

VOLUME I

RESEARCH COMPONENT

**DEPRESSIVE SYMPTOMS IN ADOLESCENTS WITH TYPE 1
DIABETES**

by

KIRAN BALI

A thesis submitted to

the University of Birmingham

for the degree of

DOCTORATE OF CLINICAL PSYCHOLOGY

Department of Clinical Psychology
School of Psychology
The University of Birmingham
May 2014

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Overview

This thesis is submitted in partial fulfilment of the requirements for the degree of Doctorate of Clinical Psychology from the University of Birmingham. The thesis consists of two volumes. Volume I includes a literature review and an empirical paper. Volume II consists of four Clinical Practice Reports and an abstract for a fifth, an oral presentation. Each report relates to work undertaken at different clinical placements.

Volume I

Volume I comprises three chapters. The first chapter is a systematic literature review synthesising evidence on the longitudinal relationship between depressive symptoms and metabolic control in adolescents with Type 1 diabetes. The focus of the review is on issues of directionality within this relationship over time and ascertaining factors that may influence identified longitudinal associations. This chapter was prepared with the intention of submission to the journal *Diabetes Research and Clinical Practice* (See Appendix i for instructions for authors). The second chapter is an empirical paper exploring the role of cognitive mechanisms in depressive symptoms in adolescents with Type 1 diabetes. The cognitions that were investigated were negative automatic thoughts as proposed by Beck's cognitive theory of depression (1967), and self-efficacy, as per Bandura's social cognitive theory (1997). This chapter was prepared for submission to the *Journal of Paediatric Psychology* (See Appendix ii for instructions for authors). The third chapter is a public domain briefing document that provides a summary of the findings of the systematic literature review and the empirical paper.

Volume II

This volume presents four written Clinical Practice Reports and an abstract for a clinical practice report that was presented orally. The first report details the case of a 24-year

old male with a mild learning disability who was experiencing elevated levels of anxiety. The assessment is described alongside cognitive behavioural and psychodynamic formulations of the presenting difficulty. The second report presents the evaluation of a community learning disabilities service based on recommendations for service provision to adults with profound and multiple learning disabilities. The third report documents an A-B single-case experimental design employed to evaluate the effectiveness of behavioural activation for depression in a 69-year old man at a specialist psychiatric unit for individuals with dementia. The fourth report presents a case study of a 38-year old man who was experiencing increased levels of anxiety. The assessment, formulation and intervention, all informed by the cognitive behavioural model, are described in addition to an evaluation of the intervention. The fifth report is the abstract of an orally presented case study describing the case of a 17-year old young woman with Anorexia Nervosa and low mood who was a patient at a specialist eating disorders unit. A cognitive behavioural intervention was implemented to target the cycle perpetuating her low mood. All names and identifying information in Volume II have been altered to maintain confidentiality.

Dedication

To Mum and Dad.

Thank you for giving me the opportunities you never had.

Acknowledgements

I would like to express my utmost gratitude to the young people and families who took part in this research. Without them, this project would not have been possible. I am also grateful to the staff at the Diabetes clinics for their assistance and kindness during the recruitment period.

I would like to thank my research supervisors, Dr Gary Law and Dr Arie Nouwen, for their guidance and invaluable feedback throughout the research process.

Thank you to my family and friends for the encouragement and support over the course of my clinical training.

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CHAPTER ONE

THE LONGITUDINAL RELATIONSHIP BETWEEN DEPRESSIVE SYMPTOMS AND METABOLIC CONTROL IN ADOLESCENTS WITH TYPE 1 DIABETES

LITERATURE REVIEW

Abstract

Background. There is evidence of an association between depressive symptoms and metabolic control in adolescents with Type 1 diabetes (T1D). Yet the directionality within this relationship is unclear. The current review aimed to synthesise longitudinal research to gain clarity regarding issues of directionality in addition to identifying factors that influence associations.

Method. Embase, PsycINFO, MEDLINE, Web of Science and PubMed were searched using variations of the constructs related to the aims of this review (i.e. depression, T1D, metabolic control, adolescents, and longitudinal research). Suitability of research was assessed against inclusion and exclusion criteria and the quality of the included studies (n=14) was evaluated.

Results. Most articles examined metabolic control in response to depressive symptoms. Findings were mixed with some studies suggesting a negative association between earlier depressive symptoms and subsequent metabolic control, an association that was reported to decrease over time. Furthermore, this relationship was not independent of the influence of other variables (e.g. illness specific factors, interpersonal factors).

Conclusions. Directionality within the longitudinal relationship between depressive symptoms and metabolic control in adolescents with T1D remains unclear. Further research should consider influencing variables and more frequent follow-ups to facilitate a closer examination of longitudinal associations.

Keywords: Depression, Type 1 diabetes, metabolic control, adolescents, longitudinal research

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Introduction

Type 1 diabetes. Type 1 diabetes (T1D) is a metabolic illness caused by the destruction of insulin producing cells in the pancreas resulting in a permanent deficiency of insulin (American Diabetes Association, 2013). Approximately 26 500 children and young people in the UK have been diagnosed with this autoimmune disease (Diabetes UK, 2012), with numbers rising by an average of 3 – 4% per year across Europe (Patterson et al., 2012). Hypotheses for these increasing figures have predominantly focussed on environmental factors such as the surge in Vitamin D deficiency (Virtanen & Knip, 2003) and the lack of exposure to infections hindering the maturation of the immune system thus increasing susceptibility to developing autoimmune diseases (Gale, 2002). Irrespective of the possible causes, receiving a diagnosis of T1D during childhood and adolescence has lifelong implications including rigorous self-management through an often complex treatment plan. This self-care regimen typically involves a combination of the daily monitoring of blood glucose levels and regulating insulin with injections or an insulin pump, in addition to making comprehensive dietary and lifestyle changes (Silverstein et al., 2005).

Metabolic control. Clinical treatment targets for individuals with T1D are based on achieving a level of blood glucose that is as close to normal levels, relative to age, and is assessed by examining metabolic control. Metabolic control is generally measured by calculating levels of glycosylated haemoglobin, also known as HbA1C (Rewers et al., 2009). HbA1c is the proportion of glucose molecules attached to haemoglobin in the blood and represents an average level of blood glucose over an eight to twelve week period. It has been reported as the most useful way of monitoring metabolic control (Saudek & Brick, 2009). It is recommended that children and adolescents attain HbA1c values of less than 7.5% (Rewers et al., 2009), with healthcare professionals tailoring treatment to facilitate this (NICE, 2005). Yet

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young people are likely to struggle with meeting treatment goals, with a marked trend towards deterioration in HbA1c during adolescence (Bryden et al., 2001).

Depression and Type 1 diabetes. Depression is a mood disorder characterised by the presence of symptoms including low mood, loss of interest in activities and feelings of worthlessness, in addition to changes in sleep, appetite and activity (American Psychiatric Association, 2013). The prevalence of depression is reported to be higher in individuals with T1D (Barnard, Skinner, & Peveler, 2006) and adolescents with T1D are particularly vulnerable to developing depressive symptoms (Grey, Whittemore, & Tamborlane, 2002). A higher prevalence of depression has been demonstrated within this sample compared to the general adolescent population (Kanner, Hamrin, & Grey, 2003) with incidence rates of up to a threefold occurrence (Johnson, Eiser, Young, Brierley, & Heller, 2013). These numbers are a cause for concern as young people with both T1D and depression are more likely to have suicidal ideation and, consequently, are at increased risk of suicide (Goldston, Kovacs, Ho, Parrone, & Stiffler, 1994).

Numerous sequelae for adolescents with T1D and depressive symptoms (i.e. not reaching diagnostic criteria but displaying symptoms) have been identified. For instance, these young people are more likely to develop other psychiatric disorders including anxiety (Dantzer, Swendsen, Maurice-Tison, & Salamon, 2003) and eating disorders (Olmsted, Colton, Daneman, Rydall, & Rodin, 2008). The presence of depressive symptoms in this population has also been associated with poor adjustment to diagnosis and treatment demands (Lernmark, Persson, Fisher, & Rydelius, 1999) and reduced self-management which increases the risk of hospitalisation (Stewart, Rao, Emslie, Klein, & White, 2005).

Depression and metabolic control. A number of hypotheses aim to explain the links between depressive symptomatology and disease related outcomes in diabetes. One possible

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theory attributes abnormalities in the functioning of the neuroendocrine system as a direct consequence of depressive symptoms. Musselman and colleagues (2003) suggest an excess of stress related hormones present in those with depression (e.g. cortisol) interfere with effective insulin functioning by further raising levels of blood glucose. More specifically, it is thought that the development of complications associated with depression and diabetes is caused by the effect depression has on metabolic control (de Groot, Anderson, Freedland, Clouse, & Lustman, 2001). The behavioural features of depression, e.g. reduced motivation, offer a plausible explanation for this as they result in poor self-management and compliance to treatment (Lin et al., 2004).

Alternative hypotheses demonstrate the maladaptive self-perpetuating relationship between depression and metabolic control. A cycle has been identified where increased levels of HbA1c cause low mood related to not achieving treatment targets, which then creates further difficulties in achieving metabolic control goals by exacerbating behavioural symptoms and decreasing motivation to comply with treatment plans (Lustman & Clouse, 2005). Cognitions specifically associated with depression are also thought to be a factor in this relationship. Farrell and colleagues (2004) found that an increase in depressive thinking styles was associated with an increase in general stress, resulting in higher levels of HbA1c in adolescents by impacting on self-management behaviour.

The extant evidence base predominantly consists of cross-sectional research demonstrating that higher levels of depressive symptoms are associated with poorer metabolic control in adolescents with T1D (e.g. Hood et al., 2006; Kovacs et al., 1995; La Greca & Bearman, 2002). Existing reviews have further suggested factors that may influence this relationship at single time points. These include treatment compliance and the occurrence of significant life events (Dantzer, Swendsen, Maurice-Tison, & Salamon, 2003),

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neuropsychological factors and maternal depression (Kanner et al., 2003), and intrapersonal characteristics such as coping skills and self-efficacy (Neylon, O'Connell, Skinner, & Cameron, 2013). However, a significant gap identified in the literature is the need for more longitudinal research to clarify causal relationships (Delamater et al., 2001) and to further explore associations between depression and metabolic control over time (Grey et al., 2002).

Aims. Adolescents with T1D are vulnerable to developing depressive symptoms. The current literature offers a more-or-less consistent picture with regard to associations between metabolic control and depressive symptoms, but there are mixed reports and theories as to the mechanisms of effect and directionality of results. The apparent lack of longitudinal studies within this area creates difficulty in drawing conclusions with confidence. Furthermore patterns over time in this association in adolescents remain a relative unknown. Building on the limitations identified in the literature, this review investigated the relationship between depressive symptoms and metabolic control in adolescents with T1D, with a particular focus on longitudinal research to help explore issues of directionality. Factors influencing this relationship in relation to longitudinal changes were identified. To determine the confidence in claims of identified associations, the quality of the research was assessed. A systematic review was therefore undertaken of the adolescent T1D evidence base with a focus on the following aims:

- (i) Identify findings in the literature regarding directionality in the longitudinal relationship between depressive symptoms and metabolic control.
- (ii) Identify factors in the literature that influence the relationship between depressive symptoms and metabolic control over time.

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Method

Search strategy. Five electronic databases (Embase, PsycINFO, MEDLINE, Web of Science, PubMed) were searched using variations of constructs related to the aims of the review, that is: depression, Type 1 diabetes, adolescents, longitudinal and metabolic control. Searches were conducted by combining variations using ‘AND’ as the Boolean operator. Variations of terms, entered in parentheses, were combined using ‘OR’ and truncation. Figure 1 illustrates the variations of each construct and the application of Boolean operators.

		Main constructs										
		Depression		Diabetes		Type 1		Adolescent		Longitudinal		Metabolic control
Variations of main construct												
		Depress* OR Mood OR Wellbeing	AND	Diabetes	AND	Type 1 OR Insulin dependent OR IDDM	AND	Child* OR Adolescen*	AND	Longitudinal* OR Predict* OR Follow up	AND	Metabolic control OR Glycemic control OR Glycosylated haemoglobin OR Glucose regulation OR Management OR Blood glucose monitoring OR BGM OR HbA1c OR Adher* OR Regimen

Figure 1. Variations of each construct entered in the search using Boolean operators.

Databases were searched individually from their inception, as no other review of this nature had been done, to January 2014. The ‘Keyword’ searches of Embase, PsycINFO and MEDLINE resulted in 148, 33 and 99 articles respectively. The ‘Topic’ search of Web of

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Science produced 83 articles and a search in ‘All Fields’ of PubMed resulted in 188 articles. The total number of articles (including duplicates) retrieved from the five databases was 551.

Inclusion and exclusion criteria. After the removal of 229 duplicate articles across the databases, the abstracts of articles were scanned against inclusion criteria to determine their relevance. To ensure the most appropriate research was included, and as some abstracts did not clearly evidence the criteria for inclusion, exclusion criteria were applied to the full texts of the remaining articles. Bibliographies of included articles were examined but yielded no further results. The inclusion and exclusion criteria are detailed in Table 1.

Table 1

Criteria for inclusion and exclusion.

Inclusion criteria	Exclusion criteria
✓ Adolescents aged between 8 and 18 years of age	✗ Findings were based on a sample that consisted of participants with T1D in addition to participants with other health chronic conditions e.g. asthma
✓ Diagnosis of T1D	✗ Age range of the sample at baseline was below 8-years and exceeded 18-years of age*
✓ Metabolic control data included	✗ HbA1c data not reported
✓ Depressive symptoms were assessed	✗ No assessment of depressive symptomatology
✓ Associations between depression and HbA1c were examined over time with at least one follow up	✗ Depression and HbA1c were assessed at only one time point
✓ Published in English in a peer-reviewed journal, describing empirical research	✗ Direct association between depression and metabolic control was not explored or when depression was not entered as the independent variable when HbA1C was the dependent variable, and vice versa

*Limiting studies to omit those where the samples were over 18 years of age at follow-up but not at baseline would have excluded two papers (Kovacs et al., 1996; Northam et al., 2004) that followed the adolescent baseline sample for a period of ten years or more. Therefore the two papers in question were included.

Final selection. Application of the inclusion criteria, removal of duplicates and examination of full texts using the exclusion criteria resulted in 14 articles. Figure 2 illustrates the number of articles excluded at each stage of the screening process.

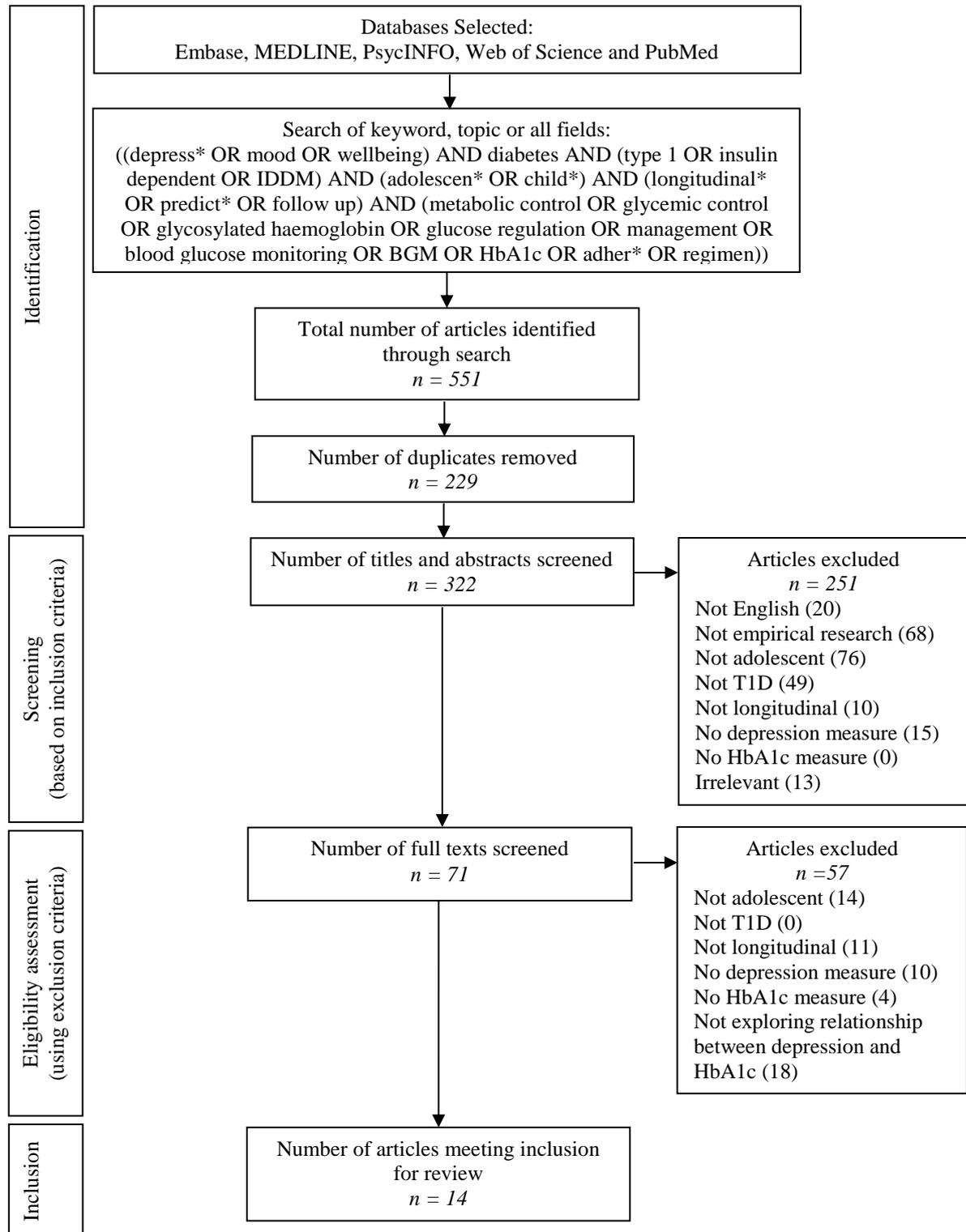


Figure 2. Illustration of the screening process (adapted from Moher et al., 2009).

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Quality assessment. The quality of the final subset of articles was assessed using a formal critical appraisal tool. The ‘Checklist for Measuring Study Quality’ (Downs & Black, 1998) was developed for evaluating a range of research studies. Certain items were not applicable to the research methods employed in the reviewed articles, particularly items concerning interventions. Consequently 12 items¹ were removed to produce an alternative quality score incorporating appropriate items to facilitate quality comparison across studies. The item regarding power (no. 27) was re-worded to increase its relevance to the review. Justification for the adoption of this framework is provided in Figure 3.

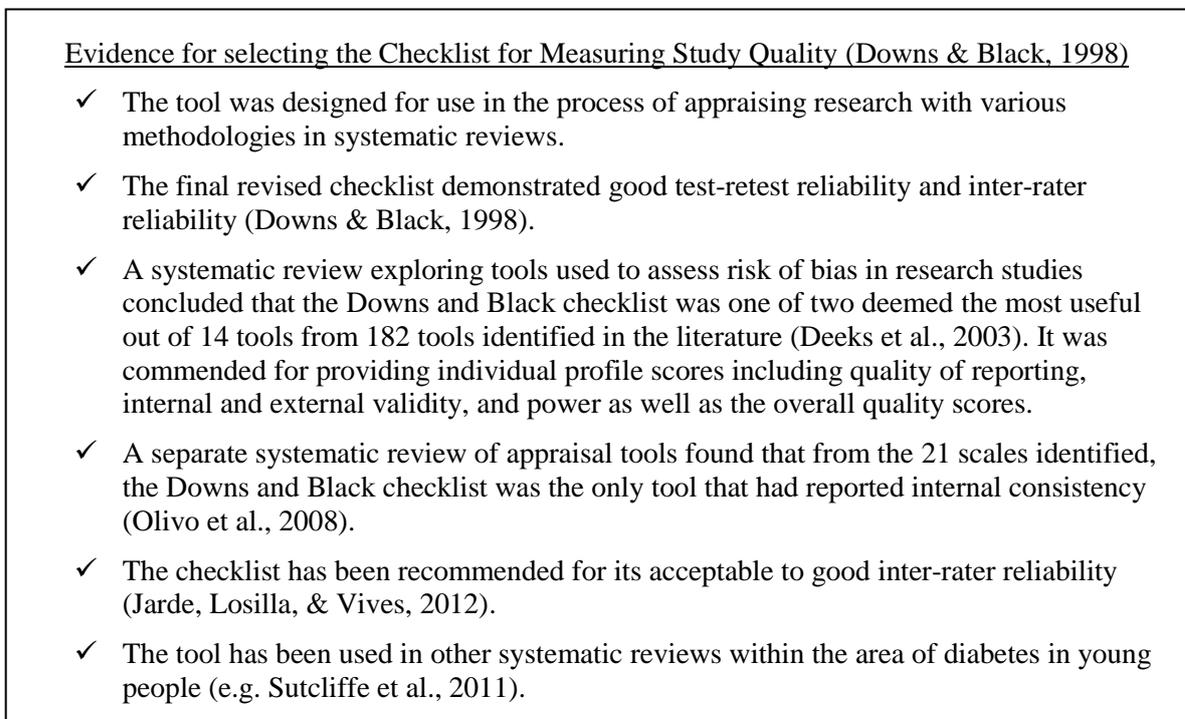


Figure 3. Justification for selection of The Checklist for Measuring Study Quality

Data extraction. Data from articles were extracted and tabulated to provide a framework for the review. The first table (Appendix A) addressed the aim of directionality in the longitudinal relationship between depressive symptoms and metabolic control. The second table (Appendix B) focussed on variables reported to influence this relationship.

¹ The items removed as numbered on the original tool (Appendix C) were: 4, 5, 8, 14, 15, 17, 19, 21, 22, 23, 24, 25.

Results

Study aims. Of the 14 papers included for review, two examined the relationship between earlier levels of metabolic control and later depressive symptoms. The remaining 12 studies explored the converse. Differences emerged in terms of primary aims and how the relationship of interest was explored e.g. through direct analysis or via predictor models. An overview of the aims is provided in Figure 4.

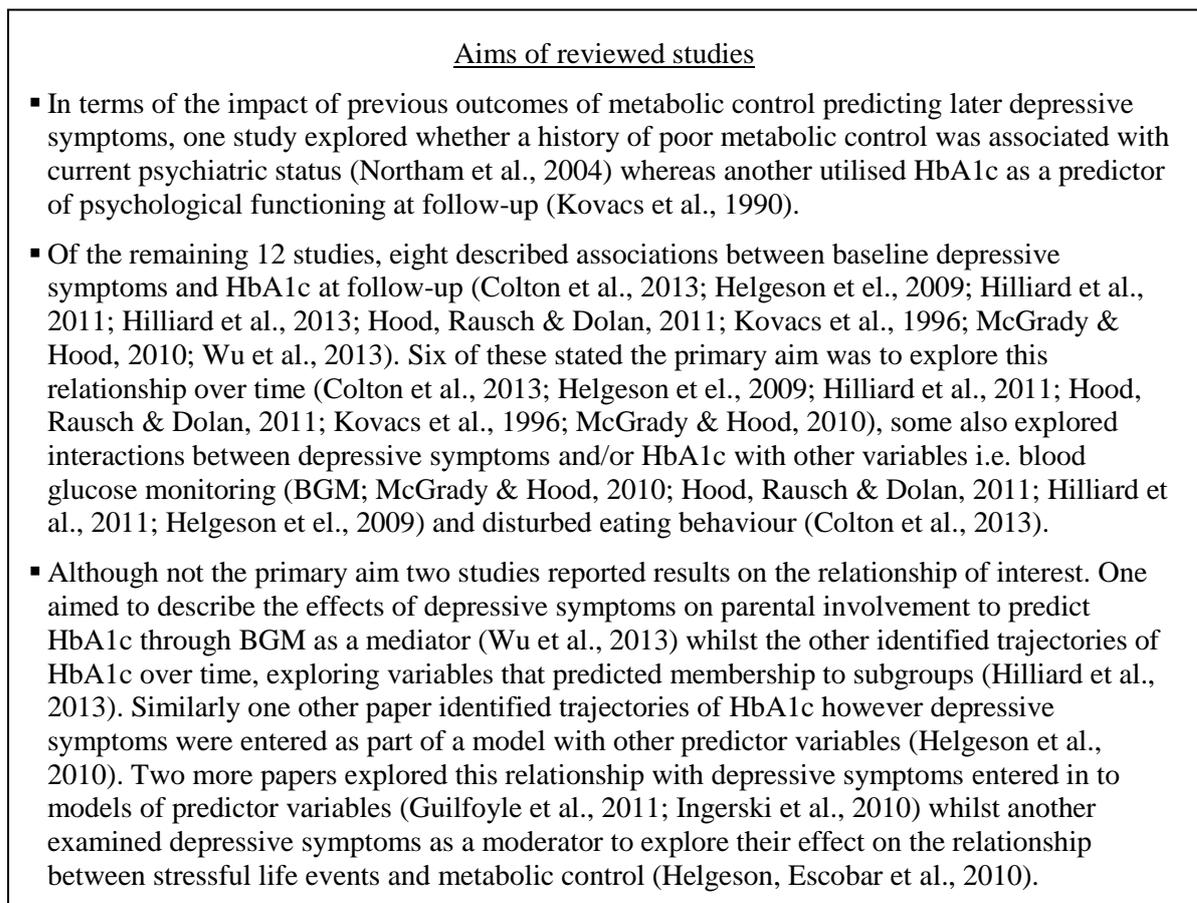


Figure 4. Overview of the aims of studies included for review.

Assessment of depressive symptoms. The majority of the studies ($n = 11$) used the Children's Depression Inventory (CDI) or variations of the tool (Kovacs 1985, 1992, 2001, 2003). Three studies used cut off scores² on the CDI to describe the number of participants with clinical levels of depressive symptoms, one of which reported analyses on both cut-off

² Scores higher than or equal to 13 on the CDI suggest depressive symptoms that are clinically significant.

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scores and continuous scores. The remaining studies explored the variance in continuous CDI scores. Parental reports of adolescent depressive symptoms were assessed using the parent version of the CDI (CDI:P; Kovacs, 1992) in one study. In addition to using the CDI, one study also used the Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman et al., 1997). Other methods of assessing depressive symptoms were based on diagnostic criteria, with one study using the Interview Schedule for Children and Adolescents (Kovacs, 1985) followed by consensus of diagnosis amongst researchers. A diagnostic method, the Diagnostic Interview for Children and Adolescents-IV (Herjanic & Reich, 1997), was also used in another study at follow-up with the Child-Behaviour Checklist (Achenbach, 1991) at baseline. Table 2 provides a summary of the assessment methods used.

Table 2

Methods of assessment for depressive symptoms utilised across the reviewed studies.

	Kovacs et al. (1990)	Kovacs et al. (1996)	Northam et al. (2004)	Helgeson et al. (2009)	Helgeson, Escobar et al. (2010)	Helgeson et al. (2010)	Ingerski et al. (2010)	McGrady & Hood (2010)	Guilfoyle et al. (2011)	Hilliard et al. (2011)	Hood et al. (2011)	Colton et al. (2013)	Hilliard et al. (2013)	Wu et al. (2013)
Children's Depression Inventory - Continuous score	✓			✓	✓	✓	✓		✓	✓		✓	✓	✓ ^P
Children's Depression Inventory - Cut off score								✓		✓	✓			
Kiddie-Schedule for Affective Disorders and Schizophrenia												✓		
Interview Schedule for Children and Adolescents		✓												
Diagnostic Interview for Children and Adolescents-IV			✓											
Child Behaviour Checklist			✓											

^P Parent version of CDI also administered.

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With regard to clinical application, the consideration of cut-off scores or continuous scores is dependent on the primary purpose of the assessment. Whilst cut-off scores demonstrate clinical levels of depression according to diagnostic criteria, continuous scores demonstrate the severity and level of depressive symptoms experienced.

Study duration. The reported study duration ranged from 3-months to those that assessed participants over a period of 10-years. Similarly, the number of follow-ups conducted varied between the studies. Table 3 provides a summary of the number of follow-ups over the study duration reported in each article and demonstrates that most of the studies with fewer follow-up assessments were those with a relatively short study duration. Eight studies reported results on multiple follow-ups. However, despite a longer study duration of 5-years, Colton and colleagues (2013) only conducted one follow-up assessment. Other studies failed to clearly report how many follow-ups were conducted.

Table 3

Study duration and number of follow-ups (in ascending order of study duration).

	Guilfoyle et al. (2011)	Hood et al. (2011)	Ingerski et al. (2010)	McGrady & Hood (2010)	Hilliard et al. (2011)	Wu et al. (2013)	Hilliard et al. (2013)	Helgeson et al. (2009)	Colton et al. (2013)	Helgeson, Escobar et al. (2010)	Helgeson et al. (2010)	Kovacs et al. (1990)	Kovacs et al. (1996)	Northam et al. (2004)
Study Duration	3M	6M	6M	6M	12M	18M	18-24M	4Y	5Y	5Y	5Y	6Y	8.6Y*	10Y
Follow-ups	1	1	1	1	1	3	3	3	1	4	4	U	U	3M**

M = Months; Y = Years; U = Unclear

*Average study duration

**Every 3 months

Quality assessment. The scores from the critical appraisal tool are presented in Table 4 to aid direct comparison between studies. Implementation of the tool across the reviewed

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articles revealed that most of the papers provided sufficient information with regard to the reporting of the research conducted. One paper (McGrady & Hood, 2010) did not clearly describe the main aims of the study. Most discrepancies between the scores were a consequence of failing to report exact probability values. With regard to external validity, all papers were similar however only one study (Northam et al., 2004) described a sample that was representative of the population they were recruited from thus increasing the generality of the findings. Samples in the other studies were skewed in terms of ethnicity. All studies demonstrated good internal validity based on the measures used and the statistical analyses conducted. Five papers did not make reference to power when reporting significant findings.

The paper by McGrady and Hood (2010) scored relatively lower than the other papers, however the areas in which points were not awarded were not deemed as being critical to the credibility of the findings. The main limitation established across the literature was concerning the external validity of findings. This was not applicable to the paper by Northam and colleagues (2004) which rated similarly to other papers with a score of 14, only losing a mark for failing to report exact probability values. Therefore on the basis of a greater generality and applicability of findings to the wider population, this paper was considered as the strongest from those reviewed.

Overall, the quality of the papers was broadly similar as they rated fairly highly with scores suggesting no need for exclusion from this review based on quality. The papers will be discussed accordingly, referring to issues of quality based on the outcome of the critical appraisal tool, with regard to the aims of the review, that is: (i) Identify findings in the literature regarding directionality in the longitudinal relationship between depressive symptoms and metabolic control; and (ii) Identify factors in the literature that influence the relationship between depressive symptoms and metabolic control over time.

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Table 4

Quality assessment summary adapted from Downs and Black (1998).

	Kovacs et al. (1990)	Kovacs et al. (1996)	Northam et al. (2004)	Helgeson et al. (2009)	Helgeson, Escobar et al. (2010)	Helgeson et al. (2010)	Ingerski et al. (2010)	McGrady & Hood (2010)	Guilfoyle et al. (2011)	Hilliard et al. (2011)	Hood et al. (2011)	Colton et al. (2013)	Hilliard et al. (2013)	Wu et al. (2013)
Reporting (Yes = 1; No = 0)														
1. Is the hypothesis/aim/objective of the study clearly described?	1	1	1	1	1	1	1	0	1	1	1	1	1	1
2. Are the main outcomes to be measured clearly described in the Introduction or Methods section?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3. Are the characteristics of the patients included in the study clearly described?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
6. Are the main findings of the study clearly described?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
7. Does the study provide estimates of the random variability in the data for the main outcomes?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
9. Have the characteristics of patients lost to follow-up been described?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
10. Have actual probability values been reported (e.g. 0.035 rather than <0.05) for the main outcomes except where the probability value is less than 0.001?	1	1	0	1	0	1	0	0	1	1	1	1	0	1
External Validity (Yes = 1; No/Unable to determine = 0)														
11. Were the subjects asked to participate in the study representative of the entire population from which they were recruited?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
12. Were those subjects who were prepared to participate representative of the entire population from which they were recruited?	0	0	1	0	0	0	0	0	0	0	0	0	0	0
13. Were the staff, places, and facilities where the patients were treated, representative of the treatment the majority of patients receive?	1	1	1	1	1	1	1	1	1	1	1	1	1	1

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	Kovacs et al. (1990)	Kovacs et al. (1996)	Northam et al. (2004)	Helgeson et al. (2009)	Helgeson, Escobar et al. (2010)	Helgeson et al. (2010)	Ingerski et al. (2010)	McGrady & Hood (2010)	Guilfoyle et al. (2011)	Hilliard et al. (2011)	Hood et al. (2011)	Colton et al. (2013)	Hilliard et al. (2013)	Wu et al. (2013)
Internal validity – bias (Yes = 1; No/Unable to determine = 0)														
16. If any of the results of the study were based on “data dredging”, was this made clear?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
18. Were the statistical tests used to assess the main outcomes appropriate?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
20. Were the main outcome measures used accurate (valid and reliable)?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Internal validity – confounding (selection bias) (Yes = 1; No/Unable to determine = 0)														
26. Were losses of patients to follow-up taken into account?	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Power (Yes = 1; No/Unable to determine = 0)														
27. Did the study report consideration of power to report clinical significant effects?	1	0	1	1	1	1	1	0	0	0	0	1	1	1
Total score (out of 15)	14	13	14	14	13	14	13	11	13	13	13	14	13	14

Note: Item numbers refer to those on the original quality assessment tool by Downs and Black (1998).

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(i) Identify findings in the literature regarding directionality in the longitudinal relationship between depressive symptoms and metabolic control. Articles reporting associations between earlier depressive symptoms and later HbA1c values (n=12) will be discussed first. They will be grouped into those reporting a direct statistical analysis between metabolic control and depressive symptoms (n=6) or a diagnosis of depression (n=2), where depressive symptoms were included as a predictor of metabolic control subgroup membership (n=3) or entered into a model alongside other predictor variables (n=4). Articles may be described under multiple headings where relevant. Articles describing associations between previous HbA1c values with later depressive status (n=2) will be discussed with regard to results that either reported depressive symptoms (n=1) or a diagnosis of depression (n=1).

Metabolic control measured in response to depressive symptoms: Direct analysis. Of the six studies that aimed to explore the direct relationship between depressive symptoms and HbA1c, two described results over a 6-month period. Hood, Rausch and Dolan (2011) explored this relationship with a focus on change scores from baseline to follow-up. The results of the general linear model showed that an increase in depressive symptoms over time was associated with increasing levels of HbA1c ($p < 0.001$). More specifically, an increase of one point from baseline to follow-up on the CDI was associated with a 0.11% increase in HbA1c. However, McGrady & Hood (2010) reported no significant association between depressive symptoms and metabolic control despite 23% of the sample reporting clinically significant depressive symptoms on the CDI. An analysis using individual subscales of the CDI further failed to demonstrate significant associations with HbA1c. Discrepancies between results may be due to Hood and colleagues utilising change scores to determine associations as opposed to the follow-up scores reported by McGrady and Hood. With regard to bias, McGrady and Hood failed to provide exact p values and a clear study aim. Hood and

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colleagues provided a clearer account of their findings and also stated details of an unplanned, exploratory analysis (i.e. data dredging). However it was unclear whether power was considered when describing findings. Both papers reported that results were based on a homogenous sample in terms of being predominantly White American, raising caution with regard to issues of generalisability. McGrady and Hood further questioned the use of self-report measures to assess depressive symptoms.

Despite higher HbA1c being associated with increased depressive symptoms at baseline, Hilliard et al. (2011) reported that baseline depressive symptoms were not associated with HbA1c at 12-months. A strength of this paper was the analyses of both cut-off scores and continuous scores on the CDI. In contrast to these results, Helgeson and colleagues (2009) found that baseline depressive symptoms predicted increases ($B=1.18$, $p<0.01$) and changes ($B=-0.71$, $p<0.05$) in HbA1c at 12-months. Despite annual assessments over a 4-year period the predictive ability of depressive symptoms decreased over time, with the strongest predictive value at baseline for HbA1c at 12-months. The account of internal consistency for the CDI at each assessment point increased the internal validity of the study. Both papers described limitations with regard to sample characteristics and the use of predominantly White American participants. The main difference between the articles was that power was explicitly considered in the Helgeson paper.

Where the relationship between depressive symptoms and HbA1c was not the main aim of the study, Wu and colleagues (2013) found that self-reports of depressive symptoms at baseline were not associated with HbA1c at 18-months, however, parental reports of child depressive symptoms were significant ($p=0.008$). The use of reports from carers in addition to self-report was highlighted as a methodological strength. Limitations included the lack of a diverse sample (i.e. not representing minority groups) and whether the findings from a study

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based in the USA, with treatments and practices specific to the target recruitment clinic, can in fact be generalised to other contexts and cultures.

In a study exploring the relationship between depression and disturbed eating behaviour on metabolic control, Colton and colleagues (2013) reported that depressive symptoms measured at baseline did not predict metabolic control at 5-years. However results were based on a female sample and the authors stated that the relatively small sample size has implications for the power of the study. A strong retention rate was noted as a strength, with no losses at follow-up, in addition to the use of standardised diagnostic interviews for depression alongside questionnaires.

In summary, when based on baseline CDI scores (as opposed to change scores), results demonstrate a significant association between depressive symptoms and HbA1c at 6-months. No significant associations at 12-months were reported when using cut-off scores and continuous scores. Conversely the predictive value of baseline depressive symptoms was significant at 12-months but decreased over time. Differences in results emerged through administration of self and parental reports of child depressive symptoms. The latter demonstrated an association between earlier depressive symptoms and HbA1c at follow up. Association ceased to exist over longer follow-up periods of up to 5-years with no significant results reported. Overall, earlier symptoms of depression appeared to be associated with poorer metabolic control, with this association decreasing over time.

Metabolic control measured in response to a diagnosis of depression: Direct analysis. Two studies described the direct relationship between a diagnosis of depression and HbA1c. The study by Colton and others (2013) described above reported that a history of depression over a 5-year period or current diagnosis was not associated with poorer metabolic control compared to those who were not depressed. However the groups were not of equal

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size (n=30 history or diagnosis of depression; n=68 no history or diagnosis). Kovacs and colleagues (1996) reported similar findings where a diagnosis of major depressive disorder had no significant longitudinal association with HbA1c over an average period of 8-years. The authors suggested that depressive symptoms elicit caring behaviours from carers who then support the young person with diabetes self-care tasks, hence serving as a protective factor for metabolic control. However a measure of parental/carer involvement was not implemented to assess this formally. Diagnostic status was monitored throughout the study however, similar to Colton et al., a relatively small number (n=24) had a diagnosis.

In summary, a current or previous diagnosis of depression does not appear to be associated with HbA1c over a longer period of up to 10-years. However findings are based on small sample sizes thus reducing power and risking a type 2 error.

Membership to metabolic control subgroup as predicted by depressive symptoms. Of the three studies describing whether depressive symptoms predicted membership to a subgroup of metabolic control, Hilliard and colleagues (2013) identified subgroups using data from trajectories over 24-months. Participants belonged to one of three subgroups identified as: meeting treatment targets, normatively similar (HbA1c moderately out of range), and high risk (HbA1c severely deviant from targets). Higher levels of depressive symptoms at baseline significantly predicted membership of the ‘normatively similar’ subgroup at 18 – 24 months compared to membership of the meeting treatment targets group ($p < 0.05$), but not to the high risk group. However negative emotions specifically related to blood glucose values as measured by the Blood Glucose Monitoring Communication Questionnaire (Hood, Butler, Volkening, Anderson, & Laffel, 2004) predicted membership to the high risk group compared to the meeting treatment targets group. According to the trajectories, membership of subgroups did not change longitudinally. However the authors report that this was

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inconsistent with previous findings where deteriorations over time were noted, thus attributing their findings to a shorter follow-up period compared to previous studies. The authors note that the diverse HbA1c values of the sample provide an accurate representation of the wider population. On the other hand they comment that the sociodemographic characteristics of the sample do not reflect those of the population from which they were recruited as those who participated were predominantly White American, with minority groups being proportionally under-represented.

In a study identifying trajectories of HbA1c over 5-years, Helegson et al. (2010) reported that depressive symptoms at baseline were not found to influence membership to the two trajectories identified ('stable/good control' and 'poor control'). Negative emotions related to diabetes increased the risk of membership to the 'poor control' group however this was based on a single item developed by the researchers and not a standardised questionnaire. The authors acknowledged the possibility of identifying additional trajectories however fluctuations in HbA1c prevented this. In comparison to the other articles reviewed, the authors do not provide participant characteristic data (demographic and diabetes specific) but instead refer readers to the original paper on which the follow-up is based. Despite collecting data over a period of 5-years, changes in the variables overtime, including depressive symptoms, were not reported and this was recognised as a limitation in addition to the lack of ethnic diversity within the sample.

In predicting membership to a metabolic control subgroup ('good control', 'poor control' or 'hypoglycaemia only'), baseline parental reports of internalising behaviour including depressive symptoms were not found by Northam and others (2004) to be associated with metabolic control over a period of 10-years. The paper provided a scarce description of the sample, ignoring ethnicity and details reported in the other reviewed articles

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(e.g. marital status of caregiver). Of the studies described in this review, this study used a relatively small sample however participants were reported to be representative of the target population. Data were described over the longest follow-up period, which has disadvantages in terms of attrition rates; therefore the paper can be praised for a clear comparison of results between participants lost at follow-up.

In summary, at 24-months higher levels of baseline depressive symptoms were associated with subgroup membership where metabolic control was moderately out of range but not high risk. This could suggest that depressive symptoms have a relatively mild effect on HbA1c over time whereas diabetes specific negative emotions, assessed using formal and informal measures, are associated with a more severe effect on metabolic control. Associations ceased to exist at later follow-ups however these were based on parental reports and where fewer trajectories were identified.

Metabolic control measured in response to depressive symptoms as part of predictor model(s). Four studies described models of variables including depressive symptoms to predict HbA1c. As these studies describe the influence of additional variables, the findings are more relevant to be discussed with regard to the second aim of this review.

Depressive symptoms or diagnosis of depression explored in response to metabolic control. In a study aiming to identify predictors of psychological adjustment to T1D, Kovacs and colleagues (1990) found no significant association between metabolic control and depressive symptoms over a 6-year period. However, follow-up intervals were irregular, which the authors attribute to logistical difficulties. The paper can be praised for a detailed description of the sample, including documentation of cognitive ability, and for providing information on participants who withdrew from the study. Average CDI scores at each assessment point over time were reported. However this revealed attrition rates at each

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follow-up and demonstrated relatively small numbers at the final follow-up (n=24). Similar to the other articles reviewed, the sample was not representative of the wider target population.

Northam and colleagues (2004) reported no significant association between HbA1c scores over 10-years and a psychiatric diagnosis (including mood disorder) at the 10-year follow-up. However 'mood disorder' included major depressive disorder in addition to other mood related diagnoses (Dysthymia and Mania/Hypomania). The authors do not provide details on how many were diagnosed with individual disorders or how changes in HbA1c may have been associated with mood/depressive symptoms. Assessing pre-existing emotional difficulties and also following a cohort of participants from diagnosis of T1D is a strength of the study. The authors recognise weaknesses with regard to a small sample size resulting in limited statistical power.

In summary, historical HbA1c values are not associated with later outcomes of depressive symptoms or a diagnosis of depression. However, these findings are based on a limited number of papers that reported findings on small sample sizes.

(ii) Identify factors in the literature that influence the relationship between depressive symptoms and metabolic control over time. Articles exploring the influence of additional variables including depressive symptoms on metabolic control over time will be discussed (n=9). All articles investigated the impact of historical depressive symptoms on later values of HbA1c. Articles will be discussed in respect to whether depressive symptoms were entered alongside other variables in predictor models of HbA1c (n=6) or whether mediating and moderating variables were explored within the longitudinal relationship between depressive symptoms and HbA1c (n=3).

Metabolic control measured in response to depressive symptoms as part of predictor models. In a model of predictor variables that were significantly correlated with blood glucose

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monitoring (BGM), Guilfoyle and colleagues (2011) reported that depressive symptoms significantly predicted HbA1c at 3-months ($p<0.05$) and accounted for 4.76% of the variance in HbA1c scores ($p<0.01$). BGM frequencies, as reported by carers, accounted for 13.42%. The use of objective reports of BGM (i.e. carer reports) was recognised as a limitation although the difficulties with using BGM meter data was acknowledged. This study can be praised for describing a sub-sample used in some of the analyses who had complete data from BGM meters ($n=90$). It is also one of the few papers in this review to clearly state conducting post hoc analyses. However results were based on the shortest follow-up period from the reviewed studies (3-months). As HbA1c values reflect the past 8 – 12 weeks, conclusions regarding the predictive value of depressive symptoms for HbA1c over time based on these results should therefore be made with caution.

In a regression model predicting HbA1c at 12-months, Hilliard and colleagues (2011) found that depressive symptoms were not a significant predictor of HbA1c but did significantly predict BGM. This was explained in terms of the behavioural features of depression (e.g. poor motivation, lethargy) hindering the upkeep of self-management tasks. It is of interest that those who reported higher depressive symptoms at baseline received more visits from mental health professionals. When the number of visits was added to the model, depressive symptoms no longer predicted BGM at 12-months suggesting that psychological treatment to reduce depressive symptoms increased self-management as measured by BGM.

Ingerski and others (2010) found that after controlling for demographic and disease specific variables, the regression model of baseline depressive symptoms was not found to be a significant predictor of HbA1c at 6-months. Significant predictors of HbA1c at 6-months included adolescent reports of insulin delivery mode ($B=-0.21$, $p<0.05$), duration of illness ($B=0.17$, $p<0.05$), and adolescent ($B=0.19$, $p<0.05$) and parental ($B=0.31$, $p<0.001$) reports of

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family conflict. Again a limitation of this study is with regard to the participants with the authors highlighting the predominantly white, female sample from high functioning families. They also acknowledge the influence of social desirability as a risk of bias from families. A strength of the paper is the description of the characteristics of the sample both at baseline and follow-up.

Depressive symptoms were described as being “marginally significant” (p. 263) by Helgeson and others (2009) in predicting HbA1c over 4-years. However this association was influenced by other variables including bulimic symptoms ($B=0.27$, $p=0.01$), body mass index at baseline ($B=0.04$, $p=0.01$) and negative relationships with peers ($B=0.35$, $p=0.01$). In terms of the latter, negative relationships with friends also predicted changes in HbA1c over time ($B=0.49$, $p<0.001$). These findings were discussed with regard to the distress associated with turbulent peer relationships negating from self-care behaviours. The authors concluded that the model suggests the association between HbA1c and depressive symptoms over time is not independent from the influence of other factors.

In another study exploring eating disorders, Colton and colleagues (2013) found that after controlling for BMI at 5-years and duration of T1D, a multiple regression model revealed that CDI scores and eating disorder related psychopathology did not predict HbA1c at 5-years. Disturbed eating behaviour was associated with depressive symptoms but not HbA1c. The differences in these results and those reported by Helgeson and others (2009) may be associated with Colton’s study including a range of eating disorders as opposed focussing solely on bulimia.

A logistic regression model by Hilliard et al. (2013) of demographic, psychological and family predictor variables demonstrated a number of factors predicted membership to subgroups of metabolic control. In terms of psychological predictors, HbA1c levels that were

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severely out of range throughout the study duration were associated with negative emotions specifically related to blood glucose values as measured by the Blood Glucose Monitoring Communication Questionnaire (BGMCO; Hood et al., 2004). These diabetes specific negative emotions predicted membership to the high risk group over the meeting treatment targets group whereas higher depressive symptoms predicted membership of the normatively similar group compared to the meeting treatment targets group. Diabetes specific family conflict also predicted this in addition to predicting HbA1c levels that were severely deviant from the target range ($p < 0.01$). Separate assessment of diabetes specific distress is a clear strength of this study.

In summary, the literature suggests that depressive symptoms are not exclusive of other variables in their involvement in HbA1c outcomes at follow-up. From the literature reviewed, the variables that were found to be part of significant models were mostly associated to diabetes specific factors, particularly with regard to self-care i.e. frequency of BGM and negative emotions related to self-care. There also appears to be some suggestion for the role of other disorders (eating disorders) as well as systemic and relational influences on metabolic control.

Metabolic control measured in response to the influence of mediator/moderator variables. Wu and colleagues (2013) entered the frequency of BGM at 12-months as a mediator of the relationship between depressive symptoms and parental involvement to predict HbA1c. Findings suggested that parental reports of adolescent depressive symptoms at baseline influenced the relationship between adolescent perceptions of parental involvement as reported at baseline (measured using the Diabetes Family Responsibility Questionnaire; Anderson et al., 1990) and BGM to impact on HbA1c at 18-months. However the influence of depressive symptoms on the relationship between parental involvement and BGM was only

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significant when fewer symptoms of depression were reported on the CDI (less than or equal to 6). As self-reports of depressive symptoms were not associated with HbA1c at follow-up there may be a suggestion that parental perceptions of the child and the nature of the caregiver-child relationship is a factor to consider when interpreting results. Obtaining child and parental reports was reported as a strength of the study however no comment was made on the discrepancy between the two reports of depressive symptoms.

In a post-hoc analysis Hood, Rausch and Dolan (2011) found that the interactions between contextual variables and CDI change scores were not moderators of change in HbA1c over time. However as depressive symptoms increased, higher frequencies of BGM at baseline moderated the effect of depressive symptoms on HbA1c. The positive influence of BGM reduced the risk of raised HbA1c levels. The authors comment on the protective role of BGM as an indicator of self-care levels in reducing the effects of depressive symptoms on HbA1c over time. Conversely, a decrease in BGM results in depressive symptoms having a greater effect, causing HbA1c levels to increase. The interaction between the variables was demonstrated with baseline HbA1c levels interacting with baseline BGM and change in CDI to predict change in HbA1c over time ($p < 0.001$).

In a study examining the effects of stressful life events on HbA1c over a period of 5-years, Helgeson, Escobar et al. (2010) found that depressive symptoms were associated with metabolic control over time ($p < 0.05$). However this association was only reported when the depressive symptoms were predicted by stressful life events. Depressive symptoms were not identified as a significant mediator in the relationship between stressful life events and metabolic control. An association was identified between BGM and metabolic control over time when stressful life events predicted BGM. However BGM was not found to be significant in mediating the relationship between stressful life events and HbA1c. The authors

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raise caution to the generality of findings due to the relatively homogenous sample not representing the ethnic diversity of the target population. They also comment on how results based on annual assessments may be a limitation.

In summary, the relationship and interactions between BGM and depressive symptoms appear to have an impact on HbA1c over time. There is also evidence regarding external stressors exacerbating depressive symptoms to influence HbA1c.

Discussion

This review explored the evidence base for the longitudinal relationship between depressive symptoms and metabolic control in adolescents with T1D. Literature was reviewed and discussed in terms of findings regarding directionality in the associations between depressive symptoms and metabolic control and identifying factors that have a significant influence on this relationship.

Directionality in the longitudinal relationship between depressive symptoms and metabolic control. With regard to clarification on issues of directionality, the literature revealed mixed results. Where later depressive symptoms or depression status was explored in response to earlier HbA1c values, no significant results were identified. However this was based on a small number of articles (n=2). The majority of the papers identified in this review explored historical depressive symptoms and later values of metabolic control, again revealing mixed findings. There was some support for associations between depressive symptoms at baseline and metabolic control at follow-up (e.g. Wu et al., 2013; Hilliard et al., 2013) however there was evidence that these associations decrease over time. For example, it was suggested that the predictive value of depressive symptoms on HbA1c is limited to earlier follow-ups, decreasing after a year (Helgeson et al., 2009). Conversely, results describing associations that remain fairly consistent overtime were also reported (Hilliard et al, 2013).

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The main conclusions regarding significant associations were attributed to the behavioural symptoms of depression with lack of motivation creating a barrier to self-care and thus resulting in poor metabolic control. It was interesting to note that negative emotions specifically related to T1D were found to have a greater impact on metabolic control than depressive symptoms (Hilliard et al., 2013). This suggests that metabolic control may be influenced to a greater or lesser extent depending on the cause of distress.

The inconsistent findings are possibly the result of differences in design, such as variations in the method of assessing depressive symptoms. Similarly, differences emerged where metabolic control was grouped into categories representing the degree of control (e.g. good control, poor control) or where HbA1c and changes in values were described. Furthermore variances in terms of study duration and number of follow-ups conducted prevented direct comparison between studies.

Factors influencing the longitudinal relationship between depressive symptoms and metabolic control. Disturbed eating behaviour, relational issues with caregivers and stressful life events were found to be involved in the relationship between depressive symptoms and metabolic control. The extent of their influence varied and was not consistent across the articles reviewed. The factor that appeared to be most common in influencing the relationship of interest across the literature was BGM, including negative emotions related to BGM. The reported capacity of its influence varied. For example, there was support for BGM as a moderator between depressive symptoms and HbA1c where depressive symptoms were found to predict BGM, which in turn would influence HbA1c values. Decreased BGM appeared to be the result of decreased motivation levels, a common symptom of depression. However, much of the research in this review only explored associations up to 12-months therefore conclusions regarding longer periods of time cannot be made. Furthermore the role

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of BGM was not independent of external factors such as stressful life events, family conflict and parental involvement (Guilfoyle et al., 2011).

The findings regarding BGM appear to be mixed and demonstrate the complex interactions between BGM, depressive symptoms and additional factors on metabolic control. The protective role of BGM within the relationship was highlighted and was consistent with previous research, where increased BGM leads to increased levels of HbA1c (Hood, Peterson, Rohan, & Drotar, 2009). In a previous review, Borus and Laffel (2010) identified factors in addition to depression that may impact on HbA1c by influencing self-care. These included non-modifiable variables such as age, gender and illness duration, and modifiable factors such as the role of peers and family. Not all of these factors were identified in the current review, which again may reflect the limited number of articles reviewed.

Limitations. Limitations of the reviewed research were similar to those noted in other systematic reviews within T1D. For example, Barnard, Skinner and Peveler (2006) commented on the lack of control groups increasing the risk of recruitment bias as those consenting may be doing so in the hope that it will be beneficial for their treatment. The absence of groups for comparison purposes also increases the difficulty in explicating whether the associations found would be dissimilar to children with depressive symptoms but without T1D, thus reducing the generality of the findings.

The overall quality of the literature was not a concern however the similar scores on the critical appraisal tool for outcome measures and the sample could be associated with papers reporting data from the same sample in prospective studies. Also with regard to participants, the majority of papers reported limitations in terms of having samples that were not representative of the population from which they were recruited, limiting the applicability of findings to wider contexts. In addition to this, the sample sizes were relatively small thus

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limiting the power of studies. Furthermore, the adolescent period comes with its own demands, independent of T1D. These challenges include hormonal and emotional changes, developing a sense of self and becoming more independent and autonomous (Cameron, 2006; Grey et al., 2002). However these factors were not acknowledged in the reviewed articles.

In terms of the measures used, all of the studies utilised HbA1c as an indicator of metabolic control, however Hilliard et al. (2013) comment that HbA1c fails to reflect more severe difficulties such as hyper- and hypoglycaemia. The self-report measures of depressive symptoms were validated for use within the adolescent population yet formal assessment by clinicians may have provided more comprehensive information (McGrady & Hood, 2010).

Despite being longitudinal in the sense of monitoring changes over time, a number of studies reported results over a short follow-up period (e.g. Guilfoyle et al., 2011; McGrady & Hood, 2010). Where longer follow-up periods were reported, results and changes over time were not fully described (Helgeson et al., 2010), possibly risking the discovery of patterns and associations.

Implications for clinical practice and research. Despite the outcomes of this review presenting mixed results, the vulnerability of the target population was evident. In line with implications from previous research (e.g. McGrady, Laffel, Drotar, Repaske, & Hood, 2009; Stewart, Rao, & White, 2005) and the recommendations in the reviewed papers, the current review suggests the need for assessment, regular monitoring and consequent treatment of depressive symptoms in adolescents with T1D. The aim would be to reduce the negative influence of depressive symptoms on diabetes related outcomes, including self-care and BGM. Hilliard and colleagues (2011) emphasise how screening questionnaires can take as little as ten minutes to complete yet have important implications for treatment.

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Early screening of emotional difficulties and additional problems (e.g. disturbed eating behaviour) is recommended to prevent further difficulties (Colton et al., 2013). The administration of parental reports of adolescent emotional functioning alongside adolescent self-reports will facilitate the identification of difficulties that might be missed or not disclosed by the adolescent. Additionally, the use of continuous scores as opposed to cut-off scores is recommended to ensure that underlying difficulties are not overlooked (Hilliard, Herzer et al., 2011) as even mild symptoms of emotional distress affect the ability to adapt to T1D (Kovacs et al., 1990). Aside from screening for depressive symptoms, monitoring and encouraging discussions regarding the impact of stress on T1D by healthcare professionals is suggested (Helgeson, Escobar et al., 2010).

Frequent monitoring allows for early intervention and preventative techniques, such as coping skills training (Hood, Rausch & Dolan, 2011). Family-based interventions are also helpful (Hilliard et al., 2013; Wu et al., 2013) with particular regard to improving BGM (Hood, Rausch & Dolan, 2011). These interventions involve promoting the involvement of caregivers to share responsibility of self-management tasks in addition to reducing diabetes-related family conflict (Ingerski et al., 2010).

Further longitudinal research on the relationship between depressive symptoms and metabolic control is warranted with consideration of the impact of variables that have been found to influence this relationship (e.g. peer and family relationships, BGM, disturbed eating behaviour). Research focuses on how depressive symptoms lead to poor metabolic control, suggesting a need for more studies investigating how metabolic control can exacerbate depressive symptoms (Stewart, Rao & White, 2005). It would be helpful for future work to build on the limitations of previous studies by aiming to recruit representative samples and

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conducting more frequent follow-ups, allowing for the identification of changes and fluctuations that might otherwise go undetected.

Conclusions. There are inconsistencies in the literature regarding the long-term relationship between depressive symptoms and metabolic control in adolescents with T1D. What is clear is that this relationship is not independent from the influence of other factors. Although there is growing research investigating issues regarding metabolic control and depressive symptoms in adolescents with T1D, few studies report the nature of this relationship longitudinally, with fewer actively monitoring this interaction at multiple points over time. Confirmation of associations between depressive symptoms and metabolic control, alongside the absence of rigorous scientific research exploring directionality, cannot be stated with confidence.

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CHAPTER TWO

THE ROLE OF NEGATIVE AUTOMATIC THOUGHTS AND SELF-EFFICACY IN DEPRESSIVE SYMPTOMS IN ADOLESCENTS WITH TYPE 1 DIABETES

Abstract

Background. Beck's cognitive theory of depression (1967) posits that negative automatic thoughts (NATs) guide responses to specific stimuli and lead to depression. Bandura's social cognitive theory (1997) proposes that depression stems from the beliefs an individual holds regarding their competence to achieve a particular goal (self-efficacy). Adolescents with Type 1 diabetes (T1D) are particularly vulnerable to developing depression.

Aims. This study explored the involvement of NATs and self-efficacy in depressive symptoms in adolescents with T1D. The influence of general and diabetes specific stress was considered, as was general and diabetes specific self-efficacy.

Method. A cross-sectional questionnaire based study was conducted. Participants were 54 adolescents with T1D aged between 13 and 18 years old.

Results. Higher levels of stress were associated with depressive symptoms and NATs. General stress was associated with general self-efficacy whereas diabetes specific stress was associated with diabetes specific self-efficacy. General self-efficacy and NATs were associated with depressive symptoms. However, NATs, general and diabetes specific self-efficacy did not mediate the relationship between stress and depressive symptoms.

Conclusions. The findings did not support the mediatory role of the cognitive constructs proposed by Beck and Bandura. Further research exploring the cognitive mechanisms that underlie depressive symptoms in adolescents with T1D is required.

Introduction

Type 1 diabetes. Type 1 diabetes (T1D) is an autoimmune disorder where the insulin producing beta cells in the pancreas are destroyed, creating a permanent deficiency of insulin (American Diabetes Association, 2013). The trend in prevalence rates suggests that the number of adolescents diagnosed with this chronic disease is rising yearly across Europe by an average of 3 – 4% (Patterson et al., 2012) with approximately 26 500 children and young people with this diagnosis in the UK alone (Diabetes UK, 2012). The management of T1D requires a complex treatment plan involving self-care tasks such as monitoring blood glucose levels, regulating and adjusting insulin via a specified mode of treatment (i.e. injections or insulin pump), and making important dietary adaptations and lifestyle changes to enhance general health (Silverstein et al., 2005). The adolescent period is a complex developmental stage in which many changes occur (e.g. hormonal, physical) in addition to other challenges experienced by adolescents such as concerns regarding acceptance within the peer group, developing an identity, and a growing desire for autonomy and independence (Cameron, 2006). It is perhaps for these reasons that the demanding self-management regimen of T1D is less of a priority for some adolescents and why a decline in diabetes related outcomes is often observed during this time (Bryden et al., 2001).

Depression. Depression is a mood disorder characterised by a cluster of behavioural, cognitive and somatic features including loss of interest in activities, thoughts of worthlessness and hopelessness, disturbances to regular patterns of sleep, changes in appetite and activity, and more overtly, low mood (American Psychiatric Association, 2013). The number of children and adolescents with a diagnosis of depression in the UK is estimated to be approximately 80 000 (Green, 2005). The extent to which depression affects this population extends to poor academic performance and social difficulties (Verboom, Sijtsema,

Verhulst, Penninx, & Ormel, 2014), increased risk of self-harm (Claes, Luyckx, & Bijttebier, 2014) and consequently suicide (Brent et al., 1993).

Type 1 diabetes and depression. Adolescents with T1D are at increased vulnerability to developing depressive symptoms (Johnson, Eiser, Young, Brierley, & Heller, 2013) and have demonstrated higher rates of depression compared to the general adolescent population (Hood et al., 2006; Kanner, Hamrin, & Grey, 2003; Whittemore et al., 2002). The co-existence of depression in adolescents who have T1D has been reported to have negative connotations for the management of the illness (Hood et al., 2006), a possible consequence of the behavioural features such as a reduction in motivation interfering with the ability to perform self-care tasks (Lin et al., 2004). Depression can also hinder the initial process of adapting to the diagnosis (Lernmark, Persson, Fisher, & Rydelius, 1999) also leading to a reduction in diabetes self-care activities (Kovacs, Goldston, Obrosky, & Iyengar, 1992). Poor self-care creates further diabetes specific complications such as increased risk of developing retinopathy (Kovacs, Mukerji, Drash, & Iyengar, 1995) and, ultimately, hospitalisation (Stewart, Rao, Emslie, Klein, & White, 2005). The severity of the complications associated with depression in this population is evident particularly as adolescents with T1D are at increased risk of suicide (Goldston, Kovacs, Ho, Parrone, & Stiffler, 1994).

A number of hypotheses have been proposed offering explanations for the development of depression in T1D. One possible reason is related to the highly demanding nature of the treatment and maintenance of self-management leading to difficulties in meeting treatment targets. This can create low mood which further prevents the meeting of treatment demands through the reduced motivation associated with depression (Lustman & Clouse, 2005). Other hypotheses include the stress of having a chronic illness and the negative consequences that occur as a result of such a condition such as illness specific cognitions

affecting resilience and the ability to cope (Nouwen, Urquhart Law, Hussain, McGovern, & Napier, 2009; Talbot & Nouwen, 2000).

The recommended psychological treatment for depression in young people is cognitive behavioural therapy (Department of Health, 2005), which places focus on altering the dysfunctional cognitions characteristic of depression in order to change the associated negative affect (Beck, 1963). The emphasis on the role of cognitions in the treatment for depression suggests the need for consideration of cognitive theories and the underlying psychological mechanisms of depression in order to facilitate an understanding of its development in adolescents with T1D. Two leading cognitive theories of depression are Beck's cognitive theory (1967) and Bandura's social cognitive theory (1997).

Beck's cognitive theory of depression. Beck's (1967) cognitive theory of depression posits that depression is the result of negative automatic thoughts representing underlying dysfunctional core beliefs developed in childhood. Core beliefs are defined as cognitive patterns that guide responses to a specific set of stimuli. In an individual with depression, the core beliefs include negative beliefs (cognitive distortions) that form the cognitive triad of depression (Beck, 1976) and skew the way they think about themselves, the world and the future. The relationship between core beliefs and behaviour is mediated by conditional statements often in the form of 'if...then...' statements labelled as dysfunctional assumptions that are developed from negative core beliefs (Greenberger & Padesky, 1995). When presented with a stressful event, the negative core beliefs are triggered and, through dysfunctional assumptions, lead to the production of negative automatic thoughts. These cognitions are rapidly available within the conscious mind and offer a negatively tinged evaluation of events (Westbrook, Kennerley & Kirk, 2007). Negative automatic thoughts that are fuelled by and represent the core beliefs that form the cognitive triad of depression lead to

the development of depressive symptoms. Figure 1 illustrates the causal pathway to depression as suggested by Beck's model.

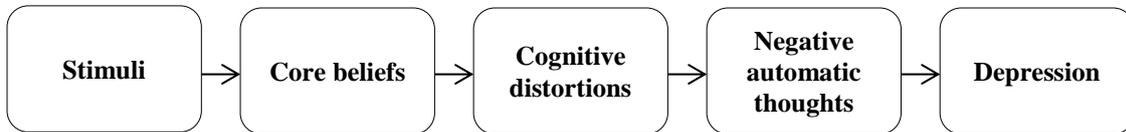


Figure 1. The pathway to depression as proposed by Beck's cognitive model of depression.

The validity of the mediating cognitive constructs in Beck's model has been investigated with regard to the development of depression in adolescents. Moilanen (1995) found that higher levels of depressive symptoms were associated with a greater degree of dysfunctional assumptions, increased frequency of negative automatic thoughts and negative core beliefs. A stronger endorsement of dysfunctional assumptions was also related to greater levels of depressive symptoms in a study where core beliefs and negative automatic thoughts were not examined (Abela & Sullivan, 2003).

In terms of the validity of Beck's model within the diabetic population, Clarke and Goosen (2009) investigated the role of regulating emotional stress in the development of depression in adults with diabetes. The findings showed that emotion focussed coping (i.e. strategies that attempt to regulate emotional stress) played a mediating role in the relationship between negative automatic thoughts and depression. Consistent with the cognitive model, the frequency of negative automatic thoughts was positively correlated with depressive symptoms. However the sample was not exclusive to those with T1D, but also included people with Type 2 diabetes. Furthermore, the age range of the sample (28 – 88 years) suggested a wide representation of the population of adults with diabetes but added little to clarify mechanisms in adolescents with T1D. Farrell and colleagues (2004) also reported that an increase in depressive thinking styles, mainly cognitive distortions in the form of negative

automatic thoughts, were associated with increases in the level of diabetes specific and general stress reported by adolescents. General stress was thought to influence self-management behaviour whereas diabetes specific stress was found to affect metabolic control directly, although the exact mechanisms of this relationship were not clear (Farrell, Hains, Davies, Smith, & Parton, 2004). The results suggest support for the role of the cognitions proposed by Beck in depression in T1D, however behavioural changes were measured (e.g. self-management) as opposed to depressive symptoms in response to distorted cognitions.

Bandura's social cognitive theory. Self-efficacy is a major component of Bandura's (1997) social cognitive theory. Self-efficacy refers to the beliefs an individual holds regarding their competence to achieve given courses of action. According to this theory, depression results from the belief that one is inefficacious to gain valued outcomes. Figure 2 illustrates the pathway to depression as suggested by Bandura.

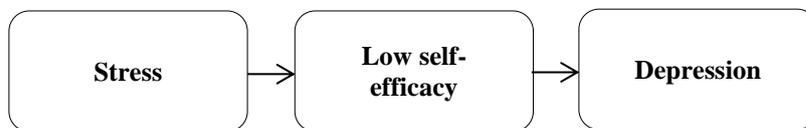


Figure 2. The pathway to depression as proposed by Bandura's social cognitive theory.

Bandura's theory has been tested in the general adolescent population. Consistent with the model, lower self-efficacy has been reported to be significantly associated with greater depressive symptoms (Muris, 2002). Similarly, Ehrenberg and colleagues (1991) found that self-reported levels of depression were negatively correlated with perceived general self-efficacy in adolescents as well as academic and physical self-efficacy. In terms of the ways in which self-efficacy influences depression in adolescents longitudinally, the results of a path analysis showed that lower self-efficacy (social and academic) was associated with higher levels of depressive symptoms at baseline and at 12-months (Smith & Betz, 2002).

In adolescents with T1D, lower self-efficacy and greater symptoms of depression were present in those who reported that diabetes had a greater impact on their quality of life (Grey, Boland, Yu, Sullivan-Bolyai, & Tamborlane, 1998). Stewart and colleagues (2000) explored self-efficacy as a mediator between emotional distress and metabolic control in adolescents with T1D in a cross-sectional, matched participants controlled study. Emotional distress was found to be significantly correlated with lower self-efficacy, which was associated with diabetes self-care. A follow-up study (Stewart, Wang, Wang & White, 2009) replicated these results demonstrating the relationship between emotional distress and self-efficacy, which was associated with self-care measured through metabolic control. However, metabolic control was the primary outcome measure as opposed to depressive symptoms and the age range of the sample used was relatively broad (9 to 21 years of age), covering varied developmental stages not exclusive to the adolescent period.

Limitations of previous research. The evidence base exploring the role of cognitions readily available in conscious awareness (i.e. negative automatic thoughts and beliefs of self-efficacy) in the development of depressive symptoms in adolescents with T1D is limited. Previous research did not encompass all the factors that are of interest in the current study. For example, although there is support for the model suggested by Beck and the role of negative automatic thoughts in depression in adolescents (Moilanen, 1995), this work has not been fully extended to adolescents with T1D. The mediating role of emotion-focused coping in the relationship between negative automatic thoughts and depression has also been demonstrated (Clarke & Goosen, 2009), but only within an adult sample. Self-efficacy has been found to be associated with metabolic control in adolescents with T1D (Grossman, Brink & Hauser, 1987), but little is known about its relationship to depression within this population. In children without diabetes, self-efficacy is negatively correlated with depression

(Ehrenberg, Cox & Koopman, 1991), however no distinction has been made between general self-efficacy and self-efficacy specifically related to diabetes. Despite some of the papers incorporating measures of emotional adjustment, depressive symptoms were not always evaluated as the primary outcome measure. This is somewhat contrary to what is suggested by the respective models proposed by Beck and Bandura which recognise depression as a consequence of the mediating cognitive constructs. The previous literature also fails to differentiate between whether the depressive symptoms were the result of general stress or the stress experienced as a result of having T1D.

In summary, the extent to which specific cognitive processes involved in the respective pathways described by Beck's and Bandura's models of depression, namely negative automatic thoughts and self-efficacy, have not been fully explored in adolescents with T1D. Equally, little attention has been paid to whether depression is a response to general stress or diabetes specific stress within this population. Determining the role of cognitive factors in depressive symptoms in adolescents with T1D could potentially help identify adolescents who may be at risk of developing depression. This will have implications for possible screening procedures and facilitate prevention and treatment interventions.

Aims. This study aimed to investigate the role of cognitions readily available in conscious awareness, with a focus on negative automatic thoughts and self-efficacy, in the development of depressive symptoms within the same sample of adolescents with T1D. The aims of this study were therefore as follows:

- (i) To explore the cognitive mechanisms (negative automatic thoughts and self-efficacy) that underlie depressive symptoms in the respective theories of depression to establish how much the mediating constructs of each theory are involved in depressive symptoms in adolescents with T1D;

- (ii) To investigate whether it is general self-efficacy or diabetes specific self-efficacy that is more strongly associated with depressive symptoms in this sample;
- (iii) To establish the extent to which general stress and diabetes specific stress influence depressive symptoms with regard to the cognitive mechanisms described by Beck and Bandura.

Methodology

Design. The research was a cross-sectional questionnaire based study. This design permitted the investigation of numerous variables at one time point and, as no research has been done in the area of interest, the design allowed for the initial discovery and identification of associations that can then be studied further.

Measures. The self-report measures consisted of a questionnaire for sociodemographic data in addition to six questionnaires relating to the aims of the research.

Sociodemographic information. Demographic and biographic data included age, gender, additional medical problems, history of depression, duration of T1D and treatment of diabetes (See Appendix 1).

Depressive symptoms. The primary outcome measure was depressive symptoms assessed using the Patient Health Questionnaire-9 (PHQ9; Spitzer et al., 1999; See Appendix 2). This is a 9-item scale that assesses depressive symptoms over the past two weeks. Respondents used a 4-point Likert scale ranging from 0 ("Not at all") to 3 ("Nearly everyday") to rate symptoms of depression with higher scores indicating higher depressive symptoms. The measure has been found to be suitable for adolescents (Johnson, Harris, Spitzer, & Williams, 2002; Richardson et al., 2010). Cronbach's alpha for the current sample was 0.82.

General stress. The Adolescent Stress Questionnaire (ASQ; Byrne, Davenport, & Mazanov, 2007; See Appendix 3) was used to assess general stress. The scale lists 58 stressful events and respondents were required to rate how stressful they found events experienced in the past year. Ratings were made using a 5-point Likert scale (1 = Not at all stressful/is irrelevant to me; 5 = Very stressful) with higher scores indicative of higher levels of stress. In addition to producing a total score, there are 10-subscales that assess stress in the following domains: Home Life, School Performance, School Attendance, Romantic Relationships, Peer Pressure, Teacher Interaction, Future Uncertainty, School/Leisure Conflict, Financial Pressure, and Emerging Adult Responsibility. Cronbach's alpha for the total score within the current sample was 0.96 and ranged from 0.71 to 0.89 for the subscales.

Diabetes specific stress. Stress specifically associated with diabetes was assessed using the Problem Areas in Diabetes scale for teenagers (PAID-T; Polonsky et al., 2005; See Appendix 4). Respondents used a 5-point Likert scale to rate 20 items assessing diabetes specific emotional distress (0 = Not a problem; 4 = Serious problem). Higher scores indicated higher emotional distress specific to diabetes. Cronbach's alpha for the current sample was 0.94.

Negative automatic thoughts. In line with Beck's cognitive theory of depression, negative automatic thoughts were assessed using the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980; See Appendix 5). A 5-point Likert scale (1= Not at all; 5 = All the time) was used to rate how frequently respondents experienced 30 different negative automatic thoughts. A higher total score suggests a higher level of negative automatic thoughts. There were also four subscales that assess negative automatic thoughts related to: personal maladjustment and desire for change, negative self-concepts and negative

expectations, low self-esteem, and helplessness. Cronbach's alpha for the total score was 0.93 for the current sample and ranged from 0.41 to 0.82 for the subscale scores.

General self-efficacy. With regard to Bandura's theory, the Self-Efficacy Questionnaire for Children (SEQ-C; Muris, 2001; See Appendix 6) was used to assess general self-efficacy. This 24 item scale focuses on self-efficacy across three domains, namely social, academic and emotional. Respondents were required to rate how well they feel they do in each situation using a 5-point Likert scale (1 = Not at all; 5 = Very well); higher scores indicated greater self-efficacy. A total score is produced alongside three subscale scores. Cronbach's alpha for the total score in the current sample was 0.90 and ranged between 0.81 and 0.85 for the subscale scores.

Diabetes specific self-efficacy. Self-efficacy related to diabetes was assessed using the Self-Efficacy for Diabetes Management Scale (SEDM; Iannotti et al., 2006; See Appendix 7). The SEDM consists of 10 items related to diabetes care. Respondents were required to rate how sure they are that they can do each of the diabetes care related activities using a 10-point Likert scale (1= Not at all sure; 10 = Completely sure). Higher scores indicated stronger self-efficacy related to diabetes specific tasks. Cronbach's alpha for the current sample was 0.86.

Procedure. Ethical approval was obtained from the East Midlands Leicester Health Research Authority National Research Ethics Service (study reference: 13/EM/0066; See Appendix 8). Recruitment took place over eight months from August 2013 to March 2014. Young people who were deemed eligible based on the inclusion criteria were identified through diabetes clinics by their Paediatrician at routine medical appointments. Those who met the inclusion criteria were then provided with an invitation to participate (Appendix 9) and a Participant Information Sheet (Appendix 10) during their appointment. The invitation included a reply slip explaining that they could meet with a member of the research team after

the current appointment or following their next appointment if they wished to take part, in addition to an option to decline to participate. If they wished to take part they met with a member of the research team following their routine appointment to complete the consent procedure. Eligibility was confirmed against exclusion criteria of: (1) having a diagnosis of a psychiatric condition, with the exception of major depression and dysthymia; (2) presence of a symptomatic co-morbid disease requiring treatment or self-management (e.g. asthma, cancer); (3) having a documented neurocognitive disorder; (4) less than 12-months post diagnosis³; and (5) not fluent English speakers. For those who remained eligible and were happy to continue, a consent form was signed (Appendix 11). Participants were requested to complete the questionnaires described above at the diabetes clinic. Participants were given the opportunity to have parents or carers present during the completion of the questionnaires. Following the completion of questionnaires, the HbA1c value from the clinic appointment was recorded. HbA1c is a measure of metabolic control and is the proportion of glucose molecules attached to haemoglobin in the blood and represents an average level of blood glucose over an eight to twelve week period (Rewers et al., 2009).

Participants. Participants were adolescents with a diagnosis of T1D aged between 13 and 18 years who were recruited from five diabetes clinics in the West-Midlands where they were receiving care from a multi-disciplinary team. A total of 99 young people were approached to take part, 21 of whom declined to participate and five left the clinic before discussing the research. Ten young people requested to meet at their next appointment at the clinic, however, due to logistical reasons, this was not possible. Of the remaining young people who consented to participate (n = 63), one withdrew from the study before completing the questionnaires, four failed to fully complete the questionnaires and four were excluded as

³ Depressive symptoms are more prevalent following initial diagnosis. They can resolve between 6 – 12 months post diagnosis but then reappear after 12-months, which may represent the end of the T1D ‘honeymoon period’.

they were approached in error and were either too young, had Type 2 diabetes or had cognitive difficulties that lead to difficulty when completing the questionnaires. Data were therefore collected from a sample of n=54 young people. The social, demographic and diabetes specific characteristics of the sample are presented in Table 1.

Table 1

Sample characteristics.

	N = 54
Gender (N, %)	
Male	28 (51.9)
Mean Age in years (SD)	15.39 (1.73)
Ethnicity (N, %)	
White	24 (44.4)
Asian	18 (33.4)
Black	6 (11.1)
Chinese	1 (1.9)
Mixed background	4 (7.4)
Other	1 (1.9)
Employment/Education status (N, %)	
In education	49 (90.7)
Full time employment	0 (0)
Part time employment	1 (1.9)
Not employed	4 (7.4)
Mean diabetes duration in years (SD)	6.78 (4.11)
Diabetes control (N, %)	
Tablets	2 (3.7)
Insulin injections	41 (75.9)
Insulin pump	11 (20.4)
Mean HbA1c in % (SD)*	10.2 (2.0)
Co-morbid health problems	
Number with additional health problems	12 (22.2)
Depression status (based on self-report)	
No current/previous diagnosis	49 (90.7)
Current/previous diagnosis	5 (9.3)
Currently taking medication for depression	0

*HbA1c values above 14 were not detected by the apparatus used to calculate HbA1c. N=4 had values of >14 therefore this figure is not entirely representative.

Statistical analysis. Sociodemographic information and the scores from the questionnaires were described using descriptive data. The dependent variable in the statistical analysis was depressive symptoms as measured by the PHQ-9. Tests for the normality of the

data and the parametric inference assumptions for all of the variables were undertaken using the Kolmogorov-Smirnov Z test. The results reported that all data were normally distributed apart from the ATQ total score. A logarithmic transformation was used to transform this variable and corrected the deviation in ATQ total scores, which was then found to be normally distributed. Parametric tests were used to analyse the data. Bivariate statistics (Pearsons correlation) were used to explore correlations between all of the variables.

The two theories investigated in this study suggest that cognitions are mediators in the development of depression. Therefore the most appropriate statistical analysis to explore the relationship between stressors and depressive symptoms as mediated by cognitions was a mediation analysis as described by Preacher and Hayes (2008). This method allowed for the examination of a range of pathways including the direct effect of stress on the mediating variables (i.e. negative automatic thoughts and self-efficacy) and on depressive symptoms, and the effect of the mediating variables on depressive symptoms.

The mediational approach was used to test two models: a general stress model and a diabetes specific stress model. For the general stress model, the analysis examined whether negative automatic thoughts, general self-efficacy and diabetes specific self-efficacy mediated the relationship between general stress and depressive symptoms. For the diabetes specific stress model, the analysis examined whether negative automatic thoughts, general self-efficacy and diabetes specific self-efficacy mediated the relationship between diabetes specific stress and depressive symptoms. The variables included in the mediation analysis were transformed into standard scores (mean = 0, SD = 1) to facilitate direct comparison between parameter coefficients within the mediation models. A bias correcting bootstrapping procedure permitted the examination of each model as a whole and the impact of the

mediating constructs on the relationship between stress (general and diabetes specific) and depressive symptoms.

The proposed mediation model for general stress is presented in Figure 3. According to the model proposed by Beck (1967) the relationship between general stress and depressive symptoms is mediated by negative automatic thoughts. In contrast, the model proposed by Bandura (1997) depicts the relationship between general stress and depressive symptoms as mediated by beliefs of self-efficacy, both general self-efficacy and self-efficacy specific to diabetes. Accordingly, the predictions of Beck and Bandura may be directly compared in the mediation model presented in Figure 3. The parameter estimation and the mediation analysis were conducted according to the procedure described by Preacher and Hayes (2004, 2008).

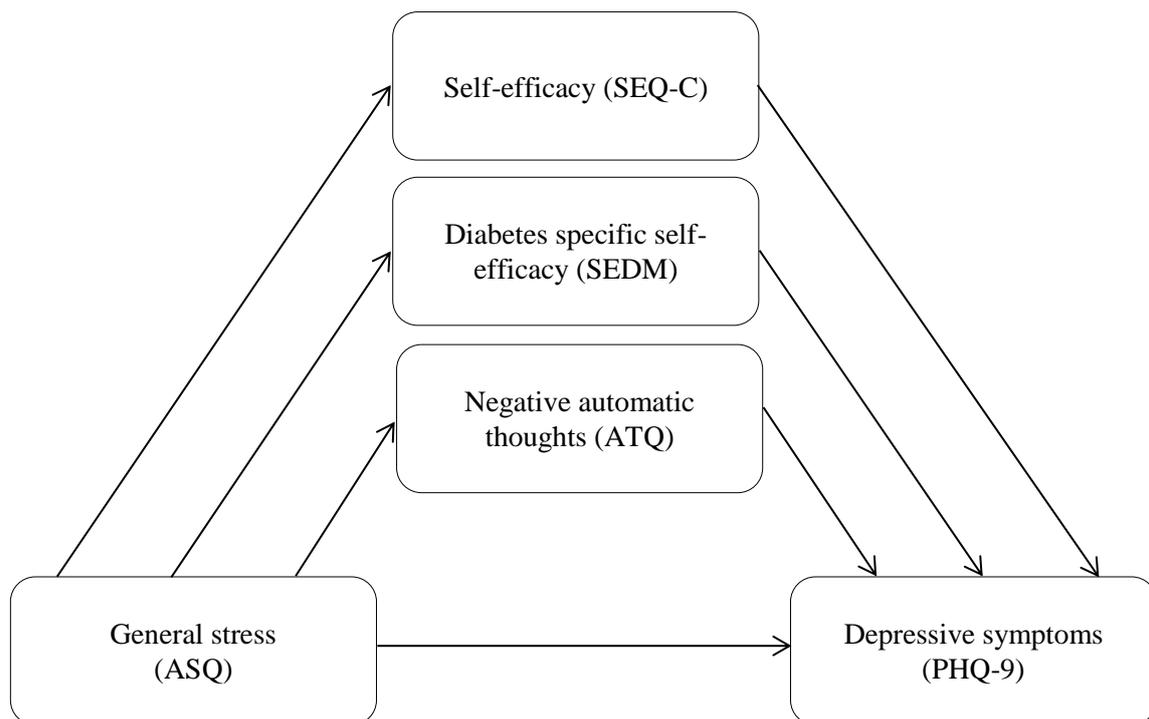


Figure 3: The proposed model of the mediating effects of self-efficacy beliefs and negative automatic thoughts on the relationship between general stress and depressive symptoms.

A second mediation analysis was undertaken to explore the mediating effects of self-efficacy beliefs and negative automatic thoughts upon the relationship between diabetes specific stress and depressive symptoms. The proposed mediation model is presented in Figure 4.

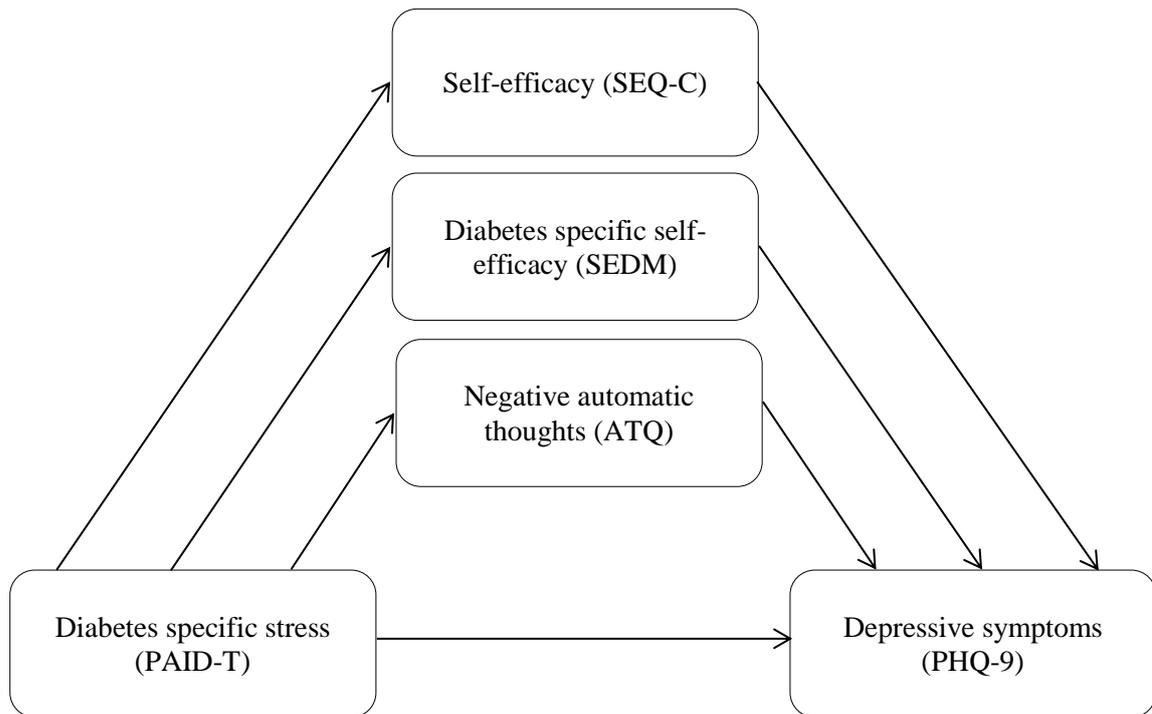


Figure 4: The proposed model of the mediating effects of self-efficacy beliefs and negative automatic thoughts on the relationship between diabetes specific stress and depressive symptoms.

To establish mediation effects, the relationship between stress and depressive symptoms must be significant. There must also be associations between stress and the mediating variables in addition to associations between the mediating variables and depressive symptoms. Mediation effects can only be established if the association between stress and depressive symptoms diminishes after inclusion of the mediating variables. The path coefficients of this model were calculated using a series of ordinary least squares

regressions. The tests of significance of the mediated pathways were calculated using the bootstrap method (MacKinnon & Fairchild, 2009; Preacher & Hayes, 2004, 2008), and the Beta coefficient and associated bias corrected confidence intervals were calculated. The bias correcting bootstrapping technique tested for mediation by using 5000 bootstrapped samples. The mediation tests were reported as ‘indirect effects’ and represented the combined influence of general and diabetes specific stress upon the mediator(s) and the mediator(s) upon depressive symptoms. Mediation tests are considered statistically significant through 95% confidence intervals at $\alpha=0.05$ if the confidence interval does not include zero.

Power calculation. To determine an appropriate sample size based on the planned statistical analysis of a bias-corrected bootstrap mediation analysis, the guidance by Fritz and MacKinnon (2007) was used which outlines the number of participants required for different effect sizes. The current research was interested in robust phenomena that can be utilised clinically therefore medium effect sizes and above were selected. Accordingly, in order to detect a medium effect size to achieve a power of 0.80, a sample of size of $n=71$ was proposed for this study.

Results

Questionnaire scores. The descriptive statistics of the scores for the measures used to assess depressive symptoms (dependent variable), general and diabetes specific stress (independent variables), and the mediating variables of negative automatic thoughts, general self-efficacy and diabetes specific self-efficacy are presented in Table 2. The scores suggest that the current sample reported mild levels of depressive symptoms. The amount of general and diabetes specific stress experienced also appeared to be relatively low. However the maximum score on the ASQ is 290 compared to 100 on the PAID-T suggesting that this sample may have experienced higher levels of diabetes specific stress compared to general

stress. Low self-efficacy, general and diabetes specific, was not reported by the sample as a whole. It is important to note that the standard deviations for all of the respective scores suggested a relatively large degree of variability within the data.

Table 2

Means (SD) on study questionnaire variables.

	Mean	SD
Depressive symptoms (PHQ-9) ⁴	5.41	4.62
General Stress (ASQ)	88.30	31.20
Diabetes specific stress (PAID-T)	64.52	26.08
Negative automatic thoughts (ATQ)	44.26	15.26
General self-efficacy (SEQ-C)	82.61	15.98
Diabetes specific self-efficacy (SEDM)	61.37	17.02

Stress and depressive symptoms. With regard to the relationship between general stress and depressive symptoms, the Pearson's correlation test showed a significant positive correlation ($r=0.70$, $p\leq 0.01$). There was also a significant positive correlation between diabetes specific stress and depressive symptoms ($r=0.72$, $p\leq 0.01$). These results suggest that an increase in general and diabetes specific stress was associated with an increase in depressive symptoms. Both diabetes specific stress and general stress appeared to demonstrate a strong association with depressive symptoms.

Beck's cognitive theory of depression. A significant positive correlation was found between the Beck's negative automatic thoughts and general stress ($r=0.54$, $p\leq 0.01$) and also diabetes specific stress ($r=0.56$, $p\leq 0.01$), both demonstrating moderate associations. In terms

⁴ Cut-off scores corresponding to depression severity on the PHQ-9 (Kroenke & Spitzer, 2002) are as follows: 0 – 4 = None-minimal; 5 – 9 = Mild; 10 – 14 = Moderate; 15 – 19 = Moderately Severe; 20 – 27 = Severe.

of the relationship between negative automatic thoughts and depressive symptoms, the scores from the measures were significantly positively correlated ($r=0.48$, $p\leq 0.01$), indicating that higher depressive symptoms are associated with higher levels of negative automatic thoughts.

Bandura's social cognitive theory. The relationships between general stress and mediating variables revealed a significant negative correlation for general self-efficacy ($r=-0.38$, $p\leq 0.01$), a relatively moderate association, but no significant association with diabetes specific self-efficacy. Conversely, diabetes specific stress was negatively correlated with diabetes specific self-efficacy ($r=-0.33$, $p\leq 0.05$), but not with general self-efficacy. Again the strength of the association was moderate. A weak but significant negative correlation was found between general self-efficacy and depressive symptoms ($r=-0.28$, $p\leq 0.05$). No significant result was found between depressive symptoms and self-efficacy related to diabetes.

Mediation analysis: General stress model. The association between general stress and depressive symptoms before the mediating effects of general and diabetes specific self-efficacy and negative automatic thoughts were considered was significant (Beta = 0.539, 95%CI 0.305 to 0.773). With the inclusion of the three mediating variables the direct link between general stress and depressive symptoms increased to Beta = 1.679 (95%CI 0.524 to 2.835). Negative automatic thoughts was the only variable that was identified as a potential mediator in this analysis as it demonstrated a significant association with stress and depressive symptoms, respectively. However, the direct effect remained significant and none of the mediated paths were statistically significant (Beta = -1.140, 95%CI -2.780 to 0.312). Therefore, neither self-efficacy (general and diabetes specific) nor negative automatic thoughts mediated the relationship between general stress and symptoms of depression (Table 3). The results are presented diagrammatically in Figure 5.

Table 3

Summary of mediation results for self-efficacy and negative automatic thoughts on the relationship between general stress and depressive symptoms.

Mediator	Effect of General Stress on Mediator (95% CI)	Effect of Mediator on depressive symptoms (95% CI)	Indirect effect (95% CI)	Direct effect in mediation model (95% CI)	Total effect (95% CI)
Self-Efficacy	Beta = -0.293 (-0.559 to -0.027)	Beta = -0.069 (-0.320 to 0.181)	Beta = 0.020 (-0.084 to 0.216)		
Diabetes specific self-efficacy	Beta = -0.120 (-0.396 to 0.156)	Beta = -0.138 (-0.380 to 0.103)	Beta = 0.017 (-0.019 to 0.109)		
Negative automatic thoughts	Beta = 0.979 (0.924 to 1.035)	Beta = -1.201 (-2.350 to -0.052)	Beta = -1.177 (-2.88 to 0.23)	Beta = 1.679 (0.524 to 2.835)	Beta = 0.539 (0.305 to 0.773)

Note: See Appendix 12 for SPSS output.

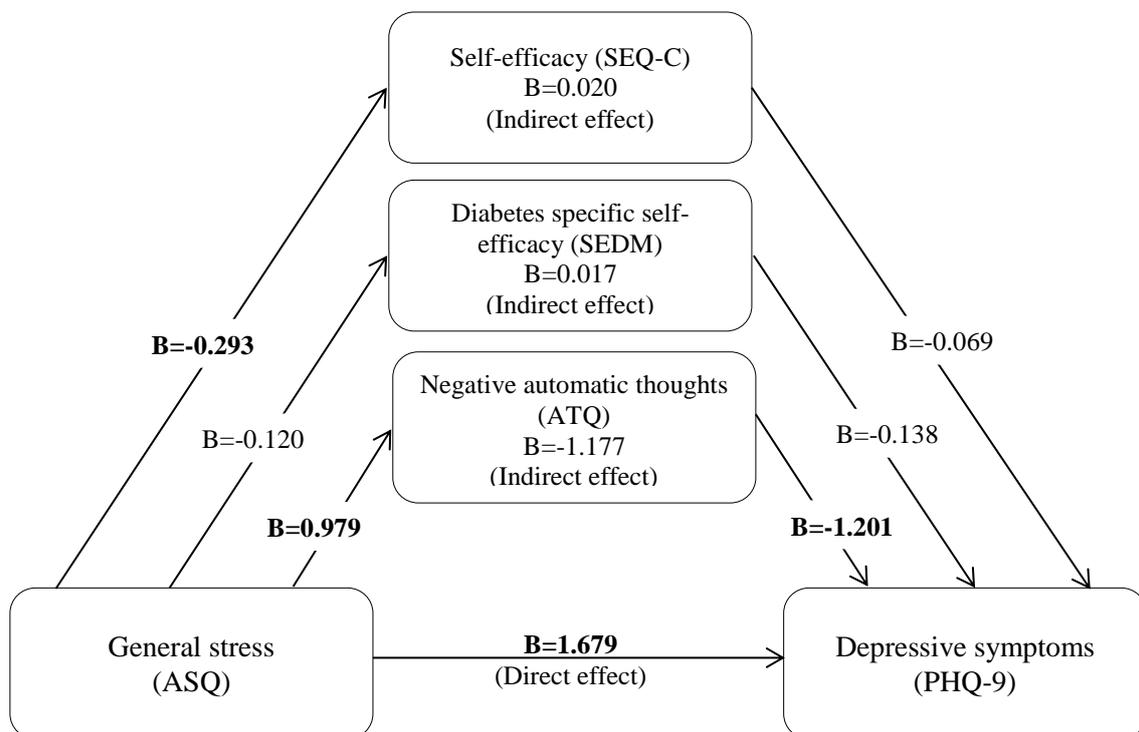


Figure 5. Model demonstrating the mediation results for self-efficacy and negative automatic thoughts on the relationship between general stress and depressive symptoms.

Mediation analysis: Diabetes specific stress model. As with the general model, a significant total effect (Beta = 0.717, 95%CI 0.522 to 0.911) between diabetes specific stress and symptoms of depression was observed before the inclusion of mediating effects of self-efficacy and negative automatic thoughts. When these variables were considered, the direct effect between diabetes specific stress and depressive symptoms were Beta = 0.958 (95%CI 0.409 to 0.9069). Again the direct effect remained significant but none of the mediated paths were statistically significant (Beta = 0.059, 95%CI -0.095 to 0.217) indicating that self-efficacy and negative automatic thoughts did not mediate the relationship between diabetes specific stress and symptoms of depression (Table 4).

Table 4

Summary of mediation results for self-efficacy and negative automatic thoughts on the relationship between diabetes specific stress and depressive symptoms.

Mediator	Effect of Diabetes Specific Stress on Mediator (95% CI)	Effect of Mediator on depressive symptoms (95% CI)	Indirect effect (95% CI)	Direct effect in mediation model (95% CI)	Total effect (95% CI)
Self-Efficacy	Beta = -0.262 (-0.531 to 0.007)	Beta = -0.103 (-0.316 to 0.111)	Beta = 0.027 (-0.030 to 0.277)		
Diabetes specific self-efficacy	Beta = -0.330 (-0.593 to -0.067)	Beta = 0.067 (-0.1492 to 0.2826)	Beta = -0.022 (-0.135 to 0.045)		
Negative automatic thoughts	Beta = 0.559 (0.328 to 0.789)	Beta = 0.096 (-0.145 to 0.337)	Beta = 0.054 (-0.100 to 0.208)	Beta = 0.658 (0.409 to 0.907)	Beta = 0.717 (0.522 to 0.911)

Note: See Appendix 13 for SPSS output.

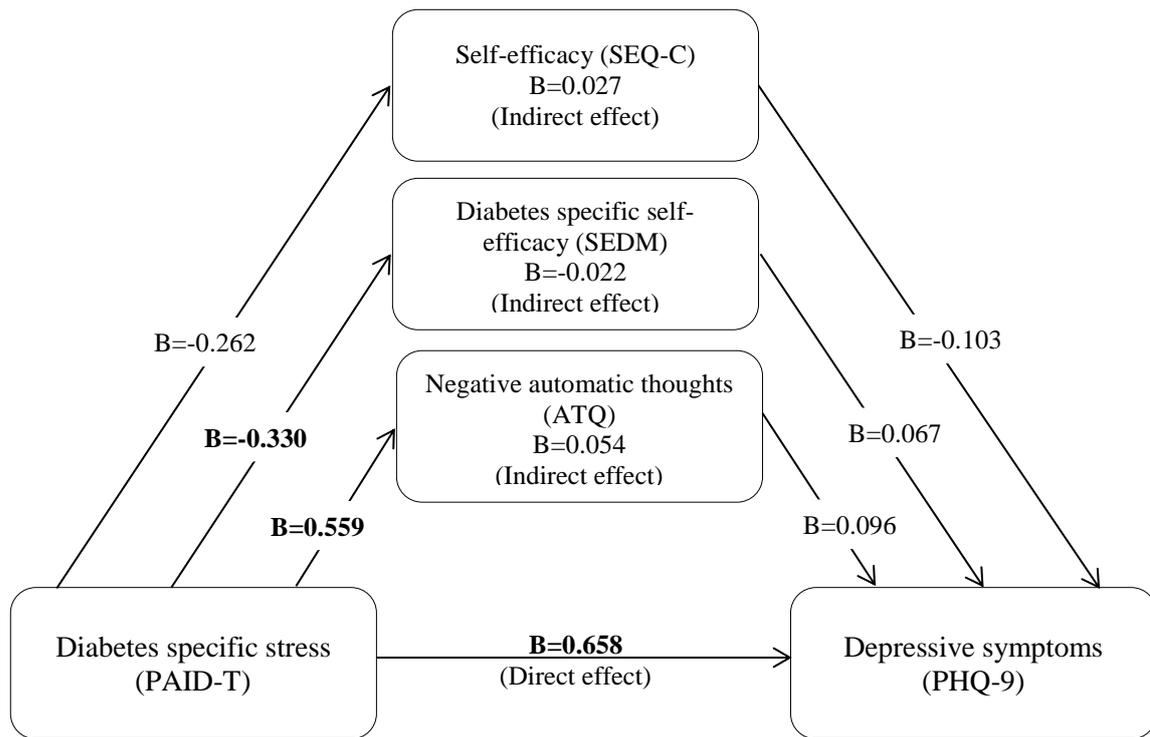


Figure 6. Model demonstrating the mediation results for self-efficacy and negative automatic thoughts on the relationship between diabetes specific stress and depressive symptoms.

Discussion

This study set out to investigate the role of cognitions in depressive symptoms in adolescents with T1D. The mediating constructs of negative automatic thoughts (as proposed by Beck's cognitive theory of depression) and the notion self-efficacy (as per Bandura's social cognitive theory) were explored within the same sample of adolescents with T1D to determine the degree of their involvement in depressive symptoms. The role of general self-efficacy and diabetes specific self-efficacy were also tested to identify the extent to which each type is associated with depressive symptoms, similarly general stress and stress specifically related to diabetes were explored.

The findings demonstrated that both general stress and diabetes-specific stress, independent of the cognitive mediators, were associated directly with depressive symptoms suggesting that higher levels of stress are related to higher reported depressive symptoms. This association remained significant after the inclusion of negative automatic thoughts and self-efficacy (both general and diabetes specific), suggesting that these cognitions did not mediate the relationship between stress and depressive symptoms in this sample. However, associations were identified when examining the individual relationships between stress, mediators and symptoms of depression.

Higher levels of general and diabetes specific stress were associated with more frequent negative automatic thoughts, consistent with previous findings within this population (Farrell et al., 2004). This is consistent with the theory proposed by Beck whereby depression results from negative automatic thoughts fuelled by underlying beliefs that guide responses to a specific set of stimuli, in this case, general stress. However, these cognitions only appeared to be associated with depressive symptoms in the mediation model of general stress. Thus supporting previous results from a non-diabetic sample (Moilanen, 1995) but in contrast to research examining the role of negative automatic thoughts in those with T1D (Clarke & Goosen, 2009). It should be noted that the study by Clarke and Goosen did suggest that there was an extra dimension to the process in the form of emotion focussed coping that mediated the relationship between negative automatic thoughts and depression, a factor that was not explored in the current study. The discrepancies between findings may be a consequence of previous research employing different methodologies and different outcome measures. Despite a positive correlation with depressive symptoms, the lack of association in the mediation model of diabetes specific stress suggested that experiencing stress solely associated with T1D does not activate pre-existing core beliefs represented by negative

automatic thoughts. A possible reason for this may be that the diabetes specific stressors were not strong enough to activate the engrained, underlying core beliefs. Similarly, as the average level of depressive symptoms within the sample was relatively mild, the mediating pathway was perhaps inactive as there were no depressive symptoms in response to stress to mediate.

With regard to self-efficacy, an increase in general stress was only associated with lower beliefs of general self-efficacy, whereas diabetes specific stress was associated with lower self-efficacy related to diabetes specific tasks. This suggests that general stress may have an effect on a broader set of beliefs whereas stress associated with T1D can have a negative impact on the management of the illness as a result of poor self-efficacy and beliefs in the ability to carry out self-care tasks. However the results were not as expected based on Bandura's (1997) social cognitive theory whereby depression results from low self-efficacy as neither type of self-efficacy was associated with depressive symptoms in the mediation analysis. Furthermore, they were not in-line with the previous literature where lower self-efficacy was associated with more symptoms of depression in the general adolescent population (Ehrenberg et al., 1991; Muris, 2002; Smith & Betz, 2002). This may be a result of difficulties with low self-efficacy not being present in this sample in addition to the mild levels of depressive symptoms.

In summary, the findings were mainly inconsistent with the theories proposed by Beck and Bandura in that the only cognitions associated with depressive symptoms in the mediation analysis were negative automatic thoughts but only when the stressor was general stress. Yet, as suggested by the theories proposed by Beck and Bandura, the presence of poor self-efficacy and more frequent negative automatic thoughts are associated with depressive symptoms in this sample. In contrast to the findings of the mediation analysis, previous research has supported the two theories and the mediating roles of the investigated cognitions,

alongside significant associations between stress and the mediating variables. Therefore this population appears to be vulnerable to developing depressive symptoms due to the presence of the mediating variables that are associated with stress, a relationship that appears to be cyclical in nature.

Limitations. The discrepancies between the conclusions drawn from the current study and previous work may be the result of a number of methodological limitations. Firstly, with regard to the measures that were used, although reliability and validity had been demonstrated for use within the adolescent population, the measures that were not specifically looking at diabetes-related factors (e.g. PHQ-9, ASQ, SEQ-C, ATQ) had not been standardised within the adolescent T1D population. This limitation can also be applied to the fact that the sample was relatively diverse in terms of ethnicity and the measures may not have been sensitive to different cultural backgrounds. Secondly, the questionnaires were all self-report measures that were completed at attendance at routine clinic appointments. An element of social desirability bias may have threatened internal validity, where difficulties may have been minimised and scores were not reflecting the difficulties experienced. Finally, the measure for depressive symptoms has been used widely as a screening tool and perhaps assessment of depressive symptoms through a more formal diagnostic interview would have provided a more comprehensive assessment of depression, allowing opportunity for exploration of individual areas of difficulties. Similarly, Bandura and colleagues (1999) found that different types of self-efficacy (social, academic, educational) were related to greater or fewer depressive symptoms. An in depth examination of these individual subscales as opposed to using total scores could have provided a clearer picture of the mechanisms associated with depressive symptoms in this population.

Further limitations were in relation to the sample. In comparison to other studies within this field, the sample size was relatively small and the number of those who participated was lower than anticipated. This not only reduced the power of the study but also increased the risk of a type 2 error i.e. failing to detect a difference where one is present. Furthermore, the characteristics of the sample in terms of the sociodemographic features suggest that the results cannot perhaps be generalised to other populations. Despite being relatively small, the diversity of the sample in terms of age also raises questions whether the developmental stages covered were too broad. For example, the views and life experiences of a 13-year old are likely to be incomparable to those of an 18-year old yet age was not explored in the current study as a covariate. Also, older participants may be required to be more autonomous in the management of their diabetes, which potentially could increase stress levels. Similarly the difference in diabetes duration could suggest that those with a longer time since diagnosis may be better equipped for dealing with the demands of the illness and perhaps less likely to experience elevated levels of stress related to diabetes. Inclusion of a control group of non-diabetic adolescents would have facilitated in establishing how much stress is related to diabetes and how much is general teen angst. Although measures were used to differentiate between the two types of stress, there was likely to be some crossover as having T1D may exacerbate the stress experienced in general situations.

The lack of consideration of external factors could have impacted on the variables of interest that were explored in this study. For example, a history of depression has been found to increase the risk of stress impacting on self-efficacy, which in turn leads to greater vulnerability in developing future depressive symptoms (Maciejewski, Prigerson, & Mazure, 2000). Data on previous depression was collected however was not included in the analysis in the current study given the relatively small numbers of young people who reported a history

of depression. There is also evidence of a relationship between parental cognitive mechanisms and the cognitive mechanisms of young people. For example, depression in mothers with children who have T1D is associated with child depressive symptoms (Jaser, Whitemore, Ambrosino, Lindemann, & Grey, 2008). Low parental mood has also been found to have a negative effect on parental perceptions of adolescent self-efficacy, which is associated with the adolescent having a negative perception of their own self-efficacy (Butler et al., 2009). Again, these factors were not considered in the current research.

Clinical implications. Implications for clinical practice can be inferred from the results, despite the mediation models not being statistically significant. Stress appears to have a negative association with the cognitions involved in the development of depressive symptoms. A screening tool incorporating measures of stress and low mood may be helpful in identifying those who may require intervention and perhaps prevention approaches. Issues may arise with self-report tools within this population (Esbitt, Tanenbaum & Gonzalez, 2012) therefore more practical approaches to monitor mood may be used within the clinical setting. For example, Fogel and Weissberg-Benchall (2010) suggest that asking questions such as “Have you been feeling more sad or down than usual?” and “Have you noticed that things that used to be fun are not so much fun anymore?” (p. 437) can be used as indicators of when further exploration and intervention may be required.

In terms of interventions for stress, techniques could be collaboratively established with healthcare practitioners to focus on strategies the young people already have in place to reduce stress (e.g. engaging in an activity, relaxation based strategies). Additional interventions may be based on psychoeducation on how to recognise stress (Hood, Rausch, & Dolan, 2011). This will then serve as an indicator of when to implement strategies to reduce stress and is more of a preventative intervention. It may be helpful to offer this type of

psychoeducation to parents and carers to increase their awareness of signs that the young person is experiencing levels of stress and may require additional support or prompting to utilise coping techniques.

Self-efficacy has been found to be key in the self-management of diabetes in adolescents (Iannotti et al., 2006) and can also be reinforced by healthcare practitioners at routine appointments. It has been suggested that this is done through active involvement of the young person in their care (e.g. through addressing them during appointments), providing education, and helping them to tackle hindrances and barriers with non-judgemental problem solving (Stewart, Rao & White, 2005). In terms of more formal interventions for adolescents with T1D, research suggests that coping skills training with a focus on resolving problems that are encountered in terms of managing T1D is effective (Davidson, Boland & Grey, 1997). This approach has been found to improve self-efficacy and diabetes specific health outcomes (Grey, Boland, Davidson, Li, & Tamborlane, 2000; Whittemore, Jaser, Guo, & Grey, 2009).

With regard to negative automatic thoughts, cognitive behavioural interventions stemming from Beck's model have been found to be effective in reducing depressive symptoms within individuals with diabetes (Balhara & Verma, 2013). This approach targets the negative automatic thoughts that are triggered by stress with a view to reduce an individual's vulnerability to developing depressive symptoms.

As the results of the current study were mixed and inconsistent with previous work, further investigation of cognitions and their role in the development of depressive symptoms in adolescents with T1D is warranted. Exploration of the factors that influence these cognitions in adolescents with T1D, such as parental cognitions, were beyond the scope of the current study and would benefit from further investigation in future work.

Conclusions. The current study has highlighted variables that potentially identify those who may be at risk of developing depressive symptoms. In terms of the mediating role of the cognitions investigated, neither automatic thoughts nor self-efficacy (general or diabetes specific) were found to be significant mediators in the relationship between stress and depressive symptoms. Yet associations were established between individual parts of the models. The main findings were that general stress leads to more negative automatic thoughts and lower self-efficacy, with the former creating an increase in depressive symptoms whereas diabetes specific leads to lower diabetes specific self-efficacy. Furthermore, both general and diabetes specific stress were strongly associated with depressive symptoms. The limitations discussed may explain why results were not entirely consistent with the theories of Beck and Bandura in terms of the mediating roles of the investigated cognitions. Despite the findings, adolescents who have T1D demonstrate significantly higher rates of depression compared to the general population (Kanner et al., 2003) and are a particularly vulnerable sample that require consistent monitoring and appropriate intervention. Effective screening procedures are required to identify and monitor young people at risk of developing depressive symptoms (Schwartz, Axelrad, Cline, & Anderson, 2011). Preventive measures may reduce the economic burden that T1D and depression place on the healthcare system (Egede & Ellis, 2010). More importantly, awareness and promotion of the psychosocial wellbeing of this group provides healthcare practitioners and family members with the means to support young people and empower them through shared decision making and collaboration with those involved with their care (Stewart, Rao & White, 2005).

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CHAPTER THREE

PUBLIC DOMAIN BRIEFING DOCUMENT

Overview

This document provides a summary of the two research components that were submitted as part of a thesis in partial fulfillment for the qualification of Doctorate of Clinical Psychology (Clin.Psy.D.) at the University of Birmingham. The literature review and empirical paper explored depressive symptoms in adolescents with Type 1 diabetes (T1D). Adolescents with T1D are more vulnerable to developing depressive symptoms than their peers (Johnson, Eiser, Young, Brierley & Heller, 2013; Kanner, Hamrin, & Grey, 2003) and the presence of depressive symptoms can have a negative influence on the self-management of T1D (Hood et al., 2006). For example, a reduction in motivation (a symptom of depression) can interfere with self-care tasks (Kovacs, Goldston, Obrosky, & Iyengar, 1992; Lin et al., 2004) which can then lead to further diabetes-related complications. More specifically, higher levels of depressive symptoms in adolescents with T1D have been found to be associated with poorer metabolic control (e.g. Hood et al., 2006; Kovacs et al., 1995; La Greca & Bearman, 2002). It is therefore important to gain an understanding of the processes that underlie depressive symptoms in adolescents with T1D and also to examine the relationship between depressive symptoms and metabolic control.

Literature review: The longitudinal relationship between depressive symptoms and metabolic control in adolescents with Type 1 diabetes

The literature review synthesised and evaluated research that was conducted over a period of time (longitudinal research) investigating the relationship between depressive symptoms and metabolic control. The aim of the review was to gain a clearer understanding of directionality within this relationship (i.e. Do depressive symptoms lead to poorer

metabolic control or vice versa?). Additional factors that may influence this relationship were also explored.

The fourteen studies that were identified from the evidence base revealed mixed findings, with most research exploring the influence of depressive symptoms on metabolic control. A number of studies suggested that depressive symptoms at baseline had a negative effect on subsequent values of metabolic control at follow-up but that this association decreased over time. However, it was important to note that this relationship was not independent of the influence of other factors, including: the frequency of blood glucose monitoring, the occurrence of stressful life events, family conflict, parental involvement in diabetes self-care, and peer relationships. Further research is needed in order to clarify the nature of the relationship between depressive symptoms and metabolic control over time in adolescents with T1D.

Empirical paper: The role of negative automatic thoughts and self-efficacy in depressive symptoms in adolescents with Type 1 diabetes

Background. Two leading theories of depression suggest that particular styles of thinking lead to the development of depression. Beck's cognitive theory of depression (1967) suggests that unhelpful thought patterns, called negative automatic thoughts, influence an individual's interpretation of events and lead to depression. This theory has been tested such that an increase in the frequency of negative automatic thoughts has been found to be associated with higher levels of depressive symptoms in adolescents (Moilanen, 1995) and adults with diabetes (Clarke & Goosen, 2009). Bandura's social cognitive theory of depression (1997), states that the beliefs an individual holds regarding their ability to achieve a particular goal (self-efficacy) are associated with the development of depression. Lower

self-efficacy has been reported to be associated with greater depressive symptoms in the general adolescent population (Ehrenberg et al., 1991; Muris, 2002; Smith & Wood, 2007) and in adolescents with T1D who reported that diabetes had a greater impact on their quality of life (Grey, Boland, Yu, Sullivan-Bolyai, & Tamborlane, 1998).

Aims. The research aimed to explore negative automatic thoughts and self-efficacy to establish how much they are involved in depressive symptoms in adolescents with T1D. Stress (both diabetes specific and general) was explored with regard to its influence on depressive symptoms. General self-efficacy and diabetes specific self-efficacy were also investigated to determine whether one is more strongly related to depressive symptoms than the other.

Method. A cross-sectional, questionnaire based design was used. Participants were recruited from five paediatric and young adult diabetes clinics in the West Midlands, and consisted of 54 adolescents with T1D aged between 13 and 18 years old.

Results. The results showed that increases in general and diabetes specific stress increased were associated with increases in negative automatic thoughts. In terms of self-efficacy, as general stress increased general self-efficacy decreased, whereas increases in diabetes specific stress were associated with a reduction in diabetes specific self-efficacy. Higher depressive symptoms were associated with higher levels of negative automatic thoughts and lower beliefs of general self-efficacy but not with self-efficacy related to diabetes. Both general stress and diabetes specific stress were strongly associated with depressive symptoms. There was no evidence to suggest that negative automatic thoughts and/or self-efficacy mediated (explained) the relationship between stress and depressive symptoms, however negative automatic thoughts were associated with depressive symptoms but only when the stressor was general stress.

Conclusions. The findings were mixed in terms of supporting the respective theories proposed by Beck and Bandura. There was evidence to support the role of negative automatic thoughts and self-efficacy in depressive symptoms in adolescents with T1D. Increases in both types of stress were associated with an increase in reported negative automatic thoughts. However, general stress was only associated with lower general self-efficacy and, similarly, diabetes specific stress was associated with a reduction in diabetes specific self-efficacy. Higher levels of stress (general and diabetes specific) were associated with higher levels of depressive symptoms. Therefore this population appears to be vulnerable to developing depressive symptoms due to the association with negative automatic thoughts and lower self-efficacy triggered by stress. Further research exploring the thought patterns that underlie depressive symptoms in adolescents with T1D is required.

Implications for clinical practice

The findings of the literature review and empirical paper highlight the vulnerability of adolescents with T1D. It is therefore recommended that they receive ongoing monitoring for emotional difficulties (Colton, Olmsted, Daneman, & Rodin, 2013). To ensure vulnerabilities are detected, the administration of parental reports of adolescent distress in addition to adolescent self-reports is recommended. This will help to identify difficulties not disclosed by the adolescent. Furthermore, the use of continuous scores as opposed to cut-off scores is recommended to ensure that underlying difficulties and mild symptoms of emotional distress are not overlooked (Hilliard, Herzer et al., 2011) and to enable more accurate monitoring.

Discussions regarding the impact of stress on T1D by healthcare professionals will be helpful (Helgeson, Escobar et al., 2010). Questions such as “Have you been feeling more sad or down than usual?” and “Have you noticed that things that used to be fun are not so much

fun anymore?” can be asked at clinic appointments to monitor mood and stress levels (Fogel & Weissberg-Benchell, 2010). Self-efficacy can also be reinforced at appointments through actively involving the young person in their care, providing education and helping them tackle hindrances and barriers with non-judgmental problems solving (Stewart, Rao & White, 2005). Parents and carers can also support adolescents in recognising signs of stress to act as cues of when to implement stress reduction strategies.

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Appendix 1

Sociodemographic questionnaire

Part One: About you and your health

Are you: Please tick

- Male
- Female

How old are you? _____

Which of the following best describes your ethnicity? Please tick.

- White
- Asian or Asian British
- Black or Black British
- Chinese
- Any other Asian background
- Mixed background
- Other (please state): _____

Do you go to school or college?

- Yes
- No

If you ticked 'No', please tick the box that applies to you.

- Working full-time
- Working part time
- Not employed
- Other (please state): _____

How old were you when you were diagnosed with Type 1 diabetes? _____

How long have you had Type 1 diabetes? _____

What do you do to control your diabetes? You can tick more than one box.

- I have only changed what I eat (my diet).
- I take tablets.
Please write down how many tablets you take per day: _____
- I have insulin injections.
Please write down how many injections you have per day: _____
- I use an insulin pump.
- I do other things to control my diabetes.

Please describe the other things that you do to control your diabetes:

Apart from diabetes, do you have any other health problems? Please tick.

- Yes
- No

If you ticked 'Yes', please write down the other health problems you have.

Have you ever been diagnosed with depression?

- Yes
- No

If you ticked 'Yes', are you currently taking any medication for depression?

- Yes
- No

Appendix 2

Adolescent Patient Health Questionnaire (PHQ-9)

Instructions: How often have you been bothered by each of the following symptoms during the past **two weeks**? For each symptom put an "X" in the box beneath the answer that best describes how you have been feeling.

	(0) Not at all	(1) Several Days	(2) More than half the days	(3) Nearly every day
1. Feeling down, depressed, irritable, or hopeless				
2. Little interest or pleasure in doing things				
3. Trouble falling or staying asleep				
4. Poor appetite, weight loss or overeating				
5. Feeling tired or having little energy				
6. Feeling bad about yourself – or feeling that you are a failure or have let yourself or your family down				
7. Trouble concentrating on things, like school work, reading, or watching TV				
8. Moving or speaking so slowly that other people could have noticed. Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual				
9. Thoughts that you would be better off dead, or hurting yourself in some way				
In the past year have you felt depressed or sad most days, even if you felt okay sometimes? <input type="checkbox"/> Yes <input type="checkbox"/> No				
If you are experiencing any problems on this form, how difficult have these problems made it for you to do your work, take care of things at home or get along with other people? <input type="checkbox"/> Not difficult at all <input type="checkbox"/> Somewhat difficult <input type="checkbox"/> Very difficult <input type="checkbox"/> Extremely difficult				
Has there been a time in the past month when you have had serious thoughts about ending your life? <input type="checkbox"/> Yes <input type="checkbox"/> No				
Have you EVER in your WHOLE LIFE, tried to kill yourself or made a suicide attempt? <input type="checkbox"/> Yes <input type="checkbox"/> No				

Appendix 3

Adolescent Stress Questionnaire (ASQ)

Here are some statements about things or situations which you might find stressful. Please tell us how stressful each of these things or situations has been for you **in the past year**, by circling one number from 1-5 depending on whether you have found this:

- | | |
|---|---|
| 1 | Not at all stressful (or is irrelevant to me) |
| 2 | A little stressful |
| 3 | Moderately stressful |
| 4 | Quite stressful |
| 5 | Very stressful |

Please respond to all items in this section. If you have not experienced something, circle 1 = not at all stressful (or is irrelevant to me).

1. Disagreements between you and your father	1	2	3	4	5
2. Not being taken seriously	1	2	3	4	5
3. Getting up early in the morning	1	2	3	4	5
4. Little or no control over your life	1	2	3	4	5
5. Having to study things you do not understand	1	2	3	4	5
6. Teachers expecting too much from you	1	2	3	4	5
7. Concern about your future	1	2	3	4	5
8. Being hassled for not fitting in	1	2	3	4	5
9. Keeping up with school work	1	2	3	4	5
10. Employers expecting too much of you	1	2	3	4	5
11. Having to take on new family responsibilities as you get older	1	2	3	4	5
12. Difficulty of some subjects	1	2	3	4	5
13. Abiding by petty rules at home	1	2	3	4	5

- 1 Not at all stressful (or is irrelevant to me)
 - 2 A little stressful
 - 3 Moderately stressful
 - 4 Quite stressful
 - 5 Very stressful
-

14. Having to concentrate for too long during school hours		1	2	3	4	5
15. Inadequate school resources		1	2	3	4	5
16. Having to study things you are not interested in		1	2	3	4	5
17. Being ignored or rejected by a person you want to go out with		1	2	3	4	5
18. Disagreements between you and your teachers		1	2	3	4	5
19. Not enough time to have fun	1	2	3	4	5	
20. Putting pressure on yourself to meet your goals		1	2	3	4	5
21. Disagreements with your brothers and sisters		1	2	3	4	5
22. Pressure to work to make money		1	2	3	4	5
23. Not enough time for leisure activities	1	2	3	4	5	
24. Too much homework		1	2	3	4	5
25. Not getting enough feedback on schoolwork in time to be helpful		1	2	3	4	5
26. Not enough time for activities outside school hours		1	2	3	4	5
27. Making the relationship work with your boyfriend/girlfriend		1	2	3	4	5
28. Being judged by your friends	1	2	3	4	5	

- 1 Not at all stressful (or is irrelevant to me)
 - 2 A little stressful
 - 3 Moderately stressful
 - 4 Quite stressful
 - 5 Very stressful
-

29. Disagreements between your parents	1	2	3	4	5
30. Changes in your physical appearance with growing up	1	2	3	4	5
31. Arguments at home	1	2	3	4	5
32. Pressure to fit in with peers	1	2	3	4	5
33. Compulsory school attendance	1	2	3	4	5
34. Having to make decisions about future work or education	1	2	3	4	5
35. Living at home	1	2	3	4	5
36. Satisfaction with how you look	1	2	3	4	5
37. Disagreements between you and your mother	1	2	3	4	5
38. Not enough money to buy the things you want	1	2	3	4	5
39. Going to school	1	2	3	4	5
40. Not enough time for your boyfriend/ girlfriend	1	2	3	4	5
41. Teachers hassling you about the way you look	1	2	3	4	5
42. Abiding by petty rules at school	1	2	3	4	5
43. Pressure of study	1	2	3	4	5
44. Lack of trust from adults	1	2	3	4	5

- 1 Not at all stressful (or is irrelevant to me)
- 2 A little stressful
- 3 Moderately stressful
- 4 Quite stressful
- 5 Very stressful

45. Not being listened to by teachers	1	2	3	4	5
46. Parents expecting too much from you	1	2	3	4	5
47. Having to take on new financial responsibilities as you grow older	1	2	3	4	5
48. Lack of understanding by parents	1	2	3	4	5
49. Parents hassling you about the way you look	1	2	3	4	5
50. Work interfering with school and social activities	1	2	3	4	5
51. Not enough money to buy the things you need	1	2	3	4	5
52. Getting along with your boyfriend/ girlfriend	1	2	3	4	5
53. Lack of freedom	1	2	3	4	5
54. Peers hassling you about the way you look	1	2	3	4	5
55. Lack of respect from teachers	1	2	3	4	5
56. Disagreements between you and your peers	1	2	3	4	5
57. Getting along with your teachers	1	2	3	4	5
58. Breaking up with your boyfriend/ girlfriend	1	2	3	4	5

Appendix 4

Problem Areas In Diabetes Questionnaire (PAID-T)

INSTRUCTIONS: Which of the following diabetes issues are currently a problem for you?
Circle the number that gives the best answer for you. Please provide an answer for each question.

	Not a problem	Minor problem	Moderate problem	Somewhat serious problem	Serious problem
	0	1	2	3	4
1. Not having clear and concrete goals for your diabetes care	0	1	2	3	4
2. Feeling discouraged with your diabetes treatment plan?	0	1	2	3	4
3. Feeling scared when you think about living with diabetes?	0	1	2	3	4
4. Uncomfortable social situations related to your diabetes care (e.g., people telling you what to eat)?	0	1	2	3	4
5. Feelings of deprivation regarding food and meals?	0	1	2	3	4
6. Feeling depressed when you think about living with diabetes?	0	1	2	3	4
7. Not knowing if your mood or feelings are related to your diabetes	0	1	2	3	4
8. Feeling overwhelmed by your diabetes?	0	1	2	3	4
9. Worrying about low blood sugar reactions?	0	1	2	3	4
10. Feeling angry when you think about living with diabetes?	0	1	2	3	4
11. Feeling constantly concerned about food and eating?	0	1	2	3	4
12. Worrying about the future and the possibility of serious complications?	0	1	2	3	4
13. Feelings of guilt or anxiety when you get off track with your diabetes management?	0	1	2	3	4
14. Not "accepting" your diabetes?	0	1	2	3	4
15. Feeling unsatisfied with your diabetes physician?	0	1	2	3	4
16. Feeling that diabetes is taking up too much of your mental and physical energy every day?	0	1	2	3	4
17. Feeling alone with your diabetes?	0	1	2	3	4
18. Feeling that your friends and family are not supportive of your diabetes management efforts?	0	1	2	3	4
19. Coping with complications of diabetes?	0	1	2	3	4
20. Feeling "burned out" by the constant effort needed to manage diabetes?	0	1	2	3	4

Appendix 5

Automatic Thoughts Questionnaire (ATQ)

Listed below are a variety of thoughts that pop into people's heads. Please read each thought and indicate how frequently, if at all, the thought occurred to you over the last week. Please read each item carefully and fill in the blank with the appropriate number; using the following scale:

- 1 = Not at all
- 2 = Sometimes
- 3 = Moderately often
- 4 = Often
- 5 = All the time

- _____ 1. I feel like I'm up against the world.
- _____ 2. I'm no good.
- _____ 3. Why can't I ever succeed?
- _____ 4. No one understands me.
- _____ 5. I've let people down.
- _____ 6. I don't think I can go on.
- _____ 7. I wish I were a better person.
- _____ 8. I'm so weak.
- _____ 9. My life's not going the way I want it to.
- _____ 10. I'm so disappointed in myself.
- _____ 11. Nothing feels good anymore.
- _____ 12. I can't stand this anymore.
- _____ 13. I can't get started.
- _____ 14. What's wrong with me?
- _____ 15. I wish I were somewhere else.
- _____ 16. I can't get things together.
- _____ 17. I hate myself.
- _____ 18. I'm worthless.
- _____ 19. I wish I could just disappear.
- _____ 20. What's the matter with me?
- _____ 21. I'm a loser.
- _____ 22. My life is a mess.
- _____ 23. I'm a failure.
- _____ 24. I'll never make it.
- _____ 25. I feel so helpless.
- _____ 26. Something has to change.
- _____ 27. There must be something wrong with me.
- _____ 28. My future is bleak.
- _____ 29. It's just not worth it.
- _____ 30. I can't finish anything.

Appendix 6

Self-Efficacy Questionnaire for Children (SEQ-C)

Circle the answer that best shows how well you do in each of the following situations.

	Not at all				Very well
	1	2	3	4	5
1. How well can you get teachers to help you when you get stuck on schoolwork?	1	2	3	4	5
2. How well can you express your opinions when other classmates disagree with you?	1	2	3	4	5
3. How well do you succeed in cheering yourself up when an unpleasant event has happened?	1	2	3	4	5
4. How well can you study when there are other interesting things to do?	1	2	3	4	5
5. How well do you succeed in becoming calm again when you are very scared?	1	2	3	4	5
6. How well can you become friends with other children?	1	2	3	4	5
7. How well can you study a chapter for a test?	1	2	3	4	5
8. How well can you have a chat with an unfamiliar person?	1	2	3	4	5
9. How well can you prevent to become nervous?	1	2	3	4	5
10. How well do you succeed in finishing all your homework every day?	1	2	3	4	5
11. How well can you work in harmony with your classmates?	1	2	3	4	5
12. How well can you control your feelings?	1	2	3	4	5
13. How well can you pay attention during every class?	1	2	3	4	5
14. How well can you tell other children that they are doing something that you don't like?	1	2	3	4	5
15. How well can you give yourself a pep-talk when you feel low?	1	2	3	4	5
16. How well do you succeed in understanding all subjects in school?	1	2	3	4	5
17. How well can you tell a funny event to a group of children?	1	2	3	4	5
18. How well can you tell a friend that you don't feel well?	1	2	3	4	5
19. How well do you succeed in satisfying your parents with your schoolwork?	1	2	3	4	5
20. How well do you succeed in staying friends with other children?	1	2	3	4	5
21. How well do you succeed in suppressing unpleasant thoughts?	1	2	3	4	5

22. How well do you succeed in passing a test?	1	2	3	4	5
23. How well do you succeed in preventing quarrels with other children?	1	2	3	4	5
24. How well do you succeed in not worrying about things that might happen?	1	2	3	4	5

Appendix 7

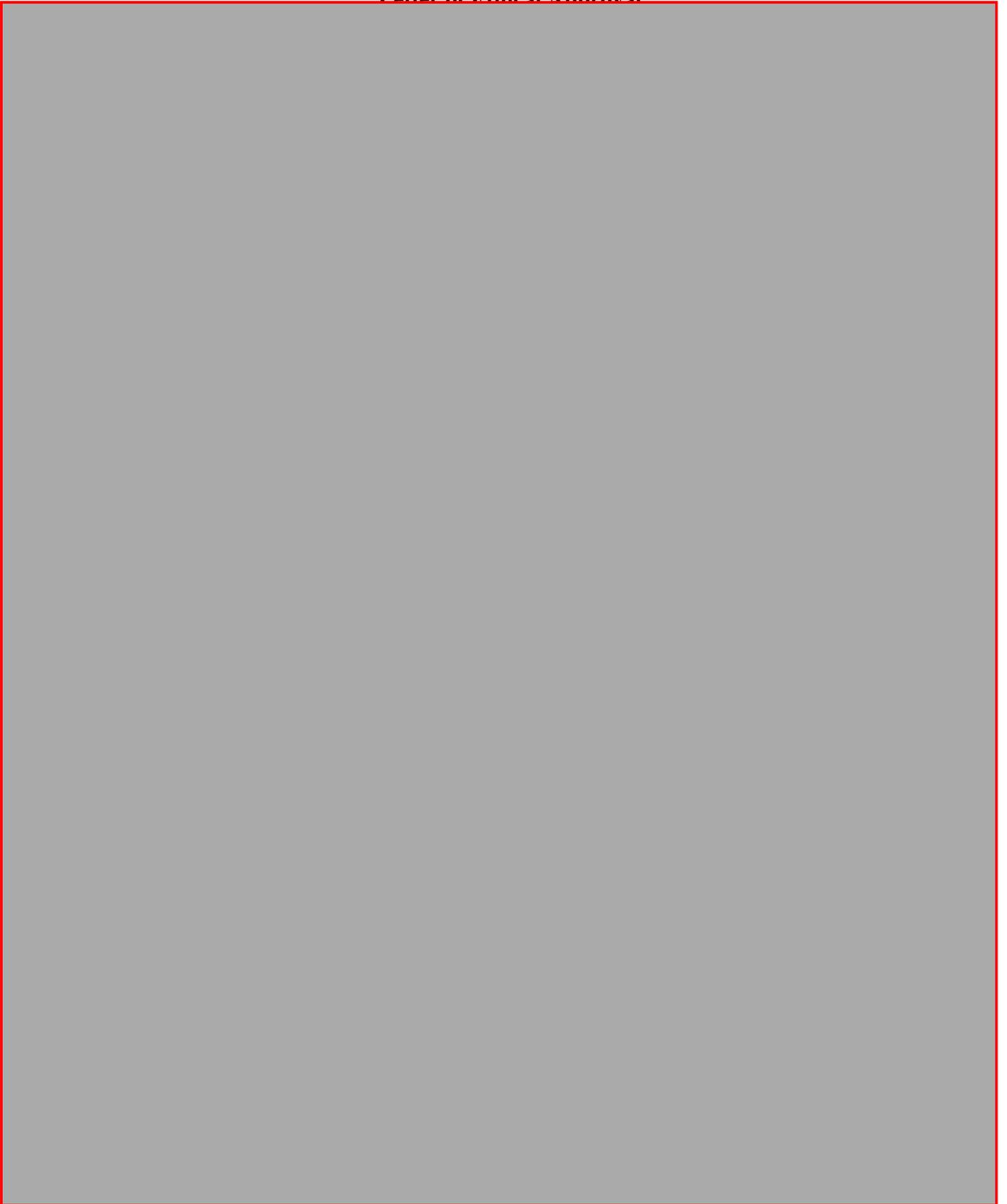
Self-Efficacy for Diabetes Self-Management Questionnaire (SEDM)

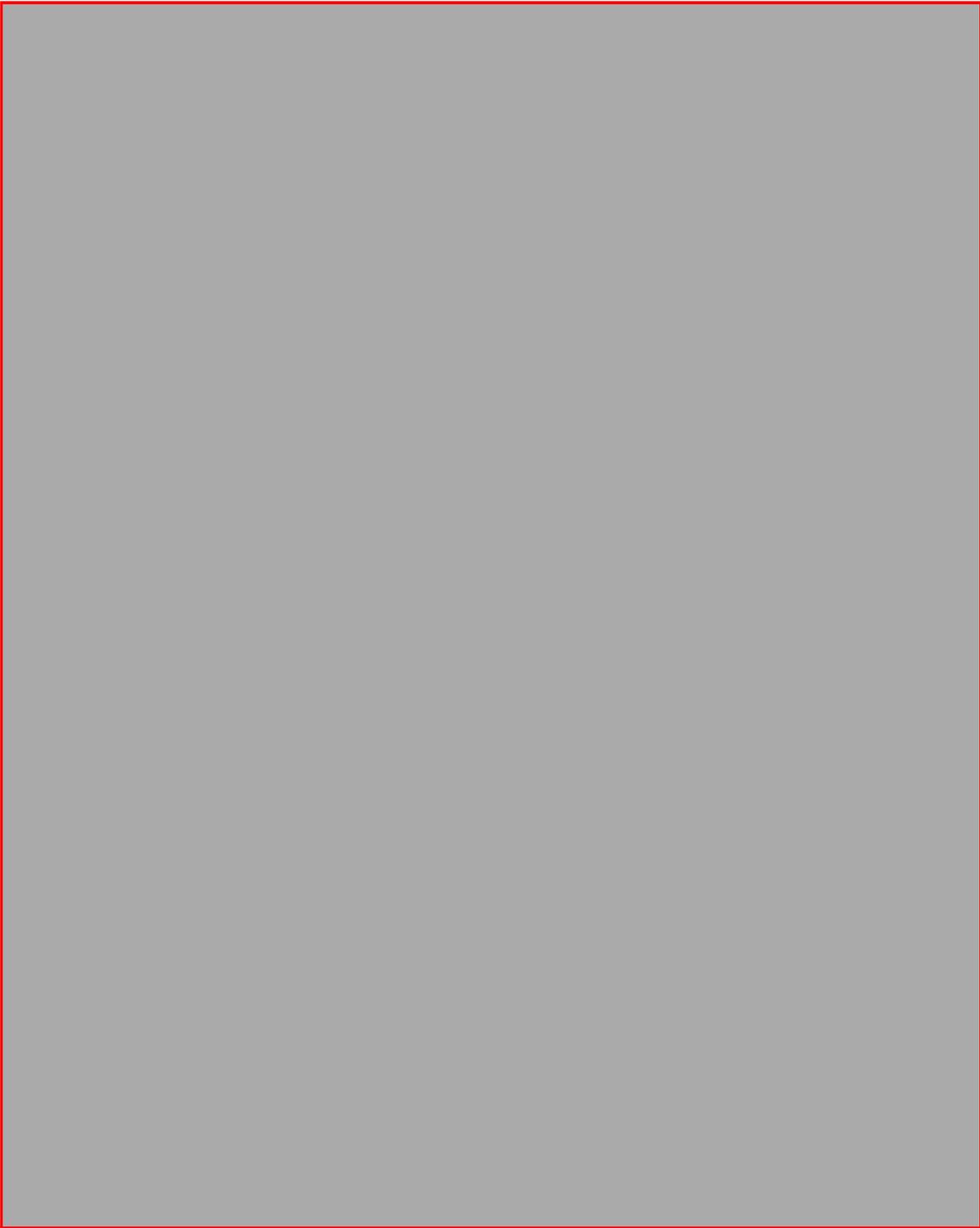
Please answer the following questions about taking care of your diabetes.

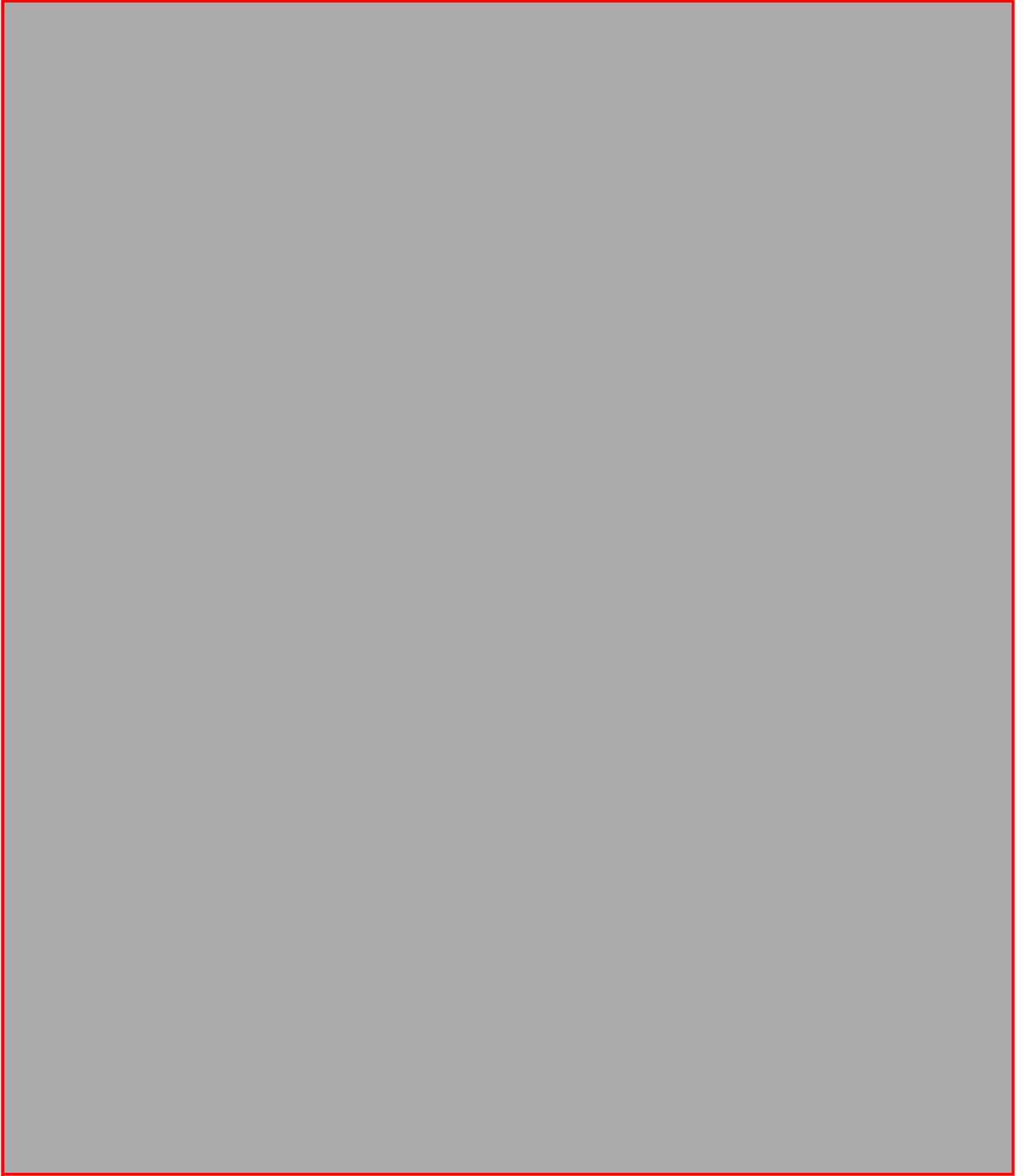
How sure are you that you <u>can</u> do each of the following, almost all of the time?	<u>Not at All Sure</u>									<u>Completely Sure</u>
1. Adjust your insulin correctly when you eat more or less than usual?	1	2	3	4	5	6	7	8	9	10
2. Choose healthful foods when you go out to eat?	1	2	3	4	5	6	7	8	9	10
3. Exercise even when you don't really feel like it?	1	2	3	4	5	6	7	8	9	10
4. Adjust your insulin or food accurately based on how much exercise you get?	1	2	3	4	5	6	7	8	9	10
5. Talk to your doctor or nurse about any problems you're having with taking care of your diabetes?	1	2	3	4	5	6	7	8	9	10
6. Do your blood sugar checks even when you are really busy?	1	2	3	4	5	6	7	8	9	10
7. Manage your diabetes the way your health care team wants you to?	1	2	3	4	5	6	7	8	9	10
8. Manage your diabetes even when you feel overwhelmed?	1	2	3	4	5	6	7	8	9	10
9. Find ways to deal with feeling frustrated about your diabetes?	1	2	3	4	5	6	7	8	9	10
10. Identify things that could get in the way of managing your diabetes?	1	2	3	4	5	6	7	8	9	10

Appendix 8

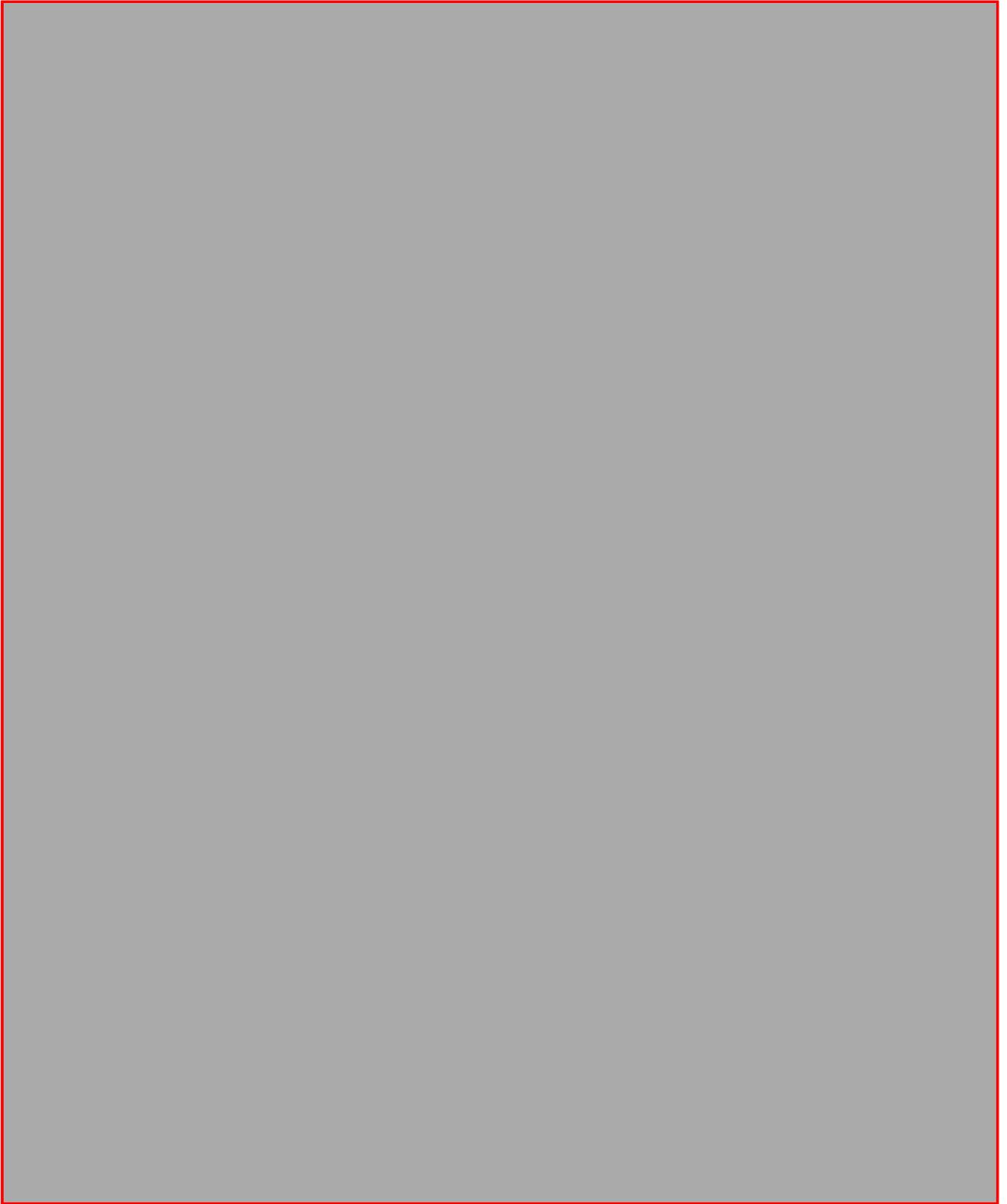
Letter of Ethical Approval







Appendix 9
Invitation Letter





Appendix 10

Participant Information Sheet

Version 2 (18/02/2013)

If you have any questions or want to discuss any part of the research, please contact the researcher:

Kiran Bali
(Trainee Clinical Psychologist)
School of Psychology
University of Birmingham
Edgbaston
B15 2TT
Telephone: 0121 414 7124
E-mail: KXB183@bham.ac.uk

What if I want to make a complaint?
If have any worries about the way you have been treated during your participation or wish to make a complaint, you can contact :

Sean Jennings,
(Team Manager)
Research and Governance,
University of Birmingham,
Room 119 Aston Webb Building, Edgbaston,
B15 2TT,
Telephone: 0121 415 8011.
E-mail : s.jennings@bham.ac.uk.

Information sheet for young people (13 - 18 years old)

This leaflet is an invitation to take part in a research project about:

Wellbeing in young people with type 1 diabetes



This research project is supervised by:

Dr Arie Nouwen & Dr Gary Unquhart Law
School of Psychology
University of Birmingham
Edgbaston
B15 2TT
Telephone: 0121 414 7124

The research project is organized and funded by the Doctoral Course in Clinical Psychology at the University of Birmingham.

This leaflet will provide you with information that may help you to decide whether or not you would like to take part.

Before you decide, take the time to read through the leaflet carefully. You may find it helpful to talk to someone about the study before you make a decision.

This study has been reviewed by the NRES Committee East Midlands—Leicester.

Participant Identification Number:

What is the purpose of the research?



The research wants to find out about young people's thoughts about having type 1 diabetes and how these might affect their wellbeing. The research is also interested in what your parent/carer thinks about diabetes.

Why have I been invited to take part?

Young people who attend this clinic, with type 1 diabetes who are aged between 13 years and 18 years old, and their parents/carers have been invited to take part.

Do I have to take part?

No. It is up to you if you want to take part. If you decide that you do not want to take part, your diabetes care at the hospital will stay the same. If your parent/carer does not want to take part, you can still take part.

What will happen to me if I take part?

If you decide to take part, the researcher will meet with you at this clinic appointment or your next appointment. You will be given a form to read through and sign. The form says that you understand what you will be doing as part of the research and that you would like to take part. After signing the form, you and your parent/carer will be asked to complete some questionnaires, preferably during your hospital visit.



What will I have to do?

You will be asked to complete a questionnaire booklet at the type 1 diabetes clinic. The questionnaires should take no longer than 30 minutes and can be done before or after your appointment.



What are the possible benefits for taking part?

Although there are no direct benefits for you, learning about the thoughts and views of young people and their parents/carers about having diabetes will help us to develop services to support young people with type 1 diabetes.



If you do decide to take part, your details will be entered in to a prize draw to win £50 worth of vouchers for iTunes.

What are the possible disadvantages or risks of taking part?

There are no disadvantages or risks for taking part. If you become upset by taking part in the research, you can talk to your family and friends. You can also talk to the diabetes nurse/doctor at the clinic or the researcher.

What will happen if I do not want to carry on with the study?

You can tell the diabetes nurse/doctor or the researcher at any stage if you change your mind about taking part.

This can be at any time and you can contact the researcher using the details on this leaflet.

Will my taking part in this study be kept confidential?

The information collected will be kept confidential. You will be given a code number so that you cannot be identified in the results of the study.

If we are worried about your safety based on your answers to the questions, we will discuss this with you. We will also inform your pediatrician.

What will happen to the information that is collected?

The results from the study will be reported in a thesis and kept in the University of Birmingham library. They may also be submitted for publication in a diabetes journal.



What happens if I have any further concerns?

If you want to discuss any part of the research before your next appointment, you can contact the researcher using the contact details on this leaflet.



Thank you for taking the time to read this leaflet.

Appendix 11

Consent Form

UNIVERSITY OF
BIRMINGHAM

Consent Form (Young Person 13 – 18 years)

Title of Research: Wellbeing in adolescents with type 1 diabetes

Name of Researcher: Kiran Bali

If you have decided that you would like to take part in this research, please read each statement and place an initial in the corresponding box to show that you have understood what the research is about.

- I confirm that I have read and understood the information sheet dated 13/02/13 (version 2) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.
- I understand that participation is voluntary and that I am free to withdraw at any time during the research, without giving any reason, without my medical care or legal rights being affected.
- I understand that sections of my medical notes that are relevant to taking part in the research (e.g. metabolic control) may be looked at by the researcher and agree that this can be done.
- I agree to take part in the above study.
- I agree to be contacted in 12 months to take part in a further study.

Name of Young Person

Date

Signature

**Name of Researcher/Person taking
consent**

Date

Signature

Appendix 12

SPSS Output: General model mediation analysis

Run MATRIX procedure:

***** PROCESS Procedure for SPSS Release 2.11 *****

Written by Andrew F. Hayes, Ph.D. www.afhayes.com
Documentation available in Hayes (2013). www.guilford.com/p/hayes3

Model = 4
Y = ZPHQTota
X = Zasqlog
M1 = ZSEQCTot
M2 = ZSEDMTot
M3 = ZATQTota

Sample size
54

Outcome: ZSEQCTot

Model Summary

	R	R-sq	F	df1	df2	p
	.2932	.0860	4.8913	1.0000	52.0000	.0314

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.1313	.0000	1.0000	-.2636	.2636
Zasqlog	-.2932	.1326	-2.2116	.0314	-.5593	-.0272

Outcome: ZSEDMTot

Model Summary

	R	R-sq	F	df1	df2	p
	.1200	.0144	.7592	1.0000	52.0000	.3876

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.1364	.0000	1.0000	-.2737	.2737
Zasqlog	-.1200	.1377	-.8713	.3876	-.3962	.1563

Outcome: ZATQTota

Model Summary

	R	R-sq	F	df1	df2	p
	.9797	.9599	1244.9604	1.0000	52.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.0275	.0000	1.0000	-.0552	.0552
Zasqlog	.9797	.0278	35.2840	.0000	.9240	1.0355

Outcome: ZPHQTota

Model Summary

	R	R-sq	F	df1	df2	p
	.6111	.3734	7.3001	4.0000	49.0000	.0001

Model	coeff	se	t	p	LLCI	ULCI
constant	.0000	.1120	.0000	1.0000	-.2251	.2251
ZSEQCTot	-.0694	.1248	-.5562	.5806	-.3202	.1814
ZSEDMTot	-.1383	.1204	-1.1490	.2561	-.3803	.1036
ZATQTota	-1.2011	.5719	-2.1002	.0409	-2.3503	-.0518
Zasqlog	1.6792	.5750	2.9202	.0053	.5237	2.8348

***** TOTAL EFFECT MODEL *****
 Outcome: ZPHQTota

Model Summary	R	R-sq	F	df1	df2	p
	.5395	.2910	21.3447	1.0000	52.0000	.0000

Model	coeff	se	t	p	LLCI	ULCI
constant	.0000	.1157	.0000	1.0000	-.2321	.2321
Zasqlog	.5395	.1168	4.6200	.0000	.3052	.7738

***** TOTAL, DIRECT, AND INDIRECT EFFECTS *****

Total effect of X on Y	Effect	SE	t	p	LLCI	ULCI
	.5395	.1168	4.6200	.0000	.3052	.7738

Direct effect of X on Y	Effect	SE	t	p	LLCI	ULCI
	1.6792	.5750	2.9202	.0053	.5237	2.8348

Indirect effect of X on Y	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	-1.1398	.7494	-2.7801	.3121
ZSEQCTot	.0204	.0774	-.0841	.2160
ZSEDMTot	.0166	.0312	-.0191	.1096
ZATQTota	-1.1767	.7540	-2.8800	.2333
(C1)	.0038	.0950	-.1426	.2259
(C2)	1.1971	.7641	-.2186	2.8692
(C3)	1.1933	.7562	-.2522	2.8522

Partially standardized indirect effect of X on Y	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	-1.1398	.7306	-2.5151	.4396
ZSEQCTot	.0204	.0733	-.0948	.1978
ZSEDMTot	.0166	.0329	-.0197	.1353
ZATQTota	-1.1767	.7287	-2.5530	.3474

Completely standardized indirect effect of X on Y	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	-1.1398	.7178	-2.4621	.4379
ZSEQCTot	.0204	.0726	-.0957	.1911
ZSEDMTot	.0166	.0314	-.0202	.1266
ZATQTota	-1.1767	.7164	-2.5048	.3688

Ratio of indirect to total effect of X on Y	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	-2.1128	1.5882	-5.7156	.6091
ZSEQCTot	.0377	.1417	-.1794	.3869
ZSEDMTot	.0308	.0599	-.0339	.2638
ZATQTota	-2.1813	1.6018	-5.8731	.5035

Ratio of indirect to direct effect of X on Y	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	-.6787	6.9853	-.8548	1.0190

ZSEQCTot	.0121	.7015	-.0607	.2802
ZSEDMTot	.0099	.3518	-.0162	.0929
ZATQTota	-.7008	6.6297	-.8806	.8216

Specific indirect effect contrast definitions

(C1)	ZSEQCTot	minus	ZSEDMTot
(C2)	ZSEQCTot	minus	ZATQTota
(C3)	ZSEDMTot	minus	ZATQTota

***** ANALYSIS NOTES AND WARNINGS *****

Number of bootstrap samples for bias corrected bootstrap confidence intervals:
5000

Level of confidence for all confidence intervals in output:
95.00

----- END MATRIX -----

Appendix 13

SPSS Output: Diabetes specific model mediation analysis

Run MATRIX procedure:

***** PROCESS Procedure for SPSS Release 2.11 *****

Written by Andrew F. Hayes, Ph.D. www.afhayes.com
Documentation available in Hayes (2013). www.guilford.com/p/hayes3

Model = 4
Y = ZPHQTota
X = ZPAIDTTo
M1 = ZSEQCTot
M2 = ZSEDMTot
M3 = ZATQTota

Sample size
54

Outcome: ZSEQCTot

Model Summary

R	R-sq	F	df1	df2	p
.2621	.0687	3.8370	1.0000	52.0000	.0555

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.1326	.0000	1.0000	-.2660	.2660
ZPAIDTTo	-.2621	.1338	-1.9588	.0555	-.5307	.0064

Outcome: ZSEDMTot

Model Summary

R	R-sq	F	df1	df2	p
.3298	.1088	6.3462	1.0000	52.0000	.0149

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.1297	.0000	1.0000	-.2603	.2603
ZPAIDTTo	-.3298	.1309	-2.5192	.0149	-.5925	-.0671

Outcome: ZATQTota

Model Summary

R	R-sq	F	df1	df2	p
.5586	.3120	23.5795	1.0000	52.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.1140	.0000	1.0000	-.2287	.2287
ZPAIDTTo	.5586	.1150	4.8559	.0000	.3277	.7894

Outcome: ZPHQTota

Model Summary

R	R-sq	F	df1	df2	p
---	------	---	-----	-----	---

.7303 .5333 13.9994 4.0000 49.0000 .0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.0967	.0000	1.0000	-.1943	.1943
ZSEQCTot	-.1025	.1062	-.9646	.3395	-.3160	.1110
ZSEDMTot	.0667	.1075	.6208	.5376	-.1492	.2826
ZATQTota	.0960	.1198	.8013	.4268	-.1447	.3366
ZPAIDTTo	.6580	.1238	5.3142	.0000	.4092	.9069

***** TOTAL EFFECT MODEL *****

Outcome: ZPHQTota

Model Summary

	R	R-sq	F	df1	df2	p
	.7165	.5134	54.8539	1.0000	52.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	.0000	.0958	.0000	1.0000	-.1923	.1923
ZPAIDTTo	.7165	.0967	7.4063	.0000	.5224	.9106

***** TOTAL, DIRECT, AND INDIRECT EFFECTS *****

Total effect of X on Y

Effect	SE	t	p	LLCI	ULCI
.7165	.0967	7.4063	.0000	.5224	.9106

Direct effect of X on Y

Effect	SE	t	p	LLCI	ULCI
.6580	.1238	5.3142	.0000	.4092	.9069

Indirect effect of X on Y

	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	.0585	.0877	-.0947	.2166
ZSEQCTot	.0269	.0702	-.0297	.2768
ZSEDMTot	-.0220	.0425	-.1346	.0452
ZATQTota	.0536	.0782	-.1004	.2078
(C1)	.0489	.1025	-.0669	.3688
(C2)	-.0267	.1187	-.2097	.3091
(C3)	-.0756	.0792	-.2396	.0720

Partially standardized indirect effect of X on Y

	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	.0585	.0940	-.0961	.2292
ZSEQCTot	.0269	.0651	-.0368	.2483
ZSEDMTot	-.0220	.0435	-.1275	.0544
ZATQTota	.0536	.0853	-.0935	.2291

Completely standardized indirect effect of X on Y

	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	.0585	.0900	-.0979	.2196
ZSEQCTot	.0269	.0629	-.0347	.2353
ZSEDMTot	-.0220	.0417	-.1228	.0508
ZATQTota	.0536	.0825	-.0951	.2248

Ratio of indirect to total effect of X on Y

	Effect	Boot SE	BootLLCI	BootULCI
TOTAL	.0816	.1301	-.1302	.3177
ZSEQCTot	.0375	.0905	-.0477	.3479
ZSEDMTot	-.0307	.0601	-.1786	.0708
ZATQTota	.0748	.1175	-.1283	.3179

Ratio of indirect to direct effect of X on Y

Effect	Boot SE	BootLLCI	BootULCI
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TOTAL	.0888	.2866	-.1152	.4656
ZSEQCTot	.0408	.1266	-.0583	.4744
ZSEDMTot	-.0334	.0805	-.2098	.1015
ZATQTota	.0815	.2431	-.1264	.4817

Specific indirect effect contrast definitions

(C1)	ZSEQCTot	minus	ZSEDMTot
(C2)	ZSEQCTot	minus	ZATQTota
(C3)	ZSEDMTot	minus	ZATQTota

***** ANALYSIS NOTES AND WARNINGS *****

Number of bootstrap samples for bias corrected bootstrap confidence intervals:
5000

Level of confidence for all confidence intervals in output:
95.00

----- END MATRIX -----