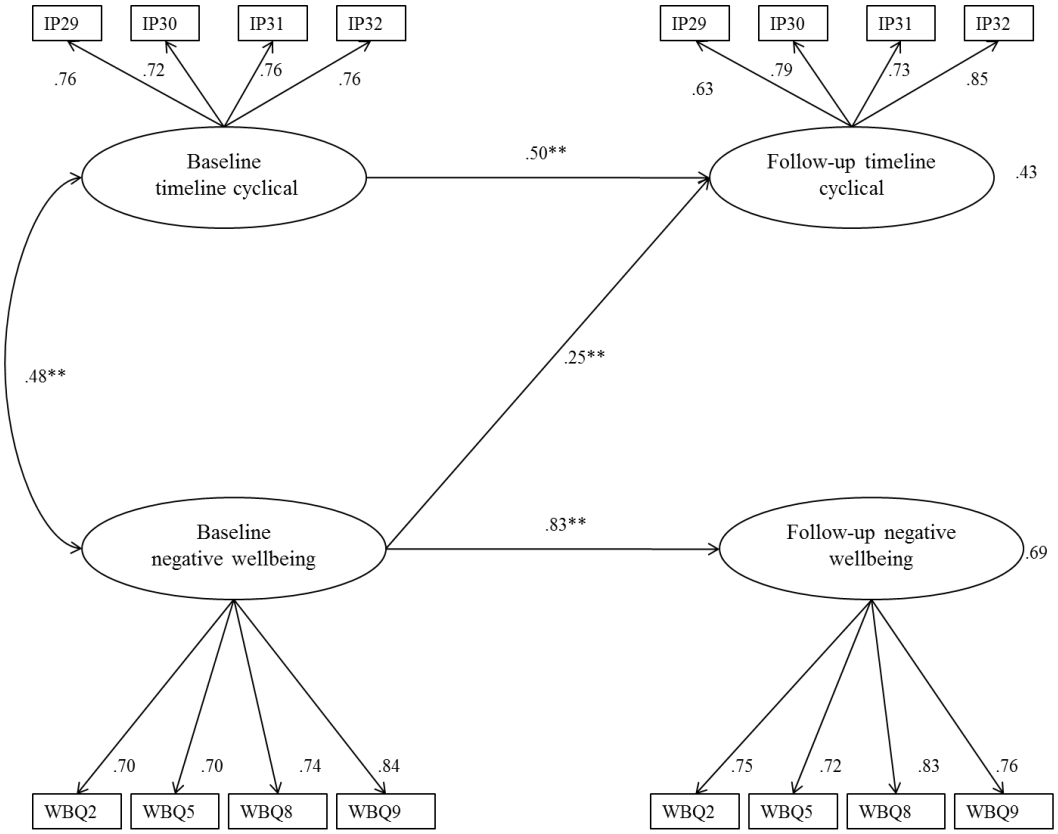


Figure 8.2: Negative well-being's directional effect on illness coherence

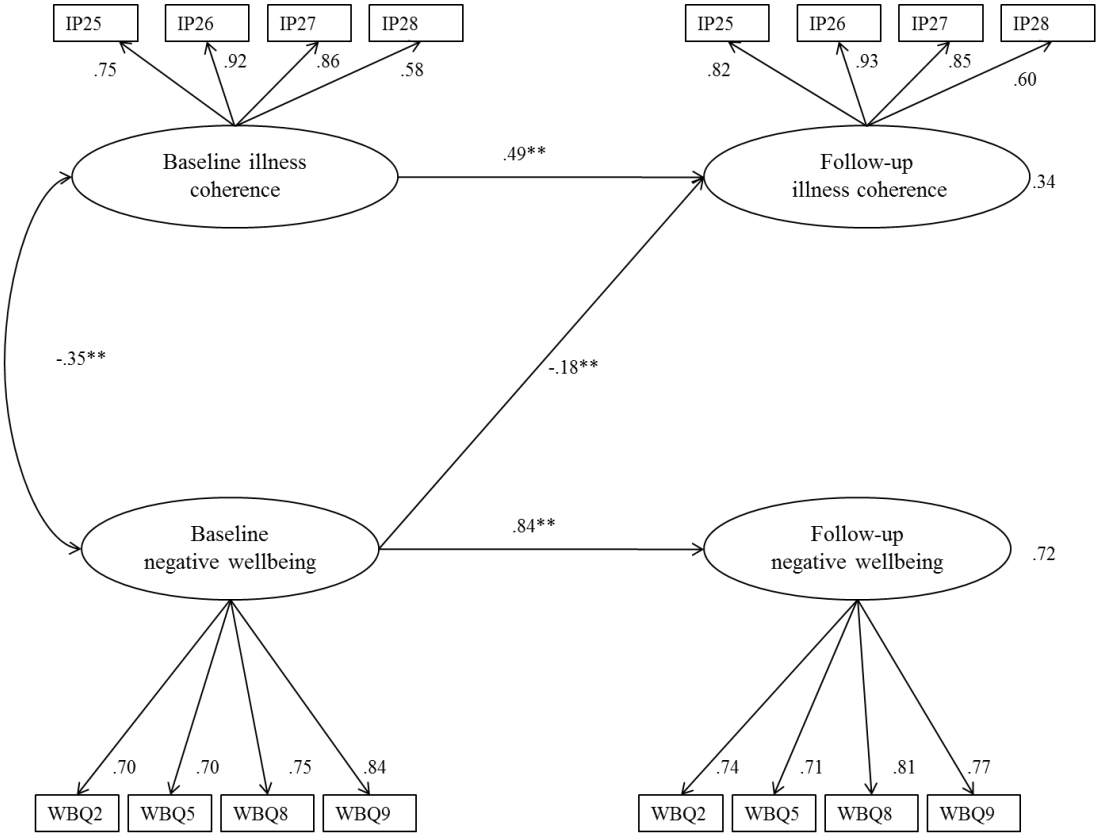
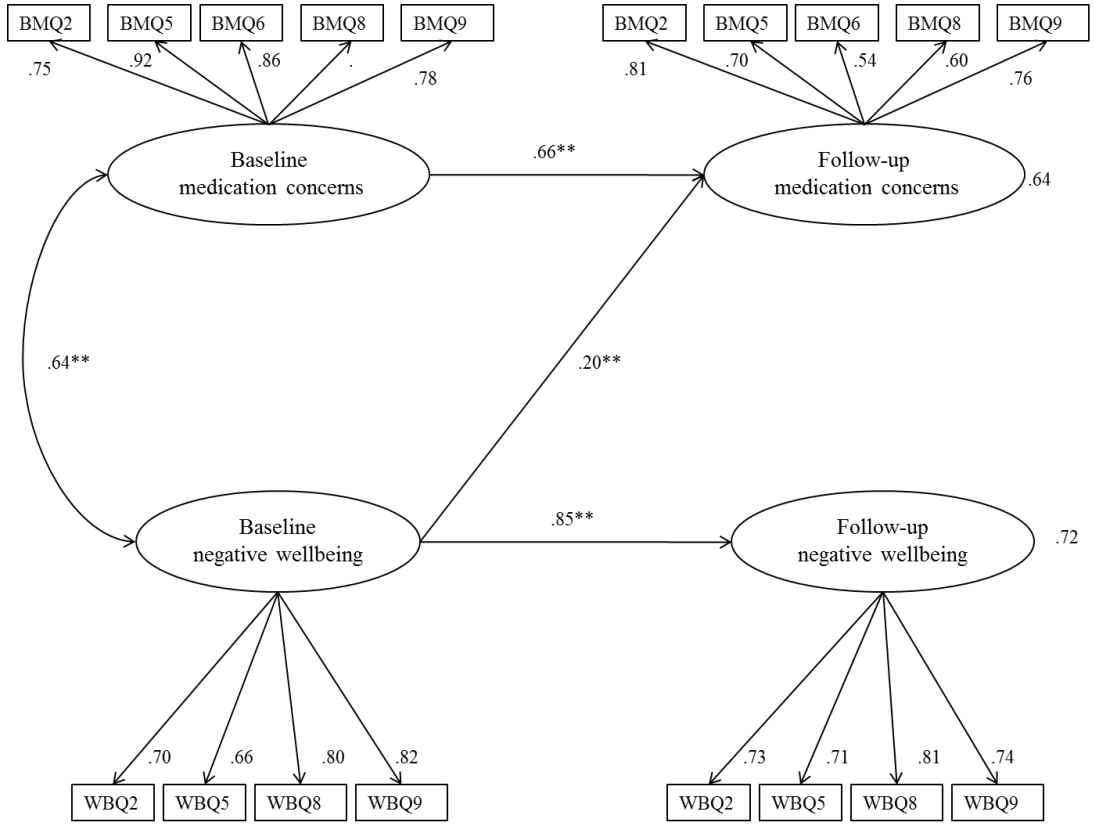


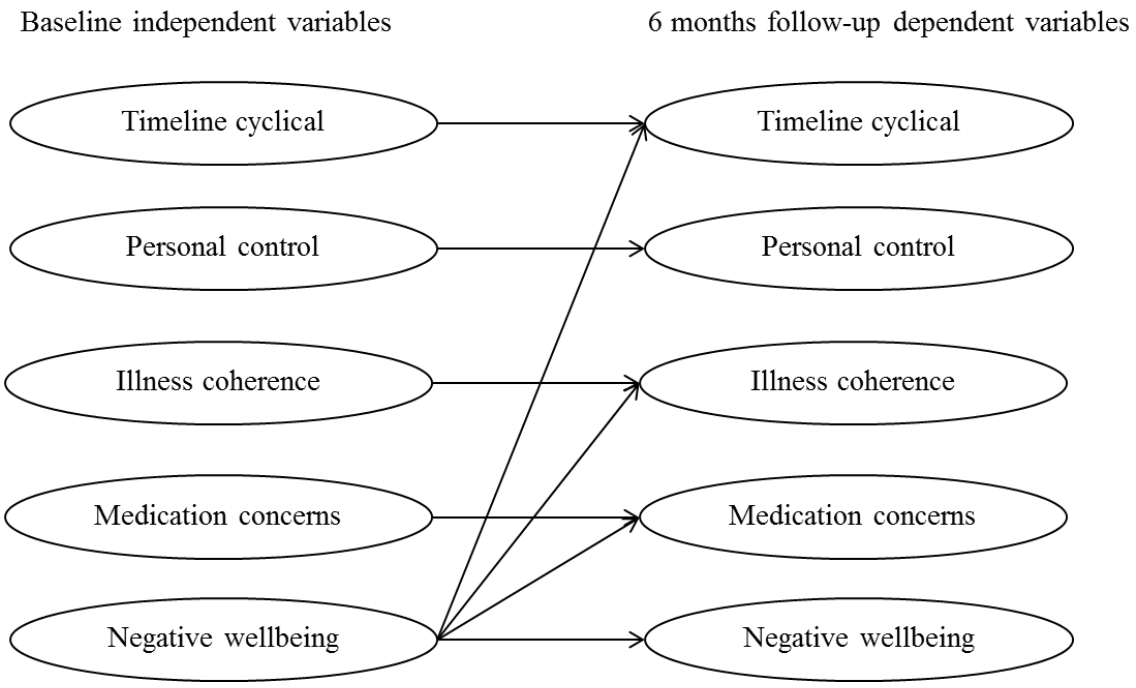
Figure 8.3: Negative well-being's directional effect on medication concerns



8.3.6 *Summary of paired cross-lagged findings*

A summary of the findings from the four separate cross-lagged analyses presented above is provided in Figure 8.4. Negative well-being demonstrated a longitudinal effect on timeline cyclical, illness coherence, and medication concerns. No relationships were observed in either direction relative to the auto-regressive model for personal control. In addition no evidence was found to support the directional effect of baseline cognitions on negative well-being at follow-up.

Figure 8.4: Summary of findings from the paired negative well-being – cognition directional models



8.4 *Results of the simultaneous structural equation models: cognitions, negative well-being, and global diabetes self-care*

The paired cross-lagged negative well-being-cognition findings indicated a need to test a model which explored the simultaneous effect of baseline negative well-being on timeline cyclical, illness coherence, and medication concerns at follow-up whilst also incorporating the independent effect of baseline personal control on global diabetes self-care at follow-up (this relationship was identified from the analyses performed in Chapter Seven, Section 7.4.3). There was no evidence of longitudinal mediation according to Cole and Maxwell's (2003) criteria.

Consistent with the final model presented in Chapter Seven it was not possible to specify multiple indicator latent variables for the cognition and negative well-being concepts because of a lack of statistical power. Item parcelling was therefore applied to reduce the number of parameters being estimated by the model (See Chapter Three, Section 3.4.4 for a description of item parcelling).

8.4.1 *Complete cases model: cognitions, negative well-being, and global diabetes self-care*

The final model of the simultaneous effect of cognitions, negative well-being, and global diabetes self-care is shown in Figure 8.5. This simultaneous model provided a more rigorous test of the CS-SRM's claims about the types of directional pathways that occur between cognitions, emotional and self-care. As can be seen from the figure the model does not include a pathway leading from baseline negative well-being to medication concerns at follow-up. This was because this pathway became statistically non-significant when entered simultaneously with the remaining variables in the model. To retain parsimony the pathway was therefore removed from the model (Byrne, 2010). Please see Statistical Appendix D, Table D6 for a tabular summary of the parameter estimates for the originally specified model.

The final model (See Figure 8.5), had evidence of good fit on all model fit indices excluding the model chi-squared index (χ^2=325.2, df_m=225, p≤0.01; RMSEA=0.05, CFI=0.94, SRMR=0.07, AIC=475.2; N=164). All directional pathways remained statistically significant, specifically:

i. Increased scores on baseline negative well-being were associated with higher scores on timeline cyclical beliefs (β=0.19, p≤0.01) and decreased perceived illness coherence (β=-0.14, p≤0.01) at six months follow-up.
ii. Baseline negative well-being and timeline cyclical explained 54% of the variance (R^2) in timeline cyclical beliefs at follow-up. Similarly, baseline negative well-being and illness coherence explained 43% of the variance (R^2) in illness coherence beliefs at follow-up.
iii. Increased perceptions of personal control at baseline had a negative relationship with global diabetes self-care at six months follow (β=-0.29, p≤0.01).
iv. Baseline personal control and global diabetes self-care explained 62% of the variance (R^2) in global diabetes self-care at follow-up.
v. All baseline variables demonstrated a longitudinal association with themselves at six months follow-up. Standardised beta coefficients for baseline illness cognition domains predicting their scores at follow-up ranged from 0.58 to 0.64. For negative well-being and global diabetes self-care standardised beta coefficients were 0.91 and 0.79 respectively.
vi. Correlations between baseline latent variables are shown in Table 8.5. A tabular output of parameters estimated in in Figure 8.5 can be found in Statistical Appendix D, Table D7.

Table 8.5: Correlations between baseline latent variables shown in Figure 8.5

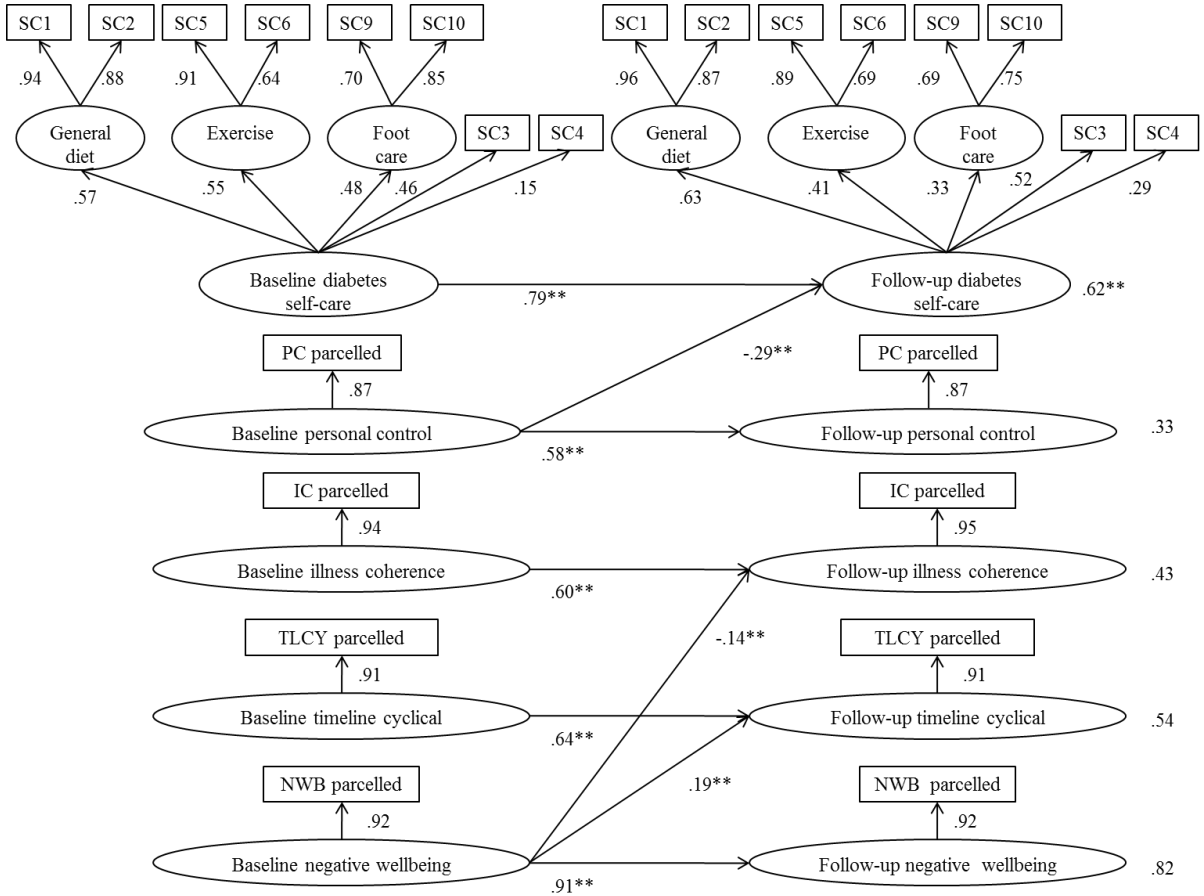
Variables	Global diabetes	Personal control	Illness coherence	Timeline cyclical
Personal control	0.19			
Illness coherence	0.44**	0.47**		
Timeline cyclical	0.04	-0.28*	-0.44**	
Negative well-being	0.05	-0.18	-0.32**	0.42**

Key: *p≤0.05, **p≤0.01, based on bootstrapped p values

Figure 8.5: Model of the simultaneous effect of negative well-being, cognitions, and global diabetes self-care

Key: IC, Illness coherence; PC, Personal control; TLCY, Timeline cyclical; NWB, Negative well-being and see Table 8.4

For simplicity co-variances are not displayed in the figure (see Table 8.5).



8.4.2 *Statistical control for confounders: clinical severity*

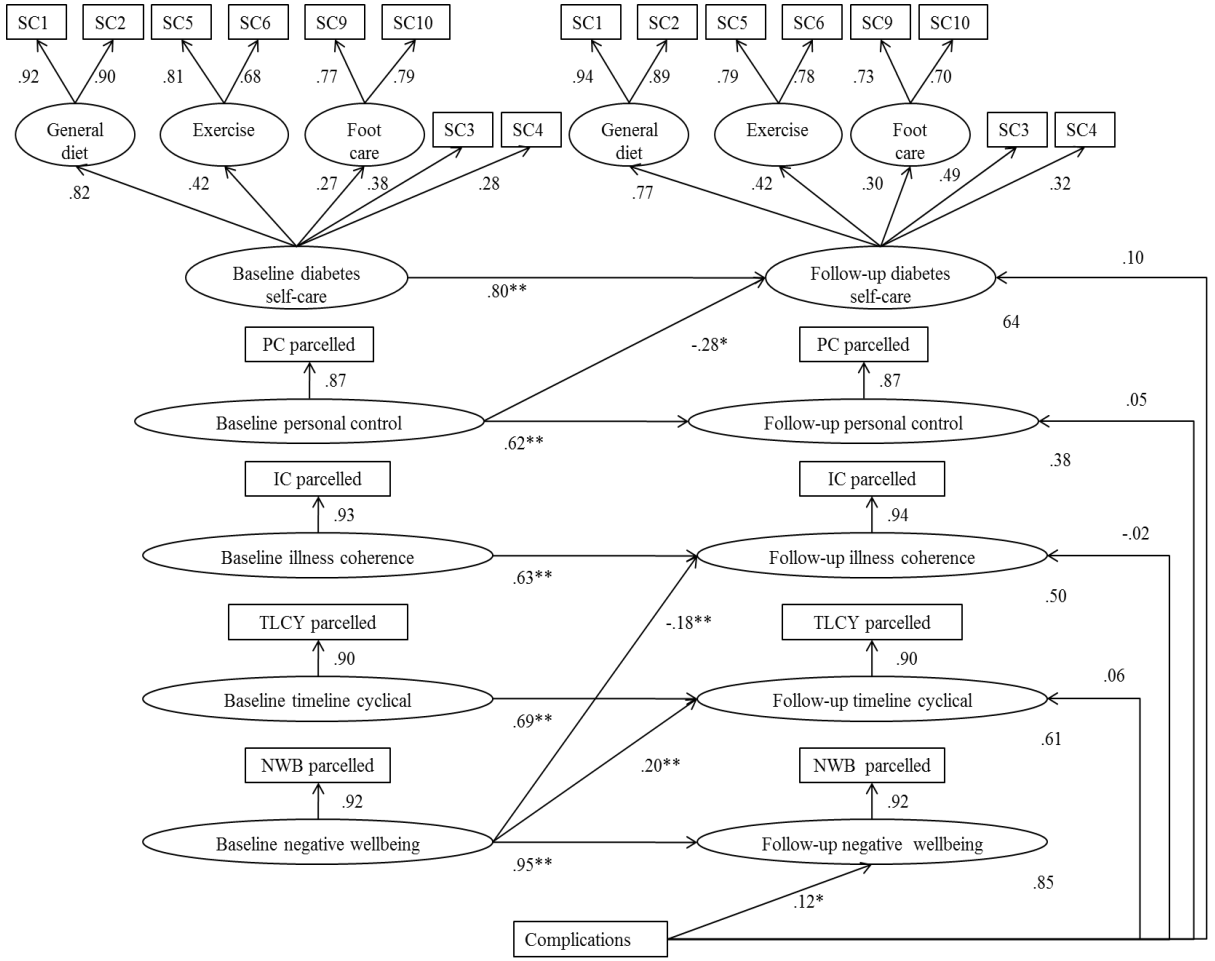
Number of diabetes complications (as a marker of severity) was added to the final model and specified to have a directional effect on all dependent variables and to correlate with each independent variable in the model (See Figure 8.6). This analysis was performed to explore whether the observed effects for the final model could be accounted for by the potentially confounding variable: number of diabetes complications.

When number of diabetes complications was added as a covariate to the final model the overall fit became marginally worse on all model fit indices apart from the SRMR which remained the same (χ^2=363.1, df_m=240, $p \leq 0.01$; RMSEA=0.06, CFI=0.93, SRMR=0.07, AIC=533.1; N=157). All cognition, negative well-being, and diabetes self-care pathways remained statistically significant but with marginal changes observed in their standardised beta coefficients. For those pathways in which differences in effect size (beta coefficients) were observed changes occurred in the range of ±0.01 to 0.05. Directional pathways leading from baseline number of diabetes complications to each of the dependent variables were statistically non-significant, apart from negative well-being (See Figure 8.6). Based on these findings the model which excluded baseline number of diabetes complications was retained as the best fitting model and there was no evidence of confounding. Statistical Appendix D, Table D8 presents a tabular summary of the parameters estimated in Figure 8.6.

Figure 8.6: Model of the simultaneous effect of negative well-being, cognitions, and global diabetes self-care controlling for the effect of number of diabetes complications

Key: IC, Illness coherence; PC, Personal control; TLCY, Timeline cyclical; NWB, Negative well-being and see Table 8.4

For simplicity co-variances are not displayed in the figure (see Statistical Appendix D, Table D8).



8.4.3 *Sensitivity analyses: missing data*

Direct maximum likelihood estimation was applied to the final model to establish whether implementing complete cases analysis impacted on the findings in relation to model fit and parameter estimates for the final simultaneous model. This imputation method was applied for all participants who returned baseline questionnaires (n=261).

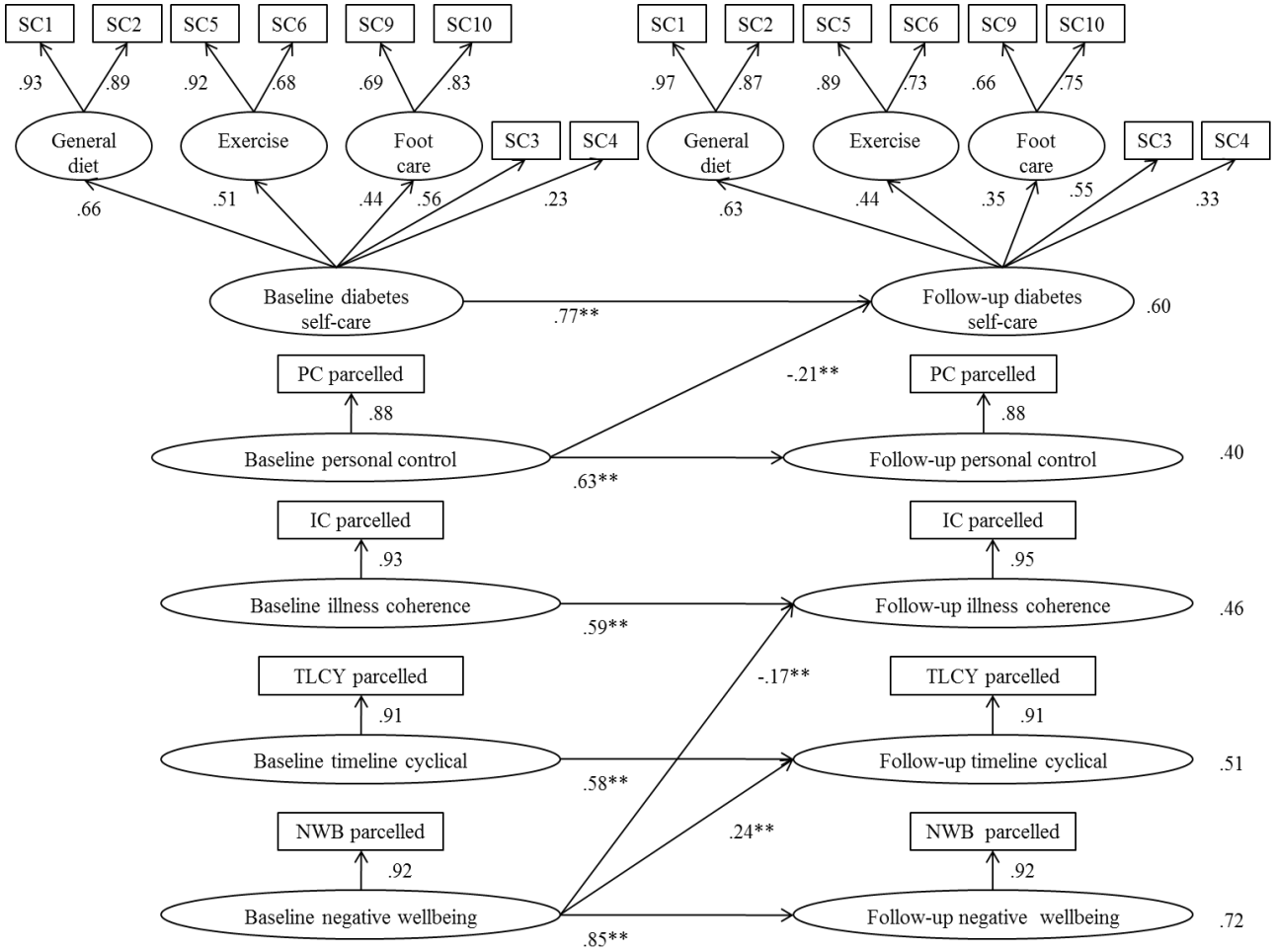
Similar to the complete cases analysis model, all goodness of fit statistics were well fitting excluding the model χ^2 statistic (χ^2=306.4, df_m=225, $p \leq 0.01$; RMSEA=0.04, CFI=0.96, SRMR=NA, AIC=504.4, N=261). Relative to the complete cases model marginal improvements in the model chi-squared, RMSEA and CFI fit index were observed, however the AIC fit index worsened. The statistical significance of pathways remained unchanged but changes in effect size estimates were observed. For those parameters in which changes were observed these ranged from ±0.01 to 0.08 (standardised beta coefficients) with the largest changes being observed for the pathways associated with the global diabetes self-care construct. Bootstrapping could not be applied in conjunction with direct maximum likelihood estimation therefore estimates of statistical significance may be biased for this model. The findings of this model are presented in Figure 8.7 and a tabular output of the parameter estimates can be found in Statistical Appendix D, Table D9.

Direct maximum likelihood was also applied to the model which included the directional effect of baseline negative well-being on medication concerns at follow-up to establish whether the non-statistically significant effect occurred as a result of a lack of statistical power. This pathway still remained statistically non-significant when based on a sample size of 261 (please See Statistical Appendix D, Table D10).

Figure 8.7: Model of the simultaneous effect of negative well-being, cognitions, and global diabetes self-care with direct maximum likelihood estimation applied

Key: IC, Illness coherence; PC, Personal control; TLCY, Timeline cyclical; NWB, Negative well-being and see Table 8.4

For simplicity co-variances are not displayed in the figure (See Statistical Appendix D, Table D9).



8.5 *Chapter summary*

This chapter performed the necessary statistical analyses to address the primary research hypotheses for the competing conceptualisation of poor emotional health as negative well-being.

The final model of the simultaneous relationships between cognitions, negative well-being, and diabetes self-care found evidence in support of the following pathways:

i. Negative well-being had a directional effect on illness coherence and timeline cyclical beliefs. Participants with high levels of negative well-being at baseline felt they had a poor understanding of their diabetes and perceived it to be an unpredictable condition.
ii. Personal control had a directional effect on global diabetes self-care. Individuals who felt more in control of their diabetes adhered less to their treatment regimens.

No evidence was found to support:

i. The directional effect of cognitions on negative well-being.
ii. The directional effect of negative well-being on diabetes self-care or vice versa.

These findings provide partial support for research hypotheses one and two. Whilst, a bi-directional relationship between cognitions and poor emotional health was absent, negative well-being did have an explanatory effect on cognitions at follow-up. Negative well-being had no relationship with diabetes self-care either directly or indirectly via diabetes cognitions. Personal control acted independently from negative well-being to determine adherence to diabetes self-care behaviours at six months follow-up.

9 *Structural models of the simultaneous effect of diabetes cognitions and poor emotional health on HbA1c*

9.1 *Chapter outline*

This chapter reports the statistical analyses that address secondary research hypothesis one. A reminder of this hypothesis is provided below.

Baseline diabetes cognition domains and poor emotional health have a combined and independent longitudinal effect on HbA1c at follow-up, specifically:

i. Diabetes cognitions have a direct and indirect effect (via poor emotional health) on HbA1c (cognitions → HbA1c, *and* cognitions → emotions →HbA1c).
ii. Poor emotional health has a direct and indirect effect (via diabetes cognitions) on HbA1c (emotions → HbA1c, *and* emotions →cognitions → HbA1c).

The analytical procedures performed to address this hypothesis are reported first followed by the results.

9.2 *Statistical analyses*

9.2.1 *Identifying variables for testing using SEM*

Consistent with the preceding two chapters it was not possible to pre-specify which diabetes cognition domains should be included in the structural equation model (SEM) as explanatory variables of HbA1c at six months follow-up because of a lack of empirical studies exploring these relationships longitudinally (McSharry et al., 2011). Bivariate *and* multivariate regression analyses were therefore used to empirically identify baseline cognition and emotion variables for more rigorous testing using SEM.

For the bivariate regression models baseline cognition or emotion variables (for all conceptualisations of poor emotional health e.g. depression, anxiety, and negative well-being) were singularly entered as a predictor of HbA1c at follow-up, whilst statistically controlling for baseline HbA1c only. This allowed for an examination of the independent effects of cognition and emotion variables on HbA1c.

For the multivariate regression model the relative effects of baseline cognitions and poor emotional health variables on HbA1c at follow-up were tested by simultaneously entering these variables into a backwards elimination regression model, whilst statistically controlling for baseline HbA1c levels. Two separate backwards elimination regression models were performed with poor emotional health defined as either: i) depression and anxiety or ii) negative well-being. All subscales of the Revised Illness Perception Questionnaire (excluding the emotional representation and cause subscales), Beliefs about Medicines Questionnaire, and Well-being Questionnaire (WBQ) were included as predictors of HbA1c in the multivariate models. The energy and positive well-being subscales of the WBQ were included to establish whether poor emotional health had an effect on HbA1c independent of the other WBQ constructs.

Variables that demonstrated a longitudinal relationship with HbA1c at follow-up at the level of $p \leq 0.10$ were retained for testing using SEM. Bootstrapped bias corrected standard errors and associated p values were used because HbA1c was non-normally distributed.

9.2.2 *Structural equation modelling*

Variables identified from the regression analyses were then tested using SEM. A l model was generated consisting of:

i. the relevant baseline cognition and poor emotional health latent variables and
ii. baseline HbA1c

Each of the above variables was hypothesised to have an effect on HbA1c at six months follow-up. If there was evidence of longitudinal mediation according to Cole and Maxwell's (2003) criteria (described in Chapter Seven, Section 7.3.2) this was incorporated into the model for the relevant cognition – emotion variables. If both competing conceptualisations of poor emotional health demonstrated a longitudinal relationship with HbA1c then two competing structural models were generated: i) cognitions, depression, anxiety, and HbA1c and ii) cognitions, negative well-being, and HbA1c.

9.2.3 *Statistical control for confounding variables*

Once a well-fitting model of the relationships between cognitions, poor emotional health, and HbA1c had been identified baseline number of diabetes complications was added to the model (as a marker of diabetes severity). This analysis provided a test of whether the modelled relationships remained independent of illness severity. Number of diabetes complications was specified to co-vary with baseline independent variables and to have a direct effect on HbA1c.

9.2.4 *Sensitivity analyses*

To establish whether using complete cases analysis impacted on the findings for secondary research hypothesis one imputation of missing data using direct maximum likelihood estimation was used to allow all participants who returned a questionnaire at baseline (N=261) to be included in the model.

9.3 *Results: cognitions, poor emotional health, and HbA1c regression models*

A summary of the findings from the bivariate and multivariate regression models used to identify diabetes cognitions and poor emotional health variables as likely predictors of HbA1c is provided in Table 9.1. The table presents the standardised beta coefficient and associated p value for each baseline diabetes cognition domain and poor emotional health concepts explanatory effect on HbA1c at six months follow-up. Variables in italics demonstrated a relationship with HbA1c at the level of $p \leq 0.10$.

Three cognition domains were identified as having a longitudinal relationship with HbA1c and these included: timeline acute/chronic, illness coherence, and medication necessity beliefs. However the illness coherence domain only demonstrated a relationship with HbA1c in the multivariate backwards elimination regression model when poor emotional health was conceptualised as two distinct constructs (depression and anxiety). Baseline poor emotional health when conceptualised as either depression and anxiety or negative well-being did not demonstrate a longitudinal relationship with HbA1c.

Table 9.1: A summary of the findings from the bivariate and multivariate regression models used to identify cognition and poor emotional health variables as predictors of HbA1c at six months follow-up.

Cognition & Emotion →HbA1c	Cognition & Emotion →HbA1c
Emotion defined as depression and anxiety	*Emotion defined as negative well-being*
Bivariate findings	*Bivariate findings*
Identity: β=0.02, p=0.76	Findings for cognitions are consistent with the bivariate findings reported in the column to the left.
Timeline acute/chronic: β=0.14, p=0.03	
Timeline cyclical: β=0.00, p=0.97	Negative well-being: β=0.04, p=0.58
Consequences: β=0.07, p=0.26	
Personal control: β=0.01, p=0.88	
Treatment control: β=-0.03, p=0.59	
Illness coherence: β=-0.05, p=0.40	
Medication necessity: β=-0.21, p=0.00	
Medication concerns: β=-0.03, p=0.67	
Depression: β=0.00, p=1.00	
Anxiety β=0.06, p=0.35	
Multivariate findings	*Multivariate findings*
Identity: β=-0.01, p=0.91	Identity: β=0.02, p=0.82
Timeline acute/chronic: β=0.16, p=0.01	*Timeline acute/chronic: β=0.16, p=0.02*
Timeline cyclical: β=-0.01, p=0.94	Timeline cyclical: β=0.01, p=0.88
Consequences: β=0.09, p=0.29	Consequences: β=0.08, p=0.26
Personal control: β=0.01, p=0.90	Personal control: β=0.03, p=0.74
Treatment control: β=-0.01, p=0.95	Treatment control: β=-0.03, p=0.71
Illness coherence: β=-0.10, p=0.10	Illness coherence: β=-0.11, p=0.11
Medication necessity: β=-0.26, p=0.00	*Medication necessity: β=-0.23, p=0.00*
Medication concerns: β=-0.11, p=0.25	Medication concerns: β=-0.10, p=0.24
Depression: β=-0.19, p=0.17	Negative well-being: β=0.04, p=0.68
Anxiety β=0.07, p=0.26	

9.4 *Results of the simultaneous structural equation model: cognitions, poor emotional health, and HbA1c*

Based on the findings from the regression models a structural model was generated consisting of the three latent variable cognition domains at baseline (timeline acute/chronic, illness coherence, and medication necessity) each having a directional effect on HbA1c at follow-up. Latent variables were generated in accordance with the model specifications identified in Chapter Six. The directional effect of baseline HbA1c on itself at six months follow-up was included in the model. All baseline variables were specified to co-vary with each other at baseline.

9.4.1 *Complete cases model: cognitions, poor emotional health, and HbA1c*

The model testing the simultaneous effect of baseline HbA1c, timeline acute/chronic, illness coherence, and medication necessity beliefs demonstrated evidence of good fit on all fit indices excluding the model chi-square statistic based on a complete cases analysis of sample size n=128 (χ^2=140.6, df_m=110, $p \leq 0.03$; RMSEA=0.05, CFI=0.97, SRMR=0.07, AIC=226.7, N=128). However the l pathway leading from baseline illness coherence to HbA1c at six months follow-up was not statistically significant at the level of $p \leq 0.05$. It was therefore removed in order to retain model parsimony based on the recommendations of Byrne (2010). A tabular summary of parameters estimated in the originally hypothesised model can be found in Statistical Appendix E, Table E1.

The revised model excluding illness coherence demonstrated good fit to the sample data on all model fit indices with the exception of the model chi-square statistic (χ^2=82.3, df_m=60, $p \leq 0.03$; RMSEA=0.05, CFI=0.96, SRMR=0.07, AIC=144.3). Figure 9.1 depicts the standardised parameter estimates for each of the pathways specified in the model, a summary of unstandardised and standardised parameter estimates can be found in Statistical Appendix E, Table E2. A key to aid with the interpretation of Figure 9.1 is shown in Table 9.2 and applies to all subsequent figures also.

All pathways specified in the model shown in Figure 9.1 were statistically significant specifically:

i. Increased timeline acute/chronic beliefs were associated with increased levels of HbA1c at follow-up (β=0.18, $p \leq 0.01$) and increased medication necessity beliefs were associated with decreased levels of HbA1c at follow-up (β=-0.23, $p \leq 0.01$)

ii. Participants baseline HbA1c levels were statistically significant predictors of HbA1c at follow-up (β=0.61, $p \leq .01$)

iii. Baseline timeline acute/chronic, medication necessity beliefs, and baseline HbA1c were able to explain 45% of the variance (R^2) in participants HbA1c scores at follow-up.

iv. All correlations between baseline independent variables were statistically non-significant.

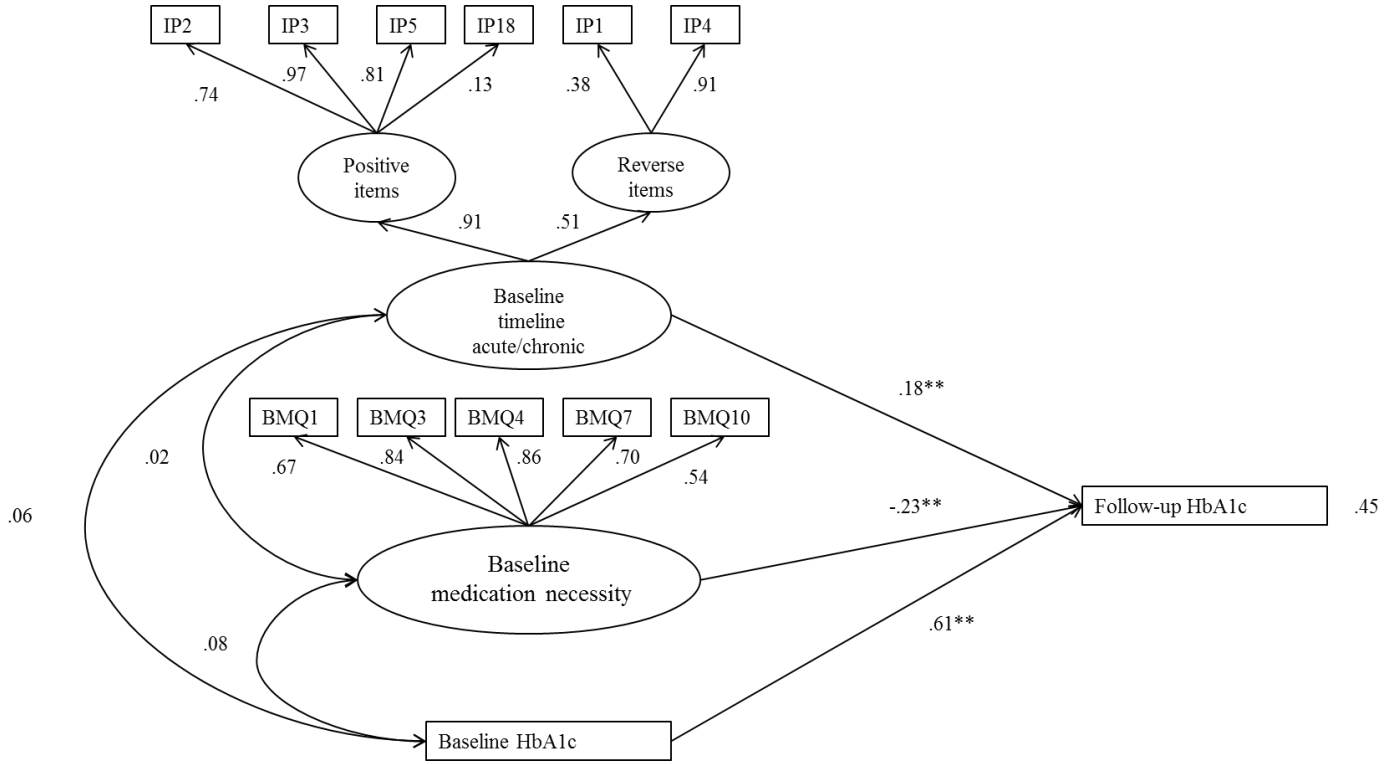
v. All questionnaire items had statistically significant factor loadings onto their latent variables excluding item 18 of the timeline acute/chronic subscale.

Table 9.2: Key to aid with the interpretation of Figures 9.1 to 9.3

Abbreviation/Symbol	Definition
IP(N)	Used to represent questionnaire item numbers from the IPQ-R (e.g. IP1)
BMQ(N)	Used to represent questionnaire item numbers from the BMQ (e.g. BMQ1)
	Indicates the direction of effect
	Indicates that variables are assumed to co-vary but the causal of effect is not explicitly stated
	Squares indicate measured variables
	Ellipses/circles indicate latent variables
*	$p \leq 0.05$
**	$p \leq 0.01$

All numbers displayed on the figures excluding those adjacent to HbA1c at follow-up are standardised beta coefficients. The statistical significance of factor loadings is not depicted on the figures but all are statistically significant at $p \leq 0.05$ unless otherwise stated in the text (based on bootstrapped p values). Numbers adjacent to HbA1c at follow-up represent the variance explained in the dependent variable (R^2).

Figure 9.1: Model of the simultaneous effect of cognitions on HbA1c at follow-up

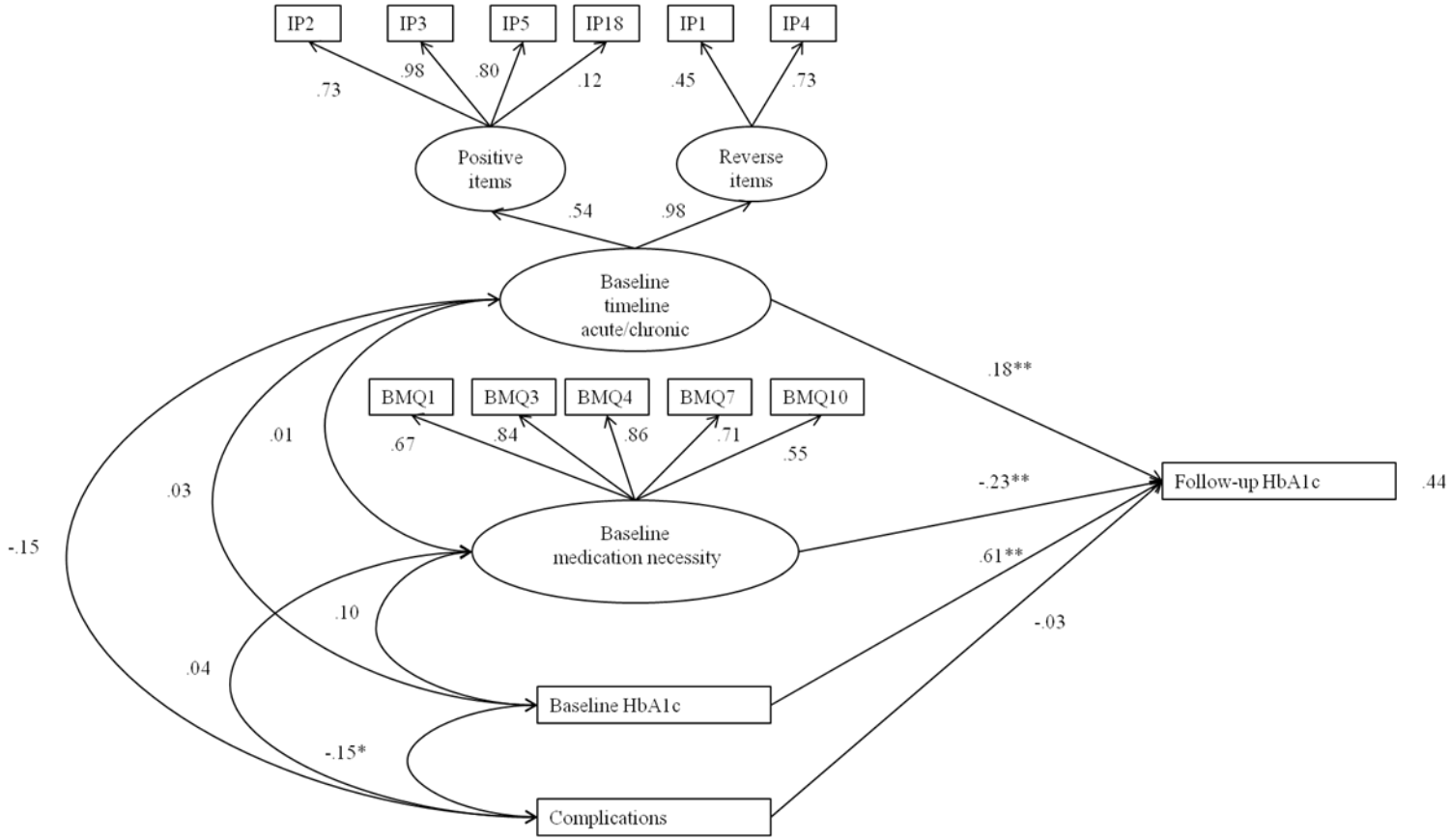


9.4.2 *Statistical control for confounders: clinical severity*

Baseline number of diabetes complications (as a marker of clinical severity) was added as a covariate into the model to establish whether cognition domains were able to demonstrate a relationship with HbA1c independent of diabetes severity. Baseline number of diabetes complications was specified to co-vary with each baseline independent variables and to have a direct effect on HbA1c at follow-up. This analysis was performed on a complete cases sample size of n=126.

The addition of baseline number of diabetes complications to the model had marginal impact on model fit indices (χ^2=102.1, df_m =69, p≤0.01; RMSEA=0.06, CFI=0.94, SRMR=0.07, AIC=174.1) however model fit became worse on the following fit indices: model chi-squared statistic, CFI and AIC compared with the model in which number of diabetes complications was absent. All directional pathways remained statistically significant and their effect sizes remained largely unchanged compared with the model in which number of diabetes complications was absent (See Figure 9.2). The directional pathway leading from baseline number of diabetes complications to HbA1c at follow-up was statistically non-significant and therefore there was no evidence of confounding by number of diabetes complications. Statistical Appendix E, Table E3 presents unstandardised and standardised estimates associated with this model.

Figure 9.2: Model of the simultaneous effect of cognitions on HbA1c whilst statistically controlling for baseline number of diabetes complications

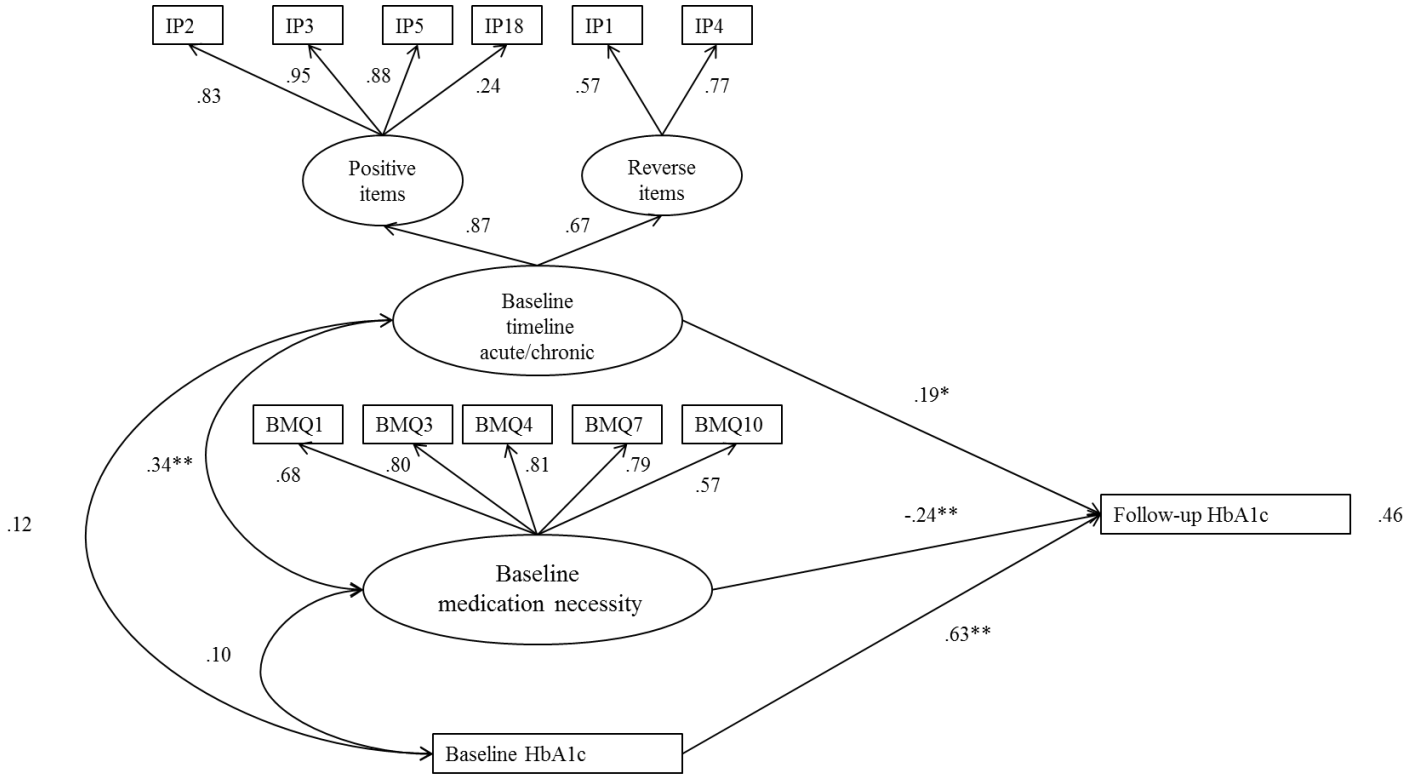


9.4.3 *Sensitivity analyses: missing data*

To examine whether using complete cases analysis impacted on the model fit and parameter estimates obtained for cognitions effect on HbA1c, direct maximum likelihood estimation was used to provide a data set with a sample size of 261. This was based on the number of participants who returned a questionnaire at baseline.

As with the complete cases model all model fit indices demonstrated evidence of good fit with the exception of the model chi-squared index (χ^2=91.1, df_m=60, $p \leq 0.01$; RMSEA=0.05, CFI=0.98, SRMR=.NA, AIC=179.1). Marginal improvements were observed for the CFI fit index. The RMSEA remained unchanged and the model χ^2 statistic and AIC fit index marginally increased. All directional pathways remained statistically significant according to non-bootstrapped p values (bootstrapping cannot be applied in conjunction with direct maximum likelihood estimation by AMOS). Standardised effect size estimates for each of the directional pathways increased by up to ±0.02. Item 18 became statistically significant for the direct maximum likelihood data set. Figure 9.3 presents the standardised parameter estimates for this model and Statistical Appendix E, Table E4 shows both the unstandardised and standardised parameter estimates.

Figure 9.3: Model of the simultaneous effect of cognitions on HbA1c with direct maximum likelihood estimation



9.5 *Chapter summary*

This chapter performed the necessary statistical analyses to address secondary research hypothesis one: baseline diabetes cognition domains and poor emotional health have a combined and independent longitudinal effect on HbA1c at follow-up. The final model showed:

i. Baseline timeline acute/chronic and medication necessity beliefs predicted change in HbA1c levels at six months follow-up. Participants who more strongly believed in the chronicity of diabetes had poorer HbA1c outcomes at follow-up. Conversely, participants who perceived medication to be necessary to the management of diabetes had better HbA1c outcomes at follow-up.
ii. No effect was observed for baseline poor emotional health (conceptualised as depression, anxiety or negative well-being) on HbA1c.

These findings found no evidence to support the combined effect of baseline cognitions and emotions on HbA1c at follow-up. Diabetes cognitions had an explanatory effect on HbA1c independent of poor emotional health.

10 *Discussion*

Poor emotional health is prevalent in adults with diabetes mellitus (Anderson, et al., 2001; Grigsby, et al., 2002). Its presence is associated with worse diabetes health outcomes (Anderson, et al., 2002; de Groot, et al., 2001; Lustman, et al., 2000b). One hypothesis for why this occurs is because negative emotional states cause a person to adhere less to their recommended treatment regimens (Ciechanowski, et al., 2000; Gonzalez, et al., 2008b; Lin, et al., 2004). This thesis aimed to identify the pathways through which poor emotional health impacts negatively on adherence to diabetes self-care behaviours. The Common Sense Self-Regulation Model (CS-SRM; Leventhal, et al., 1980; 1984) was used as a theoretical framework. This thesis explored the:

i) Types of diabetes cognitions associated with poor emotional health.
ii) The direction of effects between diabetes cognitions and poor emotional health.
iii) Combined and independent effect of diabetes cognitions and poor emotional health on adherence to diabetes self-care behaviours.

10.1 *Summary of findings*

10.1.1 *Systematic review with meta-analysis*

The systematic review with meta-analysis (see Chapter Two) studied the relationships between diabetes cognitions, poor emotional health, and diabetes self-care in adults with diabetes. The review had three objectives:

i) To explore which diabetes cognitions were associated with poor emotional health.
ii) To establish whether intervention studies which targeted diabetes cognitions impacted on poor emotional health *and* vice versa.
iii) To examine the relative effects of diabetes cognitions and poor emotional health on diabetes self-care behaviours.

Overall the systematic review identified robust associations between poor emotional health and the cognition domains: timeline cyclical, consequences, seriousness, and personal control. Perceptions that diabetes is a threatening condition with unpredictable symptoms, that negatively impact on functioning, in addition to perceptions of little mastery over

diabetes have salient relationships with poorer emotional health. These findings are consistent with the CS-SRM claims that the cognitive appraisal of a health threat can determine a person's emotional response (Leventhal et al., 1980; 1984). In addition the cognitions identified as having associations with poor emotional health centred on themes of threat, helplessness, and hopelessness, cognitive themes consistently identified among clinically depressed and anxious individuals (Beck et al., 1979; 1987).

The cognition domains identified by the review as having consistent associations with poor emotional health may be important therapeutic targets to improve negative emotional states and consequentially diabetes self-care behaviours. But the direction of effect between illness cognitions and poor emotional health could not be determined because all identified studies were cross-sectional, excluding one. However the longitudinal study showed promise for the directional influence of cognitions on depression, specifically consequences and illness coherence beliefs (Skinner et al., 2011). In addition findings from two intervention studies identified by the review which successfully targeted and improved illness cognitions (personal control and illness coherence beliefs) also reported corresponding improvements in poor emotional health. These findings further reinforce the argument for illness cognitions to have a directional effect on poor emotional health, yet no formal mediation analyses were performed.

Firm conclusions about the potential efficacy of psychological interventions to improve poor emotional health by targeting diabetes cognitions and consequentially influence adherence to diabetes self-care behaviours could not be determined from the systematic review findings. Only two studies which examined the associative relationships between cognitions and emotions went on to explore their relative effects on adherence to diabetes self-care behaviours. However both studies had methodological limitations. One study's statistical analysis assumed complete independence between cognitions and depression. It did not formally explore the potential for mediation to occur between cognitions and emotions (Hampson et al., 2000). Whilst the second study did explore the potential for cognitions to mediate the relationship between depression and diabetes self-care behaviour the alternative mediatory pathway (cognitions → depression → diabetes self-care) was not explored (Nsereko et al., 2013). Without an understanding of how cognitions and poor emotional health operate in combination to determine adherence to diabetes self-care optimal therapeutic targets cannot be identified.

To summarise:

i) Observational studies have identified consistent associations between poor emotional health and the diabetes cognition domains: timeline cyclical, consequences, personal control, and seriousness.
ii) All observational studies reported cross-sectional associations between diabetes cognitions and poor emotional health excluding one.
iii) Intervention studies have not performed mediation analyses to explore whether diabetes cognitions can account for the effect of the intervention on poor emotional health.
iv) Studies have failed to fully explore how cognitions and poor emotional health in combination determine adherence to diabetes self-care behaviours.

10.1.2 ***Main empirical findings***

The systematic review with meta-analysis showed that the CS-SRM's hypothesised bi-directional relationship between cognitions and poor emotional health had not been rigorously tested among adults with diabetes mellitus. Equally, it highlighted that the theorised combined (mediated) and independent (direct) effect of diabetes cognitions and poor emotional health on diabetes self-care behaviours was under-researched. This thesis aimed to advance our theoretical understanding of the CS-SRM and in addition identify plausible targets for clinical intervention to improve both poor emotional health and diabetes self-care. Two primary research hypotheses were tested:

Research hypothesis one: Diabetes cognitions and poor emotional health have a longitudinal bi-directional relationship (cognitions ↔ emotions).

Research hypothesis two: Diabetes cognitions and poor emotional health have a combined and independent longitudinal effect on diabetes self-care, specifically:

a. Diabetes cognitions have a direct and indirect effect (via poor emotional health) on diabetes self-care (cognitions → diabetes self-care *and* cognitions → emotions →diabetes self-care).
b. Poor emotional health has a direct and indirect effect (via diabetes cognitions) on diabetes self-care (emotions → diabetes self-care *and* emotions →cognitions → diabetes self-care).

Research hypotheses one and two were evaluated simultaneously using the robust statistical approach: structural equation modelling (SEM). Two longitudinal models were tested to explore the effects of two alternative conceptualisations of poor emotional health: i) depression and anxiety defined as two conceptually distinct constructs and ii) depression and anxiety defined as two overlapping constructs, negative well-being.

The model with depression and anxiety defined as two distinct constructs showed that participants who were more anxious perceived their diabetes to be more unpredictable and were more worried about their medications. Participants' beliefs about diabetes did not alter participants' levels of depression or anxiety over time. This finding partially supports research hypothesis one and emphasises the dominant role of anxiety in determining a person's appraisal of their diabetes and not vice versa in this population. The absence of an effect of cognitions on depression and anxiety over time is inconsistent with the claims of the CS-SRM (Leventhal et al., 1980; 1984) and cognitive models of emotion which emphasise the importance of negative cognitive appraisals in the onset and maintenance of poor emotional health (Beck, 1967; Beck, et al., 1979).

Contrary to other published findings of a relationship between poor emotional health and adherence to diabetes self-care, neither depression nor anxiety predicted change in adherence to diabetes health behaviours either directly or indirectly via diabetes cognitions. However, the model showed that personal control remained independent of depression and anxiety and predicted change in adherence to diabetes self-care behaviours. Research hypothesis two was thus partially supported. Participants who felt more in control of their diabetes adhered less to their diabetes self-care behaviours. This negative association is inconsistent with previously reported findings which have shown increased mastery beliefs to be associated with increased adherence to self-care behaviours both in diabetes (Broadbent et al., 2011; Watkins et al., 2000) and other physical long term conditions (Hagger and Orbell, 2003).

The findings for the alternative model with poor emotional health conceptualised as negative well-being were consistent with those found for the depression and anxiety model. However a relationship between negative well-being and illness coherence replaced the relationship that was observed between anxiety and medication concerns (see Figures 10.1 and 10.2). Specifically, participants with higher levels of negative well-being felt they had a poorer understanding of their diabetes. This finding is consistent with the

relationship reported by French et al. (2008) using the same measure of negative well-being.

The Aikaike Information Criterion (AIC) fit index was used to evaluate which of the two conceptualisations of poor emotional health best accounted for the relationships between cognitions, poor emotional health, and diabetes self-care. This test showed that the model of the relationships between anxiety, cognitions, and diabetes self-care had the best fit.

Summary of longitudinal findings (See Figures 10.1 and 10.2):

i. There was no evidence of a bi-directional relationship between diabetes cognitions and poor emotional health over time (when conceptualised as either depression and anxiety or negative well-being).

ii. Baseline anxiety and negative well-being predicted change in specific diabetes cognitions at six months follow-up and not vice versa.

iii. Depression showed no relationship with diabetes cognitions in either direction.

iv. The findings did not support the combined effect of diabetes cognitions and poor emotional health on diabetes self-care behaviours.

v. Poor emotional health did not predict change in diabetes self-care behaviours directly.

vi. Personal control had a direct effect on global diabetes self-care and remained independent of poor emotional health (for both conceptualisations of poor emotional health).

vii. The model of the relationship between anxiety, diabetes cognitions, and global diabetes self-care had a better statistical fit compared with the model of the relationship between negative well-being, diabetes cognitions, and global diabetes self-care, and is therefore the preferred model.

Whilst the longitudinal findings showed that: i) the main direction of effect was from poor emotional health to diabetes cognitions, and ii) only personal control predicted change in diabetes self-care behaviours at follow-up; examination of changes in individual patient scores over time and of the auto-regressive pathways in the structural models showed that diabetes cognitions, poor emotional health, and diabetes self-care behaviours remained quite stable over six months. Because of the stability in the variables measured over time, the baseline cross-sectional correlations provide useful insight into the cognitive,

emotional, and behavioural profiles that occur among individuals when they are not experiencing external events that trigger changes in any of these variables.

The baseline cross-sectional analyses as a whole showed that both depression and anxiety had statistically significant associations with the majority of cognition domains. Of these a number of cognition domains had moderate effect size relationships with poor emotional health according to Cohen's (1988) criteria (e.g. $r \geq 0.30$). Specifically, greater identity, consequences, psychological causal attributions, and medication concern beliefs were associated with higher levels of depression, anxiety, and negative wellbeing. This was also true for timeline cyclical beliefs but in relation to anxiety and negative well-being only. Similarly, greater illness coherence beliefs were associated with lower levels of depression and negative well-being but not anxiety. These baseline cross-sectional findings are consistent with the longitudinal findings, but additionally show that identity, consequences, and psychological causal attributions play a key role among participants who are in a stable diabetes self-management illness episode.

Similarly, baseline cross-sectional correlation analyses between global diabetes self-care and cognitions and between poor emotional health showed that a number of diabetes cognitions and depression had statistically significant associations with global diabetes self-care, associations with anxiety and negative well-being were non-significant[7]. However their effect sizes were small ranging from ±0.14 to 0.20 and none of these associations occurred in the longitudinal analyses. In contrast to the longitudinal findings there was no baseline cross-sectional relationship between global diabetes self-care and personal control beliefs.

Secondary research hypothesis findings

As a secondary analysis, a model was generated to test the combined and independent effects of poor emotional health and diabetes cognitions on HbA1c at six months follow-up. The final model showed that diabetes cognitions, not poor emotional health (defined as either depression and anxiety or negative well-being) had an independent effect on HbA1c. Specifically, participants who perceived diabetes to be a chronic condition had elevated HbA1c at follow-up. In contrast those participants who perceived a greater need for

[7] Based on the revised conceptualisation of global diabetes self-care whose univariate and bivariate findings are shown in Statistical Appendix A, Tables A20 to A27

diabetes medications had lower HbA1c at follow-up. This model highlights the potential for diabetes cognitions to act independently of mood to impact on an objective diabetes health outcome. However, the absence of an effect of mood on HbA1c is inconsistent with prior longitudinal studies (Dirmaier et al., 2010; Richardson et al., 2008). Of note, the baseline cross-sectional correlations showed statistically significant relationships between a number of cognitive domains and HbA1c but neither timeline acute/chronic beliefs nor medication necessity beliefs had a relationship with HbA1c at baseline. In addition increased depression, anxiety, and negative wellbeing were associated with elevated HbA1c at baseline although these effects were small ranging from 0.17 to 0.25.

Figure 10.1: Summary of the relationship between anxiety, diabetes cognitions, and diabetes self-care

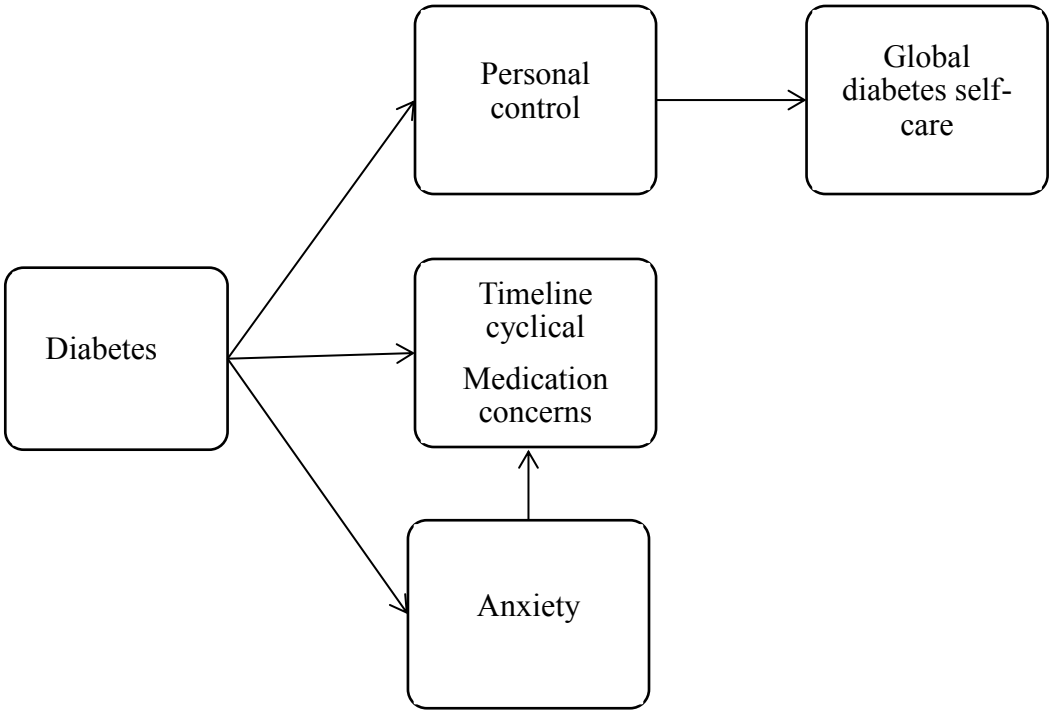
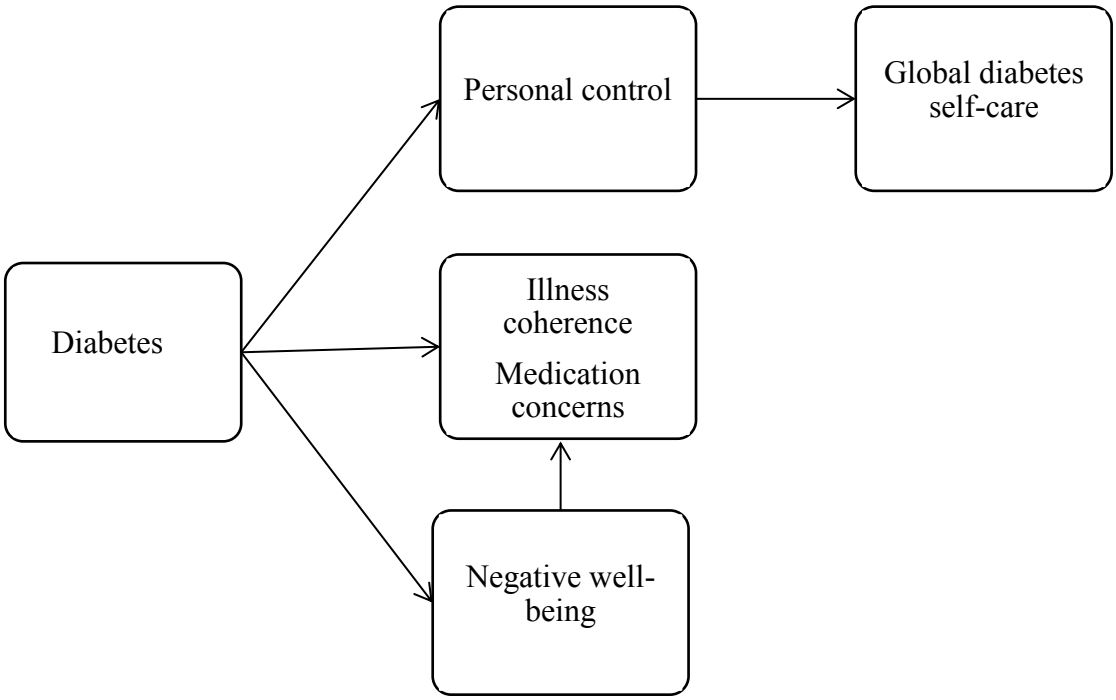


Figure 10.2: Summary of the relationship between negative well-being, diabetes cognitions, and diabetes self-care



10.2 *Methodological strengths and limitations*

10.2.1 *Strengths*

This thesis is the first study to longitudinally test the CS-SRM's hypothesised bidirectional relationship between illness cognitions and poor emotional in adults with diabetes mellitus. The longitudinal design allows greater confidence in the identified relationships because the predictor variables preceded the modelled outcome variables in time (Kenny, 1979).

A large consecutive sample was recruited and retained at six months follow-up (n=194, response rate 74.3%). The sample can be considered typical of outpatients who attend for their routine appointments in UK NHS secondary care. A third of the sample recruited at baseline was from black and minority ethnic groups. This demographic group are typically under-represented in diabetes research (Hussain-Gambles, et al., 2004; Khunti, et al., 2009) despite the higher prevalence of Type 2 diabetes among minority ethnic groups in the UK (Diabetes UK, 2012). The face to face recruitment of participants and providing assistance with the completion of the questionnaire possibly aided with the recruitment of this demographic group. Relatedly, the large sample size allowed sufficient power to detect statistically significant relationships despite effect sizes being small. A statistically significant relationship was observed for a modelled pathway whose standardised beta coefficients was as small as β=-0.14.

The use of SEM strengthens the empirical findings of this thesis. Firstly, latent variables allowed the measurement properties of all self-report measures to be rigorously evaluated using confirmatory factor analysis. This identified problems with specific subscales and allowed them to be corrected before testing their directional relationships. In particular these analyses identified that the treatment control subscale of the Revised Illness Perception Questionnaire caused model estimation failures. It is likely that the treatment control subscale requires further elaboration in the context of diabetes perhaps because of the multifaceted nature of diabetes treatment regimens.

Secondly, health psychology concepts are not directly observable; their measurement is reliant on self-report measures which are susceptible to error. SEM can account for measurement error with the use of latent variables and allows more reliable estimates of relationships compared with traditional statistical approaches (e.g. multiple regression).

Thirdly, because SEM tests multiple dependent variables simultaneously it generates estimates of the entire hypothesised model (e.g. goodness of fit indices). The global model estimates provided a stringent test of the CS-SRM's hypothesised relationships between diabetes cognitions, poor emotional health, and diabetes self-care. Similarly, the concurrent estimation of multiple predictor and outcome variables permitted shared variance between hypothesised model variables to be statistically controlled (Preacher and Hayes, 2008). This prevented erroneous conclusions about the importance of a particular modelled pathway because it was evaluated relative to all other hypothesised pathways.

Fourthly, because nested model comparisons were performed (See Chapter 7, Section 7.2.3 for an explanation of this approach) the statistical significance of hypothesised relationships were evaluated at the model level (e.g. goodness of fit statistics), and not the parameter level (e.g. the statistical significance of pathways). This approach promotes model parsimony. It can account for why statistically significant relationships found in the linear regression analyses ($p \leq 0.05$) were not retained in the structural equation models.

Given that this thesis was limited to two time points, mediation could not be fully evaluated longitudinally. However, Cole and Maxwell's (2003) recommendations for testing mediation longitudinally when limited to only two time points of data collection were applied (See Chapter Seven, Section 7.2.5 for a full description of this approach). This statistical approach allowed the effects of baseline scores on the outcome variable to be statistically controlled for and reduced the potential for spurious relationships to occur.

Sensitivity analyses were performed to ensure that the variance in the outcome variables reflected "true" variance as opposed to capturing changes in the measurement properties of the constructs (e.g. unstable measurement constructs over time). These sensitivity analyses did not alter model conclusions and provide empirical support for the longitudinal validity of the self-report measures used in this thesis. However, studies explicitly focussed on evaluating the psychometric properties of self-report measures overtime would benefit from applying the series of tests recommended by Little et al.(2007).

10.2.2 ***Limitations***

Methodological

The study's sampling frame was not limited to only participants who met diagnostic thresholds for depression and/or anxiety. The reported relationships between diabetes cognitions, poor emotional health, and diabetes self-care therefore apply across a broad continuum of poor emotional health. Based on previous prevalence estimates (Anderson, et al., 2001; Grigsby et al., 2002), it was expected that approximately 20% of the sample would experience clinically significant levels of depression and anxiety. However, because the well-being questionnaire (Bradley, 1994) was used it was not possible to determine the prevalence of depression and anxiety in this study. The well-being questionnaire does not have a validated diagnostic cut-point to categorise participants into those experiencing clinically significant levels of emotional distress from those who are not. This measure was chosen because the systematic review (Chapter Two) identified that it was commonly used by health psychology researchers to explore associations between diabetes cognitions and poor emotional health. Its use allowed this study's findings to be compared with others.

Because the sample was not restricted to participants meeting clinical thresholds for depression and/or anxiety, this may account for why no effect of diabetes cognitions on poor emotional health was observed. Indeed, across the whole sample mean levels of depression and anxiety were low indicating the absence of severe negative mood states. The mean depression, anxiety, and negative well-being scores found in this thesis are comparable with other studies that have used the WBQ among both outpatients with Type 2 diabetes (Paschalides et al., 2004) and among Type 2 diabetes patients attending for their primary care appointments (French et al., 2008). Thus the conclusions drawn from this thesis may apply only to Type 2 diabetes populations who attend for their routine diabetes appointments.

Others have found that biased cognitive appraisals occur only when a person is experiencing an acute depressive episode (Ingram, et al., 1998). The sample in this study may not have included sufficient enough numbers of participants experiencing acute negative mood states to capture bias in participants' appraisal of their diabetes. Similarly, studies which have identified a relationship between poor emotional health and adherence to diabetes self-care have observed these effects among individuals meeting diagnostic thresholds for depression and/or anxiety (Katon, et al., 2009b; Katon, et al., 2010; Lin, et

al., 2004; Mollema, et al., 2001; Zambanini, et al., 1999). Thus poor emotional health may only impact on adherence to diabetes self-care when a certain threshold of severity is reached and may account for why no effect was observed in this study.

The sampling frame of this study was restricted to only outpatients with Type 2 diabetes. This recruitment setting was chosen for practical reasons (e.g. to have greater access to a wider pool of eligible participants), but also for theoretical reasons. Individuals treated in secondary care are more likely to have developed diabetes complications (Diabetes UK, 2010) and studies have found relationships between diabetes complications and depression (de Groot, et al., 2001).

Outpatients typically include patients with more severe or complicated illness of longer duration and in this study participants' mean diabetes duration was 14 years. Because of their experience with the condition the sample had developed a homogenous representation of their diabetes as evidenced by the lack variance in the baseline diabetes cognition measures. This may account for why no effect of diabetes cognitions on poor emotional health was observed. The extreme negative thinking styles that cause, and maintain, negative emotional states in this sample were absent (Beck, 1967; 1976; Beck, et al., 1979). Leventhal et al. (2011) acknowledge that the utility of illness/medication cognitions as predictors of outcomes is limited when there is a lack of variance in how the sample cognitively represent a health threat.

The degree of interaction between diabetes cognitions, poor emotional health, and diabetes self-care may vary according to a person's illness trajectory (e.g. change when diabetes complications develop). Clinical characteristics were extracted from medical records but no formal model subgroup analyses were performed because of an insufficient sample size. Others have found a relationship between diabetes complications and cognitive and emotional responses to diabetes (Rees, et al., 2012; Searle, et al., 2008; Vileikyte, et al., 2009). Exploratory baseline analyses were performed, however. Neuropathy, respiratory or musculoskeletal conditions were associated with a negative cognitive appraisal of diabetes and/or poorer emotional health compared with those participants without these conditions. Equally, participants treated with injectable medications had a pessimistic appraisal of diabetes and adhered more to their diabetes self-care compared with those participants treated with diet/oral medications. Clinical characteristics may be important moderators of a person's cognitive, emotional, and behavioural response to diabetes. Adopting a stratified

sampling strategy in future studies will allow these relationships to be thoroughly evaluated.

The research protocol for this study had specified that inpatients with Type 2 diabetes would be recruited in addition to outpatients. The recruitment of inpatients may have allowed greater change in diabetes cognitions, poor emotional health, and diabetes self-care to be observed in response to the acute illness episode. However it was not feasible to continue with the recruitment of inpatients because the available population of inpatients with Type 2 diabetes was limited and they were often deemed too ill by their clinical team to be approached. In addition access to the wards to non-clinical staff was only permissible at limited times so as not to interfere with clinical procedures. This highlights the difficulty of conducting 'real world' applied research when using ill people for participants.

Longitudinal relationships were tested over a time interval of six months. Gollob and Reichardt (1987) emphasise the importance choosing a correct measurement interval in longitudinal studies. Too short or too long a time interval may mean that a directional effect is missed. The temporal relationships that exist between diabetes cognitions, emotions, and diabetes self-care are unknown because observational studies applying the CS-SRM as a theoretical framework have largely used cross-sectional designs. The directional relationships identified in this study may apply only to those that occur over a six month time interval.

To be comparable with studies that have found a relationship between poor emotional health and diabetes health outcomes (Anderson, et al., 2002; de Groot, et al., 2001; Lustman, et al., 2000a) this study measured depression and anxiety using the diabetes well-being questionnaire (Bradley, 1994). However the CS-SRM describes emotional responses specifically provoked by a health threat. Had diabetes specific measures of distress been used (e.g. Diabetes Distress Scale; Polonsky, et al., 2005a; Problem Areas in Distress Scale, Welch, et al., 1997) then a greater degree of interaction between diabetes cognitions, emotions, and diabetes self-care may have been observed. Therefore disease specific distress should be studied alongside psychiatric models of mood (e.g. depression and anxiety) to tease apart the role that each of these emotional states play.

Similarly, Moss-Morris et al. (2002) encourage researchers to adapt the IPQ-R to specific illness populations to allow greater validity in the constructs being measured. Indeed, as discussed earlier the confirmatory factor analyses showed that the treatment control

subscale was particularly problematic in this sample. Had an adapted version of the IPQ-R been used then this may have increased the ability to detect important relationships between diabetes specific cognitions and both poor emotional health and diabetes self-care. Future studies should use preliminary qualitative approaches for example the "think-aloud" technique used by McCorry et al. (2013) in adults with type 2 diabetes and/or qualitative interviews used by Lynch et al.(2011) to develop the IPQ-R for use in depression. Using preliminary qualitative approaches will allow the content of the IPQ-R items to be tailored to specific health conditions and provide more meaningful findings.

A related measurement issue concerns the summary of diabetes self-care activities scale (SDSCA; Toobert et al., 2000). Toobert et al. (2000) advise against creating a global measure of diabetes self-care and instead recommend using each SDSCA subscale individually. This study used a global measure of diabetes self-care because confirmatory factor analyses indicated that it was statistically acceptable to do so once the medication adherence and self-monitoring of blood glucose subscales were excluded from the model. Gonzalez et al's (2008b) meta-analysis of the relationship between depression and diabetes self-care found that the largest effect sizes were observed among studies that used a global measure of diabetes self-care. Thus the global conceptualisation of diabetes self-care likely increased the chances of this study detecting a statistically significant relationship between diabetes self-care and poor emotional health specifically.

The exclusion of measures of medication adherence and self-monitoring of blood glucose is a clear limitation of this study particularly because medication adherence is a key component of diabetes self-management. Because medication adherence was measured with only a single item it demonstrated a ceiling effect with almost all participants reporting that they adhered to their medication seven days per week (see normality tests in Statistical Appendix A). This prevented the use of this item for any further testing using SEM. Likewise, participants tended to score at the extremes of the self-monitoring of blood glucose scale reporting either total adherence to self-monitoring or none at all (see normality tests Statistical Appendix A). This response pattern may reflect the varied advice patients receive in relation to self-monitoring of blood glucose which can depend on their medication regimen (NICE, 2008) and/or the behaviours of their health care professional (Peel, et al., 2007). This pattern of responding prevented the use of this subscale on its own for any further testing using SEM. Measures of adherence to self-monitoring of blood glucose regimens need to be supplemented with a screening question (e.g. does your

doctor/nurse encourage you to self-monitor your blood glucose levels?) to ensure that this measure of adherence is used only among participants in whom it is relevant. In addition objective pharmacy claims data for self-testing strips will help reduce the effect of social response bias.

Additionally, the self-report nature of the SDSCA may have caused participants to respond in a socially desirable manner (Kristal, et al., 1990). Response bias for this study may have been heightened given that participants were recruited whilst attending for their clinical appointments. Studies that have used objective measures of diabetes self-care (e.g. pharmacy claims data) reported larger effect sizes between depression and diabetes self-care compared with studies that have relied on self-report measures (Gonzalez et al., 2008b). Had this study had access to such data, then more statistically significant relationships may have been observed.

The use of number of diabetes complications as a measure of diabetes severity may also be problematic. There is no gold standard for measuring diabetes severity or co-morbidity. Based on Hall's (2006) recommendations, diabetes complication and other health co-morbidity categories were generated that were mutually exclusive to avoid double counting (see Chapter Four, Table 4.1). However, the categories were not weighted according to the degree to which each impacted on diabetes health outcomes. Different weightings may apply depending on the outcome of interest (e.g. glycaemic control, mortality, emotional health, quality of life, and health care use). There is a diabetes complication severity index (Young, et al., 2008), but this measure was validated for examining the impact of diabetes complications on both mortality and re-hospitalisations. In addition access to (US based) pharmacy records was needed to use this measure and therefore was not feasible for this thesis.

Diabetes complications and other health co-morbidities were extracted from participants' medical records. This measurement approach may have been biased because the information may not have been fully recorded. However because these variables were not the main outcome measures it was felt justified. Future studies should validate the use of medical note extraction with full medical interviews.

Similarly, relying on medical records to record HbA1c allowed less control over the temporal lag between the measurement of baseline and follow-up HbA1c. These concerns can be slightly attenuated however because the mean number of days from baseline

recruitment to the measurement of baseline HbA1c was ± 2.6 days. Equally, at follow-up the mean number of days from the follow-up recruitment date to the measurement of HbA1c was ± 12.5 days. In addition, because follow-up measures of HbA1c were extracted one month after the completion of baseline questionnaires it allowed the measurement of cognitions and emotions to precede the measurement of HbA1c.

Statistical considerations

The CS-SRM provided a framework to identify directional pathways of interest between diabetes cognitions, emotions, and diabetes self-care. The precise diabetes cognition domains that were of key importance for the model could not be determined a priori based on empirical studies. All studies (excluding one) that had previously studied the relationship between diabetes cognitions and poor emotional health used cross-sectional designs (see chapter two). This limited conclusions about the directional relationships that occur between these constructs. As a consequence this thesis empirically identified directional relationships for study with structural equation models using univariate and multivariate regression analyses. Although these relationships could themselves have been identified using SEM, a regression approach was adopted for efficiency and to reduce the very considerable workload that would have been involved. Conservative (e.g. inclusive) criteria were used for the regression results to ensure that potentially important relationships were not excluded prior to SEM modelling.

This study's findings may only apply to the particular patient groups sampled for this thesis. The models need to be cross-validated in an alternative outpatient sample with diabetes. The approaches used in this thesis can shape the design of such a cross-validation study.

Whilst the relatively large sample size of 194 participants at follow-up can be considered a strength it is also a limitation given the complexity of the statistical analyses performed. A sample size of 200 participants at follow-up is recommended to allow stable parameter and model fit estimates to be obtained. This sample size at follow-up was almost achieved. However because SEM requires complete cases on all data points sample sizes across each of the evaluated models ranged from N=153 to N=174 once list wise deletion was applied. This raises concerns about the reliability of the statistical estimates reported in this thesis for the structural models as they may have been susceptible to sampling error.

This thesis did however make use of statistical approaches to manage missing data. Firstly item parcelling (described in Chapter Three, Section 3.4.4) was applied to all cognition and emotion latent variables when evaluating the larger combined models of the simultaneous relationships between diabetes cognitions, emotions, and diabetes self-care. Subscale means were used to generate item parcels thus allowing mean imputation to be applied so decreasing loss of cases due to item level missing data. Item parcelling also increases the participant to parameter ratio and improves the reliability of statistical estimates (Bentler and Chou, 1987).

Secondly, sensitivity analyses were performed using direct maximum likelihood estimation. Direct maximum likelihood estimation is an alternative method of handling missing data and allowed all participants who returned questionnaires at baseline (N=261) to be included in model estimates. Although concerns are sometimes expressed about imputation 'making-up' data, this approach also increases the volume of actual data in the analysis through inclusion of cases with partially-complete data. This increased the working sample size to above the minimum recommended sample size of N=200 for SEM (Anderson and Gerbing, 1988; Chou and Bentler, 1995; Gerbing and Anderson, 1984; Quintana and Maxwell, 1999; Tanaka, 1987).

Neither method of handling missing data (e.g. list wise deletion vs maximum likelihood estimation) had any impact on overall model and directional parameter conclusions, but the effect size of directional pathways in some instances changed by up to ±.08. The findings from the models with direct maximum likelihood applied should be considered more robust given that studies have shown this method of handling missing data to be more reliable compared with list wise deletion (Allison, 2009; Arbuckle, 2007). Future studies should consider not only the impact of attrition in longitudinal studies but also how best to manage the impact of missing data and report clearly the methods they applied to manage missing data. Completing questionnaires with participants or electronic questionnaires which flag up missing responses may overcome some of these issues.

The use of direct maximum likelihood estimation to address missing data meant that bootstrapping could not be applied even though variables were found to be non-normally distributed. Nevertheless the statistical significance of parameters obtained using this approach did not differ from those obtained when bootstrapping was applied. Thus concerns about inflated Type I error rates can be offset (Arbuckle, 2007; Byrne, 2010;

West, et al., 1995). In addition, item parcelling is an approach that can be applied to address non-normally distributed data but it is less statistically robust than bootstrapping.

No adjustments were made for multiple statistical testing (e.g. bonferroni correction) despite this thesis testing a large number of theoretically plausible predictive pathways using a p value of 0.05 to determine the statistical significance of each hypothesised pathway. Assuming each test to be independent (although this is unlikely) then around one in every twenty (e.g. 5%) statistically significant pathway is likely to be a Type I error. However, this thesis is the first to explore longituduinally the relationships between cognitions, emotions, and diabetes self-care, thus it was felt justified to adopt the 5% alpha level. Although the use of multiplicity adjustments, or the adoption of a more conservative alpha-level (eg 1%, or even 0.1%), would reduce number of Type I errors at the same time it would increase the Type II error rate (the probability of false negatives). In exploratory studies this can prevent theoretically important relationships from being identified (Rothman, 1990). The findings of this thesis need to be considered provisional, requiring confirmation in one or more alternative samples to have greater confidence that the identified pathways between cognitions, poor emotional health, and diabetes self-care are genuine and not the outcome of chance associations within this particular study.

10.3 *Interpretation of findings*

Contrary to hypotheses no bi-directional relationship was found between diabetes cognitions and poor emotional health: the principal direction of effect was from poor emotional health (anxiety and negative well-being) to diabetes cognitions only. Equally poor emotional health did not predict diabetes self-care either directly or indirectly via diabetes cognitions. Personal control remained independent of emotion and had a direct effect on diabetes self-care. These relationships occurred independently from number of diabetes complications.

The lack of an effect of diabetes cognitions on poor emotional health over time is at odds with both the CS-SRM's claims and cognitive models of emotional disorders which hypothesise that negative cognitions cause and maintain negative mood states (Beck, 1967; Beck et al., 1979). The CS-SRM does not precisely define when and for whom an effect of illness cognitions on poor emotional health will be observed. In contrast, Beck et al. (1979) describes that cognitions only impact on mood once a person's maladaptive schemas are activated. Maladaptive schemas are triggered by environmental factors (e.g. stressful life

events) and/or negative mood states. Negative schema activation increases as the severity of negative mood states increase. Once active, schemas function as a framework to appraise and understand situational factors.

It is plausible that no effect of diabetes cognitions on poor emotional health was observed in this study because participants' negative illness representation schemas were dormant. Consistent with Beck et al. (1979) this study highlights the potential for negative mood states to provoke the activation of negative illness representation schemas, because the main direction of effect was from poor emotional health to diabetes cognitions and not vice versa. However, because poor emotional health had a small effect on diabetes cognitions (standardised beta coefficients ranged from ±0.17 to 0.25) the samples' negative mood states were not severe enough to activate negative illness representation schemas. Indeed the effect sizes observed for baseline diabetes cognitions as predictors of themselves at six months follow-up were moderate to large (standardised beta coefficients ranged from 0.57 to 0.67). This indicates that diabetes cognitions were mainly determined by participants' prior beliefs about diabetes. Equally, baseline poor emotional health predicted itself at six months follow-up, with standardised beta coefficients of β=0.91 and β=0.85 for anxiety and negative well-being respectively. This demonstrates that negative mood states were stable over a six month period and were not affected by diabetes cognitions.

The stability of cognitions and emotions is further reinforced by the relatively little individual change in scores over time. The unchanging cognition, emotion, and behaviour profiles of the sample limited the ability to detect important changes in outcome variables over time because the external event which triggered change in individuals cognitive, emotional, and behavioural response to diabetes likely occurred at an earlier point in time, for example diagnosis (Hertzog and Nesselroade, 1987; Little, et al., 2006; Maruyama, 1997). Nevertheless, cross-sectional findings do provide some indication of the cognition-emotion relationships that occur when a person has an established illness representation schema and has emotionally adjusted to their diabetes.

Theories of emotion (Beck 1965; Beck et al. 1979; Forgas, 2000; Teasdale, 1983; 1988) can explain how poor emotional health impacts on diabetes cognitions. Negative mood states can alter a person's attentional processes by causing individuals to attend to mood congruent information (Beck et al., 1979; Teasdale, 1983, 1988). Anxiety and negative

well-being may have led participants to direct their attention to bodily symptoms and attribute these symptoms to:

i) the unpredictable nature of diabetes (e.g. increased timeline cyclical beliefs)
ii) being a consequence of their medications (e.g. increased medication concerns, anxiety only)
iii) a lack of comprehension of their diabetes (e.g. lower illness coherence beliefs, negative well-being only).

Poor emotional health did not predict identity beliefs. This suggests that participants did not attribute a greater number of symptoms to their diabetes but were instead hyper-vigilant to the day to day course of the symptoms that they had already attributed to their diabetes, for example the symptoms associated with hypoglycaemic episodes (e.g. sweating, confusion). Hyper-vigilance to bodily cues is consistent with cognitive models of anxiety disorders (Wells, 1997).

Poor emotional health can also cause individuals to retrieve mood congruent memories and to elaborate further on their cognitive content (Teasdale, 1983; 1988; Forgas, 2000). In this study participants may have retrieved pessimistic cognitions about chronic illness which reflect the:

i) Varying trajectory of chronic illness (timeline cyclical beliefs).
ii) Potential for medicinal treatments to have harmful effects (medication concerns, anxiety only).
iii) Overwhelming nature of chronic illness (illness coherence, negative well-being only).

Leventhal et al. (1980) also provides an account of how poor emotional health can impact on illness cognitions hypothesising that individuals may apply their mood as a heuristic to interpret their physical health. Participants experiencing greater levels of anxiety and negative well-being may experience somatic symptoms associated with negative affect e.g. shaking, sweating, lethargy, confusion. These symptoms overlap with the somatic experience of hypo/hyperglycaemic episodes leading participants to erroneously conclude that:

i) Diabetes is unpredictable (timeline cyclical).

ii) Medications have substantial side effects (medication concerns, anxiety only).
iii) They do not understand their diabetes (illness coherence, negative well-being only).

The association between poor emotional health (anxiety and negative well-being) and timeline cyclical beliefs are consistent with the robust associations found in the systematic review (see chapter two), thus decreasing concerns that model findings are subject to Type I errors. However the relationships that exist between medication concerns, illness coherence, and poor emotional health have been less frequently tested empirically. The model findings with regards to the negative correlation between medication concerns and anxiety are consistent with the directional relationships found by French et al. (2008). However the association reported by French et al. (2008) was in relation to the emotional health concept negative well-being not anxiety. Similarly, the negative correlation found between negative well-being and illness coherence is consistent with the findings of Rees et al. (2012) and French et al. (2008), yet others have shown a positive association between poor emotional health (specially depression) and illness coherence (Skinner et al., 2011). The role that illness coherence plays in relation to poor emotional health needs further investigation. It may be that a coherent understanding of illness is only adaptive for emotional health when an individual feels empowered and sufficiently resourced (both physical and treatment resources) to use effectively their illness knowledge.

The bivariate cross-sectional findings, specifically the cognition domains that had a moderate effect size relationship with poor emotional health and that did not occur longitudinally (identity, consequences, and psychological attributions) are largely consistent with the systematic review findings reported in chapter two. However the relationship between psychological causal attributions and poor emotional health has not been studied previously.

The absence of a longitudinal relationship between depression specifically and diabetes cognitions is likely to be a function of the content of the WBQ depression items. The depression subscale items largely consist of the psychological/behavioural facets of depression for example “I feel that I am useful and needed” (Bradley, 1994; Zung, et al., 1965). In contrast the anxiety subscale largely contains items that reflect affective elements of anxiety for example “I feel nervous and anxious” (Bradley, 1994; Zung, 1974).

Similarly the negative well-being subscale consists solely of items that represent the affective elements of mood (Bradley, 2000; Riazi, et al., 2006). Thus the dominance of anxiety and negative well-being in relation to cognitions may reflect the importance of affective components of mood as opposed to depression being unimportant. These findings are consistent with Beck et al's (1979) hypothesis that negative illness representation schemas are activated by emotions.

Unlike previous cross-sectional and longitudinal studies this study found no longitudinal relationship between poor emotional health and decreased adherence to diabetes self-care (Gonzalez, et al., 2008b; Katon, et al., 2009b; Lin, et al., 2004; Mollema, et al., 2001; Zambanini, et al., 1999). As discussed earlier, the null findings are likely attributed to the fact participants in this study were not sufficiently depressed and/or anxious for poor emotional health to negatively impact on functioning. Indeed impaired functioning is one of the criteria used by the ICD-10 (World Health Organization, 2010) to distinguish between individuals experiencing a mild episode of depression from those experiencing a moderate to severe episode. It is likely that the effects of poor emotional health on diabetes self-care are only observed when a person is experiencing moderate to severe levels of emotional distress. This argument is further reinforced by the absence of a bivariate cross-sectional relationship between poor emotional health domains and global diabetes self-care excluding depression, however the effect size was small (r=-0.14).

Consistent with the study's hypotheses diabetes cognitions had a direct effect on diabetes self-care. This relationship was observed for the cognition domain personal control only and occurred independent of poor emotional health in both models. The independence of personal control from poor emotional health is inconsistent with the meta-analysed findings of the systematic review presented in chapter two. However, the meta-analysed findings were based on cross-sectional associations. Skinner et al. (2011) reported that personal control did not predict depression at eight months follow-up when baseline personal control was included in the regression model. This study replicated Skinner et al's (2011) findings over an interval of six months using SEM. Thus, previously reported cross-sectional associations between poor emotional health and personal control may be spurious and these relationships may be accounted for by the confounding effect of prior levels of personal control. Whilst four cognition domains had statistically significant cross-sectional associations with global diabetes self-care their effect size was small. These domains may

have demonstrated longitudinal effects with diabetes self-care if participants were recruited and followed up at a critical illness episode (e.g. development of a complication).

The significance of personal control beliefs in determining adherence to problem focussed coping behaviours has been found across a number of long-term conditions (Hagger and Orbell, 2003). In the context of diabetes cross-sectional studies have shown increased personal control beliefs to be associated with greater adherence to diabetes self-care behaviours, specifically diet and exercise behaviours (Broadbent, et al., 2011; Watkins, et al., 2000). Whilst this study supports the importance of personal control as a predictor of diabetes self-care, the direction of effect observed contradicts previously reported findings. In this study greater personal control beliefs at baseline predicted lower levels of diabetes self-care at six months follow-up.

The relationship between personal control and diabetes self-care found in this study may be accounted for by the higher order cognitive construct executive functioning. Executive functioning oversees a person's response to a health threat by managing conscious deliberative processes and automatic habitual processes (Leventhal, et al., 2011). In this study participants' mean diabetes duration was 14 years, thus participants' executive functioning may have largely consisted of automatic habitual processes. That is, participants may have developed a set behavioural pattern to manage their diabetes and feel confident in their ability to perform these behaviours, but these habitual behaviours may deviate from clinical recommendations. Consistent with this explanation Phillips et al. (2013) found that a measure of habit strength was the strongest predictor of medication adherence among adults with hypertension relative to medication and coherence beliefs.

This finding has important implications for treatment interventions which promote a sense of mastery and empowerment to encourage adherence to diabetes self-care behaviours (e.g. DESMOND, Davies et al., 2008; Khunti et al., 2008; 2012). Indeed the three year follow-up report of the DESMOND trial (Khunti et al., 2012) showed that the initial benefits observed for weight (arguably a proxy measure of behavioural adherence) and smoking status at 12 months (Davies et al., 2008) were not sustained at three year follow-up.

Leventhal et al. (2011) emphasise the importance of anchoring individuals' cognitive representations with specific action plans to achieve behavioural change. Action plans provide an explicit behavioural procedure that should be performed to achieve clinical goals. Cognitive illness representations determine the detection of deviant health outcomes

via conscious declarative processing. An appropriate action plan is selected in response to this detection. However if a person does not have i) appropriate cues to detect poor health and/or ii) a detailed action plan, then a person's efforts at managing these deviations from clinical goals are ineffective. The integration of an action plan into a person's daily routine allows habitual cues to be developed and encourages the automation of self-care behaviours which require less conscious processing, for example placing medication next to a toothbrush (Leventhal et al., 2011). Nevertheless a balance needs to be maintained between automatic and declarative processing. Participants in this study may not have developed sufficient action plans to allow their personal control beliefs to be capitalised on.

The baseline cross-sectional findings further reinforce the action planning argument. There was no statistically significant relationship between baseline personal control and baseline global diabetes self-care. Thus, participants high in personal control are less likely to consciously engage in declarative processes about what it means to be personally in control of diabetes. It is likely that patients' perspectives of personal control over diabetes are distinct from medical practitioners' perspectives. Individuals with high perceptions of personal control over their diabetes may feel that they can "bend the rules" of adherence and this may account for the decrease in adherence over time in this study. However this argument is speculative and warrants further examination qualitatively.

Equally, Bandura's (1977) social cognitive theory emphasises the importance of both high self-efficacy beliefs *and* outcome expectations for determining behaviour. In the present study participants may have felt highly efficacious in their ability to perform diabetes self-care behaviours, but lacked correspondingly high outcome expectations. This explanation is consistent with Peel et al's (2007) qualitative work among patients with Type 2 diabetes who used self-monitoring of blood glucose devices. Peel et al. (2007) found participants were confused when their self-tested glucose readings were contradictory to their expectations. This caused patients to disengage with this self-care routine. These findings are likely to generalise to other diabetes self-care behaviours. For example, individuals might anticipate dramatic changes in weight loss and associated benefits (e.g. lower HbA1c, blood pressure, cholesterol) after having only adhered to a diet and exercise regimen for a short period of time.

Whilst an effect of diabetes cognitions on diabetes self-care was observed it occurred longitudinally for only one of the nine cognition domains measured. Leventhal et al. (2011) acknowledge that treatment beliefs may be more robust determinants of health behaviours than illness related beliefs. Indeed French et al. (2013) found this to be the case among adults with Type 2 diabetes. Specifically, participants with greater baseline exercise necessity beliefs and lower dietary necessity beliefs adhered more to exercise behaviours at 12 months follow-up. Illness cognitions demonstrated no effect on adherence. This thesis measured beliefs about medicines but their relationship with medication adherence could not be explored because of the lack of variance in this variable.

The secondary analyses tested longitudinally the combined and independent effect of diabetes cognitions and poor emotional health on HbA1c. Baseline diabetes cognitions (timeline acute/chronic and medication necessity) remained independent of poor emotional health and predicted HbA1c at six months follow-up. Baseline poor emotional health did not predict HbA1c.

This study is the first to test longitudinally the relationship between diabetes cognitions (measured using the IPQ-R) and HbA1c. Timeline acute/chronic and medication necessity beliefs predicted HbA1c at follow-up. These effects were not found in McSharry et al's (2011) meta-analysis of illness cognitions and HbA1c; however medication beliefs were not included in the review.

Greater baseline medication necessity beliefs predicted decreased HbA1c at follow-up. A one point increase on the self-report measure of medication necessity beliefs was associated with a 6.8mmol/mol (~0.5%) decrease in HbA1c at follow-up. It is likely that participants with greater medication necessity beliefs adhered more to their medication regimen. These relationships could not be tested using SEM because of ceiling effects in the measurement of medication adherence.

Participants with increased timeline acute/chronic beliefs had increased HbA1c at follow-up. Others have found a positive correlation between timeline acute/chronic beliefs and greater intake of sugar (Searle, et al., 2007). Participants who have had diabetes for longer are likely to have greater timeline acute/chronic beliefs. This relationship may reflect the difficulties patients with chronic medical conditions face when attempting to sustain behavioural adherence over time.

The lack of a longitudinal relationship between poor emotional health and HbA1c is inconsistent with other longitudinal studies, yet these studies performed their analyses on participants who met diagnostic thresholds for depression (Dirmaier, et al., 2010; Richardson, et al., 2008). As discussed above it is likely that poor emotional health has to reach a certain threshold of severity before it impacts negatively on both health behaviours and likewise health outcomes.

Of note the cognitions that demonstrated a statistically significant cross-sectional relationship with HbA1c at baseline (identity, timeline cyclical, consequences, medication concerns, and psychological attributions) did not demonstrate their effects longitudinally. Equally the relationship between timeline acute/chronic and medication necessity was not observed at baseline. This raises an important question about the temporal relationships that exist between cognitions and HbA1c. Arguably the cognitive constructs that had a baseline relationship with HbA1c could be provoked by the somatic experience of diabetes. Thus participants were using their concrete experience of diabetes to inform their cognitive appraisal of the condition. This may account for why a synchronous relationship occurred for these constructs. In contrast, timeline acute/chronic and medication concerns are more abstract constructs that are likely shaped by external sources of information (e.g. information from a health care professional) and inform a person's efforts at managing their diabetes over time. These findings are consistent with Leventhal et al's (1980) explanation of the concrete and abstract illness experience. Equally all poor emotional health constructs showed a statistically significant relationship with HbA1c at baseline and the temporal relationships that exist between mood and HbA1c remain unclear.

10.4 *Theoretical and clinical implications*

Whilst the CS-SRM provides an appropriate framework to explore the pathways through which the management of physical and emotional health interact this thesis has raised a number of theoretical questions and associated clinical implications. Therefore an elaborated version of the CS-SRM is proposed in Figure 10.3 and the rationale for this elaborated model is discussed below.

10.4.1 *Interaction triggers*

The CS-SRM is static in its description of how the management of physical and emotional health interact. It does not clearly define when and for whom the cognitive and emotional processing systems become dependent on each other. This thesis has shown that among participants who attend for their routine outpatient appointments the extent to which poor emotional health disrupts diabetes self-management and vice versa is limited over time.

The CS-SRM needs to be elaborated on to include "interaction triggers" to define when an interaction is likely to occur. As described earlier the extent of the interaction is likely determined by clinical events (e.g. the development of diabetes complications) and/or the severity of negative emotions. Equally an interaction may be triggered by factors external to the management of diabetes, for example bereavement. "Interaction triggers" need to be incorporated into the CS-SRM to prompt researchers to explore these factors but also to allow clinicians to better recognise the presence of negative emotional states in the context of a physical long term condition. "Interaction triggers" are shown in Figure 10.3 next to the health threat to signal that they are likely to provoke a person to engage in a process of cognitive and emotional re-appraisal of their diabetes.

10.4.2 *Translating cognitions into problem focussed behaviours*

When patients attend for health care appointments with a cognition and emotion profile consistent with one identified in this study (e.g. with no interaction occurring between diabetes cognitions and poor emotional health) clinicians can focus their efforts on improving or maintaining their patients diabetes self-management behaviours.

This thesis identified personal control and medication necessity beliefs as important and modifiable determinants of either diabetes self-care or HbA1c. How clinicians use these beliefs to encourage patients to initiate and maintain behavioural adherence needs to be

more precisely defined by the CS-SRM. This thesis showed that high perceptions of personal control to manage diabetes were insufficient on their own to promote adherence to adaptive self-care behaviours.

Leventhal et al's (1970) early work emphasised the importance of combining health communication messages with specific action plans to promote the uptake of healthy behaviours. But the significance of action plans in translating cognitive illness representations into behavioural change is not emphasised enough. Action planning is therefore explicitly represented in Figure 10.3.

Action planning should be incorporated into all clinical appointments. Clinicians need to be skilled in assessing their patients' cognitive model of diabetes. They must explore their patient's confidence in diabetes self-management behaviours, the types of behaviours they perform, their behavioural triggers (e.g. experiencing symptoms), and their outcome expectancies. This will allow the clinician to develop a tailored action plan with achievable goals (e.g. to manage outcome expectancies) and explicit prompts to promote positive habitual behaviours (e.g. checking feet after showering).

10.4.3 ***Depression/anxiety vs diabetes distress***

There is an important distinction to be made between depression and anxiety prompted by factors external to diabetes (e.g. loss of job) and depression and anxiety arising as a result of diabetes which shall be referred to as diabetes distress (e.g. feelings of hopelessness towards the management of diabetes, fear of hypoglycaemia, social anxiety about self-testing in public). Currently, the CS-SRM limits its conceptualisation of emotional states to only those provoked by the health threat. This thesis found a limited association between poor emotional health, diabetes cognitions, and diabetes self-care when measured with a generic depression and anxiety scale.

Quantitative studies have shown depression and diabetes distress to have distinct effects on diabetes self-care behaviours and health outcomes (Fisher, et al., 2008a; Fisher, et al., 2010; Gonzalez, et al., 2008a; Van Bastelaar, et al., 2010). Equally, McSharry et al's (2013) qualitative interviews among participants with diabetes and diagnosed depression identified two distinct groups of participants in terms of how they cognitively represented their diabetes and depression. Group one perceived their depression to be completely distinct from their diabetes in contrast group two integrated the presence of their

depression with the presence of their diabetes and viewed the two conditions as causally related. These studies further reinforce the need to distinguish depression and anxiety from diabetes distress. The nature of the interaction that occurs between the management of physical and emotional health may differ according to the type of emotional states experienced. By acknowledging these differences more tailored psychological interventions can be developed. This distinction is shown in Figure 10.3.

10.4.4 *Mechanisms of action and targets for intervention*

The question of how poor emotional health can be effectively managed in the context of diabetes remains unanswered. Currently in the UK, cognitive behavioural therapy (CBT) is the main recommended psychological treatment for the management of depression in physical long term conditions (NICE, 2009). However, systematic reviews of the effectiveness of CBT in the context of other physical long-term conditions, specifically coronary heart disease (Dickens, et al., 2013) and chronic obstructive pulmonary disease (Coventry, et al., 2013) have shown CBT to have small effects on depression outcomes. A standardised mean difference of 0.23 ($p \leq 0.05$) and 0.17 ($p > 0.05$) was reported by Dickens et al. (2013) and Coventry et al. (2013) respectively.

This thesis used the CS-SRM as a framework to explore how poor emotional health operates in the context of diabetes. The ultimate aim was to identify plausible targets for psychological intervention to improve poor emotional health, without hindering diabetes self-management behaviours.

This thesis showed diabetes specific cognitions to have no effects on poor emotional health (depression, anxiety or negative well-being) over time. Cognitive reappraisal therapy techniques (Beck, et al., 1979) addressing specifically the content of a person's beliefs about their diabetes will have limited effects on poor emotional health and alternative cognitive therapy techniques should be considered. This clinical implication should be interpreted cautiously and within the remit of the methodological limitations of this study (e.g. lack of severity of poor emotional health, no measure of diabetes distress, stability of the sample's cognition-emotion profile).

Nevertheless, cognitive reappraisal treatments models for the management of depression and anxiety in the context of physical long-term conditions are problematic. Some of the pessimistic and apprehensive thinking styles held by individuals about their diabetes often

are not cognitive distortions but accurate perceptions of the consequences of having a long-term condition. Because these beliefs are not erroneous they cannot be targeted using cognitive reappraisal techniques. Equally, acknowledging the presence of a physical long-term condition and its seriousness is needed to promote behavioural adherence (Detweiler-Bedell et al., 2008, Leventhal et al., 2001). Thus cognitive treatment models for depression and anxiety need to be more tailored to the needs of patients with physical long term conditions and explicitly acknowledge the potential for physical and emotional health management antagonisms to occur. Current psychiatric treatment models do not take into account these relationships.

If diabetes cognitions do cause and maintain poor emotional health among individuals experiencing clinically significant levels of emotional distress then alternative cognitive therapies for mood disorders may offer a more feasible intervention strategy. Cognitive treatments including meta-cognitive therapy (Wells, 2009), acceptance and commitment therapy (Hayes, et al., 2011), and mindfulness based interventions (Segal, et al., 2002) do not address the content of a person's thoughts but target a person's maladaptive response style to these thoughts. These treatment models argue that it is a person's ruminative response style to their negative thoughts that maintains negative emotional states. These models encourage a person to decentre from their thoughts by applying techniques such as: cognitive diffusion (e.g. observing thoughts; Hayes, 2004) and detached mindfulness (e.g. distinguishing the self from cognitions, (Segal, et al., 2002; Wells, 2009). These strategies stop a person from engaging in habitual maladaptive response styles, whilst allowing a person to maintain diabetes beliefs needed for behavioural adherence.

The potential for these alternative cognitive models to manage negative emotional states in the context of physical long-term conditions is unclear. The CS-SRM needs to be elaborated on to include rumination (See Figure 10.3) as a potential mechanism that maintains negative emotional states. Its inclusion in the CS-SRM will prompt researchers to explore ruminative processes and determine their potential worthiness as targets for clinical intervention.

This thesis showed that the main direction of effect was from poor emotional health to diabetes cognitions. Given this finding the most effective psychological interventions for managing negative emotional states in the context of diabetes may be to target these mood states directly. Mindfulness based interventions (Segal et al, 2002) may be useful

techniques to apply. Mindfulness promotes arousal reduction by encouraging the practice of meditative exercises. The practice fosters an appreciation of the present moment and explores the relationships between body and mind. It prevents a person from directing their attention in a judgemental, self-focussed manner and allows a person to i) objectively appraise situations and ii) disengage from maladaptive coping behaviours (e.g. avoidance a behaviour that has been shown to occur among individuals with diabetes; Lawson, et al., 2005).

A randomised controlled trial of mindfulness based cognitive therapy in adults with Type 1 and 2 diabetes successfully improved depression (Cohen's d=0.70), anxiety (Cohen's d=0.44), and diabetes distress (Cohen's d=0.70; van Son, et al., 2012) at 8 weeks follow-up. These findings highlight the promise of mindfulness but no associated improvements in HbA1c were found. Similarly, Hartman et al., (2012) showed that a mindfulness based stress reduction program in adults with Type 2 diabetes significantly lowered levels of depression (Cohen's d=0.71) at 12 months follow-up, but likewise to van Son et al. (2012) no corresponding improvements in diabetes health outcomes (specifically albuminuria progression, a key risk factor for the development of diabetes complications) were found.

Equally, if poor emotional health impacts on diabetes cognitions when individuals use mood as a heuristic for their physical health (Leventhal et al., 1980) then objective measures of diabetes control (e.g. self-testing devices) need to be integrated into a person's self-care routine. Objective blood glucose tests provide a behavioural experiment (Wells, 1997) to challenge potentially inaccurate timeline cyclical, medication concerns, and illness coherence beliefs as was found in this thesis. This is consistent with McAndrew et al's (2008) "bottom up" approach to designing treatment interventions for managing physical long-term conditions, whereby objective somatic cues are used to promote adaptive illness representations and behaviours.

There is an important distinction to be made between pessimistic but accurate diabetes cognitions (e.g. consequences, timeline acute/chronic beliefs) and inaccurate diabetes cognitions that occur when unreliable evidence is used to appraise health (e.g. mood). The types of diabetes cognitions held by individuals may determine the type of psychological treatments used. For example, individuals perceiving diabetes to have high social and physical health consequences may benefit from psychological treatments which focus on acceptance of these negative cognitions. In contrast individuals who are appraising their

diabetes in a negative manner based on their somatic experience (e.g. increased identity, timeline cyclical beliefs) may benefit more from psychological treatments which use behavioural experiments with objective markers of physical health. Equally, participants may hold a combination of negative beliefs and it thus may be necessary to apply both types of psychological intervention.

There is a clear need for the CS-SRM to explicitly define the mechanisms through which poor emotional health operates to impact on illness cognitions. The potential for poor emotional health to determine diabetes cognitions via attentional control processes and "mood heuristic" rules is represented in Figure 10.3. This revised model should encourage researchers to consider not only the direction of effect but their precise mechanisms of action.

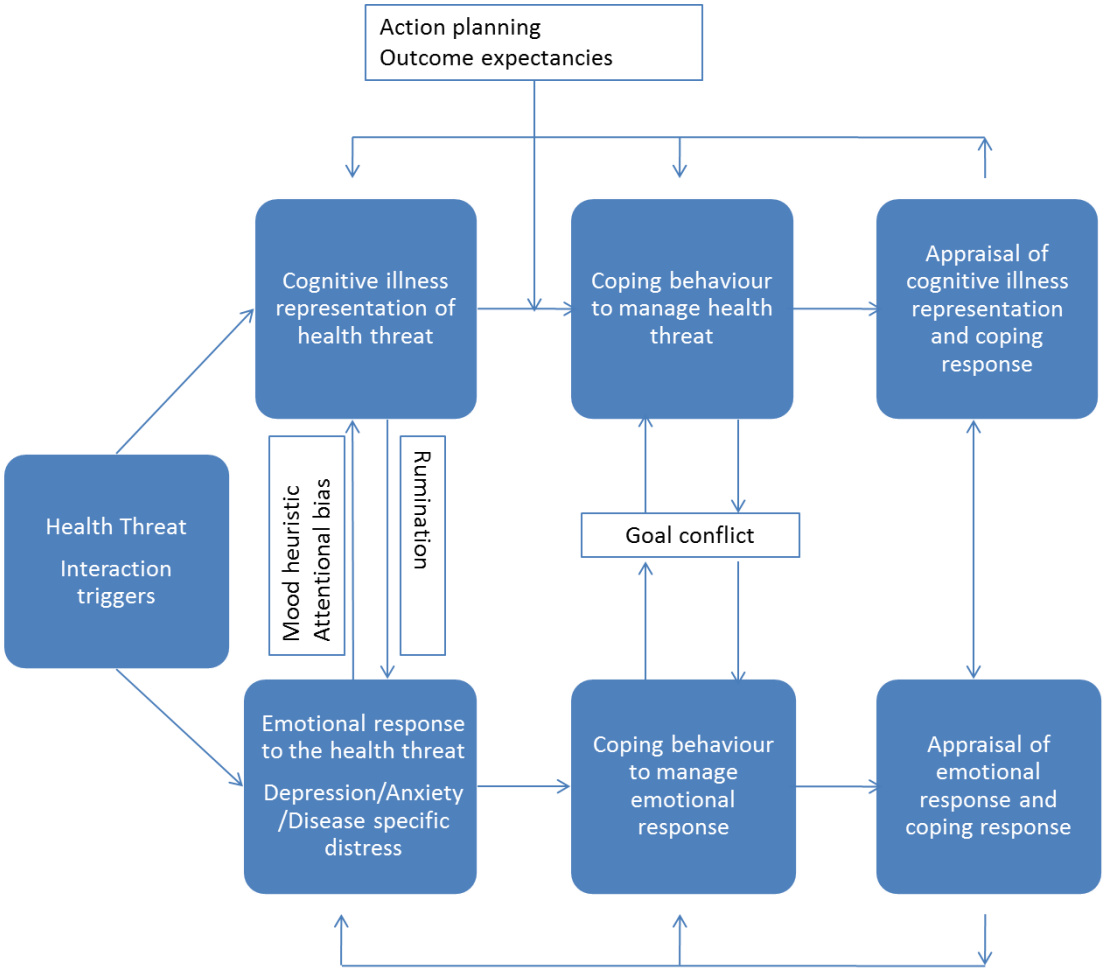
Managing both poor emotional health and diabetes can generate conflicting goals (Detweiler-Bedell et al., 2008). Not addressing conflicting goals when using psychological therapies to manage low/anxious mood in diabetes is a missed opportunity to better integrate the management of multi-morbid conditions. Indeed, current diabetes treatment policy recommendations call for better integration of physical, emotional, and social health care in the management of diabetes (NHS Diabetes, 2013).

Detweiler-Bedell et al (2008) suggests that treatment plans should be developed that allow mutual benefit for both physical and emotional health. For example, behavioural activation strategies can be implemented to schedule pleasant/reinforcing activities (e.g. walking, socialising with friends) and necessary tasks (e.g. medication taking) that allow improvements in diabetes and negative emotions to occur. However socialising with friends can generate challenges because individuals are likely to encounter situations that conflict with their routine diabetes self-management (e.g. eating out, drinking alcohol, being engaged in activities that prevent the administration of medicines). Such goal conflicts can be addressed by problem solving, which is a psychological treatment strategy used in the management of depression (Bell and D'Zurilla, 2009). The therapist can help the patient to identify solutions to managing their conflicting goals. If individuals are exhibiting social anxiety about negotiating the management of their diabetes whilst socialising then role playing may be a useful intervention strategy to apply (Wells, 1997). Role play will allow a person's negative automatic thoughts about disclosing/negotiating

their diabetes self-management to be more accessible to the patient and the therapist to allow them to be addressed.

Goal conflicts are shown in Figure 10.3. The CS-SRM needs to explicitly define goal conflicts to provide a more coherent framework for the management of multi-morbidity. The goal conflicts are likely to be disease specific, thus researchers focussing on particular conditions need to identify what these may be.

Figure 10.3: An elaborated CS-SRM



10.5 *Implications for future work*

Whilst this thesis makes an important contribution to exploring how illness/medication cognitions and poor emotional health operate to impact on diabetes self-care behaviours these findings need to be expanded on.

10.5.1 *Longitudinal studies*

A research priority should be to replicate this longitudinal study in an alternative Type 2 diabetes population. If the findings are replicated it will allow greater confidence that the conclusions drawn from this thesis are not because of sampling or measurement error alone (Kline, 2005). Future longitudinal studies of this kind should strive for measures to be completed on three occasions to allow mediatory pathways to be estimated without bias (Cole and Maxwell, 2003). If possible, objective measures of medication adherence (e.g. pharmacy claims data) should be used to improve the measurement reliability of diabetes self-care behaviours. These objective measures should be supplemented with self-report scales (e.g. the medication adherence report schedule; Horne and Weinman, 2002) to allow intentional non-adherence to be explored. This may be a salient construct among individuals managing multi-morbid conditions.

This thesis has raised the question of when and for whom does an interaction occur between illness cognitions and poor emotional health. Larger scale longitudinal studies with multiple follow-up questionnaires and clinical data extraction will help to identify the most salient clinical "interaction triggers". Studies of this kind will have sufficient statistical power to perform moderation analyses and may identify cognition and emotion profiles specific to certain clinical subgroups, thus leading to more personalised medicine once appropriate interventions are developed. The need for longitudinal studies among individuals with multi-morbidity is echoed by others (Mercer, et al., 2011). The sampling of future longitudinal studies should be stratified to include the recruitment of patients with more severe depression and anxiety to precisely explore the relationships that exist between cognitions, emotions, and diabetes self-care in this population.

Longitudinal studies should also consider nesting a more intensive longitudinal study within its design. This nested study would include fewer participants but these participants would complete measures more frequently for an intense period of time (for example fortnightly for three months, switching to six monthly thereafter to be consistent with the

larger study). Such an approach will allow directional relationships between cognitions, emotions, and diabetes self-care to be more rigorously evaluated. The temporal relationships that exist between these variables are unknown. If the wrong measurement intervals are used then potentially important directional pathways go undetected and erroneous recommendations for clinical intervention are made.

10.5.2 ***Exploring mechanisms of action***

Whilst the directional pathways between diabetes cognitions, poor emotional health, and diabetes self-care were tested in this thesis their precise mechanisms of action are unclear. An understanding of their process of action is needed to identify "key ingredients" for the design of future interventions.

Qualitative interviews

Qualitative studies have explored how individuals cognitively represent multi-morbid conditions (Bower et al., 2012; McSharry et al., 2013) but how illness specific and multi-morbid cognitions are behaviourally responded to is unknown. Key questions raised include:

-What strategies do individuals use to cope with their conflicting self-management goals if perceived to be present?

-Can salient diabetes self-management behaviours be identified that patients perceive to impact on their mood management and vice versa?

-Do situational factors determine the degree of goal conflict? For example can people manage their mood and diabetes well in their home but are challenged when faced with changes in their routine?

Using a qualitative approach will provide a rich account of how individuals behaviourally negotiate the competing demands of managing both their physical and emotional health. It will help to identify: i) key coping behaviours and hypotheses for further testing using quantitative approaches, ii) rationales used by individuals to inform their coping behaviours and the extent to which these are influenced by health care professionals, and iii) coping behaviours used by individuals that are likely to mutually benefit both physical and emotional health.

Experimental approaches and self-report measures of rumination

Experimental psychology approaches can be applied to identify how negative emotional states trigger the activation of negative illness representation schemas. Three mechanisms were described earlier: i) attentional bias, ii) mood congruent recall, and iii) mood heuristics.

The question of whether poor emotional health causes a person's attentional resources to be locked onto mood congruent information can be tested using the Stroop task (Williams, et al., 1996). The Stroop task provides an objective test of whether a person's attentional control processes are biased in the context of negative mood states by measuring colour naming response latencies to emotionally salient words (in this case words associated with diabetes). Such an approach has been applied in the context of the CS-SRM yet not in a depressed or anxious population (Henderson, et al., 2007).

Equally, tests of whether negative mood states cause a person to retrieve mood congruent memories can be examined by comparing non-depressed/anxious and depressed/anxious participants recall of catastrophic illness representation words and emotionally neutral words consistent with the approach used by Teasdale et al. (1988).

Finally, an examination of Leventhal, et al's (1980) mood heuristic hypothesis can be explored by testing whether negative mood states cause a person to wrongly appraise their diabetes control as poor by comparing their appraisal with objective measures of blood glucose control.

In addition specific measures to assess the degree of rumination on illness cognitions need to be developed. Such measures will help to determine whether a person's cognitive response style to their illness representation schema (e.g. engaging in self-focussed ruminative processes) predict poor emotional health and prevent a person from engaging in active self-management coping behaviours. Such a measure would be an adequate complement to the IPQ-R as it will help to identify whether both cognitive content and response styles need to be targeted by psychological treatment interventions to manage negative emotional states in long term conditions.

The outcome of these studies will shape the design of interventions to address poor emotional health to promote adaptive cognitive illness representations. It will determine

whether interventions should include: arousal reduction techniques (e.g. if mood effects attention and/or retrieval processes), prompts to encourage patients to self-monitor their blood glucose to provide objective cues of diabetes control, cognitive re-appraisal, cognitive acceptance based techniques or a combination of these strategies.

10.5.3 *Interventions to improve both physical and emotional health*

Currently, there is no evidence based manualised psychological therapy that has proven efficacy for synergistically managing physical and emotional health in the context of diabetes. This thesis has raised the question of whether psychological treatments other than cognitive reappraisal offer greater utility in managing poor emotional health in the context of a physical long-term condition.

Moss-Morris et al. (2013) developed a tailored CBT treatment manual for individuals with poor emotional adjustment to multiple sclerosis. Elements of the eight session program included both reappraisal and acceptance based cognitive techniques to manage participants distressing illness beliefs in addition to problem solving, relaxation, sleep hygiene, discussion of coping behaviours, and assertiveness training. Participants who received the intervention showed improvements in their levels of distress immediately after the intervention (15 weeks) and at 12 months follow-up. Such a tailored approach is needed for other physical long term conditions.

In diabetes, if poor emotional health disrupts diabetes self-management by affecting attentional control processes (e.g. rumination, self-focussed attention), then mindfulness based interventions may be a highly effective treatment model to apply.

It is equally plausible however for mood to be used as an indicator for physical health status thus influencing a person's cognitive representation of their diabetes and subsequent diabetes self-care behaviours. Such maladaptive management behaviours need to be addressed using behavioural experiments to challenge erroneous diabetes beliefs.

Which of these two psychological treatment models offers the greatest benefit for managing mood in the context of diabetes or indeed whether both elements are required needs to be explored in a three armed comparative effectiveness trial.

However it is insufficient to target negative moods in the context of diabetes without acknowledging the potential for goal conflicts to occur. The question of when these goal

conflicts should be addressed and by whom requires some thought. Because negative mood states can dramatically impair functioning and appraisal a person's emotional state needs to be addressed first before integrating the management of diabetes with emotional health.

The eight session mindfulness based cognitive therapy program (Segal et al., 2002) offers the opportunity for goal conflict management to occur in session seven when behavioural activation techniques are introduced. Activities can be scheduled that are mutually beneficial to both physical and emotional health. Because mindfulness is typically delivered in a group setting novel mutually beneficial activities may be identified by the group. A diabetes health care professional could attend session seven to help the therapist and the group problem solve conflicting diabetes and poor emotional health self-management goals.

Because behavioural experiments target diabetes cognitions directly with objective evidence there is less likelihood of generating conflicting diabetes self-management and poor emotional health treatment goals. However, regular self-monitoring of blood glucose has the potential to cause a person to feel more depressed and/or anxious about their diabetes (O'Kane, et al., 2008; Peel, et al., 2007). If participants are provided with a clear rationale for monitoring their blood glucose and their outcome expectancies are sufficiently managed then the likelihood of experiencing negative emotional states will arguably be decreased. A diabetes specialist professional trained in the principals of behavioural experiments is likely best placed to deliver this type of intervention. This professional group have the clinical expertise to manage their patient's blood glucose control outcome expectancies and to educate them on the variety of factors that can determine health outcomes other than behavioural adherence including co-morbid conditions, diabetes duration, and adequacy of medication regimens (Johnson, 1992).

10.6 *Conclusion*

Diabetes self-management occurs independently from poor emotional health among people who attend for their diabetes outpatient appointments. Personal control and medication necessity beliefs are modifiable predictors of either diabetes self-management or glycaemic control, but confidence alone is insufficient to maintain adherence to diabetes self-care behaviours.

Whilst poor emotional health did not impair diabetes self-management, it had a directional effect on specific diabetes cognitions. If more severe negative mood states are experienced it seems plausible for poor emotional health to impair diabetes self-management via diabetes cognitions.

This thesis provided an advancement on the traditional methods used to explore relationships between the CS-SRM concepts. Given that: i) health psychology concepts are susceptible to measurement error, ii) multiple directional pathways are outlined by CS-SRM, and iii) a person's illness experience is not static, future studies should consider the application of longitudinal structural equation modelling to allow theorised relationships and mechanisms of change to be rigorously evaluated. This thesis is the first to use such an approach in adults with diabetes mellitus and it is not without its limitations (see earlier). However the approaches used and lessons learned provide a platform to shape the design of future studies who choose to adopt this methodological and statistical design to develop and test their hypothesised theoretical models.

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12 *Appendices*

12.1 *Methodological Appendix A*

Systematic review search strategy

Concept One: Diabetes mellitus	Concept Two: Emotional States	Concept Three: Illness cognitions
Medline and CINAHL		
Exp Diabetes Mellitus/ Diabet$.mp, Exp Diabetes Mellitus, Type 1/ “insulin dependent diabetes mellitus”.mp. “diabetes mellitus type I”.mp T1DM.mp IDDM.mp DM1.mp Exp Diabetes Mellitus, Type 2/ “non-insulin dependent diabetes mellitus”.mp “noninsulin dependent diabetes mellitus”.mp “diabetes mellitus type II”.mp T2DM.mp NIDDM.mp DM2.mp “late onset diabetes”.mp “maturity onset diabetes”.mp “mature onset diabetes”.mp Exp Diabetes complications/ Exp Hyperglycemia/ or hyperglycaemia.mp exp Hypoglycemia/ or hypoglycaemia.mp exp Diabetic Ketoacidosis/	exp Mood Disorders/ exp Depression/ exp Depressive Disorder/ depress$.mp “low mood”.mp “negative affect”.mp “Atypical depression”.mp “Endogenous depression”.mp “Reactive depression” “Major depression”.mp “Long term depression”.mp “Affective disorder”.mp “Mixed anxiety and depression”.mp exp Anxiety/ exp Anxiety Disorders/ “Generalised anxiety disorder”.mp “Social anxiety”.mp “Social phobia”.mp exp Adjustment Disorders/ “adjustment disorder”.mp “emotional adjustment”.mp “psychosocial adjustment”.mp “psychological adjustment”.mp Adjustment.mp exp Stress, Psychological/ Distress.mp “emotional distress”.mp “Chronic stress”.mp “Diathesis stress model”.mp “Acute stress”.mp “Acute stress disorder”.mp “Critical incident stress”.mp “Mental stress”.mp exp Adaptation Psychological/ exp Affect/	Exp attitude to health/ Exp Health knowledge, attitudes, practice/ “Illness representation model”.mp “Common sense model”.mp CSM.mp “Self-regulatory model”.mp “Parallel process model”.mp “Leventhal’s model”.mp Leventhal.mp “Theory of self-regulation”.mp “Parallel processing model”.mp “Self-regulation model”.mp “Lay representation”.mp “Lay model”.mp “Illness cognition$”.mp “Illness belief$”.mp “Illness perception$”.mp “Illness representation$”.mp “Health belief$”.mp “Health cognition$”.mp “Health perception$”.mp “Health representation$”.mp “Personal model$”.mp Self-regulation.mp “Health belief model”.mp HBM.mp Exp self-efficacy IPQ.mp IPQ-R.mp “Personal model$ of diabete$”.mp “Personal model$ of illness”.mp “Diabetes illness representations questionnaire”.mp DIRQ.mp

Concept One: Diabetes mellitus	Concept Two: Emotional States	Concept Three: Illness cognitions
	exp emotions/ emotion$.mp “emotional state$”.mp “Emotional responses”.mp exp anger/ well-being.mp well-being.mp, “Well-being”.mp “positive affect”.mp psyohosocial.mp “patient health questionnaire”.mp PHQ-9.mp “Center for epidemiologic studies depression scale”.mp CES-D.mp “Problem areas in diabetes scale”.mp PAID.mp “Well-being questionnaire”.mp “Well-being questionnaire”.mp “Hospital anxiety and depression scale”.mp HADS.mp “Hamilton rating scale for depression”.mp HRSD.mp “Beck depression inventory”.mp BDI.mp ATT39.mp “Questionnaire on stress in patients with diabetes-revised”.mp QSD-R.mp “Diabetes distress scale”.mp DDS.mp “Positive and negative affect schedule”.mp PANAS.mp “Penn state worry questionnaire”.mp “Zung’s self-rating depression scale”.mp “Depression inventory”.mp “Self-rating depression scale”.mp	“Illness perception questionnaire”.mp “Illness perception questionnaire-revised”.mp “The brief illness perception questionnaire”.mp “Brief IPQ”.mp “The revised illness perception questionnaire”.mp

Concept One: Diabetes mellitus	Concept Two: Emotional States	Concept Three: Illness cognitions
Psychinfo		
Concept 1-Diabetes Mellitus	*Concept 2- Emotional states*	Exp Health attitudes/
Exp Diabetes Mellitus/	Exp affective disorders/	“Attitude to health”.mp
Exp Diabetes/	Exp atypical depression/	Exp physical illness
Diabetic$.mp	Exp Endogenous	(attitudes towards)/
“Diabetes mellitus type	depression/	“Illness representation
I”.mp	Exp reactive depression/	model”.mp
“Diabetes mellitus type	Exp Depression (emotion)/	“Common sense model”.mp
1”.mp	Exp Major depression/	CSM.mp
“Insulin dependent diabetes	“Mood disorders”.mp	“Parallel process model”.mp
mellitus”.mp	Depression.mp	“Parallel processing
IDDM	“Depressive disorder”.mp	model”.mp
DM1	Depress$.mp	“Leventhal’s model”.mp
T1DM	“Low mood”.mp	Leventhal.mp
“Diabetes mellitus type	“Negative affect”.mp	“Lay representation”.mp
II”.mp	“Long term depression”.mp	“Lay model”.mp
“Diabetes mellitus type	“Mixed anxiety and	Exp self-regulation.mp
2”.mp	depression”.mp	“Self-regulatory model”.mp
“Noninsulin dependent	Exp Anxiety/	“Theory of self-
diabetes mellitus”.mp	Exp Anxiety disorders/	regulation”.mp
“Non-insulin dependent	Exp Generalised anxiety/	“Illness cognition$”.mp
diabetes mellitus”.mp	disorder/	“Illness belief$”.mp
NIDDM	Exp Social anxiety/	“Illness perception$”.mp
T2DM	“Social phobia”.mp	“Illness representation$”.mp
DM2	Exp Stress/	“Health belief$”.mp
“Late onset diabetes”.mp	Exp Psychological stress/	“Health cognition$”.mp
“Maturity onset	Exp chronic stress/	“Health perception$”.mp
diabetes”.mp	Exp Diathesis stress model/	“Health representation$”.mp
“Diabetes	“Acute stress”.mp	Exp Health attitudes/
complications”.mp	“Acute stress disorder”.mp	“Personal model$”.mp
“Diabetic ketoacidosis”.mp	“Critical incident stress”.mp	IPQ.mp
Exp hyperglycemia/ or	“Mental stress”.mp	IPQ-R.mp
hyperglycaemia.mp	Exp Adjustment disorders/	“Personal model$ of
Exp hypoglycemia/ or	Exp emotional adjustment/	diabete$”.mp
hypoglycaemia	“Psychological	“Personal model$ of
	adjustment”.mp	illness”.mp
	“Psychosocial	DIRQ.mp
	adjustment”.mp	“Illness perception
	Exp adjustment/	questionnaire”.mp
	Exp emotional states/	“Illness perception
	Exp emotions/	questionnaire-revised”.mp
	Emotion$.mp	“The revised illness
	Exp emotional responses/	perception
	Affect.mp	questionnaire”.mp
	Emotion.mp	“The brief illness perception
	Exp anger/	questionnaire”.mp
	“Positive affect”.mp	“Brief IPQ”.mp
	Exp well being	“Health belief model”.mp

Concept One: Diabetes mellitus	Concept Two: Emotional States	Concept Three: Illness cognitions
	Well-being.mp Well-being.mp Exp distress "Emotional distress".mp Psychosocial.mp "patient health questionnaire".mp PHQ-9.mp "Center for epidemiologic studies depression scale".mp CES-D.mp "Problem areas in diabetes scale".mp PAID.mp "Well-being questionnaire".mp Well-being questionnaire.mp "Hospital anxiety and depression scale".mp HADS.mp "Hamilton rating scale for depression".mp HRSD.mp Exp beck depression inventory/ BDI.mp Exp Zung's self-rating depression scale/ ATT39 "Questionnaire on stress in patients with diabetes-revised".mp QSD-R.mp "Positive and negative affect schedule".mp PANAS.mp "Penn State worry questionnaire".mp PSWQ.mp "Depression inventory".mp "Self-rating depression scale".mp "Diabetes distress scale".mp DDS.mp	HBM.mp "Social cognitive theory".mp Exp self-efficacy
EMBASE		
Exp Diabetes Mellitus/ Diabet$.mp	Exp mood disorder/ Exp atypical depression/	Exp attitude to health/ Exp health belief

Concept One: Diabetes mellitus	Concept Two: Emotional States	Concept Three: Illness cognitions
Exp insulin dependent diabetes mellitus/	Exp endogenous depression/	"Self-regulation".mp
"Diabetes mellitus type I".mp	Exp reactive depression/	"Illness representation model".mp
"Diabetes mellitus type 1".mp	Exp major depression/	"Common sense model".mp
IDDM	Exp depression/	CSM.mp
T1DM	exp long term depression/	"Self-regulatory model".mp
DM1	"Depressive disorder".mp	Exp self-regulation model/
Exp non insulin dependent diabetes mellitus/	Depress$.mp	"Parallel process model".mp
"Noninsulin dependent diabetes mellitus".mp	"Low mood".mp	"Parallel processing model".mp
"Diabetes mellitus type II".mp	"Negative affect".mp	"Leventhal's model".mp
"Diabetes mellitus type 2".mp	Exp mixed anxiety and depression/	Leventhal.mp,au
NIDDM	Exp anxiety/	"Lay representation".mp
T2DM	Exp anxiety disorder/	"Lay model".mp
DM2	Exp generalised anxiety disorder/	"Illness cognition$".mp
Exp maturity onset diabetes/	Exp social phobia/	"Illness belief$".mp
"Mature onset diabetes".mp	"Social anxiety".mp	"Illness perception$".mp
"Late onset diabetes".mp	Exp adjustment disorder/	"Illness representation$".mp
Hyperglycaemia.mp	"Psychological adjustment".mp	"Health belief$".mp
Hypoglycaemia.mp	"Psychosocial adjustment".mp	"Health attitudes$".mp
Exp hyperglycemia/	"Emotional adjustment".mp	"Health cognition$".mp
Exp hypoglycemia/	"Adaptation psychological".mp	"Health perception$".mp
"Diabetes complications".mp	Exp stress/	"Health representation$".mp
Exp Diabetic ketoacidosis/	Exp chronic stress	"Personal model$".mp
	Exp acute stress	Exp health belief model/
	Exp acute stress disorder	HBM.mp
	Exp critical incident stress	"Social cognitive theory".mp
	Exp "Lazarus theory of stress and coping"	"Self-efficacy".mp
	Exp mental stress	IPQ.mp
	"Psychological stress".mp	IPQ-R.mp
	"Diathesis stress model".mp	"Personal model$ of diabete$".mp
	Distress.mp	"Personal model$ of illness".mp
	Exp adjustment disorder/	"Diabetes illness representation questionnaire".mp
	Exp adjustment	DIRQ.mp
	Exp emotion/	"Theory of self-regulation".mp
	"Emotional state$".mp	"Illness perception questionnaire".mp
	Emotion$.mp	"Illness perception questionnaire revised".mp
	"Emotional responses".mp	"The revised illness representation questionnaire".mp
	Exp anger/	"The brief illness perception questionnaire".mp
	Exp well-being/	
	Well-being.mp	
	exp affect/	
	"Affective disorders".mp	
	"Positive affect".mp	
	"Emotional distress".mp	
	Psychosocial.mp	

Concept One: Diabetes mellitus	Concept Two: Emotional States	Concept Three: Illness cognitions
	exp patient health questionnaire/ PHQ-9.mp exp Center for Epidemiological Studies Depression Scale/ CES.D.mp “Problem areas in diabetes scale”.mp PAID.mp Exp beck depression inventory/ BDI.mp Exp depression inventory/ exp self-rating depression scale/ “Well-being questionnaire”.mp “Well-being questionnaire”.mp exp Hamilton scale Hamilton rating scale for depression/ HRSD.mp ATT39.mp “Questionnaire on stress in patients with diabetes-revised”.mp QSD-R.mp “Diabetes distress scale”.mp DDS.mp “Positive and negative affect schedule”.mp PANAS.mp “Penn State worry questionnaire”.mp PSWQ.mp exp "Hospital Anxiety and Depression Scale"/ HADS.mp	“Brief-IPQ”.mp

12.2 *Methodological Appendix B*

Questionnaire Pack

MANCHESTER 1824
The University of Manchester

NHS
National Institute for Health Research
Collaboration for Leadership in Applied Health Research and Care (CLAHRC) for Greater Manchester

Diabetes mellitus and emotional distress

- Please follow the instructions over the page.
- Sometimes you may be asked to answer the same question but phrased in a slightly different way. This allows us to get a better picture of the way people cope with diabetes.
- If you have any questions or queries please contact Jo (the researcher) on 0161 306 7045. Leave a message on the Ansaphone if there is no reply.
- Thank you for taking the time to complete the questionnaire

Participant Identification Number:
Please leave blank office use only

☐☐☐☐

Version 2: Dated 09/05/11

Section A: Background information about yourself

A1 How old are you? ----------

A2 Are you? (Please tick) Male ☐ Female ☐

A3 How would you describe your ethnicity? (Please tick)

White British ☐

White Irish ☐

Other White Background (Please specify below) ☐

Black or Black British-Caribbean ☐

Black or Black British-African ☐

Other Black Background (Please specify below) ☐

Asian or Asian British-Indian ☐

Asian or Asian British -Pakistani ☐

Asian or Asian British Bangladeshi ☐

Other Asian Background (Please specify) ☐

Mixed- White & Black-Caribbean ☐

Mixed- White & Black-African ☐

Mixed-White & Asian ☐

Mixed- Other mixed background (Please specify) ☐

Chinese ☐

Other Ethnic Background (Please specify) ☐

Prefer not to say ☐

Section B

This section of the questionnaire is about your feelings and emotions

Please circle a number on each of the following statements to show how often you feel each statement has applied to you in the past few weeks:

B1 I feel that I am useful and needed

3	2	1	0
All the time			Not at all

B2 I have crying spells or feel like it

3	2	1	0
All the time			Not at all

B3 I find I can think quite clearly

3	2	1	0
All the time			Not at all

B4 My life is pretty full

3	2	1	0
All the time			Not at all

B5 I feel downhearted and blue

3	2	1	0
All the time			Not at all

B6 I enjoy the things I do

3	2	1	0
All the time			Not at all

B7 I feel nervous and anxious

3	2	1	0
All the time			Not at all

B8 I feel afraid for no reason at all

3	2	1	0
All the time			Not at all

B9 I get upset easily or feel panicky

3	2	1	0
All the time			Not at all

B10 I feel like I'm falling apart and going to pieces

All the time

Not at all

B11 I feel calm and can sit still easily

All the time

Not at all

B12 I fall asleep easily and get a good night's rest

All the time

Not at all

B13 I feel energetic, active or vigorous

All the time

Not at all

B14 I feel dull or sluggish

All the time

Not at all

B15 I feel tired, worn out, used up or exhausted

B16 I have been waking up feeling fresh and rested

B17 I have been happy, satisfied or pleased with my personal life

B18 I have felt well adjusted to my life situation

B19 I have lived the kind of life I wanted to

B20 I have felt eager to tackle my daily tasks or make new decisions

3	2	1	0
All the time			Not at all

B21 I have felt I could easily handle or cope with any serious problem or major change in my life

3	2	1	0
All the time			Not at all

B22 My daily life has been full of things that were interesting to me

3	2	1	0
All the time			Not at all

Please turn over for the next set of questions.

Section C

This section of the questionnaire is designed to measure the thoughts you have about having type II diabetes

Listed below are a number of symptoms that you may or may not have experienced since your diabetes. Please indicate by circling yes or no, whether you have experienced any of these symptoms since your diabetes, and whether you believe these symptoms are related to your diabetes.

	I have experienced this symptom *since my diabetes*		This symptom *is related to my diabetes*	
Pain	Yes	No	Yes	No
Sore Throat	Yes	No	Yes	No
Nausea	Yes	No	Yes	No
Breathlessness	Yes	No	Yes	No
Weight Loss	Yes	No	Yes	No
Fatigue	Yes	No	Yes	No
Stiff Joints	Yes	No	Yes	No
Sore Eyes	Yes	No	Yes	No
Wheeziness	Yes	No	Yes	No
Headaches	Yes	No	Yes	No
Upset Stomach	Yes	No	Yes	No
Sleep Difficulties	Yes	No	Yes	No
Dizziness	Yes	No	Yes	No
Loss of Strength	Yes	No	Yes	No

We are interested in your own personal views about how you see your current diabetes. Please indicate how much you agree or disagree with the following statements about your diabetes by ticking the appropriate box

	Views about your diabetes	Strongly Disagree	Disagree	Neither agree nor disagree	Agree	Strongly Agree
IP1	My diabetes will last a short time					
IP2	My diabetes is likely to be permanent rather than temporary					
IP3	My diabetes will last for a long time					
IP4	This diabetes will pass quickly					
IP5	I expect to have this diabetes for the rest of my life					
IP6	My diabetes is a serious condition					
IP7	My diabetes has major consequences on my life					
IP8	My diabetes does not have much effect on my life					
IP9	My diabetes strongly affects the way others see me					
IP10	My diabetes has serious financial consequences					
IP11	My diabetes causes difficulties for those who are close to me					
IP12	There is a lot which I can do to control my symptoms					

Please indicate how much you agree or disagree with the following statements about your diabetes by ticking the appropriate box

	Views about your diabetes	Strongly Disagree	Disagree	Neither agree nor disagree	Agree	Strongly Agree
1P 13	What I do can determine whether my diabetes gets better or worse					
1P 14	The course of my diabetes depends on me					
1P 15	Nothing I do will affect my diabetes					
1P 16	I have the power to influence my diabetes					
1P 17	My actions will have no affect on the outcome of my diabetes					
1P 18	My diabetes will improve in time					
IP 19	There is very little that can be done to improve my diabetes					
IP 20	My treatment will be effective in curing my diabetes					
IP 21	The negative effects of my diabetes can be prevented (avoided) by my treatment					
IP 22	My treatment can control my diabetes					
IP 23	There is nothing which can help my condition					

Please indicate how much you agree or disagree with the following statements about your diabetes by ticking the appropriate box

	Views about your diabetes	Strongly Disagree	Disagree	Neither agree nor disagree	Agree	Strongly Agree
IP 24	The symptoms of my condition are puzzling to me					
IP 25	My diabetes is a mystery to me					
IP 26	I don't understand my diabetes					
IP 27	My diabetes doesn't make any sense to me					
IP 28	I have a clear picture or understanding of my condition					
IP 29	The symptoms of my diabetes change a great deal from day to day					
IP 30	My symptoms come and go in cycles					
IP 31	My diabetes is very unpredictable					
IP 32	I go through cycles in which my diabetes gets better and worse					
IP 33	I get depressed when I think about my diabetes					
IP 34	When I think about my diabetes I get upset					
IP 35	My diabetes makes me feel angry					

Please indicate how much you agree or disagree with the following statements about your diabetes by ticking the appropriate box

	Views about your diabetes	Strongly Disagree	Disagree	Neither agree nor disagree	Agree	Strongly Agree
IP 36	My diabetes does not worry me					
IP 37	Having this diabetes makes me feel anxious					
IP 38	My diabetes makes me feel afraid					

In the next section we are interested in what you consider may have been the cause of your diabetes. As people are very different there is no correct answer for this question. We are mostly interested in your views about the factors that caused your diabetes rather than what others including doctors or family may have suggested to you.

On the next page is a list of possible causes for your diabetes. Please indicate how much you agree or disagree that they were causes for you by ticking the appropriate box.

Please turn over for the next section of the questionnaire.

Please indicate how much you agree or disagree that the following statements were causes for your diabetes by ticking the appropriate box.

	Possible causes	Strongly Disagree	Disagree	Neither agree nor disagree	Agree	Strongly Agree
C1	Stress or worry					
C2	Hereditary-it runs in my family					
C3	A germ or virus					
C4	Diet or eating habits					
C5	Chance or bad luck					
C6	Poor medical care in my past					
C7	Pollution in the environment					
C8	My own behaviour					
C9	My mental attitude e.g. thinking about life negatively					
C10	Family problems or worries					
C11	Overwork					
C12	My emotional state e.g. feeling down, lonely, anxious, empty					
C13	Ageing					
C14	Alcohol					
C15	Smoking					
C16	Accident or injury					
C17	My personality					
C18	Altered immunity					

Please list in rank-order the three most important factors that you now believe caused YOUR diabetes. You may use any of the items from the box above, or you may have additional ideas of your own. **The most important causes for me are:**

1. _____

2. _____

3. _____

Section D

We would like to ask you about your personal views about medicines prescribed for you for your diabetes. These are statements other people have made about their medicines. Please indicate the extent to which you agree or disagree with them by ticking the appropriate box. There are no right or wrong answers. We are interested in your personal views. If you are not prescribed any medicines for your diabetes please move on to section E.

Statements about medicines	Strongly Disagree	Disagree	Uncertain	Agree	Strongly Agree
My health at present, depends on my medicines					
Having to take medicines worries me					
My life would be impossible without my medicines					
Without my medicines I would be very ill					
I sometimes worry about long-term effects of my medicines					
My medicines are a mystery to me					
My health in the future will depend on my medicines					
My medicines disrupt my life					
I sometimes worry about becoming too dependent on my medicines					
My medicines protect me from becoming worse					

Section E

This section of the questionnaire is designed to measure your diabetes self-care activities during the past 7 days. If you were sick/ill during the past 7 days, please think back to the last 7 days that you were not sick/ill. Please answer the questions as honestly and accurately as you can. Your medical team will not have access to your responses to the questionnaire. Please tick the response most appropriate to you

DIET

D1 How many of the last SEVEN DAYS have you followed a healthful eating plan?

0	1	2	3	4	5	6	7

D2 On average, over the past month, how many DAYS PER WEEK have you followed your eating plan?

0	1	2	3	4	5	6	7

D3 On how many of the last SEVEN DAYS did you eat five or more servings of fruits and vegetables?

0	1	2	3	4	5	6	7

D4 On how many of the last SEVEN DAYS did you eat high fat foods such as red meat or full-fat dairy products?

0	1	2	3	4	5	6	7

EXERCISE

D5 On how many of the last SEVEN DAYS did you participate in at least 30 minutes of physical activity? (Total minutes of continuous activity, including walking).

0	1	2	3	4	5	6	7

D6 On how many of the last SEVEN DAYS did you participate in a specific exercise session (such as swimming, walking, biking) other than what you do around the house or as part of your work?

0	1	2	3	4	5	6	7

BLOOD SUGAR TESTING

D7 On how many of the last SEVEN DAYS did you test your blood sugar?

0	1	2	3	4	5	6	7

D8 On how many of the last SEVEN DAYS did you test your blood sugar the number of times recommended by your health care provider?

0	1	2	3	4	5	6	7

FOOT CARE

D9 On how many of the last SEVEN DAYS did you check your feet?

0	1	2	3	4	5	6	7

D10 On how many of the last SEVEN DAYS did you inspect the inside of your shoes?

0	1	2	3	4	5	6	7

SMOKING

D11 Have you smoked a cigarette-even one puff-during the past SEVEN DAYS?

No	Yes

MEDICATIONS

D12 On how many of the last SEVEN DAYS, did you take your recommended diabetes medication?

0	1	2	3	4	5	6	7

Section F

This final section of the questionnaire is about your physical health. Please tick the response that applies to you

E1 **How many years have you had diabetes?**_____

E2 **Have you ever been told by your doctor that you have a problem with your heart or circulatory system (e.g. arteries and veins)?**

No	Yes

If YES please tick if any of the following applies to you, if NO please go to question E2

	Please tick if applicable
I have had a heart attack	
I have angina	
I have heart failure	
I have peripheral arterial disease (narrowing of the arteries)	
I have had a stroke	
I have had a transient ischemic attack (TIA)	

If you have any other problems with your heart or circulatory system which are not listed above please report them in the space below

_____

_____

_____

E3 **Have you ever been told by your doctor that you have a problem with your kidneys?**

No	Yes

If YES please tick if any of the following applies to you, if NO please go to question E3

	Please tick if applicable
I am on dialysis	
I have received a kidney transplant	

Please report any other problems you may have with your kidneys in the space below if necessary

E4 **Have you ever been told by your doctor that you have a problem with the back of your eye (retina) due to diabetes?**

No	Yes

E5 **Have you ever been told by your doctor that you have a problem with your feet due to diabetes?**

No	Yes

If YES please tick if any of the following applies to you, if NO please go to question E5

	Please tick if applicable
I currently have a foot ulcer	
I have had a foot ulcer in the past	
I experience pain, numbness, tingling in my feet	
I have had an amputation as a result of my foot problems	

If you have any other problems with your feet due to diabetes which are not listed above please report them in the space below

E6 **Please use the box below to list any medications you are currently taking**

That is the end of the questionnaire. Thank you for taking the time to complete it.

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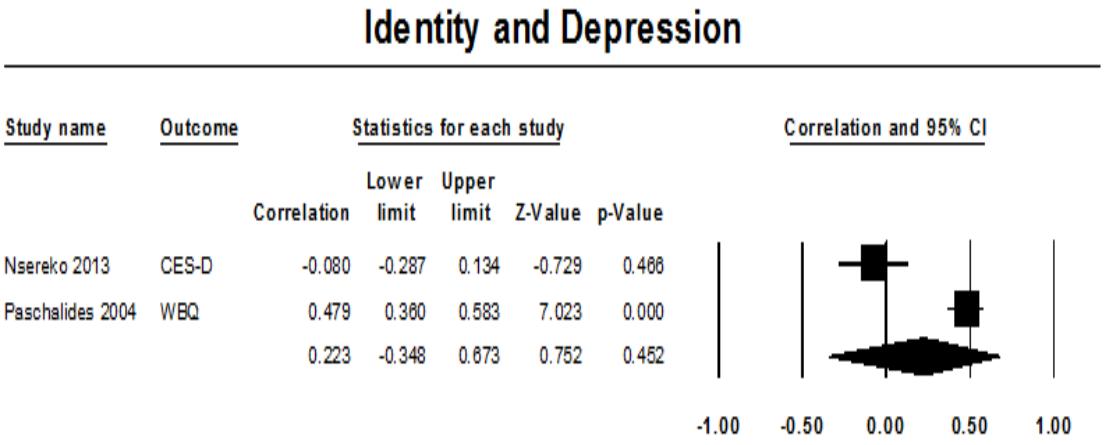
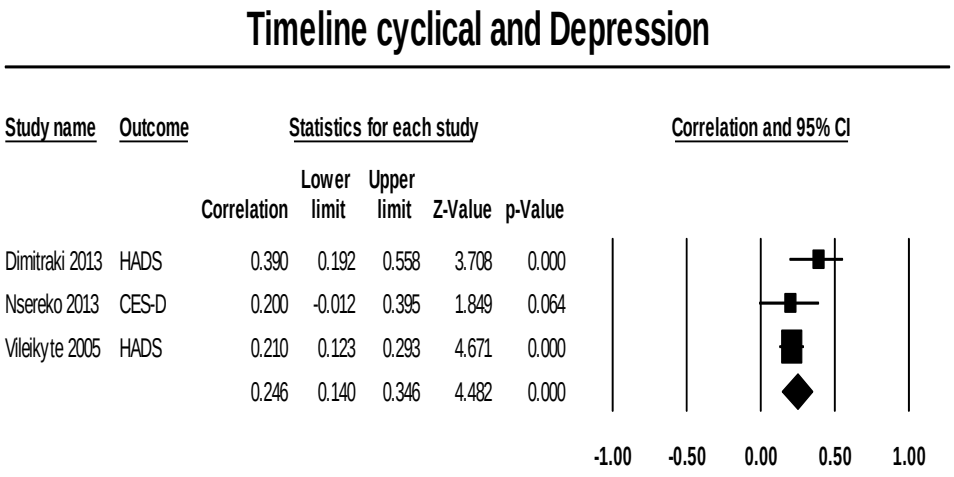
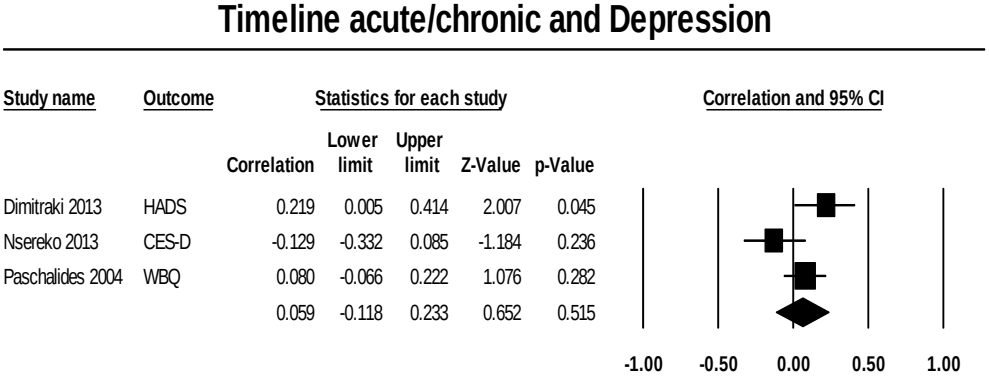
12.3 *Statistical Appendix A*

12.3.1 *Forests plots: Depression and illness cogntions*

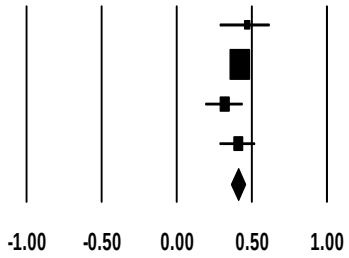






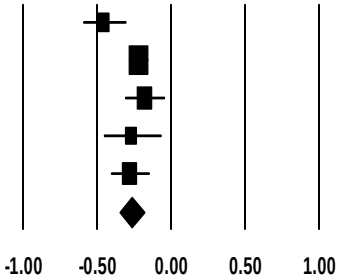
Consequences and Depression

Study name	Outcome	Correlation	Lower limit	Upper limit	Z-Value	p-Value
Dimitraki 2013	HADS	0.470	0.284	0.622	4.590	0.000
Khunti 2008	HADS	0.420	0.360	0.477	12.342	0.000
Luyckx 2010	CES-D	0.320	0.188	0.441	4.588	0.000
Paschalides 2004	WBQ	0.410	0.282	0.524	5.866	0.000
		0.407	0.359	0.453	15.042	0.000



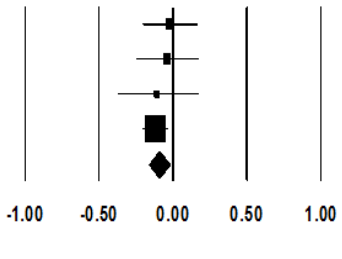
Personal Control and Depression

Study name	Outcome	Correlation	Lower limit	Upper limit	Z-Value	p-Value
Hampson 2000	CES-D	-0.460	-0.595	-0.299	-5.165	0.000
Khunti 2008	HADS	-0.219	-0.286	-0.151	-6.144	0.000
Luyckx 2010	CES-D	-0.179	-0.312	-0.039	-2.501	0.012
Nsereko 2013	WBQ	-0.270	-0.456	-0.062	-2.524	0.012
Paschalides 2004	WBQ	-0.280	-0.408	-0.141	-3.875	0.000
		-0.268	-0.350	-0.181	-5.910	0.000



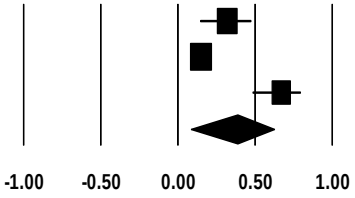
Treatment control and Depression

Study name	Outcome	Correlation	Lower limit	Upper limit	Z-Value	p-Value
Hampson 2000	CES-D	-0.020	-0.206	0.167	-0.208	0.835
Nsereko 2013	CES-D	-0.040	-0.250	0.173	-0.364	0.716
Ponzo 2006	CES-D	-0.110	-0.376	0.174	-0.754	0.451
Vileikyte 2005	HADS	-0.119	-0.206	-0.031	-2.632	0.008
		-0.095	-0.167	-0.022	-2.550	0.011



Seriousness and Depression

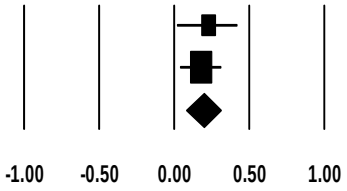
Study name	Outcome	Correlation	Lower limit	Upper limit	Z-Value	p-Value
Hampson 2000	CES-D	0.319	0.141	0.477	3.440	0.001
Khunti 2008	HADS	0.150	0.080	0.219	4.160	0.000
Ponzo 2006	CES-D	0.670	0.482	0.799	5.560	0.000
		0.384	0.081	0.621	2.456	0.014



12.3.2 ***Forest plots: Anxiety and illness cognitions***

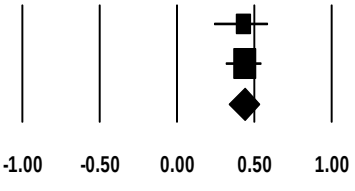
Timeline acute/chronic and Anxiety

Study name	Outcome	Correlation	Lower limit	Upper limit	Z-Value	p-Value
Dimitraki 2013	HADS	0.230	0.016	0.423	2.106	0.035
Paschalides 2004	WBQ	0.180	0.036	0.316	2.449	0.014
		0.196	0.077	0.309	3.206	0.001



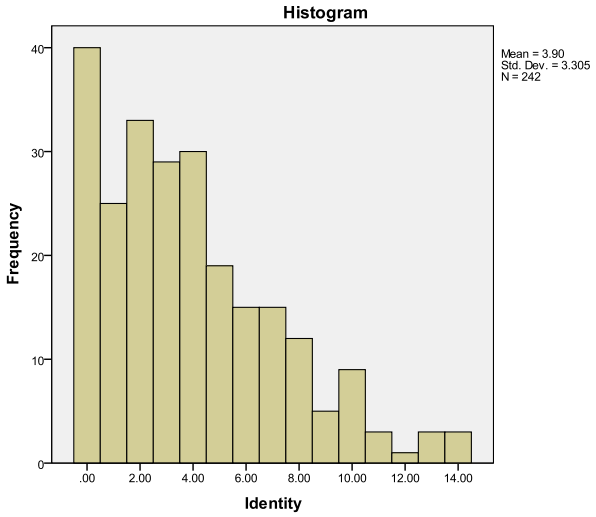
Consequences and Anxiety

Study name	Outcome	Correlation	Lower limit	Upper limit	Z-Value	p-Value
Dimitraki 2013	HADS	0.430	0.238	0.590	4.140	0.000
Paschalides 2004	WBQ	0.438	0.313	0.548	6.323	0.000
		0.436	0.333	0.528	7.558	0.000



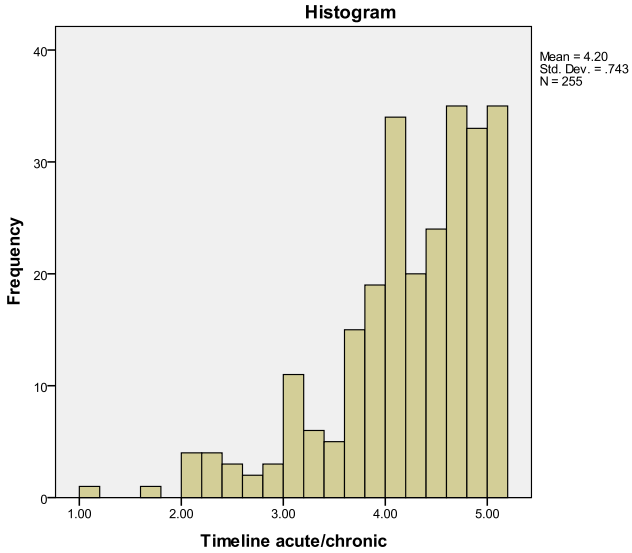
12.3.3 ***Tests of normality***

Baseline identity



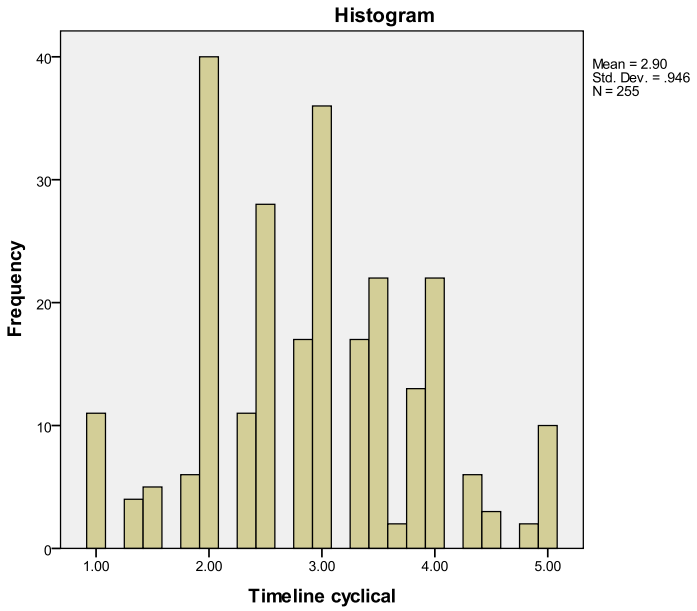
Statistical test	Test statistic	Standard error/significance
Skew	.908	.156
Kurtosis	.383	.312
Kolmogorov-Smirnov	.137	.000

Baseline timeline acute/chronic



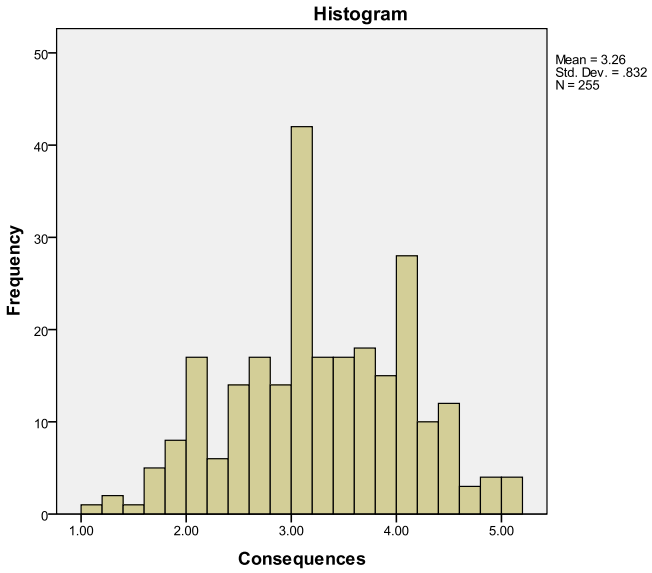
Statistical test	Test statistic	Standard error/significance
Skew	-1.294	.153
Kurtosis	1.682	.304
Kolmogorov-Smirnov	.152	.000

Baseline timeline cyclical



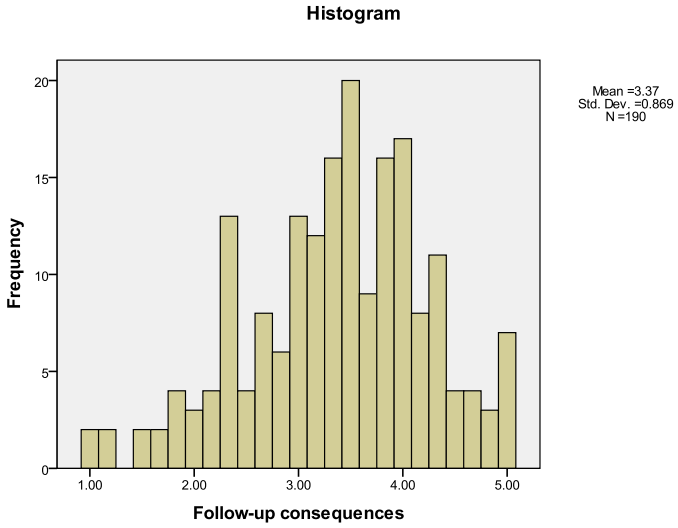
Statistical test	Test statistic	Standard error/significance
Skew	.135	.153
Kurtosis	-.357	.304
Kolmogorov-Smirnov	.087	.000

Baseline consequences



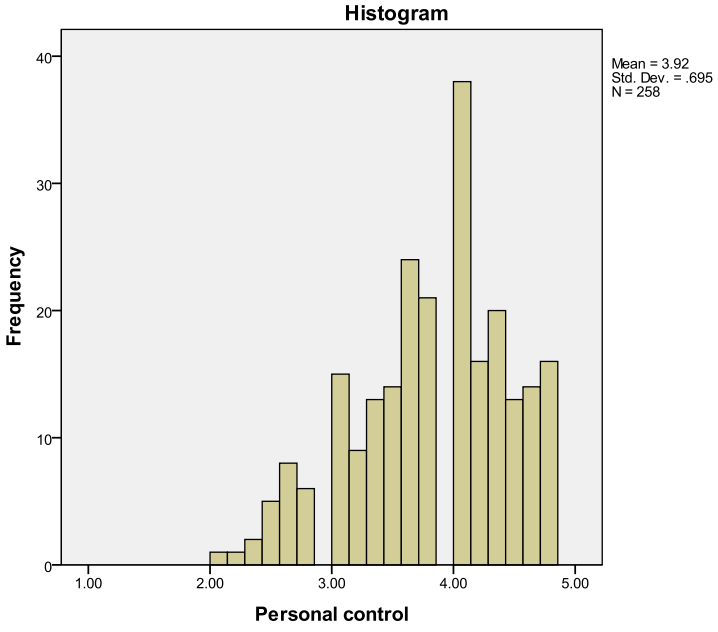
Statistical test	Test statistic	Standard error/significance
Skew	-.134	.153
Kurtosis	-.497	.304
Kolmogorov-Smirnov	.052	.086

Follow-up consequences



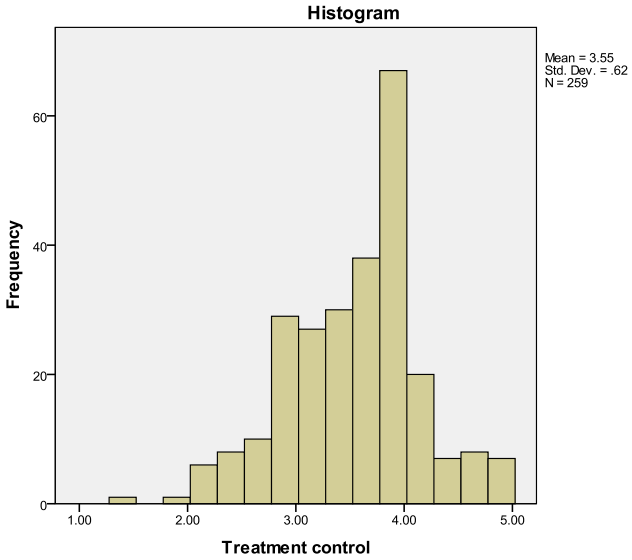
Statistical test	Test statistic	Standard error/significance
Skew	-.406	.176
Kurtosis	-.078	.351
Kolmogorov-Smirnov	.086	.002

Baseline personal control



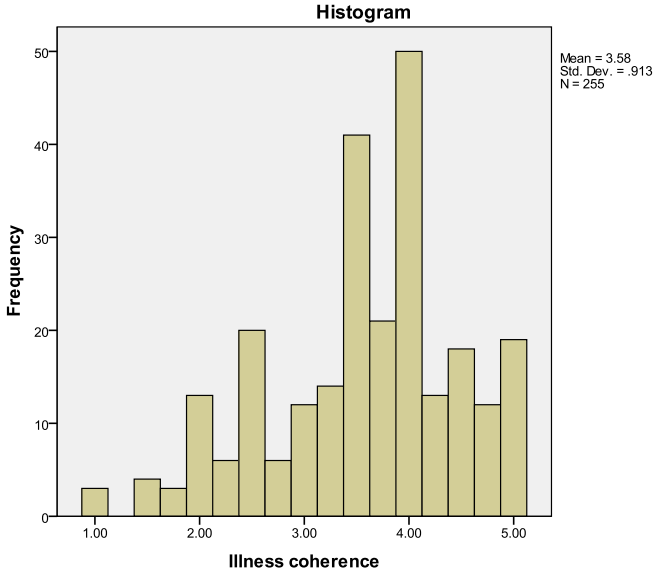
Statistical test	Test statistic	Standard error/significance
Skew	-.316	.152
Kurtosis	-.509	.302
Kolmogorov-Smirnov	.086	.000

Baseline treatment control



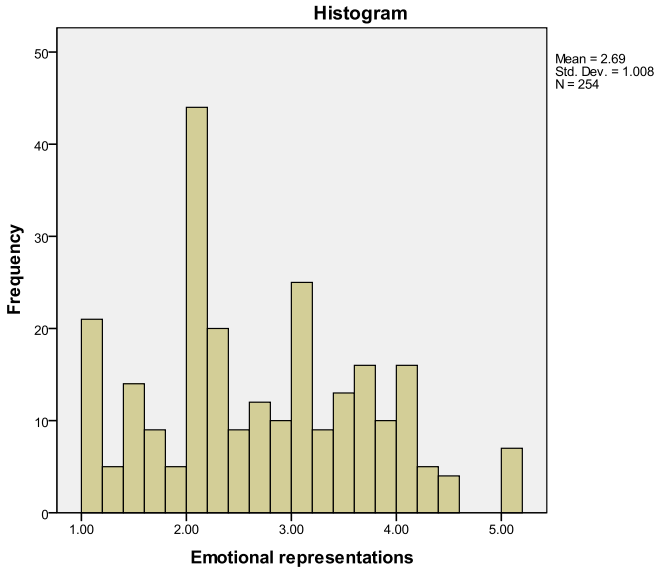
Statistical test	Test statistic	Standard error/significance
Skew	-.265	.151
Kurtosis	.197	.302
Kolmogorov-Smirnov	.100	.000

Baseline illness coherence



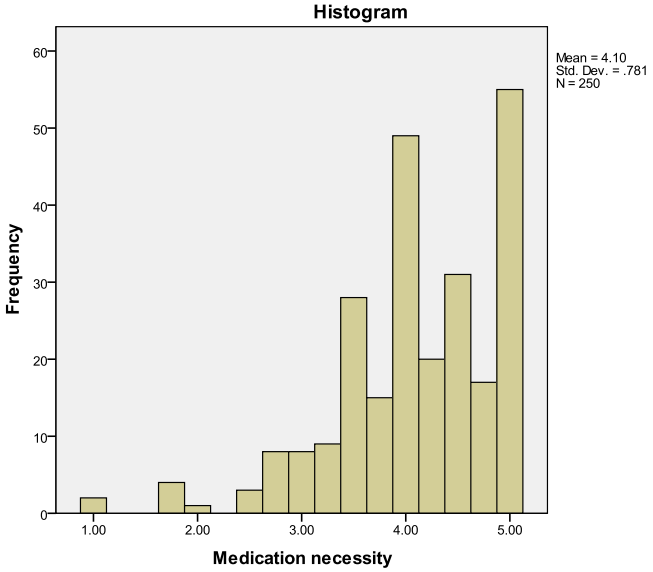
Statistical test	Test statistic	Standard error/significance
Skew	-.560	.153
Kurtosis	-.152	.304
Kolmogorov-Smirnov	.121	.000

Baseline emotional representations



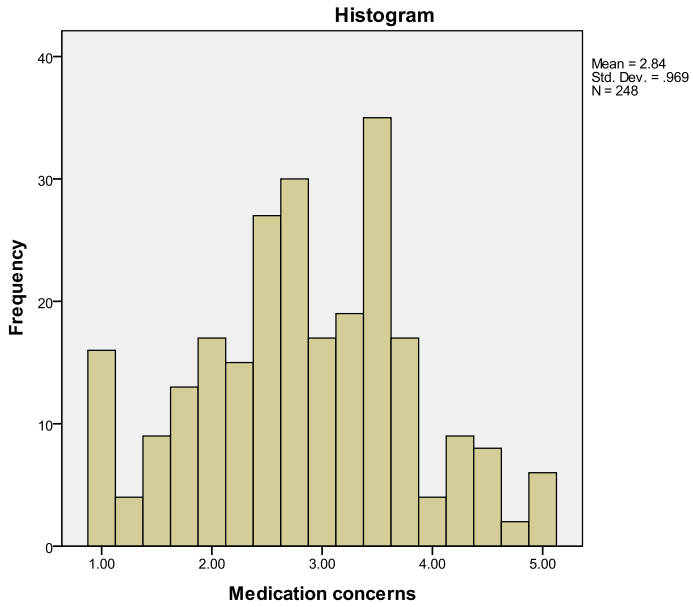
Statistical test	Test statistic	Standard error/significance
Skew	.236	.153
Kurtosis	-.704	.304
Kolmogorov-Smirnov	.101	.000

Baseline medication necessity



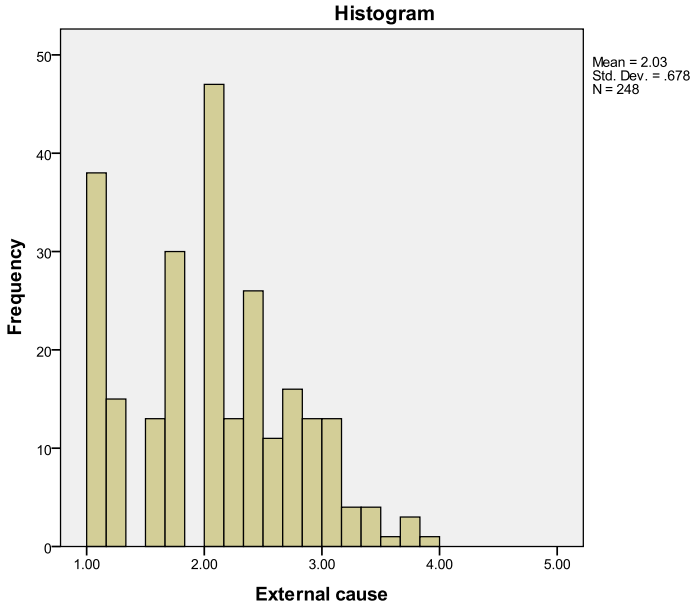
Statistical test	Test statistic	Standard error/significance
Skew	-1.031	.154
Kurtosis	1.586	.307
Kolmogorov-Smirnov	.137	.000

Baseline medication concerns



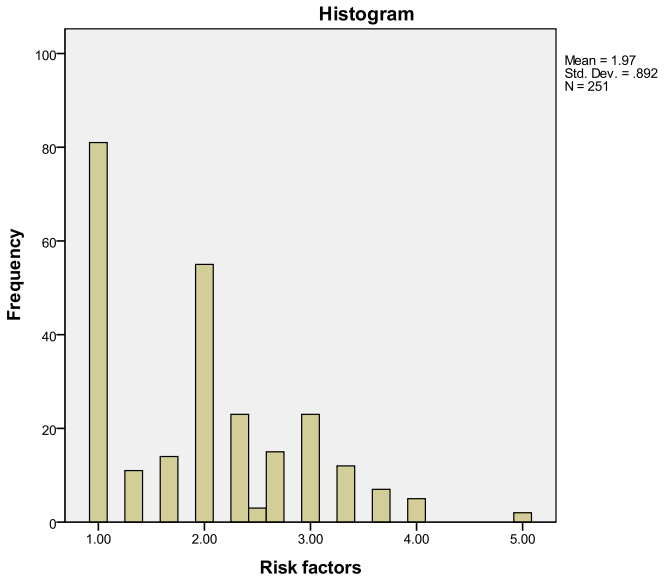
Statistical test	Test statistic	Standard error/significance
Skew	.002	.155
Kurtosis	-.423	.308
Kolmogorov-Smirnov	.076	.001

Uncontrollable cause



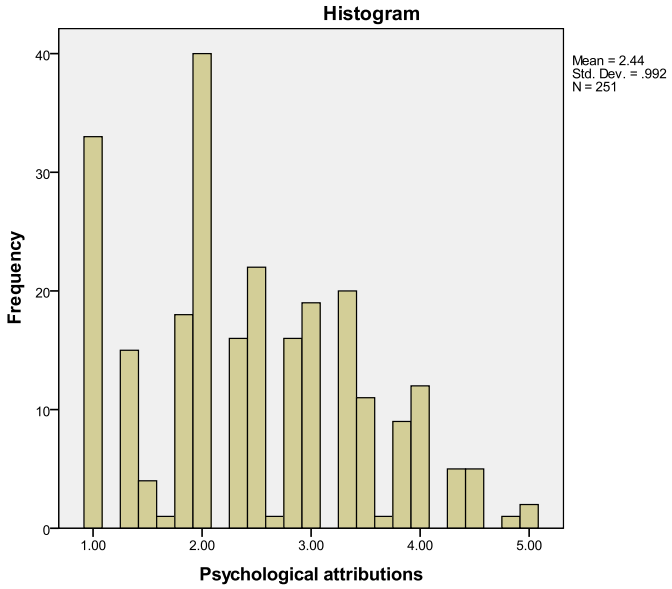
Statistical test	Test statistic	Standard error/significance
Skew	.185	.155
Kurtosis	-.498	.308
Kolmogorov-Smirnov	.096	.000

Baseline risk factors



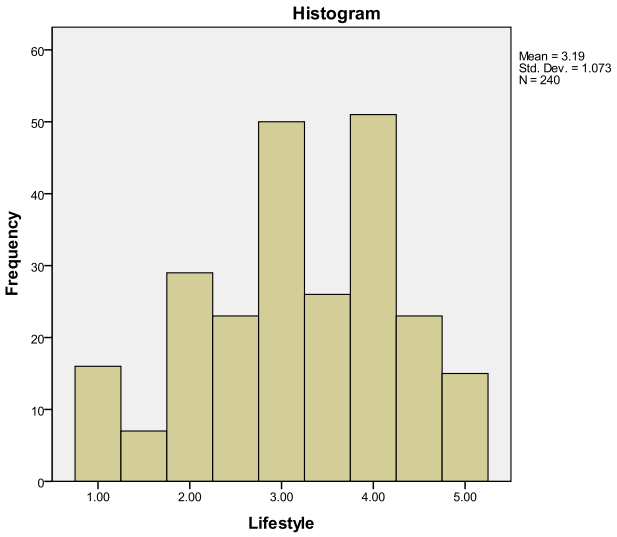
Statistical test	Test statistic	Standard error/significance
Skew	.657	.154
Kurtosis	-.088	.306
Kolmogorov-Smirnov	.185	.000

Baseline psychological attributions



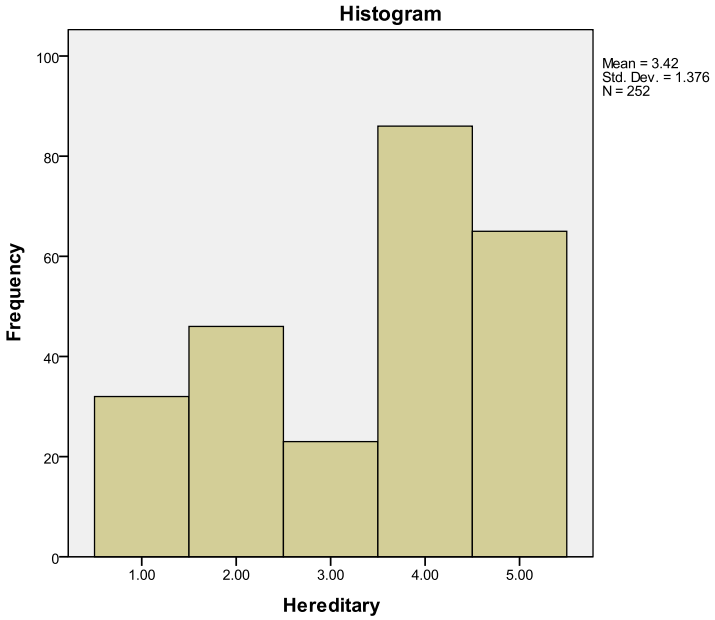
Statistical test	Test statistic	Standard error/significance
Skew	.311	.154
Kurtosis	-.655	.306
Kolmogorov-Smirnov	.115	.000

Baseline lifestyle



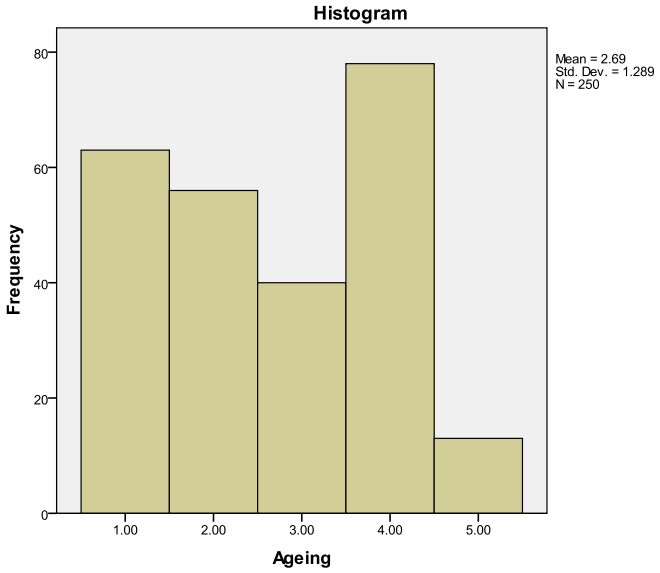
Statistical test	Test statistic	Standard error/significance
Skew	-.307	.154
Kurtosis	-.655	.306
Kolmogorov-Smirnov	.146	.000

Baseline hereditary



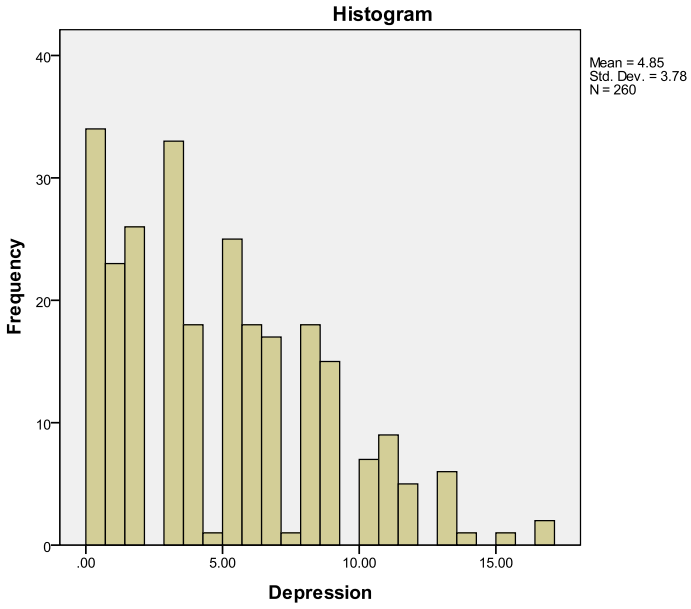
Statistical test	Test statistic	Standard error/significance
Skew	.148	.178
Kurtosis	-.802	.354
Kolmogorov-Smirnov	.262	.000

Baseline ageing



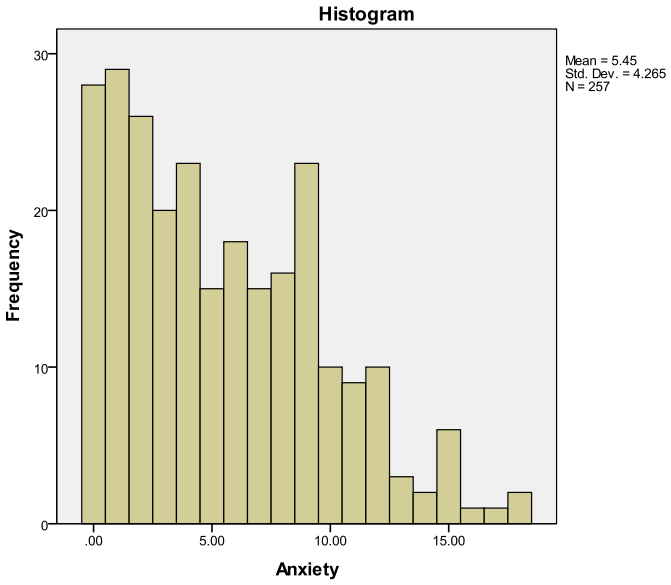
Statistical test	Test statistic	Standard error/significance
Skew	.032	.154
Kurtosis	-1.357	.307
Kolmogorov-Smirnov	.210	.000

Baseline depression



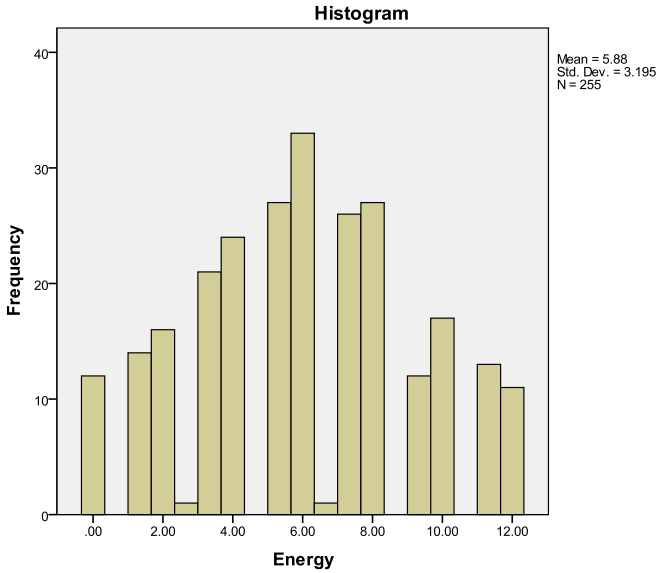
Statistical test	Test statistic	Standard error/significance
Skew	.690	.151
Kurtosis	-.072	.301
Kolmogorov-Smirnov	.133	.000

Baseline anxiety



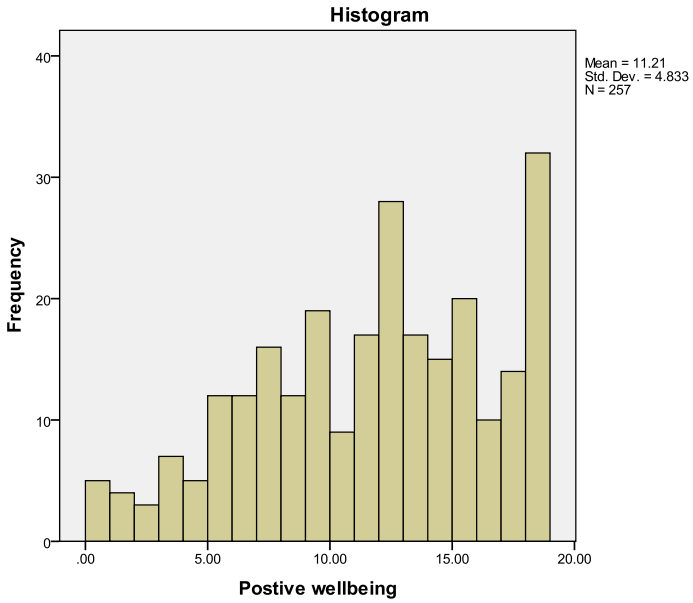
Statistical test	Test statistic	Standard error/significance
Skew	.636	.152
Kurtosis	-.303	.303
Kolmogorov-Smirnov	.123	.000

Baseline energy



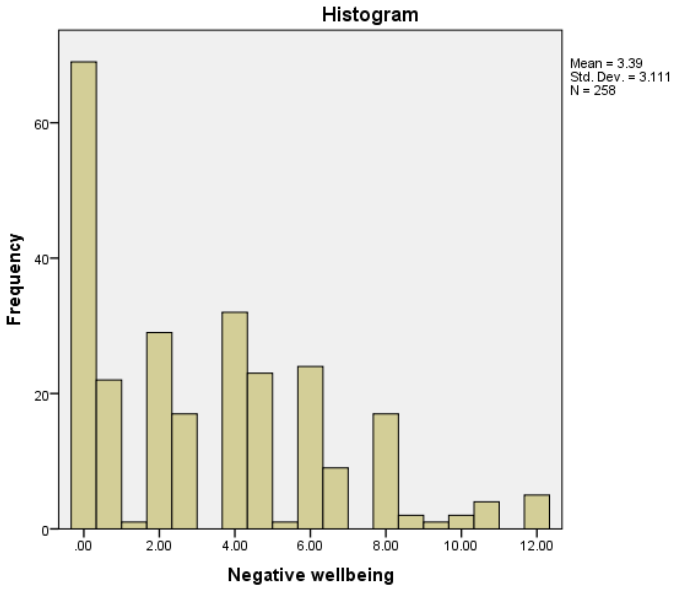
Statistical test	Test statistic	Standard error/significance
Skew	.053	.153
Kurtosis	-.762	.304
Kolmogorov-Smirnov	.067	.008

Baseline positive well-being



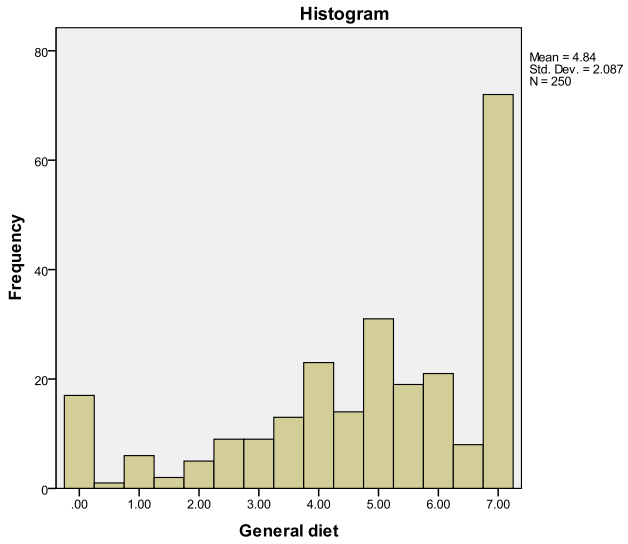
Statistical test	Test statistic	Standard error/significance
Skew	-.363	.152
Kurtosis	-.712	.303
Kolmogorov-Smirnov	.094	.000

Baseline negative well-being



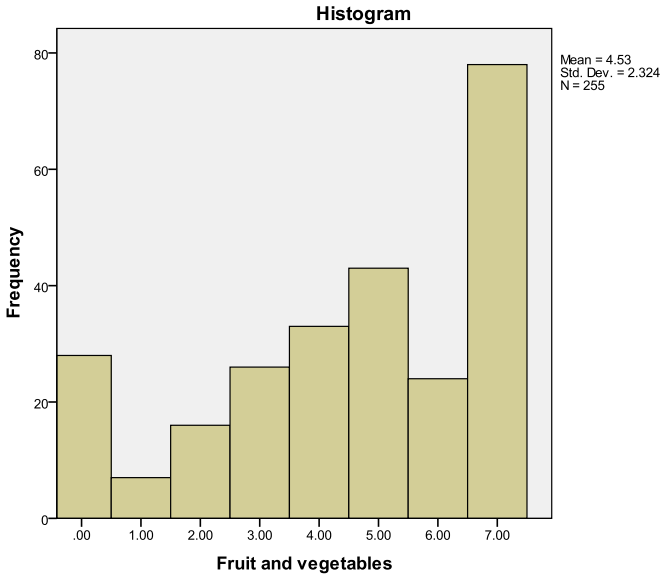
Statistical test	Test statistic	Standard error/significance
Skew	.734	.152
Kurtosis	-.127	.302
Kolmogorov-Smirnov	.142	.000

Baseline general diet



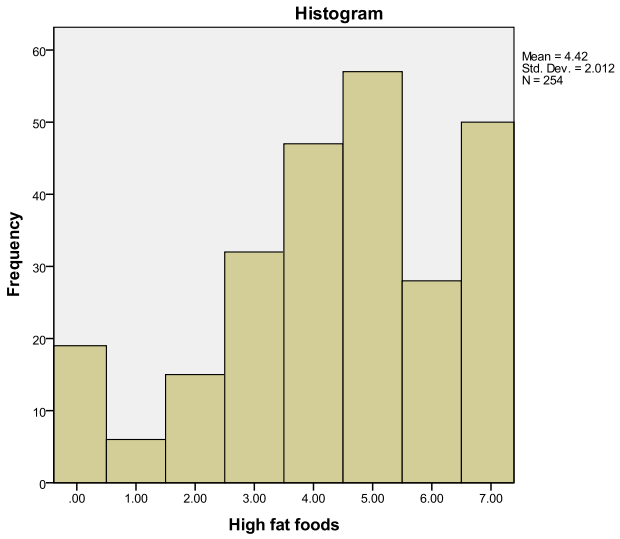
Statistical test	Test statistic	Standard error/significance
Skew	-.876	.154
Kurtosis	-.067	.307
Kolmogorov-Smirnov	.151	.000

Baseline fruit and vegetables



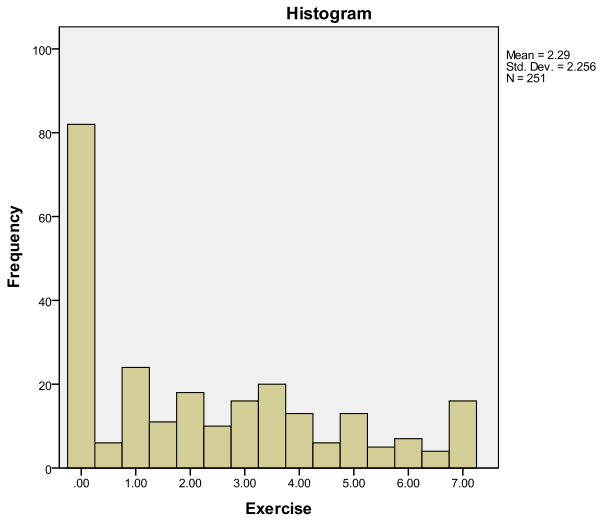
Statistical test	Test statistic	Standard error/significance
Skew	-.630	.153
Kurtosis	-.733	.304
Kolmogorov-Smirnov	.162	.000

Baseline high fat foods



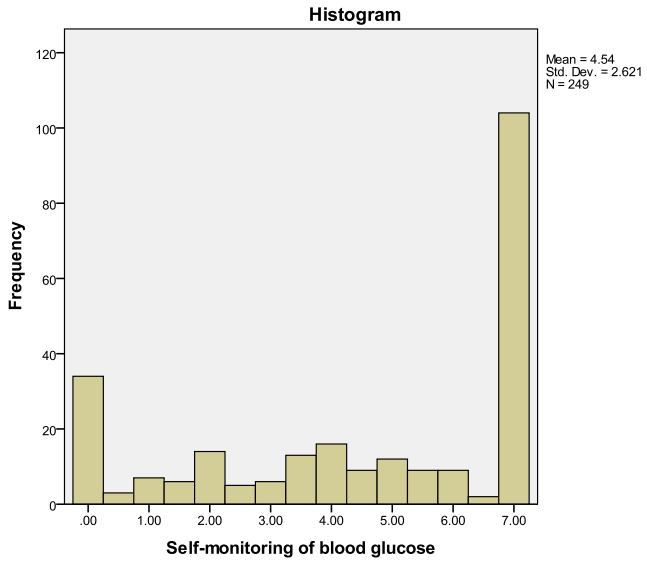
Statistical test	Test statistic	Standard error/significance
Skew	-.593	.153
Kurtosis	-.288	.304
Kolmogorov-Smirnov	.145	.000

Baseline exercise



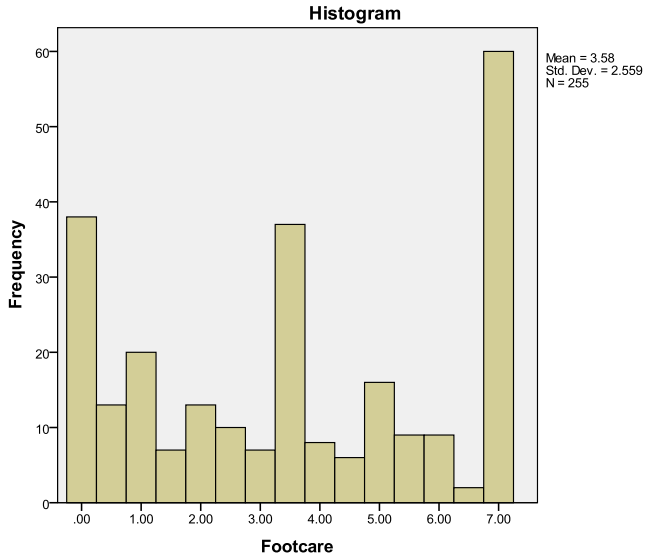
Statistical test	Test statistic	Standard error/significance
Skew	.653	.154
Kurtosis	-.757	.306
Kolmogorov-Smirnov	.171	.000

Baseline self-monitoring of blood glucose



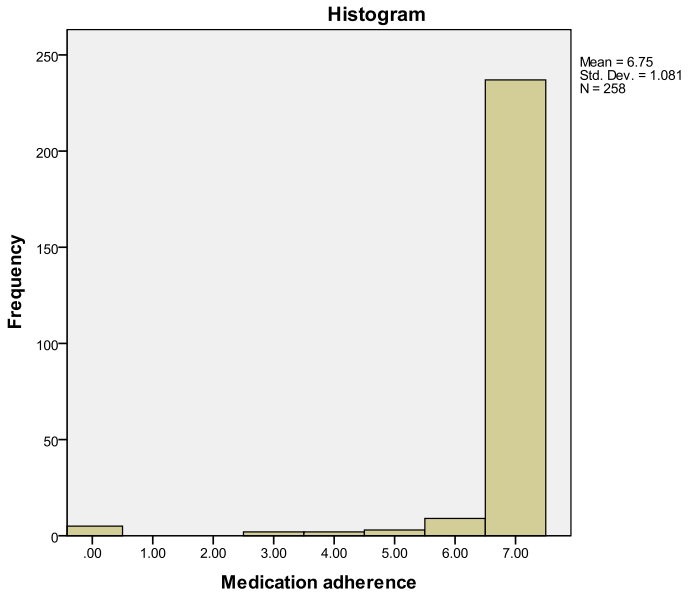
Statistical test	Test statistic	Standard error/significance
Skew	-.580	.154
Kurtosis	-1.159	.307
Kolmogorov-Smirnov	.244	.000

Baseline foot care



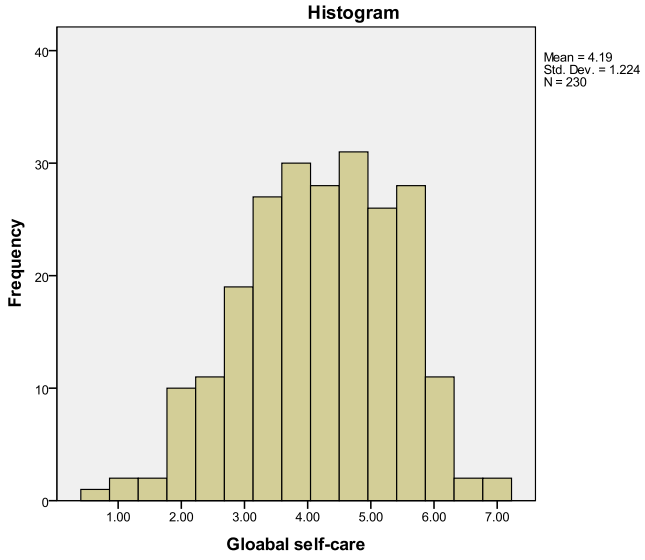
Statistical test	Test statistic	Standard error/significance
Skew	.018	.153
Kurtosis	-1.403	.304
Kolmogorov-Smirnov	.144	.000

Baseline medication adherence



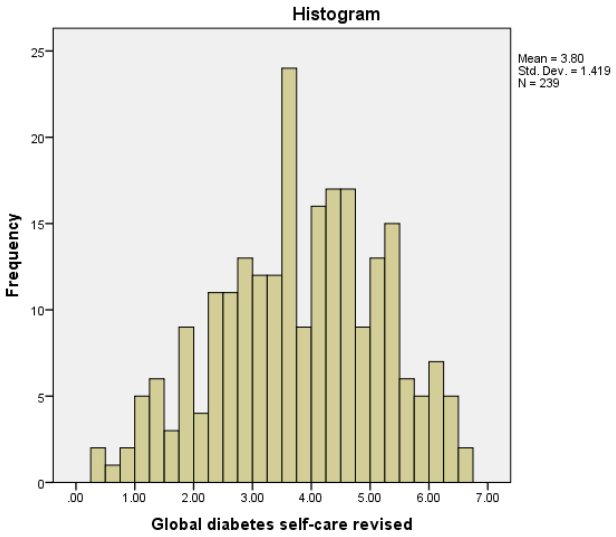
Statistical test	Test statistic	Standard error/significance
Skew	-.5.291	.152
Kurtosis	28.900	.302
Kolmogorov-Smirnov	.509	.000

Baseline global self-care



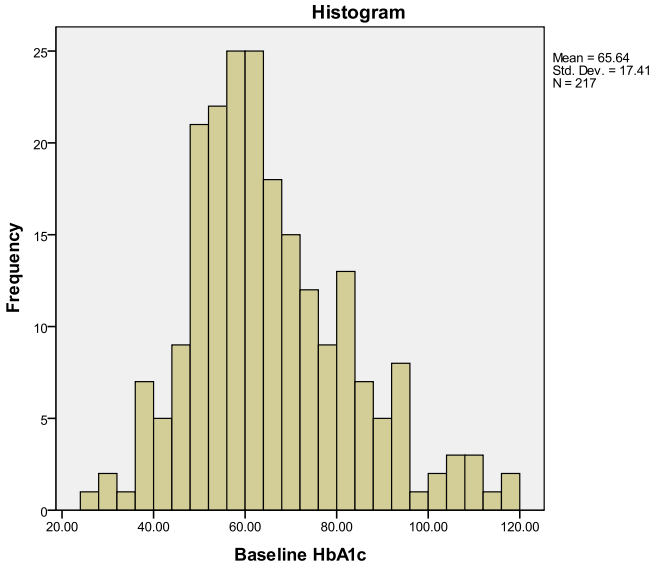
Statistical test	Test statistic	Standard error/significance
Skew	-.294	.160
Kurtosis	-.345	.320
Kolmogorov-Smirnov	.053	.200

Baseline global diabetes self-care revised



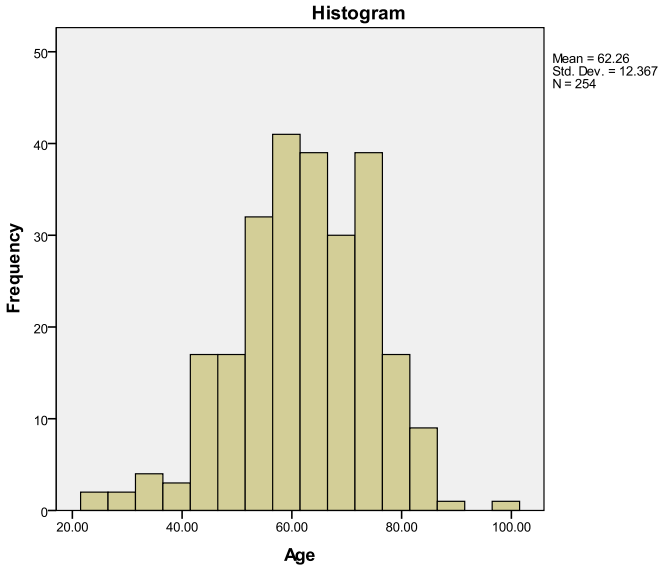
Statistical test	Test statistic	Standard error/significance
Skew	-.138	.157
Kurtosis	-.466	.314
Kolmogorov-Smirnov	.043	.200

Baseline HbA1c mmol/mol



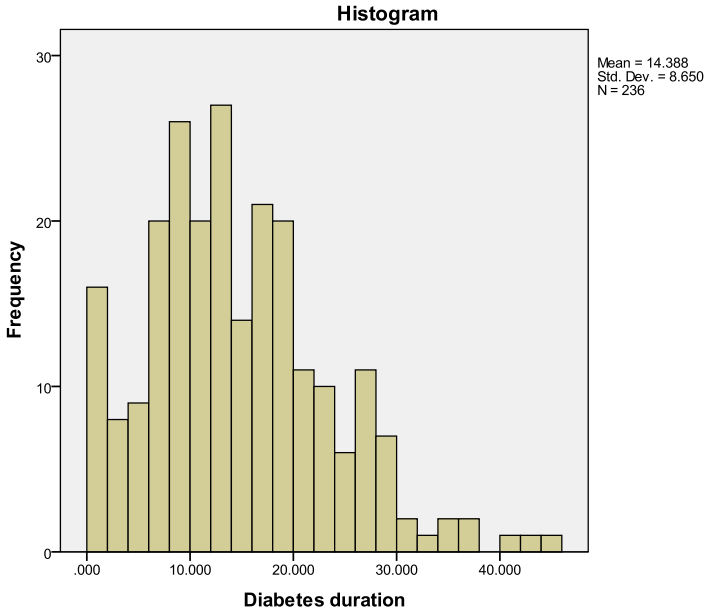
Statistical test	Test statistic	Standard error/significance
Skew	.663	.165
Kurtosis	.287	.329
Kolmogorov-Smirnov	.106	.000

Baseline age/years



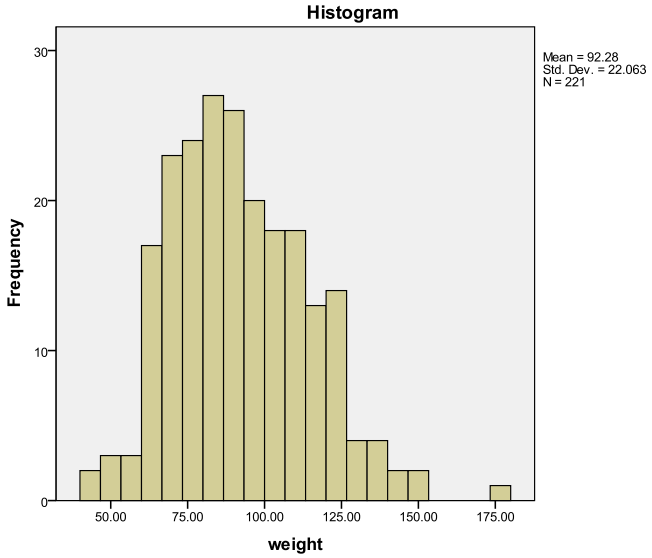
Statistical test	Test statistic	Standard error/significance
Skew	.-.361	.153
Kurtosis	.221	.304
Kolmogorov-Smirnov	.055	.057

Baseline diabetes duration (years/days)



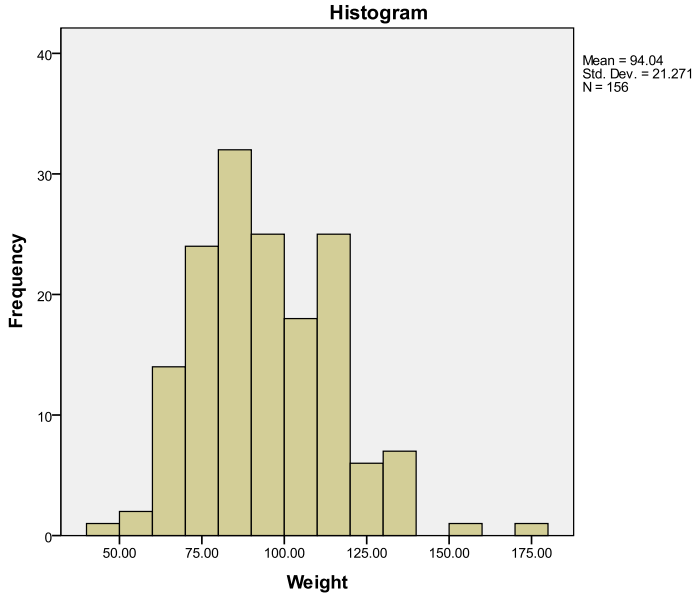
Statistical test	Test statistic	Standard error/significance
Skew	.697	.158
Kurtosis	.522	.316
Kolmogorov-Smirnov	.069	.009

Baseline Weight (Kg)



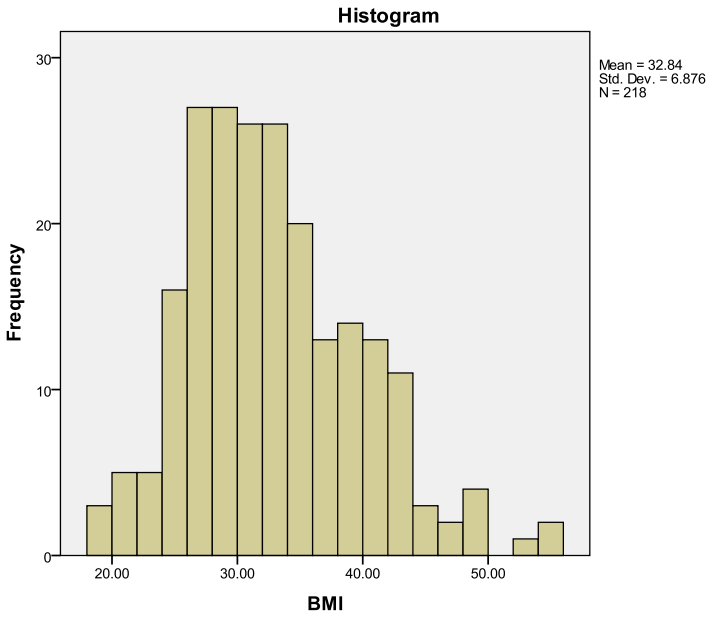
Statistical test	Test statistic	Standard error/significance
Skew	.548	.164
Kurtosis	.371	.326
Kolmogorov-Smirnov	.063	.031

Follow-up weight (Kg)



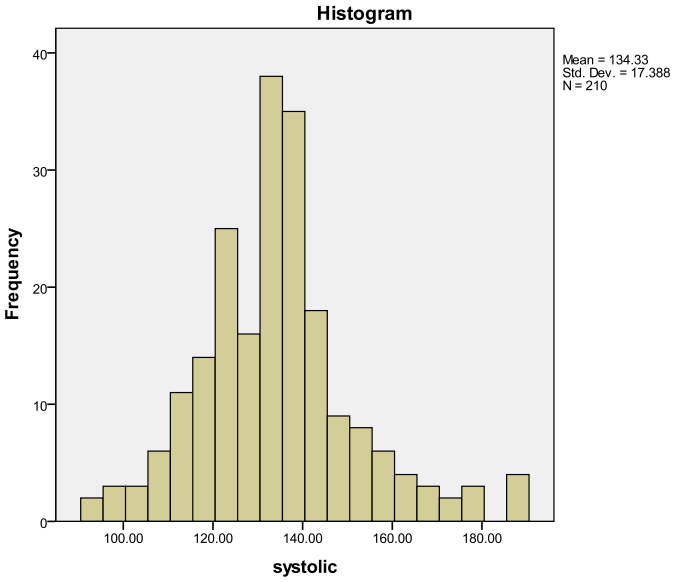
Statistical test	Test statistic	Standard error/significance
Skew	.595	.194
Kurtosis	.750	.386
Kolmogorov-Smirnov	.052	.200

Baseline BMI



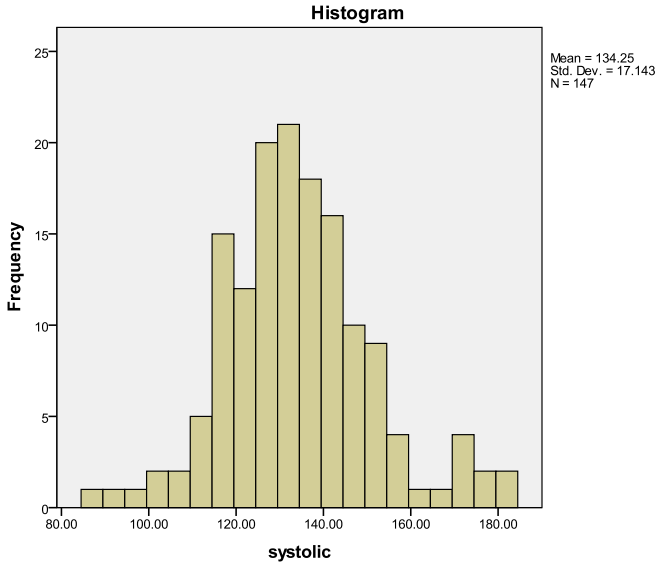
Statistical test	Test statistic	Standard error/significance
Skew	.603	.165
Kurtosis	.394	.328
Kolmogorov-Smirnov	.065	.025

Baseline Systolic (mmHg)



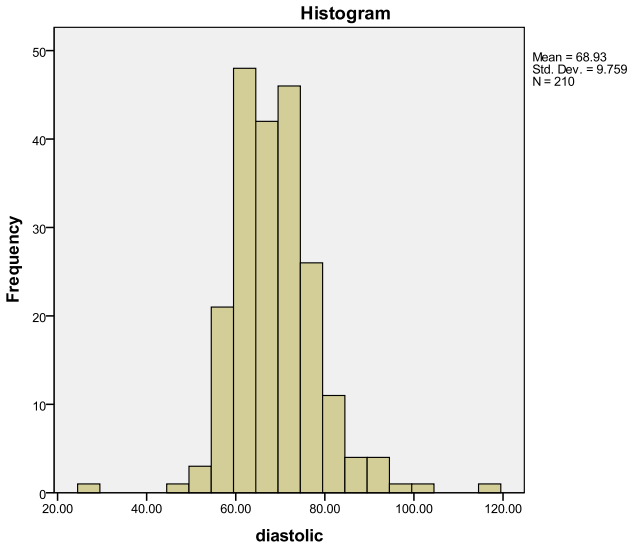
Statistical test	Test statistic	Standard error/significance
Skew	.632	.168
Kurtosis	1.156	.334
Kolmogorov-Smirnov	.113	.000

Follow-up systolic



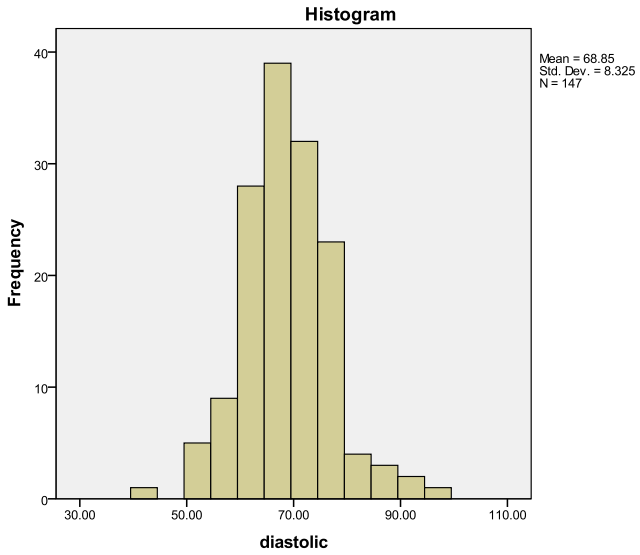
Statistical test	Test statistic	Standard error/significance
Skew	.362	.200
Kurtosis	.847	.397
Kolmogorov-Smirnov	.060	.200

Baseline Diastolic (mmHg)



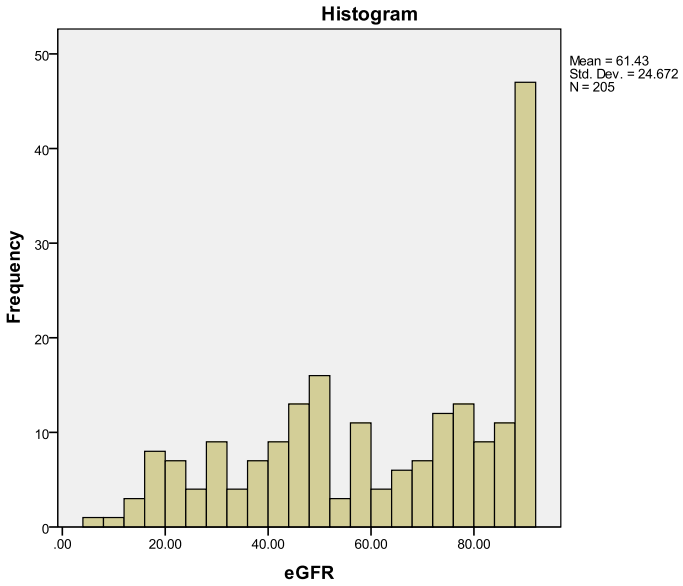
Statistical test	Test statistic	Standard error/significance
Skew	.684	.168
Kurtosis	3.650	.334
Kolmogorov-Smirnov	.078	.004

Follow-up diastolic



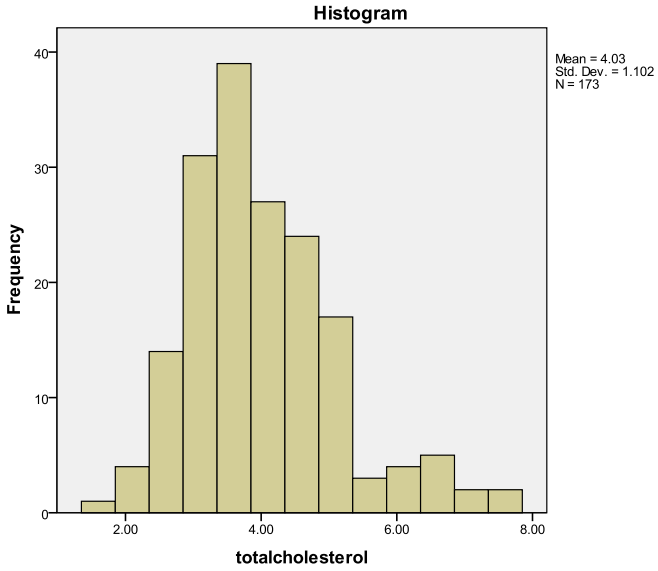
Statistical test	Test statistic	Standard error/significance
Skew	.183	.200
Kurtosis	1.125	.397
Kolmogorov-Smirnov	.055	.200

Baseline eGFR 1.73m²



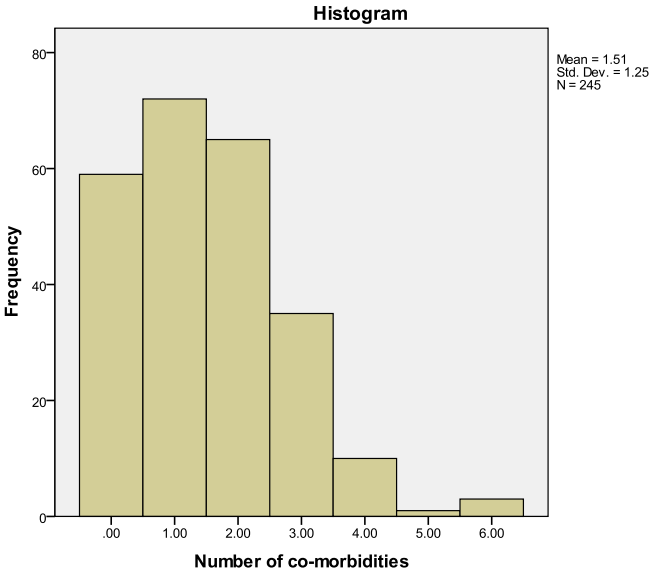
Statistical test	Test statistic	Standard error/significance
Skew	-.382	.170
Kurtosis	-1.142	.338
Kolmogorov-Smirnov	.130	.000

Baseline total cholesterol (mmol)



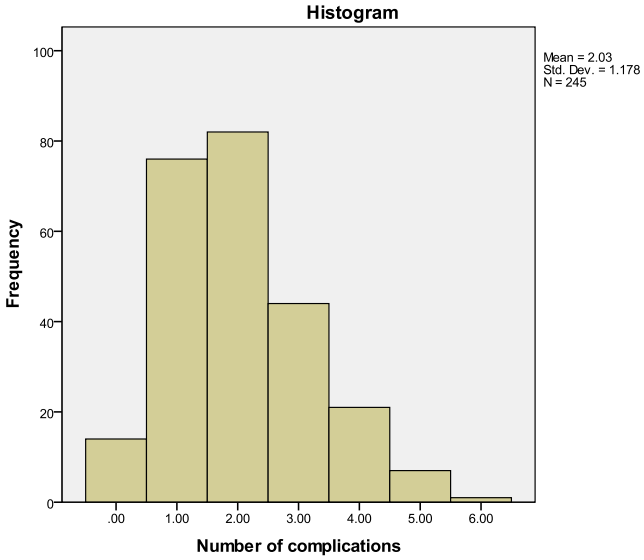
Statistical test	Test statistic	Standard error/significance
Skew	.884	.185
Kurtosis	.957	.367
Kolmogorov-Smirnov	.109	.000

Baseline number of co-morbidities



Statistical test	Test statistic	Standard error/significance
Skew	.802	.156
Kurtosis	.816	.310
Kolmogorov-Smirnov	.193	.000

Baseline number of complications



Statistical test	Test statistic	Standard error/significance
Skew	.626	.156
Kurtosis	.172	.310
Kolmogorov-Smirnov	.212	.000

12.3.4 ***Tables A1 to A26***

Table A1: Item level missing value analysis-Illness and medication cognition domains

Variables	Valid score at Time 1 across all items		Valid Score at Time 2 across all items		Valid score at Time 1 &2 across all items		Missing at Time 1 across all items		Missing at Time 2 across all items	
	N	%	N	%	N	%	N	%	N	%
Identity	242	92.7	189	72.4	173	66.3	19	7.3	72	27.6
Timeline acute/chronic	248	95.0	184	70.5	176	67.4	13	5.0	77	29.5
Timeline cyclical	251	96.2	187	71.6	181	69.3	10	3.2	74	28.4
Consequences	243	93.1	181	69.3	173	66.3	18	6.9	80	30.7
Personal control	239	91.6	181	69.3	172	65.9	22	8.4	80	30.7
Treatment control	249	95.4	185	70.9	177	67.8	12	4.6	76	29.1
Illness coherence	251	96.2	189	72.4	183	70.1	10	3.8	72	27.5
Emotional representations	251	96.2	188	72.0	184	70.5	10	3.8	73	28.0
Medication necessity	244	93.5	184	70.4	177	67.8	17	6.5	77	29.6
Medication concerns	243	93.1	185	70.9	175	67.0	18	6.9	76	29.1
Uncontrollable Cause	225	86.2	178	68.2	156	59.8	36	13.8	83	31.8
Lifestyle	240	92.0	181	69.3	166	63.6	21	7.0	78	30.6
Risk factors	247	94.6	184	70.5	177	67.8	14	5.4	77	29.5
Psychological attributions	244	93.5	180	69.0	168	64.4	17	6.5	81	31.0
Age	250	95.8	186	71.3	181	69.3	11	4.2	75	28.7
Hereditary	252	96.6	189	72.4	183	70.1	9	3.4	72	27.6

Table A1: Item level missing value analysis well-being questionnaire subscales

Variables	Valid score at Time 1 across all items		Valid Score at Time 2 across all items		Valid score at Time 1 &2 across all items		Missing at Time 1 across all items		Missing at Time 2 across all items	
	N	%	N	%	N	%	N	%	N	%
Depression	248	95.0	189	72.4	180	72.4	13	4.9	72	27.6
Anxiety	249	95.4	190	72.8	181	69.3	12	4.7	71	27.2
Energy	250	95.8	189	72.4	181	69.3	11	4.2	72	27.6
Positive well-being	254	97.3	190	72.8	184	70.5	7	2.7	71	27.2

Table A1: Item level missing value analysis-diabetes self-care behaviour subscales

Variables	Valid score at Time 1 across all items		Valid Score at Time 2 across all items		Valid score at Time 1 &2 across all items		Missing at Time 1 across all items		Missing at Time 2 across all items	
	N	%	N	%	N	%	N	%	N	%
General diet	250	95.8	185	70.9	179	68.6	11	4.2	76	29.1
Specific diet (fruit & vegetables)	255	97.7	187	71.6	183	70.1	6	2.3	74	28.4
Specific diet (fat)	254	97.3	188	72.0	183	70.1	7	2.7	73	28.0
Exercise	251	96.2	188	72.0	189	72.4	10	3.8	73	28.0
Self-monitoring of blood glucose	249	95.4	187	71.6	180	69.0	12	4.6	78	28.4
Foot care	255	97.7	191	73.2	187	71.6	6	2.3	70	26.8
Medication adherence	258	98.9	192	73.6	189	72.4	3	1.1	69	26.4
Global self-care	230	88.1	177	67.8	159	60.9	31	11.9	84	32.2

Table A1: Missing value analysis-socio-demographic and clinic characteristics

Variables	Valid score at Time 1		Valid Score at Time 2		Valid score at Time 1 & 2		Missing at Time 1		Missing at Time 2	
	N	%	N	%	N	%	N	%	N	%
Gender	261	100	194	74.3	194	74.3	0	0	67	25.7
Age	254	97.3	190	72.8	190	72.8	7	2.7	71	27.2
Diabetes Duration	236	90.4	180	69.0	180	69.0	25	9.6	81	31.0
Ethnicity	261	100	194	74.3	194	74.3	0	0	67	25.7
Diabetes treatment	239	91.6	181	69.3	181	69.3	22	8.4	58	30.7
Baseline HbA1c	217	83.1	163	62.5	140	54.0	44	16.8	98	37.1
Baseline Weight	221	84.7	156	59.8	144	55.2	40	15.3	104	39.8
Baseline BMI	218	83.5	154	59.0	140	54.0	43	16.4	108	41.4
BP systolic	210	80.5	147	56.3	127	48.7	51	19.5	106	40.6
BP diastolic	210	80.5	147	56.3	127	48.7	51	19.5	106	40.6
eGFR	205	78.5	147	56.3	122	46.7	56	21.5	106	40.6
Total cholesterol	173	66.3	135	51.7	92	35.2	88	33.7	125	48.0
Number of complications	245	93.9	185	70.5	185	70.5	16	6.13	76	29.1
Number of co-morbidities	245	93.9	185	70.1	185	70.1	16	6.13	76	29.1

Table A2: Subscale level missing value analysis-Illness and medication cognition domains

Variables	Valid score at Time 1		Valid Score at Time 2		Valid score at Time 1 &2		Missing at Time 1		Missing at Time 2	
	N	%	N	%	N	%	N	%	N	%
Identity	242	92.7	189	72.4	173	62.5	19	7.3	72	27.6
Timeline acute/chronic	255	97.7	192	73.6	188	72.0	6	2.3	69	26.4
Timeline cyclical	255	97.7	190	72.8	187	71.6	6	2.3	71	27.2
Consequences	255	97.7	190	72.8	186	71.3	6	2.3	71	27.2
Personal control	258	98.9	191	73.2	190	73.0	3	1.1	70	26.8
Treatment control	259	99.2	191	73.2	190	73.0	2	0.8	70	26.8
Illness coherence	255	97.7	190	72.8	186	71.3	6	2.3	69	26.4
Emotional representations	254	97.3	190	72.8	184	70.5	7	2.7	71	27.2
Medication necessity	250	95.8	190	72.8	184	70.5	11	4.2	71	27.2
Medication concerns	248	95.0	187	71.6	181	69.3	13	5.0	74	28.4
Uncontrollable Cause	248	95.0	187	71.6	178	68.2	13	5.0	74	28.4
Lifestyle	240	92.0	181	69.3	166	63.6	21	8.0	80	30.7
Risk factors	247	94.6	184	70.5	177	67.8	14	5.4	77	29.5
Psychological attributions	251	96.2	187	71.6	179	68.6	10	3.8	74	28.4
Age	250	95.8	186	71.3	181	69.3	11	4.2	75	28.7
Hereditary	252	96.6	189	72.4	183	70.1	9	3.4	72	27.6

Table A2: Subscale level missing value analysis-well-being questionnaire subscales

Variables	Valid score at Time 1		Valid Score at Time 2		Valid score at Time 1 &2		Missing at Time 1		Missing at Time 2	
	N	%	N	%	N	%	N	%	N	%
Depression	260	99.6	193	73.9	192	73.6	1	0.4	68	26.1
Anxiety	257	98.5	193	73.9	190	72.8	4	1.5	68	26.1
Positive Wellbeing	257	98.5	193	73.9	190	72.8	4	1.5	68	26.1
Energy	255	97.7	194	74.3	190	72.8	6	2.3	67	25.7

Table A2: Subscale level missing value analysis-diabetes self-care behaviour subscales

Variables	Valid score at Time 1		Valid Score at Time 2		Valid score at Time 1 &2		Missing at Time 1		Missing at Time 2	
	N	%	N	%	N	%	N	%	N	%
General diet	250	95.8	185	70.9	179	68.6	11	4.2	76	29.1
Specific diet (fruit & vegetables)	255	97.7	187	71.6	183	70.1	6	2.3	74	28.4
Specific diet fat	254	97.3	188	70.9	183	70.1	7	2.7	76	29.1
Exercise	251	96.2	188	72.0	184	70.5	10	1.9	74	28.4
Self-monitoring of blood glucose	249	95.4	187	76.6	180	69.0	12	4.6	74	28.4
Foot care	255	97.7	191	73.2	187	71.6	6	2.3	70	26.8
Medication adherence	258	98.9	192	73.6	189	72.4	3	1.1	69	26.4
Global self-care	230	88.1	177	67.8	159	60.9	31	11.9	84	32.2

Table A3: Correlation matrix of the 18 causal items of the IPQ-R (n=205; Key: *p≤.05, **p≤.01)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Stress or worry																	
2. Hereditary	.10																
3. Germ or virus	.21**	.06															
4.Diet or eating habit	.05	.00	-.03														
5. Chance or bad luck	.16*	-.03	.14*	-.07													
6. Poor medical care in my past	.20**	.11	.26**	.12	.31**												
7. Pollution in the environment	.23**	.08	.45**	-.02	.27**	.39**											
8. My own behaviour	.10	-.05	.00	.55**	-.02	.12	-.02										
9. My mental attitude	.38**	.03	.26**	.12	.37**	.34**	.40**	.26**									
10. Family problems or worries	.52**	.11	.24**	.07	.19**	.29**	.33**	.25**	.64**								
11. Overwork	.40**	.03	.31**	.16*	.30**	.33**	.33**	.19**	.44**	.50**							
12. My emotional state	.52**	.06	.40**	.15*	.20**	.42**	.38**	.28**	.66**	.68**	.46**						
13. Ageing	.10	.07	.29**	.09	.19**	.07	.20**	.08	.20**	.14*	.21**	.24**					
14. Alcohol	.15*	.13	.19**	.17*	.02	.12	.07	.21**	.22**	.26**	.14*	.19**	.19**				
15. Smoking	.14*	.10	.21**	.03	.01	.18*	.14*	.10	.23**	.24**	.13	.15*	.17*	.69**			
16. Accident or injury	.27**	-.08	.33**	.05	.15*	.22**	.35**	.06	.47**	.39**	.30**	.42**	.16*	.34**	.40**		
17. My personality	.19**	.05	.18**	.15*	.21**	.25**	.38**	.21**	.60**	.40**	.30**	.45**	.18*	.22**	.17*	.45**	
18. Altered immunity	.15*	-.06	.26**	-.07	.25**	.33**	.30**	.13	.34**	.19**	.32**	.37**	.22**	.07	.08	.36**	.35**

Table A4: Eigenvalues and variance explained for each component in the IPQ-R causal item PCA analysis

Component	Eigenvalues	% variance explained	% cumulative variance explained
1	5.2	32.4	32.4
2	1.8	11.2	43.6
3	1.5	9.5	53.2
4	1.1	7.0	60.1
5	1.0	6.1	66.2
6	0.9	5.6	71.8
7	0.7	4.6	76.4
8	0.7	4.2	80.5
9	0.6	3.6	84.1
10	0.5	3.3	87.5
11	0.5	2.9	90.4
12	0.4	2.7	93.1
13	0.3	2.1	95.2
14	0.3	1.9	97.1
15	0.2	1.5	98.6
16	0.2	1.4	100.0

Table A5: Principal components analysis pattern matrix of the 16 IPQ-R causal items.

	Component			
Items	I	II	III	IV
Component I				
Altered immunity	**.76**	.03	.03	.12
Pollution in the environment	**.65**	.15	.05	.12
Poor medical care in my past	**.63**	.12	.01	.04
Chance or bad luck	**.62**	.08	.15	.01
My personality	**.50**	.24	.18	.09
Germ or virus	**.42**	.22	.20	-.17
Component II				
Diet or eating habit	-.05	**.85**	.00	.02
My own behaviour	.00	**.85**	.03	-.08
Component III				
Smoking	.05	.07	**.92**	.04
Alcohol	.12	.13	**.88**	.03
Accident or injury	.33	.07	**.51**	-.14
Component IV				
Stress or worry	.20	.10	.03	**.92**
Family problems or worries	.01	.07	.09	**.84**
My emotional state	.26	.14	.01	**.69**
Overwork	.26	.12	.04	**.51**
My mental attitude	**.40**	.16	.08	**.49**

Table A6: Eigenvalues and variance explained for each of the 14 components in the IPQ-R causal item PCA

Component	Eigenvalues	% of variance explained	% cumulative variance explained
1	4.2	30.2	30.2
2	1.8	12.9	43.1
3	1.4	10.0	53.1
4	1.1	8.1	61.2
5	0.9	6.3	67.5
6	0.8	5.9	73.5
7	0.7	5.2	78.6
8	0.6	4.6	83.3
9	0.6	3.9	87.2
10	0.5	3.5	90.7
11	0.5	3.2	93.9
12	0.3	2.4	96.2
13	0.3	2.2	98.4
14	0.2	1.6	100

Table A7: Principal components analysis pattern matrix of the 14 IPQ-R causal items.

	Component			
Causal Items	I	II	III	IV
Uncontrollable cause				
Pollution in the environment	**.69**	-.14	.05	-.10
Altered immunity	**.68**	.04	-.03	.03
Poor medical care in my past	**.68**	.12	.03	.
Chance or bad luck	**.65**	-.08	-.13	.06
Germ or virus	**.49**	-.2	.22	-.15
My personality	**.47**	.25	.14	-.10
Lifestyle				
Diet or eating habit	-.04	**.87**	.	.04
My own behaviour	-.02	**.84**	.04	-.09
Risk factors				
Smoking	-.02	-.08	**.92**	.00
Alcohol	-.03	.11	**.90**	.05
Psychological attributions				
Stress or worry	-.16	-.10	-.05	**.92**
Family problems or worries	.	.06	.09	**.83**
My emotional state	.25	.14	-.01	**.70**
Overwork	.32	.13	-.04	**.48**

Table A8: Correlation matrix between identified IPQ-R causal components

Component	I	II	II
I. Uncontrollable cause			
II. Lifestyle	.06		
III. Risk factors	.16	.14	
IV. Psychological attributions	.41	.16	.26

Table A9: Mann-Whitney U Tests to explore the relationship between neuropathy and cognitions, well-being subscales, and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	3.8	3.3	3.9	3.2	-0.3	.79
	median	3.0	1.00-6.00	3.0	1.00-5.00		
Timeline acute/chronic	mean	4.2	.8	4.3	.7	-0.6	.56
	median	4.3	3.83-4.83	4.5	3.83-4.83		
Timeline cyclical	mean	2.8	1.0	3.0	.9	-1.2	.24
	median	2.8	2.00-3.50	3.0	2.25-3.54		
Consequences	mean	3.3	.8	3.2	.8	-0.8	.45
	median	3.3	2.67-3.83	3.2	2.67-3.67		
Personal control	mean	4.0	.7	3.8	.7	-3.0	.00
	median	4.0	3.67-4.50	3.8	3.33-4.17		
Treatment control	mean	3.6	.6	3.4	.6	-2.4	.02
	median	3.6	3.20-4.00	3.4	3.00-3.80		
Illness coherence	mean	3.6	.9	3.6	.9	-0.5	.63
	median	3.8	3.00-4.00	3.8	3.15-4.20		
Emotional representations	mean	2.7	1.0	2.7	1.0	-0.1	.95
	median	2.6	2.00-3.50	2.3	2.00-3.50		
Medication necessity	mean	4.1	.8	4.1	.7	-0.5	.59
	median	4.0	3.80-4.80	4.0	3.60-4.80		
Medication concerns	mean	2.8	1.0	2.8	1.0	-0.1	.94
	median	2.8	2.20-3.45	2.8	2.00-3.60		
Uncontrollable cause	mean	2.0	.7	2.1	.7	-0.3	.76
	median	2.0	1.33-2.50	2.0	1.53-2.50		
Lifestyle	mean	3.3	1.1	3.0	1.1	-1.8	.07
	median	3.5	2.50-4.00	3.0	2.00-4.00		
Risk factors	mean	2.0	1.1	1.9	1.0	-1.0	.33
	median	2.0	1.00-3.00	2.0	1.00-2.38		
Psychological attributions	mean	2.5	1.0	2.3	.9	-1.4	.15
	median	2.5	1.75-3.25	2.0	1.75-2.81		
Hereditary	mean	3.5	1.3	3.2	1.5	-1.3	.20
	median	4.0	2.00-4.00	4.0	2.00-5.00		
Ageing	mean	2.5	1.3	2.9	1.3	-2.1	.04
	median	2.0	1.00-4.00	3.0	2.00-4.00		
Depression	mean	5.0	3.6	4.4	3.7	-1.32	.19

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
	median	4.0	2.00-7.50	4.0	1.00-7.00		
Anxiety	mean	5.6	4.3	4.7	3.9	-1.42	.16
	median	5.0	2.00-9.00	4.0	1.00-7.00		
Energy	mean	5.9	3.1	6.1	3.3	-.54	.59
	median	6.0	4.00-8.00	6.0	3.00-8.00		
Positive well-being	mean	11.0	4.6	11.9	5.1	-1.78	.08
	median	11.0	7.00-15.00	12.0	8.00-17.00		
General diet	mean	4.8	2.0	5.0	2.1	-1.05	.29
	median	5.0	4.00-7.00	5.5	4.00-7.00		
Specific diet (fruit& veg)	mean	4.5	2.3	4.7	2.2	-.61	.54
	median	5.0	3.00-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.1	2.1	4.7	1.9	-1.85	.06
	median	4.0	3.00-6.00	5.0	3.00-6.00		
Exercise	mean	2.6	2.3	1.8	2.0	-2.50	.01
	median	2.5	0.00-4.00	1.0	0.00-3.50		
Self-monitoring of blood glucose	mean	4.4	2.7	4.7	2.4	-.57	.57
	median	5.5	2.00-7.00	5.0	3.50-7.00		
Foot care	mean	3.2	2.5	4.2	2.5	-3.04	.00
	median	3.5	1.00-5.38	5.0	2.00-7.00		
Medication adherence	mean	6.8	0.9	6.7	1.3	-.35	.73
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.1	1.3	4.3	1.1	-1.02	.31
	median	4.3	3.18-5.09	4.4	3.50-5.14		

Table A10: Mann-Whitney U Tests to explore the relationship between nephropathy and cognitions, well-being subscales, and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	4.0	3.3	3.3	3.1	-1.3	.18
	median	3.0	1.25-6.00	2.0	1.00-5.75		
Timeline acute/chronic	mean	4.2	.8	4.2	.7	-0.1	.93
	median	4.5	3.83-4.83	4.3	3.79-4.83		
Timeline cyclical	mean	2.9	1.0	2.6	.7	-2.1	.04
	median	3.0	2.25-3.75	2.5	2.00-3.25		
Consequences	mean	3.3	.8	3.0	.7	-2.4	.02
	median	3.3	2.67-4.00	3.0	2.46-3.50		
Personal control	mean	3.9	.7	3.9	.6	-0.6	.53
	median	4.0	3.50-4.50	4.0	3.50-4.25		
Treatment control	mean	3.6	.6	3.5	.6	-0.6	.52
	median	3.6	3.20-4.00	3.5	3.20-4.00		
Illness coherence	mean	3.6	1.0	3.7	.7	-1.0	.34
	median	3.8	3.00-4.20	4.0	3.40-4.05		
Emotional	mean	2.7	1.0	2.4	.9	-1.9	.06

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
representations							
	median	2.7	2.00-3.37	2.3	1.67-3.33		
Medication necessity	mean	4.1	.8	4.1	.7	-0.4	.72
	median	4.0	3.60-4.80	4.2	3.80-4.80		
Medication concerns	mean	2.9	1.0	2.5	.9	-2.2	.03
	median	2.8	2.20-3.60	2.7	2.00-3.25		
Uncontrollable cause	mean	2.0	.7	2.0	.6	-0.2	.85
	median	2.0	1.50-2.50	2.0	1.67-2.33		
Lifestyle	mean	3.3	1.1	2.9	1.1	-1.8	.07
	median	3.0	2.50-4.00	3.0	2.00-4.00		
Risk factors	mean	2.0	1.0	2.1	1.1	-0.5	.61
	median	2.0	1.00-2.50	2.0	1.00-3.00		
Psychological attributions	mean	2.4	1.0	2.3	.8	-1.1	.27
	median	2.5	1.75-3.25	2.0	1.75-3.00		
Hereditary	mean	3.5	1.4	3.0	1.3	-2.3	.02
	median	4.0	2.00-5.00	3.0	2.00-4.00		
Ageing	mean	2.7	1.3	2.7	1.3	-0.2	.81
	median	3.0	1.00-4.00	3.0	1.00-4.00		
Depression	mean	5.0	3.7	3.6	3.2	-2.5	.01
	median	5.0	2.00-8.00	3.0	1.00-6.00		
Anxiety	mean	5.6	4.3	3.7	3.4	-2.9	.00
	median	5.0	2.00-9.00	3.0	1.00-6.00		
Energy	mean	5.8	3.2	6.6	3.1	-1.6	.11
	median	6.0	3.00-8.00	7.0	4.75-8.25		
Positive well-being	mean	10.9	4.8	12.8	4.7	-2.6	.01
	median	12.0	7.00-15.00	13.0	9.00-17.25		
General diet	mean	5.0	2.1	4.6	2.1	-1.3	.18
	median	5.0	4.00-7.00	5.0	3.50-6.00		
Specific diet (fruit& veg)	mean	4.7	2.3	4.2	2.2	-1.8	.08
	median	5.0	3.00-7.00	5.0	3.00-6.00		
Specific diet (fat consumption)	mean	4.4	2.0	4.2	2.0	-0.6	.55
	median	5.0	3.00-6.00	4.5	3.00-6.00		
Exercise	mean	2.4	2.3	1.8	1.9	-1.7	.09
	median	2.0	0.00-4.00	1.0	0.00-3.50		
Self-monitoring of blood glucose	mean	4.5	2.6	4.9	2.5	-1.0	.33
	median	5.0	2.00-7.00	6.0	3.50-7.00		
Foot care	mean	3.6	2.6	3.8	2.5	-0.6	.52
	median	3.5	1.00-6.00	3.5	1.75-6.63		

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Medication adherence	mean	6.8	.7	6.4	1.9	-0.8	.45
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.2	1.2	4.2	1.1	-0.3	.77
	median	4.3	3.45-5.18	4.3	3.18-5.09		

Table A11: Mann-Whitney U Tests to explore the relationship between retinopathy and cognitions, well-being subscales, and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	4.0	3.2	3.3	3.3	-2.0	.05
	median	4.0	2.00-6.00	2.0	1.00-5.00		
Timeline acute/chronic	mean	4.2	.7	4.3	0.8	-0.8	.42
	median	4.3	3.83-4.83	4.5	3.95-4.83		
Timeline cyclical	mean	2.9	1.0	2.9	0.9	-0.1	.88
	median	3.0	2.00-3.50	2.6	2.00-3.75		
Consequences	mean	3.2	.8	3.3	0.8	-1.2	.21
	median	3.2	2.65-3.83	3.3	2.83-4.00		
Personal control	mean	3.9	.7	4.0	0.7	-0.7	.47
	median	4.0	3.50-4.33	4.0	3.46-4.50		
Treatment control	mean	3.6	.6	3.5	0.6	-0.5	.61
	median	3.6	3.20-4.00	3.6	3.20-4.00		
Illness coherence	mean	3.6	.9	3.7	0.9	-1.2	.23
	median	3.6	3.00-4.00	3.8	3.40-4.20		
Emotional representations	mean	2.7	1.0	2.5	1.0	-1.8	.08
	median	2.7	2.00-3.58	2.3	1.67-3.33		
Medication necessity	mean	4.1	.8	4.2	0.7	-1.3	.19
	median	4.0	3.60-4.80	4.2	3.80-4.95		
Medication concerns	mean	2.9	.9	2.7	1.0	-1.6	.12
	median	3.0	2.20-3.60	2.8	2.00-3.40		
Uncontrollable cause	mean	2.1	.7	1.9	0.7	-1.1	.27
	median	2.0	1.67-2.50	2.0	1.33-2.33		
Lifestyle	mean	3.2	1.0	3.1	1.2	-0.9	.37
	median	3.5	2.50-4.00	3.0	2.00-4.00		
Risk factors	mean	2.0	1.0	1.9	1.1	-1.1	.27
	median	2.0	1.00-3.00	2.0	1.00-2.50		
Psychological attributions	mean	2.5	1.0	2.2	1.0	-1.9	.05
	median	2.5	1.81-3.25	2.0	1.38-2.88		
Hereditary	mean	3.4	1.3	3.3	1.5	-0.2	.83
	median	4.0	2.00-4.00	4.0	2.00-5.00		
Ageing	mean	2.7	1.3	2.6	1.4	-1.0	.33
	median	3.0	2.00-4.00	2.0	1.00-4.00		
Depression	mean	5.3	3.7	3.5	3.4	-3.7	.00

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
	median	5.0	3.00-8.00	2.0	1.00-5.00		
Anxiety	mean	5.8	4.3	4.0	3.6	-3.1	.00
	median	5.0	2.00-9.00	3.0	1.00-7.00		
Energy	mean	5.6	3.0	6.7	3.3	-2.3	.02
	median	6.0	3.00-8.00	7.0	4.00-9.25		
Positive well-being	mean	10.8	4.7	12.5	4.8	-2.8	.01
	median	11.0	7.00-15.00	13.0	9.75-17.00		
General diet	mean	4.8	2.1	5.1	2.0	-1.1	.28
	median	5.0	3.50-7.00	5.0	4.00-7.00		
Specific diet (fruit& veg)	mean	4.6	2.3	4.5	2.4	-0.4	.71
	median	5.0	3.00-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.2	2.0	4.7	2.0	-1.8	.08
	median	4.0	3.00-6.00	5.0	3.00-6.75		
Exercise	mean	2.3	2.3	2.1	2.1	-0.5	.61
	median	2.0	0.00-4.00	2.0	0.00-3.50		
Self-monitoring of blood glucose	mean	4.5	2.6	4.6	2.6	-0.1	.96
	median	5.0	2.00-7.00	4.8	3.50-7.00		
Foot care	mean	3.3	2.5	4.4	2.6	-3.3	.00
	median	3.5	1.00-5.50	5.0	2.00-7.00		
Medication adherence	mean	6.8	1.0	6.7	1.2	-0.1	.89
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.1	1.2	4.5	1.2	-1.9	.05
	median	4.3	3.18-5.05	4.5	3.56-5.55		

Table A12: Mann-Whitney U Tests to explore the relationship between macrovascular disease and cognitions, well-being subscales, and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	4.3	4.2	3.8	3.2	-0.1	.91
	median	3.0	1.00-6.00	3.0	1.00-6.00		
Timeline acute/chronic	mean	4.2	1.1	4.2	0.7	-0.7	.48
	median	4.5	4.00-4.83	4.3	3.83-4.83		
Timeline cyclical	mean	2.8	0.8	2.9	1.0	-0.2	.84
	median	3.0	2.00-3.25	3.0	2.00-3.50		
Consequences	mean	3.3	1.0	3.2	0.8	-0.5	.59
	median	3.5	2.50-4.17	3.2	2.67-3.83		
Personal control	mean	4.0	0.7	3.9	0.7	-1.	.34
	median	4.1	4.08-4.54	4.0	3.50-4.45		
Treatment control	mean	3.6	0.7	3.6	0.6	-0.1	.92
	median	3.6	3.20-4.0	3.6	3.20-4.00		
Illness coherence	mean	3.6	0.8	3.6	0.9	-0.2	.83
	median	3.4	3.00-4.25	3.8	3.00-4.00		
Emotional	mean	2.9	1.0	2.7	1.0	-1.3	.20

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
representations							
	median	3.5	2.00-3.67	2.5	2.00-3.50		
Medication necessity	mean	3.8	0.9	4.1	0.8	-2.1	.04
	median	3.9	3.10-4.30	4.2	3.80-4.80		
Medication concerns	mean	3.1	0.8	2.8	1.0	-1.5	.14
	median	3.2	2.80-3.58	2.8	2.00-3.60		
Uncontrollable cause	mean	2.0	0.8	2.0	0.7	-0.2	.84
	median	2.0	1.00-2.67	2.0	1.60-2.50		
Lifestyle	mean	3.5	1.1	3.2	1.1	-1.1	.25
	median	4.0	2.63-4.00	3.0	2.50-4.00		
Risk factors	mean	2.0	1.1	2.0	1.1	-0.3	.76
	median	2.0	1.00-2.63	2.0	1.00-2.50		
Psychological attributions	mean	2.6	0.9	2.4	1.0	-1.3	.18
	median	2.8	2.00-3.25	2.3	1.75-3.00		
Hereditary	mean	3.6	1.0	3.4	1.4	-0.3	.79
	median	4.0	3.00-4.00	4.0	2.00-5.00		
Ageing	mean	2.6	1.1	2.7	1.3	-0.5	.61
	median	3.0	2.00-4.00	3.0	1.00-4.00		
Depression	mean	6.0	3.5	4.7	3.8	-1.8	.07
	median	6.0	3.00-9.00	4.0	2.00-7.00		
Anxiety	mean	6.2	4.0	5.2	4.3	-1.3	.20
	median	7.0	3.00-9.00	4.0	2.00-8.00		
Energy	mean	5.6	2.9	6.0	3.2	-0.8	.44
	median	6.0	4.00-7.00	6.0	3.25-8.00		
Positive well-being	mean	10.5	4.9	11.4	4.8	-0.8	.40
	median	10.0	7.00-15.00	12.0	8.00-15.00		
General diet	mean	4.4	2.5	4.9	2.0	-0.7	.51
	median	5.0	2.50-7.00	5.0	4.00-7.00		
Specific diet (fruit& veg)	mean	4.9	1.9	4.5	2.3	-0.4	.66
	median	5.0	3.75-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.5	2.2	4.3	2.0	-0.5	.63
	median	5.0	3.00-6.25	5.0	3.00-6.00		
Exercise	mean	2.4	2.3	2.2	2.2	-0.3	.76
	median	2.5	0.00-4.50	2.0	0.00-3.50		
Self-monitoring of blood glucose	mean	4.6	2.6	4.5	2.6	-0.1	.92
	median	5.3	2.38-7.00	5.0	2.00-7.00		
Foot care	mean	2.8	2.5	3.7	2.6	-1.7	.10
	median	3.0	0.00-4.50	3.5	1.00-7.00		
Medication adherence	mean	6.3	2.0	6.8	.9	-1.1	.26
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.1	1.3	4.2	1.2	-0.5	.60

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
	median	4.1	2.95-5.18	4.3	3.36-5.09		

Table A13: Mann-Whitney U Tests to explore the relationship between peripheral vascular disease and cognitions, well-being subscales and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	4.0	3.3	3.1	3.0	-1.5	.14
	median	3.0	2.00-6.00	2.5	0.75-5.00		
Timeline acute/chronic	mean	4.2	.8	4.4	.6	-1.5	.12
	median	4.3	3.83-4.83	4.7	4.00-4.83		
Timeline cyclical	mean	2.9	1.0	3.0	.9	-0.7	.46
	median	2.9	2.00-3.50	3.0	2.50-3.54		
Consequences	mean	3.2	.8	3.3	.8	-0.3	.78
	median	3.2	2.67-3.83	3.2	2.79-3.88		
Personal control	mean	4.0	.7	3.7	.7	-1.9	.06
	median	4.0	3.50-4.50	3.8	3.33-4.13		
Treatment control	mean	3.6	.6	3.5	.6	-0.6	.52
	median	3.6	3.20-4.00	3.6	3.00-4.00		
Illness coherence	mean	3.6	.9	3.6	.7	-0.3	.77
	median	3.8	3.00-4.20	3.6	3.15-4.00		
Emotional representations	mean	2.7	1.0	2.6	.9	-0.4	.69
	median	2.7	2.00-3.50	2.5	2.00-3.33		
Medication necessity	mean	4.1	.8	4.2	.6	-0.2	.88
	median	4.0	3.60-4.80	4.0	3.60-5.00		
Medication concerns	mean	2.9	1.0	2.5	1.0	-2.3	.02
	median	3.0	2.20-3.60	2.5	1.95-3.25		
Uncontrollable cause	mean	2.0	.7	2.1	.8	-0.3	.74
	median	2.0	1.50-2.40	2.0	1.33-2.67		
Lifestyle	mean	3.2	1.1	3.0	1.1	-1.1	.25
	median	3.0	2.50-4.00	3.0	2.00-4.00		
Risk factors	mean	2.0	1.1	1.9	.9	-0.3	.74
	median	2.0	1.00-2.50	2.0	1.00-2.50		
Psychological attributions	mean	2.4	1.0	2.3	.8	-0.3	.77
	median	2.3	1.75-3.25	2.3	1.75-3.00		
Hereditary	mean	3.4	1.4	3.2	1.3	-1.2	.23
	median	4.0	2.00-5.00	4.0	2.00-4.00		
Ageing	mean	2.7	1.3	2.6	1.3	-0.3	.75
	median	3.0	2.00-4.00	3.0	1.00-4.00		
Depression	mean	5.0	3.8	3.9	3.6	-1.6	.11
	median	4.0	2.00-7.60	3.5	0.25-6.00		
Anxiety	mean	5.4	4.3	4.5	3.9	-1.6	.11
	median	5.0	2.00-8.40	4.0	1.00-8.00		

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Energy	mean	5.9	3.2	6.4	2.9	-1.2	.25
	median	6.0	3.00-8.00	6.0	4.00-8.25		
Positive well-being	mean	11.0	4.8	12.6	4.8	-1.6	.10
	median	12.0	7.00-15.00	12.0	8.85-18.00		
General diet	mean	4.9	2.1	4.8	2.0	-1.0	.30
	median	5.0	4.00-7.00	5.0	3.50-6.00		
Specific diet (fruit& veg)	mean	4.7	2.2	3.9	2.5	-1.9	.06
	median	5.0	3.00-7.00	5.0	2.00-6.00		
Specific diet (fat consumption)	mean	4.3	2.1	4.7	1.8	-1.1	.29
	median	5.0	3.00-6.00	5.0	3.00-6.00		
Exercise	mean	2.2	2.2	2.4	2.3	-0.2	.84
	median	1.8	0.00-4.00	2.0	0.00-3.50		
Self-monitoring of blood glucose	mean	4.6	2.6	4.2	2.4	-1.1	.27
	median	5.5	2.64-7.00	4.5	2.00-7.00		
Foot care	mean	3.6	2.6	3.9	2.5	-0.5	.64
	median	3.5	1.00-6.50	3.5	2.00-6.75		
Medication adherence	mean	6.7	1.2	6.9	0.5	-0.4	.73
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.2	1.2	4.2	1.3	-0.5	.63
	median	4.3	3.30-5.18	4.3	1.34-3.45		

Table A14: Mann-Whitney U Tests to explore the relationship between respiratory conditions and cognitions, well-being subscales and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	3.7	3.2	4.4	3.3	-1.0	.30
	median	3.0	1.00-5.00	4.0	1.00-7.00		
Timeline acute/chronic	mean	4.2	.8	4.5	.6	-2.1	.03
	median	4.3	3.83-4.83	4.7	4.17-4.83		
Timeline cyclical	mean	2.8	.9	3.3	1.0	-2.1	.04
	median	2.8	2.00-3.50	3.3	2.38-4.00		
Consequences	mean	3.2	.8	3.6	.9	-2.4	.02
	median	3.2	2.67-3.83	3.7	3.00-4.33		
Personal control	mean	4.0	.7	3.7	.8	-1.7	.08
	median	4.0	3.50-4.50	3.7	3.17-4.33		
Treatment control	mean	3.6	.6	3.3	.7	-2.8	.01
	median	3.6	3.20-4.00	3.2	3.00-3.60		
Illness coherence	mean	3.6	.9	3.3	1.0	-1.6	.11
	median	3.8	3.20-4.20	3.6	2.60-4.00		
Emotional	mean	2.6	1.0	3.2	1.1	-3.0	.00

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
representations							
	median	2.5	2.00-3.46	3.3	2.50-4.00		
Medication necessity	mean	4.1	.7	4.1	.9	-0.6	.58
	median	4.0	3.65-4.80	4.1	3.60-5.00		
Medication concerns	mean	2.8	1.0	3.1	1.0	-1.7	.08
	median	2.8	2.00-3.40	3.2	2.60-3.80		
Uncontrollable cause	mean	2.0	.7	2.2	.7	-1.4	.15
	median	2.0	1.50-2.40	2.0	1.75-2.83		
Lifestyle	mean	3.2	1.1	3.3	.9	-0.2	.81
	median	3.0	2.50-4.00	3.0	2.75-4.00		
Risk factors	mean	2.0	1.0	1.9	1.1	-0.7	.49
	median	2.0	1.00-2.50	1.5	1.00-3.00		
Psychological attributions	mean	2.3	1.0	2.8	.9	-2.6	.01
	median	2.3	1.75-3.00	3.0	2.06-3.68		
Hereditary	mean	3.4	1.4	3.7	1.2	-1.0	.31
	median	4.0	2.00-4.00	4.0	2.25-4.75		
Ageing	mean	2.7	1.3	2.6	1.2	-0.4	.67
	median	3.0	1.00-4.00	3.0	2.00-4.00		
Depression	mean	4.6	3.6	6.1	4.0	-2.0	.04
	median	4.0	2.00-7.00	6.0	2.50-8.50		
Anxiety	mean	4.9	4.1	7.8	3.9	-3.5	.00
	median	4.0	1.00-8.00	8.0	7.00-9.00		
Energy	mean	6.1	3.2	5.0	3.0	-1.8	.07
	median	6.0	4.00-8.00	5.0	3.00-6.67		
Positive well-being	mean	11.6	4.7	9.3	5.2	-2.3	.02
	median	12.0	8.00-15.00	9.0	6.00-12.50		
General diet	mean	4.9	2.1	5.1	2.0	-0.7	.46
	median	5.0	4.00-7.00	5.5	4.00-7.00		
Specific diet (fruit& veg)	mean	4.5	2.3	5.0	2.0	-1.0	.31
	median	5.0	3.00-7.00	5.0	4.00-7.00		
Specific diet (fat consumption)	mean	4.3	2.1	4.8	1.6	-1.1	.29
	median	5.0	3.00-6.00	5.0	4.00-6.00		
Exercise	mean	2.3	2.3	1.9	2.0	-0.7	.46
	median	2.0	0.00-4.00	1.3	0.00-3.38		
Self-monitoring of blood glucose	mean	4.5	2.7	5.2	2.1	-1.2	.24
	median	5.0	2.00-7.00	6.0	3.50-7.00		
Foot care	mean	3.7	2.6	3.1	2.6	-1.3	.19
	median	3.5	1.00-6.88	2.5	0.50-6.00		
Medication adherence	mean	6.8	1.1	6.8	.7	-1.2	.22
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.2	1.2	4.2	1.2	-0.1	.93

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
	median	4.3	3.39-5.09	4.3	3.18-5.27		

Table A15: Mann-Whitney U Tests to explore the relationship between musculoskeletal conditions and cognitions, well-being subscales and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	3.8	3.1	4.0	3.6	-0.2	.87
	median	3.0	1.00-6.00	4.0	1.00-5.50		
Timeline acute/chronic	mean	4.2	.8	4.3	.7	-1.4	.15
	median	4.3	3.83-4.83	4.6	4.00-4.83		
Timeline cyclical	mean	2.8	.9	3.0	.9	-1.5	.13
	median	2.8	2.00-3.50	3.0	2.00-3.75		
Consequences	mean	3.3	.8	3.2	.8	-0.8	.41
	median	3.2	2.67-3.83	3.2	2.67-3.83		
Personal control	mean	4.0	.7	3.8	.7	-1.5	.13
	median	4.0	3.54-4.50	3.8	3.33-4.33		
Treatment control	mean	3.6	.6	3.4	.6	-2.3	.02
	median	3.6	3.20-4.00	3.4	3.00-3.80		
Illness coherence	mean	3.6	.9	3.5	1.0	-0.3	.74
	median	3.8	3.00-4.00	3.8	3.00-4.20		
Emotional representations	mean	2.7	1.0	2.6	1.0	-0.2	.82
	median	2.7	2.00-3.50	2.4	2.00-3.50		
Medication necessity	mean	4.1	.8	4.2	.7	-1.4	.15
	median	4.0	3.60-4.80	4.3	3.80-5.00		
Medication concerns	mean	2.8	1.0	2.8	1.0	-0.1	.93
	median	2.8	2.20-3.60	2.8	2.05-3.55		
Uncontrollable cause	mean	2.0	.7	2.1	.6	-0.8	.44
	median	2.0	1.50-2.50	2.0	1.67-2.50		
Lifestyle	mean	3.3	1.1	2.9	1.0	-2.0	.04
	median	3.5	2.50-4.00	3.0	2.00-4.00		
Risk factors	mean	2.1	1.1	1.8	1.0	-1.6	.12
	median	2.0	1.00-3.00	2.0	1.00-2.00		
Psychological attributions	mean	2.4	1.0	2.5	.9	-0.8	.40
	median	2.3	1.75-3.00	2.5	2.00-3.25		
Hereditary	mean	3.4	1.4	3.4	1.4	-0.2	.82
	median	4.0	2.00-4.00	4.0	2.00-5.00		
Ageing	mean	2.7	1.3	2.7	1.3	-0.2	.86
	median	3.0	1.00-4.00	3.0	1.25-4.00		
Depression	mean	4.7	3.8	4.8	3.5	-0.3	.76
	median	4.0	2.00-7.10	5.0	2.00-7.00		
Anxiety	mean	5.0	4.1	6.0	4.2	-1.6	.11
	median	4.0	2.00-8.00	6.0	2.20-9.00		
Energy	mean	6.1	3.1	5.7	3.3	-0.6	.52

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
	median	6.0	3.75-8.00	6.0	4.00-8.00		
Positive well-being	mean	11.4	4.9	11.2	4.6	-0.4	.71
	median	12.0	7.75-15.00	12.0	8.00-15.00		
General diet	mean	4.9	2.0	4.7	2.1	-0.7	.47
	median	5.5	4.00-7.00	5.0	3.50-6.75		
Specific diet (fruit& veg)	mean	4.6	2.3	4.5	2.2	-0.5	.65
	median	5.0	3.00-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.3	2.0	4.5	2.0	-0.8	.40
	median	5.0	3.00-6.00	5.0	4.00-6.00		
Exercise	mean	2.2	2.2	2.3	2.3	-0.2	.82
	median	1.8	0.00-3.50	2.0	0.00-4.00		
Self-monitoring of blood glucose	mean	4.5	2.6	4.9	2.6	-1.2	.22
	median	5.0	2.00-7.00	6.0	2.75-7.00		
Foot care	mean	3.8	2.5	3.1	2.7	-1.6	.12
	median	3.5	1.50-6.50	3.5	0.50-5.50		
Medication adherence	mean	6.8	1.0	6.7	1.3	-0.8	.43
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.2	1.2	4.2	1.1	-0.1	.91
	median	4.3	3.36-5.27	4.4	3.41-5.05		

Table A16: Mann-Whitney U Tests to explore the relationship between endocrine conditions and cognitions, well-being subscales and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	4.0	3.3	3.6	3.1	-0.8	.42
	median	3.0	1.00-6.00	3.0	1.00-5.00		
Timeline acute/chronic	mean	4.2	.8	4.2	.7	0.0	.98
	median	4.3	3.83-4.83	4.5	3.83-4.67		
Timeline cyclical	mean	2.9	1.0	2.9	.9	-0.2	.80
	median	3.0	2.00-3.50	3.0	2.00-3.50		
Consequences	mean	3.2	.8	3.3	.8	-1.2	.24
	median	3.2	2.58-3.73	3.3	2.67-4.00		
Personal control	mean	4.0	.7	3.9	.7	-1.2	.23
	median	4.0	3.50-4.50	3.9	3.38-4.33		
Treatment control	mean	3.6	.6	3.5	.6	-1.8	.07
	median	3.6	3.20-4.00	3.5	3.20-4.00		
Illness coherence	mean	3.6	1.0	3.6	.8	-0.3	.76
	median	3.8	3.00-4.20	3.8	3.40-4.00		
Emotional representations	mean	2.7	1.0	2.7	1.0	-0.2	.81
	median	2.5	2.00-3.50	2.6	2.00-3.50		
Medication necessity	mean	4.1	.8	4.1	.8	-0.6	.52

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
	median	4.2	3.70-4.80	4.0	3.60-4.80		
Medication concerns	mean	2.8	1.0	2.8	1.0	-0.2	.86
	median	2.8	2.20-3.50	2.8	2.00-3.60		
Uncontrollable cause	mean	2.0	.7	2.1	.6	-1.5	.13
	median	2.0	1.33-2.35	2.0	1.69-2.50		
Lifestyle	mean	3.3	1.1	3.0	1.0	-1.7	.08
	median	3.5	2.50-4.00	3.0	2.00-4.00		
Risk factors	mean	2.0	1.1	2.0	1.0	-0.6	.56
	median	2.0	1.00-2.50	2.0	1.00-3.00		
Psychological attributions	mean	2.3	1.0	2.5	.9	-1.1	.25
	median	2.3	1.31-3.00	2.5	2.00-3.25		
Hereditary	mean	3.3	1.4	3.5	1.3	-0.6	.57
	median	4.0	2.00-4.50	4.0	2.00-4.25		
Ageing	mean	2.6	1.3	2.8	1.3	-0.6	.55
	median	3.0	1.00-4.00	3.0	2.00-4.00		
Depression	mean	4.6	3.6	4.9	3.9	-0.3	.76
	median	4.0	2.00-7.00	4.0	1.88-7.40		
Anxiety	mean	5.2	4.0	5.2	4.5	-0.5	.61
	median	5.0	2.00-8.00	4.0	1.00-8.00		
Energy	mean	6.1	3.3	5.8	2.9	-0.7	.50
	median	6.0	3.00-8.75	6.0	4.00-8.00		
Positive well-being	mean	11.7	4.6	10.8	5.0	-1.2	.24
	median	12.0	8.00-15.00	12.0	7.00-15.00		
General diet	mean	4.8	2.1	5.0	2.1	-0.6	.53
	median	5.0	3.50-7.00	5.0	4.00-7.00		
Specific diet (fruit& veg)	mean	4.5	2.2	4.7	2.4	-0.9	.37
	median	5.0	3.00-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.4	2.0	4.3	2.1	-0.3	.73
	median	5.0	3.00-6.00	5.0	3.00-6.00		
Exercise	mean	2.3	2.2	2.2	2.3	-0.4	.69
	median	2.0	0.00-3.50	2.0	0.00-4.00		
Self-monitoring of blood glucose	mean	4.7	2.5	4.4	2.8	-0.3	.77
	median	5.0	3.00-7.00	5.0	2.00-7.00		
Foot care	mean	3.6	2.5	3.6	2.7	-0.1	.95
	median	3.5	1.13-5.88	3.5	1.00-7.00		
Medication adherence	mean	6.7	1.2	6.8	0.8	-0.8	.42
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.2	1.1	4.2	1.3	-0.1	.94
	median	4.4	3.45-5.09	4.3	3.18-5.29		

Table A17: Mann-Whitney U Tests to explore the relationship between genitourinary conditions and cognitions, well-being subscales and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	3.9	3.3	3.0	2.5	-1.0	.31
	median	3.0	1.00-6.00	3.5	1.00-5.00		
Timeline acute/chronic	mean	4.2	0.8	4.2	0.7	-0.2	.82
	median	4.5	3.83-4.83	4.4	3.92-4.83		
Timeline cyclical	mean	2.9	1.0	2.9	0.9	-0.4	.67
	median	3.0	2.00-3.50	3.1	2.00-3.56		
Consequences	mean	3.2	0.8	3.4	0.7	-0.7	.47
	median	3.2	2.67-3.83	3.3	2.67-4.04		
Personal control	mean	3.9	0.7	4.0	0.8	-0.6	.57
	median	4.0	3.50-4.40	4.0	3.33-4.83		
Treatment control	mean	3.6	0.6	3.5	0.6	-0.8	.44
	median	3.6	3.20-4.00	3.4	3.15-4.00		
Illness coherence	mean	3.6	0.9	3.8	0.9	-1.2	.23
	median	3.8	3.00-4.00	3.9	3.40-4.25		
Emotional representations	mean	2.7	1.0	2.6	1.0	-0.7	.46
	median	2.7	2.00-3.50	2.3	1.75-3.29		
Medication necessity	mean	4.1	0.8	4.3	0.6	-1.1	.26
	median	4.0	3.60-4.80	4.3	4.00-4.95		
Medication concerns	mean	2.9	1.0	2.6	0.8	-1.6	.12
	median	2.8	2.20-3.60	2.5	1.85-3.20		
Uncontrollable cause	mean	2.0	0.7	2.0	0.6	0.0	.97
	median	2.0	1.50-2.50	2.0	1.58-2.58		
Lifestyle	mean	3.2	1.1	3.1	1.2	-0.4	.71
	median	3.0	2.50-4.00	3.0	2.13-4.00		
Risk factors	mean	2.0	1.0	2.1	1.3	-0.2	.87
	median	2.0	1.00-2.50	1.3	1.00-3.00		
Psychological attributions	mean	2.4	1.0	2.2	0.9	-1.0	.31
	median	2.3	1.75-3.25	2.0	1.75-3.00		
Hereditary	mean	3.4	1.4	3.2	1.3	-1.1	.29
	median	4.0	2.00-5.00	3.0	2.00-4.00		
Ageing	mean	2.7	1.3	2.6	1.4	-0.2	.88
	median	3.0	1.00-4.00	2.0	1.00-4.00		
Depression	mean	4.7	3.7	4.6	3.7	-0.1	.88
	median	4.0	2.00-7.00	4.5	1.00-8.00		
Anxiety	mean	5.2	4.2	5.3	4.1	-0.2	.81
	median	4.0	2.00-8.00	6.5	1.00-9.00		
Energy	mean	6.0	3.2	6.0	3.3	-0.1	.96
	median	6.0	4.00-8.00	6.0	3.00-8.50		
Positive well-being	mean	11.3	4.9	11.6	4.3	-0.3	.79
	median	12.0	7.00-15.00	12.0	9.00-15.00		
General diet	mean	5.0	2.0	4.4	2.2	-1.3	.18
	median	5.0	4.00-7.00	5.0	3.00-6.00		

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Specific diet (fruit& veg)	mean	4.6	2.3	4.7	2.2	-0.2	.86
	median	5.0	3.00-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.3	2.1	4.6	1.8	-0.6	.57
	median	5.0	3.00-6.00	5.0	3.00-6.00		
Exercise	mean	2.4	2.3	1.3	1.5	-2.1	.04
	median	2.0	0.00-4.00	1.0	0.00-2.75		
Self-monitoring of blood glucose	mean	4.5	2.6	4.7	2.4	-0.1	.95
	median	5.0	2.00-7.00	5.0	3.50-7.00		
Foot care	mean	3.6	2.5	3.7	2.8	-0.2	.88
	median	3.5	1.00-6.00	3.8	0.50-7.00		
Medication adherence	mean	6.8	1.0	6.6	1.4	-0.8	.45
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	4.2	1.2	4.0	1.0	-1.0	.33
	median	4.3	3.36-5.27	4.3	3.64-4.68		

Table A18: Mann-Whitney U Tests to explore the relationship between other health conditions and cognitions, well-being subscales and diabetes self-care behaviours

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Identity	mean	3.9	3.3	4.1	3.1	-0.5	.60
	median	3.0	1.00-6.00	3.5	2.00-5.75		
Timeline acute/chronic	mean	4.2	0.8	4.2	0.6	-0.5	.60
	median	4.5	3.83-4.83	4.3	3.83-4.67		
Timeline cyclical	mean	2.9	0.9	2.9	1.0	0.0	.98
	median	3.0	2.00-3.50	3.0	2.50-3.50		
Consequences	mean	3.3	0.8	3.3	0.8	-0.3	.73
	median	3.3	2.67-3.83	3.3	2.83-3.83		
Personal control	mean	3.9	0.7	3.8	0.7	-0.9	.39
	median	4.0	3.50-4.50	4.0	3.00-4.33		
Treatment control	mean	3.6	0.6	3.5	0.6	-0.4	.68
	median	3.6	3.20-4.00	3.8	3.25-4.00		
Illness coherence	mean	3.6	0.9	3.7	0.9	-1.0	.30
	median	3.7	3.00-4.00	4.0	3.20-4.20		
Emotional representations	mean	2.7	1.0	2.6	1.0	-0.1	.90
	median	2.7	2.00-3.50	2.5	2.00-3.50		
Medication necessity	mean	4.1	0.8	3.9	0.9	-0.7	.50
	median	4.0	3.80-4.80	4.2	3.55-4.60		

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Medication concerns	mean	2.8	1.0	3.0	0.9	-0.7	.50
	median	2.8	2.00-3.60	2.8	2.40-3.40		
Uncontrollable cause	mean	2.0	0.7	1.9	0.6	-1.1	.28
	median	2.0	1.50-2.50	2.0	1.33-2.33		
Lifestyle	mean	3.2	1.1	3.2	1.0	0.0	.96
	median	3.0	2.50-4.00	3.3	2.50-4.00		
Risk factors	mean	2.0	1.1	1.9	0.9	-0.5	.64
	median	2.0	1.00-2.50	2.0	1.00-2.50		
Psychological attributions	mean	2.4	1.0	2.4	0.9	0.0	.96
	median	2.3	1.75-3.25	2.3	1.75-3.00		
Hereditary	mean	3.4	1.4	3.3	1.4	-0.7	.48
	median	4.0	2.00-5.00	4.0	2.00-4.00		
Ageing	mean	2.7	1.3	2.7	1.1	-0.1	.93
	median	3.0	1.00-4.00	3.0	2.00-4.00		
Depression	mean	4.8	3.8	4.9	3.5	-0.4	.69
	median	4.0	2.00-7.15	5.0	2.00-7.75		
Anxiety	mean	5.4	4.3	6.0	4.1	-1.0	.31
	median	4.0	2.00-8.20	6.0	3.00-9.00		
Energy	mean	6.0	3.3	5.4	2.6	-0.8	.41
	median	6.0	3.00-8.00	5.5	4.00-7.75		
Positive well-being	mean	11.4	5.0	10.3	3.9	-1.6	.11
	median	12.0	8.00-15.00	11.0	7.00-12.75		
General diet	mean	4.9	2.1	4.7	1.9	-0.9	.38
	median	5.0	4.00-7.00	5.0	3.50-6.00		
Specific diet (fruit& veg)	mean	4.5	2.3	4.4	2.4	-0.2	.87
	median	5.0	3.00-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.5	2.0	4.2	2.0	-0.8	.44
	median	5.0	3.00-6.00	4.0	3.00-6.00		
Exercise	mean	2.4	2.3	1.7	2.0	-1.8	.08
	median	2.0	0.00-4.00	1.0	0.00-3.00		
Self-monitoring of blood glucose	mean	4.6	2.6	4.5	2.8	-0.1	.94
	median	5.3	2.00-7.00	5.0	2.00-7.00		
Foot care	mean	3.6	2.6	3.7	2.4	-0.3	.75
	median	3.5	1.00-6.00	3.5	1.63-5.50		
Medication adherence	mean	6.7	1.2	6.9	0.4	-0.6	.54
	median	7.0	7.00-7.00	7.0	7.00-7.00		

Variables		Absent Average	SD/IQR	Present Average	SD/IQR	z	Sig
Global self-care	mean	4.2	1.2	4.0	1.1	-0.8	.41
	median	4.3	3.36-5.27	4.3	3.18-4.72		

Table A19: Mann-Whitney U Tests to explore the relationship between diabetes treatment regimens and cognitions, well-being subscales, and diabetes self-care behaviours

Variables		Diet/Oral Average	SD/IQR	Injections/combined Average	SD/IQR	z	Sig
Identity	mean	3.4	3.3	4.1	3.2	-1.9	.05
	median	2.0	1.00-5.00	4.0	2.00-6.00		
Timeline acute/chronic	mean	4.0	0.7	4.3	0.7	-3.5	.01
	median	4.2	3.50-4.67	4.5	4.00-4.83		
Timeline cyclical	mean	2.6	0.9	3.0	0.9	-2.4	.46
	median	2.8	2.00-3.00	3.0	2.25-3.75		
Consequences	mean	3.0	0.8	3.4	0.8	-3.5	.51
	median	2.8	2.33-3.67	3.3	2.83-4.00		
Personal control	mean	3.9	0.7	3.9	0.7	-0.3	.02
	median	4.0	3.67-4.48	4.0	3.42-4.45		
Treatment control	mean	3.6	0.7	3.6	0.6	-0.1	.65
	median	3.6	3.20-4.00	3.6	3.20-4.00		
Illness coherence	mean	3.8	0.9	3.5	0.9	-2.5	.62
	median	4.0	3.20-4.60	3.6	3.00-4.00		
Emotional representations	mean	2.6	1.0	2.7	1.0	-0.9	.67
	median	2.5	2.00-3.50	2.7	2.00-3.50		
Medication necessity	mean	3.7	0.8	4.3	0.7	-4.9	.15
	median	3.8	3.40-4.20	4.2	4.00-5.00		
Medication concerns	mean	2.9	1.0	2.8	1.0	-0.4	.43
	median	2.8	2.00-3.60	2.8	2.20-3.50		
Uncontrollable cause	mean	2.0	0.7	2.0	0.7	-0.7	.40
	median	2.0	1.63-2.33	2.0	1.50-2.50		
Lifestyle	mean	3.2	1.1	3.2	1.1	-0.3	.29
	median	3.0	2.38-4.00	3.0	2.50-4.00		
Risk factors	mean	2.0	1.0	2.0	1.1	-0.2	.91
	median	2.0	1.00-2.50	2.0	1.00-3.00		
Psychological attributions	mean	2.5	0.9	2.4	1.0	-0.6	.03
	median	2.5	1.75-3.25	2.3	1.75-3.25		
Hereditary	mean	3.3	1.4	3.5	1.4	-1.1	.00
	median	4.0	2.00-4.00	4.0	2.00-5.00		
Ageing	mean	2.9	1.3	2.6	1.3	-1.5	.03
	median	3.0	2.00-4.00	3.0	1.00-4.00		
Depression	mean	4.1	3.2	5.1	3.8	-1.8	.07
	median	3.5	2.00-6.00	5.0	2.00-8.00		
Anxiety	mean	4.5	3.7	5.6	4.4	-1.6	.11

Variables		Diet/Oral Average	SD/IQR	Injections/combined Average	SD/IQR	z	Sig
	median	4.0	1.00-8.00	5.0	2.00-9.00		
Energy	mean	6.8	3.0	5.6	3.2	-2.8	.01
	median	7.0	5.00-9.00	5.0	3.00-8.00		
Positive well-being	mean	12.0	4.6	11.1	4.9	-1.2	.22
	median	12.0	8.00-16.00	12.0	7.00-15.00		
General diet	mean	4.8	2.3	4.9	2.0	0.0	1.0
	median	5.5	3.50-7.00	5.0	4.00-7.00		
Specific diet (fruit& veg)	mean	4.7	2.2	4.5	2.3	-0.4	.72
	median	5.0	3.00-7.00	5.0	3.00-7.00		
Specific diet (fat consumption)	mean	4.2	2.1	4.5	2.0	-1.0	.31
	median	4.0	3.00-6.00	5.0	3.00-6.00		
Exercise	mean	2.5	2.4	2.2	2.2	-0.8	.41
	median	2.0	0.00-4.50	1.5	0.00-3.50		
Self-monitoring of blood glucose	mean	2.9	2.6	5.4	2.2	-6.8	.00
	median	2.5	0.00-5.25	7.0	4.00-7.00		
Foot care	mean	3.2	2.4	3.8	2.6	-1.7	.09
	median	3.5	1.00-5.00	3.5	1.00-7.00		
Medication adherence	mean	6.5	1.7	6.9	0.5	-1.5	.13
	median	7.0	7.00-7.00	7.0	7.00-7.00		
Global self-care	mean	3.9	1.2	4.4	1.2	-2.7	.01
	median	3.8	2.91-4.73	4.5	3.55-5.27		

Table A20: Baseline and follow-up univariate statistics for negative well-being and global diabetes self-care revised

Variables		Average	SD/IQR
Baseline			
Negative well-being	mean	3.4	3.1
	median	3.0	0.0-5.5
Global diabetes self-care revised	mean	3.8	1.4
	median	3.8	2.9-4.9
Follow-up			
Negative well-being	mean	3.3	3.1
	median	2.7	0.0-5.00
Global diabetes self-care revised	mean	3.9	1.3
	median	4.0	3.0-4.8

Table A21: Relationship between categorical socio-demographic variables and both negative well-being and global diabetes self-care revised

Variables		Male Average	SD/IQR	Female Average	SD/IQR	z	p value
Negative well-being	mean	2.9	2.9	4.2	3.2	-3.5	.00**
	median	2.0	0.0-5.0	4.0	2.0-6.3		
Global diabetes self-care revised	mean	3.9	1.4	3.6	1.4	-1.4	.17
	median	4.0	2.8-4.9	3.6	2.4-4.6		
Variables		White Average	SD/IQR	Non-White Average	SD/IQR	z	p value
Negative well-being	mean	3.4	3.1	3.9	3.1	-0.1	.99
	median	3.0	0.0-6.0	3.0	0.5-5.0		
Global diabetes self-care revised	mean	3.8	1.4	3.8	1.5	-0.2	.81
	median	3.9	2.9-4.8	3.8	2.9-5.3		

Table A22: Relationship between diabetes complications/health co-morbidities and both negative well-being and global diabetes self-care

	Condition Absent Average	SD/IQR	Condition present Average	SD/IQR	z	p
Neuropathy						
Negative well-being	3.5	3.14	2.77	2.9	-1.9	0.05
	3.0	1.0-5.7	2.00	2.0-5.0		
Global diabetes self-care revised	3.73	1.49	3.94	1.27	-0.96	0.34
	3.63	2.9-4.8	4.06	2.9-5.0		
Nephropathy						
Negative well-being	3.55	3.19	2.10	2.16	-2.76	0.01
	3.00	0.0-6.0	2.00	0.0-4.0		
Global diabetes self-care revised	3.86	1.45	3.61	1.21	-1.05	0.29
	3.88	2.9-5.0	3.75	2.6-4.6		
Retinopathy						
Negative well-being	3.62	3.15	2.41	2.67	-2.83	0.00
	3.00	1.0-6.0	1.00	0.0-5.0		
Global diabetes self-care revised	3.69	1.40	4.10	1.40	-1.76	0.08
	3.63	2.9-4.8	4.25	3.0-5.3		
Macrovascular disease						
Negative well-being	3.91	2.50	3.21	3.15	-1.58	0.11
	4.00	2.0-6.0	2.00	0.0-5.0		
Global diabetes self-care revised	3.70	1.72	3.82	1.38	-0.18	0.85
	3.69	2.4-5.1	3.88	2.9-4.8		
Peripheral vascular disease						
Negative well-being	3.32	3.06	3.09	3.25	-0.70	0.49
	3.00	0.0-5.0	3.00	0.0-5.0		
Global diabetes self-care revised	3.81	1.39	3.85	1.51	-0.37	0.71
	3.75	2.9-5.2	4.00	2.9-4.6		
Endocrine conditions						

	Condition Absent		Condition present			
	Average	SD/IQR	Average	SD/IQR	z	p
Negative well-being	3.27	3.01	3.21	3.14	-0.22	0.83
	3.00	0.0-5.0	2.00	0.0-6.0		
Global diabetes self-care revised	3.79	1.37	3.84	1.48	-0.43	0.67
	3.63	2.9-4.8	4.00	2.9-5.0		
Respiratory conditions						
Negative well-being	3.02	2.98	5.04	3.09	-3.28	0.00
	2.00	0.0-5.0	6.00	3.0-6.0		
Global diabetes self-care revised	3.83	1.41	3.69	1.44	-0.39	0.70
	3.88	2.9-4.8	3.63	2.7-4.9		
	Condition Absent		Condition present			
	Average	SD/IQR	Average	SD/IQR	z	p
Musculoskeletal conditions						
Negative well-being	3.05	2.99	3.88	3.21	-1.82	0.07
	2.00	0.0-5.0	4.00	1.0-6.0		
Global diabetes self-care revised	3.84	1.42	3.71	1.38	-0.80	0.42
	3.94	2.9-4.9	3.63	2.7-4.8		
Genitourinary conditions						
Negative well-being	3.31	3.08	2.74	2.86	-0.88	0.38
	3.00	0.0-5.0	2.00	0.0-5.1		
Global diabetes self-care revised	3.85	1.44	3.49	1.13	-1.30	0.19
	3.88	2.9-5.0	3.63	2.8-4.3		
Other health conditions						
Negative well-being	3.36	3.16	3.61	2.82	-0.80	0.42
	3.00	0.0-5.0	3.50	1.0-6.0		
Global diabetes self-care revised	3.83	1.45	3.59	1.23	-0.73	0.46
	3.88	2.9-5.0	3.75	3.0-4.4		

Table A23: Relationship between diabetes treatment regimens and both negative well-being and global diabetes self-care

		Diet/Oral		Injections/combined			
Variables		Average	SD/IQR	Average	SD/IQR	z	P
Depression	mean	2.6	2.6	3.6	3.2	-1.9	.06
	median	2.0	2.0-4.0	4.0	0.0-6.0		
Anxiety	mean	3.8	1.4	3.8	1.4	-0.1	.91
	median	3.9	3.0-4.8	3.8	2.9-5.0		

Table A24: Relationship between continuous diabetes outcomes and socio-demographic/ clinical characteristics and both negative well-being and global diabetes self-care revised

Variables	1	2
1. Global diabetes self-care revised		
2. Negative well-being	-0.03	
3. Age	0.12	-.26**
4. Diabetes duration	0.11	-0.02
5. Weight	-0.11	0.06
6. BMI	-.19**	.153*
7. Systolic	0.02	-0.07
8. Diastolic	0.00	-0.03
9. HbA1c	-.14*	.25**
10. eGFR	-0.05	.24**
11. Total cholesterol	0.00	0.10
12. N diabetes complications	0.08	-.24**
13. N diabetes co-morbidities	0.00	0.13

*p≤0.05, ** p≤0.01

Table A25: Relationship between illness and medication cognitions and both negative well-being and global diabetes self-care

Variables	1	2
1. Negative well-being	1.00	
2. Global diabetes self-care revised	-.03	
3. Identity	.46**	.06
4. Timeline acute/chronic	.08	.06
5. Timeline cyclical	.32**	-.05
6. Consequences	.40**	.09
7. Personal control	-.18**	.05
8. Treatment control	-.15*	.15*
9. Illness coherence	-.30**	.22**
10. Emotional representations	.57**	-.11
11. Medication necessity	.14*	.06
12. Medication concerns	.47**	-.19**
13. Uncontrollable cause	.19**	-.10
14. Risk factors	-.04	-.20**
15. Psychological attributions	.37**	-.09
16. Lifestyle	.12	-.14*
17. Hereditary	.14*	.06
18. Ageing	.02	-.19**

*p≤0.05, ** p≤0.01

Table A26: Relationship between well-being subscales and diabetes self-care behaviours including negative well-being and global diabetes self-care revised

Variables	1	2
1. Negative well-being	1.00	
2. Global diabetes self-care revised	-.03	
3. General diet	-.06	.63**
4. Specific diet (fruit & veg)	.00	.54**
5. Specific diet (saturated fat)	.03	.34**
6. Exercise	-.10	.62**
7. Self-monitoring of blood glucose	.13*	.14*
8. Foot care	.04	.69**
9. Medication adherence	-.04	.09
10. Energy	-.64**	.26**
11. Positive well-being	-.58**	.26**
12. Depression	.77**	-.14*
13. Anxiety	.87**	-.05

Table A27: Means and standard deviations of changes in negative well-being and global diabetes self-care over 6 months

Variables	Possible range		Average	SD/IQR
Negative well-being	0 to 12	mean	0.0	2.4
		median	0.0	-1.0 to 1.0
Global diabetes self-care revised	0-7	mean	0.1	1.04
		median	0.0	-0.5 to 0.5

12.4 ***Statistical Appendix B***

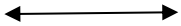
Abbreviation/Symbol	Definition
B_WBQ(N)	Used to represent questionnaire item numbers from the WBQ e.g. B_WBQ1
B_IP(N)	Used to represent questionnaire item numbers from the IPQ-R e.g. B_IP1
B_BMQ(N)	Used to represent questionnaire item numbers from the BMQ e.g. B_BMQ1
B_SDSCA (N)	Used to represent questionnaire item number from SDSCA e.g. B_SDSCA1
	Indicates the direction of effect
	Indicates that variables are assumed to co-vary but the direction of effect is not explicitly stated

Table B1: Parameter estimates for baseline timeline cyclical measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline timeline cyclical	→	B_IP29	1.00		.77		.00	1.00	1.00	...
Baseline timeline cyclical	→	B_IP30	.96	.09	.76	.00	.07	.83	1.11	.00
Baseline timeline cyclical	→	B_IP31	.91	.10	.69	.00	.11	.69	1.14	.00
Baseline timeline cyclical	→	B_IP32	.90	.09	.72	.00	.10	.72	1.10	.00

Table B2: Parameter estimates for baseline timeline acute/chronic re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline timeline acute/chronic	→	Baseline timeline acute/chronic positive	1.00		.85		.00	1.00	1.00	...
Baseline timeline acute/chronic	→	Baseline timeline acute/chronic reverse	.58	.13	.69	.00	.24	.25	1.12	.00
Baseline timeline acute/chronic positive	→	B_IP2	1.00		.81	.00	.00	1.00	1.00	...
Baseline timeline acute/chronic positive	→	B_IP3	1.12	.06	.95	.00	.11	.96	1.38	.00
Baseline timeline acute/chronic positive	→	B_IP18	.40	.10	.27	.00	.12	.18	.66	.00
Baseline timeline acute/chronic	→	B_IP5	1.09	.07	.86	.00	.14	.84	1.41	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
positive										
Baseline timeline acute/chronic reverse	→	B_IP1	1.00		.60	.00	.00	1.00	1.00	...
Baseline timeline acute/chronic reverse	→	B_IP4	1.14	.21	.75	.00	.39	.64	2.20	.00

Table B3: Parameter estimates for baseline personal control re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline personal control	→	Baseline personal control positive	1.00		.69		.00	1.00	1.00	...
Baseline personal control	→	Baseline personal control reverse	1.51	.37	.82	.00	.56	.87	2.93	.00
Baseline personal control positive	→	B_IP12	1.00		.67	.00	.00	1.00	1.00	...
Baseline personal control positive	→	B_IP13	.86	.11	.67	.00	.16	.63	1.26	.00
Baseline personal control positive	→	B_IP16	1.09	.14	.67	.00	.14	.82	1.36	.00
Baseline personal control reverse	→	B_IP15	1.00		.77	.00	.00	1.00	1.00	...
Baseline personal control reverse	→	B_IP17	.92	.17	.66	.00	.21	.56	1.37	.00
Baseline personal control positive	→	B_IP14	1.07	.13	.70	.00	.20	.76	1.54	.00

Table B4: Parameter estimates for baseline treatment control re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		Factor loadings								
Baseline treatment control	→	Baseline treatment control reverse	.52	.27	.65	.06	.76	.09	2.00	.00
Baseline treatment control	→	Baseline treatment control positive	1.00		.86		.00	1.00	1.00	...
Baseline treatment control reverse	→	B_IP23	1.94	.71	.92	.01	1.51	.91	7.18	.00
Baseline treatment control reverse	→	B_IP19	1.00		.41		.00	1.00	1.00	...
Baseline treatment control positive	→	B_IP22	.57	.15	.48	.00	.33	.25	1.25	.00
Baseline treatment control positive	→	B_IP21	1.00		.64		.00	1.00	1.00	...

Table B5: Parameter estimates for baseline consequences re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		Factor loadings								
Baseline consequences	→	Baseline health threat	1.00		.77		.00	1.00	1.00	...
Baseline consequences	→	Baseline social functioning	1.84	.59	.91	.00	.58	1.39	2.53	.00
Baseline health threat	→	B_IP6	1.00		.42		.00	1.00	1.00	...
Baseline health threat	→	B_IP8	1.15	.37	.43	.00	.41	.82	1.63	.00
Baseline social functioning	→	B_IP9	1.00		.58		.00	1.00	1.00	...

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline social functioning	→	B_IP10	1.11	.17	.60	.00	.20	.95	1.32	.00
Baseline social functioning	→	B_IP11	1.57	.24	.82	.00	.24	1.37	1.85	.00

Table B6: Parameter estimates for baseline emotional representations re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Emotional representation	→	Depression/Anger	1.00		.94		.00	1.00	1.00	...
Emotional representation	→	Anxiety	0.50	.07	.79	.00	.08	.34	.66	.00
Depression/Anger	→	B_IP33	1.00		.90		.00	1.00	1.00	...
Depression/Anger	→	B_IP34	1.04	.04	.98	.00	.04	.97	1.12	.00
Depression/Anger	→	B_IP35	0.84	.05	.77	.00	.06	.73	.96	.00
Anxiety	→	B_IP36	1.00		.55		.00	1.00	1.00	...
Anxiety	→	B_IP37	1.69	.18	.98	.00	.23	1.39	2.31	.00
Anxiety	→	B_IP38	1.33	.15	.81	.00	.20	1.05	1.81	.00

Table B7: Parameter estimates for baseline illness coherence re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		Factor loadings								
Baseline illness coherence	→	B_IP25	1.00		.77		.00	1.00	1.00	...
Baseline illness coherence	→	B_IP26	1.12	.08	.89	.00	.08	.97	1.29	.00
Baseline illness coherence	→	B_IP27	1.02	.08	.83	.00	.08	.88	1.20	.00
Baseline illness coherence	→	B_IP28	.61	.07	.55	.00	.09	.42	.77	.00

Table B8: Parameter estimates for baseline IPQ-R re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		Factor loadings								
Baseline timeline acute/chronic	→	Baseline timeline acute/chronic positive	1.00		.81		.00	1.00	1.00	...
Baseline timeline acute/chronic	→	Baseline timeline/acute chronic reverse	.80	.19	.74	.00	.45	.30	1.69	.00
Baseline consequences	→	Baseline threat	1.00		.73		.00	1.00	1.00	...
Baseline consequences	→	Baseline social	2.17	.58	.92	.00	1.46	1.05	5.27	.00
Baseline personal control	→	Baseline personal control positive	1.00		.69		.00	1.00	1.00	...
Baseline personal control	→	Baseline personal control reverse	1.55	.31	.83	.00	.59	.79	2.88	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline emotional representations	→	Baseline depression/anger	1.00		.94		.00	1.00	1.00	...
Baseline emotional representations	→	Baseline anxiety	.57	.07	.85	.00	.09	.38	.75	.00
Baseline timeline acute/chronic positive	→	B_IP2	1.00		.76		.00	1.00	1.00	...
Baseline timeline acute/chronic positive	→	B_IP3	1.22	.09	.95	.00	.17	1.00	1.65	.00
Baseline timeline acute/chronic positive	→	B_IP5	1.19	.09	.88	.00	.21	.90	1.70	.00
Baseline timeline/acute chronic reverse	→	B_IP1	1.00		.62		.00	1.00	1.00	...
Baseline timeline/acute chronic reverse	→	B_IP4	1.10	.19	.79	.00	.38	.60	2.12	.00
Baseline timeline cyclical	→	B_IP29	1.00		.77		.00	1.00	1.00	...
Baseline timeline cyclical	→	B_IP30	.98	.09	.78	.00	.07	.84	1.13	.00
Baseline timeline cyclical	→	B_IP31	.93	.10	.71	.00	.11	.71	1.16	.00
Baseline timeline cyclical	→	B_IP32	.95	.10	.75	.00	.09	.78	1.13	.00
Baseline social	→	B_IP9	1.00		.60		.00	1.00	1.00	...
Baseline social	→	B_IP10	1.18	.18	.64	.00	.23	.85	1.79	.00
Baseline social	→	B_IP11	1.31	.19	.69	.00	.22	1.01	1.86	.00
Baseline threat	→	B_IP6	1.00		.41		.00	1.00	1.00	...
Baseline threat	→	B_IP8	1.60	.43	.55	.00	1.15	.65	4.04	.00
Baseline personal control positive	→	B_IP12	1.00		.72		.00	1.00	1.00	...
Baseline personal control positive	→	B_IP13	.79	.10	.64	.00	.15	.53	1.13	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline personal control positive	→	B_IP14	1.01	.12	.70	.00	.19	.70	1.42	.00
Baseline personal control positive	→	B_IP16	1.06	.13	.67	.00	.17	.70	1.38	.00
Baseline personal control reverse	→	B_IP15	1.00		.79		.00	1.00	1.00	...
Baseline personal control reverse	→	B_IP17	.87	.13	.66	.00	.20	.54	1.31	.00
Baseline illness coherence	→	B_IP25	1.00		.80		.00	1.00	1.00	...
Baseline illness coherence	→	B_IP26	1.04	.08	.85	.00	.09	.85	1.22	.00
Baseline illness coherence	→	B_IP27	.98	.08	.83	.00	.08	.84	1.14	.00
Baseline illness coherence	→	B_IP28	.53	.08	.49	.00	.10	.33	.73	.00
Baseline depression/anger	→	B_IP33	1.00		.90		.00	1.00	1.00	...
Baseline depression/anger	→	B_IP34	1.03	.05	.96	.00	.04	.96	1.12	.00
Baseline depression/anger	→	B_IP35	.84	.06	.77	.00	.07	.71	.99	.00
Baseline anxiety	→	B_IP36	1.00		.59		.00	1.00	1.00	...
Baseline anxiety	→	B_IP37	1.55	.16	.95	.00	.22	1.25	2.10	.00
Baseline anxiety	→	B_IP38	1.23	.13	.82	.00	.20	.93	1.69	.00
Baseline timeline acute/chronic positive	→	B_IP18	.53	.12	.33	.00	.14	.31	.84	.00
Baseline Identity	→	Baseline identity parcelled	1.00		.89		.00	1.00	1.00	...
Baseline timeline acute/chronic positive	→	B_IP6	.57	.09	.42	.00	.11	.32	.76	.00

Table B9: Parameter estimates for baseline BMQ measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline medication necessity	→	B_BMQ1	1.00		.67		.00	1.00	1.00	...
Baseline medication necessity	→	B_BMQ3	1.33	.13	.80	.00	.21	1.00	1.79	.00
Baseline medication necessity	→	B_BMQ4	1.18	.11	.80	.00	.20	.84	1.62	.00
Baseline medication necessity	→	B_BMQ7	1.14	.11	.82	.00	.15	.91	1.50	.00
Baseline medication necessity	→	B_BMQ10	.73	.09	.56	.00	.15	.47	1.06	.00
Baseline medication concerns	→	B_BMQ2	1.00		.72		.00	1.00	1.00	...
Baseline medication concerns	→	B_BMQ5	.99	.10	.72	.00	.11	.78	1.22	.00
Baseline medication concerns	→	B_BMQ6	.66	.09	.53	.00	.10	.47	.86	.00
Baseline medication concerns	→	B_BMQ8	.87	.10	.65	.00	.08	.71	1.04	.00
Baseline medication concerns	→	B_BMQ9	1.03	.10	.77	.00	.10	.85	1.25	.00

Table B10: Parameter estimates for baseline medication necessity measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline medication necessity	→	B_BMQ1	1.00		.67		.00	1.00	1.00	...
Baseline medication necessity	→	B_BMQ3	1.34	.13	.81	.00	.21	1.02	1.83	.00
Baseline medication necessity	→	B_BMQ4	1.20	.11	.81	.00	.20	.85	1.69	.00
Baseline medication necessity	→	B_BMQ7	1.12	.11	.78	.00	.14	.88	1.45	.00
Baseline medication necessity	→	B_BMQ10	.73	.10	.56	.00	.14	.46	1.06	.00

Table B11: Parameter estimates for baseline medication concerns measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline medication concerns	→	B_BMQ2	1.00		.73		.00	1.00	1.00	...
Baseline medication concerns	→	B_BMQ5	.99	.10	.72	.00	.11	.79	1.24	.00
Baseline medication concerns	→	B_BMQ6	.66	.09	.53	.00	.09	.49	.86	.00
Baseline medication concerns	→	B_BMQ8	.86	.10	.65	.00	.08	.71	1.03	.00
Baseline medication concerns	→	B_BMQ9	1.04	.10	.77	.00	.10	.88	1.25	.00

Table B12: Parameter estimates for baseline WBQ measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline depression	→	B_WBQ1	1		0.51		0	1	1	...
Baseline depression	→	B_WBQ2	1.38	0.19	0.67	.00	0.26	1	2.07	.00
Baseline depression	→	B_WBQ3	0.95	0.15	0.54	.00	0.15	0.69	1.32	.00
Baseline depression	→	B_WBQ4	1.36	0.19	0.65	.00	0.17	1.09	1.77	.00
Baseline depression	→	B_WBQ5	1.41	0.19	0.67	.00	0.3	0.94	2.19	.00
Baseline depression	→	B_WBQ6	1.28	0.17	0.71	.00	0.2	0.98	1.75	.00
Baseline anxiety	→	B_WBQ7	1		0.79		0	1	1	...
Baseline anxiety	→	B_WBQ8	0.88	0.07	0.76	.00	0.07	0.73	1.02	.00
Baseline anxiety	→	B_WBQ9	1.08	0.08	0.8	.00	0.06	0.97	1.22	.00
Baseline anxiety	→	B_WBQ10	0.92	0.07	0.77	.00	0.08	0.76	1.09	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline anxiety	→	B_WBQ11	0.65	0.08	0.52	.00	0.1	0.46	0.87	.00
Baseline anxiety	→	B_WBQ12	0.62	0.09	0.44	.00	0.12	0.39	0.85	.00
Baseline energy	→	B_WBQ13	1		0.7		0	1	1	...
Baseline energy	→	B_WBQ14	0.95	0.1	0.67	.00	0.12	0.73	1.22	.00
Baseline energy	→	B_WBQ15	0.98	0.1	0.69	.00	0.12	0.77	1.23	.00
Baseline energy	→	B_WBQ16	1.2	0.12	0.76	.00	0.1	1.01	1.41	.00
Baseline positive well-being	→	B_WBQ17	1		0.82		0	1	1	...
Baseline positive well-being	→	B_WBQ18	0.97	0.07	0.8	.00	0.06	0.86	1.1	.00
Baseline positive well-being	→	B_WBQ19	1.07	0.08	0.78	.00	0.07	0.95	1.23	.00
Baseline positive well-being	→	B_WBQ20	0.88	0.07	0.71	.00	0.09	0.71	1.08	.00
Baseline positive well-being	→	B_WBQ21	0.9	0.07	0.73	.00	0.09	0.75	1.09	.00
Baseline positive well-being	→	B_WBQ22	0.98	0.07	0.8	.00	0.07	0.85	1.13	.00

Table B13: Parameter estimates for baseline depression measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline depression	→	B_WBQ1	1.00		.62		.00	1.00	1.00	...
Baseline depression	→	B_WBQ2	.96	.14	.57	.00	.21	.66	1.49	.00
Baseline depression	→	B_WBQ3	.87	.12	.61	.00	.14	.63	1.19	.00
Baseline depression	→	B_WBQ4	1.26	.14	.73	.00	.14	1.02	1.60	.00
Baseline depression	→	B_WBQ5	.96	.14	.56	.00	.22	.64	1.52	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline depression	→	B_WBQ6	1.06	.13	.71	.00	.15	.79	1.41	.00

Table B14: Parameter estimates for baseline anxiety measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline anxiety	→	B_WBQ7	1.00		.80		.00	1.00	1.00	...
Baseline anxiety	→	B_WBQ8	.89	.07	.78	.00	.07	.75	1.04	.00
Baseline anxiety	→	B_WBQ9	1.08	.08	.81	.00	.06	.97	1.20	.00
Baseline anxiety	→	B_WBQ10	.89	.07	.76	.00	.08	.75	1.05	.00
Baseline anxiety	→	B_WBQ11	.59	.08	.48	.00	.09	.41	.78	.00
Baseline anxiety	→	B_WBQ12	.56	.09	.40	.00	.11	.36	.78	.00

Table B15: Parameter estimates for baseline depression re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline depression	→	Baseline depression positive	1.49	.30	.85	.00	.35	.98	2.35	.00
Baseline depression	→	Baseline depression reverse	1.00		.73	.00	.00	1.00	1.00	...
Baseline depression reverse	→	B_WBQ4	1.29	.14	.76	.00	.15	1.05	1.63	.00
Baseline depression reverse	→	B_WBQ1	1.00		.64	.00	.00	1.00	1.00	...

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline depression positive	→	B_WBQ5	.99	.13	.76	.00	.15	.73	1.32	.00
Baseline depression reverse	→	B_WBQ3	.85	.11	.61	.00	.14	.60	1.16	.00
Baseline depression reverse	→	B_WBQ6	1.05	.13	.72	.00	.15	.77	1.39	.00
Baseline depression positive	→	B_WBQ2	1.00		.78		.00	1.00	1.00	...

Table B16: Parameter estimates for baseline negative well-being re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline negative well-being	→	B_WBQ2	1.00		.71		.00	1.00	1.00	...
Baseline negative well-being	→	B_WBQ5	1.11	.10	.78	.00	.10	.95	1.33	.00
Baseline negative well-being	→	B_WBQ8	.96	.10	.71	.00	.12	.74	1.24	.00
Baseline negative well-being	→	B_WBQ9	1.31	.12	.83	.00	.13	1.10	1.65	.00

Table B17: Parameter estimates for baseline global diabetes self-care measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline global diabetes self-care	→	General diet	1.00		.66		.00	1.00	1.00	...
Baseline global diabetes self-care	→	Exercise	.87	.20	.46	.00	.25	.43	1.41	.00
Baseline global diabetes self-care	→	SMBG	.37	.18	.22	.04	.22	.06	.86	.03

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline global diabetes self-care	→	Foot care	.72	.20	.47	.00	.22	.33	1.18	.00
General diet	→	B_SDSCA1	1.00		.94	.00	.00	1.00	1.00	...
General diet	→	B_SDSCA2	.93	.09	.87	.00	.10	.71	1.12	.00
Exercise	→	B_SDSCA5	1.00		1.00	.00	.00	1.00	1.00	...
Exercise	→	B_SDSCA6	.60	.14	.63	.00	.15	.30	.86	.00
SMBG	→	B_SDSCA7	1.00		.79		.00	1.00	1.00	...
SMBG	→	B_SDSCA8	1.24	.39	.96	.00	.68	.54	3.47	.00
Foot care	→	B_SDSCA9	1.00		.73		.00	1.00	1.00	...
Foot care	→	B_SDSCA10	1.23	.26	.82	.00	.39	.78	2.30	.00
Baseline global diabetes self-care	→	B_SDSCA3	.92	.20	.53	.00	.23	.49	1.43	.00
Baseline global diabetes self-care	→	B_SDSCA4	.42	.14	.28	.00	.16	.14	.79	.00
Baseline global diabetes self-care	→	B_SDSCA12	.11	.07	.14	.10	.09	-.01	.31	.09

Table B18: Parameter estimates for baseline global diabetes self-care re-specified measurement model

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Factor loadings							
Baseline global diabetes self-care	→	General diet	1.00		.67		.00	1.00	1.00	...
Baseline global diabetes self-care	→	Exercise	.85	.20	.48	.00	.24	.45	1.40	.00
Baseline global diabetes self-care	→	Foot care	.63	.19	.44	.00	.20	.27	1.09	.00
General diet	→	B_SDSCA2	.95	.09	.88	.00	.09	.77	1.15	.00
Exercise	→	B_SDSCA5	1.00		.94	.00	.00	1.00	1.00	...

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Exercise	→	B_SDSCA6	.67	.14	.66	.00	.14	.35	.93	.00
Baseline global diabetes self-care	→	B_SDSCA3	.97	.21	.56	.00	.28	.56	1.56	.00
Baseline global diabetes self-care	→	B_SDSCA4	.39	.13	.26	.00	.16	.10	.73	.01
Foot care	→	B_SDSCA9	1.00		.70		.00	1.00	1.00	...
Foot care	→	B_SDSCA10	1.33	.30	.85	.00	.50	.82	2.56	.00
General diet	→	B_SDSCA1	1.00		.94		.00	1.00	1.00	...

12.5 ***Statistical Appendix C***

Key to aid with the interpretation of Tables C1 to C17

Abbreviation/Symbol	Definition
WBQ(N)	Used to represent questionnaire item numbers from the WBQ (e.g. WBQ1)
IP(N)	Used to represent questionnaire item numbers from the IPQ-R (e.g. IP1)
BMQ(N)	Used to represent questionnaire item numbers from the BMQ (e.g. BMQ1)
SDSCA (N)	Used to represent questionnaire item number from SDSCA (e.g. SDSCA1)
→	Indicates the direction of effect
↔	Indicates that variables are assumed to co-vary but the direction of effect is not explicitly stated
B_	Baseline
F_	Follow-up

Table C1: Depression's directional effect on timeline cyclical model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline depression	→	Follow-up depression	.94	.17	.93	.00	.18	.64	1.37	.00
Baseline depression	→	Follow-up timeline cyclical	.32	.14	.21	.03	.19	.05	.81	.03
Baseline timeline cyclical	→	Follow-up timeline cyclical	.46	.08	.56	.00	.10	.25	.64	.00

Table C2: Depression and consequences auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline consequences	→	Follow-up consequences	1.28	.54	.82	.02	.97	.56	3.97	.00
Baseline depression	→	Follow-up depression	.99	.17	.95	.00	.22	.67	1.53	.00

Table C3: Depression and personal control auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline personal control	→	Follow-up personal control	.63	.17	.80	.00	.17	.47	.81	.00
Baseline depression	→	Follow-up depression	.87	.15	.95	.00	.17	.71	1.04	.00

Table C4: Depression and illness coherence auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline depression	→	Follow-up depression	.93	.16	.92	.00	.18	.76	1.11	.00
Baseline illness coherence	→	Follow-up illness coherence	.65	.10	.57	.00	.09	.56	.73	.00

Table C5: Depression and medication necessity auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline depression	→	Follow-up depression	.95	.17	.93	.00	.17	.80	1.13	.00
Baseline medication necessity	→	Follow-up medication necessity	.75	.12	.67	.00	.22	.57	.99	.00

Table C6: Depression and medication concerns auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline depression	→	Follow-up depression	.96	.17	.92	.00	.19	.79	1.16	.00
Baseline medication concerns	→	Follow-up medication concerns	.80	.10	.77	.00	.08	.71	.88	.00

Table C7: Anxiety's directional effect on timeline cyclical model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline timeline cyclical	→	Follow-up timeline cyclical	.41	.08	.50	.00	.10	.21	.60	.00
Baseline anxiety	→	Follow-up anxiety	.83	.09	.78	.00	.09	.66	1.02	.00
Baseline anxiety	→	Follow-up timeline cyclical	.26	.08	.27	.00	.09	.11	.47	.01

Table C8: Anxiety and illness coherence auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline illness coherence	→	Follow-up illness coherence	.66	.10	.59	.00	.09	.57	.75	.00
Baseline anxiety	→	Follow-up anxiety	.84	.09	.79	.00	.08	.77	.93	.00

Table C9: Anxiety and medication necessity auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline medication necessity	→	Follow-up medication necessity	.74	.12	.67	.00	.21	.55	.97	.00
Baseline anxiety	→	Follow-up anxiety	.85	.09	.79	.00	.08	.77	.93	.00

Table C10: Anxiety's directional effect on medication concerns model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline medication concerns	→	Follow-up medication concerns	.70	.12	.64	.00	.11	.59	.81	.00
Baseline anxiety	→	Follow-up anxiety	.84	.09	.79	.00	.09	.76	.93	.00
Baseline anxiety	→	Follow-up medication concerns	.26	.13	.19	.04	.14	.13	.39	.00

Table C11: Timeline cyclical and global diabetes self-care auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline global diabetes self-care revised	→	Follow-up global diabetes self-care revised	.60	.12	.77	.00	.15	.44	.72	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.50	.09	.67	.00	.10	.40	.60	.00

C12: Personal control's directional effect on global diabetes self-care model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline personal control	→	Follow-up personal control	.56	.18	.80	.00	.21	.24	1.04	.00
Baseline global diabetes self- care	→	Follow-up global diabetes self-care	.69	.14	.82	.00	.26	.27	1.25	.01
Baseline personal control	→	Follow-up global diabetes self-care	-.67	.25	-.32	.01	.47	-1.82	-.04	.03

Table C13: Depression and global diabetes self-care auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline depression	→	Follow-up depression	.96	.18	.97	.00	.23	.61	1.51	.00
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.69	.13	.83	.00	.18	.29	1.02	.00

C14a: Depression's directional effect on timeline cyclical-model fit and parameter estimates with equality constraints applied

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Depression and timeline cyclical (N=170)									
Auto-regressive model	251.82	163	.00	.06	.94	.08	345.82		
Timeline cyclical to depression	251.80	162	.00	.06	.94	.08	347.80	.02	.89
Depression to timeline cyclical	245.41	162	.00	.06	.94	.07	341.41	6.40	.01
Cross-lagged	245.41	161	.00	.06	.94	.07	343.41	.01	.93

*$\Delta\chi^2$ between depression to timeline cyclical and cross lagged

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline depression	→	Follow-up depression	1.06	.11	.92	.00	.12	.87	1.35	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.55	.08	.55	.00	.11	.32	.75	.00
Baseline depression	→	Follow-up timeline cyclical	.43	.17	.23	.01	.20	.10	.91	.01

Table C14b: Anxiety's directional effect on timeline cyclical-model fit and parameter estimates with equality constraints applied

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Anxiety and timeline cyclical (N=171)									
Auto-regressive model	266.233	165	0	0.06	0.94	.08	356.23		
Timeline cyclical to anxiety	265.712	164	0	0.06	0.94	.08	357.71	.52	.47
Anxiety to timeline cyclical	255.325	164	0	0.057	0.95	.07	347.33	10.91	.00
Cross-lagged *	254.751	163	0	0.058	0.95	.07	348.75	.57	.45

*$\Delta\chi^2$ between anxiety to timeline cyclical and cross lagged

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			<u>Direct effects</u>							
Baseline timeline cyclical	→	Follow-up timeline cyclical	.51	.09	.50	.00	.11	.40	.61	.00
Baseline anxiety	→	Follow-up anxiety	.80	.06	.78	.00	.07	.73	.87	.00
Baseline anxiety	→	Follow-up timeline cyclical	.29	.09	.28	.00	.09	.21	.38	.00

Table C14c: Anxiety's directional effect on medication concerns-model fit and parameter estimates with equality constraints applied

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Anxiety and medication concerns (N=162)									
Auto-regressive model	343.07	204	.00	.07	.92	.08	441.07		
Medication concerns to anxiety	339.56	203	.00	.07	.92	.08	439.56	3.51	.06
Anxiety to medication concerns	339.33	203	.00	.07	.92	.08	439.33	3.74	.05
Cross-lagged*	336.35	202	.00	.06	.92	.07	438.35	2.99	.08

*$\Delta\chi^2$ between anxiety to medication concerns and cross-lagged

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline medication concerns	→	Follow-up medication concerns	.62	.09	.65	.00	.09	.54	.72	.00
Baseline anxiety	→	Follow-up anxiety	.80	.06	.79	.00	.07	.73	.86	.00
Baseline anxiety	→	Follow-up medication concerns	.22	.11	.18	.05	.12	.10	.34	.00

Table C14d: Personal control's directional effect on global diabetes self-care-model fit and parameter estimates with equality constraints applied

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Personal control and global diabetes self-care (N=153)									
Auto-regressive model	480.03	334	.00	.05	.91	.08	624.03		
Personal control to global diabetes self-care	468.55	333	.00	.05	.92	.08	614.55	11.49	.00
Global diabetes self-care to personal control	478.15	333	.00	.05	.91	.08	624.15	1.89	.17
Cross-lagged*	466.99	332	.00	.05	.92	.08	614.99	1.56	.21

*$\Delta\chi^2$ between personal control to global diabetes self-care and cross lagged

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline personal control	→	Follow-up personal control	.70	.13	.77	.00	.15	.62	.91	.00
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.77	.10	.83	.00	.19	.49	.87	.00
Baseline personal control	→	Follow-up global diabetes self-care	-.91	.31	-.34	.00	.41	-1.35	-.53	.00

Table C15: Depression, anxiety, cognitions, and global diabetes self-care-model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline personal control	→	Follow-up global diabetes self-	-.52	.20	-.25	.01	.30	-1.14	.01	.05

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		care								
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.99	.19	.83	.00	.35	.16	1.59	.01
Baseline anxiety	→	Follow-up anxiety	.96	.06	.95	.00	.05	.87	1.09	.00
Baseline medication concerns	→	Follow-up medication concerns	.61	.10	.66	.00	.11	.42	.84	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.60	.09	.60	.00	.17	.37	.82	.01
Baseline personal control	→	Follow-up personal control	.63	.10	.58	.00	.11	.42	.84	.00
Baseline depression	→	Follow-up depression	1.03	.07	.93	.00	.08	.89	1.21	.00
Baseline anxiety	→	Follow-up medication concerns	.04	.02	.22	.03	.02	.00	.09	.06
Baseline anxiety	→	Follow-up timeline cyclical	.08	.05	.34	.13	.18	-.08	.34	.21
Baseline depression	→	Follow-up timeline cyclical	-.04	.06	-.15	.49	.21	-.37	.14	.53

Table C16: Anxiety, cognitions and global diabetes self-care-model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline personal control	→	Follow-up global diabetes self-care	-.54	.20	-.25	.01	.29	-1.17	-.06	.03
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.74	.13	.85	.00	.32	.21	1.22	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline anxiety	→	Follow-up anxiety	.94	.06	.93	.00	.05	.83	1.04	.00
Baseline medication concerns	→	Follow-up medication concerns	.60	.10	.65	.00	.11	.40	.83	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.61	.09	.61	.00	.11	.42	.82	.00
Baseline personal control	→	Follow-up personal control	.63	.10	.59	.00	.11	.42	.85	.00
Baseline anxiety	→	Follow-up medication concerns	.05	.02	.24	.02	.02	.00	.09	.05
Baseline anxiety	→	Follow-up timeline cyclical	.05	.02	.21	.01	.02	.01	.08	.01

Table C17: Anxiety, cognitions, global diabetes self-care, clinical severity-model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline personal control	→	Follow-up global diabetes self-care	-.54	.21	-.24	.01	.32	-1.16	-.02	.04
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.79	.14	.86	.00	.38	.30	1.59	.00
Number of diabetes complications	→	Follow-up global diabetes self-care	.11	.08	.11	.17	.11	-.12	.31	.30
Baseline anxiety	→	Follow-up anxiety	.94	.06	.93	.00	.06	.84	1.05	.00
Baseline medication concerns	→	Follow-up medication concerns	.60	.09	.66	.00	.11	.39	.83	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline timeline cyclical	→	Follow-up timeline cyclical	.67	.09	.66	.00	.09	.50	.85	.00
Baseline personal control	→	Follow-up personal control	.66	.10	.61	.00	.10	.46	.86	.00
Baseline anxiety	→	Follow-up medication concerns	.05	.02	.27	.01	.02	.01	.10	.02
Baseline anxiety	→	Follow-up timeline cyclical	.05	.02	.22	.01	.02	.01	.08	.01
Number of diabetes complications	→	Follow-up medication concerns	.03	.05	.05	.45	.06	-.08	.15	.60
Number of diabetes complications	→	Follow-up timeline cyclical	.04	.05	.05	.47	.05	-.06	.13	.50
			Latent variable co-variances					2.08	4.89	.01
Baseline personal control	↔	Baseline global diabetes self-care	.11	.10	.14	.24	.12	-.13	.35	.33
Baseline timeline cyclical	↔	Baseline global diabetes self-care	.07	.13	.06	.59	.14	-.19	.35	.69
Baseline medication concerns	↔	Baseline global diabetes self-care	-.24	.14	-.19	.10	.14	-.51	.06	.12
Baseline anxiety	↔	Baseline global diabetes self-care	-.09	.61	-.02	.89	.69	-1.33	1.35	.89
Baseline timeline cyclical	↔	Baseline personal control	-.17	.05	-.35	.00	.06	-.29	-.05	.01
Baseline medication concerns	↔	Baseline personal control	-.18	.05	-.35	.00	.06	-.28	-.07	.00
Baseline anxiety	↔	Baseline personal control	-.53	.23	-.23	.02	.23	-1.03	-.11	.02
Baseline medication concerns	↔	Baseline timeline cyclical	.24	.08	.32	.00	.08	.07	.41	.00
Baseline anxiety	↔	Baseline timeline cyclical	1.39	.34	.42	.00	.35	.72	2.12	.00
Baseline anxiety	↔	Baseline medication concerns	2.10	.38	.61	.00	.33	1.43	2.75	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Number of diabetes complications	↔	Baseline anxiety	-.88	.42	-.19	.04	.35	-1.61	-.22	.00
Number of diabetes complications	↔	Baseline medication concerns	-.25	.10	-.24	.01	.08	-.43	-.10	.00
Number of diabetes complications	↔	Baseline timeline cyclical	-.03	.09	-.03	.74	.08	-.17	.14	.77
Number of diabetes complications	↔	Baseline personal control	-.08	.07	-.12	.20	.06	-.21	.03	.14
Number of diabetes complications	↔	Baseline global diabetes self-care	-.06	.18	-.04	.73	.24	-.50	.45	.87

Table C18: Anxiety, cognitions, global diabetes self-care-model parameter estimates sensitivity analysis maximum likelihood imputation

Parameter			Unstandardised	SE	Standardised	P
			<u>Direct effects</u>			
Baseline personal control	→	Follow-up global diabetes self-care	-.45	.17	-.22	.01
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.69	.12	.78	.00
Baseline anxiety	→	Follow-up anxiety	.92	.06	.91	.00
Baseline medication concerns	→	Follow-up medication concerns	.63	.08	.67	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.56	.08	.57	.00
Baseline personal control	→	Follow-up personal control	.66	.09	.64	.00
Baseline anxiety	→	Follow-up medication concerns	.05	.02	.22	.01
Baseline anxiety	→	Follow-up timeline cyclical	.05	.02	.25	.00
			<u>Latent variable co-variances</u>			
Baseline personal control	↔	Baseline global diabetes self-care	.09	.08	.11	.23
Baseline timeline cyclical	↔	Baseline global diabetes self-care	-.08	.10	-.07	.44
Baseline medication concerns	↔	Baseline global diabetes self-care	-.30	.11	-.24	.01

Parameter			Unstandardised	SE	Standardised	P
Baseline global diabetes self-care	↔	Baseline anxiety	-.54	.46	-.10	.25
Baseline timeline cyclical	↔	Baseline personal control	-.17	.04	-.32	.00
Baseline medication concerns	↔	Baseline personal control	-.18	.04	-.34	.00
Baseline personal control	↔	Baseline anxiety	-.56	.18	-.24	.00
Baseline medication concerns	↔	Baseline timeline cyclical	.31	.06	.41	.00
Baseline timeline cyclical	↔	Baseline anxiety	1.40	.26	.42	.00
Baseline medication concerns	↔	Baseline anxiety	1.90	.28	.57	.00

12.6 ***Statistical Appendix D***

Key to aid with the interpretation of Tables D1 to D10

Abbreviation/Symbol	Definition
WBQ(N)	Used to represent questionnaire item numbers from the WBQ (e.g. WBQ1)
IP(N)	Used to represent questionnaire item numbers from the IPQ-R (e.g. IP1)
BMQ(N)	Used to represent questionnaire item numbers from the BMQ (e.g. BMQ1)
SDSCA (N)	Used to represent questionnaire item number from SDSCA (e.g. SDSCA1)
B_	Baseline
F_	Follow-up

Table D1: Negative well-being's directional effect on timeline cyclical model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline timeline cyclical	→	Follow-up timeline cyclical	.41	.09	.50	.00	.10	.21	.61	.00
Baseline negative well-being	→	Follow-up negative well-being	.88	.10	.83	.00	.12	.67	1.12	.00
Baseline negative well-being	→	Follow-up timeline cyclical	.28	.10	.25	.01	.10	.11	.50	.00

Table D2: Personal control and negative well-being auto-regressive model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline personal control	→	Follow-up personal control	.61	.18	.77	.00	.18	.50	.74	.00
Baseline negative well-being	→	Follow-up negative well-being	.91	.10	.87	.00	.12	.83	.99	.00

Table D3: Negative well-being's directional effect on illness coherence model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline illness coherence	→	Follow-up illness coherence	.55	.09	.49	.00	.09	.45	.64	.00
Baseline negative well-being	→	Follow-up negative well-being	.88	.10	.84	.00	.11	.78	1.00	.00
Baseline negative well-being	→	Follow-up illness coherence	-.29	.13	-.18	.02	.15	-.44	-.16	.00

Table D4: Negative well-being's directional effect on medication concerns model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline medication concerns	→	Follow-up medication concerns	.69	.11	.66	.00	.11	.49	.91	.00
Baseline negative well-being	→	Follow-up negative well-being	.91	.11	.85	.00	.14	.69	1.23	.00
Baseline negative well-being	→	Follow-up medication concerns	.34	.16	.20	.04	.20	-.01	.78	.05

Table D5a: Negative well-being's directional effect on timeline cyclical-model fit and parameter estimates with equality constraints applied

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Negative well-being and timeline cyclical (N=172)									
Auto-regressive	130.91	99	.02	.04	.98	.07	204.91		
Timeline cyclical to negative well-being	130.83	98	.02	.04	.98	.07	206.83	.08	.79
Negative well-being to timeline cyclical	122.85	98	.05	.04	.98	.05	198.85	8.06	.01
Cross-lagged*	122.76	97	.04	.04	.98	.05	200.76	.09	.76
*$\Delta\chi^2$ between negative well-being to timeline cyclical and cross lagged									

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline timeline cyclical	→	Follow-up timeline cyclical	.52	.09	.51	.00	.11	.41	.62	.00
Baseline negative well-being	→	Follow-up negative well-being	.86	.06	.84	.00	.07	.79	.92	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline negative well-being	→	Follow-up timeline cyclical	.31	.11	.25	.01	.10	.22	.41	.00

Table D5b: Negative well-being's directional effect on illness coherence-model fit and parameter estimates with equality constraints applied

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Negative well-being and illness coherence (N=172)									
Auto-regressive	172.781	99	.00	.07	.95	.07	246.78		
Illness coherence to negative well-being	172.725	98	.00	.07	.95	.07	248.73	.06	.81
Negative well-being to illness coherence	167.716	98	.00	.06	.96	.05	243.72	5.07	.02
Cross-lagged*	167.659	97	.00	.07	.96	.05	245.66	.06	.81
*$\Delta\chi^2$ between negative well-being to illness coherence and cross lagged									

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			<u>Direct effects</u>							
Baseline illness coherence	→	Follow-up illness coherence	.52	.08	.49	.00	.09	.43	.60	.00
Baseline negative well-being	→	Follow-up negative well-being	.86	.06	.84	.00	.07	.79	.94	.00
Baseline negative well-being	→	Follow-up illness coherence	-.27	.12	-.18	.02	.13	-.41	-.15	.00

Table D5c: Negative well-being's directional effect on medication concerns-model fit and parameter estimates with equality constraints applied

Nested models	χ^2	DF	P	RMSEA	CFI	SRMR	AIC	$\Delta\chi^2$	P
Negative well-being and medication concerns (N=166)									
Auto-regressive	192.07	130	.00	.05	.96	.07	274.07		
Medication concerns to negative well-being	190.27	129	.00	.05	.96	.06	274.27	1.80	.18
Negative well-being to medication concerns	188.29	129	.00	.05	.96	.06	272.29	3.79	.05
Cross-lagged*	187.01	128	.00	.05	.96	.06	273.01	1.28	.26
*$\Delta\chi^2$ between negative well-being to medication concerns and cross-lagged									

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline medication concerns	→	Follow-up medication concerns	.62	.09	.67	.00	.09	.53	.71	.00
Baseline negative well-being	→	Follow-up negative well-being	.86	.06	.85	.00	.08	.78	.95	.00
Baseline negative well-being	→	Follow-up medication concerns	.30	.15	.19	.05	.16	.15	.46	.00

Table D6: Negative well-being, cognitions and global diabetes self-care-model parameter estimates including medication concerns

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline global diabetes self-care revised	→	Follow-up global diabetes self-care revised	.79	.15	.82	.00	.38	.21	1.43	.01

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline personal control	→	Follow-up global diabetes self-care revised	-.50	.19	-.26	.01	.29	-1.20	-.03	.03
Baseline negative well-being	→	Follow-up negative well-being	.93	.06	.91	.00	.07	.79	1.08	.00
Baseline medication concerns	→	Follow-up medication concerns	.67	.10	.72	.00	.10	.46	.87	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.63	.09	.63	.00	.10	.44	.83	.00
Baseline personal control	→	Follow-up personal control	.61	.10	.57	.00	.11	.41	.83	.00
Baseline illness coherence	→	Follow-up illness coherence	.66	.08	.62	.00	.08	.50	.82	.00
Baseline negative well-being	→	Follow-up medication concerns	.04	.03	.13	.22	.03	-.03	.10	.29
Baseline negative well-being	→	Follow-up timeline cyclical	.06	.03	.18	.03	.02	.01	.10	.01
Baseline negative well-being	→	Follow-up illness coherence	-.06	.02	-.18	.02	.03	-.11	-.01	.04

Table D7: Negative well-being, cognitions and global diabetes self-care-model parameter estimates excluding medication concerns

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			<u>Direct effects</u>							
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.81	.16	.79	.00	.46	.24	1.73	.01
Baseline personal control	→	Follow-up global diabetes self-care	-.59	.20	-.29	.00	.40	-1.29	-.08	.03
Baseline negative well-being	→	Follow-up negative well-being	.92	.06	.91	.00	.07	.79	1.06	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.64	.09	.64	.00	.10	.42	.83	.00
Baseline personal control	→	Follow-up personal control	.61	.10	.58	.00	.10	.42	.81	.00
Baseline illness coherence	→	Follow-up illness coherence	.64	.08	.60	.00	.08	.49	.82	.00

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
Baseline negative well-being	→	Follow-up timeline cyclical	.06	.03	.19	.02	.02	.01	.10	.02
Baseline negative well-being	→	Follow-up illness coherence	-.05	.03	-.14	.06	.03	-.09	.01	.08

D8: Negative well-being, cognitions, global diabetes self-care, and clinical severity-model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		<u>Direct effects</u>								
Baseline global diabetes self-care	→	Follow-up global diabetes self-care	.89	.18	.80	.00	.55	.33	2.15	.00
Baseline personal control	→	Follow-up global diabetes self-care	-.59	.21	-.28	.01	.35	-1.47	-.01	.05
Number of diabetes complications	→	Follow-up global diabetes self-care	.10	.08	.10	.22	.11	-.13	.29	.35
Baseline negative well-being	→	Follow-up negative well-being	.97	.07	.95	.00	.07	.82	1.12	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.70	.09	.69	.00	.09	.53	.88	.00
Baseline personal control	→	Follow-up personal control	.65	.10	.62	.00	.11	.44	.86	.00
Baseline illness coherence	→	Follow-up illness coherence	.67	.08	.63	.00	.07	.54	.81	.00
Baseline negative well-being	→	Follow-up timeline cyclical	.06	.03	.20	.02	.02	.01	.11	.01
Baseline negative well-being	→	Follow-up illness coherence	-.06	.03	-.18	.02	.03	-.10	.00	.04
Number of diabetes complications	→	Follow-up timeline cyclical	.05	.05	.06	.37	.05	-.06	.15	.37
Number of diabetes complications	→	Follow-up illness coherence	-.01	.05	-.02	.79	.05	-.11	.09	.78
Number of diabetes complications	→	Follow-up negative well-being	.27	.14	.12	.05	.15	.00	.57	.05
Number of diabetes complications	→	Follow-up personal control	.03	.04	.05	.56	.04	-.06	.11	.58
		<u>Latent variable co-variances</u>								
Baseline personal control	↔	Baseline global diabetes self-	.11	.08	.18	.16	.09	-.08	.29	.27

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
		care								
Baseline illness coherence	↔	Baseline personal control	.23	.05	.47	.00	.06	.11	.34	.00
Baseline illness coherence	↔	Baseline timeline cyclical	-.33	.07	-.46	.00	.08	-.49	-.17	.00
Baseline illness coherence	↔	Baseline global diabetes self-care	.41	.13	.45	.00	.15	.07	.65	.02
Baseline timeline cyclical	↔	Baseline global diabetes self-care	.04	.10	.05	.70	.12	-.17	.31	.75
Baseline negative well-being	↔	Baseline global diabetes self-care	.14	.34	.05	.68	.44	-.69	1.09	.77
Baseline timeline cyclical	↔	Baseline personal control	-.16	.05	-.32	.00	.06	-.28	-.05	.01
Baseline negative well-being	↔	Baseline timeline cyclical	.96	.24	.41	.00	.25	.50	1.47	.00
Baseline illness coherence	↔	Baseline negative well-being	-.76	.23	-.32	.00	.27	-1.30	-.21	.00
Baseline negative well-being	↔	Baseline personal control	-.32	.16	-.20	.05	.17	-.67	.04	.07
Number of diabetes complications	↔	Baseline negative well-being	-.90	.30	-.27	.00	.23	-1.37	-.44	.00
Number of diabetes complications	↔	Baseline timeline cyclical	-.04	.09	-.04	.68	.08	-.19	.13	.65
Number of diabetes complications	↔	Baseline illness coherence	.10	.09	.09	.27	.07	-.04	.23	.15
Number of diabetes complications	↔	Baseline personal control	-.10	.07	-.14	.13	.06	-.23	.01	.07
Number of diabetes complications	↔	Baseline global diabetes self-care	-.08	.14	-.06	.56	.20	-.45	.35	.82

Table D9: Negative well-being, cognitions, global diabetes self-care-model parameter estimates sensitivity analysis direct maximum likelihood imputation excluding medication concerns

Parameter			Unstandardised	SE	Standardised	P
			Direct effects			
Baseline global diabetes self-care revised	→	Follow-up global diabetes self-care revised	.94	.17	.77	.00
Baseline personal control	→	Follow-up global diabetes self-care revised	-.43	.17	-.21	.01
Baseline negative well-being	→	Follow-up negative well-being	.86	.06	.85	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.57	.08	.58	.00
Baseline personal control	→	Follow-up personal control	.65	.09	.63	.00
Baseline illness coherence	→	Follow-up illness coherence	.64	.08	.59	.00
Baseline negative well-being	→	Follow-up timeline cyclical	.07	.02	.24	.00
Baseline negative well-being	→	Follow-up illness coherence	-.06	.02	-.17	.02
			Latent variable co-variances			
Baseline personal control	↔	Baseline global diabetes self-care revised	.07	.05	.11	.22
Baseline personal control	↔	Baseline illness coherence	.27	.04	.52	.00
Baseline timeline cyclical	↔	Baseline illness coherence	-.34	.06	-.46	.00
Baseline global diabetes self-care revised	↔	Baseline illness coherence	.21	.07	.25	.00
Baseline timeline cyclical	↔	Baseline global diabetes self-care revised	-.06	.07	-.07	.40
Baseline negative well-being	↔	Baseline global diabetes self-care revised	-.15	.24	-.05	.52
Baseline timeline cyclical	↔	Baseline personal control	-.17	.04	-.32	.00
Baseline negative well-being	↔	Baseline timeline cyclical	.96	.19	.40	.00
Baseline negative well-being	↔	Baseline illness coherence	-.87	.18	-.36	.00
Baseline negative well-being	↔	Baseline personal control	-.42	.13	-.25	.00

Table D10: Negative well-being, cognitions, global diabetes self-care-model parameter estimates sensitivity analysis maximum likelihood imputation including medication concerns

Parameter			Unstandardised	SE	Standardised	P
			Direct effects			
Baseline global diabetes self-care revised	→	Follow-up global diabetes self-care revised	.94	.17	.77	.00
Baseline personal control	→	Follow-up global diabetes self-care revised	-.43	.17	-.21	.01
Baseline negative well-being	→	Follow-up negative well-being	.88	.06	.86	.00
Baseline medication concerns	→	Follow-up medication concerns	.70	.09	.74	.00
Baseline timeline cyclical	→	Follow-up timeline cyclical	.57	.08	.58	.00
Baseline personal control	→	Follow-up personal control	.65	.09	.63	.00
Baseline illness coherence	→	Follow-up illness coherence	.65	.08	.60	.00
Baseline negative well-being	→	Follow-up medication concerns	.03	.03	.11	.22
Baseline negative well-being	→	Follow-up timeline cyclical	.07	.02	.24	.00
Baseline negative well-being	→	Follow-up illness coherence	-.06	.02	-.18	.01
			Latent variable co-variances			
Baseline personal control	↔	Baseline global diabetes self-care revised	.07	.05	.11	.23
Baseline personal control	↔	Baseline illness coherence	.27	.04	.52	.00
Baseline timeline cyclical	↔	Baseline illness coherence	-.34	.06	-.46	.00
Baseline medication concerns	↔	Baseline timeline cyclical	.31	.06	.42	.00
Baseline negative well-being	↔	Baseline medication concerns	1.43	.21	.58	.00
Baseline global diabetes self-care revised	↔	Baseline illness coherence	.21	.07	.24	.00
Baseline timeline cyclical	↔	Baseline global diabetes self-care revised	-.06	.07	-.07	.43
Baseline medication concerns	↔	Baseline global diabetes self-care revised	-.21	.08	-.25	.00
Baseline negative well-being	↔	Baseline global diabetes self-care revised	-.16	.24	-.06	.50
Baseline timeline cyclical	↔	Baseline personal control	-.17	.04	-.32	.00

Parameter			Unstandardised	SE	Standardised	P
Baseline medication concerns	↔	Baseline illness coherence	-.43	.06	-.57	.00
Baseline negative well-being	↔	Baseline timeline cyclical	.96	.19	.40	.00
Baseline medication concerns	↔	Baseline personal control	-.18	.04	-.34	.00
Baseline negative well-being	↔	Baseline illness coherence	-.85	.18	-.35	.00

12.7 ***Statistical Appendix E***

Key to aid with the interpretation of Tables E1 to E4

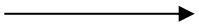
Abbreviation/Symbol	Definition
IP(N)	Used to represent questionnaire item numbers from the IPQ-R (e.g. IP1)
BMQ(N)	Used to represent questionnaire item numbers from the BMQ (e.g. BMQ1)
	Indicates the direction of effect
	Indicates that variables are assumed to co-vary but the direction of effect is not explicitly stated
B_	Baseline

Table E1: Baseline timeline acute/chronic, medication necessity, and illness coherence as predictors of HbA1c as six months follow-up-model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline HbA1c	→	Follow-up HbA1c	.63	.07	.60	.00	.09	.45	.79	.00
Baseline timeline acute/chronic	→	Follow-up HbA1c	4.78	2.13	.19	.03	4.78	1.83	18.40	.00
Baseline medication necessity	→	Follow-up HbA1c	-7.26	2.27	-.26	.00	2.15	-12.06	-3.25	.00
Baseline illness coherence	→	Follow-up HbA1c	-1.45	1.44	-.08	.31	1.34	-4.36	.76	.21

Table E2: Baseline timeline acute/chronic and medication necessity as predictors of HbA1c as six months follow-up-model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline HbA1c	→	Follow-up HbA1c	.65	.07	.61	.00	.08	.48	.80	.00
Baseline timeline acute/chronic	→	Follow-up HbA1c	4.61	2.09	.18	.03	3.68	1.84	16.35	.00
Baseline medication necessity	→	Follow-up HbA1c	-6.61	2.13	-.23	.00	1.90	-10.76	-2.97	.00

Table E3: Baseline timeline acute/chronic, medication necessity and clinical severity as predictors of HbA1c at six months follow-up-model parameter estimates

Parameter			Unstandardised	SE	Standardised	P	Unstandardised bootstrapped standard errors			
							SE	Lower	Upper	P
			Direct effects							
Baseline HbA1c	→	Follow-up HbA1c	.64	.07	.61	.00	.10	.49	.83	.00
Baseline timeline acute/chronic	→	Follow-up HbA1c	3.21	1.65	.18	.05	2.55	.20	7.97	.02
Baseline medication necessity	→	Follow-up HbA1c	-6.45	2.19	-.23	.00	2.22	-10.67	-2.49	.01
Number of diabetes complications	→	Follow-up HbA1c	-.50	1.07	-.03	.64	1.55	-2.42	2.31	.76

Table E4: Baseline timeline acute/chronic and medication necessity as predictors of HbA1c at six months follow-up- model parameter estimates sensitivity analysis maximum likelihood imputation

Parameter			Unstandardised	SE	Standardised	P
			Direct effects			
Baseline HbA1c	→	Follow-up HbA1c	.67	.07	.63	.00
Baseline timeline acute/chronic	→	Follow-up HbA1c	4.59	1.91	.19	.02
Baseline medication necessity	→	Follow-up HbA1c	-6.64	2.02	-.24	.00