# Understanding, measuring and treating eating disorders in those with type 1 diabetes

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# Understanding, Measuring and Treating Eating Disorders in those with Type 1 Diabetes

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## Declaration

I confirm that the work submitted in this thesis is my own and I confirm that the thesis references information derived from other sources.

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Jacqueline Anne Allan

# Acknowledgements

I would like to thank my partner Jon. I don't know how you've put up with me but know that although this thesis has my name on it, it's ours. There is no way this would have happened without you (me still knocking about or the PhD) and I can't put my gratitude into words, so I won't try to do so here (maybe I'll finally be able to afford a nice dinner out and tell you there). I would also like to thank my lovely mum and dad for listening to me moan about this seemingly never-ending stage of my life, never judging and always taking my side. You have been a backbone when I lost mine, regardless of what was going on in your own lives. Also, my amazing wee bro who gave me meaningful distraction at exactly the right times. Look I did it without an electric skateboard!!! (p.s. now I definitely want an electric skateboard).

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*"See me, just sucking in the Killer Bees. They chew right through me, they chew right through me"* - *The Stills*

# Abstract

The purpose of this thesis was to explore the nature of Eating Disorders in Type 1 Diabetes. Whether or not Eating Disorders are more prevalent in this demographic is a topic of contention but regardless there is a consensus that those with comorbid Type 1 have considerably worse outcomes and are significantly more difficult to treat. It has been argued that this may be due to a feature unique to this population; insulin omission for weight control.

The first aim of this thesis was to systematically review how Eating Disorders have been measured in Type 1 Diabetes, paying particular attention to whether researchers have taken the role of Diabetes regimen and insulin omission into account. Following this a comparison between two Eating Disorder scales, one Diabetes specific the other not, was made in order to compare prevalence rates, to explore which items may be potentially biased and to investigate what the effect of modification may be. The structure of the Diabetes specific scale (the Diabetes Eating Problem Scale Revised) was then explored

The second aim of this thesis was to replicate a pilot study that aimed to explore demographic, psychological and health seeking features of those with Type 1 Diabetes related Eating Disorders. This formed the basis of a structural model whereby psychological and Diabetes specific traits were hypothesised to predict Eating Disorder behaviour and elevated blood glucose levels. A questionnaire built for that study regarding patient attributions was also reanalysed using new data.

The final aim was to investigate how Eating Disorders in Type 1 Diabetes have been treated by reviewing literature from the last 2 decades, paying particular attention as to how treatment providers have accommodated the unique needs of those with T1D and whether or not programmes have been successful in relation to both psychological and biological outcomes.

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# Abbreviations

| | |
|---|---|
| APA | the American Psychological Association |
| AN | Anorexia Nervosa |
| BED | Binge Eating Disorder |
| BG | Blood Glucose |
| BPD | Borderline Personality Disorder |
| BN | Bulimia Nervosa |
| CBT | Cognitive Behavioural Therapy |
| CFC | Consideration of Future Consequences |
| DEB | Disordered Eating Behaviour |
| DKA | Diabetic Ketoacidosis |
| DOC | the Diabetes Online Community |
| DSD | Diabetes Specific Distress |
| DSM | Diagnostic Statistical Manual |
| ED | Eating Disorder |
| EDNOS | Eating Disorder not Otherwise Specified |
| HbA1c | Haemoglobin A1c |

HCP          Health Care Professional

HCRP         Health Care & Related Professional

ICB          Inappropriate Compensatory Behaviours

T1D          Type 1 Diabetes

T1ED         Type 1 Eating Disorder

# 1 Introduction: Eating Disorders including Insulin Omission for Weight Loss in those with Type 1 Diabetes

## 1.1 History

> Diabetes is a dreadful affliction, not very frequent among men, being a melting down of the flesh and limbs into urine. The patients never stop making water and the flow is incessant, like the opening of aqueducts. Life is short, unpleasant and painful, thirst unquenchable, drinking excessive, and disproportionate to the large quantity of urine, for yet more is urine passed. One cannot stop them either from drinking or making water… If for a while they abstain from drinking, their mouths become parched and their bodies dry; the viscera seem scorched up, the patients are affected by nausea, restlessness and a burning thirst, and within a short time they expire. (quoted from Poretsky 2010, p. 20)

The above is deemed to be the first clinical description of what we now recognise as Type 1 Diabetes Mellitus (T1D). It comes from the physician Aretaues of Cappadocia, a close contemporary of Hippocrates, and dates to the end of the Hellenistic period (Medvei, 1993). Although earlier descriptions of specific related symptoms had appeared in Ancient Egypt (via the Ebers papyrus) as early as 1550BCE and the term Diabetes had been used by Apollonius of Memphis after the Greek verb διαβαίνω (diabaino) meaning literally: to run through (Papaspruos, 1964) Aretaues was the first to identify the syndrome as a whole. The suffix 'Mellitus' from the Greek meaning 'sweet like honey' to reflect the sugary smell detected via the urine, was added by Thomas Willis when he dedicated a chapter of his book to 'The Pissing Evil' (Willis, 1694). The term was solidified by the Scottish military surgeon John Rollo in his 1797 work titled *An Account of Two Cases of the Diabetes Mellitus.*

By the 19th century T1D had a clinical description if not an understanding of aetiology. Those from antiquity had hypothesised that the lungs, the blood and the kidneys among others were to blame for the disease and treatment suggestions ranged from enemas, goats cheese and rice to cataplasms and the much extolled ancient remedy Theria (Karamanou, Protogerou, Tsoucalas, Androutsos, & Poulakou-Rebelakou, 2016). None of these would be successful. This was to change mid-century with the discovery that removing the pancreas in canines provoked diabetic symptomology. After returning pancreatic tissues via grafting the dogs returned to their normal state suggesting that the pancreas was the organ most likely to be critical. Further research undertaken in the latter part of the 1800s showed that pancreatic

formulations injected in to pancreatectomised dogs lowered their blood sugar (Karamanou et al., 2016). This formulation was initially given the name 'Isleton', later changed to 'Insulin'. These experiments led to the first injection of insulin using a human subject in 1922 by Frederick Banting and Charles Best, saving the young boy's life and earning the research teams involved a Nobel Prize (Bliss ,1982)

## 1.2 Aetiology

Nearly a century has passed since insulin treatment saved sufferers from what was previously a terminal illness and much more about this 'dreadful affliction' is understood. T1D is an autoimmune disorder whereby the body mistakenly attacks the insulin producing beta cells of the pancreas until they are destroyed leaving the patient unable to process carbohydrate. The absence of insulin leads to accumulating glucose in the bloodstream which if left untreated is eventually fatal. The exact aetiology of T1D is still the subject of worldwide study. Genetic factors have been suggested, in particular the high risk genotypes DR3/4 - DQ8 or DR4/DR4. Lifetime prevalence in probands is estimated to be around 6% (15 times the risk compared to the normal population) and concordance in monozygotic twins is more than 60% over the lifetime. Those with fathers who have the illness are also more at risk than those whose mothers do (Steck & Rewers, 2011). Certain viruses have been implicated. Enteroviruses such as the Coxsackie B are known to infect and inflame the beta cells of the pancreas which may lead to autoimmunity. Congenital Rubella Syndrome has also been positively associated with T1D development (Filippi & von Herrath, 2008). Environmental factors may also prove important. Studies in migrant populations have shown that prevalence concords to the host country rather than that of origin (Bodansky, Staines, Stephenson, Haigh & Cartwright, 1992). Children who have been exposed to crowds or who attended day care in infancy are also less likely to develop T1D. It is thought that this is due to immune system fortification via contact with potential antigens, also known as the hygiene hypothesis (Gale, 2002).  There is even season of birth variation with children born in in the spring/summer more likely to develop T1D than those in the autumn/ winter, suggesting a role for prenatal exposure to vitamin D. There do not appear to be significant differences in gender (Maahs, West, Lawrence & Mayer-Davis, 2010).

## 1.3 Epidemiology

Whatever the exact aetiology, the incidence of T1D globally is increasing at an estimated rate of between 2 – 5% per annum and this increase is particularly marked in infants under 5 (Maahs et al., 2010). The disease is more common in western society and in Caucasians. The highest incidence of new diagnoses in children are in Europe (20 per 1000) followed by North America and the Caribbean (16.7 per 1000) (Patterson et al., 2014). Age of onset is most frequent between the ages of 10 – 14 (Maahs et al., 2010) but around 1 in 4 are diagnosed as adults (Patterson et al., 2009). In the UK there are an estimated 370,000 adults currently living with T1D, (NICE 2015) equating the prevalence to roughly 25 out of 100,000 individuals.

## 1.4 Treatment, Outcomes and Co-Morbidity

For individuals with T1D insulin is essential for survival. Patients must attempt to mimic what the pancreas would do naturally by administering synthetic insulin into the body to regulate blood glucose levels. There are currently two main delivery methods for this, the first is multiple daily injections, whereby long acting insulin is injected to replicate a normal baseline in the blood and further short acting insulin is injected to cover any incoming carbohydrate or to correct elevated levels. The second method utilises an insulin pump which infuses constant rapid acting insulin subcutaneously and can be adjusted in real time in order to respond to meals or hypoglycaemia. Both methods require the patient to pay close attention to their diet and many utilise a carbohydrate to insulin ratio in order to estimate medication needs. It is not just food, however, that affects blood glucose. Temperature changes (Koivisto, Fortney, Hendler & Felig, 1981), weight gain (Kilpatrick, Rigby & Atkin, 2007), illness, infection, stress, hormonal changes (McDonnell & Umpierrez, 2012), injection technique, injection site and exercise (Binder, Lauritzen, Faber & Pramming, 1984) among many others things have been shown to affect glucose levels. This makes controlling T1D in everyday life more educated guess work than simple mathematics. There are high intra and inter - individual differences in how insulin absorbs and acts in the body which also vary temporally complicating successful management further (Heinemann, 2002).

Current guidelines suggest that those with T1D should keep their BG as close to that of non Diabetics as possible which is between 4 – 7 mmols and should aim for an HbA1c (glycosylated haemoglobin over 3 months) of 6.5% (NICE, 2015). In order to do this, those with T1D are advised to check their blood glucose between 4 – 10 times a day and take steps to rectify high or low levels, see a Diabetes specialist regularly, have annual checks for Diabetes related complications, follow an appropriate diet and attend structured education programmes (NICE, 2015). However, the difficulties involved in attempting to manually replicate such a complicated biological process means that two errors are common. Hypoglycaemia occurs when there is too much insulin in the blood and therefore not enough glucose in the brain leading to short term cognitive impairments and a biological response similar to 'fight or flight'. If this is not treated with glucose in an immediate manner hypoglycaemia can result in coma and death. Conversely hyperglycaemia occurs when there is not enough insulin in circulation and therefore glucose cannot be removed from the blood. Initially this causes fatigue, polyuria and weight loss but will progress to life threatening Diabetic Ketoacidosis (DKA) if insulin is not administered. As the body is not receiving energy from food due to the absence of insulin, it turns to its own stores for fuel. The process of burning fat and tissue for energy produces ketones and turns the blood acidic. At this stage DKA becomes catastrophic, massive weight loss, Kussmaul breathing[1], rapid dehydration, vomiting and cardiac trauma may further develop into multiple organ failure, coma and or death.

Over the long term, hyperglycaemia is responsible for most complications arising from T1D. High blood glucose degrades almost every bodily system, but nerve damage is the most common complaint (Diabetes Control & Control Trial, 1993). This nerve damage takes several forms. In the stomach it hampers the passage of food through the digestive tract eventually resulting in gastroparesis, in the extremities peripheral neuropathy causes numbness or painful stinging sensations, in the eyes retinopathy occurs which may lead to

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[1] Kussmaul Breathing: low, shallow breathing caused by prolonged diabetic ketoacidosis (Kussmaul 1874)

blindness, autonomic neuropathy can cause fainting and low blood pressure and nerve damage in the kidneys can advance to nephropathy. Having T1D is also a risk factor for cardio vascular disease and even with good control, mortality increases and life span is reduced significantly (Lind et al., 2014). Furthermore, as a systemic illness T1D often accompanies other chronic conditions with 33% of new cases in children presenting alongside other autoimmune diseases (Triolo et al., 2011). Celiac Disease, Autoimmune Thyroid Disease (both hypo and hyper), Addison Disease, Vitiligo and Rheumatoid Arthritis are significantly more common in those with T1D than in the general population. In some cases, these conditions and the corresponding treatment may interfere with insulin action and BG metabolism further complicating successful T1D management (Franzese, Mozzillo, Nugnes, Falco & Fattorusso 2011). Currently only around a 3$^{rd}$ of UK based T1Ds manage to attain the HbA1c targets set by their care team and 15% have levels equivalent to more than 10% (Diabetes UK., 2016).

## 1.5  Psychological and Social Implications

Possibly unsurprisingly there are many psychological and social implications of living with such a relentless chronic illness. In fact, the needs are as such that the American Diabetes Association has produced a position statement suggesting Diabetic stages of development to accompany that of typical maturation. These stages include advice such as 'reassuring the child that Diabetes is no-one's fault' and suggesting that treatment priorities include vigilance around weight and body image concerns (Please see table 1.1 below, Chiang, Kirkman, Laffel & Peters, 2014). None the less the evidence suggests that T1D is disadvantageous in a multitude of ways; educational attainment is reduced, school completion rates are lower and although there is some suggestion that psychological issues may ease with maturation, referral to mental health services are significantly more prevalent in this demographic (Dahlquist & Källén, 2007; Northam, Lin, Finch, Werther & Cameron, 2010). T1D also restricts access to particular careers such as those in the military and although the blanket ban from hiring those with T1D from emergency services in the UK has been lifted in lieu of individual health assessments (Diabetes UK, 2016b), these may still prove restrictive and disappointing to children and young people who may have had conflicting aspirations at the time of diagnosis.

*Table 1.1: Major Developmental Issues and their Effects on Diabetes in Children and Adolescents replicated from Chiang, Kirkman, Laffel & Peters, 2014.*

| Developmental Stages (Ages) | Normal Developmental Tasks | Type 1 Diabetes management priorities | Family issues in Type 1 Diabetes management |
|---|---|---|---|
| Infancy (0 – 12 months) | Developing a trusted relationship or bond with primary caregivers | Preventing and treating hypoglycaemia<br><br>Avoiding extreme fluctuations in blood glucose levels | Coping with stress<br><br>Sharing the burden of care to avoid parent burnout |
| Toddlers (13 – 26 months) | Developing a sense of mastery and autonomy | Preventing and treating hypoglycaemia<br><br>Avoiding extreme fluctuations in blood glucose levels due to irregular food intake | Establishing a schedule<br><br>Managing the picky eater<br><br>Limit setting and coping with toddler's lack of cooperation with regimen<br><br>Sharing the burden of care |
| Preschool and early elementary school (3 – 7 years) | Developing initiative in activities and confidence in self | Preventing hypoglycaemia<br><br>Coping with unpredictable appetite and activity<br><br>Positively reinforcing cooperation with regimen<br><br>Trusting other caregivers with Diabetes management | Reassuring the child that Diabetes is no – one's fault<br><br>Educating other caregivers about Diabetes management |
| Older elementary school (8 – 11 years) | Developing skills in athletic, cognitive artistic and social areas<br><br>Consolidating self – esteem with respect to the peer group | Making Diabetes regimen flexible to allow for participation in school or peer activities<br><br>Child learning short and long-term benefits of optimal control | Maintaining parental involvement in insulin and blood glucose management tasks while allowing for independent self – care for special occasions<br><br>Continuing to educate school and other caregivers |

| Developmental Stages (Ages) | Normal Developmental Tasks | Type 1 Diabetes management priorities | Family issues in Type 1 Diabetes management |
| --- | --- | --- | --- |
| Early adolescence (12 – 15 years) | Managing body changes<br><br>Developing a strong self of self-identity | Increasing insulin requirements during puberty<br><br>Diabetes management and blood glucose control becoming more difficult<br><br>Weight and body image concerns | Renegotiating parent and teenager's roles in Diabetes management to be acceptable to both<br><br>Learning coping skills to enhance ability to self-manage<br><br>Preventing and intervening in Diabetes related family conflict<br><br>Monitoring for signs of depression, Eating Disorders and risky behaviours |
| Later adolescence (15 – 18 years) | Establishing a sense of identity after high school (decisions about location, social issues, work and education) | Starting an ongoing discussion about transition to a new Diabetes team (discussion may begin in earlier adolescent years)<br><br>Integrating Diabetes into new lifestyle | Supporting the transition to independence<br><br>Learning coping skills to enhance ability to self-manage<br><br>Preventing and intervening in Diabetes related family conflict<br><br>Monitoring for signs of depression, Eating Disorders and risky behaviours |

Social relationships are also affected. T1D adolescents report less trust and intimacy with their romantic partners than controls (Jacobson et al., 1997). Romantic relationships are not the only ones that suffer. The family environment may have to adapt rapidly to cope with the diagnosis of a member and while it has been shown that increased parental involvement in T1D management relates to better BG levels (Schafer, McCaul & Glasgow, 1986) it may also lead to increased conflict (Miller-Johnson et al., 1994). The effect of diagnosis can be so overwhelming that in children and young people the risk of developing a psychiatric condition is threefold within the following six months and suicide attempts are significantly

more common than in controls (Butwicka, Frisén, Almqvist, Zethelius, & Lichtenstein, 2015).

Unfortunately, adults do not fare much better in terms of prognosis, (Bryden, Dunger, Mayou, Peveler & Neil, 2003). Both men and women with T1D are more likely to be unmarried, citing psychological pressures and job discrimination as contributory factors (Aono et al., 2000). They also have higher rates of common mental disorders, in particular those related to anxiety and depression in comparison to controls (Das-Munshi et al., 2007). Adult, child or young person, most studies find that psychosocial problems are predictive of poorer blood glucose management (Hislop, Fegan, Schlaeppi, Duck, & Yeap., 2008; Bryden et al 2003; Kakleas, Kandyla, Karayianni, & Karavanaki, 2009; Das-Munshi et al., 2007).

## 1.6 Type 1 Diabetes and Eating Disorders

Of all the mental illnesses that have been suggested to be more prevalent in those with T1D, one is particularly prominent. Recent research suggests that by the age of 25, 60% of T1D females will have experienced a clinically significant Eating Disorder (ED) (Colton et al 2015). Although at first glance this percentage may seem incredibly high, it is concordant with other research that suggests that around 40% of female T1Ds between the ages of 15 – 30 deliberately induce hyperglycaemia and DKA in order to produce weight loss[2], and that there is an increased risk of developing Anorexia and/or Bulimia. Although in males it was initially thought that EDs were not more prevalent in those with T1D more recent research suggests that this is not the case with insulin omission being reported in up to a quarter of male research respondents. It has also been shown that T1D males have a higher drive for thinness than their non diabetic counterparts (Rodin, Craven, Littlefield, Murray & Daneman, 1991; Fairburn, Peveler, Davies, Mann & Mayou, 1991; Striegel-Moore, Nicholson &

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[2] As stated above when the body does not have sufficient amounts of insulin, glucose escapes into the blood and is excreted through the urine, this means that those calories are not absorbed. Furthermore, when hyperglycaemia evolves into DKA the body burns fat and tissue for energy that is no longer provided by food resulting in substantial weight loss. This practice is commonly known as 'Diabulimia' (BBC, 2017)

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Tamborlane, 1992; Polonsky et al., 1994; Peveler et al., 2005; Jones, Lawson, Daneman, Olmsted & Rodin, 2000; Markowitz et al., 2010; Svensson, Engström & Aman, 1992).

*Figure 1.1: Young Girl with Type 1 Diabetes before and after Insulin Treatment. Reproduced from Polonsky (2012).*

There are substantial issues when investigating ED in T1D. T1D management requires the patient to think carefully and constantly about food, exercise and numbers. Getting insulin dosing wrong can have serious consequences. As such those with T1D may engage in food behaviour that to the uninitiated appear at best odd and a worst, disordered. For example, a meal may be skipped due to high blood glucose, a rigid feeding schedule may be utilised, a low carbohydrate diet may be employed, (cutting out a whole food group), both hyper and hypoglycaemic states promote hunger (and potentially bingeing especially in the impaired cognitive state which accompanies hypoglycaemia) and it is clinically recommended to keep records of what is being consumed. These actions, according to the National Eating Disorders Association (2018), are all behavioural signs of an ED.

There are also specific aspects of Diabetes management that may promote ED behaviour. Fear of hypoglycaemia may lead to a patient reducing food intake and thus injections (Ishmail & Treasure., 2010; Rodin et al., 2009; Goebel-Fabbri et al., 2008). Given that food may be restricted this can lead to moralising and shaming which promotes ED like cognitions (Goebel-Fabbri, Uplinger, Gerken & Mangham., 2009; Tierney, Deaton, & Whitehead 2009). Hypoglycaemia may trigger what is commonly seen in bulimia (a loss of control over eating) which may then promote inappropriate compensatory behaviours. (Criego, Crow & Goebel-Fabbri, 2009; Rodin et al., 2002). Weight monitoring is also a standard requirement of T1D and may be commented on or discussed in clinic. Again then, the nature of T1D may maintain unhealthy cognitions related to food, numbers and weight.

In regard to insulin behaviour, controversy occurs when defining insulin omission as an ED behaviour. The Diagnostic Statistical Manual has now included insulin omission under criteria for both Anorexia (AN) and Bulimia Nervosa (BN) but the habit of researchers has also been to classify it under Eating Disorder not Otherwise Specified (EDNOS) (Please see chapter 2). Although recent advances have ensured that HCPs should be aware of the behaviour in relation to EDs[3] there is disagreement on how to treat insulin omission as an ED symptom. Clinical diagnoses are difficult, and often applied as a measure of BMI rather than symptomology. This is particularly problematic for the patients themselves who see insulin omission for weight loss or 'Diabulimia' as a distinct, unique and incomparable illness. They also find that standard ED approaches which focus on relaxing food rules and not counting calories are incompatible with successful T1D management. (Hastings, McNamara, Allan, & Marriott 2016; MacDonald et al 2018; Staite et al 2018; Allan 2015[4]).

The issues above may help explain why standard screening measures are problematic when used in this population. Instruments may be over – sensitive to the eating behaviours that accompany hyper and hypoglycaemia and may completely exclude the mechanisms of insulin omission (Powers et al., 2012), for example; bingeing after a hypo or restricting food due to a hyper may be misattributed to an ED rather than the treatment for problematic BG. These misconceptions may also help explain why those with T1D appear to be much more resistant to standard ED treatment, have much worse clinical outcomes relating to recovery and drop out of treatment at much higher rates (please see chapter 8). Furthermore, the majority of programmes which have attempted to improve outcomes for those with T1ED have had no success in reducing HbA1c (please see chapter 8).

This is disturbing as those with T1ED face devastating physical consequences. Goebel-Fabbri et al. (2008) found that insulin restriction at baseline was associated with a threefold risk of mortality at 11 year follow up. Insulin omitters also had a much-reduced mean age of

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[3] Please Appendix A for commentary on the 2017 Nice Guidelines by the researcher
[4] Please see Appendices B - E for copies of these articles which were either authored or co-authored by the researcher

death (45 years vs 58 years). Nephropathy, Neuropathy and Retinopathy are also much more common among those with T1ED and the onset of complications was much earlier in life. (Goebel – Fabbri et al., 2008; Rydall, Rodin, Olmsted, Devenyi & Daneman, 1997)

## 1.7   The Current Study

The purpose of the current thesis is to explore several aspects related to EDs in T1D (T1EDs)

Study 1: Given the wildly variating estimates of both clinical EDs, Disturbed Eating Behaviour (DEB) and insulin omission in the T1D population a systematic review was undertaken to investigate what instruments have been used to make assumptions, whether or not they take Diabetes regimen into consideration, what diagnoses or cut-off points they utilise, how insulin omission is measured, what the prevalence of insulin omission is and what the difference is in prevalence estimates between the scales used.

Study 2: The purpose of study 2 was to compare 2 popularly used ED scales, the Eating Attitudes 26 (EAT-26) item which is not Diabetes specific and the Diabetes Eating Problem Survey Revised (DEPS-R) which is Diabetes specific using a large cross sectional sample of individuals with T1D. This study compared rates of those screening as clinically concerning, investigated a potential subscale in the DEPS-R relating to insulin omission and modified the EAT-26 by consulting experts in the field as to items which may be biased by aspects of T1D rather than relate directly to ED symptomology. It also aimed to explore the factor structure of the DEPS-R questionnaire.

An exploratory pilot study was undertaken by the researcher in 2013, in partial fulfilment of a BSc Psychology degree, in order to ascertain certain characteristics of those with T1D Diabetes who self-identified as having recovered from an Eating Disorder. The three primary aims of the study were to investigate psychiatric co-morbidity, to build a clinical profile of the participants and to create a questionnaire based on a literature search for suggestions as to why those with T1D were at a higher risk of Eating Disorders. This questionnaire (40 items) was then subjected to a exploratory factor analysis in order to reveal underlying latent variables that patients attributed to the development of their Eating Disorder. The study which included 98 participants (96 female) found high levels of psychiatric comorbidity. The

40 item questionnaire suggested a five factor solution These factors related to specific Diabetes distress, Diabetes diet, classic risk factors, self-esteem and family communication and negative attention[5].

Study 3: The purpose of study 3 was to use a large cross-sectional sample of T1Ds and several psychometric scales in order to ascertain if there were relationships between psychological and demographic factors and Diabetes specific ED symptomology and glucose control. In order to do this the Diabetes Eating Problem Survey Revised and HbA1c were employed as outcome measures. Structural Equational Modelling was utilised as the methodology. Psychometric and demographic variables were chosen as they related to the published literature and the pilot study.

Study 4: Study 4 was a replication of the factor analysis undertaken as part of the pilot study. The initial study only utilised those who had been in recovery for a period of no less than 2 years and thus attributions may have been biased in favour of retrospection. As such a sample containing both those who have recovered and those still suffering was utilised to yield a more valid attribution model which could also be subjected to a confirmatory procedure. The study also collected data on health seeking behaviour, co-morbid mental health diagnoses, clinical ED diagnoses and what participants thought their ED diagnosis should have been.

Study 5: The purpose of study 5 was to provide a brief literature review of treatment programmes that have been used in those with T1ED and provide a critical analysis given the results already reported in this thesis.

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[5] The pilot study is provided in Appendix F and parts of the results were published in Appendix E

## 2   Eating Disorders and Insulin Omission for Weight Loss in those with Type 1 Diabetes: A Systematic Review

### 2.1   Introduction

Eating Disorders in those with Type 1 Diabetes has been a subject of interest since reports of individuals with concurrent Anorexia and T1D started appearing in journals in the 1950s (Chimenes, 1955). Initially these cases were viewed as somewhat of an oddity, a rare combination that should provoke little clinical concern as the comorbidity was not associated with degradation of BG management (Crisp, 1978). However, disagreeing case reports emerged in the 1980s suggesting that EDs in the presence of T1D may represent a significant issue capable of producing 'dire consequences' (Roland & Bhanji 1982, p.1). The issue began to attract wider attention with a number of articles appearing that also warned of the impact including *Bulimia and Diabetes: a potentially life-threatening combination* (Hillard, Lobo, & Keeling, 1983) and *Bulimia, anorexia nervosa, and Diabetes. Deadly combinations* (Hillard & Hillard, 1984). The initial results painted a bleak picture, Rodin et al. (1985) in the first systematic study on the subject, suggested that in T1D adolescents, Anorexia and subthreshold variants thereof were present in 8.7% of the sample representing a 6 fold increase compared to the non T1D population while Bulimia and subthreshold syndromes were present in 10.8%, representing a two fold increase. Reading this paper with a contemporary understanding of issues in researching this population however foreshadows many of the problems still apparent; insulin omission for weight control is not mentioned, the instruments used (EAT-26, EDI) are not modified in any way to accommodate T1D regimen effects, the follow up clinical interview does not appear to have been modified either and the sample size is arguably too small to make meaningful conclusions.

The state of research into T1ED currently faces similar challenges. Methods of reporting vary widely, diagnoses of clinical, subclinical, Disordered Eating Behaviour (DEB) and the role of insulin omission is still contentious and sample sizes are often small. While some studies use self-report questionnaires and suggested cut-off points for further examination, others use clinical or semi-structured interviews. The issues of using self-report measures are well documented with issues such as validity, bias, reporting errors and boredom effects (for a

review please see Gerald & George, 2010) and as such the clinical interview is deemed the gold standard of diagnostic procedures. However, the use of interviews can be costly, timely and resource intensive, whereas self-reports offer a quicker, cheaper and easier to disseminate option. Plus, interviews are not free of issues; a working understanding of the needs of certain populations must be known to the questioner or demographic specific aspects (such as insulin omission) may stay uncovered, furthermore participants may be less willing to admit behaviours to an interviewer (d'Emden et al., 2012; Sevenson et al., 2003).

Eating Disorder research utilises both self-report and interview data in order to describe patterns of cognition and behaviour related to the illness. While some offer the potential of clinical diagnosis others rely on cut-off points which indicate the need for further assessment. While instruments such as the Eating Attitudes Test (Garner & Garfinkel, 1979) or the Eating Disorders Examination Questionnaire (Fairburn & Beglin, 1994) or any other number of instruments may be entirely suitable for measuring ED symptomology in the general population, the nature of T1D has not been taken into account in many studies employing these measures in this population. As stated in the introduction, for example, many T1Ds deliberately employ a low carbohydrate diet thus avoiding whole food groups and utilise diet products which has been shown to improve blood glucose control (Brand-Miller, Hayne, Petocz & Colagiuri, 2003). Hypo and hyperglycaemic states promote biological hunger and in the case of hypoglycaemia can lead to eating which is out of control (Daneman et al., 2006) and potentially shame inducing as an indication of failure to adhere to Diabetes regimen (Sparud, Lundin, Öhrn, & Danielson, 2010). Blood glucose concerns mean that patients may avoid a meal or have the outward appearance of adopting 'strange' eating patterns and it could be argued that the very nature of T1D creates an environment where a patient is reliant on being preoccupied with food. This is problematic in research which utilises instruments that deem these sorts of behaviours as indicative of an ED.

In T1EDs, diagnostic problems are also apparent. One of the main issues of contention is whether insulin omission for weight loss purposes is included as a feature of an ED. In order to explain these issues further it is necessary to look at how changing definitions in the Diagnostic Statistical Manual (DSM) have affected the diagnostic criteria for Eating Disorders and the role of insulin omission within them. The DSM III (1980) has no mention

of insulin omission in the guidelines for EDs and neither does the revised version (1987). Insulin omission is first mentioned in the ED section in the DSM IV (1994) within the notes for Bulimia, the same is published in the DSM IV revised (2000):

> Individuals with Diabetes mellitus and bulimia nervosa may omit or reduce insulin doses in order to reduce the metabolism of food consumed during eating binges. (p. 546)

Insulin omission may be viewed as a form of purging within the Bulimia framework. In its most recent incarnation, the DSM V ( 2013) insulin omission is included as a clinical feature of both Anorexia and Bulimia. While the section in Bulimia remain identical, in the clinical features of Anorexia the following is written:

> Individuals with anorexia nervosa may misuse medications, such as by manipulating dosage, in order to achieve weight loss or avoid weight gain. Individuals with Diabetes mellitus may omit or reduce insulin doses in order to minimize carbohydrate metabolism (p. 376)

The changing status of insulin omission is significant and may contribute to the widely fluctuating estimates in prevalence of EDs in this demographic. Further problems are revealed when asking how some with T1ED for whom insulin omission is the main weight loss mechanism, would actually be diagnosed with an ED as Allan and Nash (2014) state:

> Under these diagnostic criteria, one may ask: "what is the difference between people with Diabetes and anorexia and those with Diabetes and bulimia?" Simply put, the answer is weight; however, determining Eating Disorder severity by weight is not relevant to people with type 1 Diabetes who omit insulin. The measure of severity for this demographic would more accurately be HbA1c. Furthermore, these diagnostic criteria propagate the idea that one simply has anorexia or bulimia with Diabetes as a footnote. We know that there are Diabetes-specific environmental factors that contribute to the development of diabulimia and, perhaps more importantly, that Eating Disorder treatment

programmes that do not address the Diabetes-related factors fail abjectly (p. 386) 6

## 2.2    The Current Study

A comprehensive systematic review and meta-analysis of the prevalence of Eating Disorders in Type 1 Diabetes was published by Nielson in 2002. The author was primarily interested in demonstrating whether AN, BN, EDNOS and subthreshold EDs were more prevalent in the T1 population when analysing studies that employed clinical interviews supplemented by validated self-reports between 1986 – 2000. The author concluded that while AN was not more prevalent in those with T1, BN was and T1D carried a threefold risk. It was also found that subthreshold ED and EDNOS were more common carrying a twofold risk. The author also highlights that insulin omission is an important mechanism but that studies examining this as a feature of an ED are scarce. Mannuci et al. carried out a similar metanalysis in 2005 but they only reviewed papers that utilised clinical interviews with non T1D controls, reported diagnoses of AN or BN, EDNOS was not considered (the authors state it had an 'uncertain definition' p. 417), males were excluded and only 8 papers were included in the analysis. They concluded that while AN was more prevalent in T1D participants the differences between controls was non-significant. For BN there was a significant difference, those with T1D had higher prevalence as did they for the 2 conditions combined. Insulin omission is not mentioned anywhere in the text (Mannucci et al., 2005).  By 2013 a more relaxed approach to what constitutes ED behaviour was taken by Young et al. who conducted a systematic review and meta-analysis comparing T1D adolescents to controls in rates of both clinical EDs and DEB. They also specifically investigated the relationship between these measures and glycaemic control and included measures that were modified for T1D and took insulin omission into account. They highlighted many of the problems associated with researching these behaviours in this demographic:

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6 Please see Appendix G for full article

Commonly used generic measures of eating problems validated in a general population might inflate the prevalence of eating problems in those with Diabetes, because they assess the extent to which individuals worry about their diet, reduce the intake of certain food groups and eat when they are not hungry. As a consequence of low blood glucose, or their insulin regimen, individuals with Type 1 Diabetes may score highly on such items, as they may restrict foods with high carbohydrate content or eat when they are not hungry (often because of the need to treat hypoglycemia). Furthermore, generic measures are not designed to acknowledge insulin omission as either weight loss behaviour or general non-adherence to treatment. Many previous studies have used generic measures validated in the general population, and this may contribute to higher or biased prevalence estimates. (Young et al., 2013, p. 190)

The authors conclude that both ED and DEB are more common in adolescents with T1D and that both are associated with impaired glycaemic control regardless of whether the instrument was modified for T1D.

Given the reservations of previous researchers and their highlighting of the substantial issues faced when researching T1EDs the purpose of the current study is to provide an updated broad overview of how EDs in the T1 population have been investigated since the publication of the 2002 Neilsen review. As such the current study will systematically review how Eating Disorders in Type 1 Diabetes have been researched and whether previous assumptions made about this population are valid by considering the sample size, age of participants, the primary methodology, sample recruitment , the measurement instrument used, whether that measurement has been modified for T1D regimen, the prevalence of diagnoses or number of those scoring above designated cut-off points reported if any, whether and how insulin omission was measured and what the prevalence of insulin omission is.

## 2.3 Methodology

### 2.3.1 Search Terms and Databases

A comprehensive search was run using the MEDLINE & PubMed Databases using the following terms. (Diabetes Mellitus, type 1* [MeSH Terms]) AND (Feeding and Eating Disorders* [MeSH Terms]), Diabetes mellitus, type 1/ psychology [MeSH Terms], Diabetes mellitus, type 1/ psychology [MeSH Terms] AND "Eating Disorder*" [All Fields] and Feeding and Eating Disorders* [MeSH Terms] AND "Type 1 Diabetes" [All Fields]. A

search was similarly run on PSYCH INFO using the terms 'Eating Disorders', 'Type 1 Diabetes', 'Type 1 Diabetes Mellitus'

Following this a hand search of the references section of relevant papers was used to identify any other papers which may have been unidentified by the original search.

### 2.3.2   Inclusion/ Exclusion Criteria

Inclusion Criteria

1)      English Language

2)      Has some measure of Eating Disorders or Disordered Eating Behaviour in Type 1 Diabetes

Exclusion Criteria

1)      Review Articles (although used in identifying references)

2)      Individual Case Studies

3)      Do not measure Eating Disorders in Type 1 Diabetes

4)      Studies with Diabetes type not differentiated

5)      Studies not in English language

213 papers were initially identified by the search. Of these 47 duplicates were removed. Of the remaining 166 papers, all were read by 2 postgraduate research assistants who wanted to gain experience in the methodology of systematic reviewing and reviewed again by the researcher to ascertain suitability for inclusion in the review. Papers using the same sample of (n = 4) were removed, 60 papers were retained for the analysis (please see figure 2:.1 below)

### 2.3.3 Data Extraction

From every study the authors, country of origin, sample size, recruitment methods, study design and gender were extracted, The measures utilised by each paper were recorded alongside whether or not the instruments were modified in any way to account for the effects of Type 1 Diabetes. The diagnoses or cut-off criteria and the prevalence of insulin omission observed in the sample was then reported if it was available.

*Figure 2.1: Flow Chart of Search Process*
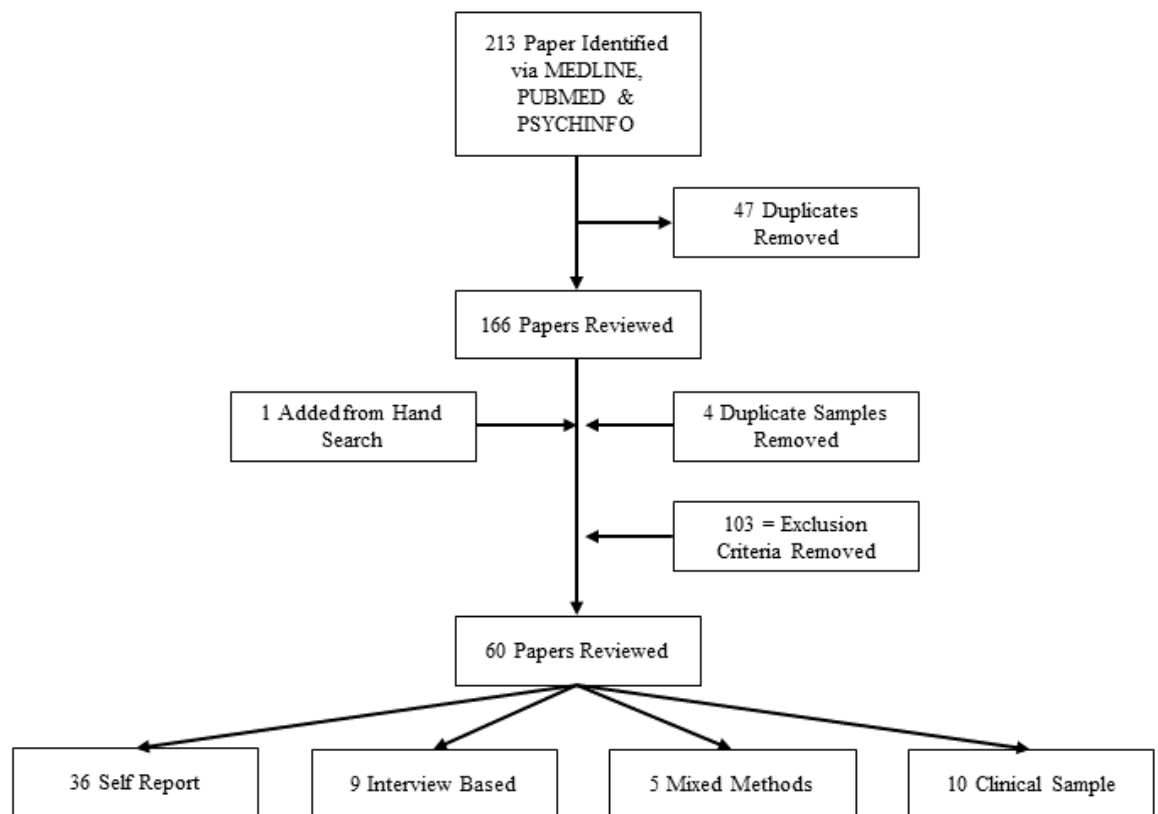
## 2.4    Results

*Table 2.1: Self Report Research*

| Author/ Year/ Country of Origin | n (f/m) | Age Range or x̄ (sd) | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adapted? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| Altinok et al., 2017 (Turkey) | 110/ 90 | 9 - 18 | Cross Sectional | Routine Clinic Visit | DEPS-R | 29. 1%f > cut-off 17.8%m > cut-off | Yes | Yes | 22%f, 25%m |
| Doyle et al., 2016 (US) | 27/ 33 | 18 -28 | Observational | Routine clinic visit | DEPS-R | 29.6% f > cut-off 18.2% m > cut-off | Yes | Yes | n/r |
| Bachle et al., 2016 (Germany) | 402/ 417 | 11 - 21 | Population | Nationwide Survey | SCOFF | 30.9%f/ 11.1% m > cut-off | No | Yes | 6.9%f, 7.2%m |
| Powers et al., 2016 (US) | 153/ 115 | x̄ = 19 (9.2) | Cross Sectional | Questionnaire via health records | EDEQ | Probable ED 7.8%* Possible ED 34%* No ED 58.2% * | No | n/r | n/r |
| Bachle et al., 2015 (Germany) | 126/ 85 | 18 – 21 | Population Study, Control | Nationwide Survey | SCOFF | 30.2%f, 9.5%m > cut-off | No | No | n/a |
| Caccavale et al., 2015 (US) | 73/78 | 13 – 18 | Cross Sectional | Routine clinic visit | DEPS-R | n/r | Yes | Yes | n/r |
| Zuijdwijk et al., 2014 (Canada) | 43f | x̄ = 15.8 (1.7) | Scale Validation | Not stated | EDI SCOFF | EDI high risk 23.2% Scoff at risk 27.9% | Yes | Yes | n/r |
| Baechle et al., 2014 (Germany) | 289/ 340 | 11 – 17 | Population | Nationwide Survey | SCOFF | DEB 31.2%f/ 11.7%m | Yes | Yes | 20.5%f 18.5%m |
| Johnson et al., 2014 (UK) | 57/ 39 | 19 – 21 | Observational | 2 clinics: Not Reported | DEPS-R | Elevated DEB 35.1% * | Yes | Yes | n/r |
| Sivertsen et al., 2014 (Norway) | 21/ 19 | 16 – 19 | Control | population/ registry | EDS | n/a | No | No | n/a |
| Merwin et al., 2014 (US) | 189/ 112 | 18 – 76 | Model Validation | 2 Diabetes Clinics: Email invitation | DEPS-R, Researcher Constructed | < DEPS-R cut-off 22%* | Yes | Yes | n/r |

| Author/ Year/ Country of Origin | n (f/m) | Age Range or x (sd) | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adapted? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| Williams et al, 2014 (Australia) | 42/ 19 | 13 – 17 | Cross sectional | Advertising through charity | EAT-40 | n/r | No | No | n/a |
| d'Emden et al., 2013 (Australia) | 66/ 58 | 13 – 18 | Cross Sectional | Routine clinic visit | YEDEQ, EDI 3RC | YEDEQ, DEB 32.3%f, 25.9%m, EDI - 3RC, n/r | Yes | Yes | 7.6%f 3.4%m |
| Phillipi et al., 2013 (Brazil) | 141/ 48 | 12 – 56 | Cross Sectional | 3 Diabetes Clinics: Invitation | BITE, EAT-26, BES | cut-off EAT 45% BITE 40% BES 16% Cumulative 58.7%* | No | Yes | 14.8%f 0%m |
| Bernstien et al., 2013 (US) | 73/ 77 | 11 – 25 | Cross sectional | Routine clinic visit | Eating Disorder Screen for Primary Care | DEB 20.7%* | No | Yes | 13.30%* |
| Markowitz et al., 2013 (US) baseline | 19f/ 24m | 10 – 17 | Longitudinal | 3 Diabetes Centres: Not Reported | DEPS-R | n/a | Yes | Yes | 14%* |
| Wisting et al., 2013 a/b (Norway) | 390/ 380 | 11 – 19 | Population | Routine clinic visit | DEPS-R, EAT12 | DEPS-R cut-off 27.8% f, 8.6%m | EAT no | Yes | 36.8%f, 9.4% m |
| Tse et al., 2012 (US) | 72/ 79 | 8 - 13 | Cross sectional | Routine clinic visit | DEPS | Low Risk 85% At Risk 15%* | Yes | Yes | n/r |
| Quick et al., 2012 (US) | 20f | 18 – 26 | Control | Flyers in 3 universities | EDEQ, TFEQ, NEQ | n/a | EDEQ yes, TFEQ no, NEQ no | Yes | n/r |
| Goebel-Fabbri et al., 2011 (US) | 207f | X = 44 (12) | Longitudinal | Routine clinic visit | BITE Researcher Constructed | n/a | No | Yes | 36% |
| Alice Hsu et al., 2009 (Taiwan) | 42/29 | 10 - 22 | Control | Routine clinic visit | BITE, EAT-26 | BITE cut-off 4.8% f / 3.4% m EAT cut-off 23.8% f/ 17.2% m | No | No | n/a |

| Author/ Year/ Country of Origin | n (f/m) | Age Range or x (sd) | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adapted? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| Markowitz et al., 2010 (US) | 63/ 49 | 13 – 19 | Scale Validation | Routine clinic visit | DEPS-R | n/a | Yes | Yes | 24%m, 29%f |
| Ackard et al., 2008 (US) | 73/73 | 12 - 21 | Control | Routine clinic appointment, letter | AHEAD | Any unhealthy weight control behaviour 4.3% m/ 20.7f Less extreme unhealthy weight control 3.7%m/ 16.1%f Extreme unhealthy weight control 2.3%m/ 5.1% f | Yes | Yes | 2.8%m 17.7%f |
| Sim et al 2009., (US) | 20f | 15.8 (20.11 months) | Cross Sectional | 3 outpatient clinics: Invitation | EDEQ | n/r | No | No | n/r |
| Markowitz et al., 2009 (US) | 90f | 12 - 18 | Cross Sectional | Paediatric Clinic or Summer Camp: Invitation | EDEQ, TFEQ, PFS | EDEQ 20% within Clinical Range | No | No | n/r |
| Goebel – Fabbri et al., 2008 (US) | 234f | 13 – 60 | Longitudinal | Routine clinic visit | BITE, EDI | n/r | No | Yes | 30% |
| Smith et al., 2008 (UK) | 40f | 11 – 19 | Comparison/ control | 4 Clinic: Routine clinic visit | EDEQ | BN 15% BED 12.5% | No | No | n/a |
| Ryan et al., 2008 (France) | 16/ 27 | x = 38.3 | Cross Sectional | Outpatients: sent an invitation | QWEPR TFEQ | Over Eating or Binge Eating 26%m 0% female | No | No | n/a |
| Battaglia et al., 2006 (US) | 69f | 12 – 18 | Cross Sectional | Routine clinic visit | EDI, EAT-26 (Dietary Restraint Subscale) | n/a | EAT-26 No, EDI Yes | Yes | 15% |
| Pinar et al 2005., (Turkey) | 23/ 22 | 12 – 18 | Control | Outpatient Clinic | EAT-40 | 68.9% > cut-off * | No | Yes | 40%* |

| Author/ Year/ Country of Origin | n (f/m) | Age Range or x (sd) | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adapted? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| Pollock-BarZiv & Davies, 2005 (Canada) | 52f | 12 – 27 | Cross Sectional | Adverts via the internet and the community | EDEQ, DSMED | Combined 27% > cut-off | Yes | Yes | 27.50% |
| Iafusco et al., 2004 (Italy) | 92/ 101 | 8 – 18 | Control | Not Reported | EDEQ | Clinical Eating Disorder 0% Subthreshold 5% | Yes | Yes | n/r |
| Maharaj et al., 2003 (Canada) | 88f | x = 15 (2.2) | Cross Sectional | Children's Hospital | EDI, DSMED | Combined Highly Eating Disturbed 20.5%, Mildly Eating Disturbed 34%, Non-Eating Disturbed 45.5% | No | Yes | 17% |
| Schwartz et al., 2002 (US) | 45f | 12 - 18 | Cross Sectional | Routine clinic visit | EDEQ | n/a | No | No | n/r |
| Neumark – Sztainer et al., 2002 (US) | 70/ 73 | 12 – 21 | Cross Sectional | Routine clinic appointment, letter | AHEAD | No weight control 46.4% m / 7.6% f, Healthy weight control 37.7% m/ 54.5% f, Unhealthy weight control 13% m / 18.2% f, Very Unhealthy weight control 2.9% m /19.7% f | Yes | Yes | 1.4% m/ 10% f |

DEPS-R = Diabetes Eating Problem Revised, SCOFF = Sick, Control, One, Fat, Food. YEDEQ/ EDEQ = The Eating Disorders Examination and Youth Eating Disorders Examination Questionnaire, EAT = Eating Attitudes Test, EDI = The Eating Disorder Inventory, EDS = The Eating Disturbance Scale, BITE = Bulimia Inventory Test Edinburgh, BES = The Binge Eating Scale, TFEQ = The Three Factor Eating Questionnaire, NEQ = The Night Eating Questionnaire, AHEAD = Assessing Health and Eating among Adolescents with Diabetes, QWEPR = Questionnaire of Weight and Eating Patterns, DSMED = The Diagnostic Survey for Eating Disorders, PFS = Power of Food Scale

n/r = not reported, n/a = not applicable,

DEB = Disordered Eating Behaviour.

*Gender split not reported, n/r = not reported, n/a = not applicable, sub = subthreshold

*Table 2.2: Interview Based Research*

| Author/ Year/ Country of Origin | n (f/m) | Age Range or x | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adaptation? | Insulin Omission Measured? | Insulin Omission (%) |
|---|---|---|---|---|---|---|---|---|---|
| Wisting et al 2017 (Norway) | 61/ 43m | x=15.7 (1.8) | Cross Sectional | Nationwide Study | ChEDE | n/r | Yes | Yes | n/r |
| Wisting et al 2015 (Norway) | 61/ 44 | 12 – 20 | Cross Sectional | Nationwide Study | ChEDE | n/a | Yes | Yes | n/r |
| Colton et al 2015 (Canada) | 71f | 19.2 – 27.8 | Longitudinal | 1 Clinic: Invitation | EDE | AN 2.8% AN BN 1.4% EDNOS 28.2% subED 8.5% DEB 59% | Yes | Yes | 26.80% |
| Wilson et al 2014 (Ireland) | 30/ 20 | 14 – 16 | Cross Sectional | 1 Clinic: Invitation | ChEDE | no ED %100 | Yes | Yes | 0% |
| Colton et al 2013 (Canada) | 98f | 14 – 18 | Longitudinal | 1 Clinic: Invitation | EDE | ED criteria 13.3% BN 3.1% EDNOS 3.1% subED 7.1% DEB 35.7% | Yes | Yes | n/r |
| Grylli et al 2010 (Austria) | 76f | x=17.2 (2.1) | Cross Sectional | Outpatients: Invitation, Various Districts | EDE | EDNOS 11.8% subED 18.4% | No | No | n/a |
| Olmsted et al 2008, (Canada) baseline | 126 f | 9 – 13 | Longitudinal | Routine clinic visit | ChEDE | DEB 15% | Yes | Yes | 2.40% |
| Peveler et al 2005 (UK) | 87f | 11 – 38 | Longitudinal | Follow up | EDE | Clinical ED 14.9%, subED 11.1% | Yes | Yes | 35.60% |
| Colton et al 2004 (Canada) | 101f | 9 -14 | Control | Routine clinic visit | ChEDE | EDNOS % 2 subED % 6 | Yes | Yes | 2% |

*Gender split not reported, n/r = not reported, n/a = not applicable, sub = subthreshold, AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified, DEB = Disordered Eating Behaviour, EDE/ ChEDE = Eating Disorder Examination/ The Child Eating Disorder Examination

*Table 2:3: Mixed Methods Research (Self Report & Clinical Interview)*

| Author/ Year/ Country of Origin | n (f/m) | Age Range or x̄ (sd) | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adapted? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| d'Emden 2012 (Australia) | 66/ 58 | 13 – 18 | Scale Validation | Routine clinic visit | YEDEQ | correlational design | Yes | Yes | 3.4%m |
| | | | | | EDI | | | | 7.6%f |
| | | | | | chEDE | | | | 3.90% |
| Grylli et al 2005 & 2004(Austria) | 96/ 103 | 14.1 (2.6) | Cross sectional | 10 Clinics: Sent Letter, approached during Routine Clinic Visit or Diabetes Camp | EAT-26, EDI | > cut-off 31.25f%, 4.6%m | No | No | n/a |
| | 30/ 5 | | Observational | | EDE | No ED, 30%f BN 6.6%f EDNOS 6.6%f subED 40%f, 1%m | | | 26%f 0%m |
| Garcia – Reyna 2003 (Spain) | 38/ 60 | 12 – 16 | Control | Not Reported | EAT-40 | > cut-off 3.1% | No | No | n/a |
| | 13* | | Clinical Interview | | EDE | EDNOS 5.3%f, 1.7%m subED 10.5%f, 10%m | | | |
| Sevenson et al 2003 (Sweden) | 141m | 14 – 18 | Control | Routine clinic visit | EDI – C | DFT (EDI) 1.4% | Yes | Yes | 2.10% |
| | | | | | RABT | Clinical ED (RAB) 0% | | | 0.00% |

*Gender split not reported, n/r = not reported, n/a = not applicable

sub = subthreshold AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified, DEB = Disordered Eating Behaviour

EDE/ ChEDE = Eating Disorder Examination/ The Child Eating Disorder Examination, YEDEQ/ EDEQ = The Eating Disorders Examination and Youth Eating Disorders Examination Questionnaire, EDI = The Eating Disorder Inventory, EAT = Eating Attitudes Test, DFT = Drive for Thinnes, RABT = Rating for Anorexia and Bulimia

### 2.4.1 Country of Origin

Research came from 16 different countries with America producing the majority of articles. The UK only produced 3 pieces of research that was included in the systematic review. In non-clinical samples, the highest level of self-reported symptomology in young people came from Turkey with 68.9% of the 45 mixed gender respondents scoring above the cut-off point for clinical concern on the EAT 40. This research also provided the highest level of insulin omission seen in young people with 40% of the sample reporting this behaviour (Pinar et al 2005). Researchers from Ireland using a mix gender sample and the Eating Disorder Examination, and from Sweden using a male only sample and the RABT found 0% ED symptomology (Wilson et al., 2014; Sevenson et al., 2003). The same participants in the Irish study reported 0% insulin omission and an Austrian sample reported the same for young males (Wilson et al., 2014; Grylli et al., 2005, 2004).

In adult and mixed samples, the highest level of symptomology was found using a composite of risk measures in Brazil at 58.7% (Phillipi et al., 2013) and the highest rate of insulin omission reported was 36% in the US (Goebel- Fabbri et al., 2011). The lowest rates reported was 0% binge eating symptomology in females using the QWEPR in France (Ryan et al., 2008) and 0% insulin omission in males in Brazil (Phillipi et al., 2013)

This having been said there is a lack of international diversity represented to be able to extrapolate these conclusions. Several countries only produce 1 piece of eligible research for the systematic review and there is no representation from large swathes of the globe as can be seen in figure 2.2.

*Table 2.4: Country of Origin*

| Country | No of Articles |
|---|---|
| US | 21 |
| Canada | 8 |
| Norway | 5 |
| Japan | 4 |
| Australia | 3 |
| UK | 3 |
| Germany | 3 |
| Austria | 3 |

51

| Country | No of Articles |
|---------|---------------:|
| Spain   | 2 |
| Brazil  | 1 |
| Taiwan  | 1 |
| France  | 1 |
| Turkey  | 2 |
| Italy   | 1 |
| Ireland | 1 |
| Sweden  | 1 |

*Figure 2.3: Country of Origin*
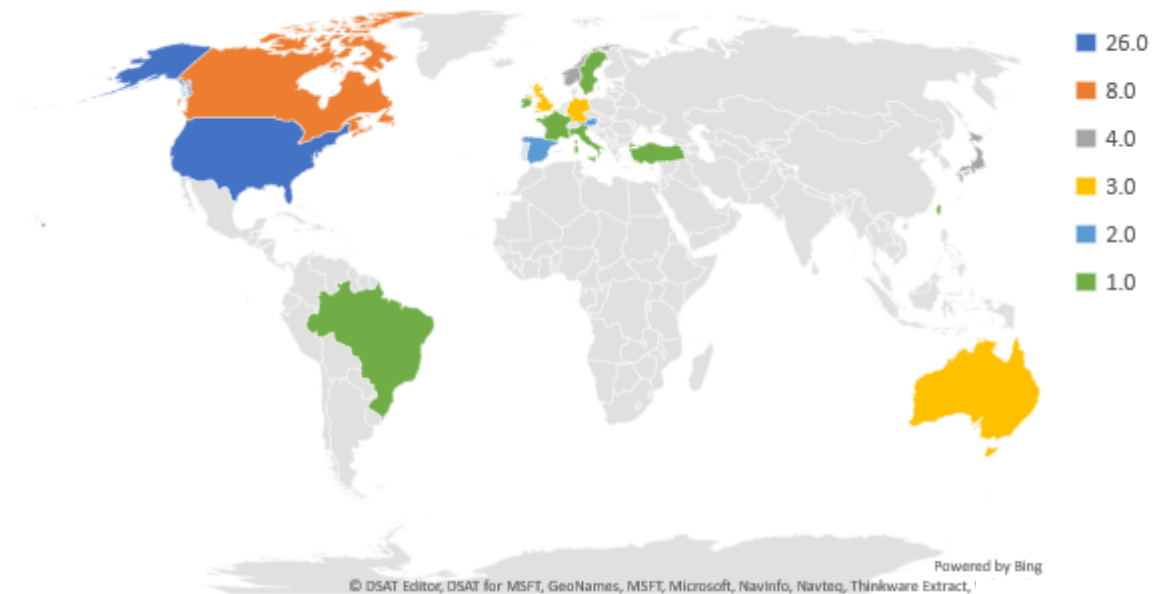


### 2.4.2 Sample Size

One of the main limitations of research in T1EDs is the small sample sizes utilised. In non-clinical samples just under half of the studies included utilised a sample size of less than 100 participants, 2 of the studies only included 20 participants and only 4 of the studies utilised a large-scale sample of 500 participants or more. As a relatively rare disease this may be understandable, but results must be approached with a certain amount of caution due to this issue. This having been said in research utilising large samples there is not a marked

difference in prevalence than in those using smaller sample sizes, they neither represent the highest or lowest estimates (as can be seen in tables 2.1 – 2.3).

*Figure 2.3: Sample Sizes in Non Clinical Samples*
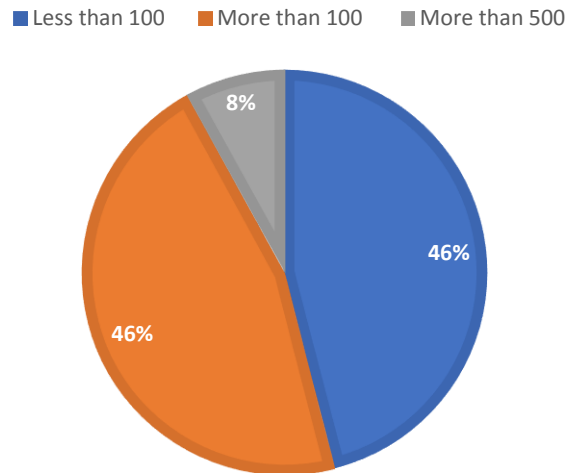


### 2.4.3   Gender

Although the majority of papers utilised a mixed gender sample, when one gender was examined separately it was typically female. In the 1 sample in this review that utilised an exclusively male sample, nearly negligible rates of ED symptomology using both self-report and clinical interview were reported, although insulin omission was evident in 2% (Sevenson et al., 2003). Rates of symptomology in males in other studies varied substantially. The highest rates of symptomology in young males was 25.9% using a composite of the YEDEQ and the EDI by d'Emden et al. (2013) and the lowest was 0% using the EDE (Wilson et al., 2014; Grylli et al 2005, 2004). Also, among young males, insulin omission rates vary with the highest levels reported as 24% (Markowitz et al., 2010) and the lowest as 0% (Grylli et al., 2005, 2004). In the 1 article reporting any gender separated results in adults, 0% insulin omission was found (Phillipi et al., 2013). For males it is important to note that rates of internal consistency may be lower using self-report scales such as the EDI and YEDEQ potentially because these instruments have typically been validated in those with clinical Eating Disorders who are typically female (d'Emden et al., 2012).

There do appear to be gender differences in regard to the type of ED symptomatology reported. Levels of ED symptomology are much higher in females than males in youth samples, using the EDI at least mild levels of Eating Disturbance was found in 54.5% of the female group (Maharaj et al., 2003) and insulin omission seen in as many as 36.8% (Wisting et al., 2013 a/b). In the adult samples using the EDE high levels of clinical behaviour was also seen, in one study 60% of the female sample demonstrated disturbed eating behaviour (Colton et al., 2015). In two studies insulin omission of 36% was reported (Peveler et al., 2005; Goebel-Fabbri et al., 2011). Alice Hsu et al. (2008) reported higher levels of vomiting and use of diet pills in T1D adolescent females than males but they also found higher levels of bulimia like behaviour in T1D boys than in the general population.  Similar results were found by Pinar et al. (2005). In research utilising the DEPS-R, females score higher than males even if the difference between genders is insignificant (Doyle et al 2016). In two studies however, insulin omission was found to be more prevalent in males than in females (Altinok.  2017; Bachle et al., 2015). Given that this is more recent research it highlights the need for further research into male only samples.

In the clinically diagnosed T1ED samples, research subjects were nearly completely females, in the 10 articles only 12 of the total participants were male as can be seen in table 4.

*Figure 2.4: Gender of Participants in Non-Clinical Samples*
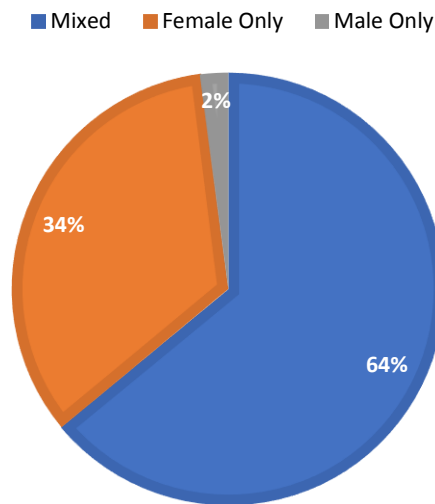
### 2.4.4    Age Range

The majority (n = 38) of research utilising non-clinical samples into T1EDs focussed on children, adolescents and young adults aged 8 – 21 years old. The remainder (n = 12) focussed on either a mixed age range or adults 11 – 76 years old. In general, it seems that ED symptomology and rates of insulin omission are higher in adult samples than in samples utilising exclusively children and young people. In clinical samples (n = 10) the majority of research used adolescent and adult samples with ages ranging from 14 - 44 but this is maybe to be expected as this research was primarily conducted in patients attending some sort of clinic for their ED.

*Figure 2.5: Age Range of Participants in Non-Clinical Samples*
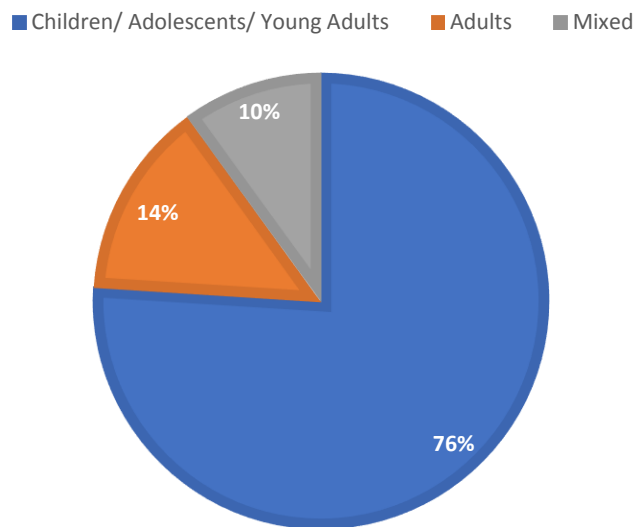


### 2.4.5    Design

#### *2.4.5.1   Methodology*

##### *2.4.5.1.1   Cross Sectional*

Most of the research undertaken used a cross sectional design and utilised only patients with Type 1 Diabetes. It should be noted that the cross-sectional research all utilised point prevalence with some of the scales asking what behaviour had been carried out over the last

month or the last 3 months. None of the studies were looking at lifetime prevalence. Since there appears to be little consensus as to what the most appropriate instrument to use in there is also little agreement among the cross-sectional research as to prevalence rates. Phillipe et al. (2013) used a composited measure of the Binge Eating Scale, the BITE and the EAT and found that cumulatively 58.7% of the sample was at risk for ED behaviour whereas Grylli et al. (2004, 2005) using a combination of the EAT and the EDI found ED behaviour in only 4.6% of male respondents. Only 2 papers attempted to describe clinical diagnoses in the sample, Wilson et al. (2014) found a 0% rate of clinical EDs using the chEDE in an adolescent sample but Grylli et al. (2010) found 11.8% of EDNOS and 18.4% subthreshold EDs using the EDE. Insulin omission rates were reported by 8 of the cross-sectional papers with the highest level (27.5%) reported by Pollock-BarZiv (2005) and the lowest level (0%) reported by Wilson et al (2014).

### 2.4.5.1.2 *Observational Studies*

3 observational studies were reported in the literature. 2 of these studies used the DEPS-R and found as many as 35% of the mixed samples of young people/ young adults scoring above the cut-off point (Doyle et al., 2016; Johnson et al., 2014) Another study used the EAT-26 and the EDI to screen youth who would then go on to attend a clinical interview if they scored above the cut-off point. Of those who did subthreshold EDs and insulin omission were found in 40% and 56% of the females respectively (Grylli et al., 2004, 2005).

### 2.4.5.1.3 *Control Studies*

Controlled studies were primarily concerned with comparing rates of Eating Disorders and Eating Disorder symptomology in those with and without T1D. There is conflicting evidence as to whether this is the case.

Using an unmodified version of the EDI Kaminsky and Dewey (2013) found no differences in adolescent ED behaviour and body image ratings but this research only utilised a T1D control group of 27. A similar result was found using the Eating Disturbance scale, but this research also utilised a very small sample size of Diabetics (n = 40) compared to a control group of 9843 (Sivertsen et al., 2014). Using a much larger population based study and the

SCOFF, Baechle at al. (2014) came to the same conclusion even though high levels of those with T1D were above the cut-off point (31.2%f, 11.7%m). Ackard et al. (2008) used the AHEAD protocol within project EAT (eating amongst teens) in order to ascertain the differences between healthy adolescents and their diabetic peers, they assessed whether any kind of weight control was present including behaviour that they deemed 'healthy' such as exercising. They found that those with T1D were more likely to endorse healthy behaviours (such as having regular meals) they also found that they were less likely to report weight dissatisfaction concluding that T1 may be a protective factor, although they still found significant prevalence of insulin omission. In a study where adolescent girls with T1D were compared with healthy controls and a group with scoliosis it was found that Bulimia and binge eating were significantly more prevalent than in either of the other groups (Smith et al., 2008).

Of the other control studies, though there was significant evidence that T1D is a risk factor for EDs, where adults with T1D have been encapsulated into a larger sample of patients with 'Diet Related Chronic Health Conditions' it was found that the latter group were twice as likely to have been diagnosed with an Eating Disorder than healthy controls (Quick et al., 2012). Using the EAT-26 and the BITE it was found that in a Taiwanese control study T1D females and males scored higher on the BITE and on the Bulimia subscale of the EAT-26 than their non diabetic counterparts. Pinar et al. (2005) found that Disordered Eating Behaviour was 4 times as common in diabetic adolescents than their non-diabetic peers. A study using only male adolescents found that Type 1 boys were heavier and had a higher drive for thinness using the EDI DFT subscale than their non-diabetic peers (Sevenson et al., 2003).  In a mixed gender study using a modified version of the EDEQ it was found that adolescents with T1D were more likely to have sub threshold EDs than their non T1D counterparts (Iafusco et al., 2004). In Spanish research both EDNOS and subthreshold EDs were found in significantly more T1D adolescents than in non-diabetic controls (Garcia – Reyna et al 2003).

### 2.4.5.1.4 Longitudinal Studies

There is a paucity of longitudinal studies in this demographic, in one of the few reviewed Goebel-Fabbri et al. (2008) found that insulin restriction at baseline predicted a number of negative outcomes such as rates of nephropathy. It also increased the risk of mortality by 3.2 times and decreased life span from an average of 58 to 44 years at 11 year follow up. Similar results were found by Peveler et al. (2005), insulin restriction and Eating Disorder symptomology at baseline indicated higher levels of mortality and microvascular complications at 8 – 12 year follow up.

In terms of prevalence Colton et al describe a series of studies where 126 females with T1D were followed for a 14 year period. They found that there was a cumulative probability of 60% of ED onset before the age of 25 and a high chance of recurrence regardless of whether patients had initially demonstrated remission. (Colton et al., 2015). In a shorter-term longitudinal study of baseline, 1 and 6 months it was found that initiation of insulin pump therapy led to less endorsement of Eating Disorders behaviours (Markowitz et al., 2013).

### 2.4.5.1.5 Population Based Studies

There was very little in the way of large scale population studies reviewed. Of the 3 undertaken in Germany using the SCOFF (Baechle et al., 2014, 2015, 2016) there was a large proportion of participants scoring above the cut-off point, particularly females. In a population study of Norwegian patients using the DEPS-R it was found that 27.8% of females were above the cut-off point and 36.8% reported at least occasional insulin restriction. For males 8.6% scored above the cut-off point and 9.4% reported insulin omission (Wisting et al., 2013)

### 2.4.5.1.6 Model Validation

One study used a model generating methodology in order to ascertain if there are relationships between Eating Disorder symptomology, eating patterns, hypoglycaemia, insulin omission and other appetite factors (Merwin et al., 2014). The authors concluded that disinhibited eating led to more maladaptive cognitions and behaviours.

*2.4.5.1.7 Scale Validation*

Three studies were undertaken in order to validate the use of certain instruments in the T1D population. Markowitz et al. (2010) proposed a shorter and updated version of the DEPS (Antisdel et al., 2001) that would consider newer ways of managing T1D, such as MDI and pump therapy. In order to validate the DEPS-R several measures and a youth sample was used, parents and clinicians were also consulted. The Diabetes Family Conflict Scale (Hood, Butler, Anderson & Laffel, 2007), the Blood Glucose Monitoring Communication questionnaire (Hood, Butler, Volkening, Anderson & Laffel, 2004), the Paediatric Quality of Life Inventory (Varni, Seid & Rode, 1999), the Problem Areas in Diabetes Survey–Parent version (Antisdel, 2000), and the Diabetes Quality of Life for Youth questionnaire (Ingersoll & Marrero, 1991) were all completed by participants and their parents while medical records were consulted regarding other relevant medical information and a consultant completed the a questionnaire relating to compliance which included items on insulin omission (Jacobson et al., 1990). The authors argue that the use of these particular instruments demonstrate concurrent validity of the DEPS-R as the presence of an ED would cause probable disruption that can be can be measured thusly. While they state that the instrument is sensitive to the presences of EDs as can be seen by the correlation with the aforementioned factors and that physician adherence rating is related to DEPS-R scores they did not take clinical ED diagnoses or any other ED behaviour into account during the validation process.

d'Emden et al also used a scale validation methodology to assess whether the YEFEQ and the EDI3RC were suitable for use in adolescent T1Ds by comparing them with the chEDE. The authors kept the original questions in the YEDEQ and the EDI3RC but added in questions on insulin omission to be similarly rated as a disturbed eating behaviour. The authors conclude that both subscales are suitable for use in the T1D demographic, even though they state that there are items which could be potentially biased (d'Emden et al 2012).

Zuijdwijk et al. (2014) developed a modified version of the SCOFF that replaced the 5th question with another on insulin omission and validated it via scores on a modified EDI which removed questions on dietary restrictions which could potentially be attributed to T1D. The authors conclude that as there is agreement between the two instruments that their

version of SCOFF is suitable for use in the population. However, they deliberately excluded those who had a clinical ED diagnosis which would have provided a reference category and better ecological validity.

*Figure 2.6: Methodology Utilized in Non-Clinical Samples*
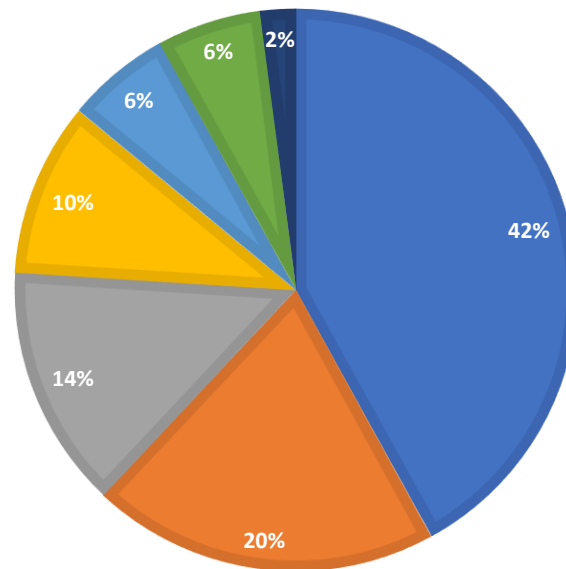


### 2.4.5.2   Sample Recruitment

The method of sample recruitment may go some way to explain the amount of variation reported in symptomology. Most of the research utilises samples from small geographical areas such as a paediatric clinic or a few hospitals in the region. The most common type of recruitment took place during a routine clinical visit. This may have to be considered in terms of selection bias, as only those who regularly attend clinic during the study period may have been approached. It may be that as such research has been based on more typically adherent patients. This is alarming as the conclusions made in the literature would therefore be conservative.

*Figure 2.7: Recruitment Type Utilized in Non-Clinical-Samples*
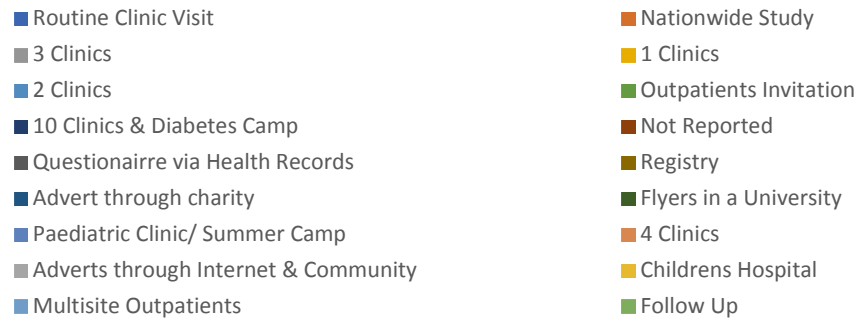
### 2.4.6    Scales Used

*Figure 2.8: Scale & Interview Instruments used in Non Clinical Samples*
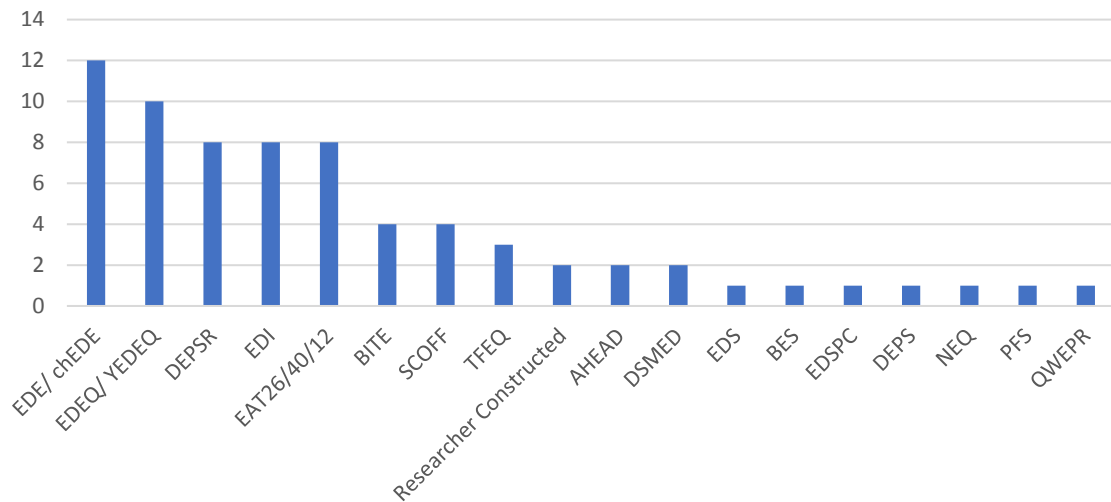


### 2.4.7    Not Diabetes Specific

### *2.4.7.1    The Eating Disorders Examination and Youth Eating Disorders Examination—Questionnaire (EDEQ/ YEDEQ)*

The EDEQ and the YEDEQ were the most commonly used self-report measures. They are short self-reports based on the main tenets of the EDE proposed by Fairburn and Beglin in 1993 (please see section 2.4.9.1 below). As such the items measure the EDE's 4 core restraint, eating concern, shape concern and weight concern. Questions include *Has thinking about food, eating or calories made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?, Over the past 28 days how many times have you eaten what other people would regard as an unusually large amount of food (given the circumstances), Over the past 28 days, on how many days have you eaten in secret (ie furtively)? Do not count episodes of binge eating* and *How much would it upset you if you had been asked to weigh yourself once a week (no more, or less often) for the next four weeks*. Responses are based on a 6 item Likert scales with responses from '0 days' to 'everyday' and scores to the subscales are then divided and reported as means and standard deviations. A global score can also be attained by summing the subscales and

dividing by 4. While Fairburn and Belgin do not explicitly recommend cut-off points they do provide community norms in order to compare scores (Bohn et al., 2008).

The EDEQ has been modified by some researchers to include insulin misuse as a disturbed eating behaviour while keeping the original scoring intact however what these items are is often not reported.

> Questions regarding insulin misuse for weight control were added by us with permission from the authors and rated similarly as a disturbed eating behaviour, but the original scoring of the tools remained intact (d' Emden et al., 2012, p. 974).

Similarly, other researchers claim that the EDEQ has been modified to account for T1D but do not explain how (Iafusco et al., 2004). Quick et al. (2012) consider insulin omission as a compensatory behaviour in the binge eating scale of the EDE where 4 or more incidents within the defined time is considered of clinical importance.

Out of the papers reviewed using either the EDEQ or its youth version only 5 provided information regarding diagnoses or cut-off points. In a youth sample using an unmodified version of the EDEQ Markowitz et al. (2010) found that 20% of the female only sample scored above the cut-off point. Using a smaller but mixed age sample of females and a modified version of the EDEQ, Pollock-BarZiv et al. (2005) found a higher rate of participants scoring above the cut-off threshold at 27%. Utilising an adult mixed gender sample and a modified version of the EDEQ over 40% of participants were screened as potentially clinically concerning (Powers et al., 2016) and in the one study that used the EDEQ for diagnostic purposed (unmodified) in a small sample of females it was found that 15% had probable BN and 12.5% BED. In another article, while no cases of full threshold ED were found in the mixed gender sample using a modified version of the EDE, 5% of the sample were reported to have subthreshold ED behaviours (Iafusco et al., 2004).

### 2.4.7.2   The Eating Disorder Inventory

The EDI was proposed by Garner et al. in 1983 as a comprehensive 64 item self-report answered on a 6 point Likert scale from 'always' to 'never'. It was designed to detect

cognitive and behavioural indicators of AN and BN. The Scale has been revised with the most recent version being released in 2004. The current version, EDI3, comprises 91 items which make up 12 subscales and 6 composite scores. The subscales are Drive for Thinness (DT), Bulimia (B), Body Dissatisfaction (BD), Low Self-Esteem (LSE), Personal Alienation (PA), Interpersonal Insecurity (II), Interpersonal Alienation (IA), Interoceptive Deficits (ID), Emotional Dysregulation (ED), Perfectionism (P), Asceticism (A), and Maturity Fears (MF). The composite scores are computed. They are the Eating Disorder Risk Composite (EDRC) which includes the DT, B and BD scales, Ineffectiveness Composite (IC) which includes LSE and PA, Interpersonal Problems Composite (IPC) which comprises II and IA, Affective Problems Composite (APC) which uses ID and ED, Overcontrol Composite (OC) which sums the scores from P and A, and General Psychological Maladjustment (GPMC) which uses all of the psychological subscales to provide a composite. The scores generated are then compared to non-clinical samples using percentiles in order to ascertain the clinical profile. It also comes with another self-report form which investigates frequency of behaviours and therefore has the potential to be used for DSM diagnostic purposes.

There were early attempts to modify certain parts of the EDI to account for T1D specific effects. In 1989 Steel, Young, Lloyd & Macintyre utilised the following protocol to identify potentially biased items:

> Ten members of the medical staff working in the diabetic clinic were asked to complete the EAT and EDI as though they were sensible young Diabetics without an Eating Disorder, so as to identify any questions to which respondents might give biased answers simply by following recommended diabetic treatment guidelines. A question was considered to be biased if over half the medical staff independently suggested it to be so (Steel et al., 1989, p. 515)

The questions removed were all from the drive for thinness scale *I eat sweets and carbohydrates without feeling nervous. I think about dieting,* and *I feel extremely guilty about over – eating*. Jones et al. report removing potentially similar items and suggesting cut-off points for the EDI which is a protocol adopted by other researchers (Jones et al., 2000; Zuijdwijk et al., 2014)

Other studies do not claim to have altered the original structure of the EDI or its composites but rather have added to them, while still keeping the original scoring matrix intact, by asking questions regarding insulin omission. The protocol adopted by Meltzer has been emulated in several other research papers (d'Emden et al 2012, 2013; Battaglia et al 2006).

> Two additional questions asking about insulin manipulation were included on the EDI ("I skip insulin shots to lose weight" and "I take less insulin than I am supposed to, to lose weight"). Participants responded to questions on a six-point Likert scale; response options ranged from "always" to "never". (Meltzer et al., 2001, p.679)

Although the measures have been modified or added to, only two of the papers reviewed actually provided prevalence rates (cut-offs or otherwise) using exclusively either the EDI or an EDI composites (as can be seen in table 2.1). Zuijdwijk et al. (2014) found that 23.2% of the female only sample was at risk and Sevenson et al. (2003) found that males with Type1 Diabetes have a higher drive for thinness than their non diabetic peers although only 1.4% scored above the cut-off point for the drive for thinness subscale.

### 2.4.7.3   Eating Attitudes Test

The EAT was originally developed in 1979 to detect AN and sub AN behaviour (Garner & Garfinkel. 1979). The EAT is one of the oldest and most widely used self-report screening tests. It was initially comprised of 40 items on a 6 point Likert scale from 'always' to 'never' and was later reduced by Garner et al. in 1982 to 26 items. It is advised to be used as a risk assessment tool rather than a diagnostic instrument with those scoring more than 20 advised to see a qualified ED specialist (Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990; Dotti & Lazzari, 1998). The questions in the EAT-26 also form 3 subscales, dieting, bulimia and food preoccupation and oral control. Items include *Am terrified about being overweight*, *Find myself preoccupied with food* and *Avoid eating when I am hungry*. A cut-off point of 20 is recommended for further clinical investigation. A 12 item version was also constructed in Norway which utilised a total score of 10 and a cut-off point of 9 as clinical concern (Wisting et al., 2013 a/b).

Proportion of those scoring above the cut-off point for the EAT varies by study, one Tawainese (Alice Hsu et al., 2009) study reported a prevalence of 23.8% females and 17.2% males while a study from Turkey suggested that 68.9% of the mixed sample screened above (Pinar et al., 2005). In a sample including predominantly male participants only 3.1% of the sample screened above the cut-off point (Garcia – Reyna et al., 2003). In a mixed sample from Brazil it was found that 45% of the sample screened above the cut-off point (Phillipi et al., 2013). It should be noted that none of the research reviewed modified the EAT to take Diabetes specific effects into consideration.

### 2.4.7.4  Sick, Control, One, Fat, Food (SCOFF)

The SCOFF was designed to give non ED specialists a brief measure of whether a patient is at risk using the main tenets of AN and BN and 5 yes/ no questions. *Do you make yourself Sick because you feel uncomfortably full? Do you worry that you have lost Control over how much you eat? Have you recently lost more than One stone (14 lb) in a 3-month period? Do you believe yourself to be Fat when others say you are too thin? Would you say that Food dominates your life?*  If a participant answers yes to two or more questions then the authors argue that they are likely either AN or BN (Morgan, Reid  & Lacey, 1999)

4 papers reviewed included the SCOFF or a modified version of the SCOFF as a measure with 3 coming from the same first author. These studies found a high level of participants scoring above the cut-off point with around 30% of females and 10% of males answering in the affirmative to two or more questions.  Several researchers offered specific modifications for the scales that would make them less biased to Diabetes specific regimen and psychological effects for example a modified SCOFF (MSCOFF) was proposed whereby the last question was removed and replaced by a question addressing insulin *Do you ever take less insulin than you should?* (Zuijdwijk et al 2014) or *How often did you inject too little or no insulin after carbohydrate intake during the last week?* with the response categories 'never', 'once or twice a week', '3 to 5 times a week', '(almost) every day', or 'more than once a day' (Baechle et al., 2014).

### 2.4.7.5 Bulimia Inventory Test Edinburgh (BITE)

The BITE is a self-report form based on the DSM 3 criteria for Bulimia and is designed to show risk of BN and sub BN using yes/ no questions and a 7 point Likert scale, with a threshold cut-off for further clinical investigation. The scale is divided into two subscales, 'symptoms' which contains 27 items and 'severity' which contains 6 items. Items include *Do you ever fast for a whole day? (symptom)* and *If yes, how often is this? (severity)* as well as items such as *Do you feel a failure if you break your diet once?* and *Do you count the calories of everything you eat, even when not on a diet?* The total score is then computed with scoring over 15 indicating a probable Eating Disorders. Three questions are also used to compute the severity (6, 7 and 27) if the score of those three items is more than 5 then that is also indicative of an Eating Disorder (Henderson & Freeman 1987).

Of the 4 papers using the BITE to investigate rates of EDs in the T1D population none were modified to take T1D into account and only 2 were used in a way that provided cut-off points. Alice Hsu et al. (2008) found that 4.8% f / 3.4% m scored above the cut-off point in a youth sample and Phillipe et al. (2013) found that 16% scored above the cut-off point in a larger scale sample using a mixed age sample.

### 2.4.7.6 The Three Factor Eating Questionnaire

The TFEQ (Stunkard & Messick, 1985; Karlsson, Persson, Sjostrom & Sullivan, 2000) is an 18 item scale devised of 3 subscales cognitive restraint (CR), uncontrolled eating (UE), and emotional eating (EE) using a 4 point Likert scale including responses such as 'Definitely True' to 'Definitely False' for 17 questions and a $1 - 8$ rating for question 18. Questions include *When I smell a delicious food, I find it very difficult to keep from eating, even if I have just finished a meal, I consciously hold back at meals in order not to weight gain* and *When I feel anxious, I find myself eating.* This scale is not generally used to make clinical assessments, rather to look at relationships between food related behaviour and as such no cut-off points have been suggested. In the three papers that reported using the TFEQ no cut-off information was provided and no amendments to account for T1D were reported.

### 2.4.7.7  The Diagnostic Survey for Eating Disorders

The purpose of the DSED is to ascertain whether participants are engaging in behaviours that coincide with the DSM diagnostic criteria for Eating Disorders. As such it asks questions regarding the frequency of behaviours.

The DSED has been modified for use in the diabetic population by including questions on insulin omission (Rodin et al., 1991) which was later expanded on by Rydal et al. (1997) as they state:

> Three mutually exclusive, hierarchical categories were used. Highly disordered eating was defined as the occurrence of one or more of the following forms of disordered behavior at least twice per week during the preceding three months: binge eating, omission or underdosing of insulin to promote weight loss, self induced vomiting, or use of laxatives. Moderately disordered eating was defined as the occurrence of one or more of these forms of disordered behavior at least twice per month, but less than twice per week, during the preceding three months. Non - disordered eating was defined as the absence of disordered behavior or its occurrence less than twice per month during the preceding three months (Rydall et al., 1997, p. 1850)

This protocol has been used by another two researchers (Maharaj et al., 2003; Pollock-BarZiv et al., 2005) but neither researcher reported cut-off points using only this measure, rather they used a composite of this and other scales to determine Eating Disorder behaviour.

### 2.4.7.8  The Power of Food Scale

The PFS was designed to assess desire for highly palatable foods and the 'psychological impact of living in food-abundant environments' using three different proximity subscales food available, food present, and food tasted. It rates answers on a 5 point Likert scale from 'don't agree at all' to 'strongly agree' over 15 items. Items include *It's scary to think of the power that food has over me* and *It seems like I have food on my mind a lot* (Cappelleri et al., 2009) This scale is not generally used to clinically suggest Eating Disorders but rather to explore relationships with other factors such as BMI in obesity research (Lowe et al., 2009) and cut-off points are not recommended by the authors. Only one paper used the power of

food scale, unmodified, and found that higher scores were implicated in those who were overweight (Markowitz et al., 2009).

### 2.4.7.9  The Eating Disorder Screen for Primary Care

The ESPC was constructed to allow for quick screening of patients presenting in primary care. The screener is 5 questions long and are answered yes or no. These questions represent 'abnormal' or 'normal' and questions include *Does your weight affect the way you feel about yourself?* Any abnormal answers are considered indicative of Eating Disorder risk (Cotton, Ball & Robinson, 2003). Only one paper utilised this scale, unmodified and found a prevalence of 20.7% of Eating Disorder in the mixed gender sample.

### 2.4.7.10 The Eating Disturbance Scale

The EDS is a brief 5 item scale based on a likert scale ranging from 1 – 7 with response options that are varied to the question being asked and investigates behaviour over the preceding 30 days. Items include *Are you satisfied with your eating habits?* and *Have you felt guilty about eating?* The authors suggest that scores of 16 and above are indicative of an Eating Disorder (Rosenvinge et al., 2001). Only one study utilised this scale which was not edited to account for Diabetes and did not report those screening above the cut-off point.

### 2.4.7.11 The Binge Eating Scale

The BES was initially devised for use amongst obese patients to detect abnormal eating patterns by Gormally, Black, Daston & Rardin in 1982. The scale comprises 16 items with differing responses that are then scored to demonstrate higher levels of binge eating. The answers are given on a three or four point Likert scale which asks participants to describe which is most like them for example question 1 asks them to select between *I don't feel self-conscious about my weight or body size when I'm with others, I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself, I do get self-conscious about my appearance and weight which makes me feel disappointed in myself* and *I feel very self-conscious about my weight and frequently, I feel intense shame and disgust for myself. I try to avoid social contacts because of my self-consciousness.* The items are then

scored, and the following categorisations given, Non-binging; less than 17, Moderate binging; 18-26 or Severe binging; 27 and greater. Only one paper reported rates of binge eating as being clinically concerning in 16% of mixed gender T1 patients and the scale was not adapted for use among those with T1D (Phillipi et al., 2013).

### 2.4.7.12 The Night Eating Questionnaire

The NEQ evaluates whether Night Eating Syndrome is likely by asking 14 questions using response scales on a 5 point Likert scale with responses varying per question. Questions include *How much control do you have over your eating between supper and bedtime?* And *When you snack in the middle of the night, how aware are you of your eating?* Scores are added and there are 2 cut-off points, Scores above 25 indicate that Night Eating Syndrome is likely and scores above 30 indicate that NES is highly likely. (Allison et al., 2008). Only one sample used the NEQ in order to compare night eating in those with diet related health conditions to healthy controls of which no significant differences were found. The questionnaire was not modified for Diabetes (Quick et al., 2012).

### 2.4.7.13 Questionnaire of Weight and Eating Patterns

The QWEP was initially construed to be able to diagnose Binge Eating by investigating various components needed for a DSM IV BED or BN diagnosis. It utilises 26 questions using a variety of responses ranging from yes/ no to 5 point Likert scales including *During the past six months, did you often eat within any two-hour period what most people would regard as an unusually large? amount of food?* And *During the past six months, on average, how often did you have times when you ate this way-that is, large amounts of food with the feeling that your eating was out of control?* 5 of the questions relate to a diagnosis of BED and 8 of the questions relate to BN and the scoring relates to DSM IV criteria. (Spitzer et al., 1992) In the one paper reporting using the QWEP binge eating was found in 26% of the male respondents and 0% of the female participants. The questionnaire was not modified for Diabetes. It should be noted that parts of the QWEP have been used in larger studies utilising a number of scales such as the AHEAD study (Neumark – Stzainer et al., 2002).

### 2.4.8  Scales Used – Diabetes Specific

#### 2.4.8.1  *Diabetes Eating Survey Revised*

In order to overcome some of the issues with diagnosing EDs in the T1D population a new survey was proposed. The first version of the Diabetes Eating Problem Survey (DEPS) included 28 self-report questions on a Likert scale. Published in 2001 (Antisdel, Laffel & Anderson, 2001), it was one of the first scales to recognise that generic screening instruments for Eating Disorders may over diagnose Bulimia based on Diabetes factors while simultaneously avoiding the important role of insulin omission. The scale was shortened to 16 items and renamed the Diabetes Eating Problem Survey Revised (DEPS-R) by Markowitz et al. in 2009. Examples of questions are *Losing weight is an important goal to me, I feel that it's difficult to lose weight and control my Diabetes at the same time. I alternate between eating very little and eating huge amounts* and *I would rather be thin than to have good control of my Diabetes.* The scale utilises responses on a 6-point Likert scale from 'never' to 'always' and scores over 20 are deemed to be clinically concerning.

The DEPS-R was a popular choice for screening instruments with 9 researchers utilising it this having been said only 5 of the studies published cut-off points. In one large scale study using registry data from children and adolescents 27.8% of females and 8.6% of males screened above the cut-off point (Wisting et al/. 2013 a/b). In a sample using a younger age group $(8-13)$ 15% screened above the clinical cut-off point. In studies using adults, Merwin et al. (204) found 22% of respondents screened above the cut-off point and Doyle et al. (2017) found a greater prevalence in women (29.6%) than in men (18.2%). The questionnaire also asks specific questions about insulin omission. Merwin et al (2014) suggest that:

> Five items on the DEPS-R assess manipulation of the diabetic treatment regimen for weight control purposes or related attitudes….including: *"I try to keep my blood sugar high so that I will lose weight"* (Keep BG High: DEPS-R9), *"I try to eat to the point of spilling ketones in my urine"* (Spill Ketones: DEPS-R10), and *"I feel fat when I take all of my insulin"* (Feel Fat: DEPS-R11). The remaining two DEPS-R items that assess manipulation of the diabetic treatment regimen for weight control purposes include: *"After I overeat, I don't take enough insulin to cover the food"* and *"After I overeat, I skip my next insulin dose."* (Merwin et al., 2014, p. 125)

However, researchers do not generally separate out different types of ED behaviour using the DEPS-R.

### 2.4.8.2  *Assessing Health and Eating among Adolescents with Diabetes (AHEAD)*

The AHEAD study was undertaken by the Children's Diabetes Clinic at St Paul Hospital in Minnesota. Part of the AHEAD study asked questions similarly to that in Project Eat; a large scale study aimed at assessing adolescent Eating and Health practices. It was modified for Diabetes by including two additional questions that indicate 'extreme weight control behaviours' *1) Skipped insulin doses,* and *2) Took less insulin that prescribed.* It also measures behaviours like current dieting status, and frequency of meals (Akard et al., 2008) The response scales and cut-off points vary depending on the question being asked[7]. 2 studies used the AHEAD protocol to assess levels of Eating Disturbance in T1Ds. The AHEAD questionnaire divides symptomology into subcategories of weight control in one study there were distinct gender differences seen with nearly half of the males displaying no weight control whatsoever compared to just 7.6% of females.

### 2.4.9  Interviews Used

### 2.4.9.1  *The Eating Disorder Examination (Fairburn, Cooper & O'Connor 2014).*

The EDE, currently on its 17th updated version is a semi structured interview with emphasis placed on the fact that it is an investigator rather than respondent led interview that encourages some fluidity around the core questions in order to get a full picture of the respondents eating attitudes and behaviour. It can be used for current symptomology by

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[7] This was an extremely large study that used a huge variety of scales. The whole list can be found at the University of Minnesota website:

(http://docs.sph.umn.edu/epich/eat/EAT2010_FEAT_Psychometrics.pdf)

focusing on a previous 28 day period or for diagnostic purposes by extending this to 3 months. Interviewers need specific training to administer the interview, as the author states:

> With investigator-based interviews interviewers need training to ensure that they fully understand the concepts being assessed. The structure in such interviews lies in the detailed specifications provided for the interviewer of the concepts to be rated and the rating scheme, rather than in the precise wording of individual questions. In summary, investigator-based interviews such as the EDE require that interviewers be trained both in the technique of interviewing and in the concepts and rules governing the ratings. (Fairburn et al., 2014, p. 3)

The EDE measures the frequency of specific ED behaviours such as binging and purging and also more general information based on 4 subscales, restraint, eating concern, shape concern and weight concern. Most updates have reflected changes to ED diagnosis and are in line with changes in the DSM, for example the most recent version removed amenorrhea from the diagnostic portion of the interview as it was also omitted from the DSM V's criteria for AN (Fairburn et al., 2014).

Researchers have modified the EDE in order to account for Diabetes specific aspects of ED behaviour as Grylli et al note:

> The EDE version that we used takes into consideration the potential presence of insulin omission/misuse as a compensatory measure and contains an appropriate semi-structured module. (Grylli et al., 2004, p. 231)

There is no further information on how this interview has been modified or what questions are asked regarding insulin omission. Similarly, Colton et al. (2013, 2015) describes insulin omission in terms of a 'Disturbed Eating Behaviour' but don't explain how the EDE was modified in order to assess prevalence rates This having been said this was part of a longitudinal study so this may reflect evolving attitudes on nomenclature. Peveler et al. (2005) also describe insulin omission prevalence rates but do not report modifications.

Several researchers make no mention of any modifications to the interview to account for Diabetes specific factors (Garcia – Reyna et al., 2003; Grylli et al., 2010).

### 2.4.9.2 *The Child Eating Disorder Examination (chEDE)*

The ChEDE (Bryant- Waugh, Cooper, Taylor & Lask, 1996) applies the same principles as the EDE but uses modified language in order to deal with a younger demographic. They also use a sort task to assess ideas about weight and shape where by the investigator makes lists of *things that are important to you in how you see yourself or think about yourself* and *things that are important to you when you think about how good you are as a person*. Further modifications look at intent rather than action given that children's lives are more likely to be structured by their parents.

While it appears apparent that insulin omission or other Diabetes specific aspects have been taken into account, some researchers do not explain how this was done (d'Emden et al., 2012; Wilson et al., 2014) or whether it was done as part of a modified chEDE or as an adjunct (Wisting et al., 2017). In 2008 Olmsted et al. describe a longitudinal study whereby questions about the use of insulin omission for weight control were added to the 38 standard questions in order to assess disturbed eating behaviour. Other questions were modified to ensure that behaviours described were in fact due to weight and shape concerns rather than due to T1D regimen and several other researchers utilise this protocol (Wisting et al., 2015; Colton et al., 2013).

Other researchers are equally as vague when it comes to describing how insulin omission is approached for example Colton et al (2004) state:

> Insulin dosage omission or reduction was diagnosed only if it was primarily a purging strategy to promote weight loss and not if it was occurring for other reasons. All ratings of disturbed eating behavior were based on the cEDE interview (Colton et al., 2004, p. 1656).

It should be noted that in mixed method research whereby insulin omission is reported by both self-report and then verified in an interview that reported rates are lower in interview representing a possible limitation of the interview (d'Emden et al., 2012).

### 2.4.9.3 *Rating for Anorexia and Bulimia (RABT)*

The authors of the RABT state that it was designed in order to account for the fact that most interviews used in Eating Disorders were designed for English Speakers and thus had little diagnostic use outside English speaking environments (Clinton & Norring, 1999). It consists of 56 items addressing 4 subscales; *Body- shape and Weight Preoccupation, Binge- Eating, Anorexic Eating Behaviour* and *Compensatory Behaviour*. Only one paper used this interview and it found a 0% of Eating Disorders and 0% insulin omission and did not explain how or if Diabetes specific aspects of ED were addressed (Svenson et al, 2009).

### 2.4.10 Mixed Methods Research

In research utilising both self-report and a clinical interview lower levels of insulin omission are reported via interview compared to the self-reports in the same sample (d'Emden et al., 2012) In research where not only those who screen as clinically concerning are then clinically interviewed there are substantial levels of Eating Disorders seen in both male and female participants versus controls (Garcia- Reyna et al., 2003). It may be that a mixed method approach whereby questionnaires are used for screening purposes and all are followed up with an interview would give a clearer clinical picture.

### 2.4.11 Reported Data

*Figure 2.9: Number of Papers which used Adapted Measures*
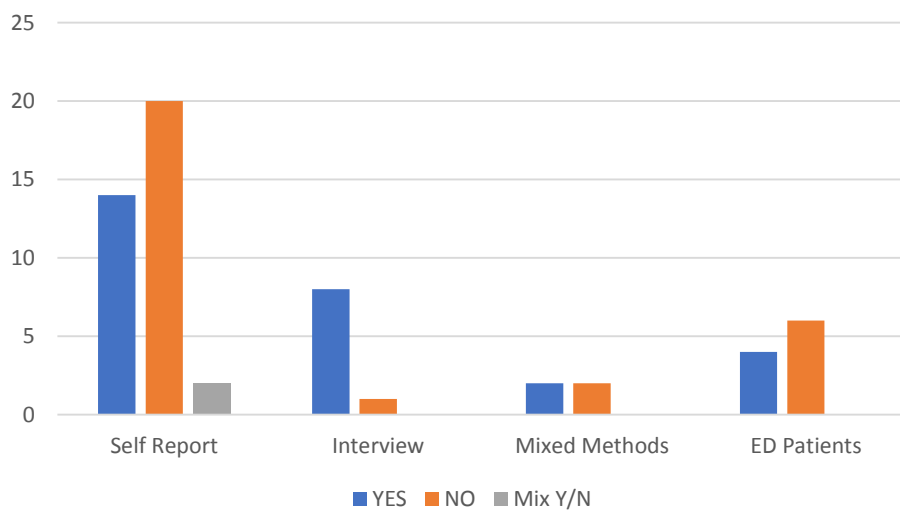
*Figure 2.10: Number of Papers Reporting Insulin Omission*
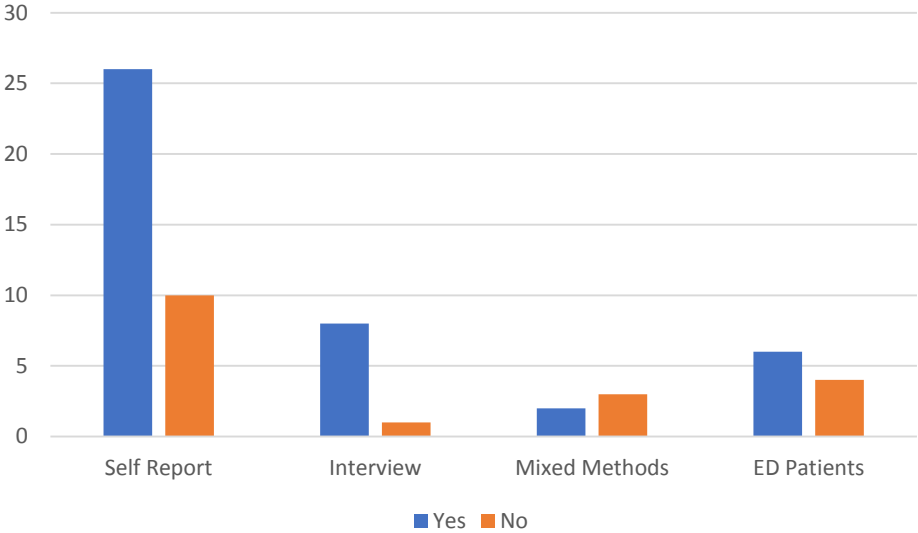
*Figure 2.11: Percentage of Participants Scoring above the cut-off point in Self-Report Research*
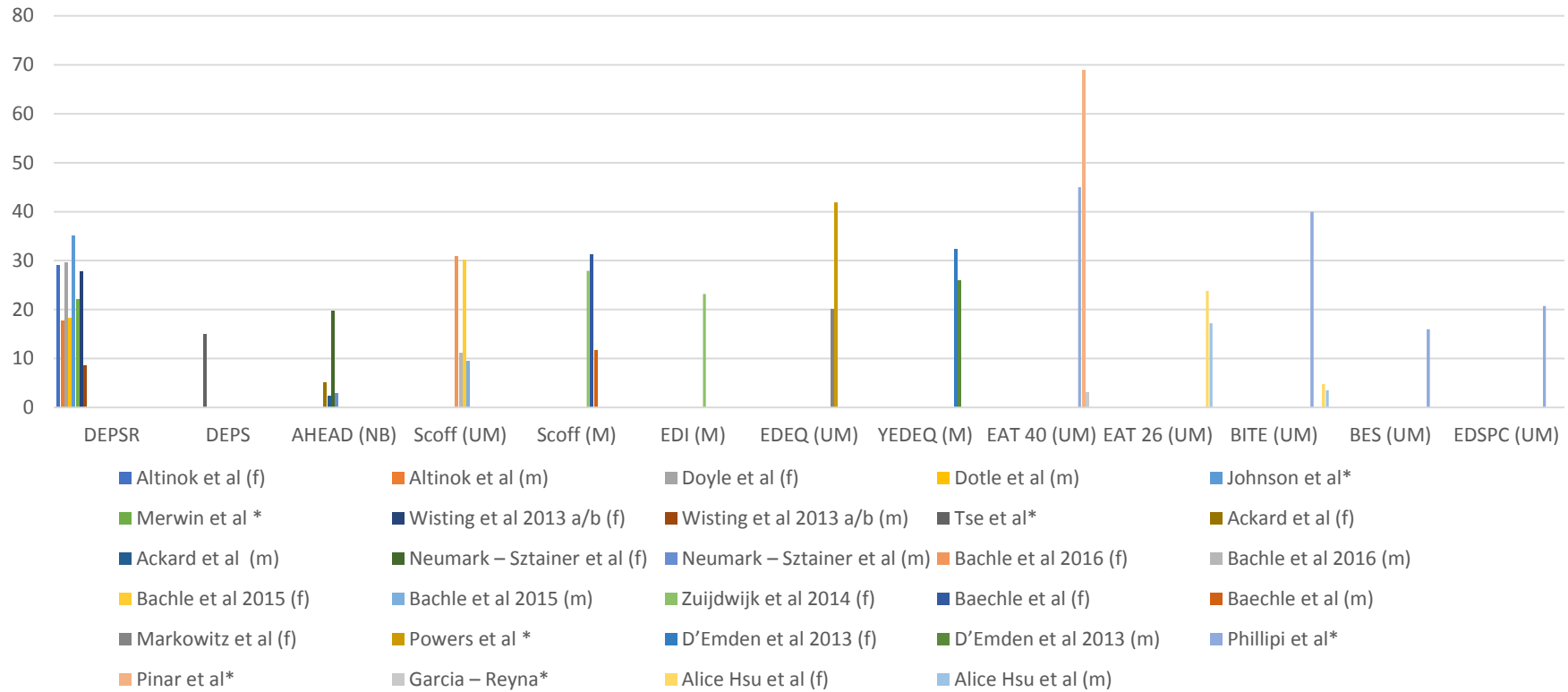
*= Gender split not reported, UM = unmodified

*Figure 2.11: Percentage of Participants meeting Clinical ED Criteria in Interview Based Research*
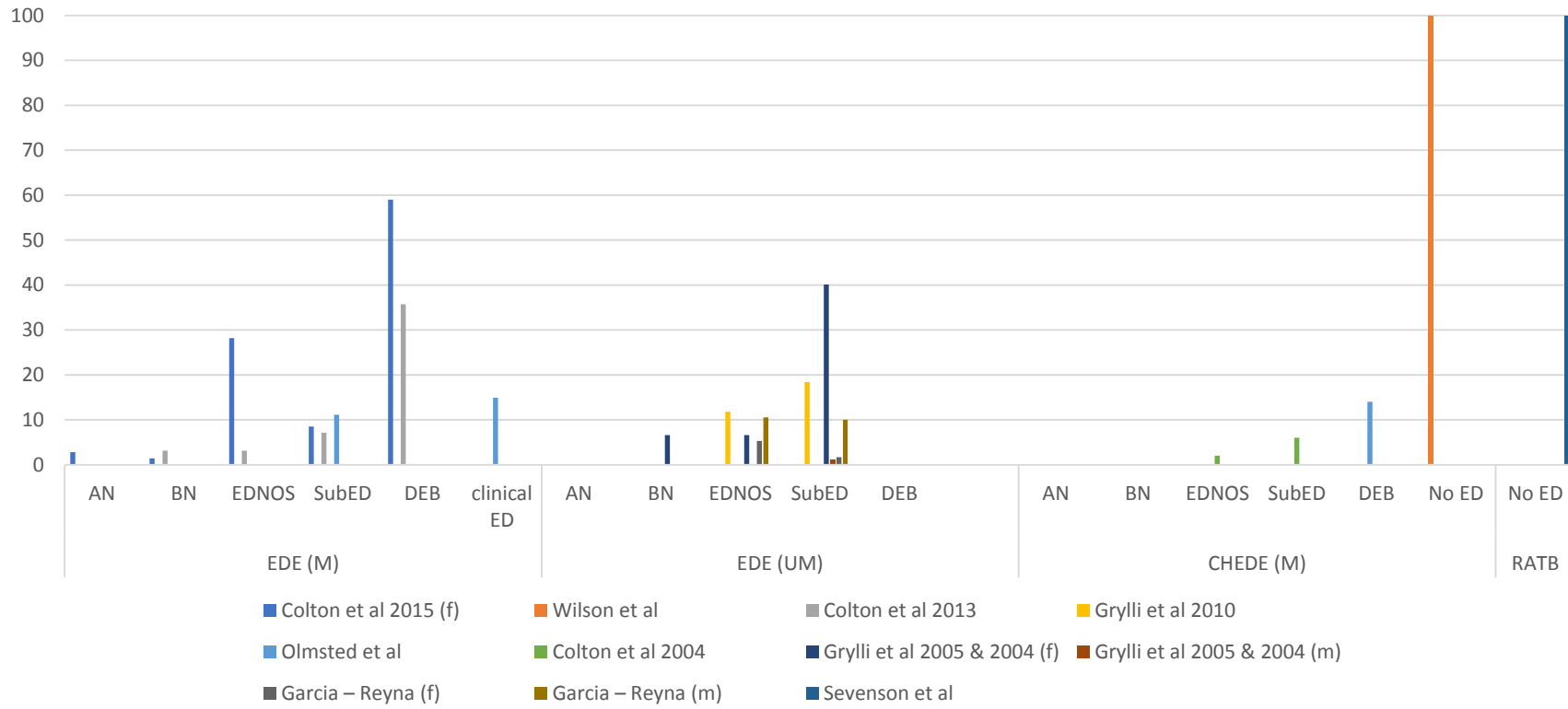
*= Gender split not reported, sub = subthreshold, AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified, DEB = Disordered Eating Behaviour, No ED = No Eating Disorder Present, EDE/ ChEDE = Eating Disorder Examination/ The Child Eating Disorder Examination, RATB = Rating for Anorexia and Bulimia

### 2.4.12 Diagnoses & Insulin Omission

There are conflicting prevalence rates of EDs in the T1 population by clinical diagnosis. It is important to note that regardless of insulin omission being mentioned it is not clear in any of the papers how this behaviour has been categorised in terms of diagnosis. This is perhaps not surprising given the vague terminology of the DSM IV and DSM V (please see section 2.1) which gives little instruction on aspects such as degree or frequency of omission.

Several authors (Grylli et al., 2004, 2005, 2010; Garcia-Rayna et al., 2003; Colton et al., 2004, 2015) reported utilising an operationalised definition of EDNOS and subthreshold ED defined by Jones et al. in a 2000 research article, the authors state;

> Based on DSM-IV criteria a minimum of four clinical symptoms over the past three months were necessary for the diagnosis of anorexia or bulimia nervosa. DSM-IV provides broad suggestions for diagnosis of Eating Disorders not otherwise specified. We operationalised the diagnosis of these disorders more precisely based on DSM-IV criteria and criteria used in previous controlled prevalence studies…Subthreshold Eating Disorders were considered to be milder eating disturbances with a lower frequency or severity of symptoms over the past three months than those specified in DSM-IV. (Jones et al., 200, p.1564)

Colton et al. (2004) later reproduced the definitions in greater detail

> Eating Disorder not otherwise specified
>
> 1. All the criteria for anorexia nervosa except for amenorrhea; or
>
> 2. All the criteria for anorexia nervosa except the subject does not report a fear of weight gain or does not report a disturbance in the way in which their body weight and/or shape is experienced; or
>
> 3. All the criteria for bulimia nervosa except that the subject does not report self-evaluation being unduly influenced by shape and/or weight; or
>
> 4. All the criteria for bulimia nervosa except that the frequency of binge eating and purging behavior occurred at least once per week for 3 months, or two times per week over the previous 4 weeks; or
>
> 5. An individual regularly engages in inappropriate compensatory behavior in the absence of binge eating (e.g., recurrent self-induced vomiting or insulin omission for shape and weight control at least one time per week for the past 3 months, or twice weekly over the previous 4 weeks); or

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6. An individual engages in recurrent episodes of objective binge eating (at least one time per week for the past 3 months, or twice weekly over the previous 4 weeks).

Subthreshold Eating Disorders

1. An individual engages in occasional (three or more times) binge eating, and/or purging over the past 3 months; or

2. An individual whose self-evaluation is unduly influenced by shape or weight, and who regularly engages in extreme dietary restraint (<500 kcal/day); or

3. An individual whose self-evaluation is unduly influenced by shape or weight, and who regularly engages in intense, excessive exercise for the purpose of weight control (at least five times weekly) over the past 3 months (Colton et al., 2004, p. 1656)

Though these definitions have been commonly used that does not mean that they are appropriate, for example the original authors found that although insulin omission for weight loss was present in a higher number of patients with a diagnosed ED and subthreshold disorders they also found it in those who they deemed to have no ED (Jones et al., 2000). It is also worrying that those who are withholding insulin but are diagnosed with subthreshold ED or no ED are deemed to be less clinically concerning given that DKA is a momentary event. It is, to a certain extent, irrelevant if it is a chronic behaviour. DKA is a life-threatening condition that these participants are at obvious risk of. Further issues are apparent aside from insulin omission, both Grylli et al. (2004, 2005) and Colton et al. (2004) highlight that intense exercise may be a prescribed behaviour and that it is beneficial to T1D management but may be described as pathological in such a paradigm. They also highlighting that other typical bulimic behaviours are much less common (Grylli et al., 2004, 2005, Colton et al., 2004).

Only one self-report gave diagnostic estimates. It utilised the EDE-Q (Smith et al., 2008) finding that 15% of the female only sample had probable BN and 12.5% had probable BED. In interview-based research in youth samples no participants were diagnosed with AN but in one sample using adults a 2.8% prevalence was found (Colton et al., 2015). BN and EDNOS was more commonly found in youth samples and subthreshold Eating Disorders were found in as many as 40% (Grylli et al., 2005, 2004) the EDE or chEDE was used to ascertain these diagnoses but it should be noted that as well as varying rates of diagnoses being found, one study also found a 0% rate of any clinical ED (Wilson et al., 2014). This research was

undertaken with T1D teenagers and their parents however potentially leading to under-reporting of behaviours. Similarly, in 1 study using an alternative interview the RABT, 0% clinical diagnoses were found, although this used a male youth sample only. In adult samples, only 1 article reported diagnoses rates (Colton et al., 2015) so it would be unwise to extrapolate from that. Only 11 papers reported clinical diagnoses. As can be seen in figure 2.12 the estimates of clinical diagnoses vacillate too wildly to make any general estimates of prevalence. Furthermore, it could be argued that given the substantial issues in measuring EDs in this population, which have never been appropriately addressed, one would be advised against making any conclusions. Also rates of insulin omission for weight loss, arguably the most dangerous behaviour those with T1ED can engage in, do not equate to rates of ED reported, for example Grylli et al. (2010) found only a 13.2% rate of clinical diagnoses (BN, EDNOS) but 26% of their sample reported insulin omission for weight control, Sevenson et al. (2003) found a 0% rate of clinical EDs but a 2.1% rate of insulin omission and Peveler et al. (2005) found only a 14.9% rate of clinical diagnosis but a 35.5% rate of insulin omission.

There are further issues when looking at how insulin omission has been measured as can be seen in table 2.7. There is no consensus as to what constitutes insulin omission in a clinically significant way. There is no mention as to how omission is encapsulated into ED diagnoses.

What also seem to be apparent is that in clinical samples of T1EDs who have an ED diagnosis insulin omission is a common feature regardless of diagnosis type (please see table 2.6) suggesting that this is either a transdiagnostic feature or that this is the key tenet of T1ED. It is also notable that despite reservations that AN is not commonly seen in T1D, it is observed by all reporting prevalence except for Takii et al. (2002, 2003) who were utilising those with BN only. Also, research using those with diagnosed EDs tends to focus on adult samples in a reversal of what is seen in other research into T1ED.

*Table 2.5: Diagnoses in non-Clinical Samples*

| Author/ Year/ Country of Origin | n (m/f) | Age Range or x̄ | Primary Method | DSM | Instruments | Diagnoses and Prevalence | Adaptation? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| Colton et al 2015 (Canada) | 71f | 19.2 – 27.8 | Longitudinal | DSM-IV-TR | EDE | AN 2.8% BN1.4% EDNOS 28.2% subED8.5% | Yes | Yes | 26.80% |
| Wilson et al 2014 (Ireland) | 30f/ 20m | 14 – 16 | Cross Sectional | n/r | ChEDE | no ED %100 | Yes | Yes | 0% |
| Colton et al 2013 (Canada) | 98f | 14 – 18 | Longitudinal | DSM IV | EDE | BN 3.1% EDNOS 3.1% subED 7.1% | Yes | Yes | n/r |
| Grylli et al 2010 (Austria) | 76f | x̄ = 17.2 (2.1) | Cross Sectional | DSM-IV-TR | EDE | EDNOS 11.8%, subED 18.4% | No | No | n/a |
| Smith et al 2008 (UK) | 40f | 11 – 19 | Comparison/ control | DSM - IV | EDEQ | BN 15%, BED 12.5% | No | No | n/a |
| Peveler et al 2005 (UK) | 87f | 11 – 38 | Longitudinal | DSM IV | EDE | Clinical ED, 14.9% subED, 11.1% | Yes | Yes | 35.60% |
| Colton et al 2004 (Canada) | 101f | 9 -14 | Control | DSM - IV | ChEDE | EDNOS 2, % subED 6% | Yes | Yes | 2% |
| Grylli et al 2005 & 2004(Austria) | 30f/ 5m | 14.1 (2.6) | Clinical Interview | DSM - IV | EDE | BN 6.6%f, EDNOS 6.6%f subED 40%f, 1%m | No | No | 26%f 0%m |
| Garcia – Reyna 2003 (Spain) | 13* | 12 – 16 | Clinical Interview | n/r | EDE | EDNOS 1.7%m, 5.3%f subED 10%m, 10.5%f | No | No | n/r |
| Svenson et al 2003 (Sweden) | 141m | 14 – 18 | Control | DSM - IV | RABT | Clinical ED 0% | Yes | Yes | 0.00% |

*Gender split not reported, n/r = not reported, n/a = not applicable, sub = subthreshold. sub = subthreshold AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified,

*Table 2.6: Research with Clinical ED Patients*

| Author/ Year/ Country of Origin | n (m/f) | Age Range or x̄ (sd) | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adapted? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| Colton et al 2015 (Canada) | 95f | 27.1 (9.1) | Retrospective Chart Review | Attendees of Day Hospital | EDE | BN 47.4% AN 14.7% EDOS37.9% | Yes | Yes | 78.80% |
| Dickens et al 2015 (US) | 29f | 25.55 (9.03) | Retrospective Chart Review | Inpatient | EDI | BN 79.3% AN 10.3% EDNOS 10.3% | No | No | n/a |
| Merwin et al 2015 (US) | 73/ 10 | 18 – 68 | Momentary | Patient registries, online advertisements, and flyers placed in nearby clinics | DEPS-R, EDE | n/a | No | No | n/a |
| Custal et al 2014 (Spain) | 20f | 25.3 (8.0) | Treatment Outcome | Outpatients/ Day patients | Semi Structured Interview, EDI | An & subAN 10.0% BN & subBN 25.0% BED10% EDNOS 55% | No | Yes | 90% |
| Powers et al 2013 (US) | 47/ 1 | 26.2 (10.3) | Scale Comparison | Initial Screening | EDE-Q, EDI | n/r | No | No | n/a |
| Powers et al 2012 (US) | 47/ 1 | 26.2 (10.3) | Exploratory retrospective case – control | Inpatient, partial, or outpatient | EDE-Q, EDI | EDNOS 58.3% BN 37.5% AN 4.2% | No | Yes | 47.90% |
| Takii et al 2011 (Japan) | 53f | 14 – 44 | Exploratory retrospective case – control | Outpatients | ED module of the Structured Clinical Interview for DSM 1V | BN 96.2% AN 3.8% | Yes | N/r | n/r |
| Takii et al 2008 (Japan) | 109f | 22.9 (5.2) | Exploratory retrospective case – control | Outpatients | ED module of the Structured Clinical Interview for DSM 1V | BN 64.2% BED 25.7% AN 6.4% EDNOS 3.7% | Yes | Yes | 67.90% |
| Takii et al 2003 (Japan) | 19f | 23.8(5) | Follow up | Inpatient | Clinical Interview  EDI | BN 100% | No | Yes | 77.80% |
| Takii et al 2002 (Japan) | 53f | 14 -36 | Cross Sectional | Outpatients | Clinical Interview | BN 100% | Yes | Yes | 83% |

| Author/ Year/ Country of Origin | n (m/f) | Age Range or x (sd) | Primary Method | Sample | Instruments | Diagnoses and Prevalence | Adapted? | Insulin Omission Measured? | Insulin Omission (n) |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | EDI | | | | |

n/r = not reported, n/a = not applicable, sub = subthreshold AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified DEPS-R = Diabetes Eating Problem Revised, YEDEQ/ EDEQ = The Eating Disorders Examination and Youth Eating Disorders Examination Questionnaire, EDI = The Eating Disorder Inventory, EDE/ ChEDE = Eating Disorder Examination/ The Child Eating Disorder Examination

*Table 2.7: How was Insulin Omission Measured in Papers where Rates are Reported?*

| Author/ Year | How was insulin measured? | Clinical ED/ DEB | Insulin Omission |
|---|---|---|---|
| Altinok et al 2017 | Questions on the DEPS-R | > DEPS-R cut-off 29. 1%f, 17.8%m | 22%f, 25%m |
| Bachle et al 2016 | In this study, all patients answered questions on the frequency of IR and the number of carbohydrate exchange units consumed without insulin coverage during the previous week. Frequent IR defined as IR occurring more than five times a week was used as proxy for regular, and thus likely intentional, IR. | >SCOFF cut-off 30.9%f, 1.1% m | 6.9%f, 7.2%m more than 5 times per week |
| Colton et al 2015 | DEB was defined as reporting any of the following during the 28 days before the EDE interview: dieting; objective binge-eating episodes; self-induced vomiting; abuse of laxatives, diuretics, or diet pills; insulin omission or underdosing for weight control... | AN 2.8% <br><br> BN 1.4% <br><br> EDNOS 28.2% <br><br> subED <br><br> DEB 59% (all f) | 26.80% f |
| Baechle et al 2014 | we assessed insulin restriction in Diabetes patients by the additional question "How often did you inject too little or no insulin after carbohydrate intake during the last week?" with the response categories "never," "once or twice a week," "3 to 5 times a week," "(almost) every day," or "more than once a day." | > SCOFF cut-off 31.2%f, 11.7%m | 18.5%m, 20.5%f more than 3 times per week |
| Wilson et al 2014 | Not stated | clinical ED 0% | 0%* |

| Author/ Year | How was insulin measured? | Clinical ED/ DEB | Insulin Omission |
|---|---|---|---|
| Phillipi et al 2013 | The patients also answered socio-demographic questions and questions related to T1D, such as time elapsed since their Diabetes diagnosis, whether they use fastacting or long-acting insulin, whether they use the carbohydrate counting method, and whether they restrict or omit insulin in order to lose weight. | cut-off 45% EAT, 40% BITE, 16% BES, Cumulative 58.7% * | 14.8%f/ 0%m |
| D'Emden et al 2013 | For this study, the Youth EDE-Q and EDI-3 RC were adapted for Diabetes with additional questions pertaining to insulin misuse, which were endorsed by the authors of each tool. These included questions similar to Meltzer 'I take less insulin than I should to influence my shape or weight'; 'I skip insulin shots to influence my shape or weight', with an additional question about intention to manipulate insulin: 'I have thought about taking less insulin to modify my shape or weight.' The Insulin questions were analysed separately. | (YEDEQ) Disturbed Eating Behaviour 32.3%f, 25.9%m, | 7.6%f/ 3.4%m |
| Bernstien et al 2013 | Participants were also screened for intentional insulin omission or dose reduction with the question, "Do you take less insulin than you should?" | (EDS) Disordered Eating 20.7% * | 13%* |
| Wisting et al 2013 a/b | we operationally defined insulin restriction and insulin omission according to the following two DEPS-R items: "When I overeat, I do not take enough insulin to cover the food" and "After I overeat, I skip my next insulin dose." | > DEPS-R cut-off 27.8% f, 8.6%m | 36.8%f, 9.4% m |
| Markowitz et al 2013 | Based on the researcher assumption that the question 'When I overeat, I don't take enough insulin to cover the food' measures insulin omission | n/r | 14% |
| d'Emden et al 2012 | (YEDEQ) specific questions are also asked about the presence and frequency of disturbed eating behaviours, such as restraint, binge eating, self-induced vomiting, use of diuretics or laxatives and driven exercise for the purpose of weight control. Questions regarding insulin misuse for weight control were added by us with permission from the authors and rated similarly as a disturbed eating behaviour, but the original scoring of the tools remained intact. (EDI - 3RC) The EDI-3RC takes 5 min to complete. Questions regarding insulin misuse were added, with permission from the author; however, the integrity of the original scoring was maintained. | n/r | 3.4%m / 7.6% f |

| Author/ Year | How was insulin measured? | Clinical ED/ DEB | Insulin Omission |
|---|---|---|---|
| Goebel - Fabbri et al 2011 | We used responses to the screening statement, "I take less insulin than I should," to determine insulin restriction status in this cohort. Responses were on a 6-point Likert scale ranging from "never," "rarely," "sometimes," "often," "usually," to "always." We decided on the following definition of insulin restriction, because we believed that social desirability pressures could influence women to underreport insulin restriction as a symptom, particularly as part of research being done in a specialty Diabetes center... We used their responses at baseline to categorize women as insulin restrictors if they reported any form of restriction from, "rarely" to "always." They were categorized as appropriate insulin users if they endorsed "never." | n/r | 36% |
| Markowitz et al 2010 | To rate adherence to insulin therapy, clinicians indicated whether or not they thought the patient was "skipping shots, misusing insulin, or 'forgetting' to bolus on the pump." Such patients are categorized as those missing or restricting insulin. This rating of insulin restriction was based on clinician assessment and lab results, but did not specify that insulin restriction was for purposes of weight management. | n/r | 24%m, 29%f |
| Goebel- Fabbri et al 2008 | The screening item "I take less insulin than I should" in a self-administered survey developed for the original study was used to determine insulin restriction status in this patient cohort. Based on their responses at baseline, women were categorized as insulin restrictors if they reported restriction at any level of frequency from "rarely" to "always" in response to this statement. They were categorized as appropriate insulin users if they endorsed "never" on this same item. | n/r | 30%f |
| Ackard et al (2008) | Two additional 'extreme' weight control behaviors were included in the AHEAD survey only and were included as 'extreme'weight control behaviors: skipped insulin dose(s) and took less insulin than prescribed. | Any unhealthy weight control behaviour 4.3% m/ 20.7f,<br><br>Less extreme unhealthy weight control 3.7%m/ 16.1%f<br>Extreme unhealthy weight control 2.3%m/ 5.1% f | 2.8%m 17.7%f |
| Olmsted et al 2008, Baseline | The cEDE consists of 38 items including questions that were added regarding the use of insulin dosage manipulation or omission as a weight control strategy | (ChEDE) 15% DEB (all f) | 2.40% |

| Author/ Year | How was insulin measured? | Clinical ED/ DEB | Insulin Omission |
|---|---|---|---|
| Battaglia et al 2006 | Two additional questions ("I skip insulin shots to lose weight" and "I take less insulin than I am supposed to, to lose weight") were included to address insulin omission (Meltzer et al., 2001). | n/r | 15% |
| Pinar et al 2005 | The questions in the second part were for diabetic patients and were related to Diabetes and adherence to Diabetes regimen. These questions were on duration of Diabetes, level of HbA1c, adherence to recommended diet, strict diet restriction, and intentional insulin misuse to lose weight. | >EAT cut-off 68.9%* | 40% |
| Pollock - BarZiv et al 2005 | Subjects with Eating Disorder: symptoms included those in whom one or more of the following behaviors occurred at least twice a month during the preceding 3 months: binge eating, insulin omission or manipulation of an insulin dose to promote weight loss, self-induced vomiting, and laxative use. | (EDE - Q, DSMED) Combined > cut-off 27% (all f) | 27.5%f |
| Peveler et al 2005 | Not stated | Clinical ED, 14.9% Subclinical ED, 11.1% (all f) | 35.60% |
| Colton et al 2004 | Insulin dosage omission or reduction was diagnosed only if it was primarily a purging strategy to promote weight loss and not if it was occurring for other reasons | EDNOS % 2, SubED % 6 (all f) | 2% |
| Maharaj et al 2003 | Highly eating disturbed included… engagement in one or more disordered eating and/or weight loss behaviours, including binge eating, self-induced vomiting, laxative/diuretic use, insulin omission to promote weight loss, or complete food avoidance, at a frequency of at least 2–3r/month to >1r/day, over the preceding 3 months. Mildly eating disturbed included girls who reported one or both of the following: (1) engagement in one or more of the above disordered eating and/or weight loss behaviours at a frequency of 1r/month or less, over the preceding 3 months | Combined Highly Eating Disturbed = 20.5% Mildly Eating Disturbed = 34% Non-Eating Disturbed = 45.5% | 17% |
| Sevenson et al 2003 | The patients also answered anonymously a few questions concerning their Diabetes treatment and whether they had ever taken less insulin than they should in order to lose weight. | DFT (EDI) 1.4% (all m) | 2.1%m |
| Neumark – Sztainer et al 2002 | Items assessing weight perceptions and weight control behaviors were drawn from the Project EAT (Eating Among Teens) Survey (24). The Diabetes Eating Problems Survey (DEPS) (2) was used to further assess disordered eating attitudes and behaviors and manipulation of insulin or weight control purposes among the respondents. | No weight control = 46.4% m / 7.6% f Healthy weight control = 37.7% m/ 54.5% f Unhealthy weight control = 13% m / 18.2% f | 1.4%m/ 10%f |

| Author/ Year | How was insulin measured? | Clinical ED/ DEB | Insulin Omission |
|---|---|---|---|
| | | Very Unhealthy weight control = 2.9% m /19.7% f | |

n/r = not reported, n/a = not applicable,

DEPS-R = Diabetes Eating Problem Revised, SCOFF = Sick, Control, One, Fat, Food. YEDEQ/ EDEQ = The Eating Disorders Examination and Youth Eating Disorders Examination Questionnaire, EAT = Eating Attitudes Test, EDI = The Eating Disorder Inventory, EDS = The Eating Disturbance Scale, BITE = Bulimia Inventory Test Edinburgh, BES = The Binge Eating Scale, TFEQ = The Three Factor Eating Questionnaire, NEQ = The Night Eating Questionnaire, AHEAD = Assessing Health and Eating among Adolescents with Diabetes, QWEPR = Questionnaire of Weight and Eating Patterns, DSMED = The Diagnostic Survey for Eating Disorders, PFS = Power of Food Scale

sub = subthreshold AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified, DEB = Disordered Eating Behaviour

## 2.5 Discussion

### 2.5.1 Summary

This is the largest systematic review to date investigating how EDs are measured in the T1 population. This review shows that there are many inconsistencies between research papers in a number of important aspects and that there are serious methodological issues in the canon. The majority of research used a cross-sectional approach and came from the US followed by Canada. The UK seems to be somewhat behind with very few articles. Sample size and recruitment methodology were often flawed with most of the research utilising too small a sample from a small geographical area to adequately make population assumptions. There does appear to be a general consensus that females are more susceptible to T1EDs but the review also found a worrying trend with more males displaying higher levels of ED symptomology and insulin omission in more recent years. This is especially concerning as only one paper focussed exclusively on males. Researchers seemed to be predominantly interested in young adults and adolescents which is interesting as it is understood that EDs appear later in the Diabetic population or at least have a higher age threshold when looking at risk (Goebel-Fabbri et al., 2011). When looking at research with patients who have been diagnosed with EDs this trend appears with most of the papers focussing on adults. Among this group BN and EDNOS were the most frequent diagnoses and although it has been argued that AN is less frequently seen several papers reported diagnoses.

In self-report based research it was less likely that clinical diagnoses would be offered, rather a plethora of risk categories were reported such as 'probable ED' (Powers et al., 2016), 'Elevated Eating Disorder Behaviour' (Johnson et al., 2014), 'Very Unhealthy Weight Control' (Schwartz et al., 2002) and 'Mildly Eating Disturbed' (Maharaj et al., 2003). A large variety of instruments used to make these assumptions with the EDEQ, DEPS-R and the EDI being the most popular. In interview-based research the EDE or the chEDE were used nearly exclusively. While most researchers reported adapting their instruments to account for the effects of Diabetes there were still a substantial number who did not, furthermore while insulin omission was measured by the majority of researchers it was not necessarily reported. Rates of insulin omission varied widely but generally samples including adults reported

higher levels than those based on adolescents and children. In those utilising a clinical sample, much higher levels of insulin omission were seen with rates reported to be as high as 90% suggesting that for those diagnosed with an Eating Disorder, this is a key tenet of the illness.

## 2.5.2  Interview Research

Although clinical interview is thought of as the gold standard when diagnosing EDs there are sizable issues utilising this methodology in T1ED. The most probable issue of using clinical interviews with the T1D population is awareness of insulin omission as a weight loss tool. Even then, someone who is omitting for weight loss may not screen as being clinically concerning, indeed they may not score high enough on any of the EDE subscales *because* they are omitting insulin. In T1ED the food may be less fear inducing than administering insulin, or it may be the combination of food and insulin that elicits psychopathology (Goebel – Fabbri, 2017). The most recent version of the EDE (ver 17) does not mention insulin omission directly. Similarly, to its adult counterpart the chEDE interviewer would need an operational knowledge of how Eating Disorders present in those with T1D in order to make an accurate assessment. Regardless of this several researchers do not mention adapting the interview to account for insulin omission (Grylli et al., 2010; Grylli et al., 2005), which may account for a number of 'no ED diagnosis' cases. Other researchers do adapt the interviews but amalgamate insulin omission into 'Disordered Eating Behaviour' without reporting individual levels (Wisting et al., 2015; Colton et al., 2013).  It is important to note that using interviews may lead to a lower reporting of insulin omission than self-report, and this may be more pronounced in males. It should also be considered that this demographic are acclimatised to talking to HCPs about their health and potentially hiding aspects that they do not want to discuss (Svenson et al., 2003; d'Emden et al., 2012). For clinical purposes this may suggest that a self-report measure administered prior to an invitation to interview may be more appropriate for use as a tool, once insulin omission has been confirmed by questionnaire it can be discussed. It may be suggested that an entirely new diagnostic interview specific to T1ED should be developed that can separate the nuances of T1D from what is *true* Eating Disorder pathology and insulin omission for weight loss.

### 2.5.3   Regimen Effects

This is not however to say that self-reporting was free from issues. One of the main problems is that the questionnaires that are not Diabetes specific do not take into account how Diabetes regimen may bias responses. For example, a study undertaken using those with T1ED and those with ED suggested that around 50% of the questions in the EDEQ could be influenced by T1D (Powers et al., 2013). There are ambiguous items in the scale whereby 'abnormal' eating behaviour may be due to T1D and rather than an ED (Items 7,9 13, 14) which would inflate estimates of prevalence. However, one of the potential benefits of using the EDEQ lies in the wording. The specifier 'because of weight and shape' eradicates any potential for regimen related effects to be scored as Eating Disorder symptomology. For example, 'Have you tried to exclude from your diet any foods that you like' may elicit a false positive among those with T1D who use a low carbohydrate diet to control blood glucose. Similar issues arise when using the EDI. The most common composite used was EDI-3RC (Drive for Thinness, Bulimia Scale, and Body Dissatisfaction Scale). The use of composites rather than the full scale allows for a shorter questionnaire which may be advantageous especially in large scale studies using multiple instruments. In this composite however, there are items that may relate to Diabetes regimen (Items 1, 5 and 7 Drive for thinness). This having been said a study undertaken comparing those with T1ED and those with ED suggested that only around 7% of the questions in the EDI3 could be influenced by T1D (Powers et al., 2013). Issues are also apparent in the SCOFF questionnaire which only utilises five questions, one of which (question 2) may be sensitive to T1D regime effects. Regardless some authors have used this to measure with little mention of potential problems (Bachle et al., 2015). The EAT suffers from similar issues. A T1D may avoid eating when hungry depending on their BG reading, similarly they are advised by specialists to be aware of the nutritional content of their food and many follow a low carbohydrate diet which may include consuming 'diet foods' avoiding high carbohydrate/ sugar items. This is an issue when using any of the subscales, particularly dietary restraint (Battaglia et al., 2006). Research that does employ this instrument has found startlingly increased ED symptomology in T1EDs compared to those without T1, some suggesting that EDs may be four times as common (Pinar et al., 2005). Hunger may also be dysregulated in T1D, episodes of hypo and hyper glycemia markedly affect appetite, increasing it drastically, so asking questions regarding 'being out

of control' or 'eating lots of food at once' needs to take that into account which this scale, the BITE and the EDI do not. Even questions such as 'can you leave food' are inappropriate as that may be determined by what point the participant takes insulin; the carbohydrate counting approach to T1D management advises patients to inject for the amount they are going to consume at the start at the meal.

Given that all of the scales used are fundamental flawed it could be argued that none are appropriate, they pathologise behaviour that is normal in a T1D context while missing aspects (and often the most dangerous behaviour, insulin omission) specific to that demographic. Furthermore, prevalence estimates cannot be trusted using these instruments for the same reason.

### 2.5.4   Psychological Effects

Regimen effects are not the only T1 related issues when utilising standard questionnaires. Type 1 Diabetes carries a heavy psychologically burden often in relation to issues addressed in these scales such as feelings and thoughts around food. T1Ds often proclaim that food is a domineering influence in their lives, that they are preoccupied with it and give too much thought to it, while also displaying self-control in order to manage BGs. There is guilt around breaking a strict diet that may be for BG control rather than weight loss. Many consultants encourage their patients to keep food diaries with nutritional information. This may influence responses to questions on all of the scales. For example, the PFS asks the participants to rate the question 'It's scary to think of the power that food has over me' (de Cássia, Sparapani, Jacob & Nascimento, 2015; Rohan et al., 2013; Rossi et al., 2013; Goebel-Fabbri, 2017)

Furthermore, T1EDs which are defined behaviourally by insulin omission may circumvent the intentions of this screening instrument. Battaglia et al., (2006) for example removed the question "I eat sweets and carbohydrates without feeling nervous" from the EDI when it could be argued that those who are insulin restrictors happily consume carbohydrates and sweets, that is the exact mechanism via which they increase their blood glucose and thus lose weight. If they had added 'and then take insulin' or some variant thereof they may have a better understanding of how T1EDs operate. These issues further highlight the need to eradicate the use of non T1D specific instruments from research in this population.

### 2.5.5   Modifications & Questions on Insulin Omission.

Several researchers offered specific modifications for the scales that would make them less biased to Diabetes specific regimen and psychological effects, for example a modified SCOFF (MSCOFF) was proposed whereby the last question was removed and replaced by a question addressing insulin 'Do you ever take less insulin than you should?' (Zuijdwijk et al., 2014) or 'How often did you inject too little or no insulin after carbohydrate intake during the last week?' with the response categories 'never,' 'once or twice a week', '3 to 5 times a week', '(almost) every day', or 'more than once a day' (Baechle et al., 2014) however it may still generate a false positive by failing to inquire whether insulin omission is due to weight and shape concerns or other issues rather than other issues such as fear of hypoglycaemia (In this particular piece of research patients with a known ED history were excluded from the analysis which removed a potential reference category).  Research using this questionnaire has claimed that disturbed eating behaviour is not more common in youth with T1D (Baechle et al., 2014).

As the EDE and the YEDE has a subscale relating to disturbed eating behaviours it may be ideally placed to add in questions surrounding insulin omission as a DEB. Indeed, researchers have done this and kept the original scoring intact, concluding that insulin omission for weight and shape concerns may be over reported due to the omission of this specifier (d'Emen et al., 2012).  Goebel-Fabbri et al (2008) asked participants to rate the statement 'I take less insulin than I should' with a Likert response that ranged from 'never' to 'always' in a longitudinal study which aimed to ascertain the associated complications and mortality rate of insulin restriction. They demonstrate that those who have higher levels of insulin restriction also score higher on a number of ED measures but also on psychological issues regarding T1D that are not necessarily related to ED for example 'fear of hypoglycaemia'. Snyder (2016) surveyed adolescents with T1D in order to find out how many young people were misusing insulin and found that out of 11 participants who admitted to intentionally under or over dosing on insulin only 2 did so out of a desire for weight control. However Goebel-Fabbri (2011) justifies the use of such questioning by pointing out that directly asking whether insulin omission is due to weight and shape concerns may elicit a false answer from participants who feel pressure to be compliant and it should be added that Snyder asked his

questions to adolescents with their parents in a room next door which may have biased results.

When questions on insulin omission are added they may be so as an addendum and not part of any particular subscale or overall eating pathology (Battaglia et al., 2006). Some researchers have attempted to circumvent these issues by creating their own questionnaires with questions such as 'I am afraid that getting my blood sugars in good control will cause me to gain weight' and 'Taking insulin makes me gain weight' (Goebel – Fabbri 2011). However, there is not enough consensus on how and when modifications should be made nor is there enough research to contrast unmodified and modified versions of instruments to recommend which scales or specific questions are suitable to be used. As a result, it maybe concluded that indeed none are suitable and a T1D specific instrument is the only acceptable option or alternatively, something new must be conceived.

### 2.5.6 Diabetes Specific Instruments

There appears to be more consensus on levels of disturbed eating behaviour when using T1D specific instruments. The DEPS-R in female population consistently reports symptomology of around 30 % although rates in males are less consistent. However, the DEPS-R does not provide diagnoses estimates and instead opts for a cut-off point for clinical concern.. The studies using the AHEAD protocol found much lower rates of Eating Problems but that may be due to the researcher recruiting much younger participants. It could be argued then that currently the DESPR produces the most consistent results and thus should be recommended for use (please see chapter 9 for a larger discussion on the use of the DEPS-R).

### 2.5.7 Diagnoses

Perhaps due to changing attitudes regarding insulin omission, Eating Disorders in T1D are described differently. Several authors use the moniker *Disordered Eating Behaviour (DEB)* and a scale of severity to describe unhealthy weight control practices in this group. Olmsted et al define DEB as 'dieting for weight control binge eating, self -induced vomiting, or the use of diuretics, laxatives, insulin omission, or intense exercise for weight control' (2008, p. 1978).  Nomenclature tends to depend on the instrument used however. Many researchers

are quick to highlight that insulin misuse is the differential factor for Eating Disorder between those who do and do not have T1D. What is potentially worrying is that even when insulin omission is present and is the primary symptom, diagnoses vary. Thus, two patients displaying exactly the same pathology and behaviour may vary on diagnosis by one factor such as BMI (Custal et al., 2014). BMI is particularly problematic for use in this population as T1 carries a weight penalty of around 15% (DCCT, 1995).

A longitudinal study by Olmsted et al found that *Disordered Eating Behaviour* may predict later insulin omission (2008). They highlight this while stating that despite nearly half of their study participants developing DEB none of them met the criteria for a clinical diagnosis. Similarly, a large prevalence of DEB in T1Ds may be found and yet no increase in prevalence of EDs reported (Iafusco et al., 2004).

The use of the diagnostic label Eating Disorder Not Otherwise Specified is often used for T1s who 'only' omit insulin but do not display any other Eating Disorder symptoms (Colton et al., 2013). This is problematic as research has shown that T1EDs who engage in 'Diabulimia' do not see themselves as having EDNOS (Allan, 2014), furthermore there are treatment implications for this diagnosis. It is often seen as a less serious variant of Eating Disorders or not a 'full' Eating Disorder (Colton et al., 2004) and while this may hold true for the general population, given the dangers associated with insulin omission it may be inappropriate and potentially hazardous for this demographic. Similarly diagnoses of 'subclinical Eating Disorders' are also common (Grylli et al., 2010; Colton et al., 2013; Colton et al., 2015) and perhaps more problematic. Given that insulin omission events are often equated to excessive exercise or purging and are thus rated on frequency it could be argued that a subclinical diagnosis is inappropriate. DKA is frequently fatal, one episode of omission could lead this biological state, so even if this occurs only once a month (extremely low frequency) the risk is much higher and therefore must be dealt with as a matter of urgency, something that may not occur with the nomenclature 'subclinical'. This can also be seen in questionnaires that ask about frequency of insulin omission without explaining how boundaries have been met or if this has been done with any clinical consideration or just completely arbitrarily, for example severe omission is seen by Takii et al., (2002, 2003) as omission of at least 25% of the total dose of insulin prescribed while others set other limits

(Baechle et al., 2014; Pollock-BarZiv & Davis, 2005). It may be that this attempt to amalgamate insulin omission into wider Eating Disorder pathology is in order to demonstrate that other than this behaviour, EDs appear in this population as it does in the general population, however when looking at patient attributions of what contributed to the development of their Eating Disorders (see chapter 7) Diabetes specific aspects feature heavily. That they do shows that by nature EDs in this population are different. In research focussing on patients with confirmed ED it is consistently reported that T1Ds have much worse outcomes and higher dropout rates than their non T1D counterparts (Colton et al., 2015a; Colton et al.,2015b). This may suggest that the treatment is not working because regardless of the diagnoses, in T1D EDs are fundamentally different. It could therefore be argued that what is required in order to treat these patients is an approach separate from that taken in standard ED. In T1EDs who omit insulin food is a mechanism by which to lose weight, the more one eats the quicker blood glucose rises and the more weight can be lost. For these patients, food may not be, as assumed in standard models of EDs, 'the enemy'. It is in fact the insulin and taking the correct dosage that is the source of pathology.

It should be noted that in some of the surveys non-responders were more likely to have higher HbA1c levels (Svenson et al., 2003; Neumark – Sztaine et al., 2002; d'Emden et al., 2014), in some cases this is taken to suggest higher levels of ED symptomology but again there is an issue with boundaries between high and normal HbA1c. For example, one study may use above the recommended NICE guideline and another may select one somewhat arbitrarily stating that these are 'generally accepted' (Bernstien et al., 2013, p. 11) but not by whom. It must also be considered that not all raised HbA1c are due to EDs, there are many factors that may affect that BG, periods of stress, periods of growth, bereavement to name but a few.

The above may explain why diagnoses vacillate widely. As such it could be argued that diagnostic categories are currently of little use in this population. They do not describe the main feature of insulin omission appropriately, they do not weight it in any considered manner and there is not any consensus on what constitutes severity either.

### 2.5.8 Strengths

The purpose of the current review was to provide the broadest overview possible of how EDs in the T1D population have been measured since Nielson's 2002 article. In doing so this is the largest systematic review to date in this area and it las looked at every aspect possible. Unlike other studies it has not focussed on controlled research, clinical samples or interview research only and as such can make broader assumptions.

### 2.5.9 Limitations

The purpose of this review was to be as broad as possible but in doing so there may have been some finer details that have been omitted. It was deemed inappropriate to only review controlled studies as there is a strong argument that for this group, there is no appropriate or equivalent control in the general population. Further research may wish to focus on the differences between those with T1D who do and do not have EDs, rather than on a control group of non T1Ds.

As the aim of the current study was to be as broad as possible, quality was not considered as a prerequisite for inclusion. On reviewing the literature, it became apparent that the majority of assumptions made regarding T1ED were based on fundamentally flawed instruments. Therefore, even studies with excellent methodology are essentially problematic. It is for this reason that alpha levels reported in the studies were considered irrelevant (this is further expanded in the proceeding chapter).

### 2.6 Conclusion

The canon of research investigating Eating Disorders in Type 1 Diabetes is, for lack of a better term, messy. There is little consensus as to which instrument to use, if or how it should be modified, what diagnoses should correspond to insulin omission and sample sizes tend to be problematically low and from small geographical regions. Prevalence rates of both clinical diagnoses and insulin omission vary wildly and conclusions regarding this group as a whole are difficult to make.

The vast majority of research investigating treatment programmes for these patients have concluded that standard models don't work and that relapse and dropout rates are higher (Colton et al., 2015a; Colton et al., 2015b). That would seem a relatively obvious outcome if we accept that the reason for this failure is an error in what actually needs to be treated. This would also concur with evidence that shows that patients themselves differentiate between diagnoses, they see 'Diabulimic' as behavioural (insulin restriction or omission), just as they appear to differentiate as bulimic when they binge and purge (Allan, 2014). There is not a scale for these participants. Even Diabetes specific questionnaires have issues. It may be that a serious effort to explore these patients qualitatively would bear more accurate instruments to describe the psychopathology of this group. For example, Peveler et al state

> It is clear that insulin misuse for the purpose of weight control is not confined to subjects with a clinical Eating Disorder. (Peveler et al., 2005, p. 87)

And herein may lie the fundamental problem. For this group Eating Disorders and maladaptive weight and shape control are fundamentally different from the general population *because* of insulin omission and other T1D specific factors; their 'clinical' is not something that is adequately described by looking to standardised models. Validation studies stress the importance of assessing the 'full spectrum of Eating Disorder pathology' (d'Emden et al., 2012, p 997) but it appears that for those with T1ED we do not understand that full spectrum yet.

# 3 Scale Comparison

## 3.1 Introduction

The systematic review undertaken in the previous chapter demonstrates that from the 60 papers reporting ED symptomology more than 21 different measurement instruments were used. Among these articles though, few investigate the potential problems of utilising the scales in a population who by nature have what may be viewed as abnormal eating patterns, in a systematic or meaningful way despite acknowledgement that these are genuine issues. As previously discussed people with T1D for example may utilise more restrictive diets, avoid certain food groups, feel like their eating is out of control or experience negative emotions around food that are due to the pressures of the Diabetes regimen rather than as a result of an ED. Furthermore, insulin omission for weight loss is a behaviour uniquely available to this population and as such is not covered by standardised Eating Disorder questionnaires with researchers often adding items regarding this action as an adjunct.

Powers et al. (2012, 2013) sought to ascertain the effect that T1D would have on two common self-report scales; the Eating Disorders Examination Questionnaire (EDEQ) and the Eating Disorder Inventory (EDI). They did this by first comparing screening scores between those with T1ED who had been treated at an Eating Disorder clinic and those with ED but no T1 (2012) and then by investigating the effects T1D may have on individual items by way of an expert panel (2013). The 2012 study using 2:1 matched controls found that those with T1D actually fared better on EDEQ and EDI scores and were less 'psychologically compromised' (Powers et al., 2012, p. 252) particularly on the questions relating to food restriction and control. The authors conclude that this may be due to a healthier approach to meal planning and health monitoring that accompanies T1D, however as nearly half of their participants were insulin omitters an alternative explanation would be that they do not need to exercise restraint as they can control their weight easily via insulin manipulation.  The authors claim that they managed to control for participants who utilised insulin omission as their sole ED behaviour by using participants without T1D who had 'diverse compensatory behaviours' (Powers et al., 2012, p. 253) but there is an argument that insulin omission is a unique feature of T1ED that has specific biological and psychological mechanisms that cannot be controlled

for and thus these results may not be ecologically valid. The study only investigated the difference between responses on these questionnaires and did not emphasise the difference between clinical diagnoses which might have usefully highlighted the different behaviours within the diagnostic categories.

The 2013 paper by the same team utilised an expert panel who assessed the questions on the EDEQ and the EDI and rated whether they thought T1D would influence the answers. They suggested that half of the items in the EDEQ could be heavily influenced by T1D. The EDI fared better with only around 7% of the questions deemed potentially influenced by T1D.

Another ED questionnaire that is used routinely is the Eating Attitudes Test (Garner, Olmsted, Bohr & Garfinkel, 1982; Garner & Garfinkel, 1979). Steel et al (1989) were among the first to highlight that T1D could potentially bias answers to an ED measurement instrument. They also used an expert panel to complete the EAT 40 as if they were 'sensible young Diabetics without an Eating Disorder' (Steel et al., 1989, p. 515) and concluded that 6 of the questions were potentially sensitive to T1D. They found in a cross-sectional, controlled sample that the unadjusted EAT-40 classified more T1Ds as clinically concerning in comparison to controls, than the modified version. In women there was still a significantly higher proportion scoring as concerning compared to controls but for men the difference became insignificant. A reduced item version of EAT has been more commonly used in recent years. The EAT-26 (Garner et al., 1982; Garner & Garfinkel, 1979) is widely reported as one of the most commonly used Eating Disorder screening measures. It consists of 26 items that form 3 subscales, *Dieting, Bulimia and Food Preoccupation* and *Oral Control* scored on a 6 point Likert scale with answers ranging from 'Always' to 'Never'. Items include *Am terrified about being overweight*, *Find myself preoccupied with food* and *Avoid eating when I am hungry*. A cut-off point of 20 is recommended for further clinical investigation (Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990; Dotti & Lazzari, 1998) unlike the EDI and the EDEQ no one has seriously considered the effect of T1D in this questionnaire. There have been various attempts but no general consensus as to which questions should be added regarding insulin omission or subtracted regarding the effects of Type 1 Diabetes (Jones et al., 2000).

A perhaps obvious solution to the issue of screening and detecting T1ED is to make sure that any instrument used takes regimen effects into consideration. Such an instrument would also need to be cognisant of the availability of insulin omission to this demographic. Such a scale was proposed by Antisdel et al. in 2001 and revised by Markowitz et al. in 2010. The Diabetes Eating Problem Survey Revised is one of the most commonly used instruments in the T1ED population. The DEPS-R is a unidirectional scale that is designed to give a global score of ED symptomology. In this sense it treats Diabetes related Eating Disorders as a homogenous syndrome. However, researchers have pointed out that sections of the DEPS-R deal exclusively with Eating Disorder symptomology attributed to insulin administration. Merwin (2014) states that the five items on the DEPS-R relating to insulin omission are *I try to keep my blood sugar high so that I will lose weight*, *I try to eat to the point of spilling ketones in my urine*, *I feel fat when I take all of my insulin*, *After I overeat, I don't take enough insulin to cover the food* and *After I overeat, I skip my next insulin dose.*. Wisting et al. (2013) describe a large-scale registry study whereby they utilise a sample of T1D adolescents to explore the structure of the DEPS-R suggesting a 3-factor solution broadly relating to eating habits, drive for thinness and insulin omission. Similarly, Altinok et al. (2017) state that they performed a confirmatory factor analysis on the DEPS-R using a Turkish sample of a similar age, but they do not state the factor structure found.

## 3.2   The Current Study

The purpose of the current study was to examine the properties of the EAT 26 and the DEPS-R using a large cross-sectional sample of adults with T1D.

The EAT 26 was first used to investigate how many participants screened above the cut-off point for clinical concern compared to the DEPS-R. Then questions that could be considered biased by T1D by decision of an expert panel were removed from the EAT-26 and the analysis repeated to explore whether this reduced the number of participants scoring above the cut-off point. Gender differences were also explored. It was hypothesised that initially the EAT-26 should identify more participants above the cut-off point than the DEPS-R but by removing items potentially biased by T1D should reduce that proportion.

The factor structure of the DEPS-R was then investigated using exploratory factor analysis with methodology replicated from Wisting et al. (2013) in order to ascertain whether the DEPS-R performed similarly in an adult sample (as compared to the adolescent samples used by Wisting et al. (2013) and Altinok et al. (2017) and to explore whether subscales relating to different behaviours may be viable. The response patterns to the questions suggested by Merwin et al. (2014) that relate directly to relating to insulin omission were then described in terms of whether the behaviour is ever present and the potential of using them as a subscale was tested. It was hypothesised that the questions in the DEPS-R would be related to underlying factors that potentially describe different types of ED behaviour and that the questions suggested by Merwin (2014) would form a subscale relating directly to insulin omission.

## 3.3    Methodology

### 3.3.1    Design

For the first section comparing global EAT-26 score to global DEPS-R score, as the variables were categorical and within subjects, McNemar's test was used to investigate the difference in proportions of those scoring above the cut-off point for clinical concern. Following this a modified version of the EAT 26 was analysed to determine if this lowered the proportion scoring above the modified cut-off point. Then in line with Wisting et al. (2013) a Principal axis factoring with oblimin rotation was performed on the DEPS-R data to ascertain any underlying latent structure. Next as the DEPS-R utilises a Likert scale with the response options 'never', 'rarely', 'sometimes', 'often', 'usually', and 'always', the data relating to the five items on the relating to insulin omission as defined by Merwin et al (2014) were transformed so that any presence of the behaviour was coded as yes response and no presence as defined by a 'never response' was coded as a no response.

### 3.3.2    Ethical Approval, Setting, Procedure, Survey Participants

Please note that the data collected for the current study was part of a larger data collection that informs chapters 4 – 7.

### 3.3.3 Ethical Approval

Ethics approval for this study was granted by the Birkbeck departmental ethics committee on the 27th January 2014, approval number 131462

### 3.3.4 Setting

Recruitment took place over the period of 04/05/2015 – 01/10/2015. An advert was posted on the social media pages of the registered charities: Diabetes UK, the Juvenile Diabetes Research Foundation, Diabetics with Eating Disorders. The same advert was also posted on every Facebook page offering support and information to those with Type 1 Diabetes and the biggest online support forums for Type 1 Diabetes: Diabetes.co.uk, TuDiabetes and Diabetessupport.co.uk. A shorter version was also put on Twitter using the popular Diabetes hashtags #T1D, #DOC and #GBDOC. Participants were invited to share the advert on their social media pages and notify their Type1 friends/colleagues/health care professionals. (please see appendix H).

### 3.3.5 Procedure

If participants decided to take part they were taken to the website www.typeoneandpsychology.org where specially designed 'questioner' software was hosted. The website was built using Wordpress and the software was built in the python programming language. The resultant database utilised MySQL and was held on a private secure virtual server owned by the researcher. The landing page gave information about the study and asked for consent, on completing the ethics questions, the participants were taken through to the Questioner programme (please see appendix I). The Questioner programme was designed so that after the demographics questions were answered, if they chose, participants could create a user account that allowed them to sign in and out of the programme at their convenience as the study was long. A number of scales were presented for completion (that inform other sections of this research) including those used in the present chapter and then the participants were debriefed (please see appendix J).

### 3.3.6 Participants

Eligibility criteria for the participants were being over the age of 16, having been diagnosed by a medical professional as having Type 1 Diabetes and having a good grasp of English. As the study was conducted online internet access was also essential. This may have been via a computer/ table/ laptop/ smartphone. All of the participants fulfilled this criteria none were excluded from the final analysis.

### 3.3.7 Expert Panel

An expert panel including 2 Diabetes consultants, a Diabetes dietician, a diabetic specialist nurse, 2 Diabetes consultant psychiatrists, a psychiatrist with experience of Diabetes and the researcher, 2 of whom also have T1D rated each of the items on the EAT 26 as potentially biased by Diabetes via email communication with the researcher.

### 3.3.8 Materials

The Diabetes Eating Problem Survey Revised (see section 2.4.8.1) and the Eating Attitudes test 26 Item was used (see section 2.4.7.3).

### 3.3.9 Software

IBM statistics SPSS ver 23 (IBM Corp. 2016) was used to perform the analyses

### 3.4 Results

Please note that the demographic information for this study is also noted in section 6.2

### 3.4.1 Sample Characteristics

#### 3.4.1.1 Initial Numbers

687 participants started the questionnaire having being recruited mostly by social media (610/ 88.8%). All participants completed the gender question. 92 (13.4%) were male, 592 (86.2%) were female, 2 were transgendered (0.3%) and 1 selected prefer not to say (0.15%). 686 participants gave their age. They had mean age of 33.73 (12.01) years (range:16 – 69). Many

nationalities were reported but most were UK/ Eire and US nationals living in those countries all participants answered these questions and the ethnicity question, the majority (599/ 87.2%) were white and a large minority selected prefer not to say (81/ 11.8%) (for the full characteristics of the participants please see appendix K).

### 3.4.1.2   Medical Status

686 participants reported a mean age at diagnosis of 15.48 (11.58)[8] years (range: 0 – 61) and a mean duration T1D of 18.25 (11.98) years (range: 0 – 59). 659 participants reported a mean HbA1c% of 8 (2.24, range: 4 – 29%). 530 participants reported their methods of blood sugar management. 319 (60.2%) utilised a pump, 200 (37.7%) were on multiple daily injections, 4 (0.75%) used a mixtard9, 6 (1.13%) used syringes and vials and 1 (0.19%) used inhaled insulin. 222 (41.89%) of these patients also utilised a continuous glucose measurement device. 684 participants reported a mean of 0.67 (1.12) Diabetes related complications.  The most commonly reported were Retinopathy and other eye issues (Macular Oedema, Cataracts and Glaucoma) (162/ 23.7%), Neuropathy (101/ 14.7%), Gastroparesis (47/ 6.9%) and Nephropathy (36/ 5.3%). The 684 participants were taking an average of 0.23 (0.84) medications to manage these conditions. Of the 687 asked, 325 (47.3%) participants reported having more than Diabetes as a medical diagnosis. Of these the most common conditions experienced were Hypothyroidism (95/ 13.8%), Asthma (56/ 8.15%) and Coeliac Disease (23/ 3.3%) (for full participant medical status please see appendix K).

### 3.4.1.3   Participants Mental Health Information

439 participants answered the question 'have you ever had a mental health diagnosis (except an Eating Disorder diagnosis)' 193 (44%) answered yes so participants had a mean of 0.678

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[8] Please note that this statistics in this thesis follows the following convention when reporting means. MEAN (STANDARD DEVIATION)

[9] A mixtard is an injectable form of insulin that contains both long acting and short acting insulin.

(.913). Of those who did report other diagnoses the most common were depression (155/ 35.3%), anxiety (79/ 18%) borderline personality disorder (14/ 3.2%), bipolar disorder (10/ 2.3%), and post-traumatic stress disorder (10/ 2.3%) for full participant mental health characteristics please see table 1.6. Participants who had reported having another mental health diagnosis had a mean of 1.52 (0.78 min 1 – max 5) conditions. 435 participants answered the question 'to the best of your knowledge does anyone in your family have a mental health diagnosis?'. Of them 217 (49.9%) answered yes, 173 (39.7%) answered no and 45 (10.3%) stated that they weren't sure. 435 participants answered the question 'to your knowledge has anyone in your family ever had an Eating Disorder?' of these 60 (13.8%) answered yes, 354 (81.4%) answered no and 21 (4.8%) stated that they weren't sure. 439 participants answered the question 'have you ever been diagnosed with or thought that you had an Eating Disorder' of these 130 (29.9%) answered yes (for full participant mental health status please see appendix K).

### 3.4.2 Modified EAT 26

An item was deemed potentially sensitive to T1D if it was selected by 5 out of the 8 expert panel. The panel concluded that 50% of the items as detailed in the table below were potentially biased

*Table 3.1: EAT-26 and Potentially Biased Items.*

| Question | Subscale | Remove |
|---|---|---|
| 1) Am terrified about being overweight. | Dieting | |
| 2) Avoid eating when I am hungry. | Oral Control | x |
| 3) Find myself preoccupied with food. | Bulimia and Food Preoccupation | x |
| 4) Have gone on eating binges where I feel that I may not be able to stop. | Bulimia and Food Preoccupation | x |
| 5) Cut my food into small pieces. | Oral Control | |
| 6) Aware of the calorie content of foods that I eat. | Dieting | x |
| 7) Particularly avoid food with a high carbohydrate content (i.e. bread, rice, potatoes, etc.) | Dieting | x |
| 8) Feel that others would prefer if I ate more. | Oral Control | |

| Question | Subscale | Remove |
| --- | --- | --- |
| 9) Vomit after I have eaten. | Bulimia and Food Preoccupation | |
| 10) Feel extremely guilty after eating. | Dieting | x |
| 11) Am preoccupied with a desire to be thinner. | Dieting | |
| 12) Think about burning up calories when I exercise. | Dieting | |
| 13) Other people think that I am too thin. | Oral Control | |
| 14) Am preoccupied with the thought of having fat on my body. | Dieting | |
| 15) Take longer than others to eat my meals. | Oral Control | |
| 16) Avoid foods with sugar in them. | Dieting | x |
| 17) Eat diet foods. | Dieting | x |
| 18) Feel that food controls my life. | Bulimia and Food Preoccupation | x |
| 19) Display self-control around food. | Oral Control | x |
| 20) Feel that others pressure me to eat. | Oral Control | |
| 21) Give too much time and thought to food. | Bulimia and Food Preoccupation | x |
| 22) Feel uncomfortable after eating sweets. | Dieting | x |
| 23) Engage in dieting behaviour. | Dieting | x |
| 24) Like my stomach to be empty. | Dieting | |
| 25) Have the impulse to vomit after meals. | Bulimia and Food Preoccupation | |
| 26) Enjoy trying new rich foods. | Dieting | |

### 3.4.3   Completion

516 participants completed the Eating Attitudes Test 26 in its entirety. 491 participants completed the Diabetes Eating Problem Survey Revised in its entirety. Only whole responses were analysed, data was missing completely at random (Littles test Chi-Square = 2901.456, df = 3356 p =1)

### 3.4.4    EAT 26 vs DEPS-R

#### *3.4.4.1    Reliability*

In this sample the total EAT 26 scale showed good internal reliability (Cronbach's alpha = .908). The dieting subscale also demonstrated good validity (Cronbach's alpha = .877) as did the Bulimia and food preoccupation subscale (Cronbach's alpha = .813) but was reduced for oral control (Cronbach's alpha = .718). The modified EAT containing the 13 questions which were assumed to be unbiased also had good validity (Cronbach's =.833), the dieting subscale had good validity (Cronbach's = .817), as did the oral control subscale (Cronbach's alpha = .719) and the bulimia and food preoccupation subscale (Cronbach's alpha = .816)

The DEPS-R demonstrated very good validity (Cronbach's alpha =.936)

#### *3.4.4.2    Cut-Off Points*

As the EAT-26 has a cut-off point of 20 and 50% of the items were deemed to be potentially biased the cut-off point for the modified EAT 26 was 10.

*Table 3.2 Means and Frequencies of those Scoring above the Cut-Off Point*

|  | Mean (sd) | Above cut-off | % (valid) | Missing | % (total) |
|---|---|---|---|---|---|
| Total Sample |  |  |  |  |  |
| DEPS R | 23.19 (17.24) | 217 | 44.2 | 196 | 28.5 |
| EAT-26 | 38.55 (16.1) | 443 | 85.9 | 171 | 24.9 |
| Modified EAT | 28.32 (8.48) | 475 | 92.1 | 171 | 24.9 |
| Males |  |  |  |  |  |
| DEPS-R | 15.89 (14.48) | 13 | 20.6 | 29 | 31.5 |
| EAT-26 | 47.55 (20.73) | 62 | 93.9 | 26 | 28.3 |
| Modified EAT | 27.15 (10.96) | 63 | 95.5 | 26 | 28.3 |
| Females |  |  |  |  |  |
| DEPS-R | 24.33 (17.39) | 204 | 47.9 | 166 | 28.0 |
| EAT-26 | 37.16 (14.84) | 378 | 84.6 | 145 | 24.5 |
| Modified EAT | 22.7 (7.9) | 409 | 91.5 | 145 | 24.5 |

### 3.4.5  McNemar Chi Square

A McNemar Chi square showed that there was a significant difference between those scoring yes and no for further investigation with participants scoring yes significantly more often using the EAT-26 than the DEPS-R. (n = 459, chi square = 109.829, p <0.001) when looking at the population as a whole. This assumption held when looking individually at male participants (n = 56, chi square = 36.54, p <0.001) and female participants (n = 401, chi square = 75.52, p <0.001).

A McNemar Chi square also showed that there was a significant difference between those scoring yes and no for further investigation with participants scoring yes significantly more often using the modified EAT than the DEPS-R (n = 459, chi square = 158.305, p<0.001) when looking at the population as a whole. This assumption held when looking individually at male participants (n = 56, chi square = 35.021, p <0.001) and female participants (n = 401, chi square = 120.831, p<0.001).

As expected the unmodified EAT 26 identified more participants as clinically concerning than the DEPS-R but unexpectedly, using a modified version of the EAT actually increased the rates of those scoring as clinically concerning. More males were also identified as concerning than females.

### 3.4.6  Factor Structure of the DEPS-R

#### 3.4.6.1  Data Screening

Item correlations between all of the items were checked to ensure that they had values above r = .3 indicating that they had sufficient relationships. No variables were removed from the analysis (please see appendix L for the full correlation matrix).

#### 3.4.6.2  Sampling Adequacy

The Kaiser-Meyer-Olkin measure of sampling adequacy was over the recommended value of .5  (KMO = .945) indicating that there is a sufficient proportion of variance in the sample which may be attributable to underlying factors. Bartlett's test of sphericity was significant

Chi Square = 5449.596 df = 120 p < 0.001 suggesting that there are related variables and therefore a factor analysis is a suitable procedure.

### 3.4.6.3   Extraction and Rotation

In replication of Wisting et al (2013) a Principal axis factoring with oblimin rotation was performed. Using the Kaiser – Gutman Criteria of retaining factors with an Eigen value over 1 a 2 factor solution was suggested, factor 1 explained 53.1% of the variance and factor 2 8.6% with a cumulative explanatory power of 61.7%.

*Figure 3.1 Scree Plot of Suggested Factors*
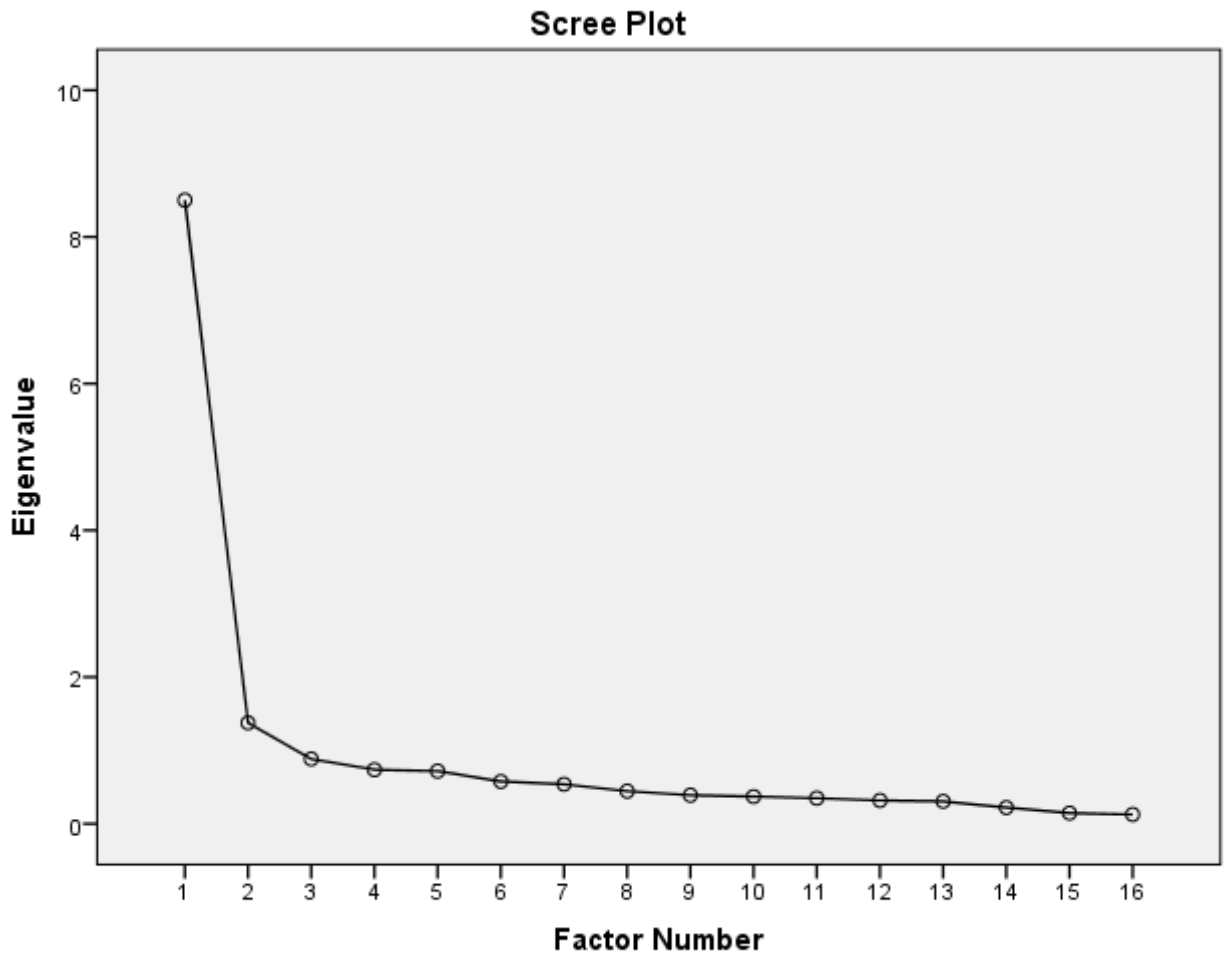
### 3.4.6.4  Factor Loadings

*Table 3.3: Factor Structure*

|  | Factor | |
| --- | --- | --- |
|  | 1 | 2 |
| DEPS-R1 Losing weight is an important goal to me | | 0.717 |
| DEPS-R2 I skip meals and/or snacks | | |
| DEPS-R3 Other people have told me that my eating is out of control | 0.448 | |
| DEPS-R4 When I overeat, I don't take enough insulin to cover the food | 0.671 | |
| DEPS-R5 I eat more when I am alone than when I am with others | | 0.530 |
| DEPS-R6 I feel that it's difficult to lose weight and control my Diabetes at the same time | | 0.728 |
| DEPS-R7 I avoid checking my blood sugar when I feel like it is out of range | 0.605 | |
| DEPS-R8 I make myself vomit | 0.471 | |
| DEPS-R9 I try to keep my blood sugar high | 0.995 | |
| DEPS-R10 I try to eat to the point of spilling ketones in my urine | 0.950 | |
| DEPS-R11 I feel fat when I take all of my insulin | 0.569 | |
| DEPS-R12 Other people tell me to take better care of my Diabetes | 0.613 | |
| DEPS-R13 After I overeat, I skip my next insulin dose | 1.021 | |
| DEPS-R14 I feel that my eating is out of control | | 0.516 |
| DEPS-R15 I alternate between eating very little and eating huge amounts | | 0.468 |
| DEPS-R16 I would rather be thin than to have good control of my Diabetes | 0.676 | |

### 3.4.6.5  Reliability

The first factor which could be conceptualised as the more severe behaviours such as insulin omission, vomiting and neglecting Diabetes self-care behaviour had high reliability (Cronbach's alpha = .923). The second factor which could be conceptualised as the less severe behaviours surrounding ED like cognitions and actions around food demonstrated good validity (Cronbach's alpha = .857).

### 3.4.6.6  Insulin Subscale

#### 3.4.6.6.1  Insulin Subscale Reliability

The five items as a scale showed good validity (Cronbach's alpha = .905) which would have been improved with the removal of DESPR11 I feel fat when I take all of my insulin (Cronbach's alpha = .91).

#### 3.4.6.6.2  Insulin Subscale Cut-Off

As the cut-off point for clinical concern in the DEPS-R was 20 the cut-off point for the insulin subscale equalled 6.25, which was rounded down to 6 as the responses were only possible in whole numbers, to ascertain how many participants were identified by the subscale as clinically concerning.

*Table 3.4 Mean Scores and Cut- off Points by Total Sample and Gender*

|  | Mean (sd) | Above cut-off | % (valid) | Missing | % (total) |
|---|---|---|---|---|---|
| Total Sample Insulin Subscale | 4.34 (6.02) | 116 | 16.9 | 185 | 26.9 |
| Males Insulin Subscale | 2.8 (4.91) | 8 | 12.5 | 28 | 30.4 |
| Females Insulin Subscale | 4.54 (6.08) | 107 | 24.6 | 157 | 26.5 |

Females scored higher on the insulin subscale with nearly a quarter scoring above the cut-off point than men with around an eighth. This is similar to rates of insulin omission seen in the systematic review (please see previous chapter). It should be noted however that the original responses on the DEPS-R are in Likert formula. In order to investigate whether the behaviours/ thoughts described by the subscale were ever present in the sample the items were transposed into binary format.

*Table 3.5: Response Patterns to the Insulin Subscale*

| | Total Sample | | Males | | Females | |
|---|---|---|---|---|---|---|
| | yes | % | yes | % | yes | % |
| When I overeat, I don't take enough insulin to cover the food | 345 | 67.9 | 45 | 69.2 | 299 | 68 |
| I try to keep my blood sugar high so that I will lose weight | 123 | 24.2 | 7 | 10.8 | 115 | 26.1 |
| I try to eat to the point of spilling ketones in my urine | 79 | 15.6 | 8 | 12.5 | 70 | 15.9 |
| I feel fat when I take all of my insulin | 212 | 41.7 | 11 | 16.9 | 200 | 45.4 |
| After I overeat, I skip my next insulin dose | 117 | 22.9 | 6 | 9.2 | 110 | 24.9 |

There were inconsistent responses to the insulin omission behaviour subscale and males scored higher than females on the first item which is inconsistent with previous research suggesting that maladaptive insulin behaviour is more common in females (please see previous chapter). A significant majority of participants stated that they don't take enough insulin to cover overeating but far less participants stated that they deliberately overeaten in order to spill ketones. This suggests that a further investigation of the questions is warranted in order to ascertain what they are actually measuring and how closely that matches ED.

### 3.4.7 Figure 3.2: Percentage of Participants Scoring above the Cut-off Point for Clinical Concern
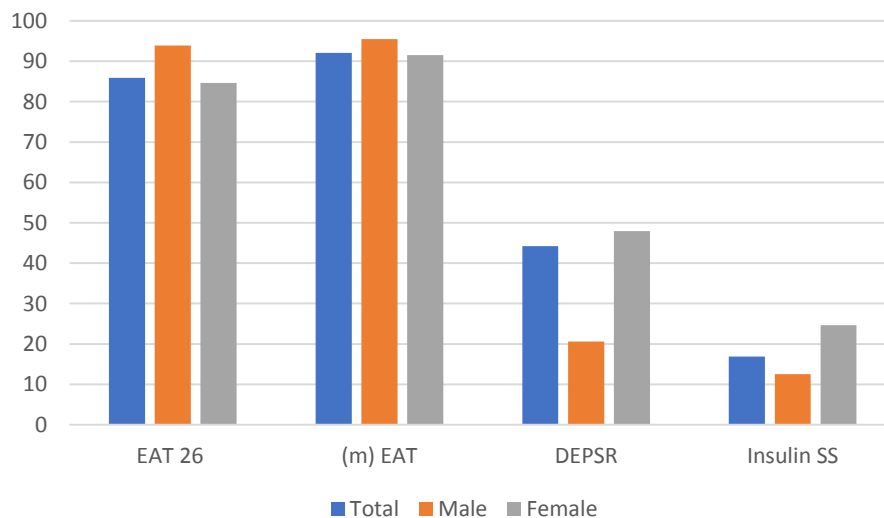


ss = subscale, m = modified

## 3.5 Discussion

This study hypothesised that the EAT-26 would screen more participants as clinically concerning than the DEPS-R and this was the case. This held for both men and women, but a larger proportion of men screened positive on the EAT-26 which is a reversal of what would normally be expected. When using the DEPS-R a higher proportion of Females screened as clinically concerning which is concordant with previous research (See previous chapter). It was then hypothesised that removing items on the EAT-26 which were deemed to be potentially influenced by T1D that the amount of those screening clinically concerning would be reduced but the opposite was seen, and the modified version screened nearly the whole sample as clinically concerning.

The factor structure of the DEPR was then investigated and a 2-factor solution suggested. This is different from previous research using a child and adolescent sample. It appears that the first and largest factor relates to behaviours which are situated at the more severe end of the ED spectrum such as omitting insulin, vomiting and deliberately inducing ketones whereas the second and smaller factor is more related to feelings and behaviours around food which are less severe behaviourally, also it is notable that this factor contained no items relating to insulin behaviour.

The insulin items suggested by Merwin (2014) formed a reliable scale. Less participants scored above the cut-off point than for the full DEPS-R indicating that the items were potentially are measuring a different behaviour. Females also scored higher than males which is consistent with other research measuring insulin omission (please see previous chapters). It was also interesting that the only question which would have improved reliability via removal related to feelings rather than action *I feel fat when I take all of my insulin.* It could possibly be argued that the other four questions are specifically measuring the performative aspects of 'Diabulimia'.

When investigating binary response patterns to the 5 questions that relate directly to insulin an interesting pattern was found, there were much lower levels of participants saying that they tried to keep their blood sugar high, tried to attain ketones or skip their insulin dose after over eating than not covering their overeating with appropriate insulin. Also, more males said

that they under dosed following overeating than women. however nearly half of the female sample stated that they felt fat when taking their insulin which was much higher than males.

The high levels of clinically concerning behaviour using the EAT-26 in this sample is not seen in previous research using this scale. This having been said a study utilising the EAT-40 in an adolescent sample found that around 70% of participants screened above the cut-off point (Pinar et al., 2005) while Phillipi et al (2013) found rates of around 45% in adults, neither of these studies modified the scale in any way. It should be noted that these studies also recruited from Diabetes clinics whereas this study utilised an anonymous cross-sectional internet sample. As such the results may be interpreted in one of 2 ways, the first, as this is an internet sample and completely anonymous participants felt more able to be truthful about their thoughts and behaviours, the second, this sample is qualitatively different than previous research.

It could be that the nature of T1D simply makes one vulnerable to displaying ED behaviour and having ED like cognitions as measured by the EAT and thus the cut-off point represents too low a threshold. It should be noted that the cut-off is supposed to be diagnostically relevant and aid in the recognition of clinically significant behaviour and perhaps T1D results in thoughts and actions that in other populations are seen as pathological. Parents of Type 1 children report more meal time related behavioural problems than parents of children without T1D and children with other clinical feeding problems, including intense disruptive behaviour (Patton, Dolan & Powers, 2006). It may be that some sort of behavioural pattern, set early on following the onset of T1D is influencing the patterns seen using this instrument. This having been said not all T1Ds are diagnosed in childhood or even adolescence.

Based on the findings of this study it may be justifiable to recommend against the use of any version of the EAT in those with T1D. It is relatively inconceivable that all those with T1D have an ED.

The DEPS-R provided a more conservative prevalence estimate and the insulin subscale lower still. However, in this sample there are inconsistencies when looking at statements pertaining to the manipulation and omission of insulin. It maybe that these inconsistencies are due to the wording of the questions, participants are asked whether they *try* to keep their

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blood sugar high or spill ketones into their urine and this implies intent. Also missing an entire dose may be argued to be on the more severe end of ED behaviour which is why a lower level of participants answer in the affirmative. Furthermore, it could be argued that T1Ds are more aware of when they overeat as if their blood sugar after a meal is higher than expected that would indicate that they have underestimated the amount they consumed. Also, as insulin is normally administered before eating it may follow that participants are cognisant that they have under dosed which may affect answers to that question. This having been said the rates of those screening as clinically concerning is higher but not overly dissimilar to that reported by other studies (please see previous chapter)

What does seem to be apparent is that a large proportion of participants display at least some level of insulin manipulation and that this is in direct relation to at least the perception of over eating. Given that only around 40% of participants screened as being concerning using the DEPR this is significant. It could be that this demographic by nature are more pathological and that unlike the general population those with T1D have a way to negate the effects of overeating. Alternatively, it maybe that the Likert scale format needs to be revaluated as 'rarely' performing an action that is as physically damaging as insulin omission is not weighted appropriately. That nearly half of the female participants in this sample stated that taking their insulin correctly made them feel fat is also noteworthy and potentially justifies some kind of behavioural intervention where insulin can be seen in a more positive manner or disassociated from negative weight perceptions.

### 3.5.1   Strengths

The sample size of the current study is a significant strength as stated in the previous chapter very little large scale research into this population has been carried out, it also utilised a global sample which is significantly different from other research which tends to focus on clinical participants from defined geographical areas. This is the first study which has utilised an expert panel of T1ED specialists to assess the suitability of the EAT-26 for use in this demographic. Also, little deconstruction has been done of the DEPS-R and the current study represents a significant progression in further enquiring on the nature of this instrument.

### 3.5.2   Limitations

There is no gold standard by which comparisons can be made, although that is not an issue unique to this study but rather to all research utilising those with T1D. As stated in the previous chapter, investigating EDs in this population is complicated and fraught with methodological issues.

Part of this study aimed to investigate gender differences in Eating Disorder symptomology as measured by the EAT-26 and DEPS-R but there was a relatively small sample size for males so any conclusions regarding gender specific ED behaviour should be interpreted with caution. This may have been influenced by the recruitment process which advertised the study as measuring psychological variables. It maybe that the males responding to the advertisement represent a biased sample for which the participants are not representative of the male gender but rather those who are willing to confront psychological issues or have some sort of insight in to their own psychological profile. It has been well reported that males are less likely to report psychological difficulties. Also, this sample was recruited mostly via the internet and the Diabetes Online Community (DOC). Membership of the DOC may have exposed participants to subjects relating to psychological aspects of Type 1 Diabetes and therefore the whole sample may be generally representative of those members who are comfortable discussing these issues or who have joined the DOC because of experience in these matters.

The limitations observed in this study could be overcome by a few modifications, for example a clinical sample using patients from nationwide Diabetes clinics may be able to produce a larger more gender equal representation of those with T1D. Carefully worded clinical interviews may also be able to untangle a more accurate picture of what is happening with these patients. It maybe that neither of the EAT-26 or any kind of modified version is suitable for use in this population or alternatively that what we are actually perceiving as ED symptomology is some other type of behavioural and cognitive phenomena unique to T1D. Qualitative researchers may want to take note of these findings and explore the structure of ED scale item responses in a more focussed manner.

Although the DEPS-R in this sample seemed to be more reliable there are still issues with its use that should be further investigated (please see chapter 9 for a larger discussion on the use of the DEPS-R)

## 3.6    Conclusion

Measuring Eating Disorders in Type 1 Diabetes is a complicated process that appears to be heavily affected by the nature of the illness. The EAT-26 may be unsuitable for use in this population given that even when it is modified to allow for potential T1D regimen effects it identifies almost all participants as high in ED symptomology. The DEPS-R while identifying a much lower range of participants as below the cut-off point, still has issues and those who demonstrate lower levels of ED symptomology that still have significant negative clinical implications may screen out. This having been said, the DEPS-R is arguably the most suitable instrument for use in this population currently.  It could be suggested, given the severity of the consequences observed in those with T1ED, that a dual measure is warranted, one which can highlight problematic cognitions and behaviours and one which can highlight clinical risk such as the HbA1c. These two measures combined would give a clearer indicator of what kind of intervention the patient needs.

# 4 Chapter 4: Risk Factors and Co-morbidities

## 4.1 Introduction

As stated in previous chapters the literature on Eating Disorder in Type 1 Diabetes is complicated but there appears to be a general consensus that Eating Disorders and Disordered Eating Behaviour are more common in this population. This suggests that Diabetes specific aetiology and risk factors are important. Given that the pilot study[10] reported a complicated psychological profile consisting of co/multimorbid mental health issues and several Diabetes specific latent variables as attributional to ED development, the following section reviews these issues in more detail and then explores a structural model based on these factors that predicts HbA1c and DEPS-R scores.

## 4.2 Demographic Risk Factors

### 4.2.1 Age

Age specific factors may act as protective or risky. In children and young people there is research that suggests that pre pubertal T1D diagnosis is predictive of worse glycaemic control and earlier onset of retinopathy (Kordonouri, Danne, Enders & Weber, 1998). There is also evidence that in those diagnosed in infancy <6 years T1D contributes to cognitive impairments (Gaudieri, Chen, Greer & Holmes, 2008). Puberty comes with many physiological challenges for those with T1D, it has been shown that it is often delayed which may have psychosocial implications, dawn phenomenon, whereby the liver secretes glucose in the early morning (Chowdhury, 2015) and insulin resistance is at its most pronounced during this time making insulin requirement estimations difficult and variable (Amiel, Sherwin & Simonson, 1986). For females particularly, the onset of menarche can prove particularly challenging as rapid weight gain and fluctuating hormones adds to changing insulin requirements. T1D onset in adulthood is not problem-free, however, and research

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[10] please see appendices E & F

suggests that Diabetes control gets worse the longer the duration of the illness (Kovacs, Goldston, Obrosky & Iyengar, 1992). These pubertal difficulties for those with T1D also coincide with what is deemed to be a risky time for the development of EDs in the general population. Eating Disorders have been somewhat popularised in the media to affect young teenage girls, and there is evidence suggesting that this may be the case. Early attempts by researchers to ascertain the age of onset in AN hypothesised that there may be a bimodal distribution whereby risk is highest at the age of 14 and then 18 for females (Halmi, Casper, Eckert, Goldberg, & Davis, 1979). A recent large-scale review of Primary Care registers in the UK found that for females the peak incidence of diagnoses was between the ages of 15 – 19 for girls but was much lower for boys at 10 – 14 (Micali, Hagberg, Petersen & Treasure, 2013).

However, age of onset for T1EDs may be higher than that for EDs in the general population. One study assessing inpatients found that the average age of assessment for those with T1D was 26.2 (Powers et al., 2012), which coincides with the considerable evidence that in the majority of cases T1D diagnoses preceded that of an ED (Powers et al., 2012). Furthermore Goebel-Fabbri., (2011) found that in a longitudinal study of the onset and remission of insulin restriction, new restriction occurred in a significant number of older T1D females at follow-up (average age of the sample was 44). ED behaviour in the T1 population also appears to increase with age post puberty. Longitudinal studies show that while levels remain relatively low in childhood and early adolescence they rise exponentially until in females, by the age of 25, one particular group of researchers found a 60 % chance that a clinically significant Eating Disorder will have occurred (Wisting et al., 2013, Colton et al., 2015, Olmsted et al., 2008). It should be noted that females are hugely overrepresented in this research and as such, the evidence should only be extrapolated to the female population of T1Ds. As such it is unclear whether age will be a predictive variable in the structural model.

### 4.2.2 Gender

Several studies have suggested that certain psychological issues which are commonly comorbid are more prevalent in T1 women than in T1 men. These include studies on depression (Anderson et al., 2001; Trief et al., 2014) and anxiety (Grigsby et al., 2002;

Rechenberg, Whittemore, & Grey, 2017). Adult T1D females are also more likely to seek medical help for mental/ emotional problems and drugs or alcohol problems (Shin, Poltavskiy, Kim, Hasan, & Bang, 2017) although this potentially means that they are more likely to seek help, not necessarily that they are more likely to develop those issues. In line with research in the general population, T1D adult males are significantly more likely to commit suicide than T1D adult females (Wang, An, Shi & Zhang, 2017).

Eating Disorders were historically seen as illnesses that affected nearly exclusively females. (Bruch, 1978). As such much of the early research focusses on this demographic only. In more recent years however the incidence of EDs in males has seen a substantial rise. In the UK the increase in males was around 27% between 2000 and 2009 in the General Practice Research Database (Micali et al., 2013). There is still a huge gender divide in prevalence however, in the aforementioned study ED diagnosis reached a peak of 63.8 diagnoses per 100,000 female patients in 2008 compared to the peak diagnosis of 7.1 per 100, 000 males in 2009.  It may be that there are gender based behavioural differences for those with EDs also. Twin studies have shown that females twins are more likely to report classical symptoms such as dieting or purging food and report higher levels of weight dissatisfaction than their male brothers (Anderson & Bulik, 2004). This is similar to research in the T1D population which also suggests differences in the type of Disordered Eating Behaviour reported (please see section 2.4.3)

As can also be seen in chapter 2 there appears to be a higher proportion of males with Disordered Eating Behaviour in T1D than in the general population and the gap between the percentage of female and male sufferers is lower. Insulin omission has been reported by males in increasing numbers). In a recent Australian survey nearly a fifth of males were above the cut-off point for the DEPS-R, a level, which has also been found in similar studies and over half of male respondents reported binge eating in the previous week. Furthermore, while in this study over three quarters of the male participants reported being dissatisfied with their body shape nearly a third expressed a desire to be bigger. This may represent an important differential factor that is currently poorly understood (please see section 2.4.3; Doyle et al.,2017; Araia et al., 2017). It may be then, that in those with T1D gender is not such a strong predictor of Eating Disorder behaviour than in the general population.

## 4.3 Psychosocial Risk Factors

### 4.3.1 Depression

Depression is one of the most commonly diagnosed mental illnesses in the general population with around 3.3% screening as positive (Moran, Rooney, Tyrer & Coid, 2014). Adults with T1D are at a significantly higher risk of depression than the general population (Gendelman et al., 2009). Depression predicts decreased self-care and blood sugar control (Anderson, Freedland, Clouse, & Lustman, 2001; Barnard, Skinner & Peveler, 2006; Maia et al., 2014) and has been significantly associated with Diabetes related complications such as neuropathy, (Bai et al., 2017). Depression also affects children and adolescents with T1D where prevalence may be double than in the general population, a recent meta-analysis found depressive symptomology in around a third of patients. Furthermore, the presence of symptomology negatively affects self-care and is predictive of worse glycaemic control (Hood et al., 2006; Whittemore et al., 2002; Adal et al., 2015; Buchberger et al., 2016; Bernstein, Stockwell, Gallagher, Rosenthal & Soren, 2013). Studies have also found that the presence of depression in young people with T1D can negatively affect other aspects of development related to illness perceptions, illness functioning, and self-esteem (Oris et al., 2016). Some researchers have pointed out, however, that depression may be confused with Diabetes specific distress and that overdiagnosis is a significant problem (please see section 4.4 below). They have also suggested that levels of depression are much lower than reported in the research literature (Fisher et al., 2016). There is also conflicting evidence that higher depression levels do not predict poorer T1D management (Strandberg, Graue, Wentzel-Larsen, Peyrot & Rokne, 2014).

Depression is a common comorbidity in EDs in the non diabetic population also. (Braun, Sunday & Halmi, 1994; Lewinsohn, Striegel-Moore & Seeley 2000) in one study investigating the characteristics of female inpatients depression was found in nearly all of the patients (Blinder, Cumella & Sanathara, 2006). It is not entirely clear how depression affects Eating Disorder symptomology in those without T1D; some researchers have argued that it mediates the relationship between ED behaviours and body dissatisfaction (Brechan & Kvalem, 2015) while others have stated that the cultural overvaluation of thinness promotes

depression especially in young women, which then leaves them vulnerable to EDs (McCarthy, 1990). Depression has also been shown to directly predict binge and emotional eating (Brechan & Kvalem, 2015; Goossens, Braet, Van Vlierberghe & Mels, 2009). In adolescents with T1ED, less positive attitudes towards life, lower joy in life and higher depressive mood is found than in those without ED (Grylli et al., 2005). Longitudinal research has suggested that while ED and depression may co occur in younger individuals with T1, such symptoms are 'commonly but not universally associated' therefore this might be indicative of an underlying vulnerability (Colton et al., 2013, p. 375). Other research has found a link between depression and Eating Disorder onset in T1D (Olmsted et al., 2008). It should be noted that depression levels considered average or only slightly elevated, may be predictive, particularly in adolescent females of later eating issues (Olmsted et al., 2008). When investigating relationships between depression and T1ED it has been argued that while depression scores and ED symptomology are significantly positively related, for women at least this does not translate to a worsening of HbA1c (Bächle et al., 2015; Colton et al., 2013). Furthermore, there is some evidence that those without T1D related Eating Disorders may display higher levels of depression compared to those with T1ED (Powers, 2012) but this should be interpreted with caution due to sample size. In the pilot study (appendix E & F) depression was found in nearly half of the participants so it is assumed that depression will be a predictive factor in the structural model.

### 4.3.2   Anxiety

Anxiety encompasses a number of syndromes. In the DSM V these include phobias, social anxiety disorder, panic disorder, agoraphobia and generalised anxiety disorder. It should be noted that in DSM IV, Obsessive Compulsive Disorder (OCD) was also included under the anxiety label (APA, 2013) and the pilot study in which this model is based treated it as such. In the DSM V there is also an option for Unspecified Anxiety Disorder and research often looks at subsyndromal or subclinical representation (APA, 2013).

Research has shown that both clinical and subclinical presentations of anxiety disorders are significantly more present in adults with T1D than in the general population with Generalised Anxiety Disorder being the most frequently diagnosed (Grigsby, Anderson, Freedland

Clouse & Lustman, 2002). The presence of anxiety is related with decreased Diabetes control and elevated HbA1c (Friedman, Vila, Timsit, Boitard, & Mouren-Simeoni, 1998; Maia et al., 2014). Similar results have been found in adolescents (Herzer & Hood, 2009; Adal et al., 2015; Bernstein et al., 2013). A 2017 systematic review found similar results but added poorer self-care behaviours including BG testing, elevated depressive symptoms and fear of hypoglycaemia. The authors also found that state anxiety and trait anxiety affected Diabetes care differentially. Family conflict and suboptimal monitoring was better predicted by state anxiety and fear of negative Diabetes outcomes by trait anxiety (Rechenberg, Whittemore & Grey, 2017). In children and adolescents with T1D a similar pattern is found, a recent systematic review estimated that around a third of patients showed anxiety symptoms and that this was a risk factor for poor BG control (Buchberger et al., 2016). There is conflicting evidence, however, that suggests higher anxiety levels do not predict poorer T1D management (Strandberg et al., 2014)

Anxiety disorders are also highly comorbid with Eating Disorders in the Non T1D population (Braun et al., 1994). A 2004 study found elevated symptoms in both AN and BN and that anxiety disorders appear before the development of ED suggesting that such a diagnosis maybe a sign of vulnerability and that 'childhood anxiety represents one important genetically mediated pathway' to AN particularly (Kaye et al., 2004, p. 2215). In one study evaluating female ED inpatients anxiety was found in over half of the sample (Blinder et al., 2004). Several relationships between anxiety symptomology and EDs have been proposed in BN, binging and purging may be anxiolytic and body dissatisfaction may lead to significant social anxiety. In AN anxiety is apparent particularly around food and any perceived weight gain. (Bulik, 2002). There is a paucity of research relating to the relationship between T1ED and anxiety, but generally higher levels are seen (Takii et al., 2011). There is some evidence that drive for thinness and bulimic symptomology predict anxiety (Helgeson et al., 2007) and further evidence that shows that anxiety is predictive of weight preoccupation in subjects with T1ED (Pollock-BarZiv, & Davis, 2005). A study investigating momentary predictors of insulin omission also found that anxiety and nervousness before eating predicted insulin omission at the proceeding meal (Merwin et al., 2015). Although there is little research focusing on the relationship between anxiety and ED in T1Ds it has been suggested that treatment for T1ED lowers anxiety scores suggesting that

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there may be a comorbid relationship (Gagnon, Aimé, Bélanger & Markowitz, 2012). In the pilot study anxiety had been diagnosed in just under 30% of patients which is much higher than in the general population (Moran, Rooney, Tyrer & Coid, 2014). It is thus hypothesised that anxiety will be predictive of HbA1c and DEPS-R scores.

### 4.3.3    Borderline Personality Disorder

Borderline Personality Disorder (BPD) is a cluster B personality diagnosis in the DSM. There has been considerable debate about the validity of the diagnosis in recent years, but it survived the cull observed in the DSM 1VR personality disorders and is currently diagnosable under the DSM V. A BPD diagnosis is hallmarked by extreme emotional instability, an intense feeling of abandonment, suicide ideation and or self-harm and it may or may not be accompanied by dissociative episodes (APA, 2013). In the last adult psychiatric morbidity survey, 2.4% of 16 – 64 year olds screened positive for Borderline Personality Disorder (Moran et al., 2014).  There is a notable lack of published research on BPD in relation to T1D but what there is suggests that it hinders T1D management substantially. In a 2005 review Leichter & Dreelin discuss the substantive issues of treating these co morbid T1D and BPD patients.

> When seen in the health care setting, patients with BPD are often a challenge to provider organizations. They arouse intense emotional reactions from medical staff and others. They can pit care providers against each other. They often occupy substantial amounts of time and resources in frequently futile attempts to help them solve either their medical conditions or their recurrent dissatisfactions with the care process. And usually, but not always, they end their stormy course with a health care organization with a negative termination of the therapeutic relationship, complete with a vigorous litany of the many failings of the health care group. They are more apt to litigate against health care providers than are other patients. (Leichter & Dreelin, 2008, p. 101)

Although there is a scarcity of literature exploring the relationship between T1D and BPD, there is some evidence that cluster B personality disorders in general predict worse glycaemic control (Orlandini et al., 1997). There is more evidence linking BPD particularly to EDs in the general population and rates are reported at around 30%, (Herzog, Keller, Lavori, Kenny & Sacks, 1992; Sansone, Levitt & Sansone, 2004). In T1ED perhaps somewhat unsurprisingly there is also a lack of research into the relationship with BPD but in a rare

study Pollock-BarZiv & Davis (2005) found that borderline traits significantly predicted whether or not patients would engage in ED behaviours such as binge eating and insulin omission. They also highlight that given that self-harm is a symptom of BPD, in patients with both BPD and T1D overdose by insulin may be reported, and there may also be crossover between what is deemed to be ED behaviour and what is being driven by BPD (Pollock-BarZiv & Davis, 2005). In the pilot study, personality disorders were reported by around a fifth of participants of which the most common was borderline. The relationship between BPD and T1ED is unclear but should be considered in modelling ED behaviour given the high comorbidity.

### 4.3.4   Self-Esteem

Self-esteem broadly relates to how much value and worth one finds in oneself (Hewitt, 2009). There appears to be conflicting evidence regarding self-esteem among those with T1D. Some researchers have found lower levels of Self-esteem in T1D females compared to their non-D peers and a converse or no relationship with males, suggesting that male gender may be protective for these issues in T1D (Rassart, Luyckx, Moons & Weets, 2014; Kaminsky & Dewey, 2014). There is alternative evidence that there is no difference in self-esteem levels between those with and without T1 in both adults and children/ adolescents (Powers et al., 2013; Vlachioti et al., 2010).

This having been said longitudinal studies have provided some support for the idea that higher levels of self-esteem are predictive of better HbA1c levels and cross sectional research has produced similar findings (Luyckx & Seiffge-Krenke 2009; Zoffmann, Vistisen & Due-Christensen, 2014). In females particularly, it also seems that higher BMI is associated with lower levels of self-esteem in T1D (Kaminsky & Dewey, 2014). Self-esteem may also act as a mediating factor for treatment outcomes in this demographic (Jaser et al., 2013). Other authors have argued that there are aspects of self-esteem that are explicitly related to T1D and that T1D specific aspects of self-esteem predict BG control (Schneider et al., 2008). In those with T1ED Low self-esteem is especially related to weight and shape; this may be predictive of ED onset in T1D and should be seen as a clinical red flag (Olmsted et al., 2008; Racicka & Bryńska, 2015). In adolescents who have been diagnosed with ED and T1 lower

rates of self-esteem and social withdrawal are more commonly found than in those who do not have ED (Grylli et al., 2005). Similar results have been found in T1ED adolescents in relation to body image concerns with those with ED showing lower levels (Pinar et al., 2005), and this was particularly pronounced in female T1Ds. These issues are similarly found in the general ED population where socially, people with EDs appear to have higher sensitivity toward social stimuli compared to their non-ED counterparts. The authors suggest that they are more vulnerable to negative social comparison, which may have a significant relationship with self-esteem (Cardi, Di Matteo, Gilbert & Treasure, 2014). Lower self-esteem has been found to predict lower levels of recovery in those undergoing CBT treatment (La Mela, Maglietta, Lucarelli, Mori & Sassaroli, 2013).

Low self-esteem has been positively related to T1ED symptomology in adolescent samples (Colton, Olmsted, Daneman, Rydall & Rodin, 2007; Maharaj et al., 2003) and models of T1ED include low levels of self-esteem as a preceding factor (Treasure et al., 2015). It should be noted that even levels considered average or only slightly elevated of low self-esteem and weight and shape concern may be predictive, particularly in adolescent females of later eating issues (Olmsted et al., 2008). Lower levels of self-esteem were found to be important in the development of Eating Disorders in those participating in the pilot study and thus are included in the structural model.

### 4.3.5 Perfectionism

Hollender (1965) defined perfectionism as 'demanding of oneself or others a higher quality of performance than is required by the situation' (Hollender 1965, p.384). While there is not an extensive literature on perfectionism in T1D, Diabetes support group facilitators have reported that part of their job is to disseminate that 'perfectionism as neither possible nor desirable in self-management' (Costello, 2013, p. 178). Similarly, parents of T1D females state that combatting perfectionism in relation to disease management is a major challenge of dealing with their children (Mellin, Neumark-Sztainer & Patterson, 2004). Other measures of psychological functioning in T1Ds such as Diabetes specific distress (DSD) (please see section 4.4) are related to perfectionism with higher levels of distress coinciding with higher levels of perfectionism (Powers, Richter, Ackard & Craft, 2017). It is worth noting however

that there is conflicting evidence that perfectionism is not elevated in adolescents with T1D compared to their non-T1D peers (Sivertsen et al 2014).

For some authors, perfectionism is seen as preceding risk factor in T1ED (Treasure et al., 2015) and this is also seen in those with ED but no T1D, since early researchers such as Bruch, claimed that such patients are chasing 'super perfection' (Bruch, 1979, p. 56). Interventions for T1EDs have partly focussed on reducing perfectionism arguing that it is important for the maintenance of ED behaviour (Wilksch, Starkey, Gannoni, Kelly & Wade, 2013) and it has been shown that it is positively associated to attitudinal factors of Eating Disorder such as preoccupation with weight (Pollock-BarZiv & Davis, 2005). This is also seen in the general population (Stice, 2002). When looking at differences between patients who do and do not use inappropriate compensatory behaviours (ICB) such as insulin omission it has been found that perfectionism is higher among those who do not use ICB suggesting that perfectionists may be more concerned with blood sugar control (Takii et al., 2002). However, it may be the pressure of this perfectionistic attitude and the near impossible task of managing BG that pushes patients into T1ED. In her recent book Goebel-Fabbri interviewed 25 T1ED women who had experienced Diabulimia and states:

> Many (participants) described internalizing unrealistic, perfectionistic ideas about Diabetes management and giving up when they proved to be unattainable. They were self – critical and felt like they were failing at their health. (Goebel-Fabrri, 2017, p. 21)

Perfectionism was highlighted as important across several dimensions when asking participants what they considered relevant to the development of their EDs and thus may predict ED symptomology if modelled.

### 4.3.6   Family Functioning

A diagnosis of T1D can be a major familial event. There are major challenges to rearing a child with T1D and similar to other milestones, a developmental pathway for increasing autonomy has been suggested (please see table 1.1).  Age appropriate parental support promotes good Diabetes management, as does parental warmth. parental ability to adopt the child's perspective and higher levels of family cohesiveness (Helgeson et al., 2007; Davis et al., 2001; Mackey et al., 2011; Blicke et al., 2015). Conversely, conflict, restrictiveness and

premature passing of responsibility to the child is correlated with decreased self-efficacy, treatment adherence and worsened BG control (Anderson, 2012; Davis et al., 2001). This is the case regardless of developmental stage but problems may arise particularly around the time of puberty as insulin needs increase and BG is more difficult to control due to rising levels of hormones. A recent analysis of over 7000, 13–19 year-olds with T1D demonstrated that only around a fifth attained HbA1c level of < 7.5% (T1D Exchange Clinic Network, 2013) and conflict during adolescence has been shown to predict BG control longitudinally, demonstrating that family level effects are important across the life span (Hilliard et al., 2011). This is especially unfortunate as good BG control throughout adolescence is predictive of lesser levels of microvascular complications later in life, even if post adolescence this control is not maintained (Cleary, Dahms, Goldstein, Malone, & Tamborlane, 2001).

Conflict specifically around dietary regimen may lead to food related issues that would have been avoided without a T1 diagnosis. Furthermore, adolescents with T1D may avoid self-care behaviours, such as checking blood sugar, in order to limit such conflict (Hilliard et al., 2011). The family food environment also seems to be important for the development of T1ED. It may be understandable that parents would restrict certain foods in the house for their diabetic children, but the presence of such items predicts higher Eating Disorder symptomology as do low priority of family meals and less parental modelling of healthy eating (Alice Hsu, Chen, Huang, Lin & Lin, 2009; Caccavale et al., 2015). For those without T1D research has suggested that ED patients report on the existence of dysfunction more often than controls (Holtom-Viesel & Allan, 2014). Large scale registry studies have also shown that there is a higher risk for ED development in children (particularly daughters) of mothers who have experienced an ED themselves, potentially suggesting either a genetic effect or a replication of familial environment (Bould et al., 2015). Similarly disturbed parental eating attitudes and disturbed attachment profiles are associated longitudinally with disturbed eating behaviour while higher cohesion is protective in T1ED (Colton et al., 2007; Neumark-Sztainer et al., 2002). Maternal concern with weight and shape and dysfunctional mother daughter relationships are also predictive of higher Eating Disorder symptomology in adolescent T1D females. The authors suggest that as T1D carries a weight penalty these girls are even more vulnerable in an environment that espouses the thin ideal (Maharaj et al., 2003). Weaker attachments are also associated with the development of T1ED (Olmsted et

al., 2008). In the pilot study a latent variable constructed of items associated to family functioning was found to be important to the attributions participants made to ED development and as such it may be related to other variables in a model.

### 4.3.7 Summary

There are several psychosocial factors that are associated with both suboptimal blood sugar management and Eating Disorders in Type 1 Diabetes. These aspects were also reported in the pilot study preceding the current project (please see appendix E & F). It is also notable that these particular psychosocial variables are also associated with EDs in the general population. It is conceivable then that these variables may be representative of an underlying psychological vulnerability that can be modelled as a latent variable to predict both blood glucose levels and Eating Disorder symptomology.

### 4.4 Diabetes Specific Psychological Factors
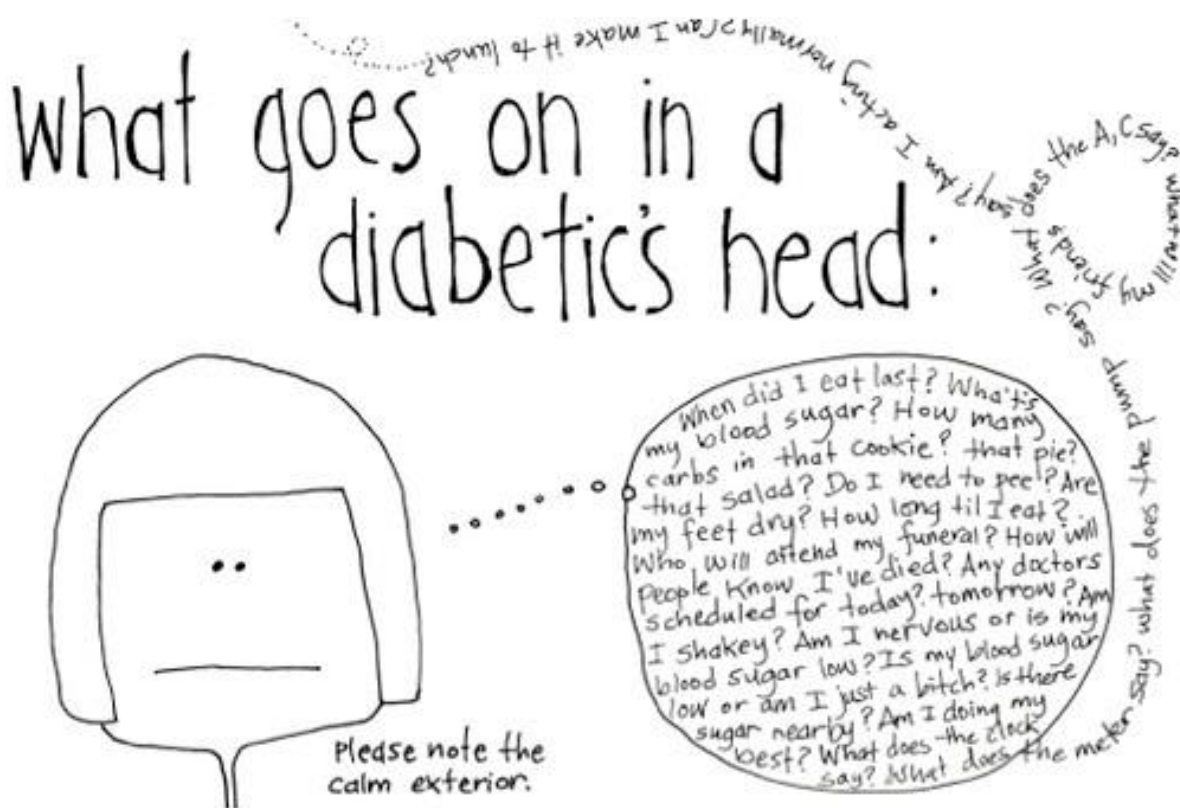


*Figure 4.1 Cartoon Reproduced with Permission of the Artist Haidee Merrit (2012)*

The above cartoon has become a popular meme in the Diabetes Online Community as it is representative of the multitude of complex and varied cognitions that may accompany a diagnosis of T1D. As such some researchers have proposed that there are Diabetes specific psychological factors that affect those with T1D. For example, Dr William Polonsky of the Behavioural Diabetes institute discusses Diabetes Burnout:

> …what happens when you feel over-whelmed by Diabetes and by the frustrating burden of Diabetes self care. People who have burned out realise that good Diabetes care is important for their health, but they just don't have the motivation to do it. At a fundamental level, they are at war with their Diabetes – and they are losing (Polonsky, 1999, p. 9)

Polonsky, on the proposition of Diabetes specific psychological burden proposed the Diabetes Distress Scale (DDS) This scale is divided into 4 aspects of Diabetes related life, Emotional Burden, Interpersonal Burden, Regimen Burden and Physician related Burden (Polonsky et al., 2005). These subscales reference Diabetes specific distress (DSD)

High levels of DDS are related to suboptimal control, particularly the regimen related distress that relates to the day-to-day mechanics of managing T1 (Strandberg et al., 2014; Strandberg et al., 2015). Importantly in several studies DDS operated as an independent predictor of HbA1c, regardless of other variables such as anxiety or depression (Strandberg et al., 2014). This suggests that DSD is a specific aspect of life unique to this population which is incomparable to controls. Any model attempting to explore relationships between psychological aspects of T1D should take in unique aspects of this population's experience and several DDS related items were highlighted in the pilot study as attributional (please see appendices E & F).

### 4.4.1 Regimen Related Distress

It is notable that dieting behaviour is a major risk factor for the development of EDs in the general population. Arguably a diagnosis of T1D requires a regimen that to the wider world may be considered dieting behaviour. Comparisons have been made between the prescribed advice given to those with T1D and their carers and the tell-tale signs of ED development. Rigidity, food group restriction, limiting common 'treats', timed meal and counting nutritional values share commonality with good Diabetes care (B-eat, 2018). Carbohydrate

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counting whereby a patient matches their insulin intake to the amount of carbohydrate they are consuming is the most recent advice for dietary T1D management, but a recent Brazilian study found that for some patients this practice led to obsessive behaviour and anxiety around issues such as portion sizes. The same study also found high levels of ED behaviour in those not utilising carbohydrate counting suggesting that this was due to the extended flexibility of the approach (Philippi et al., 2013). It appears then that dietary management in T1D regardless of the approach can lead to maladaptive behaviours and cognitions.

Other regimen issues can affect blood sugar control and ED behaviours. Unsurprisingly blood, needle or injury phobias have a significant impact on adherence to T1D regimen with those expressing injection anxiety citing this as a reason for insulin avoidance (Zambanini, Newson, Maisey & Feher, 1999). Patients are also much less likely to check their blood sugar as it involves pricking the fingertips with a small needle (Berlin et al., 1997; Babler & Strickland, 2015).

### 4.4.2 Emotional Burden of Diabetes

There are several aspects of T1D that contribute to emotional burden. A qualitative study by Browne and colleagues in 2014 demonstrated that those with T1D feel that they are stigmatised, the results of this study showed that association with T2 was the largest source of stigmatisation with patients going as far as advocating for a change of nomenclature. They state:

> Reasons for this included wanting to distance themselves from those with T2DM so as to avoid the negative judgements and stereotypes (e.g. 'fat', 'lazy', 'eat too much'), wanting people to understand the seriousness of T1DM as a health condition (Browne et al., 2014, p. 3).

Other themes included blame, negative social judgements, stereotyping, exclusion, rejection and discrimination. Notably, the sources of stigmatisation were far reaching as participants reported the media, family and friends, HCPs and teachers (Browne, Ventura, Mosely, & Speight, 2014). In Australia the Juvenile Diabetes Research Foundation (JDRF) released a 2014 report stating that 46% of participants they surveyed had experienced bullying or perceived social exclusion as a result of their condition, mostly due to misconceptions around

Type 1 Diabetes; namely that it is caused by overeating sugar, being fed too much sugar as a child or as a result of an unhealthy lifestyle (JDRF, 2014), which corresponds to the same misconceptions mentioned above.

This may partly account for findings that children with T1D may have issues with social confidence compared to healthy peers (Helgeson et al., 2007). It has also been found that adolescents with T1D may be vulnerable to peer pressure when adherence to T1D conflicts with perceived social acceptance (Thomas, Petterson & Goldstien, 1997).

### 4.4.3   Physician Related Distress

It is of note that those with T1D like those with other chronic conditions spend more time in hospital than the general population. T1Ds are supposed to have access to a multidisciplinary team of nurses, dietitians and consultants to help manage their condition and the quality *and* quantity of these relationships can be predictive of both successful and problematic Diabetes management (Findley, Cha, Wong & Faulkner, 2015; Care Quality Commission, 2014).

#### 4.4.3.1   *Paediatrics*

If a patient is diagnosed in childhood then they should attend a paediatric clinic with T1D specialists. This generally includes a consultant, specialist nurse, dietician and also potentially a psychologist (Findley et al., 2015). The UK has one of the worse paediatric HbA1c outcomes in Europe and the 2017 paediatric Diabetes audit found that the average HbA1c of those under 18 was 8.4% (RCPCH, 2017), higher than the NICE recommended maximum of 6.5% (NICE, 2014). One potential explanation for this is the clinic environment. Patients have reported feeling 'judged', ignored in favour of their parents, and fearful of potential conflict when attending clinic (Findley et al., 2015). Communication skills are perceived as lacking and consultants are particularly viewed as not being patient-centred and ignorant of the day-to-day stresses of living with and parenting for T1D. In general clinicians are seen as unable to offer the sort of emotional support needed (Lowes et al., 2015). The procedures in clinic may also within themselves be problematic, a 2011 analysis suggested that girls found the physical examinations such as weighing to be intrusive. The authors also highlight other issues:

The discussion groups with teenage and young girls voiced feelings of being a 'disappointment' to parents and healthcare professionals, particularly when blood glucose results were high or they had put on weight … Being measured (height and weight) in relatively public areas was particularly unpopular with teenage girls. (Hawthorne et al., 2011, p. 1104, 1106)

### *4.4.3.2   Transition*

Adolescence and emerging adulthood can be a turbulent time. Hormonal changes, weight changes and psychosocial factors can all adversely affect individuals. This is also well known as a risk period for adopting unsafe behaviours in areas such as sex, drugs and alcohol (Strang, Chein & Steinberg, 2013). Adolescents and Emerging Adults with T1D are not immune to these challenges and all of the above aspects also affect the ability to successfully manage the condition. Health care professional involvement at this time is extremely important but often fraught with issues as the needs of the patient change.

Transition describes a period where a patient is moving from pediatric services, which are often more intensive, into adult services. Some areas of the UK employ a specific clinic whereby young adults progress gently from one service into another, for other areas the change is abrupt and dictated by a cut-off age, it is broadly agreed that transition should occur at some point between the ages of 14 – 25 (Findley et al., 2015). A 2014 report by the Care Quality Commission found services for young adults in this stage of their illness to be woefully lacking.

> This report describes a health and social care system that is not working, that is letting down many desperately ill youngsters at a critical time in their lives. We have put the interests of a system that is no longer fit for purpose above the interests of the people it is supposed to serve. (Care Quality Commission, 2014 p. 1)

In T1D this lack of support can be devastating to long term outcomes.  A recent systematic review reported that many patients transitioning between paediatric and adolescent settings found the change distressing and the clinic environment cold. They also reported a lack of resources compared with the paediatric clinic, shorter appointment times and inattentive specialists. Moreover, those who chose as a result to receive community treatment by a GP

had even worse HbA1c levels (Findley et al., 2015). Support during the transition phase is shown to help adolescents 'normalise' their experience of their Diabetes (Babler & Strickland, 2015).

### 4.4.3.3 Adults

If transition is not handled appropriately there is a risk that younger adults will disengage with services altogether (White, O'Connell & Cameron, 2017). This group of people have been dubbed 'the lost tribe' by The Association of British Clinical Diabetologists (ABCD) They state:

> (there were) concerns that people with Type1 Diabetes had inadvertently been let down by wholesale system change, discharging people from secondary care clinics without providing robust support to primary care colleagues to manage this condition (ABCD, 2016, p. 1).

This lack of care means that there may be a sizable minority of those with T1D who are not seeing appropriate services and therefore may have no idea what their physical status is. A dedicated T1D consultant will monitor the risk of ongoing complications such as neuropathy, retinopathy and nephropathy, whereas ABCD warn that other health care professionals may not (ABCD, 2016).

### 4.4.4   Interpersonal Distress

As explained above there are several family level factors that affect Type1 Diabetes and Eating Disorders in Type1 Diabetes. Several other interpersonal relationships are potentially significantly affected by T1D, however. Peer relationships are understood to be important to development and a recent meta-analysis investigating such relationships in adolescents with T1D found that there are links between peers and self-care. The results of the analysis suggest that although the relationship is not clear there appears to be more support for peer conflict being harmful than peers support assisting with management (Palladino & Helgeson, 2012). This is potentially worrisome as although adolescent T1Ds report less peer support than controls they report similar levels of peer conflict. Furthermore, female T1Ds report less romantic support than controls although there appears to be no difference for males, higher

levels of romantic conflict are also associated with higher Eating Disorder behaviours in T1Ds. (Helgeson et al., 2014 a/b). It should be noted that there is conflicting research, which suggests that peer relationships have little effect of T1D related outcomes (Helgeson et al., 2013)

### 4.4.5   Consideration of Future Consequences

During a blog analysis co-authored by the researcher (please see appendix D) it was noted that when Diabulimia was mentioned in the press it was often accompanied by statements suggesting that sufferers had little concern regarding the future outcomes of their Eating Disorders. For example, an interviewee in a women's health magazine stated

> I didn't consider that the consequences could be deadly (Macellari, 2018, para. 10)

And in a metro interview, the bereaved family of a young T1ED stated

> She always thought it would never happen to her. She just didn't want to accept what she had – she was willing to take the risk… She thought she was invincible – she lived life her way. (Harley – Parkinson, 2018, para. 22)

This poses questions around whether or not there is a personality trait that is associated with T1ED in relation to whether sufferers acknowledge that the damage their ED is inflicting on their bodies will have proceeding ramifications. Consideration of future consequences (CFC) was proposed as a concept by Strathman, Gleicher, Boniger & Edwards in 1994 as 'a stable individual difference in the extent to which people consider distant versus immediate consequences of potential behaviors' (Strthman et al., 1994, p. 742) The authors argue that this trait can be measured on a continuum and that those who exhibit low levels are more concerned with immediate gratification than long term outcomes. Conversely those with higher levels are said to be willing to tolerate short term discomfort in pursuit of long term benefit. Given the significant long term consequences of T1D management which are highlighted often by HCPs this variable is a perhaps a novel predictor of HbA1c. If it also predicts DEPS-R scores then there are possible implications for the clinical treatment of T1ED.

### 4.4.6 Summary

The sub components that comprise Diabetes distress as measured by the Diabetes Distress Scale (Polonsky et al., 2005) are related to a number of poor outcomes for those with Type 1 Diabetes including those that are associated with Eating Disorders. Consideration of future consequences has also been suggested as a potential factor in those who engage in T1ED. Aspects relating to Diabetes specific distress were also suggested as attributional to the development of T1ED in the pilot study (please see appendix E & F). Diabetes specific distress should therefore predict both HbA1c as a measure of Diabetes control and Eating Disorder symptomology. Furthermore, the relationship between these two variables and consideration of future consequences should be investigated as this is something which has not been explored in other research.

### 4.5 The Current Study

Several variables were identified by the pilot study as associated with Eating Disorders in Type 1 Diabetes and the preceding literature review suggests further evidence that these issues are related. These variables could broadly be defined under the umbrella terms demographic, psychosocial and Diabetes specific distress. The current study therefore aims to model these variables as they are predictive of T1ED. In order to do this, it is first investigated whether the psychosocial aspects measured individually (depression, anxiety, borderline personality disorder, self-esteem, perfectionism & family functioning) relate to an underlying latent factor which could be described as general psychological functioning. Then the Diabetes Distress Scale is also factor analysed in order to show that the individual subscales related to an underlying factor. These latent factors are then treated as predictors of Eating Disorders and are hypothesised to predict both BG control and DEPS-R scores. Relationships with demographic factors such as gender are also explored in an alternative modeling fashion. As the role of the family is also seen to be important the relationship between family functioning and the latent variables are explored. The novel variable consideration of future consequences is also explored as a potential predictor of DEPS-R scores and HbA1c.

As discussed in chapters 2 and 3 measuring Eating Disorders in Type 1 Diabetes is complicated and there is no gold standard. The DEPS-R does appear to offer the most Diabetes specific symptomology and as such it has been chosen for the current study. There is also a lack of general agreement as to what level of insulin manipulation is clinically concerning as related to EDs and as such the current study accepts HbA1c as a variable related to insulin omission. Given how little is known about the nature of T1EDs the purpose of the current study is to provide an exploration of how these variables are related, whether they have any predictive value and if certain variables may act as mediatory.

# 5     Latent Variable Modelling

## 5.1    Methodology: Latent Variable Modelling
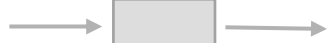
Given that Latent variable modelling has a number of conflicting recommendations and the proceeding chapters use the methodology, a brief review of the literature is given below.

## 5.2    Nomenclature and Graphic Representation

There is slightly different nomenclature used when generating latent and structural models than in other statistical methods. One key feature of the analysis is that relationships are often denoted by graphical representations whereby different classes of variables are presented using different standard forms.

*Table 5.1: Nomenclature and Graphic Representation used in Latent Variable Modelling.*

| Unit | Description | Graphic |
|---|---|---|
| Latent Variables, Latent Factors, f | Latent variables and other non-observed variables (i.e. error) are indicated by an ellipsis in the path diagram | |
| Errors, Residuals | Error/ Residuals are often denoted in brackets | () |
| Observed Variables, Indicators, y | Variables measured directly in the analysis and that are indicators of latent factors are in a rectangular box | |
| Uni Directional Arrows | Denotes a causal relationship in the form of a regression between one variable (or error) and another. It is important to note that in Factor Analysis variations in the latent trait predicts the relationship. The indicators themselves are not predictive. | |
| Double Headed Arrows | Indicates a relationship (i.e. correlation/ covariance) between variables. | |

| Unit | Description | Graphic |
|---|---|---|
| Independent Variable, Regressor, Predictor, Exogenous | A variable that emits an arrow. | |
| Dependent Variable, Predicted | A variable that receives an arrow, i.e. is regressed on. | |
| Endogenous Variable | A variable that is regressed on but can be dependent and independent depending on model type | |

## 5.3   Exploratory Factor Analysis (EFA)

There have been methodological arguments over the appropriateness of using exploratory factor analysis (EFA) to model data, with researchers highlighting that only in rare cases is there little knowledge of potential underlying structures. In the current data set however there are no a priori assumptions. The suggestions made as to why EDs may be more prevalent in the T1D population are speculative. Although there have been arguments made regarding the use of EFA there is also a general consensus that when the underlying pattern is poorly understood an exploratory procedure is valid (Gerbin & Hamilton, 1996, Byrne, 2012). Furthermore, EFA is often cited as the most common form of latent variable analysis in psychological research and is particularly useful for Likert data (Pohlmann, 2004).

### 5.3.1   Sample size

Sample size has been one of the most contentious issues in the Factor Analysis literature with most of the arguments focusing on 'rule of thumb' sample sizes ($N$) and item to $N$ ratio.  A Sample size of 100 is recommended by Gorsuch. (1982) and echoed by Kline. (1979), however other researchers have suggested a minimum sample of 200 (Guilford, 1954) or 250 (Cattell, 1978) while such a size is deemed simply 'fair', 300 'good', 500 'very good' and 1000 or more 'excellent' according to Comrey and Lee. (1992). In terms of ratios Everitt. (1975) argued that the item to $N$ should be at least 10:1. Gorsuch. (1983) halved that recommendation to 5:1 and Cattel. (1978) reduces this recommendation to at least 3:1. In

their review paper on sample size in factor analysis however MacCallum, Widaman, Zhang and Hong. (1999) state 'A fundamental misconception about this issue is that the minimum sample size, or the minimum ratio of sample size to the number of variables, is invariant across studies' (MacCallum et al., 1999, p.84). Accordingly most current conclusions on sample size appropriateness are made by assessing how strong the data is. Costello & Osborne. (2005) state that this can be measured by 'uniformly high communalities without cross loadings, plus several variables loading strongly on each factor' (Costello & Osborne, 2005 p. 4) and this sentiment is echoed by MacCallum et al. (1999) in their review. Strong data with those properties are widely understood to be able to withstand EFA regardless of sample size, $N$ between $100 - 200$ is adequate under the conditions above and some studies have found that this is the case even with samples under $N = 50$ (MacCalum et al., 1999; Jung, 2013). Furthermore, even under ideal conditions using a ratio of 20:1 EFA can produce high error rates (Costello & Osborne, 2005). Rather than using rules of thumb to determine whether of not sample size is sufficient there are two tests available in SPSS which asses the suitability of the data for factor analytic procedure. The Kaiser-Meyer-Olkin test measures how much variance in each variable maybe common and as such the lower the portion of common variance the more suitable the data. The test produces a statistic between 0 and 1 and Kaiser denoted the values as '0.00 to 0.49 unacceptable. 0.50 to 0.59 miserable. 0.60 to 0.69 mediocre. 0.70 to 0.79 middling. 0.80 to 0.89 meritorious. 0.90 to 1.00 marvellous'. (Kaiser 1974; Cerny & Kaiser 1977). Bartlett's test of sphericity tests the correlation matrix against the null hypothesis that no factors could explain the relationships, therefore a significant test $p<0.05$ suggests that there is an underlying factor structure and the data is suitable for analysis (Cramer & Howitt, 2004).

### 5.3.2   Rotation

Rotation of factors in EFA are generally divided into orthogonal and oblique solutions. Orthogonal rotation such as the most common method, the Varimax solution (Kaiser, 1958) assumes no correlation between factors. In oblique rotation, some level of correlation between items is expected and thus unique variance is separated from common variance. While historically orthogonal rotation has been used, as this solution offers the most easily interpretable output, more recently researchers have argued that as little in psychological

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sciences is unrelated oblique rotation is more theoretically sound (Browne, 2001; Costello & Osborne, 2005).

### 5.3.3 Extraction

Much debate has also surrounded the method of factor extraction. The most common methods in psychological research are Principle Components Analysis (PCA), Principle Axis Factoring (PAF) and Maximum Likelihood (ML). It should be noted that PCA is theoretically distinct from the other methods. PCA only seeks to explore the current sample data by reducing items into smaller components, thus summarising the data without taking measurement error into consideration and assessing the total variance in the sample. Factor analytic methods such as PAF and ML primarily utilise the communalities between items, assuming that variation in individual items scores are driven by an underlying variable not directly measurable and as such analyses the common variance within the sample (Haig, 2005; Matsunaga, 2010; McNiesh, 2015). Although PCA is the most common method reported (possibly because it is the default in SPSS and SAS) (Pohlmann, 2004) and some researchers argue that there is little difference between methods (Gerbin & Hamilton, 1996; Zhang & Preacher, 2015), factor analytic (FA) methods are widely believed to be more theoretically sound (Costello & Osborne, 2005). There are various methods of extraction under the FA umbrella of which the most common are Maximum Likelihood and Principle Factor Methods also called Principle Axis Factoring (PAF).

### 5.3.4 Factor Construction

Whether an item is retained as a component of a factor is determined by various properties namely, correlations with other items, communalities, factor loading and cross loading. Velicer and Fava, (1998) recommend that high communalities (the extent to which a variable is related to the other variables entered into the model) are those of 0.8 or greater but as Costello & Osborne, (2005) highlight this level of communality is rare and in social sciences communalities more often lie between 0.4 -0.7 with those that are 0.4 or below being considered low. MacCallum et al, (1999) suggest that with sample sizes between 100 – 200 a solid factor solution can be recovered as long as communalities are above 0.5 but recommend that the mean level of communality should be 0.7. In a later paper they also state

that high communalities can also help overcome sampling error that may be present in the data (MacCallum, Widman, Preaher & Hong, 2001). Factor loading can broadly be described as the strength of the relationship between the latent factor and the item. It follows from this that items that are strongly related with a factor are desirable and that others that are not may be screened out. An issue arises again as there is no general consensus as to what boundary levels should be used to define appropriate loading. Tabachnick and Fidell, (2001) propose .32 as a minimum loading. In a recent review the author states that a cut, off of 0.4 is liberal while 0.6 – 0.7 is conservative while concluding that 'there is a certain degree of judgement – call involved in this procedure' (Matsunaga, 2010, p. 101). Cross loading is another issue in item retention and it occurs when an item loads on more than one factor. Tabachnick and Fidell, (2001) consider an item to be cross loading if it has correlations on more than two or more factors but they also state that this is a 'rule of thumb'. A cross loading factor may be a good candidate for exclusion if there are higher loading items constituting the factor (Costello & Osborne, 2005). Alternatively, an approach that involves a first and second loading cut-off may be utilised, for example an item may be retained if it has a loading of 0.6 on it's primary factor and a loading of .4 or any other factor (Matsunaga, 2010) this may solve any potential issues with cross loading.

Again there is conflicting advice as to the exact number of items to retain in a factor. Byrne (2013) using the principle of overidentification suggests that a 3 item factor is under-identified a 4 item factor is just identified and a 5 item factor is the minimum required for an overidentified factor which is the ideal situation. Costello and Osborne, (2005) quantify this further by recommending that these 5 items should have a minimum factor loading of equal to or more than 0.5.

### 5.3.5 Dimensionality

There are various recommendations regarding how many factors should be retained post extraction and rotation. The most common are the Kaiser – Gutman rule which advocates keeping factors that have an eigen value over 1, however it has been argued that this over identifies factors (Matsunaga, 2010) and the scree test (Cattell, 1958, 1966) which involves looking for an inflection point whereby after there is an insignificant cluster of results. Those

above the inflection point are retained as factors. Regarding the Kaiser – Gutman rule Costello & Osborne, (2005), in their review of best practice in EFA, state that this method is seriously problematic claiming 'there is broad consensus in the literature that this is *among the least accurate methods'* (Costello & Osborne, 2005, p.2), although they then go on to recommend the scree test followed by multiple FAs using the suggested number of points above inflection and using the 'cleanest solution'. They then state is a factor solution 'with item loadings above 0.3, no or few cross loadings (and) no factors with fewer than three items' (Costello & Osborne, 2005, p.3). A newer more validated approach to factor retention uses Parallel analysis (Horn, 1965; Turner, 1998). Computer programmes run a monte carlo simulation with a predefined number of randomised data sets with similar parameters to the expected solution and show the eigen values expected when there is no latent structure assumed. Research suggests that this is the most accurate method (Henson & Roberts, 2006).

## 5.4 Confirmatory Factor Analysis (CFA)

Confirmatory factor analysis (CFA) is mainly used to confirm an a prioi hypothesised factor structure, often suggested by an EFA. Due to the nature of CFA a model is deemed appropriate if the predicted parameters fulfil a number of 'goodness of fit' criteria. Similar to EFA when the researcher judges which extraction and rotation methods are most appropriate for the sample, CFA also uses different techniques of parameter estimates that are data led.

### 5.4.1 Parameters of the measurement model

In confirmatory factor analysis MPLUS (Muthén & Muthén, 1998-2016) fits the data to a number of specified parameters in order to asses fit. These parameters are factor loadings (slope), for observed variables, intercepts of the observed variables, error variance of the observed variables and error covariance (Geiser, 2013).

### 5.4.2 Estimation

Maximum Likelihood (ML) is the most common estimation method in CFA especially for continuous data, it's also the default for most programmes such as Mplus (Muthén & Muthén 2010). However, there are assumptions that should be met such as normal distribution,

although it has been argued that even under non-ideal circumstances ML may be appropriate, there are also corrections that can be utilised in order to overcome non normality. In MPlus Satorra-Bentler corrections can be utilised using the MLM estimator however this procedure requires complete data. In the case of missing data however, Huber/Pseudo ML/sandwich corrections can be employed using the MLR estimator. MLR is recognised as the most robust method of parameter estimation when there are missing data which is either Missing at Random (MAR) or Missing Completely at Random (MCAR) and for this reason it is recommended when also dealing with non-normally distributed data (Rosseel, 2010; Byrne, 2012; Geiser, 2013; Muthén & Muthén, 1998-2016).

### 5.4.3 Goodness of Fit Statistics.

In CFA, unlike other statistical procedures the null hypothesis is desired as it predicts that the model fitted holds in the population to which the sample belongs. Thus, conversely to standard tests, insignificance is sought (p>0.05). Chi-square is one of the most common fit measurements used to test the null hypothesis and it is standard in most SEM programmes. MPlus (Muthén & Muthén, 1998-2016) provides a 'baseline model' whereby there is zero covariation between items, called the independence or null model and also provides a chi-square goodness of fit for that model. In essence, if the proposed model has a lower chi square than the baseline model then that represents a better fit. As chi-square is sensitive to sample size and non-normality some researchers have advocated for the use of the normed chi-square which is the chi-square value divided by the degrees of freedom. There are debates over which cut-off point to use for significance, Ullman (2001) proposes less than 2 as appropriate while Schumacker & Lomax. (2004) suggest under 5. However, as Byrne. (2012) highlights, particularly in relation to CFA and SEM, null hypothesis significance testing is particularly problematic, especially with non-normally distributed data and therefore further measures of fit are required.

There are two main types of fit indices outside the use of chi square, incremental, whereby fit is measured in comparison to a generated more restricted model, and absolute, where fit is measured by the specified model parameters being able to reproduce the sample data (Byrne 2012).

The Bentler Comparative Fit Index (CFI) is the most commonly used incremental fit statistic followed closely by the Tucker and Lewis Fit Index (TLI), these tests are similar in computation and interpretation with values closer to 1 being indicative of best fit (Byrne, 2012; Geiser 2010). As with other fit test there has been considerable debate as to an appropriate cut-off point, early research suggesting that values of over 0.9 should be considered good fit (Bentler, 1990) with this later being revised to 0.95 (Hu & Bentler 1995; Kline 1999).

Root Mean Square Error of Approximation (RMSEA) represents the most common absolute fit index. This test seeks to explain the difference between the proposed model and a model that perfectly fits the population data RMSEA = 0. Various boundaries have been suggested for good fit cut-off. Stieger (1990) argues that ideally RSMEA < 0.05. Absolute fit indices have been argued to over reject good models when the sample size used is small according to Hu and Bentler (1999) and as such they argue a less conservative value of 0.6 with some researchers stating that values of up to 0.8 (Browne and Cudek, 1993) may be acceptable or at least the upper confidence interval should not exceed that boundary (Hu & Bentler, 1995). Mplus (Muthén & Muthén, 1998-2016) also provides a significance test of the RMSEA whereby insignificance demonstrates that RMSEA is <0.05 in the population, this is termed 'closeness of fit' (Geiser, 2013). The Standardised Root Mean Square Error of Approximation (SRMSEA) is often used in conjunction with the RMSEA, it standardises the differences between the observed and predicted variances. Hu and Bentler. (1999) advise an upper boundary of 0.8 as indicative of good fit.

## 5.5  Structural Equation Modelling (SEM)

The confirmatory or exploratory factor models, called the measurement model in SEM represent the development and construction of latent factors. The structural model represents the relationship between, and potential predictive properties of, latent variables. The relationships in the model can be noted as paths, hence the use of 'path models'. Paths typically annotate directional regressions or covariances between variables (please see table 5.1). The SEM approach allows for further modelling by (among other things) allowing these latent factors to regress on to each other and also to act as predictors for other variables

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(observed and latent). Theoretically SEMs maybe used in a strictly confirmatory fashion, whereby the research is only fitting the model to data in an a priori fashion, Alternative modelling, where several theoretically guided assumptions are tested are used far more commonly in a model Generating way which attempts to find the model best fitted to the data (Joreskog, 1993).

### 5.5.1 Assumptions

As with any statistical testing procedure SEM also is optimal under a number of assumptions, namely that the data is normally distributed, mostly complete and independent. However even in datasets where assumptions are violated programmes such as MPLUS provide options to counteract non-normality such as MLR estimation (Muthén & Muthén, 1998-2016).

### 5.5.2 Model Identification

As with factor analysis the goal of a SEM is to describe an over identified model which is a model whereby there are more data points than there are parameters to be estimated thus leaving degrees of freedom that can be used to assess model fit statistically (Byrne, 2012). In SEM models the parameters estimated, excluding those for the measurement model, are variance for exogenous factors, factor means of exogenous (independent) factors, covariance in exogenous factors, latent path coefficients where the latent variable is also predictor, latent intercepts for endogenous factors, residual variances on latent endogenous factors, residual covariances for endogenous latent factors (Geiser 2013).

### 5.5.3 Sample Size

In general SEM is advised only for use in larger sample sizes. Kline (2005) argues that this is influences by contextual factors, i.e. complexity of the model and estimation technique but generally should be > 200. The original rule of thumb often followed by researchers is that there should be at least 10 subjects per parameter (Nunally, 1967). More recent arguments state that ratios should be applied to the number of free parameters in the model i.e. 5:1 (Bentler, 1989). Modern interpretations state that rules of thumb are not appropriate as sample size cannot be calculated via a linear function (Westland, 2010).

### 5.5.4 Latent Variable Scaling

Parameters for latent variables are typically specified using one of two methods. A reference item from each latent variable construction may have their loading (typically) constrained to 1 in order to translate that scale to the latent variable. Alternatively, the fixed factor approach may be utilised where by the variances of latent variables are constrained to 1 leaving all other parameters to be estimated freely (Byrne, 2012). Little et al. (2006) argue that latent variable scaling need not be arbitrary and provide a calculation to ensure this, but it can only be used where the indicator variables utilise the same response scale.

### 5.5.5 Goodness of Fit Statistics

The Fit statistics for SEM are the same as for CFA.

# 6 Methodology, Results & Discussion: Structural Equation Model

## 6.1 Methodology

### 6.1.1 Ethical Approval

Ethics approval for this study was granted by the Birkbeck departmental ethics committee on the 27th January 2014, approval number 131462.

### 6.1.2 Setting

Recruitment took place over the period of 04/05/2015 – 01/10/2015. An advert was posted on the social media pages of the registered charities: Diabetes UK, the Juvenile Diabetes Research Foundation, Diabetics with Eating Disorders (please see appendix H). The same advert was also posted on every Facebook page offering support and information to those with Type 1 Diabetes and the biggest online support forums for Type 1 Diabetes: Diabetes.co.uk, TuDiabetes and Diabetessupport.co.uk. A shorter version was also put on Twitter using the popular Diabetes hashtags #T1D, #DOC and #GBDOC. Participants were invited to share the advert on their social media pages and notify their Type1 friends/colleagues/health care professionals.

### 6.1.3 Procedure

If participants decided to take part they were taken to the website www.typeoneandpsychology.org where specially designed 'questioner' software was hosted. The website was built using Wordpress and the software was built in the python programming language. The resultant database utilised MySQL and was held on a private secure virtual server owned by the researcher. The landing page gave information about the study and asked for consent (please see appendix I) on selecting OK the participants were taken through to the Questioner programme. The Questioner programme was designed so that after the demographics questions were answered, if they chose, participants could create a user account that allowed them to sign in and out of the programme at their convenience as the study was long. The order in which each scale was presented was also randomised in order to counteract order effects. Following completion of the scales a second questionnaire

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appeared asking about the mental health history of the participant in relation to formal diagnoses made by a HCP, this was asked at the end in order to avoid biasing answers to the scales. They were then asked if they had ever felt that they had an Eating Disorder. If they selected yes they were taken to the next stage of the study (Chapter 7) and if they said no they were directed to the debrief screen (please see appendix J).

### 6.1.4 Participants

Eligibility criteria for the participants were being over the age of 16, having been diagnosed by a medical professional as having Type 1 Diabetes and having a good grasp of English. As the study was conducted online internet access was also essential this may have been via a computer/ table/ laptop/ smartphone. All of the participants fulfilled this criteria none were excluded from the final analysis.

### 6.1.5 Variables

#### 6.1.5.1 Demographic information

Age, gender, recruitment method, nationality, current country of residence and ethnicity was recorded.

#### 6.1.5.2 Medical Information

Age at diagnosis, duration of diagnosis, HbA1c, method of insulin administration, Contiguous Glucose Monitoring usage, T1D related complications, medications, other health conditions, mental health diagnoses, familial mental health and Eating Disorder status were recorded.

#### 6.1.5.3 Psychometric Variables

Depression: The Center of Epidemiological Studies Depression 10 Items was used (Andersen, Malmgren, Carter, & Patrick, 1994). This scale has shown good internal consistency (Cronbach's alpha = 0.86) and has been used in T1ED samples in other research (Powers et al., 2017). Responses are given on a 4-point Likert scale from 0=rarely or none of the time, to 3=all of the time, with a minimum score of 0 and a maximum score of 30

indicating higher levels of depression. Items include 'I was bothered by things that usually don't bother me.'

Anxiety: the Generalised Anxiety Disorder 7 Item scale was used (Spitzer, Kroenke, Williams & Löwe, 2006). This scale demonstrates good internal validity (Cronbach's alpha = 0.92) and has been utilised in other studies researching T1D (Löwe, Decker, Müller, Brähler, Schellberg, Herzog, & Herzberg, 2008). Responses are given on a 4-point Likert scale from 0=not at all to 3=nearly every day. This renders a minimum score of 0 and a maximum score of 21. Higher scores indicate higher levels of anxiety. Items include 'Being so restless that it is hard to sit still'.

Borderline Personality traits: The Mclean Screening Instrument for Borderline Personality Disorder (MSI-BPD) was used (Zanarini et al., 2003). This scale demonstrates good internal validity (Cronbach's alpha = 0.74) and has been used for screening (Chanen et al., 2008). The scale consists of 10 yes/no questions with a minimum score of 0 and a maximum of 10, higher scores indicate more borderline personality traits. Items include 'Have you deliberately hurt yourself physically (e.g., punched yourself, cut yourself, burned yourself)? How about made a suicide attempt?'

In order to assess perfectionism, the Almost Perfect Scale Revised was used (Slaney, Rice, Mobley, Trippi & Ashby 2001). This scale consists of 3 subscales which have all shown good internal validity: Order (Cronbach's alpha= 0.86); Standards (Cronbach's alpha = 0.85); and Discrepancy (Cronbach's alpha = 0.92). It has been used in Eating Disorder samples (Paulson & Rutledge, 2014). The discrepancy scale consists of 12 questions, order 4 and standards 7. All of these utilise a 7-point Likert scale ranging from 1=strongly disagree to 7=strongly agree. The minimum score for the scale is 23 and the maximum is 161 with higher scores being indicative of higher levels of perfectionism. Items include 'I often worry about not measuring up to my own expectations.', 'If you don't expect much out of yourself, you will never succeed.' and 'I like to always be organized and disciplined.'

In order to measure Diabetes related distress, the Diabetes Distress Scale (DDS) was used (Polonsky, et al., 2005). This questionnaire consists of 4 subscales: Emotional Burden (5 questions); Physician Related Distress (4 questions); Regimen Related Distress (5 questions)

and Interpersonal distress (3 questions). The scale shows good internal validity (Cronbach's alpha = 0.87). The 17 questions that constitute the scale are measured on a 6-point Likert scale ranging from 1= not a problem to 6= A very serious problem. The minimum score for this questionnaire is 17 and the maximum is 102.  Higher scores indicate higher levels of Diabetes related distress. Items include 'Feeling that I will end up with serious long-term complications, no matter what I do.', 'Feeling that I don't have a doctor who I can see regularly enough about my Diabetes.', 'Not feeling motivated to keep up my Diabetes self-management.' and 'Feeling that friends or family don't give me the emotional support that I would like.'

Consideration of Future Consequences (CFC): In order to measure how concerned participants were regarding their disease outcomes the Consideration of Future Consequences questionnaire was used (Strathman, Gleicher, Boninger & Edwards, 1994). This scale has shown good internal validity (Cronbach's alpha = 0.8) and has been used in studies investigating health related outcomes (Von Wagner, Good, Whitaker, & Wardle, 2011). This scale constitutes 12 questions with a minimum score of 12 and a maximum of 60, rated on a 5-point Likert scale, with 1=extremely uncharacteristic to 5=extremely characteristic. Some of the items were reverse scored.  Higher scores indicate higher levels of consideration of future consequences. Items include 'I think it is more important to perform a behaviour with important distant consequences than a behaviour with less-important immediate consequences.'

Family Functioning: The Family Adaptability, Cohesion Evaluation Scale (FACES) 3 (family version) was used; this measures 2 subscales (Olson, 1985). Adaptability (Cronbach's alpha = 0.62) and Cohesion (Cronbach's alpha = 0.77) and has been utilised in a number of studies in health psychology (Kouneski, 2000).  Each of these subscales contain 10 items and utilise a 5-point Likert scale going from 1=almost never to 6=almost always. The minimum score is 10 and the maximum is 50. Higher scores represent higher family functioning. Items include 'Children have a say in their discipline' and 'Family members like to spend free time with each other'.

Self-esteem: the Rosenberg self-esteem scale (RES) was used (Rosenberg, 1965). This has shown good internal reliability (Cronbach's alpha = 0.88) and has been used in a number of similar studies (Baumeister, Campbell, Krueger & Vohs, 2003). The scale is measured over 10 questions on a 4-point Likert scale from 0=strongly disagree to 3=strongly agree. Some of the items are reverse scored and participants can receive a minimum of 0 and a maximum of 30. Higher scores indicate higher levels of self-esteem. Items include 'I feel that I'm a person of worth, at least on an equal plane with others.'

In order to assess Eating Disorder symptomology the Diabetes Eating Problem Scale Revised was used (Antisdel, Laffel & Anderson 2001). This scale has demonstrated high internal reliability (Cronbach's alpha 0.86) and has been validated in other related studies (Wisting et al 2013 a/b). 16 questions constitute the scale and they are scored on a 6-point Likert scale from 0=never to 5=always. The minimum score is 0 and the maximum is 80 with scores over 21 deemed clinically concerning. Items include 'I feel that it's difficult to lose weight and control my Diabetes at the same time'.

In order to compare Diabetes related Eating Disorder symptoms to those found in the general population the Eating Attitudes Test 26 was used (please see chapter 3).

### 6.1.5.4 Variables for Analysis

The main outcome variables of interests were HbA1c as self-reported by the participants from their last medical appointment and DEPS-R scores as a measure of Eating Disorder symptomology, with all other variables being examined as possible predictors/ mediators.

## 6.2 Results

### 6.2.1 Overview of Analysis

Firstly, the sample characteristics were described in terms of demographics, medical details and mental health information. Following this the descriptive data, distribution, proportion of missing data and scale alphas of the potential variables for structural modelling as identified by the pilot study and the literature review were reported. Relationships between the variables for structural modelling and the main outcome variables DEPS-R scores and

HBA1C levels were then explored via Pearson's correlation coefficients and significant associations selected.

The hypothesised variables relating to psychological functioning and Diabetes specific distress were then factor analysed to ascertain whether there were underlying latent variables to account for variations in scale responses. Latent factors were identified; 'Psychological functioning' comprising of anxiety, depression, borderline traits, maladaptive perfectionism and self esteem scores and 'Diabetes specific distress' comprising of Emotional Burden, Physician Related Distress and Interpersonal Distress. These latent variables were then explored in SEM by using them as predictors of HbA1c and DEPS-R scores. As suggested by the literature gender was also entered into the model. Following this the relationship with family functioning and the latent variables was explored via regression and mediation analysis. An alternative model was considered in order to ascertain if the novel variable 'Consideration of Future Consequences' predicted HbA1c and DEPRS scores. A further alternative model was then considered by regressing 'Age' onto the outcome variables also.

### 6.2.2 Software

All data was processed and analysed using IBM SPSS version 23 (IBM Corp. 2016) and Mplus version 8 (Muthén & Muthén 1998-2016).

### 6.2.3 Sample Characteristics

#### 6.2.3.1 Initial Numbers

687 participants started the questionnaire having being recruited mostly by social media (610/ 88.8%). All participants completed the gender question. 92 (13.4%) were male, 592 (86.2%) were female, 2 were transgendered (0.3%) and 1 selected prefer not to say (0.15%). 686 participants gave their age. They had mean age of 33.73 (12.01) years (range:16 – 69). Many nationalities were reported but most were UK/ Eire and US nationals living in those countries all participants answered these questions and the ethnicity question, the majority (599/ 87.2%) were white and a large minority selected prefer not to say (81/ 11.8%) (for the full characteristics of the participants please see appendix K).

### 6.2.3.2  Medical Status

686 participants reported a mean age at diagnosis of 15.48 (11.58) years (range: $0 - 61$) and a mean T1D duration of 18.25 (11.98) years (range: $0 - 59$). 659 participants reported a mean HbA1c% of 8 (2.24, range: $4 - 29\%$). 530 participants reported their methods of blood sugar management. 319 (60.2%) utilised a pump, 200 (37.7%) were on multiple daily injections, 4 (0.75%) used a mixtard[11], 6 (1.13%) used syringes and vials and 1 (0.19%) used inhaled insulin. 222 (41.89%) of these patients also utilised a continuous glucose measurement device. 684 participants reported a mean of 0.67 (1.12) Diabetes related complications.  The most commonly reported were Retinopathy and other eye issues (Macular Oedema, Cataracts and Glaucoma) (162/ 23.7%), Neuropathy (101/ 14.7%), Gastroparesis (47/ 6.9%) and Nephropathy (36/ 5.3%). The 684 participants were taking an average of 0.23 (0.84) medications to manage these conditions. Of the 687 asked, 325 (47.3%) participants reported having more than Diabetes as a medical diagnosis. Of these the most common conditions experienced were Hypothyroidism (95/ 13.8%), Asthma (56/ 8.15%) and Coeliac Disease (23/ 3.3%) (for full participant medical status please see appendix K).

### 6.2.3.3  Participants Mental Health Information

439 participants answered the question 'have you ever had a mental health diagnosis (except an Eating Disorder diagnosis)' 193 (44%) answered yes so participants had a mean of 0.678 (.913). Of those who did report other diagnoses the most common were depression (155/ 35.3%), anxiety (79/ 18%) borderline personality disorder (14/ 3.2%), bipolar disorder (10/ 2.3%), and post-traumatic stress disorder (10/ 2.3%) (for full participant mental health characteristics please see appendix K).  Participants who had reported having another mental health diagnosis had a mean of 1.52 (0.78 min 1 – max 5) conditions. 435 participants answered the question 'to the best of your knowledge does anyone in your family have a mental health diagnosis?'. Of them 217 (49.9%) answered yes, 173 (39.7%) answered no and

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[11] A mixtard is an injectable form of insulin that contains both long acting and short acting insulin.

45 (10.3%) stated that they weren't sure. 435 participants answered the question 'to your knowledge has anyone in your family ever had an Eating Disorder?' of these 60 (13.8%) answered yes, 354 (81.4%) answered no and 21 (4.8%) stated that they weren't sure. 439 participants answered the question 'have you ever been diagnosed with or thought that you had an Eating Disorder' of these 130 (29.9%) answered yes. However, of the 491 participants who completed the DEPS-R questionnaire 217 (44.2%) screened positive (a score of 20 or above) for further clinical investigation.

### 6.2.4 Main Outcome Variables and Potential Predictors/ Mediators for Structural Modelling

#### 6.2.4.1 Missing Data

The data was missing completely at random (MCAR) Littles test Chi-Square = 2901.456, df = 3356 p =1) as such pairwise deletion was used for the correlation analysis, confirmatory factor analysis and structural equation model. Please see table 6.1 below for the distribution of missing data.

#### 6.2.4.2 Distribution

The Distribution of some variables showed non normality, particularly HbA1c which had a kurtosis of 22.03, however this is to be expected given that the normal range for blood sugar is between 4 – 8 mmols. Furthermore, MPLUS provides an MLR estimation that is robust to non normality (Muthén & Muthén, 1998-2016). Please see table 6.1 below for distribution statistics

#### 6.2.4.3 Descriptive Statistics, Distribution Statistics, Missing Data and Scale Alphas for Variables of Interest in the Sample

*Table 6.1: Descriptive Statistics, Distribution Statistics, Missing Data and Scale Alphas for Variables of Interest in the Sample*

| | n | Mean | Std. Deviation | Skew | SE | Kurtosis | SE | Missing Count | Missing Percent | Scale Alpha |
|---|---|---|---|---|---|---|---|---|---|---|
| Outcome Variables | | | | | | | | | | |
| HbA1c | 659 | 8 | 2.24 | 3.427 | 0.095 | 22.03 | 0.19 | 28 | 4.1 | |
| DEPS-R Scores | 491 | 23.19 | 17.25 | 1.227 | 0.11 | 0.976 | 0.22 | 196 | 28.5 | 0.936 |
| Psychosocial Variables | | | | | | | | | | |
| Depression | 505 | 11.78 | 7.06 | 0.5 | 0.109 | -0.424 | 0.217 | 182 | 26.5 | 0.866 |
| Borderline Traits | 468 | 4.36 | 3.21 | 0.486 | 0.113 | 0.026 | 0.225 | 219 | 31.9 | 0.831 |
| Generalised Anxiety | 512 | 8.74 | 6.03 | 0.425 | 0.108 | -0.88 | 0.215 | 175 | 25.5 | 0.929 |
| Perfectionism (Standards) | 497 | 40.64 | 7.15 | -1.532 | 0.11 | 3.321 | 0.219 | 190 | 27.7 | 0.872 |
| Perfectionism (Order) | 501 | 20.59 | 5.21 | -0.705 | 0.109 | 0.014 | 0.218 | 186 | 27.1 | 0.862 |
| Maladaptive Perfectionism | 496 | 54.22 | 18.37 | -0.248 | 0.11 | -0.944 | 0.219 | 191 | 27.8 | 0.96 |
| Self Esteem | 488 | 17.39 | 7.3 | -0.202 | 0.111 | -0.673 | 0.221 | 199 | 29 | 0.938 |
| Consideration of Future Consequences | 480 | 40.94 | 8.81 | -0.409 | 0.111 | -0.195 | 0.222 | 207 | 30.1 | 0.869 |
| Family Cohesion | 471 | 35.48 | 8.51 | -0.661 | 0.113 | -0.092 | 0.225 | 216 | 31.4 | 0.897 |
| Family Adaptability | 411 | 23.88 | 6.3 | 0.254 | 0.12 | 0.491 | 0.24 | 276 | 40.2 | 0.777 |
| Diabetes Specific Distress | | | | | | | | | | |
| Diabetes Emotional Burden | 504 | 18.47 | 7.35 | -0.049 | 0.109 | -1.17 | 0.217 | 183 | 26.6 | 0.92 |
| Diabetes Physician Related Distress | 498 | 8.91 | 5.41 | 1.059 | 0.109 | 0.13 | 0.218 | 189 | 27.5 | 0.87 |
| Diabetes Regimen Related Distress | 504 | 14.94 | 7.38 | 0.525 | 0.109 | -0.879 | 0.217 | 183 | 26.6 | 0.89 |
| Diabetes Interpersonal Burden | 503 | 9.26 | 4.51 | 0.396 | 0.109 | -0.963 | 0.217 | 184 | 26.8 | 0.855 |
| Demographic Variables | | | | | | | | | | |
| Age | 686 | 33.73 | 12.01 | 0.818 | 0.093 | -0.025 | 0.186 | 1 | 0.1 | |
| Diagnosis Age | 686 | 15.48 | 11.58 | 1.412 | 0.093 | 2.05 | 0.186 | 1 | 0.1 | |
| Diabetes Duration | 686 | 18.25 | 11.98 | 0.734 | 0.093 | 0.097 | 0.186 | 1 | 0.1 | |

### 6.2.5 Relationships between Psychosocial, Diabetes Specific and Demographic variables and HbA1c and DEPS-R scores

Pearson's bivariate correlation tests were run on all of the relevant variables (please see appendix M for the full correlation matrix). Those who were younger, had been diagnosed at an earlier age and had a lesser duration of Diabetes had higher DEPS-R scores. Higher levels of anxiety, Diabetes specific distress (all subscales), maladaptive perfectionism, borderline personality traits, depression and lower levels of consideration of future consequences and self-esteem were all correlated with higher Eating problem symptomology. Perfectionism (standards and order) were not significantly related. Higher levels of family cohesion were related with lower DEPS-R scores but family adaptability was not significantly correlated.

Those who were younger and had a shorter duration of Diabetes had higher HbA1c levels but there was no significant correlation with age at diagnosis. Higher levels of anxiety, Diabetes specific distress (all subscales), maladaptive perfectionism, borderline personality traits, depression and lower levels of consideration of future consequences and self-esteem were all correlated with higher HbA1c levels. Higher standards (perfectionism) was related with lower levels but order (perfectionism) was not. Family variables were not significantly related to HbA1c levels.

The two dependent variables DEPS-R scores and HbA1c levels were significantly correlated

*Table 6.2: Correlations between Outcome Variables and Potential Predictors/ Mediators*

|  | HbA1c | | | DEPS-R Scores | | |
|---|---|---|---|---|---|---|
|  | n | r | p | n | r | p |
| Outcome Variables |  |  |  |  |  |  |
| Eating Problem Symptomology (DEPS-R Scores) | 476 | .54$^{**}$ | <0.01 |  |  |  |
| HbA1c |  |  |  | 476 | .54$^{**}$ | 0 |
| Psychosocial Variables |  |  |  |  |  |  |
| Self Esteem Scores | 469 | -.35$^{**}$ | <0.01 | 437 | -.60$^{**}$ | <0.01 |
| Generalised Anxiety Scores | 495 | .32$^{**}$ | <0.01 | 461 | .49$^{**}$ | <0.01 |
| Depression Scores | 487 | .33$^{**}$ | <0.01 | 460 | .51$^{**}$ | <0.01 |
| Perfectionism Discrepancy Scores | 478 | .26$^{**}$ | <0.01 | 450 | .51$^{**}$ | <0.01 |
| Perfectionism (standards) Scores | 479 | -.11$^{*}$ | 0.02 | 452 | -0.03 | 0.52 |
| Perfectionism (order) Scores | 483 | -0.02 | 0.69 | 452 | -0.06 | 0.24 |

| | HbA1c | | | DEPS-R Scores | | |
|---|---|---|---|---|---|---|
| Consideration of Future Consequences Scores | 464 | -.29** | <0.01 | 435 | -.45** | <0.01 |
| Borderline Personality Scores | 452 | .35** | <0.01 | 421 | .56** | <0.01 |
| Family Cohesion Scores | 455 | 0.08 | -0.08 | 425 | -.21** | <0.01 |
| Family Adaptability Scores | 399 | 0.07 | 0.18 | 371 | -0.06 | 0.26 |
| Diabetes Specific Distress | | | | | | |
| Emotional Burden of Diabetes Scores | 484 | .37** | <0.01 | 460 | .56** | <0.01 |
| Physician Related Burden Scores | 479 | .21** | <0.01 | 453 | .29** | <0.01 |
| Diabetes Regimen Burden Scores | 485 | .49** | <0.01 | 459 | .74** | <0.01 |
| Interpersonal Distress due to Diabetes Scores | 483 | .26** | <0.01 | 459 | .42** | <0.01 |
| Demographic Variables | | | | | | |
| Age | 658 | -.18** | <0.01 | 490 | -.32** | <0.01 |
| Age at Diagnosis | 658 | -0.06 | 0.14 | 490 | -.16** | <0.01 |
| Diabetes Duration (years) | 658 | -.13** | <0.01 | 490 | -.17** | <0.01 |

*Significant <0.05, **Significant <0.01

## 6.3  Structural Equation Model

### 6.3.1  Factor Analysis

As proposed the psychological variables (anxiety, depression, borderline traits, maladaptive perfectionism and self-esteem) were added to the model and regressed on the latent trait 'Psychological Functioning'. The variables all had factor loadings > 0.7 (please see table 6.3). The 3 scales of the Diabetes Distress Scale 'Emotional Burden', 'Physician Related Distress' and 'Interpersonal Distress' were then regressed onto the latent trait 'Diabetes specific distress' and all variables had loadings > .5. As there was a .74 correlation between DEPS-R scores and Regimen Related Distress this variable was not entered into the model due to potential issues with collinearity (please see discussion).  This revealed a relatively well-fitting model: $x^2$ (19) =82.203, p< 0.000, RMSEA = 0.074, CFI = 0.962, TLI = 0.945. In order to assess whether a better fit was possible, modification indices were consulted, which suggested allowing self-esteem to correlate with both maladaptive perfectionism and general anxiety scores. This yielded a better fitting model: $x^2$ (17) =29.770, p< 0.0281, RMSEA = 0.035, CFI = 0.992, TLI = 0.987.

*Table 6.3: Latent Factor Construction (standardised)*

| | Estimate | S.E. | P-Value | 95% CI Lower 2.5% | 95% Upper 2.5% | R square |
|---|---|---|---|---|---|---|
| Psychological Functioning | | | | | | |
| Anxiety | 0.823 | 0.022 | <0.001 | 0.779 | 0.867 | 0.678 |
| Self Esteem | -0.844 | 0.02 | <0.001 | -0.884 | -0.804 | 0.712 |
| Maladaptive Perfectionism | 0.712 | 0.026 | <0.001 | 0.661 | 0.764 | 0.507 |
| Borderline Traits | 0.741 | 0.029 | <0.001 | 0.684 | 0.799 | 0.55 |
| Depression | 0.871 | 0.014 | <0.001 | 0.844 | 0.897 | 0.758 |
| Diabetes Distress | | | | | | |
| Emotional Burden | 0.876 | 0.021 | <0.001 | 0.835 | 0.918 | 0.768 |
| Physician Burden | 0.526 | 0.04 | <0.001 | 0.447 | 0.604 | 0.276 |
| Interpersonal Distress | 0.703 | 0.03 | <0.001 | 0.644 | 0.763 | 0.495 |

*Figure 6.1: Construction of Latent Factors 'Psychological Functioning' and 'Diabetes Specific Distress' (standardised)*
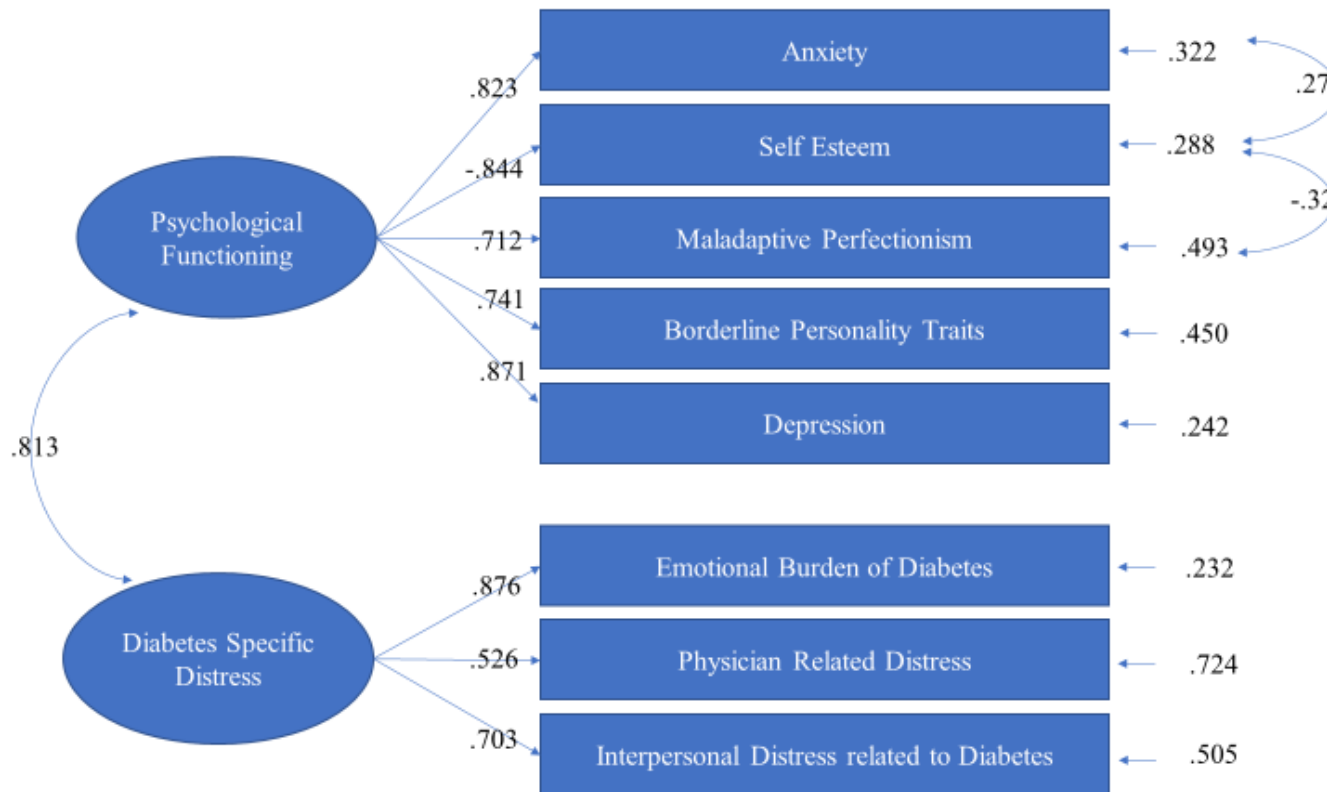


### 6.3.2 Structural Model One

In order to predict the role of psychological functioning (PF) and Diabetes specific distress (DSD) on BG control and Eating Disorder symptomology a full structural model was specified. PF and DDS were regressed on HbA1c and DEPS-R while controlling for gender as the literature suggests that women are at higher risk for both EDs and poor control and a well-fitting model was revealed ($x^2$ (37) =90.873, p< 0.000, RMSEA = 0.046, CFI = 0.975, TLI = 0.963).

The results suggest that psychological functioning predicted HBA1C  (β=0.193 SE=0.030, 95% CI = 0.030, 0.357) and DESPR (β=0.431 SE=0.082, 95% CI = 0.271, 0.590). As suggested by the literature higher levels of impaired psychological functioning predicted higher HBA1C levels and DESPR scores. Similarly, higher levels of diabetic specific distress

predicted increased levels of HBA1C (β=0.245 SE=0.091, 95% CI = 0.066, 0.424) and DESPR (β=0.261 SE=0.081, 95% CI = 0.106, 0.424). Gender predicted DESPR (β=0.220 SE=0.094, 95% CI = 0.035, 0.406), but did not predict HBA1C. This suggest that women are .20 of a standard deviation more likely than men to experience Eating Disorder symptomatology, but that diabetic specific distress was not associated with gender. Overall the model explained 17.4% of variance in HBA1C and 44.6% of variance in DESPR.

*Table 6.4: SEM Model 1 Construction (standardised)*

|  | Estimate | S.E. E | P-Value | 95% CI Lower 2.5% | 95% CI Upper 2.5% |
|---|---|---|---|---|---|
| Psychological Functioning |  |  |  |  |  |
| Anxiety | 0.815 | 0.022 | <0.001 | 0.771 | 0.859 |
| Self Esteem | -0.854 | 0.02 | <0.001 | -0.893 | -0.814 |
| Maladaptive Perfectionism | 0.717 | 0.027 | <0.001 | 0.665 | 0.769 |
| Borderline Traits | 0.749 | 0.029 | <0.001 | 0.692 | 0.806 |
| Depression | 0.861 | 0.014 | <0.001 | 0.834 | 0.888 |
| Diabetes Distress |  |  |  |  |  |
| Emotional Burden | 0.882 | 0.021 | <0.001 | 0.841 | 0.923 |
| Physician Burden | 0.517 | 0.04 | <0.001 | 0.438 | 0.595 |
| Interpersonal Distress | 0.697 | 0.031 | <0.001 | 0.637 | 0.756 |
| HBA1C |  |  |  |  |  |
| Psychological functioning | 0.193 | 0.083 | 0.02 | 0.03 | 0.357 |
| Diabetes Specific Distress | 0.245 | 0.091 | 0.007 | 0.066 | 0.424 |
| DEPS-R |  |  |  |  |  |
| Psychological functioning | 0.431 | 0.082 | <0.001 | 0.271 | 0.59 |
| Diabetes Specific Distress | 0.265 | 0.081 | 0.001 | 0.106 | 0.424 |
| HBA1C |  |  |  |  |  |
| Gender | 0.017 | 0.091 | 0.852 | -0.161 | 0.195 |
| DEPS-R |  |  |  |  |  |
| Gender | 0.22 | 0.094 | 0.02 | 0.035 | 0.406 |

*Figure 6.2: Structural Model 1 (standardised)*
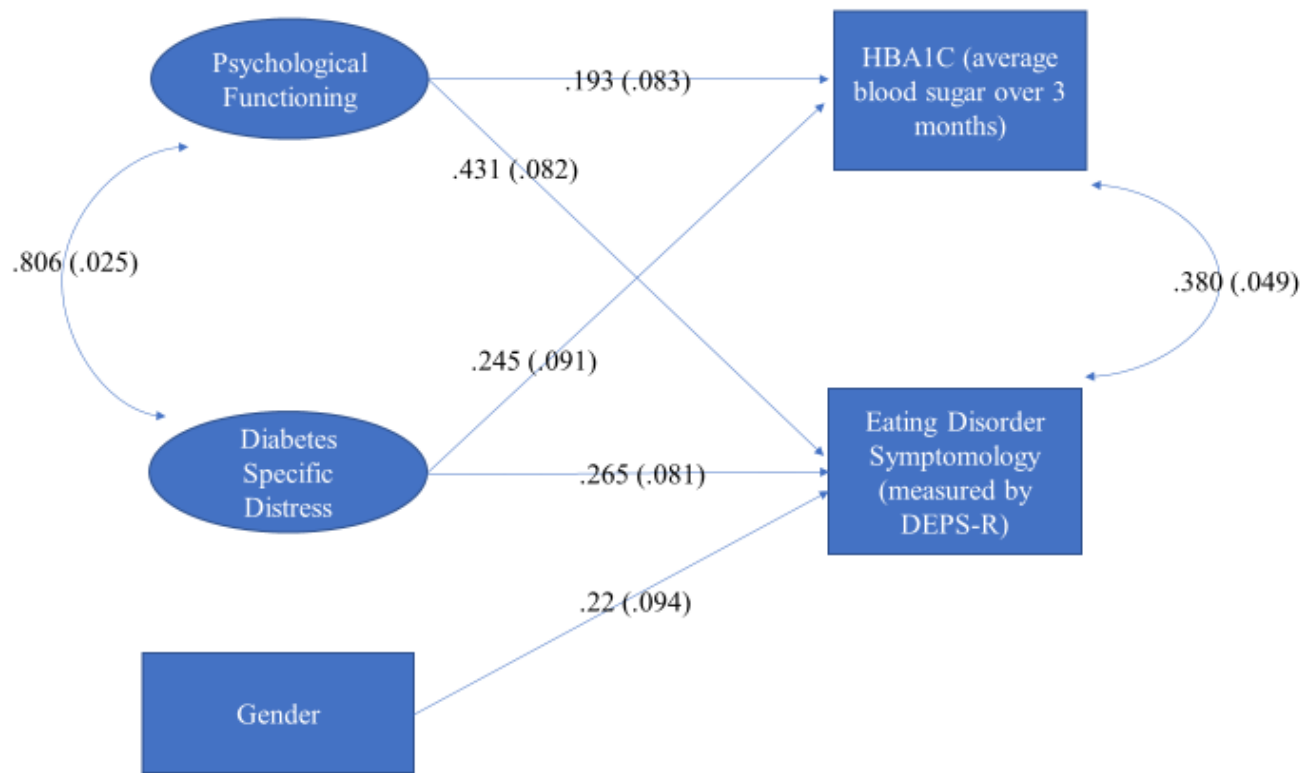


### 6.3.3 Structural Model 2: The Role of Family Functioning

Family functioning is related to psychological functioning and potentially then it may also be to HBA1C and DEPS-R. The next step tested the role of family functioning on HbA1c and DEPS-R. While there was no significant relationship between Diabetes specific distress and Family cohesion (estimate = -0.099 SE = 0.056 p = 0.080) there was for Psychological Functioning. As such the model was prespecified to only consider family functioning in relation to general psychological functioning.

Following this indirect effects were tested to ascertain whether psychological functioning mediate the effects of family cohesion on HBA1C and DESPR levels. A relatively well-fitting model was found ($x^2$ (46) =118.198, p< 0.000, RMSEA = 0.058, CFI = 0.963, TLI = 0.948).

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The results suggest that family cohesion acted as a protective factor, predicting reduction in problematic psychological functioning ($\beta$= -0.235 SE=0.039, 95% CI = -0.312, -0.158). What is more, indirect effects were found, suggesting that psychological functioning mediated the effects of family cohesion on HBA1C levels ($\beta$=-0.045 SE=0.023, 95% CI = -0.090, 0.000), and DEPS-R levels ($\beta$=-0.115 SE=0.029, 95% CI = -0.171, -0.058). The effects were just significant in the case of HBA1C, but significant at the p< .05 in the case of DEPS-R, this suggests that higher levels of cohesion in the family predicts less psychological problems, which in turn predict reduced levels of HBA1C and DEPS-R.

*6.5: Structural Model 2, Family Cohesion (standardised)*

| | Estimate | S.E. | P-Value | 95% CI Lower 2.5% | 95% CI Upper 2.5% |
|---|---|---|---|---|---|
| Psychological Functioning | | | | | |
| Anxiety | 0.809 | 0.024 | <0.001 | 0.762 | 0.855 |
| Self Esteem | -0.843 | 0.022 | <0.001 | -0.886 | -0.801 |
| Maladaptive Perfectionism | 0.701 | 0.029 | <0.001 | 0.644 | 0.758 |
| Borderline Traits | 0.744 | 0.031 | <0.001 | 0.684 | 0.804 |
| Depression | 0.864 | 0.014 | <0.001 | 0.836 | 0.892 |
| Diabetes Distress | | | | | |
| Emotional Burden | 0.904 | 0.023 | <0.001 | 0.858 | 0.949 |
| Physician Burden | 0.487 | 0.045 | <0.001 | 0.399 | 0.574 |
| Interpersonal Distress | 0.666 | 0.035 | <0.001 | 0.598 | 0.734 |
| Psychological Functioning | | | | | |
| Family Cohesion | -0.235 | 0.039 | <0.001 | -0.037 | -0.019 |
| HBA1C | | | | | |
| Psychological Functioning | 0.19 | 0.088 | 0.03 | 0.019 | 0.362 |

|  | Estimate | S.E. | P-Value | 95% CI Lower 2.5% | 95% CI Upper 2.5% |
|---|---|---|---|---|---|
| Diabetes Distress DEPS-R | 0.259 | 0.091 | 0.004 | 0.081 | 0.437 |
| Psychological Functioning Diabetes Distress | 0.488 | 0.08 | <0.001 | 0.33 | 0.645 |
| DEPS-R Gender | 0.211 | 0.08 | 0.008 | 0.055 | 0.367 |
| Indirect Effects HbA1c Psychological Functioning | 0.231 | 0.036 | 0.04 | 0.011 | 0.45 |
| Family Cohesion DEPS-R Psychological Functioning | -0.045 | 0.023 | 0.05 | -0.09 | 0 |
| Family Cohesion | -0.115 | 0.029 | <0.001 | -0.171 | -0.058 |

*Figure 6.3: Structural Model 2, Family Cohesion (standardised)*
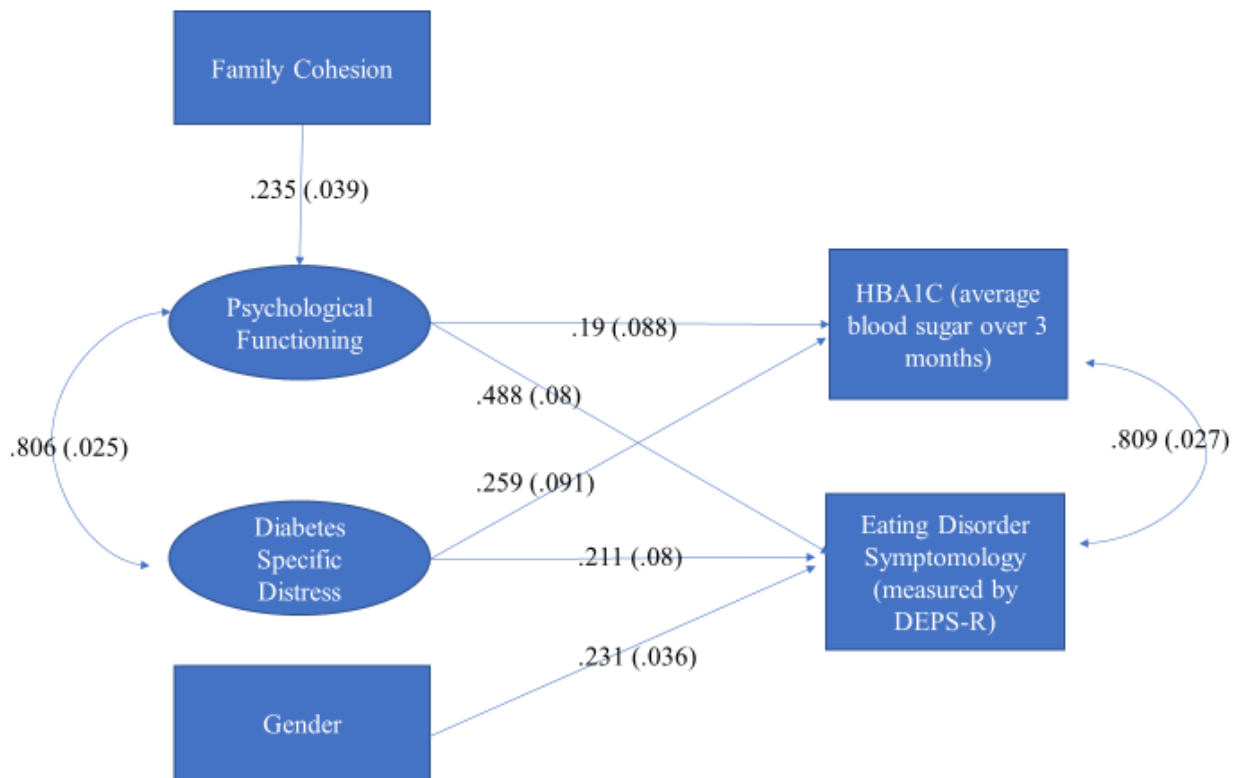


### 6.3.4  Structural Model 3: Consideration of Future Consequences

As stated in the literature review the relationship with consideration of future consequences is not well defined in the academic literature regarding T1ED but rather suggested by media source. As such in an alternative model CFC was added as an independent predictor of HBA1C and DEPS-R. A moderately well fitting model was found  ($x^2$ (46) = 144.833, p< 0.000, RMSEA = 0.067, CFI = 0.953, TLI = 0.933). Higher levels of consideration of future consequences predicted both lower HbA1c (β=-0.237 SE=0.046, 95% CI = -0.328, -0.146) and DEPS-R scores latter (β=-0.307 SE=0.043, 95% CI = -0.391, -0.223) suggesting that this maybe a protective factor. Also, on inclusion of consideration of future consequences to the model, psychological functioning no longer significantly predicted HbA1c.

*Table 6.6: Structural Model 3, Consideration of Future Consequences (standardised)*

| | Estimate | S.E. | P-Value | 95% CI Lower 2.5% | 95% CI Upper 2.5% |
|---|---|---|---|---|---|
| Psychological Functioning | | | | | |
| Anxiety | 0.818 | 0.023 | <0.001 | 0.771 | 0.858 |
| Self Esteem | -0.845 | 0.022 | <0.001 | -0.893 | -0.814 |
| Maladaptive Perfectionism | 0.718 | 0.028 | <0.001 | 0.665 | 0.769 |
| Borderline Traits | 0.768 | 0.027 | <0.001 | 0.692 | 0.806 |
| Depression | 0.865 | 0.014 | <0.001 | 0.834 | 0.888 |
| Diabetes Distress | | | | | |
| Emotional Burden | 0.879 | 0.023 | <0.001 | 0.84 | 0.923 |
| Physician Burden | 0.510 | 0.044 | <0.001 | 0.438 | 0.595 |
| Interpersonal Distress | 0.679 | 0.034 | <0.001 | 0.637 | 0.757 |
| HBA1C | | | | | |
| Psychological | 0.096 | 0.093 | 0.308 | 0.034 | 0.36 |
| Functioning Diabetes Distress | 0.242 | 0.091 | <0.001 | 0.062 | 0.421 |
| DEPS-R | | | | | |
| Psychological | 0.324 | 0.086 | <0.001 | 0.275 | 0.594 |
| Functioning Diabetes Distress | 0.310 | 0.080 | <0.001 | 0.101 | 0.42 |
| HBA1C | | | | | |
| Consideration of Future Consequences | -0.237 | 0.046 | <0.001 | -0.328 | -0.146 |
| DEPS-R | | | | | |
| Consideration of Future Consequences | -0.307 | 0.043 | <0.001 | -0.391 | -0.223 |

*Figure 6.4: Structural Model 3 (standardised): Consideration of Future Consequences*
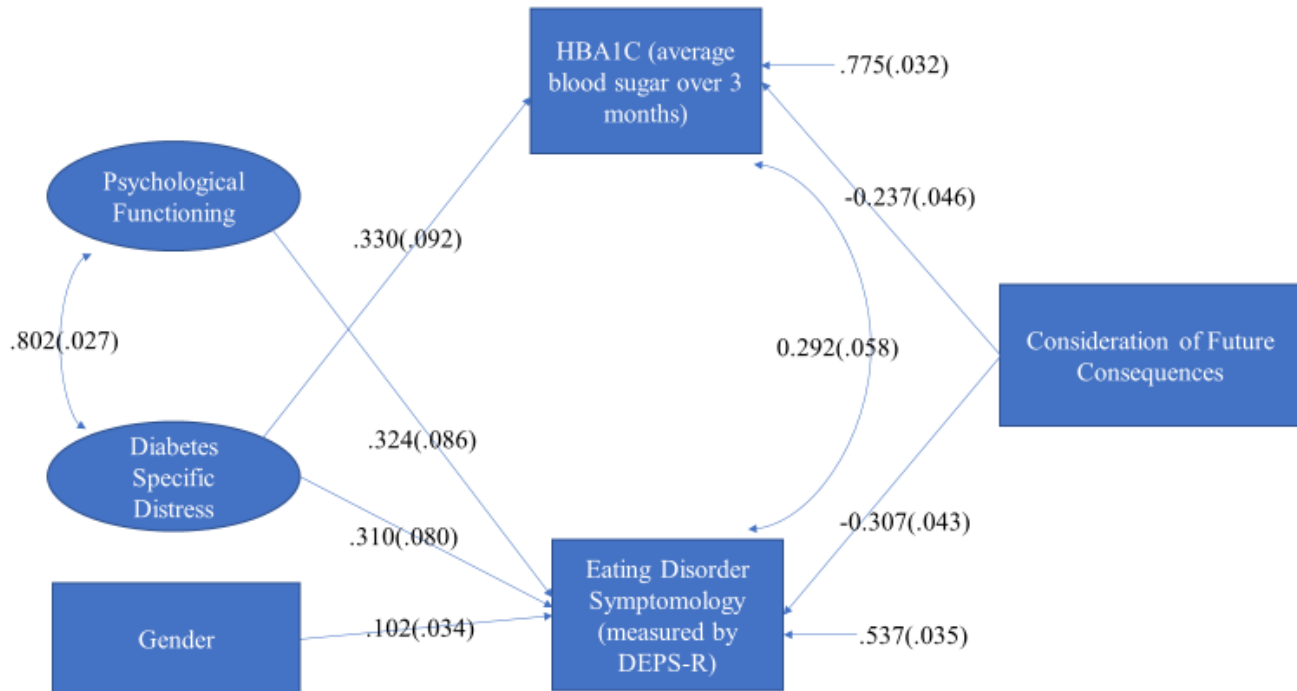


### 6.3.5 Relationship with age

In order to ascertain the relationship between HbA1c and DEPS-R and Age, Age was regressed onto those variables. The inclusion of age however significantly reduced the fit of the model: $x^2$ (58) =423.009, p< 0.000, RMSEA = 0.116, CFI = 0.822, TLI = 0.763 and thus it was not a predictor.

### 6.4 Discussion

As hypothesised, there were significant relationships between the variables highlighted in the literature review and the pilot study and both blood sugar management and Eating Disorder symptomology. These two variables were chosen as while there is no gold standard of defining EDs in T1D, the DEPS-R appears to be the measure that produces the most consistent results and HbA1c is higher in those who have Type 1 related Eating Disorders, this is consistent with the current study which shows a significant relationship between the two outcome variables. Also, as predicted individual scale scores formed two reliable latent

variables 'Psychological Functioning' and 'Diabetes Distress'. Psychological functioning predicted both HbA1c and DEPS-R scores the latter stronger than the former as did Diabetes distress in the initial structural model. The two latent variables were also strongly related. Gender predicted higher ED scores (only by .2 of a standard deviation which is lesser than would be suggested by the literature) but not worse blood sugar control. Family cohesion was related to psychological functioning but not Diabetes distress. A mediation analysis also suggested that higher levels of cohesion in the family predicted less psychological problems, which in turn predicted reduced levels of HBA1C and DEPS-R. Consideration of future consequences which was suggested as important by media reports was revealed to be related as higher levels predicted both lower blood sugar levels and Eating Disorder symptomology but inclusion of the variable into the model altered the predictive value of psychological functioning to HbA1c as no longer significant. Age however, when added to the model decreased model fit substantially and therefore could not be considered as a predictor in the current sample.

The latent variable 'Psychological functioning' was comprised of higher levels of anxiety, depression, borderline traits and maladaptive perfectionism and lower levels of self-esteem which was all suggested by the pilot study and the literature review. All of these variables were strongly related to the latent factor suggesting that in these patients there may be an underlying vulnerability which makes them less psychologically stable or more prone to the symptoms of mental illness. Given that all of these patients share a diagnosis of T1D it could therefore be argued that T1D is potentially causal to that vulnerability. Furthermore, these variables are also highlighted as risk factors/ comorbidities for those without T1D and ED. This may go some way to explain the higher prevalence of EDs in T1, the patients are already at a higher level of risk as they display higher levels of these traits, which have more far reaching consequences, particularly in relation to blood sugar control and Eating Disorder onset. In all of the models the two latent variables were also closely related so it may be that it is the combination of psychological dysfunction and Diabetes specific distress that is important for the development of EDs.

Psychological functioning predicted both Eating Disorder symptomology and blood sugar control and while it could be argued that a number of factors are associated with blood sugar

management, that a latent factor can predict both has substantial clinical value. While we do not have a gold standard for defining T1EDs we know that the DESPR is measuring behaviour related to EDs and that blood sugar management is significantly disrupted by the presence of these behaviours.

The latent variable 'Diabetes Distress' was comprised of the DDS subscales except regimen related distress (please see below) and significantly predicted both HbA1c and DEPS-R scores. Interestingly the modification indices did not suggest covariance between any of the psychometric variables and that of the emotional burden, physician related and interpersonal subscales.. This further points to Diabetes specific distress as an independent entity that is not beholden to other psychological vulnerability. Researchers have postulated several reasons as to why this demographic are more vulnerable to EDs and it may be that DSD is an explanation for that. This may also help explain why insulin omission can appear (particularly in women) significantly at much older ages (Goebel-Fabbri et al., 2015) than seen for standard ED behaviour in the general population. Diabetes specific distress adds an extra temporal dimension, and DSD can presumably be affected by life events for example the death of a family member or some other loss. In this case the stressor that is DSD is a lifelong vulnerability, which is unique to this population. Furthermore, in Diabetes burnout whereby you do not have the energy to look after blood glucose properly there may be a side effect, weight loss. Burnout leads to lower concern over managing blood sugars and if that translates into general higher glycaemia then it may be that this weight loss is seen as a positive change and this facilitates further ED behaviour. In this sense the patient may feel *rewarded* for not looking after their Diabetes with weight loss. That combined with maladaptive perfectionism, which is hard to imagine would be easy to deal with in the face of burning out or *failing* at T1D, and lower levels of self-esteem and other psychological variables may create the perfect storm for ED development.

That the two latent variables were strongly related is also of significance. There is evidence to suggest that while psychological conditions or Diabetes distress alone do not predict blood sugar control, when you combine them self-care is decreased, despite access to specialist services aimed at doing so (Sturt et al., 2015).

Being female was a risk factor for higher scores on the DESPR but not for higher HbA1c. It is of note that the increase in risk was not as high as would have been suggested in the literature at only .2 of a standard deviation. This may be consistent with research that suggests that EDs are more common in T1D males than in their non diabetic peers, and that the proportion of difference between male and female sufferers is lessened. If we accept HbA1c as a measure of insulin omission then the model would suggest that as there is no predictive value of gender then males and females are omitting insulin at the same rate which is in opposition to previous literature. However, this may also be due to specific limitations of the current study (please see below)

As it was suggested in the literature that family functioning was important to several Diabetes related aspects of life, family cohesion and adaptability was measured. Adaptability did not significantly relate to the variables of interest, but cohesion was found to have predictive power in relation to 'Psychological functioning'. Furthermore, cohesion was protective with increased levels mediating HbA1c and DEPS-R scores. Clinically this is important as interventions that target improving cohesion could feasibly improve both outcome variables. It was somewhat unexpected that family cohesion did not predict Diabetes distress scores however.

That consideration of future consequence is relevant in the model poses some difficult questions. Firstly, this is a novel variable that has not been measured in relation to T1D before. It has been seen in qualitative research that those with Diabulimia report feeling 'doomed' by their diagnosis or that no matter what they do they are going to be afflicted by Diabetes related complications such as retinopathy or neuropathy and this suggests that perhaps these aspects should not be overstated in clinic visits. The current model however would suggest that consideration of these aspects is an important protective factor in the development of T1ED. As two recent participants stated:

> I could already foresee the future. I was like "I'm going to be 32 one day, and 35 one day, and I'll want to have kids", but I'm not going to be able to have kids, I'm not going to have legs, I'll be blind because of what I'm doing right now (Goebel-Fabbri, 2017, p. 11)

> While I was in the hospital... one of the women that was instructing me about everything… gave me all this literature, and it was like, "On average, if you have Diabetes, 15 years is going to be cut off your life". I remember staying up in bed in the middle of the night the time I was diagnosed, reading all this stuff that was all the horror stories of Diabetes that you hear about. Why would you give this literature to a 15-year-old girl to have her read in her hospital bed? And I think that really stuck with me (Goebel-Fabbri, 2017, p. 22-23)

It is hard to imagine that these participants and others like them are not considering the future consequences of their actions and this may highlight the difference between awareness and action. One can be highly aware of future consequences without being able to do anything about them. In fact, it could be argued that this is an important part of what makes T1ED a mental illness

The inclusion of the CFC also reduced the predictive value of psychological functioning to insignificant in the case of HbA1c but not DEPS-R scores, perhaps further highlighting the previous point. This raises further questions also about the appropriateness of the DEPS-R as a screening tool also (for a further discussion of this please see chapter 9), One can feel, for example, fat when using the appropriate dose of insulin without resorting to insulin omission to rectify it. This having been said the relationship between T1ED and CFC is not clear, as this is the first study to explore this variable in relation to the T1D demographic.

Regimen related distress was not added as a predictor as it was highly related to DEPS-R scores and could have therefore skewed the model. Theoretically it could be argued that the same items that measure regimen related distress such as 'Feeling that I am not testing my blood sugars frequently enough' or 'Feeling that I am not sticking closely enough to a good meal plan' also measure T1EDs, particularly Diabulimia. It is akin in this sense to asking those with AN how they feel about the regimen of eating. Age could also not be predictably used in the model either but that coincides with evidence that insulin omission for weight loss can appear (particularly in women) significantly at much older ages than in the general population (Goebel-Fabbri et al., 2015).

### 6.4.1   Strengths

There are implications for the uncovering of these latent variables including potentially being able to indirectly measure T1ED. Given that researchers have suggested most people

'discover' Diabulimia through indirect sources (Balfe et al 2013; Goebel Fabrri, 2017), it is not out with the realms of possibility that the DEPS-R could function as one of these indirect sources. Being able to measure psychological variables *around* T1ED may be a safer way of gauging risk in the T1D population. Also this is the first model to identify that Diabetes specific distress is predictive of Eating Disorders as well as BG control highlighting that this population has unique risk factors that are not currently addressed in routine ED treatment.

### 6.4.2 Limitations

There are several limitations of the current study. As with any theoretical model it may be that there are further variables that remained unidentified by the pilot study which on inclusion would have provided a different solution. A perhaps significant exclusion is weight. BMI has been found to predict Eating Disorders in a number of studies, higher weight has been shown to predict low self-esteem and body image in the general population and this is also seen in those with T1D (Tse et al., 2012, Powers et al., 2012). Those T1D's who report being overweight also report higher levels of ED behaviour (Markowitz et al., 2009) and being overweight may predict the onset of ED in T1D (Olmsted et al., 2008). However patients have stated how distressing being weighed is and as presumably a sizeable minority of this sample were assumed to be struggling with or recovering from EDs it was omitted from the current study.

A further potential limitation is that while around 30% of the sample stated that they had current or previous experience of an ED, nearly 45% scored above the DEPS-R cut-off point, suggesting either that they are partaking in behaviour they may not be aware is related to EDS or that there are issues with the DEPS-R as a measure potentially related to the difference between feeling and actions alluded to above.

There are several key differences in the reported sample than in other research into T1EDs. Firstly this research utilised a global sample rather than a localised one, this may mean that further research may want to specify regional variations. The sample was also collected via the internet particularly through social media and as such it may be that this model is more representative of the Diabetes online community. The sample is also self-selecting and given that the recruitment advert was forthcoming regarding the psychological nature of the study

it may also be the case that the current sample is more representative of those who are more comfortable disclosing these sorts of issues. The internet also made it possible to collect a much larger sample of those with T1D, nearly 700 participants responded to the study which is considerably higher than the vast majority of studies into T1ED (please see section 2.4.2). Regardless of this however there was still an over-representation of females, had there been a more equal gender split then two models investigating each gender may have been warranted, this lack of male representation is an issue that is prevalent in other research in this area and future researchers should make a concerted effort to address this. Perhaps however, the main difference between this sample and others is the proportion of participants who utilise Diabetes technology. Given that rates of pump usage are reported at around 15% by NHS England, the rates seen in this sample at 38% may represent a significant bias. Although data on continuous glucose monitors is scarce the insulin pump awareness group (IPAG) reports that for the year 2018/19 there are only 153 individually funded in Scotland, a significantly lower proportion that the 42% of this study utilising the technology. There are implications for this in terms of both HbA1c and DEPS-R. NHS Digital reports that those using Diabetes technology are more much more likely to achieve recommended blood sugar levels and there is further evidence that pump usage reduces T1ED symptomology (NHS Digital 2018; IPAG Scotland 2018; Markowitz et al., 2013). This would suggest that the assumptions made in this study are conservative.

As stated elsewhere there is no gold standard for measuring or defining T1EDs and while it may be customary to suggest that future studies may wish to involve clinical interviews, it has been shown that participants are more likely to under report Diabetes specific Eating Disorder behaviours such as insulin omission under these conditions and currently there is no Diabetes specific ED interview (please see chapter 2). It may also be argued that there are further issues in that there is no non-diabetic control group to compare with some sort of similar ED measure, but those without T1D do not have outcomes such as HbA1c and DDS to consider, so a lack of control group may be justified.

## 6.5    Conclusion

This model tested factors around T1ED, measured by the DEPS-R and HbA1c and found that 'Psychological functioning' and 'Diabetes distress' predicted both, that family cohesion was protective for psychological function, and that higher levels of consideration of future consequences was protective in terms of DEPS-R and HbA1c. This is important particularly for clinicians who are seeking to treat this population given that the majority of the evidence suggests that standard treatment rarely works. It may be that given the unique experience of T1D and its relation to weight control behaviour that EDs in T1D are just *too* different to be even considered alongside EDs in the general population.

# 7    Patient Attributions as to what Caused their Eating Disorder

## 7.1    Introduction

As research into Eating Disorders in Type 1 Diabetes has predominantly focussed on prevalence rates and academic debate has neglected much that falls outside clinical diagnoses, little is known about the profile of those suffering. There are important questions that remain chronically under-investigated such as whether or not they have reached out for help, who they have reached out to and whether or not they have other issues that may relate to treatment outcomes such as psychological comorbidity. There is also a lack of research investigating how these patients self identify their EDs and what diagnoses are attributed to them in the face of the non clinical term that references insulin omission for weight loss 'Diabulimia', nomenclature that is widely accepted in Diabetes community if not in the Doctors office (BBC, 2017).

It is also important to understand what these patients attribute to the development of their T1ED. Presently, although NICE has recognised the need for consistent practice, the guidelines are not obligatory and there is no other national policy on how to deal with T1EDs. Furthermore HCPs have reported that they feel significantly out of their depth dealing with this population (Allan, 2017[12]; Tierney et al., 2009). Understanding attribution may inform future guidance which may lessen the impact of such factors or provide a framework for early intervention. Causal attributions can affect a number of health related behaviours and outcomes. For example, at 8 year follow up for patients who had suffered a heart attack, it was shown that those who attributed the event to stress or other people were more likely to have another attack and die than those who saw the initiating event as an indication to make behavioural changes (Affleck, Tennen, Croog & Levine, 1987). In mental health, attributions as to what is causal to the illness has also been shown to affect treatment seeking behaviour (Narikiyo & Kameoka, 1992). In T1D research attributional inquiry is extremely limited. In

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[12] Appendix A

one study though it was shown that negative attributions to perceptions of friend reactions to regimen adherence predicts poorer glycaemic control, demonstrating that attributions may be important to health behaviours (Hains, Berlin, Davies, Parton & Alemzadeh, 2006; Hains et al., 2006). Causal attributions have not been explored in T1EDs. In order to address this gap in the literature a pilot study was undertaken by the researcher in 2013[13] using participants who had at least 2 years recovery from T1ED. A questionnaire was constructed that reflected suggestions made in the research literature as to why EDs are more common in T1D and this was then subjected to an exploratory factor analysis. Other aspects were also investigated such as psychiatric comorbidity, ED diagnoses and insulin omission. Please note it is not the purpose of this study to assess the validity of the attributions that researchers suggest as aetiological in the development of T1ED, rather it is to describe them, ask the participants how much they agree with them and ascertain whether they form any useful pattern.

## 7.2    Item Generation

A review of the literature until 2013 was undertaken in order to ascertain what researchers suggested as aetiological to the development of T1EDs. The attributions took the format of 6 broad categories: Diabetes specific factors, Weight and Body Image issues, Dietary aspects of T1D, Iatrogenic factors, Family problems affected by T1D and more Classic Risk factors that can be also seen in Eating Disorders in the general population.

### 7.2.1    Diabetes Specific Factors

In searching for potential reasons as to why EDs may develop in those with T1ED many researchers point out that insulin omission as a successful weight loss tool is a unique to this demographic and this uniqueness and/ or the realisation alone of how much weight can be lost my omitting is enough to encourage this behaviour (Goebel-Fabbri et al., 2008; Ackard

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[13] Appendix F

et al 2008; Markowitz et al 2009; Nash & Skinner 2005; Daneman et al., 2002; Jones et al., 2000; Colton, Rodin, Bergenstal & Parkin, 2009; Ishmail & Treasure, 2010)

Several Diabetes specific psychological factors were suggested in the literature to be important to the development of EDs in those with T1D including general Diabetes stress, fear of hypoglycaemia, fear or embarrassment of injecting or testing, denial and/ or resentment of the diagnosis and burnout (Nielsen, Børner & Kabel, 1987; Steel et al., 1989; Goebel- Fabrri et al.,  2008; Colton et al., 2009; Ishmail & Treasure, 2010).

Other personality aspects such as perfectionism have been suggested to be detrimental to adherence, trying to attain perfection in relation to blood sugars with an illness as unpredictable as Diabetes has been related to ED development (Nielsen et al., 1987; Peveler & Fairburn, 1989; Steel et al., 1989; Pollock Barziv & Davis 2005; Young-Hyman  & Davis, 2010).

### 7.2.2   Weight/ Body Image Related Factors

Pre-diagnosis many T1Ds will lose a substantial amount of weight and as such several researchers have suggested that this is a triggering factor for the development of EDs (Pinar et al., 2005; Markowitz et al., 2009; Jones et al., 2000; Ishmail & Treasure 2010). This initial weight loss is also typically reversed on commencement of insulin therapy which can act as a second or compounding trigger (Peveler & Fairburn, 1989; Steel et al., 1989; Crow, Keel, & Kendall, 1998; Nielsen & Mølbak, 1998;  Pinar et al., 2005; Grylli et al., 2005; Battaglia et al., 2006; Jones et al.,  2000; Young-Hyman  & Davis, 2010; Ishmail & Treasure, 2010). It has also been argued that this initial loss then gain can promote a fear of increased weight which then promotes Eating Disordered behaviour (Goebel- Fabrri et al., 2008; Olmsted et al., 2008).   This is particularly problematic as T1D carries around a 15% weight penalty meaning that regardless of T1D concern over higher a higher BMI can promote ED behaviours (DCCT, 1995; Alice Hsu et al., 2008; Markowitz et al., 2009; Tse et al 2012; Schwartz et al., 2002; Nash & Skinner, 2005; Daneman et al., 2002; Meltzer et al., 2001; Colton et al., 2009). Several researchers recognise that T1D may promote body image disturbances either due to the weight penalty or as T1D may increase body awareness, which may encourage ED behaviour (Steel et al., 1989; Striegel-Moore et al., 1992; Pinar et al.,

2005; Alice Hsu et al., 2008; Schwartz et al., 2002; Daneman et al., 2002; Meltzer et al., 2001; Young-Hyman & Davis, 2010). As weight is a salient aspect of T1D management, researchers have suggested that compared with healthy peers a sense of achievement at losing weight is potentially heightened which also contributes to ED development (Peveler & Fairburn, 1989; Smith et al., 2008; Daneman et al., 2002).

### 7.2.3   Dietary Factors

Unsurprisingly the dietary aspects of T1D were suggested by many researchers as aetiological to T1ED, particularly dietary restriction (Powers, Malone, Coovert, & Schulman, 1990; Striegel-Moore et al., 1992; Crow et al., 1998; Pinar et al., 2005; Goebel-Fabbri et al., 2008; Nash & Skinner, 2005; Daneman et al., 2002; Jones et al., 2000), having to correct for hypoglycaemia which may lead to overeating/ bingeing and subsequent weight gain (Battaglia et al., 2005; Goebel-Fabbri et al 2008; Daneman et al., 2002; Young-Hyman & Davis, 2010; Criego et al., 2009). The excessive focus on food which often included adherence to strictly timed meals was also suggested by several researchers to promote a disturbed attitude which was related to ED onset (Ackard et al., 2008; Olmsted et al., 2008; Alice Hsu et al, 2008; Markowitz et al., 2009; Powers et al., 2012; Quick et al 2012; Tse et al., 2012; Schwartz et al., 2002; Colton et al., 2009).

### 7.2.4   Iatrogenic Factors

As stated in chapter 4, those with T1D have increased interactions with HCPs compared with the general population and it has been suggested that certain negative feelings regarding these interactions may be iatrogenic in the development of T1ED, particularly attention on weight, pressure to be a 'good' diabetic and an over – emphasis on future complications which can lead a patient to feel hopeless.  (Szmukler, 1984; Steel et al., 1989; Smith et al., 2008; Quick et al., 2012; Nash & Skinner ,2005; Daneman et al., 2002; Crow et al., 1998; Colton et al, 2009).

### 7.2.5   Family Factors

Family factors have been discussed as important to the development and maintenance of T1EDs (please see chapter 4). Family profiles that are high in general dysfunction and rigidity

have been suggested to encouraged ED development (Malone & Armstrong, 1985; Peveler & Fairburn, 1989; Colton et al., 2007; Daneman et al., 2002). Of particular interest is the conflict that can arise from developing a chronic illness which requires intense management at the time, developmentally, when healthy adolescents would be striving for growing autonomy and individualism. Several researchers have suggested that this conflict and disagreement over Diabetes management between parents and their T1 children is important to the development of T1ED (Malone & Armstrong, 1985; Nielsen et al., 1987; Striegel - Moore et al., 1992; Grylli et al., 2005; Grylli et al., 2010; Schwartz et al 2002; Daneman et al., 2002; Young-Hyman & Davis, 2010; Szmukler, 1984).

Examples of disturbed eating in the family is also suggested as related to T1ED (Colton et al., 2007; Daneman et al., 2002) The mother daughter relationship appears to be particularly important attributionally, especially maternal concerns with weight and shape (Quick et al., 2012; Colton et al 2007; Daneman et al., 2002).

### 7.2.6   Classical Eating Disorder Attributions

Need for control is a common attribute of EDs in the non T1D population. It is perhaps expected then, that in an illness which is as difficult to manage as T1D, that need for control would feature heavily in suggestions as to why EDs are more common (Nielsen et al., 1987; Grylli et al., 2010; Schwartz et al 2002; Nash & Skinner 2005; Young-Hyman & Davis, 2010).

There is some evidence that those who have T1D have disrupted onset of menses which is a known risk for EDs in the non T1D population, several researchers argue that in those with T1D this period of time is even more risky as pubertal hormones can substantially increase insulin requirements leading to weight gain (Smith et al., 2008; Alice Hsu et al., 2008; Grylli et al., 2010; Schwartz et al., 2002; Meltzer et al., 2001).

Some researchers have argued that while ED families may be high in disfunction that does not negate them from expecting high achievements, and this is no different in families experiencing T1ED ,with academic pressure posing a suggested risk (Neilsen et al., 1987; Daneman et al., 2002). Those with T1D are not immune to the societal pressure to conform

to a thin ideal either and as such several researchers suggested that cultural issues surrounding body composition were important for ED development, (Nielsen & Mølbak, 1998; Pinar et al., 2005: Quick et al 2012; Meltzer et al., 2001; Colton et al., 2009) neither are they immune the negative consequences of conflict in peer relationships with some researchers arguing that peer issues contribute to ED development (Szmukler, 1984; Peveler & Fairburn, 1989).

Several authors have linked comorbid mental health problems to the development of EDs in both the T1 and non T1 population as discussed in chapter 4. As the focus of the current study was to measure attributions rather than clinical diagnoses for the purposes of the questionnaire, participants were asked if they felt that mental illness had contributed to the development of their ED rather than for the individual diagnoses. A question regarding self-esteem was also asked in the same manner given numerous suggestions in the literature that is important (see chapter 4). A pre T1 diagnosis of an Eating Disorder has also been suggested as a risk factor by early researchers (Roland & Bhanji, 1982; Nielsen et al., 1987) and was thus also included.

## 7.3    The Current Study

There were considerable limitations in the pilot study that the current study seeks to address. As the original research only took those in recovery into consideration the attributions were retrospective. The study was also hampered by not having a follow up confirmation of the factor structure. The purpose of this study is to replicate the pilot study but with a larger sample size, including those who are currently suffering with T1ED, in order to get a better understanding of the profile of these patients and potentially confirm an underlying structure to what they attribute to the development of their ED.   It is hypothesised that there will be an underlying structure which can be confirmed and shows a meaningful pattern of latent factors.

## 7.4    Study Methodology

Data was collected at 2 time points in order to achieve a large enough sample for both an exploratory and a confirmatory factor analysis. At time 1 this was as part of a larger study (please see section 6.1 for ethical approval, setting, procedure and participants). On

completion of the questionnaire section of that study participants were asked if they had ever suffered with an Eating Disorder, if they answered yes to this question they were taken to the 'Factors in Eating Disorders questionnaire' where they answered demographic and other profile questions (please see appendix N) Some of the questions in the scale were worded so that the patient had to think carefully about their answer in order to avoid response bias.

For the confirmatory sample collected between May and June 2018, everything was replicated except the advert was tailored to appeal to those who had experience of an Eating Disorder (please see appendix O) as this was the only population of interest and as such only the Demographics & Factors in Eating Disorders Questionnaire was administered.

## 7.5    Results

### 7.5.1    Overview of Analysis

Firstly, the demographic details of the participants are described at time 1 (exploratory) and time 2 (confirmatory). Following this, the exploratory and confirmatory factor analyses are described and the underlying latent variables constructed. In order to further investigate specific aspects of this population the samples are then combined for descriptive purposes.

### 7.5.2    Software

IBM SPSS vs 23 was used for the Exploratory Factor Analysis.  FACTOR was used for the parallel analysis (Lorenzo-Seva & Ferrando 2006), Mplus version 8 was used for the Confirmatory Factory Analysis (Muthén & Muthén, 1998-2016).

### 7.5.3    Participants

132 participants elected that they had current or previous experience of T1ED and were presented with the Factors in Eating Disorder Questionnaire at time 1. 127 participants provided complete answers. 189 participants originally answered the questionnaire at time 2 but respondents who stated that they had completed the questionnaire before had their answers were removed. This left 161 participants providing responses. There were no

significant demographic differences between the samples using independent T- Tests (please

tables 7.1 & 7.2).

*Table 7.1: Sample Characteristics and Means Difference Tests*

| Exploratory Sample | | Confirmatory Sample | | t | df | p |
|---|---|---|---|---|---|---|
| Age (n = 130) | 32.62 (11.52) | Age (n = 161) | 31.89 (8.96) | 0.612* | 289 | 0.541 |
| Age at Diagnosis (n = 130) | 14.49 (10.05) | Age at Diagnosis (n = 161) | 13.34 (8.52) | 1.058 | 289 | 0.291 |
| Time from T1D to ED in years (n= 122) | 4.88 (5.12) | Time from T1D to ED in years (n = 160) | 5.97 (5.67) | -1.621 | 280 | 0.103 |
| HbA1c (n = 124) | 8.93 (3.29) | HbA1c (m = 160) | 9.24 (2.73) | -0.864 | 282 | 0.388 |

*Table 7.2: Sample Gender, Nationality and Ethnicity*

| | n | Valid Percent |
|---|---|---|
| Gender | | |
| Male | 12 | 4.15 |
| Female | 277 | 95.85 |
| Nationality | | |
| British | 131 | 45.0 |
| American | 105 | 36.1 |
| Australian | 11 | 3.8 |
| Canadian | 10 | 3.4 |
| Irish | 9 | 3.1 |
| New Zealander | 4 | 1.4 |
| South African | 4 | 1.4 |
| Danish | 3 | 1.0 |
| Euro/US | 2 | 0.7 |
| German | 2 | 0.7 |
| Barbadian | 1 | 0.3 |
| Norwegian | 1 | 0.3 |
| Swedish | 1 | 0.3 |
| Polish | 1 | 0.3 |
| Bulgarian | 1 | 0.3 |
| Greek | 1 | 0.3 |
| Israeli | 1 | 0.3 |
| French | 1 | 0.3 |
| Egyptian | 1 | 0.3 |

|  | n | Valid Percent |
|---|---|---|
| Finnish Ethnicity | 1 | 0.3 |
| White/Caucasian | 253 | 92.3 |
| Mixed Race | 9 | 3.3 |
| Hispanic | 4 | 1.5 |
| Black | 3 | 1.1 |
| Jewish | 3 | 1.1 |
| Asian | 1 | .4 |
| Native American | 1 | .4 |

## 7.5.4 Medical profile and Health Seeking Behaviour

291 participants provided at least partial answers to questions regarding their medical status and health seeking behaviour. For missing data patterns please see below

*Table 7.3 Missing Data*

|  | n | missing | % |
|---|---|---|---|
| Eating Disorder Diagnosis | 281 | 10 | 3.4 |
| Perceived Eating Disorders | 283 | 8 | 2.7 |
| Insulin Omission | 286 | 5 | 1.7 |
| Mental Health Comorbidities | 285 | 6 | 2.1 |
| Bullying T1D | 272 | 19 | 6.5 |
| Bullying Weight | 271 | 20 | 6.9 |

### 7.5.4.1 Eating Disorder Diagnoses & Perceived Eating Disorders

The participants who self-identified as having experience of an ED (n = 281) had been formally diagnosed with a variety of ED diagnoses. The most common diagnosis was EDNOS and co and multi morbidity was frequent although more than 40% had received no diagnoses at all. When asked what ED they thought they have the majority of participants responded with Diabulimia but again they often perceived themselves to have co morbid conditions. A sizable majority of the participants also reported insulin omission (please see figures 7.3, 7.4 & 7.5). The majority of participants did approach a HCP for help regarding their ED. Of the UK based participants, the general practitioner was the most commonly

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approached but it was also common for patients to seek out private treatment (please see figures 7.6 & 7.7).

*Figure 7.1 Eating Disorder Diagnoses*
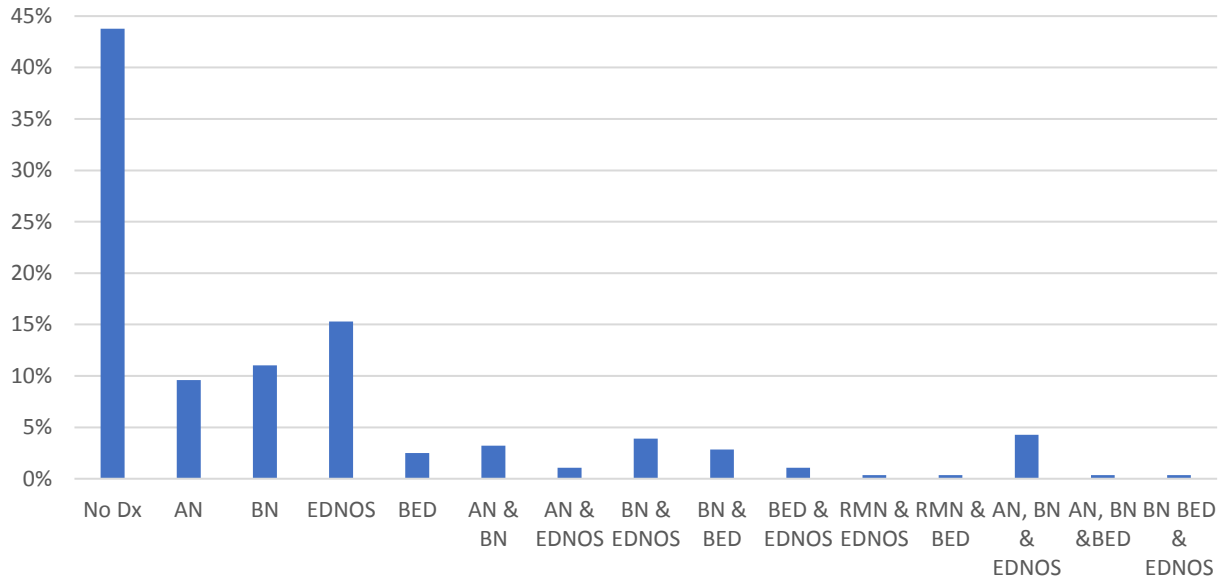


AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified, BED = Binge Eating Disorder, RMN = Rumination Disorder, No Dx = No Diagnoses

*Figure 7.2: Perceived Eating Disorder Diagnoses*



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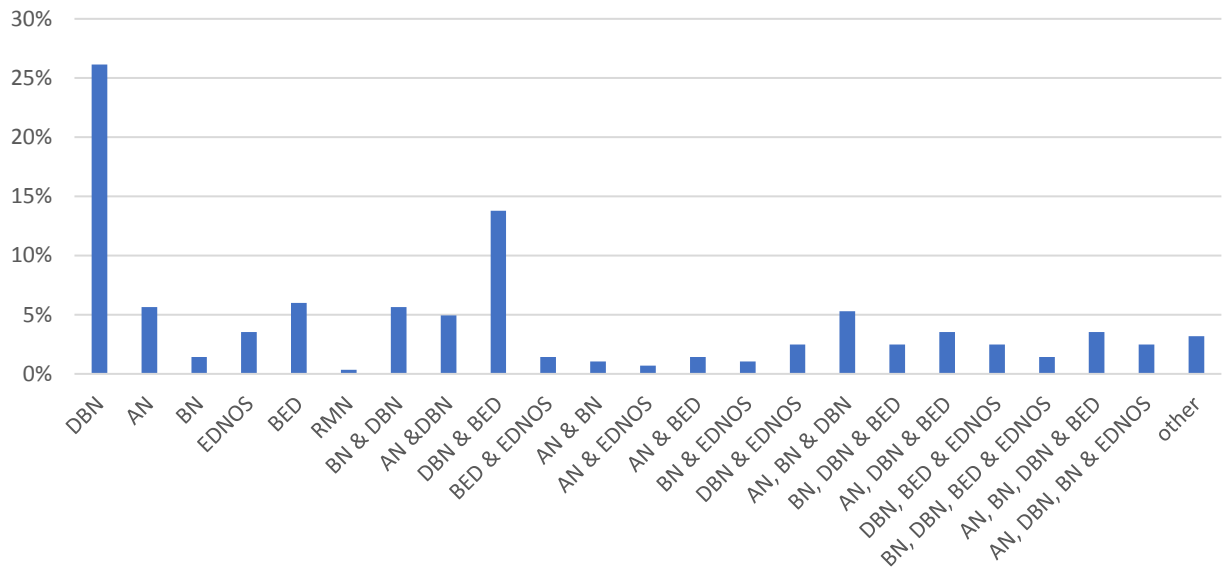
DBN = Diabulimia, AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified, BED = Binge Eating Disorder, RMN = Rumination Disorder

*Figure 7.3: Insulin Omission for Weight Loss Purposes*
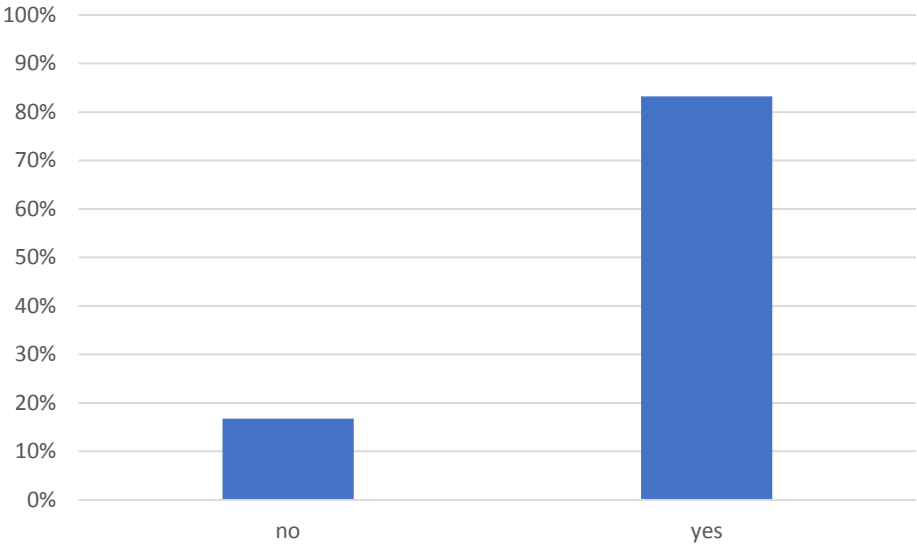


*Figure 7.4: Did you Approach a HCP for Help?*
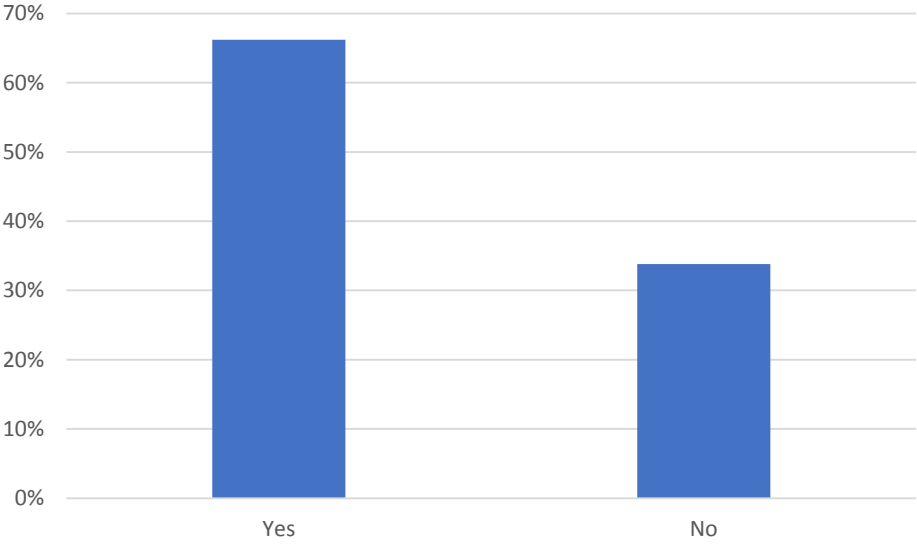
*Figure 7.5 Help Seeking Behaviour of UK Based Participants*
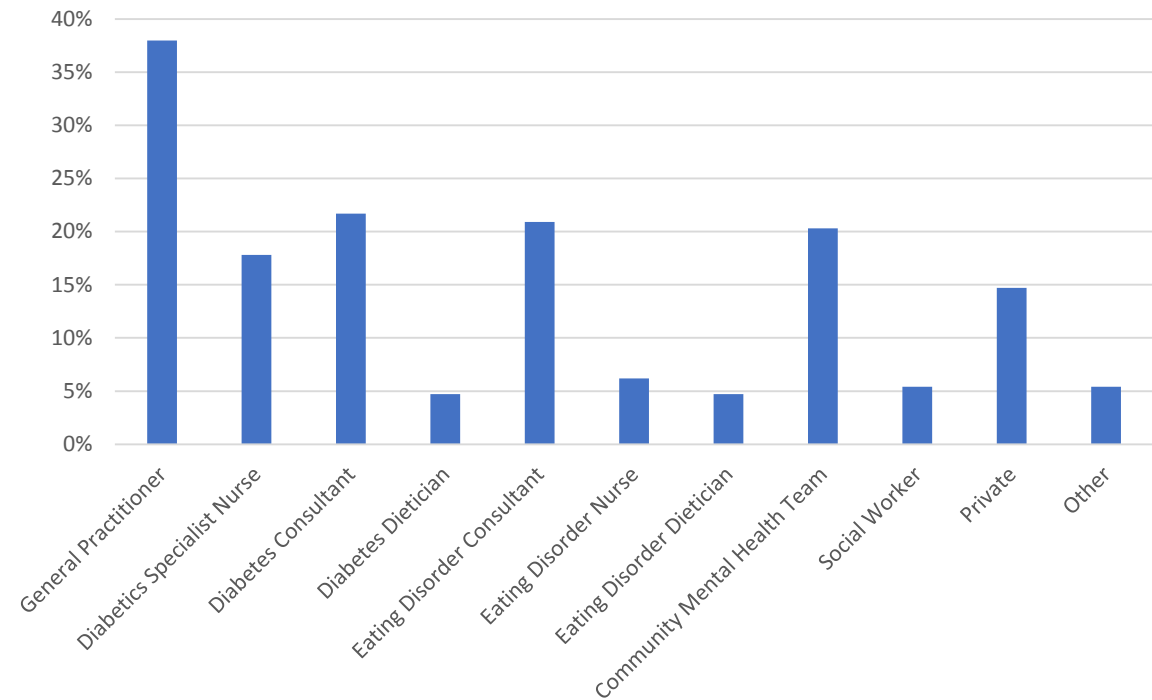


### 7.5.4.2    Mental Health Comorbidities and Bullying

Most of the participants had experience of other mental health issues with depression also being diagnosed in nearly half of the participants and anxiety in over a third. The full spectrum of diagnoses can be seen in figure 3.7 below. More than half of respondents had experienced weight related bullying and 40% had experienced bullying due to T1D (figure 3.8).

*Figure 7.8: Mental Health Comorbidity.*
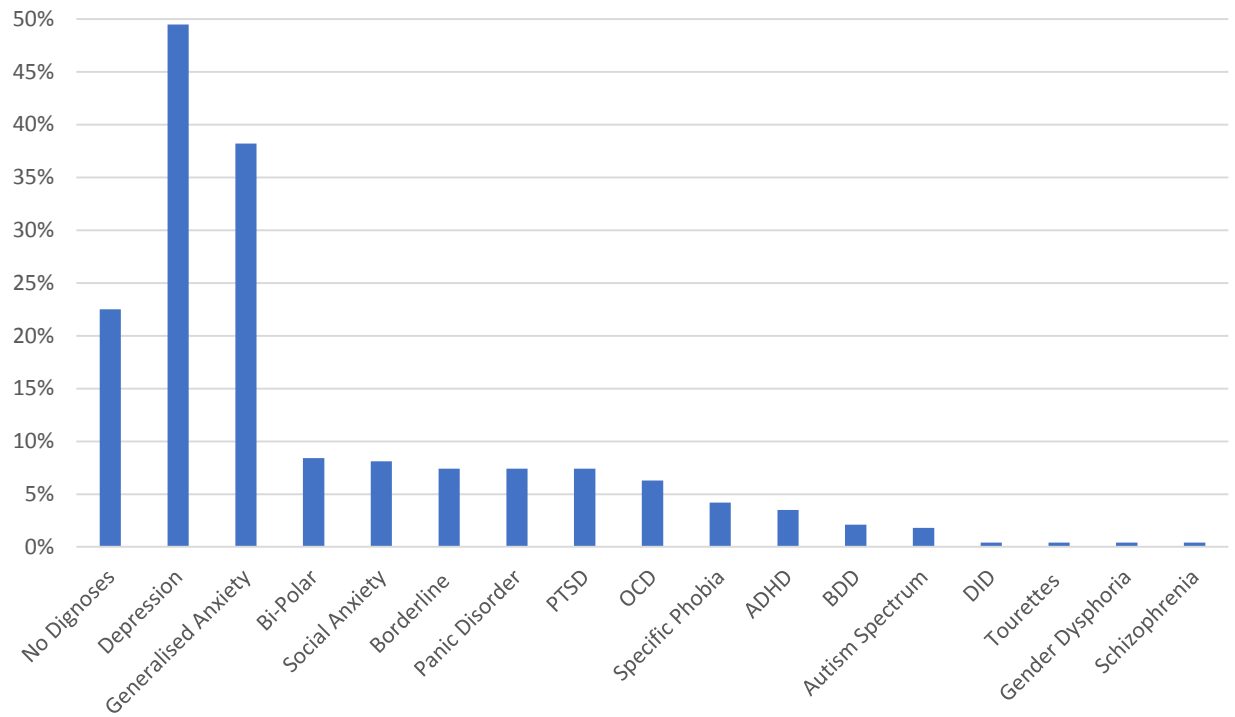


Borderline = Borderline Personality Disorder, PTSD = Post Traumatic Stress Disorder, OCD = Obsessive compulsive Disorder, ADHD = Attention Deficit Hyperactive Disorder, BDD = Body Dysmorphic Disorder, DID = Dissociative Identity Disorder

*Figure 7.9 Bullying*
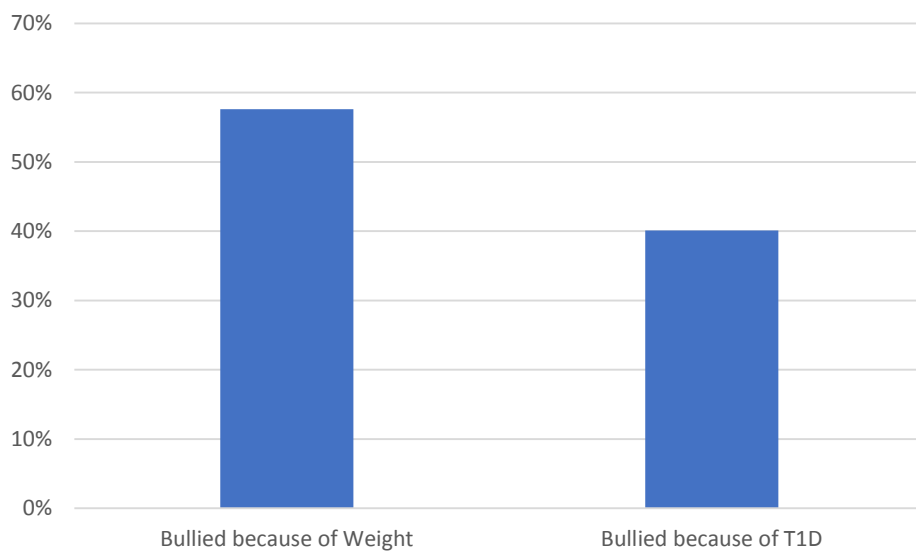
### 7.5.5    Exploratory Factor Analysis

#### 7.5.5.1    Data Properties

133 participants provided at least partial answers to the Factors in Eating Disorder Questionnaire, 127 provided complete answers. The data was found to be MCAR. Little's test: Chi-Square = 200.626, DF = 185, Sig. = .205. Missing data = 4.5% which is stated as the ideal conditions (McNiesh, 2016) as such all missing cases were treated to pairwise deletion.   The final ratio of participants to variables was over 4:1. 22 data points were missing. The Kaiser-Meyer-Olkin measure of sampling adequacy was over the recommended value of .5 (KMO = .838) Bartlett's test of sphericity was significant $x2 = (595) = 2611.715$ $p < 0.001$.

#### 7.5.5.2    Data Screening

Following data screening, several items were removed from the analysis as they yielded few correlations > 0.3 Question 5 'Early Puberty contributed to the development of my Eating Disorder', 17 'Academic Pressure did not contribute to the development of my Eating Disorder', 23 'An over – emphasis on what would happen if I didn't look after myself did not contribute to the development of my Eating Disorder', 32 'An Eating Disorder prior to developing Type 1 Diabetes contributed to the development of my Eating Disorder' & 37 'I had pre-existing Mental Health issues and this contributed to the development of my Eating Disorder'. Following the analysis items with communalities under .5 were removed which were 'Weight gain after I started insulin', 'Perfectionist attitude towards my blood sugar', 30 'A distorted body image', 19 'Conflict among my peer group' and 40 'A disturbed attitude to food'. (Please see appendix P for the correlation matrix)

#### 7.5.5.3    Extraction & Rotation

Following data screening Principal Axis Factoring Analysis with Promax rotation for oblique factors was utilised. Using the Kaiser – Gutman Criteria of retaining factors with an Eigen value over 1, a 5-factor solution was initially suggested that explained cumulatively 63% of the variance within the sample. Looking at the scree plot however the inflection point occurs at the fourth factor (please see figure 3.1 below). As such a parallel analysis was run based

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on minimum rank factor analysis (Timmerman & Lorenzo-Seva, 2011) using 500 randomly generated correlation matrices. The results suggested a four-factor solution and as such the analysis was rerun specifying this parameter. The resulting solution is presented in the tables below

*Figure 7.10: Scree Plot of Factors*
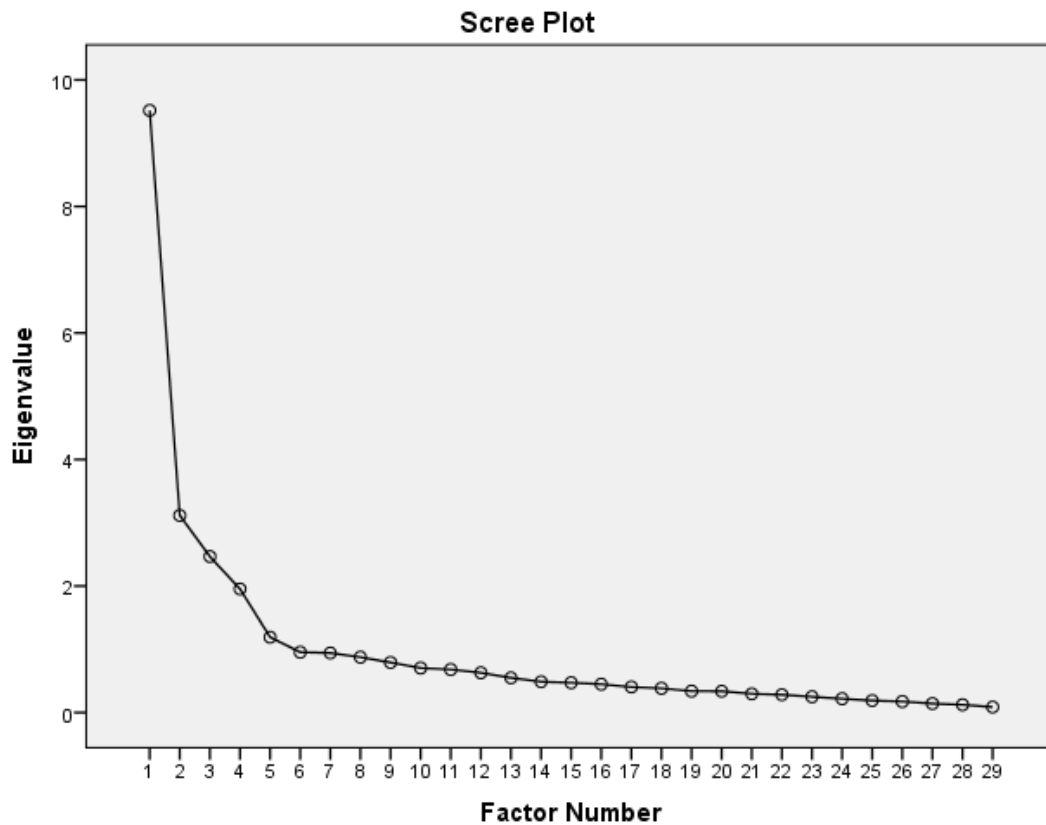


*Table 7.4: Eigenvalues and Variance Explained in 4 Factor Solution*

| Factor | Total Eigen Value | % of Variance | Cumulative % |
|--------|-------------------|---------------|--------------|
| 1 | 9.518 | 32.82 | 32.82 |
| 2 | 3.116 | 10.74 | 43.56 |
| 3 | 2.467 | 8.51 | 52.1 |
| 4 | 1.954 | 6.72 | 58.81 |

*Table 7.5: Pattern Matrix*

|  | Factor | | | |
|---|---|---|---|---|
|  | 1 | 2 | 3 | 4 |
| 34) I felt I couldn't talk to my parents | 0.826 | | | |
| 35) Family dysfunction | 0.817 | | | |
| 36) Feeling that I was not an individual within my family | 0.803 | | | |
| 11) Conflict at home within my family | 0.714 | | | |
| 39) My family was very rigid | 0.682 | | | |
| 29) Maternal concern with weight and shape | 0.557 | | | |
| 33) Disagreement with the way my parents dealt with my Diabetes | 0.537 | | | |
| 26) Examples of disturbed eating in my family | 0.473 | | | |
| 38 I didn't like the way my body looked | | 0.943 | | |
| 18) A sense of achievement at losing weight | | 0.873 | | |
| 25) Societal pressure to be thinner | | 0.823 | | |
| 6) Low self-esteem | | 0.701 | | |
| 9) Regardless of my Diabetes I was unhappy I was gaining weight | | 0.628 | | |
| 1) needed to feel in control | | 0.548 | | |
| 12) realisation I could lose weight quickly | | 0.524 | | 0.400 |
| 14) Fear of weight gain | | 0.457 | | |
| 7) Too much focus on my diet | | | 0.786 | |
| 28) Pressure from my health care professionals | | | 0.705 | |
| 3) restrained diet | | | 0.656 | |
| 27) Overeating/ binging, following episodes of hypoglycaemia | | | 0.596 | |
| 8) Attention to my weight from Medical Staff | | | 0.553 | |
| 10) Adherence to timed meals | | | 0.526 | |
| 22) Diabetes Burn Out | | | 0.507 | |
| 13) General Diabetes stress | | | 0.457 | 0.405 |
| 16) Denial that I had Diabetes | | | | 0.766 |
| 21) Embarrassment at injecting or testing | | | | 0.664 |
| 24) Fear of injecting and or self-testing | | | | 0.636 |
| 31) Resentment of having Diabetes | | | | 0.547 |
| 20) Fear of hypoglycaemia | | | | 0.522 |

The items that were cross loading in this solution were analysed in the factor in which they had the highest loading and then factor scores and scale reliability were computed

*Table 7.6: Scale Mean Scores and Reliability*

|          | N   | Mean | SD   | Cronbach's alpha |
|----------|-----|------|------|------------------|
| Factor 1 | 132 | 2.82 | 1.13 | 0.899            |
| Factor 2 | 132 | 3.89 | 0.99 | 0.877            |
| Factor 3 | 132 | 3.21 | 0.99 | 0.865            |
| Factor 4 | 132 | 2.78 | 1.04 | 0.817            |

As predicted the factors were related.

*Table 7.7: Factor Correlations (Pearson's r)*

| Factor | 1     | 2     | 3     | 4     |
|--------|-------|-------|-------|-------|
| 1      | 1.000 | 0.325 | 0.417 | 0.355 |
| 2      | 0.325 | 1.000 | 0.458 | 0.370 |
| 3      | 0.417 | 0.458 | 1.000 | 0.473 |
| 4      | 0.355 | 0.370 | 0.473 | 1.000 |

### 7.5.6 Confirmatory Factor Analysis Results

#### 7.5.6.1 Data Properties & Initial Analysis

161 participants completed the Factors in Eating Disorders Questionnaire at time 2. The final ratio of participants to items was 1:5.5. The data was missing completely at random. Little's MCAR test: Chi-Square = 451.527, DF = 447, Sig. = .431. The items and factor composition suggested by the EFA were entered into MPUS using the MLR estimator to account for missing data.

The initial analysis yielded a poorly fitted model: Chi square = (371) 640.457, p <0.001, normed chi square = 1.73, RMSEA = 0.067 (90% CI 0.058 - 0.076), Probability RMSEA <= .05 = 0.001. SRMR = 0.086. CFI = 0.744, TLI = 0.720,

#### 7.5.6.2 Data Screening

On further investigation it was apparent that several items did not meet the criteria of loading >0.05 and thus the following items were removed: from factor 1; 36) Feeling that I was not

an individual within my family, 29) Maternal concern with weight and shape and 26) Examples of disturbed eating in my family. From factor 2; 6) Low self-esteem, 9) Regardless of my Diabetes I was unhappy I was gaining weight and 14) Fear of weight gain. From factor 3; 3) Restrained diet, 22) Diabetes Burn Out and 13) General Diabetes stress and from factor 4; 31) Resentment of having Diabetes and 20) Fear of hypoglycaemia. This produced a better but not ideal fit Chi-Square Test of Model Fit: Chi Square (113) = 205.929, p <0.001, normed chi square = 1.82. RMSEA = 0.071 (90% CI = 0.056 - 0.087). SDMD = 0.068. Probability RMSEA <= .05 = 0.014. CFI = 0.864, TLI = 0.836.

### 7.5.6.3   Final Model

The modification indices produced by Mplus also suggested allowing items within the same factor to correlate so this was also entered into the analysis. This produced a much better fit (please see figure 7.2).

### 7.5.6.4   Goodness of Fit Statistics

#### 7.5.6.4.1   Chi Square

The chi square value for the proposed model = (108)123.409, p = 0.1475, (Scaling correction factor for  MLR = 0.9953) The chi square value for the independence model = (136) 819.678, p >  0.001 indicating that the proposed model represents a better fit of the data. The normed chi square value = 1.14 indicating a well-fitting model.

#### 7.5.6.4.2   Incremental Fit Indices

CFI = 0.977 indicating a well-fitting model,  TLI =  0.972 indicating a well-fitting model.

#### 7.5.6.4.3   Absolute Fit Indices

RMSEA = 0.03 (90% CI 0 - 0.052), Probability RMSEA < .05 = 0.930 indicating a close fit.

SMRSEA = 0.059 also indicating good fit.

*7.5.6.4.4 Factor Composition*

*Table 7.8: Standardised Loadings, Standard Error, Estimated Standard Error and P –*

*Values*

|  | Loading | SE | Est/SE | P- Value |
|---|---|---|---|---|
| Factor 1 | | | | |
| 34) I felt I couldn't talk to my parents | 0.746 | 0.047 | 15.892 | <0.001 |
| 35) Family dysfunction | 0.733 | 0.050 | 14.547 | <0.001 |
| 11) Conflict at home within my family | 0.641 | 0.062 | 10.306 | <0.001 |
| 39) My family was very rigid | 0.754 | 0.059 | 12.569 | <0.001 |
| 33) Disagreement with the way my parents dealt with my Diabetes | 0.786 | 0.040 | 19.598 | <0.001 |
| Factor 2 | | | | |
| 38) I didn't like the way my body looked | 0.487 | 0.139 | 3.517 | <0.001 |
| 18) A sense of achievement at losing weight | 0.724 | 0.090 | 3.517 | <0.001 |
| 25) Societal pressure to be thinner | 0.582 | 0.099 | 5.883 | <0.001 |
| 12) Realisation I could lose weight quickly | 0.599 | 0.098 | 6.093 | <0.001 |
| Factor 3 | | | | |
| 7) Too much focus on my diet | 0.641 | 0.077 | 8.303 | <0.001 |
| 3) Restrained diet | 0.613 | 0.089 | 6.868 | <0.001 |
| 28) Pressure from my health care professionals | 0.435 | 0.091 | 4.787 | <0.001 |
| 8) Attention to my weight from Medical Staff | 0.531 | 0.106 | 5.024 | <0.001 |
| 10) Adherence to timed meals | 0.697 | 0.072 | 9.669 | <0.001 |
| Factor 4 | | | | |
| 16) Denial that I had Diabetes | 0.598 | 0.091 | 6.547 | <0.001 |
| 21) Embarrassment at injecting or testing | 0.625 | 0.099 | 6.340 | <0.001 |
| 24) Fear of injecting and or self-testing | 0.587 | 0.088 | 6.703 | <0.001 |

*Table 7.9: Scale Scores and Reliability Analyses*

| Factor | Mean Score | Standard Deviation | Cronbach's Alpha |
|--------|-----------|--------------------|--------------------|
| 1 | 3.04 | 1.12 | 0.86 |
| 2 | 4.33 | .73 | 0.69 |
| 3 | 3.5 | .9 | 0.73 |
| 4 | 2.57 | 1.04 | 0.61 |

*Figure 7.11: Confirmatory Factor Model, Standardised Results – Graphic Representation*
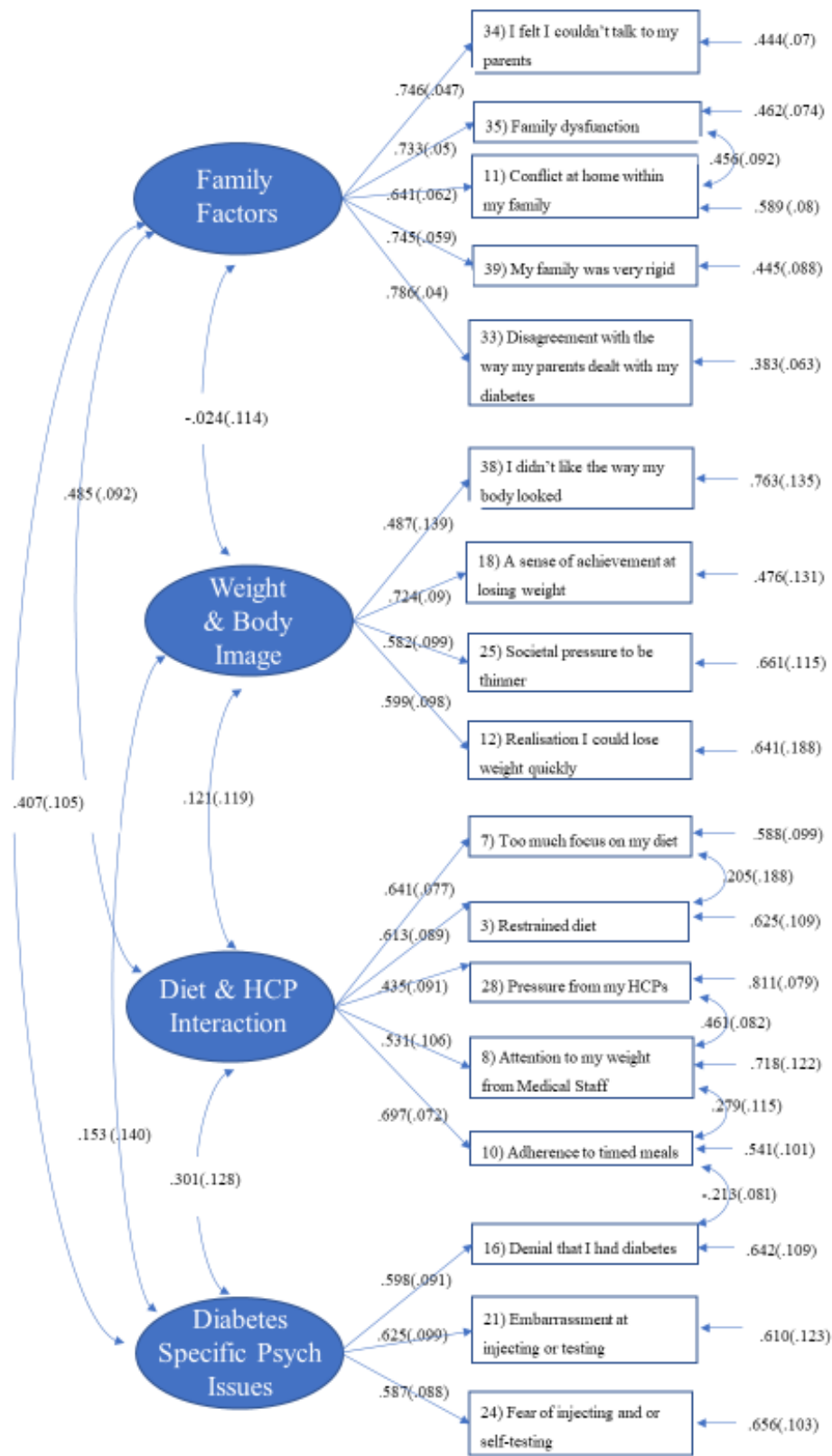
**7.6    Discussion**

As hypothesised there was an underlying structure to the attributions that participants gave for ED development and this was suggested by an exploratory factor analysis and then a confirmatory factor analysis. Initially a 5-factor structure was suggested but a monte carlo analysis suggested that the structure should be reduced to 4. The 4 confirmed factors related to issues surrounding 1) the family, 2) weight and body image, 3) Diabetes diet and HCP interactions and 4) Diabetes specific psychological issues. These factors had reliability values ranging from acceptable to excellent.

The first confirmed factor demonstrated how important the family environment is to ED development. The items included were 34) I felt I couldn't talk to my parents, 35) Family dysfunction, 11) Conflict at home within my family, 39) My family was very rigid and 33) Disagreement with the way my parents dealt with my Diabetes. This factor highlights how important family intervention may be for these patients. It should be noted that family factors are implicated in a number of T1D outcomes (please see chapter 4) further suggesting that this type of intervention is warranted. The second confirmed factor and the factor on which participants scored the highest was related to weight and body image issues and was composed of the items 38) I didn't like the way my body looked, 18) A sense of achievement at losing weight, 25) Societal pressure to be thinner and 12) Realisation I could lose weight quickly. It could be argued that these relate to classical risk factors with the added ability that those with T1D have in order to lose weight quickly. While it is understandable that approaching weight loss via insulin omission is sensitive it may be that more work needs to be undertaken around how to address this issue. The third confirmed factor Diabetes Diet and HCP Interactions contained the items 7) Too much focus on my diet, 3) Restrained diet, 28) Pressure from my health care professionals, 8) Attention to my weight from Medical Staff and 10) Adherence to timed meals. This factor suggests some kind of interaction between perceived dietary rigidity necessary for T1D and a negative clinical environment that combines to form a risk factor for T1ED development. A negative clinical experience has been shown to affect a number of adverse T1D related outcomes. (Please see chapter 4) The final confirmed factor Diabetes Specific Psychological issues contained the items 16) Denial that I had Diabetes, 21) Embarrassment at injecting or testing and 24) Fear of injecting and

or self-testing. This factor highlights that there are very specific psychological aspects to both being diagnosed with and dealing with the daily regimen of T1D that preclude the development of T1ED.

Despite do not attends being more prevalent in this demographic as reported in chapter 4, a significant majority had approached HCPs for help which is opposite to the attitudes of HCPs who often assume that these patients are not willing to discuss issues with them (Teirney et al., 2009). Respondents had also approached a number of health care professionals to report their difficulties, in the UK the most approached was the GP which is also concerning as, of the multidisciplinary team, it is feasible that the GP is the least specialised member and no research has been undertaking researching on how T1ED presents in primary care.

Participants, similarly to the pilot study also reported high levels of psychological comorbidity with only just over 20% having no other diagnoses. This is concordant that levels of mental illness are higher in those with T1D anyway (please see chapter 4). That morbidity is so high and also so varied, may go some way to explain why treatment is so difficult for this demographic. (please see chapter 8) Also bullying due to weight was reported by over half the participants while bullying because of T1D was present in 40% suggesting that school based interventions may also be justified.

### 7.6.1 Nomenclature and Diagnoses: The Importance of the term 'Diabulimia'

Worryingly over 40% of participants who perceived themselves to have an ED had not been formally diagnosed and of those who had EDNOS was the most common singular diagnoses, this is even more alarming given that nearly all of the participants had reached out for help. Of those who had been diagnosed, comorbidity was common, in some cases patients reported 4 concurrent ED diagnoses. This provides further evidence for the idea that T1ED is not homogenous, rather multiple clinical behaviours may be seen. When asked what the participants perceived their diagnoses to be, the majority stated Diabulimia or a comorbidity with Diabulimia and over 80% of the respondents had deliberately reduced or omitted their insulin in order to lose weight. This is not reflected in the diagnostic categories attributed to them and represents a substantial disconnect between what they feel they are suffering with and what they are diagnosed with. The term has gained recent notoriety due to media events

such as the release of the BBC documentary *Diabulimia: The World's most Dangerous Eating Disorder* (BBC, 2017) but before this, was used most frequently in the T1D community. The lack of a recognised clinical diagnosis appears to be massively problematic for those who consider themselves Diabulimic and even for those who don't but recognise that the regimen demands of T1D contradict with standard ED treatment and feel that AN, BN and EDNOS are not appropriate descriptors for their experience.

Recently the British Psychological Society's division of Clinical Psychology released a position statement regarding the usefulness of organising around diagnostic labels

> The DCP is of the view that it is timely and appropriately to affirm publicly that the current classification system as outlined in the DSM and the ICD, in respect of the functional psychiatric diagnoses, has significant conceptual and empirical limitations. Consequently, there is a need for a paradigm shift in relation to the experiences that these diagnoses refer to, towards a conceptual system which is no longer based on a 'disease' model. (BPS, 2013, p. 1)

They go on to claim that the disease model promotes a number of negative outcomes for patients including stigmatisation surrounding a clinical diagnosis which can negatively affect self-esteem, disempowerment over disagreements with diagnoses, marginalisation of the patients own experience and how diagnoses affect the decision-making processes of Health Care Professionals (BPS, 2013). It maybe that a paradigm shift would be useful, but it is important to recognise that *not* having a recognised clinical diagnosis with clinical symptomology can be equally as problematic. In a recent paper Hastings et al. found that those with Diabulimia see themselves as having a 'unique illness identity' of which 'insulin omission as a defining feature of their illness identity that distinguished them from other eating disorder groups' (Hasting et al., 2016, p. 2). Furthermore, participants also described a similar pattern when comparing themselves to those with T1 who were not concurrently suffering with an ED. These patients perceive their identity as isolating, a barrier to

appropriate support and not helpful to recovery in the context of the current healthcare system (Hasting et al., 2016[14])

At a group level, identification as 'Diabulimic' may promote several positive strategies for recovery. Cruwys et al. (2014) found that ED group identification could be the basis for 'normative change'. Using a group intervention protocol, they suggested that the shift from ED identification to recovery identification reduced thin – ideal internalisation, body dissatisfaction and dieting intentions via the norms of the group becoming more recovery focussed during the course of the intervention. Further positive effects of a 'recovery identity' whereby an ED sufferer shifts their perspective of themselves from someone as actively ill to someone working on recovery, have been found in lower levels of relapse and higher levels of engagement in treatment (Buckingham, Frings & Albery, 2013; Beckwith, Best, Dingle, Perryman & Lubman, 2015).

In Diabulimia, benefits of group identity also seem apparent. Members of a T1ED support group reported that the online group provided protection from the isolation of their conditions by providing an alternative, supportive and understanding community of in which there was a strong shared identity. The authors state:

> The support offered by those who share a sense of identity was perceived as qualitatively different from outside the group… Specifically participants felt that they could share experiences without being judged and that they could receive the emotional support and encouragement from group members that was absent from their other support networks (however well -intentioned the support offered may be) (Hastings et al., 2016, p. 5)

The group also validated the feelings of members, allowing them to identify as having this new and different condition. They also used the group as the primary source of illness related

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[14] Appendix B

information both related to regimen issues such as useful medications and the potential consequences of not recovering:

> Participant: It's like the start point do I look it up on google or go to the GP, no I ask the group if that makes sense, it's like the first thing I do
> Interviewer: That's great, and why is it the first place you go to over other places? Participant: Because no one knows more about Diabulimia than Diabulimics (Hasting et al 2016, p.5)

Other qualitative research has reported similar themes. A recent blog analysis has shown that healthy, understanding networks are vital for successful recovery and for Diabulimia that incudes peer support. Furthermore, these networks may encourage identity change from a 'sufferer' to a 'supporter' as one participant stated:

> They are women who, not only help encourage me to live my best life in harmony with my diabetes, but also provide me with valuable wisdom, guidance, and most importantly, a supportive shoulder to lean on when needed (Staite et al., 2018 please see appendix, p. 39)

Given then, that the use of the term 'Diabulimia' is validatory and provides access to inavaluable social resources for patients, it would seem sensible that legitimising it with a clinical diagnosis or something more formal would help sufferers further particularly as the usage of a non-medical term when engaging with medical professionals provokes a range of reactions, some patients even reporting that HCPs have claimed 'that's made up on the internet' (Allan, 2015, p. 100[15]). Furthermore, given that T1EDs represent a rarity, finding HCPs who specialise or have expertise in this area is difficult. This is felt acutely by participants in qualitative research on this subject who describe feeling ostracised by HCPs, that the treatment they receive is not suitable for their comorbidity and that clinicians are unwilling to accept that their ED is symptomatically distinct. As one participant stated

> I've tried counselling and therapy but none of them had a clue what Diabulimia was so they weren't much help whatsoever. Everyone I tried to explain to

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[15] Appendix E

they'd just ignore me and talk about other eating disorders that I may have
(Hastings et al 2016, p. 4)

Other qualitative research has found similar attitudes. McDonald et al. 2017[16] please see appendix for full article) reported that most of their participants experienced more negative than positive interactions with HCPs and that not only were these interactions unhelpful, in some instances they were potentially negligent. As one participant stated:

Then there were the incidents with things like them forgetting to give me my insulin, forgetting to do blood tests… I was in there to be treated for my eating disorder and part of that is struggling with taking it so for me to remind them that I needed to do it or that they needed to test my blood, was just ridiculous (McDonald et al., 2017, p. 226)

With this kind of treatment experience, it is perhaps understandable that in this demographic there is a level of mistrust towards medical professionals. Participants went on to state that there were issues with HCPs understanding the uniqueness of the situation they were in as T1ED and that they expected HCPs to just 'not get it'. They also complained that there was a lack of collaboration between teams which led to misunderstandings and inappropriate treatment. It would appear apparent then that HCPs are unfamiliar with the workings of T1ED and insulin omission. A distinct diagnosis would promote futher understanding, after all how do you train HCPs for something that does not officially exist.

These sentiments were inadvertently validated by HCPs (ED specialist) who in one incidence essentially stated that T1D was simply another comorbidity that should be dealt with in the same way one would deal with a condition such as depression. This demonstrates a clear conflict between what the patients and those treating them recognise as appropriate attitudes to the comorbidity (MacDonald et al., 2017). Perhaps worse still Tierney et al. reported that HCPs may be openly hostile to patients with T1ED. One of her HCP participants reported

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[16] Appendix C

> You can tell that some people won't give them the time of day and you have to be careful yourself not to think, oh no, he's in again (Tierney et al., 2009, p. 338)

Having a recognised clinical description would surely lessen some of this anxiety. Other healthcare professionals in this and other samples appear to recognise that there were potential skills and training gaps and there was a need for specialists in each discipline (Eating Disorders and Diabetes) to have at least a rudimentary understanding of the other. There was also a recognition in some of the HCPs that T1ED was challenging and this was compounded by limitations and constraints placed on their positions in the overarching NHS structure, the authors state:

> By far, the greatest challenge was working with problematic thoughts and behaviours; i.e. insulin omission, difficulties in engaging, high anxiety, unrealistic recovery goals, diagnostic ambiguities, transient nature of barriers put up and threats of self harm and suicide. Other concerns included responsibility of clinical risk and the anxiety inducing effects on their own team members. (MacDonald et al., 2017, p. 227)

The HCP participants recognise that part of the solution to these challenges is close multidisciplinary working which helps overcome treatment barriers but simultaneously they also reported that challenges in treating T1EDs extended to issues caused within the team due to patient centric effects. This included the process of 'splitting' whereby a patient will play one health care professional off against another often using nefarious means such as lying in order to achieve a desired outcome. As one Diabetes specialist HCP noted:

> Because often the patients know a lot about diabetes, so they can blind the psychiatrist or eating disorders person with the diabetes… They will cheat the nurse (MacDonald et al., 2017, p. 228)

Other qualitative research in HCP subjects has shown similar frustrations. Teirney et al (2009) highlighted that within a sample of Diabetes Specialists, patients with T1ED were anxiety inducing and identified 4 main themes; classification, detection, treatment and lack of training. It would make sense however that much of this is due to a lack of any concrete

diagnosis of insulin omission as a weight loss behaviour, Splitting is much easier when there is confusion.

An interesting finding of this research is that many of the Diabetes specialists separated 'classic' eating disorder behaviour such as bingeing and purging or severe food restriction to those more generally seen in those with T1, this potentially shows a significant difference between the attitudes of those working in Diabetes and those working in ED who may simply view diabetes as a mostly irrelevant side note. It also coincides with patient attitudes towards diagnosis. The Diabetes specialists in the Teirney et al. sample appeared to have a good working knowledge of the potential psychological mechanisms behind T1ED cases, citing focus on nutrition, obsessive diets for glycaemic control, resentment of T1D, weight gain associated with good control and were reflective about how their own practice may have been iatrogenic. This having been said they were acutely aware that they were not trained in this area, had a serious lack of expertise and were often afraid to bring up insulin omission in appointments in case they broke established rapport, this was reported in other samples also ((MacDonald et al., 2017; Balfe et al 2013). This may represent a missed opportunity as research has suggested that it is just this rapport that makes it more likely for sufferers to 'open up' to HCPs (Balfe et al 2013). Perhaps due to the relationships fostered, HCPs in this sample reported trying to contain milder T1ED problems within the diabetes clinic. The authors highlight that

> 'psychologists and other experts in eating disorders did not always have a good understanding of these problems within the context of diabetes' (Teirney et al., 2009)

This may be indicative of the core issue, despite recognising that they are ill equipped to either recognise or treat these issues, Diabetes specialists are reluctant to bring in outside help. When that help is brought in it is also seen to be ill equipped. A diagnostic term unique to this condition would surely assist in these aspects.

As such it would seem of prime importance that the we acknowledge 'Diabulimia' as a term. Regardless of how academics, clinicians or researchers may feel about the term, it has obviously been adopted by those who omit insulin for weight loss and given that it describes

a specific and unique illness, with distinct behavioural features it would appear that it does have utility.

### 7.6.2 Strengths

This is the first piece of research to examine patient attributions as to what caused their ED to develop. It used a large cross sectional, global sample which provides a wider comprehension of issues facing this demographic. It has hopefully gone some way to address a significant gap in the literature. Furthermore this research should help our understanding of where these patients appear in service and the issues they are likely to be facing when they do reach out for help.

### 7.6.3 Limitations

The pilot project on which the current study is based utilised was conducted in 2013 as part of a bachelors degree and as such it maybe that newer suggestions in the literature as to why EDs develop in T1D would have been suggested and thus a different factor structure concluded. Also, this was an international sample and as such the results should not be generalised to a specific geographical region. It should also be noted that the sample used was recruited via the internet whereby issues around T1ED are discussed openly and this may have biased results. Similarly, there were not enough male participants in this study to generalise the results to males. Although it could be argued that there is not enough literature on the male experience of T1ED to currently generalise any conclusions.

A substantial limitation of the current study is that although these suggestions are in the literature they are not necessarily coming from patients, these attributions are suggested by researchers. A meta-synthesis of qualitative data may have provided different and perhaps more valid attributions.

### 7.6.4 Future Directions

It appears obvious that the terminology that HCPs use in relation to these patients should be addressed and the taxonomy of EDs in T1D should be the subject of considerable future study given that unique Diabetes specific factors feature so prominently in patients' attributions as

to what caused development. It may be useful to redefine what EDs in T1D are separately from EDs in the general population given the substantial differences in both aetiology, physical processes and outcomes. Furthermore, as these patients are accessing a wide variety of services in order to ask for help it would be prudent to consider some kind of training programme for not only health care professionals but also those in related positions such as social services and education.

## 7.7    Conclusion

The most important finding of the current study is that while there are latent factors that those with or recovering from T1ED attribute to their ED, T1D is inextricable from most of these factors. The implications of this are that one cannot address factors in T1ED without the T1D which may go some way to explain why treatment for this group so often fails. Furthermore, it is obvious that the diagnoses that are being attributed to this group are missing the main diagnosis that they feel they have which is Diabulimia. While on the surface this may seem like an issue of nomenclature it is beyond that. Diabulimia is a descriptor of a unique behaviour that only effects T1D and given that these patients are also highly comorbid for other mental illnesses one can only hypothesise on how having an undiagnosable illness influences these patients.  They are presumably approaching their multitude of HCPs with this terminology. The next stage of research effort in this area should seek to develop a new taxonomy that takes into account the unique aspects of ED development and behaviour of those with T1D.

# 8    Treating Eating Disorders in Type 1 Diabetes

## 8.1    Introduction

There have been repeated calls for both psychological support and Diabetes education programmes from patients, caregivers and clinicians dealing with Type 1 Diabetes. Standard models of T1D care often leave the entirety of management down to what the patient and those close to them can ascertain for themselves with sporadic input from specialists. Furthermore, accessing high quality mental health support is increasingly difficult. That these resources are lacking for treatment as usual is obvious but the near complete absence of resources for the combined diagnosis of T1ED is alarming, especially as the accompanying consequences are devastating. There are various structural explanations as to why there appear to be no defined programmes or treatment pathways for T1ED in the UK; lack of resources, little professional training, issues surrounding diagnosis, changing guidelines, clinical responsibility, funding, lack of specialist knowledge, insulin omission as a clinical feature and overall recognition of the problems to name but a few (BBC, 2017; NICE, 2017a/b; Diabetes UK, 2017; Tierney et al., 2009; MacDonald et al., 2017; Goebel- Fabbri, 2017). Patient centric issues must also be taken into consideration, T1EDs are less likely to show up in clinic, have a poorer opinion of the clinical environment, are less trusting of HCPs, often have comorbid diagnoses such as anxiety and may have significant physical complications making travel to appointments difficult (Allan, 2015; NICE, 2017b; Tierney et al., 2009; MacDonald et al., 2017; Goebel-Fabbri, 2017).

### 8.1.1    Treatment Recommendations

Certain influential figures in the domain of T1EDs have made recommendations though, perhaps most notably the clinical academic Anne Goebel-Fabbri who treated numerous T1ED patients during her tenure at the Joslin Diabetes Centre, Harvard. She was primary investigator on 3 papers outlining how to treat these patients. In the first published in 2002 she states that a large multidisciplinary team of specialists from both T1D and ED must be involved and they must communicate fluently and regularly. She also argues for the hospitalisation of patients until they are stable enough to benefit from psychological input. Treatment itself she argues should be based around incremental goal setting regarding insulin

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dosing (she warns that intensive insulin titration is not suitable at this stage), DKA avoidance, increasing and regulating food intake and increasing BG monitoring.  She also highlights the importance of discussing T1D specific aspects of ED recovery such as the initial weight gain which may come from water retention, and the temptation to over treat hypoglycaemic episodes (Goebel-Fabbri et al., 2002).  In 2009, the paper Goebel – Fabbri et al. published was specifically focussed on the outpatient treatment of T1ED. The sentiments of the earlier paper are echoed but the importance of rapport with the patient who is often afraid of judgement was emphasised as was the concurrent abuse of other medications. This paper also produces a table of guidelines where food restriction and insulin restriction are differentiated in term of treatment recommendations. In a 2009 paper solely authored by Goebel-Fabbri, she argues that clinicians should be aware of further Diabetes specific stressors such as fear of hypoglycaemia, perfectionist attitudes towards BG control and the relationship between hyperglycaemia and depression (Goebel-Fabbri et al., 2009). Although Goebel-Fabbri appears to have concentrated her efforts around outpatient treatment, recommendations have been made regarding inpatient protocols. Bermudez et al. (2009) highlight that those with T1ED will present differently for IP treatment where aspects such as hyperglycaemia, DKA and severe dehydration should be expected and further T1D issues for management include T1D related complications. They also state that insulin omission is incredibly important to watch out for and recommend that patients do not be admitted to facilities that are not equipped to cope with the unique needs of T1ED. In relation to ward treatment, the authors are eager to point out what differentiates these patients, namely that initially, insulin dosing should be the responsibility of staff and BG control should be attained incrementally, pumps should be replaced with MDI and that weight restoration is not a necessary goal. They also provide a fairly comprehensive table of recommendations regarding Diabetes specific aspects of IP treatment. Interestingly these authors do not refer to patients by a standard ED diagnosis (AN, BN..etc) but rather by the moniker ED-DMT1 which stands for Eating Disorders in Diabetes Mellitus Type 1. This indicates that perhaps to this research team at least the commonality comes from Type 1 Diabetes, diagnosis type, to a large extent is irrelevant.

Much of the aforementioned recommendations were reflected in an update to the NICE guidelines for Eating Disorders published in 2017. These guidelines represented a leap

forward in the treatment of T1ED although there were still limitations (please see appendix A for a further discussion of this topic).

## 8.2 The Current Review

Any researcher who is involved in this discipline could not fail to notice that nearly every paper regarding T1ED highlights how difficult this demographic is to treat. The purpose of the current review was to provide a 20 year overview (1998 – 2018) of reported treatment programmes for those suffering with Type 1 Diabetes and an Eating Disorder in order to ascertain what types of interventions have been suggested, whether they have been modified for Type 1 Diabetes, whether HbA1c was measured or insulin omission considered, what measurement instruments have been used and what  the length of treatment was. The discussion then uses the findings of this review and that from elsewhere in this thesis to suggest whether or not the perceived wisdom that this group are harder to treat is valid.

## 8.3 Methodology

### 8.3.1 Search Terms and Databases

A comprehensive search was run using the MEDLINE & PubMed Databases using the following terms. (Diabetes Mellitus, type 1* [MeSH Terms]) AND (Feeding and Eating Disorders* [MeSH Terms]), Diabetes mellitus, type 1/ psychology [MeSH Terms], Diabetes mellitus, type 1/ psychology [MeSH Terms] AND "Eating Disorder*" [All Fields] and Feeding and Eating Disorders* [MeSH Terms] AND "Type 1 Diabetes" [All Fields]. A search was similarly run on PSYCH INFO using the terms 'Eating Disorders', 'Type 1 Diabetes', 'Type 1 Diabetes Mellitus'

Following this a hand search of the references section of relevant papers was used to identify any other papers which may have been unidentified by the original search.

### 8.3.2 Inclusion/ Exclusion Criteria

Inclusion Criteria

1)      English Language

2)      Discusses a Treatment Protocol for Eating Disorders in which there are Type 1 patients.

Exclusion Criteria

1)      Review Articles (although used in identifying references)

2)      Individual Case Studies.

3)      Does not Discuss a Treatment Protocol for Eating Disorders in which there are Type 1 patients.

4)      Prevention Programmes

5)      Studies with Diabetes Type not differentiated

6)      Studies not in English Language

293 papers were suggested by the literature search for review. Of these 49 duplicates were removed. 2 papers were added via hand search and 2 papers utilising the same treatment protocol and sample were combined (please see figure 8.1)

### 8.3.3   Data Extraction

The follow data was extracted from the literature: Author/ year/ country of origin, number of participants, gender, age range or mean age, diagnoses, type of treatment, whether the programme was designed or modified for T1D, whether HbA1c or insulin omission was measured, what ED measurements were used, whether the programme improved outcomes, if there was a control group, the length of treatment and whether there was any post treatment follow up.

Studies between 2001 – 2016 were read by 2 postgraduate research assistants who wanted to gain experience in the methodology of systematic reviewing and reviewed again by the researcher to ascertain suitability for inclusion in the review. Studies between 1998 – 2001 and 2016 - 2017 were reviewed by a PhD student who wanted to gain experience in the

methodology of systematic reviewing and reviewed again by the researcher to ascertain suitability for inclusion in the review.

*Figure 8.1: Flowchart*
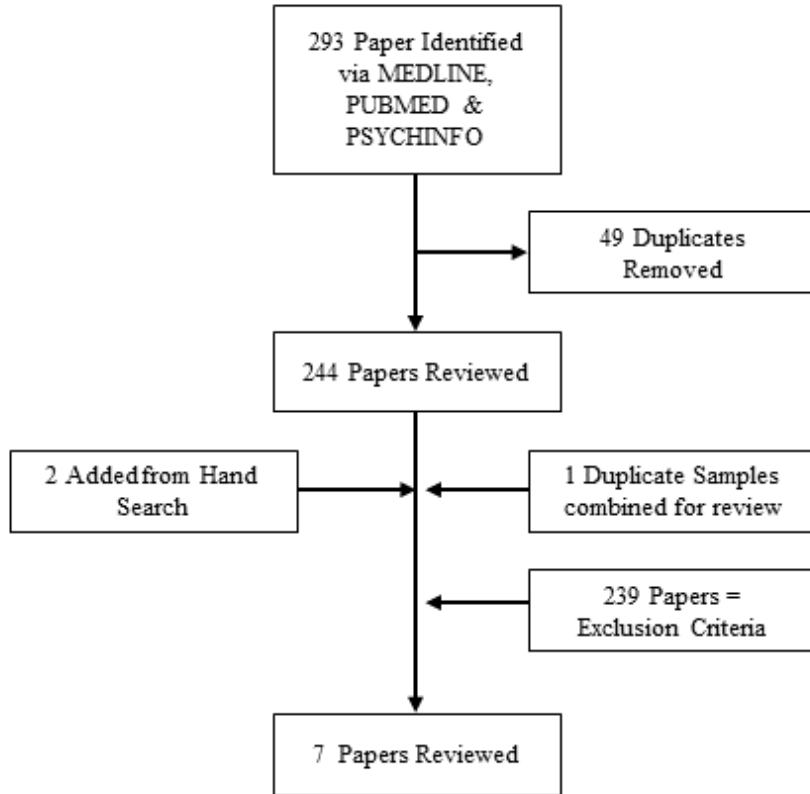
## 8.4    Results

*Table 8.1: Features of the Papers Reviewed*

| Author/ Year/ Country of Origin | n (m/f) | Age Range or x | Diagnoses | Protocol | HbA1c or IO Measured | Eating Disorder Measures | Control Group | Treatment Length | Post Treatment Follow Up |
|---|---|---|---|---|---|---|---|---|---|
| Colton et al 2015 (Canada) | 32f* | x=25.6 | BN/AN/ EDNOS | Day patient CBT | Yes | EDE | 801 no T1D | 6 – 8 weeks EDNOS, 10 – 14 weeks AN | No |
| Dickens et al 2015 (US) | 29f | x= 25.55 | BN/ AN/ EDNOS | Inpatient | Yes | EDI | No | n/s | No |
| Custal et al 2014 (Spain) | 20f | x=25.3 | AN/ BN/ BED/ EDNOS | Day patient CBT or Inpatient treatment | Yes | EDI & Semi Structured Interview | 20 non T1d | 16 sessions outpatient CBT or 3-month day patient | Mo |
| Takii et al 2002; 2003 (Japan) | 28f | IP x= 23.8 | BN/ BED | Outpatient Counselling or Integrated Inpatient Therapy | Yes | Clinical Interview & EDI | No | n/s | 6mo, 24mo, 36mo |
| Olmsted et al 2002 (Canada) | 50f | 12 - 20 | >cut- off | Psychoeducation | Yes | EDI, EDE & DSED | 35 T1D | 6 sessions | 4wks, 6mo |
| Alloway et al 2001 (Canada) | 8f | 32.5 | subED | Psychoeducation | Yes | EDI, EAT modified for T1D | 6 T1D | 6 sessions | 1 mo N=5, 6 mo |

IO = insulin omission, n/s = not stated, IP = Inpatient, EDE = The Eating Disorders Examination, EDI = The Eating Disorder Inventory, DSED = The Diagnostic Survey for Eating Disorders, EAT = Eating Attitudes Test, sub= subthreshold, mo = months
*this is the number of people who attended the programme > 4wks,

*Table 8.2: Content and Success of Programmes Reported*

| Paper | Designed for T1D | Modified for T1D | Improvements | Modification |
|---|---|---|---|---|
| Dickens et al (2015) | Yes | Yes | Yes in both ED symptomology and HbA1c | In line with current treatment recommendations (Goebel-Fabbri, 2009), clinical staff at the multi-disciplinary residential treatment center included licensed psychotherapists and their supervisees, registered dieticians, psychiatrists, physicians (including an endocrinologist), registered nurses, and mental health technicians. Therapeutic approaches included cognitive behavioral, person-centered, family, and feminist approaches. The primary goal of treatment was to teach patients cognitive and emotional skills to manage eating disorder thoughts and urges that interrupted diabetes management. Patients received medical monitoring of blood sugar levels several times daily by nursing staff. (Dickens et al., 2015, p 3)<br><br>Diabetic patients were taught to practice intuitive eating by administering insulin dose for half of their portion prior to eating, and then administering the remainder of the dose following the meal according to the amount they consumed while attending to internal hunger and satiety cues. Meals were eaten under supervision of trained staff. (Dickens et al., 2015, p 4) |
| Colton et al (2015) | No | Yes | 6 = Good outcome 14 = Intermediate outcome 12 = Poor Outcome | Day hospital treatment protocol was the same for those with and without diabetes, with the following modifications for those with type 1 diabetes: endocrinology consultation and follow-up throughout; expectation of full adherence with blood-glucose monitoring and insulin administration; and further diabetes education is provided as needed. (Colton et al., 2015, p313) |
| Custal et al (2014) | n/s | n/s | 50% Partial or Full Remission | No modifications stated. |
| Takii et al (2002;2003) | Yes | Yes | | While treating those with T1D and BED, which was diagnosed in the absence of ICBs Takii et al., (2002) utilised a counselling session at the time of screening which was focused on T1D. They state that this protocol is as follows<br><br>'1. Bring out patient feeling about diabetes by listening for a sufficient amount of time that the patient gets a feeling of ''ventilation''.<br>2. Help the patient recover from the injured self-esteem.<br>3. Contradict too pessimistic an image of diabetes and present a hopeful and acceptable image of it. |

| Paper | Designed for T1D | Modified for T1D | Improvements | Modification |
|---|---|---|---|---|
| | | | | 4. Teach the patient the importance of finding the easiest and most suitable form of diabetes self-care. |
| | | | | 5. Encourage recovery/improvement of communication between the patient and family members, especially the mother' (Takii et al, 2002., p 293.) |
| | | | | It should be noted that in the event that 1st episode counselling did not produce the desired outcome of a 1% drop in HbA1c in 6 – 12 months the patients were referred to an Integrated Inpatient Programme which focused on limiting control over injecting insulin until better eating behaviour was established. The authors give an outline: |
| | | | | 'The basic elements of integrated inpatient therapy |
| | | | | I. Recovery period for the mind and body |
| | | | | a. Recovery from mental and physical fatigue and depression |
| | | | | b. Normalization of biorhythms |
| | | | | II. Modification of behaviors and cognition |
| | | | | a. Improvement of eating behavior |
| | | | | (Therapist control stage) |
| | | | | Decision, by the patient, of the initial calorie intake and insulin dose |
| | | | | Completely and regularly eating meals |
| | | | | Not eating snacks or confectioneries |
| | | | | Incremental increases in the volume of the meal (200 kcal at a time) |
| | | | | (Patient control stage) |
| | | | | Free ingestion training |
| | | | | Snack training |
| | | | | Eating out training |
| | | | | Staying at home training |
| | | | | b. Promoting glycemic control competence |
| | | | | Self-measurement of blood glucose |
| | | | | Self-injection of insulin |
| | | | | Practical coaching and training in adjustment of the insulin dose |
| | | | | c. Modification of cognitive aspects |
| | | | | Individual counseling |
| | | | | Group therapy for eating disorders |
| | | | | III. Restoration of family relationships |
| | | | | a. Spontaneous restoration process |
| | | | | b. Family counseling |

| Paper | Designed for T1D | Modified for T1D | Improvements | Modification |
|---|---|---|---|---|
| | | | | c. Coaching family members: especially in how to understand and cope with the patient (Takii et al., 2003, p 351)' |
| Olmsted et al 2002 | No | Yes | Yes in both ED symptomology and HbA1c | The relationship between disordered eating and diabetes, body image concerns relating to diabetes , how eating attitudes and behaviour affect diabetes control, the dangers of insulin omission and family communication strategies. Strategies for change and a nondeprivaional , nondieting approach to normal eating were emphasised. (Olmsted et al., 2002, p.234) |
| Alloway et al 2001 | No | Yes | Not as an effect of the intervention | They state that 'slight modifications' were made but do not explain exactly what these modifications were and state that the programme had sessions on …eating disorders, "normal" eating, healthy body weights, assertiveness training, stress management, improving self- esteem, perfectionism, media and societal expectations of thinness, and enhancing body image. (Alloway et al., 2001. p2) |

### 8.4.1 Country of Origin

There were very few geographical regions represented in this study. 3 papers came from Canada, 1 from the US, 1 from Spain and 2 from Japan.

### 8.4.2 Sample Size

As is the case with most research in to T1ED there was a serious issue with sample sizing in most of these research articles. For example, Alloway et al. (2001) only used a sample size of 8, Takii et al. (2002) did extended their study with a sample size of 28 but this was divided into 10 BED and 18 BN patients, of which only 9 would accept inpatient treatment to be described in their later paper (Takii et al., 2003). Similarly, Custal et al. (2014) divide their 20 participants between diagnoses. Colton et al (2015) describe a sample of 32 who completed adequate treatment but it should be noted that the data from these participants were collected retrospectively over a 22 year period. Olmsted et al. (2002) reported the highest participant numbers at n= 50.

### 8.4.3 Gender

All treatment programmes exclusively treated females. Some researchers deliberately excluded males (Custal et al., 2014) although it should be noted that authors claim to do so due to such a limited sample. Colton et al. (2015) do state that 2 T1D men did come to their clinic within a 22 year period but they were deemed to not have an ED and so were excluded from research.

### 8.4.4 Age Range

Consistent with research that shows that in T1D Eating Disorders tend to manifest at older ages most of the research undertaken was with adults. Olmsted et al. (2002) used a younger cohort aged 12 -20, arguably there is a significant difference between the needs of a 12 and a 20 year old which may explain why the protocol was relatively unsuccessful.

### 8.4.5 Diagnoses

A variety of diagnoses were represented in the intervention literature. Alloway et al. (2010) utilised a sample of subED patients excluding anyone who qualified as having full clinical EDs. Takii et al. (2002) separated patients into either BN or BED depending on whether they utilised ICBs including insulin omission. Other researchers did not use diagnosed ED patients at all, preferring instead to rely on information gleaned from clinical interviews or cut-off points from ED screening interviews (Olmsted et al., 2002). Similarly to other research in this demographic however, even when diagnoses reported run the spectrum of ED disorder possibilities, insulin omission still appears to be the main feature of the illness. (Takii et al. 2002/2003; Custal et al., 2014; Dickens et al; 2015; Colton et al. 2015).

### 8.4.6 Type of Protocol

#### 8.4.6.1 *Outpatient: Counselling & Cognitive Behavioural Therapy*

Takii et al. (2002;2003) describe a treatment protocol whereby they separated patients who have BED to receive 1 session of counselling that is related to Diabetes. For BED they claim this intervention has significant effects on both behavioural and biological measures at 3 year follow up (please see below). Custal et al. (2014) describe providing their T1ED patients (who don't have AN diagnosis) 16 sessions of Cognitive Behavioural Therapy but they don't explain any further than that.

#### 8.4.6.2 *Outpatient: Psychoeducation*

Olmsted at al. (2002) describe psychoeducation as 'the provision of information about an illness or problem and strategies for changing or coping with it' (Olmsted at al., 2002, p.231). The format of their programme was a 6, 90-minute, weekly sessions whereby mothers and their T1D daughters were given information delivered by an ED specialist and a T1D specialist (subjects and their mothers in separate rooms) alongside a treatment manual adapted from a standard ED programme. They emphasise that the main approach used was 'nondeprivational' which encourages patients away from dieting behaviour. They found that following this intervention patients' attitudes towards food and body image improved but HbA1c and rates of insulin omission did not. Alloway et al. (2001) found that a

psychoeducation group was no more effective at reducing psychological distress or HbA1c than a waiting list.

### 8.4.6.3   Outpatient: Day Hospitalisation

Day hospitalisation represents a middle ground between outpatient and inpatient treatment. Patients attend intensive treatment during the day but are allowed home in the evening. Colton et al. (2015) provide a retrospective chart review of T1D patients who attended this programme, based on CBT principles and delivered via group therapy with a multidisciplinary team over a 22 year period. It should be noted however than less than half of patients offered this treatment accepted it.  Custal et al. (2014) found similar results. In a retrospective chart review they compared outcomes between those with T1D and those without who had attended treatment at a single ED unit in Spain. They subjected their AN patients' to day hospitalisation but do not extrapolate any further on that.

### 8.4.6.4   Inpatient Treatment

Takii et al. (2002; 2003) published two studies regarding the treatment of BED and BN in T1D. For those with BN they describe an 'integrated inpatient' programme. This programme consisted of many T1D specific elements such as deciding insulin dose administration and close supervision at meals regarding insulin. The authors argue that this inpatient protocol significantly improved HBA1c and ED behaviours including insulin omission to the extent that at 3 year follow up 78% of patients did not meet criteria for either full or subthreshold EDs.

Dickens et al. (2015) describe both significant ED and HbA1c improvements in a sample of T1EDs who went through a residential treatment programme at a clinic in the US. The authors report that the programme used a large multidisciplinary team which included an endocrinologist. Patients were closely medically monitored daily in relation to blood sugar. The programme involved multiple weekly individual therapy sessions, bi monthly or monthly family sessions, weekly nutritionist and psychiatrist appointments, multiple daily group therapy sessions and weekly MDT meetings to review progress. They authors do highlight

that the use of such an integrated approach makes it difficult to suggest which elements of treatments are the most successful.

### 8.4.7   Eating Disorder Measures

Alloway et al. (2001) used the EDI and the EAT to screen patients into their group psychoeducation programme for subclinical EDs and also added 2 questions regarding insulin omission. They found no significant differences between control and treatment conditions on these measures. Takii et al. (2002; 2003) also used the EDI but also a clinical interview based on the DSMIV which included questions on insulin. They found significant differences in ED behaviours using these instruments as a result of treatment. Olmsted et al. (2002) used a mix of ED measurements which they stated were modified for use in the T1D population, the EDI, the EDE and the DSED and they found a significant reduction in ED attitudes as a result of treatment. Custal et al. (2014) used the EDI and a semi structured interview. There were also questions asked about insulin omission. Dickens et al. (2014) also used the EDI and found improvements as a result of treatment. Colton et al. (2015) describe using the EDE initially to screen participants but they specify more behavioural measures in terms of outcomes. They measured both weight restoration and frequency of bingeing and purging to ascertain whether outcomes were good, intermediate or poor.

### 8.4.8   HbA1c & Insulin Omission

Alloway. (2001) excluded anyone who had ICBs as a indication of bulimia. They do not mention if that includes those who omitted insulin but they did record HbA1c at baseline. They found that following a psychoeducation program both the treatment and control had improved HbA1c as an effect of time concluding that there was no difference between groups. Olmsted et al. (2002) measured both HbA1c and frequency of insulin omission and they found no significant effect of treatment for either at 6 month follow up. Takii et al. (2002;2003) found that there were significant longitudinal effects of treatment whereby those with BN who did not enter IP treatment did not improve their HBA1c or insulin omission frequency while those in the treatment group recovered significantly and stayed as such through follow up.  For BED however one counselling session was enough to improve HbA1c at follow up. Custal et al. (2014) reported HbA1c levels and self-reported insulin

omission at baseline but did not provide any follow up data. Colton et al. (2015) reported on outcomes based on behaviours such as binging and purging but did not explain what ratio of those behaviours included insulin omission and despite measuring HbA1c at baseline they do not report it post treatment.

### 8.4.9   Control Group

Several researchers used a T1D control group but in some cases,  this was very small, Alloway et al. (2001) for example used a control group of 6. Olmsted et al. (2002) used a TAU group of N = 35 T1Ds. As seen in other research some authors utilise a non T1D control group stating that the only statistical difference is insulin omission. It should be noted that the authors claim that they have provided appropriate control they also state that nearly all (90%) of their Diabetic sample manipulated insulin for weight control (Custal et al., 2014). Colton et al. (2015) used a comparison group of 801 non Diabetics. Other researchers use no control group at all (Dickens et al., 2014). Of specific interest is Takii et al'.s assertion (2003) that 'leaving motivated patients untreated would be unethical' (p.354).

### 8.4.10  Treatment Length

Takii et al. (2002) describe the shortest intervention which was one session of counselling at the time of assessment, perhaps somewhat surprisingly the authors state that for with T1Ds and BED this is effective at long term follow up. Other researches attribute failure to shorter interventions, particularly those of a psychoeducational nature, both of those reported here were only 6 sessions long. (Alloway et al., 2001; Olmsted et al., 2002). Longer programmes extended to 3 months and that is certainly in line with standard CBT where programmes typically last no more than 3 months at 1 session per week (Custal et al., 2014). Even inpatient treatment may only last between 4 – 12 weeks (Custal et al., 2014; Dickens et al., 2016). Longer treatment length was associated with better outcomes according to Dickens et al. (2015) but no mention of actual length was given.

### 8.4.11  Post Treatment Follow Up

Follow up varied widely, Alloway et al. for example only utilised a 1month follow up with most of their participants and 6 month follow up with others, there was no significant

differences. Olmsted et al. also used a 6 month follow up time (2002). Takii et al. (2002;2003) followed up with their patient at 6, 12, 24 ad 36 months representing perhaps the most thorough post treatment assessments. Other researchers provide no follow up data (Custal et al., 2014; Colton et al., 2015; Dickens et al., 2015)

## 8.5 Discussion

### 8.5.1 Summary

There are very obvious problems with the canon of literature relating to treating Eating Disorders in Type 1 Diabetes. No publications in the last 20 years have come from the UK, this is significant and somewhat disappointing given that the most recent set of recommendations come from here (NICE, 2017). It would also be unwise to extrapolate any generalisations from any of this research given that taken as a whole it represents the treatment of only 167 patients. It is of real concern that none of these patients were males given the soaring rates of insulin omission in this demographic over recent years (please see chapter 2), and this may signal a looming crisis. Of the 7 papers reviewed, all focussed on adults apart from 1 (Olmsted et al., 2002) and while this is consistent with research that T1ED appears in adulthood there is research suggesting that insulin omission for weight loss is often present in both male and female adolescents. Diagnoses were varied but in line with other research reported in chapter 2, insulin omission was seen as the most prescient symptom.

Treatment modalities were also varied; there were 3 inpatient programmes, 2 day patient programmes and 2 psychoeducation programmes, a course of CBT and an at assessment counselling session reported. The inpatient protocols were the most successful and the psychoeducation programmes appear to be the least effective. Inpatient protocols explicitly based on Diabetes specific aspects of ED improved HbA1c and incidences of insulin omission but no other treatment was successful in improving these variables where they were reported. Of note is that one of the successful programmes stated that outcomes improved further with longer treatment. Treatment length, where reported, seemed to be relatively short with no interventions stretching longer than a period of 3 months. In terms of measurement instruments the EDI was the most common and although most authors highlight that they used either modified versions of instruments or added questions regarding insulin behaviours,

no papers utilised a Diabetes specific scale such as the DEPS-R to monitor outcomes. T1D control groups were utilised in some research but not in others and in some cases the control groups consisted of non Diabetics (please see chapter 2 for a larger discussion around the problems with these issues). Various follow up times were also utilised and while some researchers preferred to use a simple pre and post treatment paradigm others utilised slightly longer times. 1 paper reported an extensive follow up for a period of 36 months This is of interest as HbA1c generally is measured at 3 month increments and behavioural change can take time, particularly in this demographic.

### 8.5.2   Treatment Features

It is of extreme importance that three of these programmes reported success and they were primarily based on aspects of T1 that relate to EDs (please see table 8.1). While it may be unsurprising that inpatient treatment demonstrated the most efficacy as it is widely regarded as the most intensive of the treatment modalities, the nature of the programme should not be discounted, particularly as in the Takii et al. (2002;2003) research recovery was maintained at 36 month follow up. Dicken's et al. specifically referenced Goebel-Fabbri's recommendations in relation to their programme and Takii et al. (2002;2003) reference the American Diabetes Association showing that these programmes put Diabetes at the forefront. Perhaps one of the main reasons that they showed improvements is that as suggested by Goebel Fabbri et al. (2002; 2008; 2009) and Bermudez et al. (2009) initial responsibility for insulin dosing is abdicated to the ward staff or at least medically supervised until the patient is in a reasonable psychological state. Unfortunately details about the content of therapy sessions were not forthcoming in the articles published but in suggesting reasons for long term success Takii et al. (2003) suggest:

> the following elements of this inpatient therapy seem to be important in maintaining a good course after discharge: (1) calming the patient's mind and modifying behavior by controlling stimuli that lead to eating problems such as binge eating, (2) allowing incremental psychological/ behavioral conflicts by lifting the controls little by little, and (3) allowing the patient to experience recovery from the conflicts with careful coaching by the therapist. (Takii et al., 2003, p. 355)

It is of also of particular note that this research team describe that one treatment session of counselling for individuals with comorbid T1D and BED was successful at long term follow up (please see table 8.1 for details). This may provide further support for the idea that a new taxonomy for T1ED should be developed which considers severity levels and behavioural profiles. More research is certainly warranted.

In the psychoeducation treatments described the authors explain that the interventions may have been too short (Olmsted et al., 2002) or that this particular sub group require a much more targeted and intensive approach involving much one to one clinical time in order to improve (Alloway et al., 2001). However Takii et al. (2002) were successful in treating BED in just on session. It is of not that both psychoeducation interventions based on others which are designed for EDs in the general population. It appears that Alloway et al. (2010) may have fallen foul of only focussing on classic symptoms of Eating Disorders rather than those that are unique to T1ED (please see chapters 4 & 7). Similar issues may have affected Olmsted et al's. (2002) study where they also adapted a programme originally designed for use in the general population but claim to have modified for Diabetes. There is nothing in the article to suggest that they are actually doing anything to assist patients who are actively Eating Disordered though, rather this intervention appears like discussions around the topic rather than directly addressing behaviours (please see table 8.2).

Colton et al. (2015) describe the patients that they have treated over a 22 year period using a day patient protocol which they state they do not significantly change for their Type 1 patients. They state that those with T1 do significantly worse than their non Diabetic peers and that this could be due to 'low readiness to change' (p. 316). This seems more than a little presumptuous given that the programme was not tailored at all to the needs of these of patients. The authors do give further suggestions for treatment resistance however hypothesising that:

> Individuals with Type 1 diabetes may feel poorly understood by other group members or staff who do not have diabetes themselves, and who may have limited knowledge of the broad ways in which diabetes can influence daily life, long-term health and self concept. This may make it more difficult for those with type 1 diabetes to make full use of group therapy sessions or to form an

effective alliance with the team and their peers in treatment. (Colton et al., 2015, p.316)

This is a more likely explanation and congruent with qualitative research (please see appendices B, C & D). Furthermore, their programme demands absolute insulin adherence which is not only contrary to all published recommendations but also unrealistic, given that these patients only attended the treatment during the day, leaving them with complete control over night.

In programmes which do not describe content the authors nonetheless suggest why those with T1D are more resistant. For example, Custal et al. (2014) postulate that those with type 1ED do not benefit from therapeutic treatment in the way that non T1EDs do due to temperamental attributes such as low perseverance, instability and low frustration tolerance. However an alternative explanation is that these patients demonstrate these attributes in the face of treatment which they know is not going to be suitable for the nature of their ED or tailored to take into account the unique aspects of T1E. The authors state that there is no clinical difference in ED symptomology between those with T1ED and ED other than insulin omission but this thesis provides significant evidence to the contrary and thus assumptions should not be made. Also, this research does not appear to have taken notice of any recommendations made when dealing with this population.

### 8.5.3 Strengths

The purpose of the current review was to provide a broad overview of treatment programmes reported over the last 2 decades (1998 – 2018). In doing so this is the one of the largest systematic reviews to date in this area. Unlike other reviews this review has not focussed on controlled treatments only and as such can make broader assumptions. Also given the nature of the other sections of this thesis the current review maybe better placed to make suggestions as to why perceived wisdom may be incorrect.

### 8.5.4 Limitations

There were a number of case reports detailing treatment of individual T1EDs that were screened out of the review. Given the small sample sizes reported in the articles included it

could be that this was overly conservative and these case reports actually warranted inclusion. Also, as the details of the treatment programmes in terms of actual content was extremely limited it may be that the assertions made in this review are overly speculative, to a certain extent though, given the lack of information this was unavoidable. Quality was not considered as a prerequisite for inclusion, this was due to other assertions made in this thesis that there is no gold standard, and often the underlying principles of dealing with T1ED are misguided and therefore, even studies which would otherwise be deemed to have excellent methodology are essentially problematic. There may also be an argument that only randomised controlled trials should be included in any treatment review but as stated elsewhere there is no consensus on what an acceptable control group for this demographic is.

## 8.6 Conclusion

This review concludes that authors should hesitate to claim that those with Eating Disorder and Type 1 Diabetes are hard to treat when basing that claim on the treatment literature. Such a statement is based on a sample size of less than 200 patients, who for the most part have not participated in suitable treatments. A more accurate statement would be that those with Type 1 Diabetes and an Eating Disorder do not recover in treatment programmes that are designed for those with an Eating Disorder but not Type 1 Diabetes. It is significant that when these patients participate in interventions which are designed specifically for them and not based on preconceptions from traditional treatment, that not only do they get better, they can also stay better.

# 9    Final Discussion and Conclusion

## 9.1    Discussion

### 9.1.1    Measuring Eating Disorders in Type 1 Diabetes

#### 9.1.1.1    *Chapter 2: Eating Disorders and Insulin Omission for Weight Loss in those with Type 1 Diabetes: A Systematic Review*

The main aim of chapter 2 was to provide a very broad overview (in the form of a systematic review of all papers published in this area since 2002), of how Eating Disorders have been measured in Type 1 Diabetes and whether previous assumptions made about this population are valid by considering sample size, age of participants, primary methodology, sample recruitment, measurement instrument used, whether that measurement has been modified for T1D regimen, prevalence of diagnoses or number of those scoring above designated cut-off points reported if any, whether and how insulin omission was measured and what the prevalence of insulin omission was. The review showed research in this area was inconsistent and seriously methodologically flawed. There were particular issues with sample size and lack of male participants. Measurement instruments were often used 'as is' with no modifications for T1D and where modifications had been used there was little consensus as to how this should be done. When a Diabetes specific scale was used it was the DEPS-R in the majority of cases (please see below for a larger discussion on this).

BN and EDNOS were the most common diagnoses although there were varying methods used to ascertain diagnosis, the status of insulin omission was unclear in many papers, completely ignored in others and not given the appropriate weighting considering the consequences. Where diagnoses were not made a various risk categories were reported instead such as 'probable ED' (Powers et al., 2016), 'Elevated Eating Disorder Behaviour' (Johnson et al., 2014), 'Very Unhealthy Weight Control' (Schwartz et al., 2002) and 'Mildly Eating Disturbed' (Maharaj et al., 2003). These terms were concluded to be relatively ineffectual. Where insulin omission was measured the prevalence rates reported varied widely but in general this behaviour was more common in adult females than any other

demographic. In research utilising clinical samples IO was reported in as many of 90% of participants demonstrating that this is a key tenet of the illness.

### 9.1.1.2  *Chapter 3: Scale Comparison*

The aim of chapter 3 was to compare rates of T1Ds scoring above the cut-off point for clinical concern using both the EAT-26 and the DEPS-R and then to modify the EAT-26 by removing items deemed to be influenced by T1D regimen by an expert panel. It was hypothesised that the EAT-26 would identify more participants as being of clinical concern than the DEPS-R but that modification should reduce the ratio of those scoring above the cut off point. The DEPS-R was then subjected to a factor analysis to ascertain if there was underlying structure that differentiated Eating Behaviours in the T1D population and the insulin behaviour subscale identified by Merwin et al (2014) was investigated further.  As expected the EAT-26 did screen more patients as clinically concerning than the DEPS-R but unexpectedly modifications actually increased that ratio, almost screening the entire sample as probably having an Eating Disorder suggesting that the EAT-26 is inappropriate for use in the T1D population.

A 2 factor solution was found when factor analysing the DEPS-R. These subscales related to more severe and less severe ED behaviours and cognitions. The insulin items suggested by Merwin (2014) also formed a reliable scale and less participants scored above the cut-off point than for the full DEPS-R indicating that the items were potentially are measuring a different behaviour closer related to the concept of 'Diabulimia'.  This having been said there are still issues with the use of the DEPS-R (please see below).

### 9.1.2  **Understanding Eating Disorders in Type 1 Diabetes**

### 9.1.2.1  *Chapter 4-6: Risk Factors and Co-morbidities*

The aim of the study reported in these chapters was to ascertain if variables identified in a pilot study and subsequent literature review could be modelled to predict Eating Disordered behaviour as suggested by elevated HbA1c and DEPS-R Scores. Latent variable modelling demonstrated psychosocial aspects (depression, anxiety, borderline personality disorder, self-esteem, perfectionism, consideration of future consequences & family functioning),

Diabetes specific distress (measured by certain subscales of the Diabetes distress scale) and demographic variables (gender) could be modelled in a meaningful way to predict Eating Disorder indicators. The individual scale scores formed two reliable latent variables 'Psychological functioning' and 'Diabetes specific distress'. Psychological functioning predicted both HbA1c and DEPS-R as did Diabetes distress in the structural model. Female gender also predicted higher DEPS-R scores but only slightly and higher levels of family cohesiveness predicted less psychological problems, which in turn predicted reduced levels of HbA1c and DEPS-R. The novel variable 'consideration of future consequences' also predicted the outcome variables with those who were more considerate of the future scoring lower on the DEPS-R and having better BG control.

### 9.1.2.2   *Chapter7: Patient Attributions as to what Caused their Eating Disorder*

The main aim of chapter 7 was to replicate a study previously undertaken by the researcher in which suggestions as to why EDs in T1D are more common were transformed into a Likert scale and presented to those who had recovered from T1ED. The study reported used a larger and more representative sample in order to confirm that there was an underlying factor structure to this questionnaire which broadly relates to attributions patients make to what caused their ED. As hypothesised, there was an underlying structure. The 4 confirmed factors were 1) the family, 2) weight and body image, 3) Diabetes diet and HCP interactions and 4) Diabetes specific psychological issues.

### 9.1.3   Treating Eating Disorders in Type 1 Diabetes

### 9.1.3.1   *Chapter 8: Treating Eating Disorders in Type 1 Diabetes.*

The purpose of chapter 8 was to ascertain if the assertion that those with T1 are more difficult treat for EDs than those without was valid. A review of treatment protocols employed in this population over the last 20 years found that, when programmes are built explicitly with T1D in mind then this assertion if false, rather the main issue appears to be that those with T1ED are either subjected to programmes which are not initially built for them or shoehorned into standard ED treatment. A significant finding of this review is that the literature covers the treatment of less than 200 patients and therefore no generalisations should be made.

## 9.2 Recommendations

### 9.2.1 The Need for more Research around Co-Morbidities

It should be noted that many of the same risk factors/ comorbidities for Eating Disorders in the General Population also apply to those with T1D. The main difference might be that these traits are also generally higher in T1D which may help explain why ED prevalence is increased. More research needs to be focussed on the co-morbidity of mental health diagnoses in T1ED also particularly on disorders such as borderline personality which seems to be increased both in the general T1D population and in those with T1ED. As BPD is an illness marked by significant self-harm episodes this is even more important given that T1Ds are prescribed insulin, a medication that could be used dangerously for that purpose, yet only 3 articles have been written on the subject. Even where research has looked at co-morbidity there is little more than speculation as to the link between T1ED and the features of co morbid mental-health conditions.

### 9.2.2 Changes to Current Protocols

In order to avoid EDs everything in the T1D environment should be examined; it could be that understanding the impacts of T1D on mental health and the important relationship with Diabetes specific distress could significantly reduce the risk of developing EDs. At diagnosis this is not something that T1Ds or their families are informed about. Instructions on how to inject, how to carbohydrate count and even structured education programmes such as DAFNE(dose adjustment for normal eating) are useful for the practical aspects of living with T1D but this study shows that in reality how one functions is as much a product of psychological aspects if not more. All the knowledge available on T1D does little to address the daily grind of living with such a relentless illness and patients are not warned about this. Although psychologists are more frequently in the employ of Diabetes services this is still relatively rare. If psychological input was integrated from the moment of diagnosis and utilised as part of regular appointments it may be that these issues could be identified and thus acted upon earlier improving the prognosis for these patients. It would also be useful to explore the relationships between general psychological distress and Diabetes specific distress in the clinic which would be an ideal place to carry out research. Also, it is of note

that despite HCPs stating that they feel Diabulimics do not reach out for help this was not the case in the reported study with nearly all of the participants approaching HCPs for help, more psychological input would mean that these cries for help would be recognised.

The addition of a psychological input would also be beneficial for the other HCPs who so often report feeling out of their depth dealing with this population. Iatrogenic factors seem to occur due to fundamental misunderstandings around the difficult of dealing with T1ED, insensitivities around weight and complications or unrealistic expectations. We need to 'go back to the drawing board' with clinics and restructure services with these vulnerabilities in mind. It does seem that the NHS are moving in that direction, having just produced a position statement on language and Diabetes where they introduce the document by stating:

> People with Diabetes internalise messages from the media, and from those around them, but most of all from their healthcare providers. When these messages are perceived negatively, whether it is intended or not, this can lead to feelings of shame, guilt and resentment. People who are ashamed of a condition will find it much harder to engage and manage that condition proactively (NHS England, 2018, p.1)

The recommendations made in this document however are just that and more needs to be done at a level where implementation can be recorded and outcomes measured. Although there would no doubt be a financial argument against the inclusion of more paid staff this is a moot point when looking at the catastrophic costs of not dealing with these issues in a timely manner. For example, nephropathy is one of the most common complications of Diabulimia; the cost of dialysis for 1 patient for 1 year is an average of around £35.000 and kidney services utilise around 3% of the annual NHS budget (Baboolal et al., 2018; National Kidney Federation, 2018). Put simply this is too costly not to deal with and proactive solutions will save money and heartbreak down the line.

### 9.2.3   The Need for a New Taxonomy

The current argument would be that insulin omission is a feature or AN or BN or (even less defined) EDNOS, but there is no evidence for that. Rather T1ED patients describe a pattern of Diabetes specific attributional factors and unique to Diabetes psychological disturbances that affect not only the aetiology of their disorder but also barriers to recovery. In fact, it

could be argued that insulin omission for weight control is *the only* behaviour in this population that we know as a singular T1ED symptom. From purging after a hypoglycaemic related binge to starving due to fear of that same hypoglycaemia all other ED symptoms appear entrenched in factors relating directly to Diabetes regimen. This also renders current ED diagnostic criteria for T1D essentially useless and non T1D control groups irrelevant. There are also factors which are just not applicable to the general population like the use of an insulin pump or Diabetic Ketoacidosis

Due to this significant effort should to put into defining a taxonomy for T1ED, the current trend of assigning arbitrary categorisations such as 'Extremely Unhealthy Weight Loss Behaviour' or 'Disturbed Eating Behaviour' is doing nothing in terms of helping design treatment programmes or estimating true prevalence rates, some kind of consistency is needed. Similarly that 5 people with exactly the same pathology and behaviour can be defined as AN, BN or EDNOS, subthreshold ED or DEB depending on instrument used is alarming. It is time that we define EDs in this population in relation to Diabetes specific aetiological factors and biological outcomes so we can work on treatment programmes that will actually be effectual. Furthermore, nomenclature should be prioritised considering that most T1EDs describe their ED as 'Diabulimia' which is not a clinical term.

It should also be noted that in all probability EDs in T1D are not homogenous, they are marked by significantly different behaviours, for example not all T1EDs will utilise insulin omission. A new taxonomy should be entirely separate from standard Eating Disorders and rather describe behaviours for weight control in relation to Type 1 Diabetes.

### 9.2.4  The Need for New Measurement Instruments

The key issue when attempting to measure EDs in T1D is that in reality we do not know what we are measuring. There is not anywhere near enough scrutiny on the suitability of any of the current scales used and how they may be affected by the presence of T1D. Even when instruments are modified, such as the EAT-26, unexpected results can occur. It may be that simply by nature of the illness, T1Ds appear more Eating Disordered. Furthermore, many of instruments miss the key tenet of the illness; insulin omission. In any other circumstance this

would seem absurd; like measuring Anorexia without considering calorie restriction or Bulimia without compensatory behaviours.

The 2002 Meta- analysis by Nielsen found that 40% of T1Ds females (and increasing numbers of males) who screened as ED were misusing insulin for weight loss purposes and levels of insulin omission were also higher than rates of ED diagnoses in the studies described. This means that current instruments are missing inulin omission. It may be that these patients who are obviously engaging in extremely high-risk weight control behaviour represent a distinct psychological profile that is not represented *at all* by any of these instruments.

It seems apparent that everything involved in measuring Eating Disorders in Type 1 Diabetes needs to be reconsidered. The instruments included in the review were fundamentally flawed for 2 reasons. Firstly, because unfortunately sometimes appropriate treatment for T1D *looks* like an ED and secondly, because the most common (and the most harmful) behaviour for weight control is unique to this population and poorly understood by those outside the T1 community. As T1D is relatively rare it is somewhat understandable that researchers and clinicians in the ED field would not have considered it when developing ED measurements. Now we *do* know however it is absolutely essential that we develop T1D specific instruments (please see chapter 9 for a discussion on the DEPS-R) that have clinical utility.

New, T1D specific instruments must be proposed. It would be a good idea to do more qualitative research in this area in order to finetune what items are should be included. Future research may also wish to make use of the model reported in this thesis by identifying what items of the individual scales correspond most closely to the latent variables of 'Diabetes distress' and 'Psychological functioning' in order to build a short form risk questionnaire.

### 9.2.4.1   *Use of the DEPS-R*

Researchers have tried to address issues with measuring T1EDs by proposing Diabetes specific instruments such as the DEPS-R but as demonstrated in this thesis, there are still problems. A worrying aspect of using a measurement such as the DEPS-R particularly in youth samples comes from a potential iatrogenic effect. Participants in qualitative research

have reported 'finding out' about diabulimia through informal means (Balfe et al., 2013; Goebel – Fabbri, 2017).

In the original DEPS-R validation study (Markowitz et al., 2010) use clinicians to state whether or not insulin omission is present in their sample but participants are less likely to report IO to clinicians. They also validate the DEPS-R in an adolescent sample who are more likely to under report due to family and clinical pressures. Furthermore, they validate the scale against BMI, HbA1c, Diabetes-specific family conflict, youth negative affect around blood glucose monitoring, parental Diabetes-specific burden. frequency of blood glucose monitoring and quality of life which are all youth specific, (even although we know that T1ED has an older age of onset and significantly affects adults) but not any clinical diagnoses of Eating Disorders (Markowitz et al., 2010). There has only been one study to date that investigates the relationship between DEPS-R scores and clinically diagnosed Eating Disorder patients which concluded that those with clinical ED scored higher on the DEPS-R scores. However this study utilised participants with both Type 1 and Type 2 Diabetes and thus the results can not be extrapolated to just those with T1 (Pinna et al., 2017) In other validations studies the DEPS-R has only been validated against scales used in the general population which as has been covered extensively are inappropriate.

Perhaps for these reasons there are questions as to whether the DEPS-R is accurately identifying ED cases. Wisting et al. (2013) for example found a high rate of insulin omission in their sample but only 41% of restrictors screened above the cut-off point for concern and in the current study when asked have you ever been diagnosed with or thought that you had an Eating Disorder' of 29.9% of participants answered yes but 44.2% screened positive for further clinical investigation. Some of these issues lie in the way in which questions are asked for example the items regarding insulin omission 'When I overeat, I don't take enough insulin to cover the food' and 'After I overeat, I skip my next insulin dose' do not directly assess whether this is due to weight and shape concerns, there may be many reasons outside EDs that a T1D may avoid injecting, fear of hypoglycaemic for example. Further issues with utilising the DEPS-R occur when assessing what 'overeating' is. In this population, who may have skewed attitudes toward what constitutes appropriate meal size and nutrition 'overeating' may be a subjective assessment.

It should also be noted that there are no subscales in the DEPS-R indicating that the authors view EDs in T1D as homogenous, rather than having distinct behavioural patterns that represent more AN, BN or insulin omission symptomology (not negating the relationship these have to Diabetes regimen). The study reported in this thesis did find 2 latent factors in the DEPS-R the first of which related to what would constitute more severe ED behaviour and the second which pertained to more feelings based risk factors. This highlights a further issue with this scale (and other non-Diabetes specific instruments). Feelings do not necessarily equal action. Feelings may suggest a useful risk profile but, in an illness, as deadly as Diabulimia they should not be weighted in the same manner in instruments that claim to measure clinical severity. An individual who is T1D who omits insulin completely is in immediate danger of death. This is very different to an individual who purges or starves, one incident will likely not cause mortality. Also, no instrument including the DEPS-R measured HbA1c as the most prominent indicator of how severe T1ED is in the individual and an objective measure of risk. Rather they favoured BMI which while important in the general population is of much less concern in T1ED. The DEPS-R has been used in the current study due to its utility as the only commonly used instrument for this population but it is not without flaw.

### 9.2.5 The Need for New Treatment Programmes

Treatment programmes that are based on published recommendations are desperately needed. Only 2 interventions have been published in the last 20 years which were based on T1D related aspects and they were both successful. There are several main areas of concern in the literature. Men were completely unrepresented in the literature, patient feedback or collaboration appeared to be entirely absent, insulin omission is not understood properly and those with T1D are often treated alongside those without. In order to build successful treatment programmes in the future, those involved should disregard anything that comes from standard ED literature and instead focus on T1D related aspects.

### 9.2.6 The Looming Crisis of Male T1EDs

The male experience of T1ED should be prioritised for research in all areas. It could be that as researchers we are doing to males what standard Eating Disorder models are doing to

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T1EDs. From what we do know it seems apparent that EDs present differently in males and that is no different for T1ED. Most alarming is the rapid increase in the prevalence of over recent years and a total lack of reported treatment.

## 9.3    Conclusion

Concluding anything about Type 1 related Eating Disorders is difficult, other than stating that those who have this comorbidity suffer substantially. This suffering could be avoided if we accept that we have fundamentally misunderstood this group and admit that what we have been doing is not working. Every aspect of the perceived wisdom in T1ED is flawed. It is the main conclusion of this research that Type 1 related Eating Disorders must be redefined separately from Eating Disorders in the general population. Measurement should be different, treatment should be different and perhaps most importantly, it must be understood that insulin omission is qualitatively different from any other Eating Disorder symptom. It is unique psychologically, behaviourally and physically. The next stage in T1ED research should seek to define a new taxonomy and concurrent measurement instruments and treatment programmes. Males T1EDs must also be addressed. We do not know the death rates from insulin omission for weight loss as it does not have a clinical diagnosis. At this point it could be argued that nomenclature, 'Diabulimia' or some other term does not matter as essentially, undiagnosable equates to untreatable and it is the patients who pay for this, sometimes with their sight, sometimes with their limbs and all too often with their lives.

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**Appendix A: Allan, J (2017) Diabetes and Eating Disorders: Update to the NICE guideline. Journal of Diabetes Nursing 21(3), 103-107**

# Diabetes and eating disorders: Update to the NICE guideline

*Jacqueline Allan*

**Article points**

1. The NICE guideline on the management of eating disorders is due to be published in May 2017.

2. The draft guideline features, for the first time, specific sections on eating disorders in the context of diabetes.

3. Recognition of the risks associated with insulin omission and recommendations for collaborative care between the diabetes and eating disorder teams are welcome.

4. However, some issues remain, and it could be argued that diabetes-related eating disorders belong in a category of their own.

**Key words**

- Clinical guidelines
- Eating disorders
- NICE
- Type 1 diabetes

**Authors**

Jacqueline Allan is a PhD student at Birkbeck, University of London, and Founder and Managing Director of Diabetics with Eating Disorders.

The latest NICE guidance on the management of type 1 diabetes acknowledges the increased risk of eating disorders in this population. The updated guideline on eating disorders, due to be published in May 2017, is expected to follow suit, with subsections on type 1 diabetes added for all categories of eating disorder. In this article, the author reviews the diabetes-related advice that has been added in the draft guideline. While these updates are welcome, some concerns remain. This critique aims to stimulate further thought about these issues, both for practitioners and prior to publication of the final guidance.

It is well established that eating disorders are more common in people with type 1 diabetes. Despite increasing awareness of the prevalence of eating disorders and the importance of considering insulin omission when diagnosing them, there has been a distinct lack of advice for healthcare professionals on how to deal with these complicated dual diagnoses. This has been partly addressed by updates to NICE guidelines and quality standards for type 1 diabetes, both in children and young people and in adults. The guidelines feature psychological support more than ever before, and they specifically note the increased prevalence of eating disorders and the potential for insulin omission.

In addition to the diabetes guidelines, updated guidance on eating disorders is due for release in May 2017. The new guideline follows suit, with whole sections on diabetes added for the first time. While these updates are welcome and validate the unique issues faced by this demographic, it is still necessary to critically evaluate the guideline and make recommendations for the future. The latest draft has been shared with stakeholders and, while there are likely to be substantial changes before the

final publication, there are a number of issues that deserve comment.

## Diagnosis

Diabetes first appears in the guideline in the statement that people with type 1 diabetes are a high-risk population, mostly because they are more prone to serious complications as a result of eating disorders. As such, risk management should be the first priority. The guideline then suggests that screening for eating disorders in those with particular risk factors, such as type 1 diabetes, should be considered as a matter of course. This raises a problem, however, as there is no current consensus on appropriate screening tools for eating disorders in people with type 1 diabetes. Hopefully, the inclusion of this advice in the guideline will encourage future research into screening measures, or at least a general agreement on how current instruments should be adapted to account for issues such as insulin omission.

## Management and treatment

Type 1 diabetes as a comorbidity has specific sections under treatment for all categories of eating disorder: anorexia, bulimia, binge eating disorder

and eating disorders not otherwise specified (EDNOS). The guidelines under all are identical:

1. Eating disorder specialists and other care teams should collaborate when caring for people with physical or mental health comorbidities that may be affected by their eating disorder.

2. When collaborating, teams should use outcome measures for both the eating disorder and the physical and mental health comorbidities, to monitor the effectiveness of treatments for each condition and the potential impact they have on each other.

3. Eating disorder teams and diabetes teams should collaborate to explain the importance of physical health monitoring to people with an eating disorder and diabetes.

4. Consider involving family members and carers (as appropriate) in the treatment programme to help the person with blood glucose control.

5. Agree between the eating disorder and diabetes teams who has responsibility for monitoring the physical health of people with an eating disorder and diabetes.

6. Explain to the person and their diabetes team that they may need to monitor their blood glucose control more closely during the treatment for the eating disorder.

7. Address insulin misuse as part of any psychological treatments for eating disorders in people with diabetes.

8. Offer people with an eating disorder who are misusing insulin the following treatment plan:
   – A low-carbohydrate diet, so that insulin can be started at a low level.
   – Gradually increasing insulin doses to reduce blood glucose levels.
   – Adjusted total glycaemic load and carbohydrate distribution to meet their individual needs and prevent rapid weight gain.
   – Carbohydrate counting when adjusting their insulin dose (including via pumps).
   – A diabetes educational intervention, such as Dose Adjustment for Normal Eating (DAFNE).
   – Education about the problems caused by misuse of diabetes medication.

9. For more guidance on managing diabetes, refer to the NICE guidelines on type 1 and type 2 diabetes in children and young people, type 1 diabetes in adults and type 2 diabetes in adults.

## Comments
### Collaborative care

A multidisciplinary approach to dealing with eating disorders in people with type 1 diabetes has been advocated in the research literature both historically and more recently (Peveler and Fairburn, 1989; Colton et al, 2015). Practically, however, patients have complained that this type of professional collaboration is rare (Hastings et al, 2016). This is particularly important as advice for standard eating disorders (such as employing a more relaxed attitude too food, being less focused on nutritional labels and being less rigid around timings) may be in direct contradiction with advice for managing blood glucose. If specialists in one condition do not understand the ramifications of the other, then advice could be confusing and hinder, rather than help, the recovery process.

The recommendation for multidisciplinary teams to use specific health outcomes for both the eating disorder and diabetes is another welcome addition as, historically, $HbA_{1c}$ has not been considered as important as BMI. Indeed, in most research papers on the issue, $HbA_{1c}$ has be absent entirely as an outcome measure, as stated in the draft guidelines themselves:

*"No data was available on $HbA_{1c}$ scores, remission, weight, all-cause mortality, adverse events, quality of life, resource use, relapse, general psychopathology, general functioning, family functioning or service user experience."* (page 411)

Taking $HbA_{1c}$ outcomes into account will also hopefully widen treatment access to this demographic, who are often denied support by merit of a "normal" BMI, regardless of often dangerously high $HbA_{1c}$ levels.

The recommendation that the importance of physical health monitoring should be "explained" to patients is potentially problematic, and similar issues arise with "explaining" to patients that they may need to monitor their blood glucose control more closely during the treatment for the eating disorder. People with type 1 diabetes can usually be considered "expert patients", and to repeat the consequences of mismanagement and

### Page points

1. The new draft NICE guideline on eating disorders now has subsections on diabetes for all categories of eating disorder.

2. Diabetes and eating disorder teams are advised to work in collaboration to treat both conditions, and to use outcome measures such as $HbA_{1c}$, as well as BMI, to monitor the effectiveness of treatment.

3. A specific treatment plan, including structured diabetes education, is outlined for people who are misusing insulin.

the need for monitoring may seem condescending or patronising, as these people know the consequences of their actions more than most (Diabetics with Eating Disorders, 2010). Given that this group often feels marginalised by healthcare professionals, highlighting such issues may further the divide between patient and professional (Hastings et al, 2016). It also should be apparent that anyone involved in eating disorder behaviours who also has type 1 diabetes should be cared for by a specialist clinic, whereby such issues are discussed as a matter of course. The obvious exception to this are patients who have disengaged with their diabetes teams and may be seen only and sporadically in primary care, if at all. Under these circumstances, highlighting the importance of physical health monitoring may be appropriate.

Involving family members in treatment for both eating disorders and type 1 diabetes is fairly standard, but it should be considered that family problems are a risk factor for the development of eating disorders (Striegel-Moore and Bulik, 2007), and conflict over the management of type 1 diabetes and related poor glycaemic control is common (Anderson et al, 2002). Therefore, in some cases, familial involvement could ignite further issues.

Further issues arise when deciding who between the eating disorder and diabetes teams should have responsibility for monitoring the physical health of people with type 1 diabetes and eating disorders. Given that so much of each area is highly specialised, it is understandable that one team should take the lead. That said, there is so much in type 1 diabetes that diverges from the standard physical monitoring in people with eating disorders – not just blood glucose, ketones and $HbA_{1c}$, but also cerebral oedema, rapid onset of complications and glucose toxicity to, name a few – that it seems most appropriate to have the diabetes team take the lead.

### Insulin misuse

Including insulin omission as part of any psychological treatment may appear to be an obvious addition, but issues arise again when there is no suggestion of how to implement or evaluate this. Given that insulin omission is incredibly common in people with type 1 diabetes, with an estimated prevalence of around 40% in women

in particular and new evidence suggesting that 11% of adolescent males are affected (Fairburn et al, 1991; Hevelke et al, 2016), treatment recommendations are now imperative. That said, the fact that insulin omission is mentioned at all is potentially life-changing for those people for whom insulin omission comprises their primary eating disorder behaviour but who are treated as if this is not the case. The inclusion of this in the guideline should hopefully bring attention to the lack of research and, thus, treatment options. Similarly, the addition of a treatment plan that focuses on insulin omission will no doubt have a significant impact for patients.

### Structured education

That the draft guidance takes into account issues around carbohydrates, carbohydrate counting and weight gain further validates assertions that people with type 1 diabetes require a highly individualised approach that takes their diabetes-specific needs into account. However, there are issues with the treatment plan, particularly the recommendation that patients attend a DAFNE course. DAFNE improves outcomes in many people with type 1 diabetes; however, its efficacy in those with an eating disorder has no evidence base. This recommendation may be appropriate at some stages of recovery, but there is no research to support it as part of an initial plan. Given that DAFNE takes place in groups and involves activities such as group dosing and group eating, this course of action may be inappropriate and cause further problems, not least of which may be complications associated with rapid tightening of control.

### Looking ahead

The guideline review committee members ask the question "Does any intervention for an eating disorder need to be modified in the presence of common long-term health conditions?" They go on to consider evidence from a number of studies but conclude that none of the research is appropriate to draw any conclusion from. It is notable that this assumption is made for all categories of eating disorder with comorbid diabetes. Therefore, it could be argued that, as we are aware that standard treatments rarely show efficacy, especially in regard

to HbA$_{1c}$ levels, a new paradigm is warranted. The panel conclude that:

*"The ideal study design to answer the question of whether a treatment for eating disorders needs to be modified in the presence of a long-term health problem would be to randomise people with an eating disorder and diabetes to two different treatment groups: one modified to address both the eating disorder and diabetes and one non-modified eating disorder treatment."*          (page 411)

Inclusion of this discussion in the guidelines will hopefully ignite interest among researchers and funders, especially as the committee also highlights that the resource needs of people with type 1 diabetes and eating disorders increase as issues go unaddressed.

The new guideline demonstrates positive progression towards recognition that eating disorders in people with type 1 diabetes, and insulin omission specifically, pose a unique problem. Although a few particular concerns remain, the guideline constitutes a new framework from which further advancements can be made. One may ask, however, why type 1 diabetes-related eating disorders are not in a category of their own. The advice for each category of eating disorder (anorexia, bulimia, binge eating disorder and EDNOS) is modified in exactly the same way for people with type 1 diabetes, including the addition of advice on insulin omission. This suggests that eating disorders in people with type 1 diabetes are fundamentally distinct from eating disorders in the general population. Further support for this view comes from the drastic alterations to standard physical monitoring that are needed in this population.

Another potential problem is the omission of the term "diabulimia". This term is problematic for a number of reasons, not least of which is the fact that diabulimia is not a clinical diagnosis and there is no consensus on the term's use. Patients, however, identify with it. In a recent study in which women with type 1 diabetes who were in recovery from an eating disorder were asked "what type of eating disorder do you think you had?", 27.5% believed they had diabulimia, 19.4% believed they had a combination of bulimia and diabulimia, 18.4% believed they had

a combination of anorexia and diabulimia, and 28.6% believed that they had a combination of all three conditions (*Figure 1*; Allan, 2015). Only 4.1% believed they had anorexia nervosa and none reported singular bulimia or EDNOS. This suggests that the patients themselves view the act of insulin omission as behaviourally distinct from binging and purging or restricting. As such, they identify with the term diabulimia and, whether or not clinicians or researchers feel the term is appropriate, this is how patients may present in clinic or at the GP. At the very least, healthcare professionals dealing with eating disorders and type 1 diabetes should be aware and prepared. The inclusion of the term diabulimia in the guidelines would facilitate such awareness and should, therefore, be encouraged.

Alternatively, perhaps it is time that there was some consensus around how we screen, diagnose and treat eating disorders in this population that acknowledges the specific biological and psychological ramifications of type 1 diabetes which are so inherently tied to the

**Page points**

1. It could be argued that diabetes-specific eating disorders require their own categorisation.

2. "Diabulimia" is a term that, although not recognised as a diagnosis, appears to resonate with patients and may be appropriate to include in the guideline.
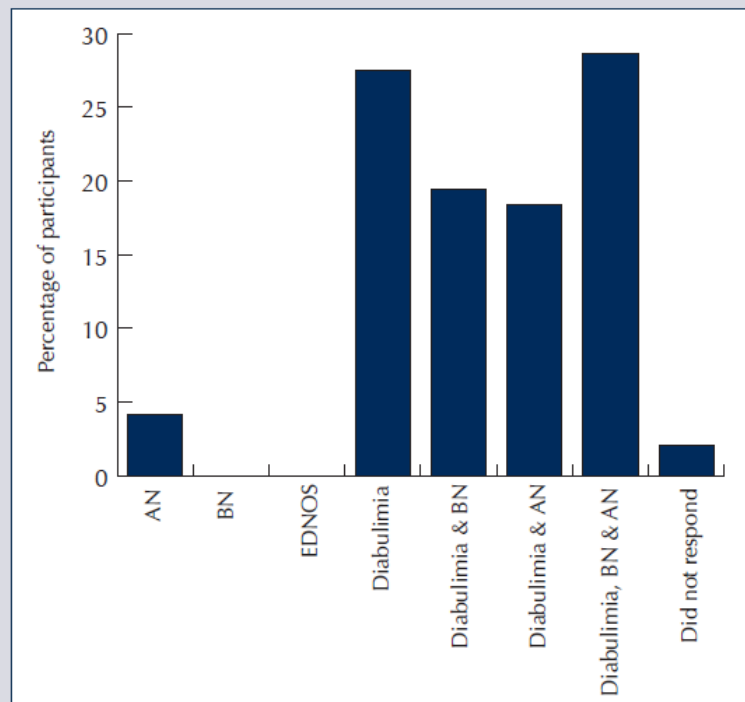


Figure 1. Participants' self-labelling of their eating disorder (n=98; Allan, 2015). AN=anorexia nervosa; BN=bulimia nervosa; EDNOS=eating disorder not otherwise specified).

*"The addition of type 1 diabetes and insulin omission in such a comprehensive manner to the NICE eating disorder guideline is representative of a cultural shift towards recognising the magnitude of this problem."*

development and maintenance of the disorders. Such a nomenclature could then separate this demographic into their own criteria and specify the type of eating disorder by behaviour (e.g. restricting, binging/purging, insulin omission, etc.). This would provide further clarification as to what is needed in terms of treatment and validate patient opinion, while avoiding the problematic term of diabulimia.

## Concluding remarks

The addition of type 1 diabetes and insulin omission in such a comprehensive manner to the NICE eating disorder guideline is representative of a cultural shift towards recognising the magnitude of this problem. Mounting political pressure from MPs such as George Howarth and MSPs such as Dennis Robertson, the continued efforts of the charity Diabetics with Eating Disorders, spearheading clinicians such as Professor Janet Treasure and Professor Khalida Ismail, and the emerging support of the larger charities such as Diabetes UK and Beat indicate that we are moving towards a more specialised approach to type 1 diabetes-related eating disorders. ∎

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**National Guideline Alliance**

Version 1.0

## Eating Disorders: recognition and treatment

**Full guideline**

*NICE Guideline*
*Methods, evidence and recommendations*
*December 2016*

Draft for Consultation
Commissioned by the National Institute for Health and Care Excellence

## About the guideline

This article is based on the draft guideline presented for consultation in December 2016. Based on comments from stakeholders, the final guidance, due to be published in May 2017, may differ from the draft.

The full draft guidance can be accessed at: **https://www.nice.org.uk/guidance/GID-CGWAVE0703/documents/draft-guideline**

**Appendix B: Hastings, McNamara, Allan, Marriott (2016) The importance of social identities in the management of and recovery from 'Diabulimia': A qualitative exploration.** *Addictive Behaviours Reports 4*, (78-86)

# The importance of social identities in the management of and recovery from 'Diabulimia': A qualitative exploration

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## ARTICLE INFO

## ABSTRACT

*Introduction:* A significant barrier to recovery for individuals with co-morbid eating disorders and type 1 diabetes is the way in which group members self-categorise. Nonetheless, identity issues are neglected during the recovery process. The aim of this paper is to explore how group memberships (and the associated identities) both contribute to and hinder recovery in this cohort.
*Method:* Transcripts from five online focus groups with 13 members of an online support group for individuals with 'Diabulimia' were thematically analysed.
*Results:* Findings suggested that those with whom one shares a recovery identity can be well placed to provide psychological resources necessary for successful recovery although such connections can be damaging if group norms are not managed. Members recognised that other important relationships (including family and friends and health professionals) are also key to recovery; these other group memberships (and the associated identities) can be facilitated through the recovery identity group membership, which allows for external validation of the recovery identity, provides encouragement to disclose the illness to supportive others, and provides information to facilitate positive service interactions.
*Conclusions:* While clinical interventions typically focus on eliminating disordered behaviours, we suggest that these should also include strengthening important group memberships that promote recovery.

## 1. Introduction

Individuals with Type 1 Diabetes Mellitus (T1DM) are at increased risk of developing an eating disorder (Custal et al., 2014; Jones, Lawson, Daneman, Olmsted, & Rodin, 2000; Peveler, 2000). A defining behavioural feature of eating disorders in individuals with T1DM is the practice of deliberately and chronically withholding insulin specifically and only for weight loss (Custal et al., 2014; Murray & Anderson, 2015; Pinhas-Hamiel, Hamiel, & Levy-Shraga, 2015; Tierney, Deaton, & Whitehead, 2009). As weight loss itself increases in importance to the individual, he or she tends to also routinely engage in other weight management strategies (Balfe et al., 2013) commonly associated with anorexic or bulimic behaviour (Allan, 2015; Custal et al., 2014; Murray & Anderson, 2015). While there has been limited research with this population, Custal et al. (2014) found that apart from insulin misuse, individuals with co-morbid eating disorders and T1DM do not differ from individuals with eating disorders on other eating disorder-related behaviours and psychopathology (such as drive for thinness, body dissatisfaction, and perfectionism).

Once diagnosed, this cohort tends not to respond to standard eating disorder treatment and is at very high risk of serious complications linked to the eating disorder and the resulting poor glycaemic control (Colton, Olmsted, Wong, & Rodin, 2015; Peveler & Fairburn, 1992; Tierney et al., 2009). Compared to individuals with eating disorders but without diabetes, this cohort shows lower partial and full recovery rates and individuals are more likely to drop out of treatment at an earlier stage (Peveler & Fairburn, 1992). Poor clinical outcomes are primarily explained as a result of low levels of persistence and motivation to change (Custal et al., 2014). However, recent research suggests that an important barrier to recovery is the way in which this cohort self-categorises as an illness group (Allan, 2015; Allan & Nash, 2014).

Within mental health in general (e.g. British Psychological Society, 2013) and eating disorders in particular (e.g. Fairburn & Cooper, 2014), there remains a critical discussion about the validity of diagnostic terminology for understanding facets of human distress. Nevertheless, for people experiencing such difficulties, group identities are commonly organised around diagnostic labels (e.g. Espíndola & Blay, 2009), and the issue of identity also lies at the heart of recovery for individuals with both diabetes and eating disorders. The diabetes community uses the

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term 'Diabulimia' to describe what they see as a unique illness identity that should be distinguished from other eating disorder subtypes (Allan, 2015; Allan & Nash, 2014; Custal et al., 2014; Murray & Anderson, 2015; Tierney et al., 2009). However, this is neither a recognised diagnostic category, nor is it used in the academic literature (Allan & Nash, 2014).[1] This absence of identity recognition can hinder recovery due to lack of tailored health services and professional training (Tierney et al., 2009), misunderstanding on the part of family and friends (Pinhas-Hamiel et al., 2015), and the inability to form connections with other individuals participating in group eating disorder interventions (Colton et al., 2015). Cruwys, Haslam, Fox, and McMahon (2015) found that in such group programmes individual progress often occurs in the context of newly forged ideas of normative changes within a group identity; however, someone with Diabulimia may not be able to share the group's recovery identity due to their perceptions of the unique nature of their particular difficulties, thus making the acceptance of newly formed group norms a much harder task.

Over time the eating disorder forms an important basis for self-definition (Abbate-Daga, Amianto, Delsedime, De-Bacco, & Fassino, 2013). Consequently, a shift from an illness to a recovery identity is an essential part of successful recovery (Bowlby, Anderson, Hall, & Willingham, 2015; Espíndola & Blay, 2009; McNamara & Parsons, 2016). However, identity change is typically conceptualised as occurring at the individual rather than the group level (Malson et al., 2011). Recent research in the area of substance misuse has highlighted the importance of social identity transition for successful recovery from addiction. It is proposed that dis-identification with an 'addict' group alongside identification with a recovery group fosters recovery and positive health outcomes (Best et al., 2016; Dingle, Stark, Cruwys, & Best, 2015; Frings & Albery, 2015). Recovery identities have been associated with lower relapse rates (Buckingham, Frings, & Albery, 2013), treatment engagement (Beckwith, Best, Dingle, Perryman, & Lubman, 2015), and greater duration of abstinence (Tomber, Shahab, Brown, Notley, & West, 2015).

It has been argued that eating disorders are a form of addiction (Davis, 2001; Davis & Claridge, 1998). From a clinical perspective, the core behavioural components of eating disorders closely resemble those of substance abuse (Davis, 2001). For those living with Diabulimia, the act of insulin omission and the performance of other disordered eating behaviours become progressively entrenched and individuals report feeling unable to cease these behaviours in spite of experiencing adverse medical consequences (Balfe et al., 2013). Given the addictive components associated with Diabulimia and the complex identity issues involved, it is argued here that the approach espoused by the Social Identity Model of Cessation Maintenance (Frings & Albery, 2015) and the Social Identity Model of Recovery (Best et al., 2016) might be effectively applied to recovery from Diabulimia.

Recent research by McNamara and Parsons (2016) has illustrated that connections with similar others online can promote recovery in individuals with eating disorders through the construction of a shared recovery identity that promotes illness disclosure and treatment engagement. However, a shared recovery identity, while central to treatment success, is only one part of successful recovery. The newly-acquired recovery identity needs to be seen in the context of other groups that are also crucial to recovery and that have implications for how individuals self-define. Therefore, the aim of the current paper is to explore the ways in which important group memberships (and the social identities derived from them) both contribute to and hinder the process of recovery in individuals with Diabulimia.

*1.1. The role of social relationships in recovery*

Social relationships are important in providing a context conducive to recovery, namely, one where the individual does not feel judged on the basis of their disorder identity but feels understood, accepted and perceives their issues are taken seriously by their social networks (Federici & Kaplan, 2008; Leonidas & dos Santos, 2014).

The Social Identity Model of Identity Change (SIMIC) proposes that group memberships have an important role to play in adjusting to change (Haslam et al., 2008; Iyer, Jetten, Tsivrikos, Postmes, & Haslam, 2009). Life transitions often involve leaving one social group to join another – a process that can be experienced as extremely stressful (Haslam et al., 2008). SIMIC proposes that the negative effects of transition can be attenuated, in part, through the adoption of a new social identity which provides the individual with a sense of meaning, purpose, and behavioural guide (Haslam et al., 2008; Iyer et al., 2009). This model forms a key element of the social identity approach to addiction and has informed both the Social Identity Model of Cessation Maintenance (Frings & Albery, 2015) and the Social Identity Model of Recovery (Best et al., 2016). These models propose that a shared sense of identity with others who are also in recovery facilitates the identity transition necessary for successful recovery. Alongside receiving social support from other group members, the recovery group membership acts as an important meaning-making tool that guides behaviour and provides the individual with specific goals and motivations that offer a sense of purpose post-transition (Buckingham et al., 2013; Frings & Albery, 2015; Dingle, Stark, et al., 2015; Jones et al., 2012).

The eating disorder literature has tended to focus on how connections with similar others, particularly those forged online, maintain disordered behaviours rather than promoting recovery (Riley, Rodham, & Gavin, 2009). Nonetheless, recent research in this area suggests that a shift away from a positively-valued eating disorder identity is facilitated by the support and understanding of similar others (Ison & Kent, 2010; Linville, Brown, Sturm, & McDougal, 2012; McNamara & Parsons, 2016; Ransom, La Guardia, Woody, & Boyd, 2010). While research on this topic is even more scarce for those dealing with Diabulimia, a qualitative study by Balfe et al. (2013) suggested that moving away from disordered behaviours was facilitated by contact with similar others which allowed participants to acknowledge the severity of their disorder. However, there is no insight into how recovery identities might promote positive health outcomes in this cohort. Given the comparative rarity of the comorbidity of eating disorders and type 1 diabetes, the potential of online groups in particular to facilitate the construction of a shared recovery identity necessitates further investigation.

Although relationships with similar others can be helpful, relying on such identities may keep individuals within the world of mental illness and addiction indefinitely, preventing a full recovery (Tew et al., 2012). While the social identity approach to recovery has recognised the importance of dis-identification with groups that promote addictive behaviours, it has yet to consider in detail how to incorporate into the recovery process group memberships that may have been negatively affected by the individual's disorder identity but are a vital part of recovery. This is an important avenue of research as the desire to repair important relationships, particularly those with family and friendship groups, can be a catalyst for recovery (Dingle, Cruwys, & Frings, 2015; Leonidas & dos Santos, 2014; Linville et al., 2012).

Family and friendship groups represent potentially important identity groups that have existed prior to the onset of the individual's addictive disorder and should be present after recovery (Haslam et al., 2008; Tew et al., 2012). These can provide a sense of identity continuity to the individual which is an important determinant of a successful identity transition (Haslam et al., 2008). The formation of a recovery identity might also be seen as more compatible with these identities – again another important determinant of a successful transition (Haslam et al., 2008). While individuals with eating disorders can withdraw from valued support networks (Leonidas & dos Santos, 2014), it is important

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that such identity networks are preserved (or re-established) during recovery.

However, support from family and friends is not always positively experienced by individuals in recovery from an eating disorder (Leonidas & dos Santos, 2014; Linville et al., 2012). There can be a mismatch between an individual's support needs and the support offered such that it is experienced as inadequate at best or hurtful at worst (Linville et al., 2012; McNamara & Parsons, 2016). Family and friends may believe that the disorder is controllable by the individual. This misperception can result in tense interactions that ultimately damage relationships (Linville et al., 2012). Those living with Diabulimia have described family and friends as "imperfect supports" (Balfe et al., 2013, p. 2033) in that dysfunctional family relationships could contribute to the development of the disorder but also that family members can reinforce disordered behaviours due to the value placed on weight loss (Balfe et al., 2013). Given the mixed findings in this area (Leonidas & dos Santos, 2014), it is important to investigate how such broader identity networks can be effectively incorporated into the recovery process alongside the recovery-oriented group.

Finally, support also comes from formal services. Issues around identity and service engagement have been investigated to a limited extent within the social cure tradition. Walter, Jetten, Dingle, and Parsell (2015) argue that engagement with some services (specifically those for homeless individuals) necessitates the adoption of a stigmatised identity which might not necessarily be associated with positive outcomes for the individual. Nonetheless, there is evidence that identification with services can enhance wellbeing in this group (Walter, Jetten, Dingle, Parsell, & Johnstone, 2016), suggesting that self-categorising in terms of a stigmatised identity might not always be negative. However, service interactions can become a site of identity conflict (Stevenson, McNamara, & Muldoon, 2014). The stigma associated with a group of service users can undermine relationships with service providers by undermining a sense of shared identity between both parties. This lack of shared identity combined with service users' anticipation of negative treatment from service providers undermines trust in services and could ultimately contribute to individual service disengagement (Stevenson et al., 2014).

Such issues are particularly pertinent for the current context. First, as mentioned earlier, healthcare professionals do not formally recognise the Diabulimia identity (Allan & Nash, 2014). Individuals with Diabulimia have reported negative experiences with professionals when they used this term with their identity being dismissed as "made up on the Internet" (Allan, 2015, p. 100). This dismissal of an individual's identity can lead to difficult interactions with professionals and a refusal to engage with treatments that are not perceived as identity-congruent (Colton et al., 2015). Furthermore, individuals with eating disorders are routinely stigmatised by professionals (Byrne, 2000; Currin, Waller, & Schmidt, 2009; McNicholas, O'Connor, O'Hara, & McNamara, 2016). They are described as difficult to treat and are not viewed as a group that clinicians enjoy interacting with. This is also the opinion displayed toward those with diabetes, thus those presenting with both conditions may be doubly-disadvantaged (Tierney et al., 2009).

However, as with family and friends, those with Diabulimia do mention strong therapeutic relationships with health professionals as important to recovery (Balfe et al., 2013; Tierney et al., 2009). Thus, as with informal support sources, it is important to determine how these relationships can also be effectively incorporated into the recovery process and understand how they exist alongside other valued identity groups.

To summarise, poor treatment outcomes for individuals with Diabulimia are typically explained in individualistic terms, neglecting the role of social factors in recovery. Group memberships (and the social identities derived from them) are at the core of the identity transition process necessary for successful recovery (Best et al., 2016; Dingle, Stark, et al., 2015). These identity networks form the context in which recovery occurs and can both facilitate and hinder this process (Haslam et al., 2008). The social identity approach to addiction recovery has so far investigated the role of identity groups relevant to the addict and recovery identity (i.e. similar others) but has paid less attention to the role of other identity groups that are crucial to recovery. Therefore, the aim of the current paper is to investigate the role played by multiple identity groups in the recovery process. Specifically, we are interested in exploring (1) how a sense of shared identity with similar others online enhances wellbeing and promotes recovery from Diabulimia and (2) how important identity networks can be successfully incorporated into the recovery process.

## 2. Method

### 2.1. Participants and recruitment

Thirteen members of an online support group for individuals who self-categorise as recovering from Diabulimia participated in this study. Of those who reported demographic information, all were female and ranged in age from 18 to 67 years (mean age = 34.9 years). Nine participants were British and two were from the USA. Participants were recruited through an advert posted on the online group's homepage. Those that indicated their willingness to participate were provided with a detailed participant information sheet by the research team which indicated that the research was being conducted as part of the requirements of the first author's Bachelor's degree. Participants were informed that their participation in this study would not affect the service they received from the online group. Informed consent was obtained from all participants prior to the focus groups. The study and its procedures were approved by an ethics committee at the lead author's institution.

### 2.2. Data collection

Five online focus groups (Gaiser, 1997) comprising two to four participants were facilitated by the first author using a secure chat room accessible only by study participants and the facilitator. The facilitator had sufficient preparation for the process through completion of core research modules and preparatory conversations in academic supervision. She had no additional relationship with the participants beyond the recruitment to – and conduct of – the focus groups. Each participant was issued a unique screen name when they logged in at the beginning of the focus group session to preserve anonymity. A semi-structured schedule was developed from a literature review and consisted of fourteen open-ended questions that explored participants' experiences of the online support group (e.g., "what have you found most useful about being part of the group?"), their impression of support services available for individuals with Diabulimia (e.g., "what other support services did you try before this group?"), and their experiences of seeking help and support for their disorder from family, friends, and healthcare professionals (e.g., "what do you find most challenging about talking to people who do not have the illness?"). Focus groups lasted for approximately 1 h each. Transcripts of chat sessions were subsequently downloaded and entered into NVivo text tagging software for analysis.

### 2.3. Analytic approach and procedures

A theoretical thematic analysis (Braun & Clarke, 2006, 2013) was conducted on the data as we were interested in exploring participants' experiences of recovery and the online group through the lens of the social identity approach. A contextualist epistemological approach (Braun & Clarke, 2013) was adopted in this study. This approach views knowledge as signifying the researcher's theoretical position and experience in addition to being 'true' in the data collection context (Braun & Clarke, 2006).

Analysis followed the steps outlined by Braun and Clarke (2006). Transcripts were read repeatedly for familiarisation purposes before all data relevant to the research question were coded, being as inclusive as possible. Themes articulating the most salient patterns occurring across the dataset were formed by grouping similar codes together. Through the use of deviant case analysis (Silverman, 2001), instances which did not fit the broader patterns were used to revise the thematic structure so that it could account for the data in its entirety. All data pertaining to each theme were collated once the thematic structure was finalised.

Braun and Clarke (2006, 2013) advocate that the process of coding and initial analysis is built upon the individual researcher developing a deep familiarisation with the data; within this methodological position, the concept of inter-rater reliability to coding is not an appropriate expectation, and so the initial stages of coding were conducted primarily by the first author without seeking to perform inter-rater reliability checks. However, samples of transcript were reviewed by the second author in her role as academic supervisor. Discussions were held at all stages of the analytical process to create an iterative process through which the analysis reflected the original data in a coherent manner that had been considered from the multiple perspectives of the different researchers.

## 3. Findings and analysis

Four themes were identified from the data. Superordinate and subordinate themes are provided in Table 3.1. These are discussed below with extracts provided to illustrate identified themes. We begin by articulating how identity can lead to a perception of being driven away from mainstream supports (including other service users and professional services) and subsequently seeking validation in the online group. While this group can assist in symptom management and enhance wellbeing, group members must also manage the risks presented by the community. Finally, we detail how the validation provided by the online group allows reintegration into other valued groups.

### 3.1. Theme 1: not like everyone else

#### 3.1.1. Self-distinction from other patients

Participants saw themselves as having a distinct (as they termed it) "Diabulimic" identity and the lack of recognition of this identity by others acted as a significant barrier to support. Participants saw insulin omission as a defining feature of their illness identity that distinguished them from other eating disorder groups and which prohibited them from developing a shared sense of identity with other individuals with eating disorders. In extract 1 below, participants discussed their experiences with either eating disorder-only or diabetes-only groups – neither of which is able to support them in the way they needed. Being directed

**Table 3.1**
Superordinate and corresponding subordinate themes.

| Superordinate theme | Subordinate themes |
| --- | --- |
| Theme 1: not like everyone else | Self-distinction from other patients |
| | Dismissal of healthcare professionals |
| Theme 2: shared identity online promotes recovery | Acceptance and validation of Diabulimic community |
| | Facilitation of symptom management |
| | Shared learning |
| Theme 3: threats to recovery | Self-management – when Diabulimic is not a helpful identity |
| | Group self-regulation |
| Theme 4: recovery outside of the Diabulimia group | Accessing services |
| | Talking to non-group members |
| | Owning the group identity |

to this form of support is reflective of the current treatment protocol, whereby illnesses are seen as co-morbid and both are treated separately (Allan & Nash, 2014; Custal et al., 2014). However, participants did not view this as helpful for recovery:

Extract 1
Focus group 1
User81 [...]the other patients did fail to grasp the effect diabetes had to play in my situation, they didn't understand the complexity of it, so it was easy to feel alone even when surrounded by support, as through no fault of their own, they could not fully understand
User58 Plus they were all non-diabetics, and didn't know what MY issues meant. There was one diabetes group, but all that happened is that the nurse scolded me for not improving my BGs[a] in a week, and THAT set me off to the point where I wouldn't go back.
User81 Sometimes I felt people would be thinking 'oh here she goes again, on about her sodding diabetes'
[a] Blood glucose levels.

#### 3.1.2. Dismissal of healthcare professionals

The fact that this combination of difficulties is not well encapsulated within existing diagnostic frameworks meant that medical help was also perceived to be inadequate which led to difficult interactions with healthcare professionals (as alluded to in Extract 1). Participants felt a lack of empathy from clinicians regarding the hold that the eating disorder had over them:

Extract 2
Focus group 3
Int What smaller things do you feel the professionals don't get?
User12 That just how randomly and dramatically blood levels can fluctuate, even when you are putting in 200%, how small things can really have a massive impact on your day and your ability to do the right thing, that making the right choice, no matter how insightful you are is still very fucking hard.

While this sentiment has been expressed in research with individuals with eating disorders (McNicholas, O'Connor, McNamara, & O'Hara, 2015), our participants also faced the lack of formal recognition of their disorder as well as a lack of professional knowledge and training (Allan, 2015; Tierney et al., 2009). This added an extra barrier to receiving what they perceived to be effective and appropriate treatment, in spite of their efforts to engage with the services offered to them:

Extract 3
Focus group 2
User93 I've tried counseling and therapy (for eating disorders) but none of them had a clue what Diabulimia was so weren't much help whatsoever. Everyone I tried to explain to they'd just ignore me and talk about other eating disorders that I may have.

This inability to find appropriate support resulted in a strong feeling of isolation among participants. While they shared that they had a strong desire for recovery, they felt that this could not be achieved without support from similar others (Best et al., 2016; Frings & Albery, 2015) and professionals who were informed about their disorder.

### 3.2. Theme 2: shared identity online promotes recovery

#### 3.2.1. Acceptance and validation of Diabulimic community

The sense of identity as 'Diabulimic' operated as a social identity that formed the basis for seeking out similar others online to access the support that they felt would be more helpful and conducive to recovery than that received from those who did not share this identity (Best et al., 2016; Frings & Albery, 2015; Haslam, Jetten, O'Brien, & Jacobs, 2004; Haslam, Reicher, & Levine, 2011; McNamara & Parsons, 2016; Read, Morton, & Ryan, 2015). Participants described the strong sense of community and sense of shared identity among the online group. This was reported to combat feelings of isolation and group members formed a key part of participants' social networks (Best et al., 2016):

**Extract 4**
Focus group 2
User1  I've got some of my best friends from it
User3  Yeah I have made some really close friends through the support group, in fact a whole new social circle offline

Focus group 3
User12  It's given me true valuable friendships with people who get it.

Similar to research in addiction recovery, Extract 4 above illustrates that as the individual wished to recover, their social networks increasingly included others in recovery (Best et al., 2016; Dingle, Stark, et al., 2015). Interactions with group members were much different to those described earlier:

**Extract 5**
Focus group 2
User10  I think the chance to really say all on my mind, to rant and just get it all out because a lot of the time healthcare professionals don't provide that space, or don't know where you're coming from. also you bond with people online, and strike up friendships, so the mutual support is really valuable. you can say your most honest shit but it's not the unbalanced relationship of a healthcare professional and client.

Focus group 5
User20  I do have support from other areas but it's not the same here I can come and rant and rage be depressed or proud of myself feel rubbish or great. No one judges or tries to tell me "I mustn't do this or that" or says "if you carry on behaving like this you'll be crippled with this disease by the time you're 50".

The support offered by those who share a sense of identity was perceived as qualitatively different from that received from those outside the group (Haslam et al., 2011; Linville et al., 2012; McNamara & Parsons, 2016). Specifically, participants felt that they could share experiences without being judged and that they could receive the emotional support and encouragement from group members that was absent from their other support networks (however well-intentioned the support offered may be).

*3.2.2. Facilitation of symptom management*
It was apparent from the data that the group provided a social context within which members could learn to manage their disorder and face the challenges of recovery (Best et al., 2016; Dingle, Cruwys, et al., 2015; Dingle, Stark, et al., 2015). The group was seen as a vital source of information about the illness and how to manage it. This was especially important given the aforementioned lack of knowledge among many clinicians (Tierney et al., 2009). Participants described the group as their primary source of advice:

**Extract 6**
Focus group 5
User81  It's like the start point do I look it up on google or go to the GP, no I ask the group if that makes sense, it's like the first thing I do
Int  That's great, and why is it the first place you go to over other places?
User81  Because no one knows more about Diabulimia than Diabulimics

*3.2.3. Shared learning*
Aside from information on medication and coping strategies, it was clear that learning about the consequences of the disorder and its complications from trusted others had an impact on participants' willingness and motivation to improve their own self-care (Frings & Albery, 2015; Haslam et al., 2004). This is exemplified below when participants were asked about the type of informational support received from the online group:

**Extract 7**
Focus group 1
User81  Mostly insight into the devastating impact of it
User93  Yes definitely the consequences of it all, people sharing their experiences first hand. It's a real eye opener.
User81  I knew the facts, but nothing like hearing first hand experiences
User58  Exactly, 81!

Thus, it would seem that a shared sense of identity with similar others assists in the successful management of the disorder, including finding comfort in the encouragement from others and having a trusted source of information and advice (Best et al., 2016; Frings & Albery, 2015; Haslam et al., 2004). It also suggests, similar to Balfe et al. (2013) that such connections can assist in individuals appreciating the negative consequences of disordered behaviour patterns related to their eating disorder.

*3.3. Theme 3: threats to recovery*

*3.3.1. Self-management – when Diabulimic is not a helpful identity*
While the group experience was generally a positive one, there could be some drawbacks whereby interactions with the group could have a negative effect. At times, group members felt that they had to regulate contact with the group if they perceived it was having a negative impact on mood:

**Extract 8**
Focus group 4
User61  I had to take a step back after my baby was born though as I was feeling very vulnerable and didn't find it helpful focusing on anything ed related.
Int  That makes sense, so can sometimes an online group make you think more about the illness?
User95  Yes. You are definitely more mindful. Sometimes that's a good thing but sometimes not. Think that depends on your frame of mind/mood.
User61  Yes. Sometimes it can be very helpful but personally I've found that if I'm feeling vulnerable and lots of people are really struggling the general mood can decline and it is easy to dwell on the negativity. But overall I've found online groups a positive experience.

This illustrates the potential for such groups to present threats to recovery, requiring the individual to regulate their contact with the group (Haas, Irr, Jennings, & Wagner, 2011; Mulveen & Hepworth, 2006). It also provides further support for the recommendation by Frings and Albery (2015) that such groups comprise a mix of participants at different stages of the recovery process.

*3.3.2. Group self-regulation*
Participants pointed to the importance of group norms (or "guidelines" as they were referred to) to highlight how the group itself can respond to instances where online group interactions can contribute to individual vulnerability and threaten the recovery of its members (Frings & Albery, 2015). Participants believed that it was important for group norms to be centred on recovery and for these to be enforced in group discussions:

**Extract 9**
Focus group 4
User61  I think it's important for there to be guidelines in place and for all members to stick to them as otherwise the group can go from being supportive to encouraging people to further their ed
Int  Thats true, so what helps to keep an online group a positive experience rather than negative?
User95  I like that [name of support group] has goals. And people are open in the group.
User61  Partially a good admin team and partially members taking responsibility for what they post and understanding the purpose of the group.

This suggests that participants understood the group identity to revolve around the principles of recovery; these principles informed the group's normative beliefs and values and were reflected in the guidelines and the agreed purpose of the group. When these norms were

violated, there were adverse consequences for group members' recovery (Cruwys & Gunaseelan, 2016; Frings & Albery, 2015). However, both group leaders and members were held jointly responsible for ensuring a group environment that was conducive to recovery.

### 3.4. Theme 4: recovery outside of the Diabulimia group

#### 3.4.1. Accessing services

Recovery occurs in the context of other social relationships and also relationships with healthcare professionals. As previously mentioned, the recovery group was generally viewed as providing the understanding and emotional support needed to promote recovery. This was in contrast to other valued social networks. However, successful recovery involves engaging with formal support services and making (or renewing) connections outside of the treatment milieu (Best et al., 2016; Dingle, Cruwys, et al., 2015; Frings & Albery, 2015; Tew et al., 2012). In our data, we noted that the online group did not just provide psychological resources necessary for managing symptoms and understanding the disorder but also provided resources that assisted in managing relationships with other valued groups.

First, many participants reported that the group had successfully impacted on their engagement with clinicians. A number of participants reported that they had increased engagement with health services as their group membership increased their motivation to recover and encouraged them to engage with health services (McNamara & Parsons, 2016). There was one exception to this broader pattern whereby one participant reported a reduction in the number of medical appointments attended but clarified that this was due to improved self-care.

It was also clear that group membership influenced interactions with clinicians in a number of ways, as exemplified below:

Extract 10
Focus group 2
Int    Because you're getting information from the online group do you feel you see health professionals more or less than you did before using online groups?
User1    More
User10    More
User1    Some of the ppl I talk to give me the encouragement I need to face hcp[a]
User10    Because i am taking control so am more motivated
User3    More but also my appointments are more useful so I see them at the right time and I'm asking them the right things
[a] Health care professionals.

As outlined in this extract, group members felt that participation in the recovery group not only gave them the motivation needed to engage with formal services (McNamara & Parsons, 2016; Read et al., 2015) but also allowed them to more appropriately manage their interactions with professionals (Read et al., 2015). In Extract 10 above, group members spoke about how their membership of the group helped them to manage their medical appointments so that they were useful and effective. In this way, participants may derive a greater sense of personal control over their health outcomes from their online group membership (Greenaway, Haslam, Cruwys, Branscombe, & Ysseldyk, 2015). Importantly, participants saw the online group as complementary to, rather than as a replacement for, health service engagement.

#### 3.4.2. Talking to non-group members

Articulating how group membership might impact on interactions with friends and family was not as straightforward. Consistent with previous research, these social networks were not perceived to be uniformly supportive (Balfe et al., 2013; Linville et al., 2012). This could lead some to withdraw completely from these groups or only engage with select members, as exemplified below, when participants discussed whether engaging with the online group encouraged them to speak to family and friends more often about their disorder:

Extract 11
Focus group 1
User58    I'm speaking to them a little more, but it has been VERY difficult to open up. I'm still not revealing the total extent of it, but I do think I'll get there. And I'm not sure that it's because of the group, or because I really AM in recovery.
User93    I feel like others have encouraged me to open up to my family and friends more but it's still hard and I still can't say everything I want to.
User81    Not much difference there I don't think.
User89    Friends definitely, and my husband. My folks or siblings, no. They don't get it, and they have made zero effort to become involved. I have tried but to them, if they don't talk about something, then it doesn't exist. And I asked for their support a year ago when I left for treatment. But my good friends are understanding and my husband tries to be supportive.

The practice of 'selective disclosure' described here has been shown to have positive associations with mental health and can therefore be an effective strategy for managing the effects of belonging to a stigmatised group (Ilic et al., 2014).

#### 3.4.3. Owning the group identity

As outlined above, some participants felt the group was pivotal in their increasing ability to speak to those friends and family members who they judged would be supportive. Participants suggested that the group facilitated relationships with important others, either by providing an alternative avenue of support or by providing a way of validating their illness identity to others:

Extract 12
Focus group 2
User3    In fact I think I continue to have wonderful friends because I can go to the group and say things that I need to say in a social space but that I don't really want my friends who I meet down the pub to know so it makes my other relationships easier, I don't feel like Im burdening them or wishing they would understand because I know there is a huge group of people who do understand online…

Focus group 4
Int    Since joining an online support group have you felt able to speak to your friends and family about your illness more or less than before?
User95    Much more! It's more validated now it has recognition. Recognition from other sufferers rather than medical. I can share [support group's facebook] status'. People can see what it is for themselves too.

Focus group 5
User20    I'm not sure. Those people I do talk more to have also told about the online group and shared some of the things I have read/learnt on here
User81    Yeah I'm the same probably like it's not just me thinks that makes me feel more confident

This latter aspect of group membership, namely, that it provides validation of the illness identity was particularly important in the absence of formal medical recognition (Tierney et al., 2009). Showing friends and family that the individual is "not the only one" with this disorder, but that it is an identity that is shared with others, can serve to legitimise their identity in the eyes of important others. Therefore, this suggests that recovery identity-relevant relationships can help to maintain other important group memberships that will persist after recovery.

## 4. Discussion

The aim of this paper was to explore the ways in which important group memberships (and the social identities derived from them) impact on recovery from Diabulimia. Our findings suggested that identity-based support from similar others is a key part of the recovery process (Best et al., 2016; Frings & Albery, 2015). A shared recovery identity formed the basis for the provision of psychological resources necessary to manage symptoms and cope with the challenges associated with recovery – in particular the lack of understanding and knowledge that exists both within informal and formal support networks. In addition, this group membership was seen as complementary to other

identity networks that were crucial to recovery and was perceived to help facilitate interactions with those who might misunderstand the nature of the disorder.

### 4.1. Including important group memberships in the treatment and recovery process

Clinical services in mental health often use group programmes as a means of achieving therapeutic change (e.g. Cruwys et al., 2015). However, the nature of difficulties presented by the participants in this study may make face-to-face group programmes an impractical solution, as the comparative rarity of the co-occurring difficulties of diabetes and eating disorders would not allow a sufficient critical mass of Diabulimics in one locality to meet with regularity. Nevertheless, the findings of this study imply the importance of clinical services who treat such individuals undertaking efforts to somehow incorporate social networks successfully into the treatment and recovery process. The accounts of these participants suggest that clinicians should not be afraid of supporting individuals to access online fora for this purpose, and perhaps future service developments could consider how such groups could be formally created and endorsed within the wider service context.

In such cases, developments require a clear understanding of the identity dynamics involved. These groups should facilitate the identity transition necessary for successful recovery (Best et al., 2016). This implies that not only should they endorse norms reflecting health-enhancing behaviours (and avoid emphasising the importance of a particular body shape or dieting, etc.) (Best et al., 2016; Cruwys et al., 2015; Cruwys & Gunaseelan, 2016; Frings & Albery, 2015), but they should also create a social context in which the individual affected feels that they are accepted and their issues are taken seriously (Leonidas & dos Santos, 2014; Linville et al., 2012).

The findings of the current study are reflective of previous research on the role played by social networks in eating disorder and Diabulimia recovery. First, our findings indicate that this cohort has developed a strong illness identity that is perceived to be separate from other eating disorder subtypes (Allan, 2015; Allan & Nash, 2014). Similar to those in recovery from addiction, establishing connections with similar others to construct a recovery identity was seen to be a crucial step in the recovery process (Best et al., 2016; Buckingham et al., 2013; Dingle, Stark, et al., 2015; Frings & Albery, 2015). The sense of shared identity established in the online group facilitated the provision of important psychological resources including emotional support, strategies for managing symptoms, and information relating to the adverse consequences of continuing to engage in disordered behaviours, such as insulin omission. However, our data does suggest that connections with a recovery-oriented group are not free from the potential to hinder recovery. Our findings emphasise the importance of establishing an identity based on the principles of recovery and ensuring enforcement of group norms related to this in order to ensure online (or any form of support group) interactions foster health-enhancing, rather than maladaptive, behaviours (Cruwys & Gunaseelan, 2016; Frings & Albery, 2015; McNamara & Parsons, 2016). These findings also echo research into the effective processes within group programmes, whereby individual changes occur in the context of newly forged ideas of normative changes within a group identity (Cruwys et al., 2015; Frings & Albery, 2015).

Second, consistent with previous literature, family and friends were not perceived as uniformly supportive (Leonidas & dos Santos, 2014; Linville et al., 2012). Clearly such networks cannot connect on the basis of a shared recovery identity and this limits the extent to which support offered can be seen as appropriate to the needs of the individual in recovery (Haslam et al., 2011; McNamara & Parsons, 2016). However, participants did acknowledge that friends and family members who understood and were willing to empathise with them were an important source of support (Linville et al., 2012). Similarly, engagement with

health professionals was also recognised as essential for recovery. However, to the extent that professionals dismissed (or were hostile to) participants' identity, the support offered was perceived as unhelpful and even detrimental to recovery (Stevenson et al., 2014; Tierney et al., 2009). This lead in some instances to participants reporting that they disengaged from support services that were not viewed as identity-congruent. Access to services is often dependent on assuming a certain social category (Walter et al., 2015). In this instance, participants resisted the category imposed by services and wished to engage with services on the basis of their 'Diabulimic' identity. Further research is needed to investigate the extent to which identification (or not) with diagnostic categories impacts on certain groups' willingness and ability to access the services necessary for health and wellbeing. Such issues are applicable to groups beyond that discussed in the current study and include those with stigmatised identities such as those with mental illness and substance abuse disorders.

Finally, one concern typically associated with membership of online groups is that they can become an inappropriate substitute for real-world relationships (Jetten, Haslam, Haslam, & Branscombe, 2009). Furthermore, identifying with a group oriented toward illness might limit the development of a complete recovery identity (Federici & Kaplan, 2008; Tew et al., 2012). Our findings address both of these issues. First, the online recovery group was seen to complement rather than replace health service engagement. Group membership facilitated positive interactions with health professionals by providing information that could be used to structure appointments and ensure that participants made the "best use" of this time (Read et al., 2015). Second, some participants also suggested the group facilitated conversations with supportive friends and family members by providing them with a validation of their illness identity and the confidence to disclose their difficulties, not just online, but to their important social relationships. While some caution is noted given the potentially damaging impact of online interactions (Haas et al., 2011; Mulveen & Hepworth, 2006), the associated norms of engagement with outside relationships could be built upon to promote the creation of links with groups that are not eating disorder or Diabulimia related to ensure the move to a recovery identity that is not based on a disordered identity (Federici & Kaplan, 2008; Tew et al., 2012).

### 4.2. Strengths and limitations of the current study

There are a number of strengths to the current study. First, there is a dearth of research on the role that social relationships play in eating disorder recovery (Leonidas & dos Santos, 2014; Linville et al., 2012). Furthermore, to our knowledge, there is no research on the experiences of this particular cohort in seeking support from social relationships during recovery. This study moves beyond understanding poor treatment engagement as a consequence of personality factors and considers the role played by group memberships and associated identities. Consistent with previous research in this tradition, the current study illustrates that connections with similar others can promote recovery and that during recovery, these connections become an important part of the individuals' social networks (Best et al., 2016; Dingle, Cruwys, et al., 2015; Dingle, Stark, et al., 2015; Frings & Albery, 2015). It also includes an exploration of the role played by other important formal and informal support networks that are crucial to recovery but that are not always fully incorporated into the recovery process. In particular, the current study illustrates the importance of identity-congruent support services to facilitate the construction of these connections and demonstrates the potential of the recovery group membership to assist in the maintenance of connections with other groups necessary for successful recovery. We suggest that these processes are not just relevant in the context of Diabulimia but are likely to be applicable across a range of addictive disorders as well as other forms of mental illness.

Second, this study highlights the importance of identity content to the recovery process. Recovery-oriented groups that engage in

discussions or behaviours that are not consistent with recovery may impede the identity transition. This is consistent with the growing literature on social identity and recovery (Best et al., 2016; Dingle, Stark, et al., 2015; Frings & Albery, 2015; McNamara & Parsons, 2016). Third, this study supports previous research by Stevenson et al. (2014) highlighting the corrosive effects of stigma on the service provider/service user relationship. Services that promote a sense of shared identity between user and provider or at least a respect for differing identities should help to foster treatment engagement.

However, the study has some limitations. First, a comprehensive understanding of the role played by wider networks suggests the need to collect data from these sources on their perspective of their role in the recovery process. Second, our data reflect retrospective accounts of group experiences, rather than investigating group dynamics in situ. Exploration of the latter could be particularly important to determine exactly how a group manages violation of group norms and ensures that the group remains recovery-oriented. Third, we are unable to precisely state how representative our sample is of the population of people experiencing Diabulimia, given the dearth of statistics for this demographic as a whole. While our sample was female, the online group described in this study is also used by males with Diabulimia. Future research should consider the male experience of living with Diabulimia as well as the prevalence of the disorder among males with Type 1 diabetes. Finally, the use of online recruitment and discussion is a strength in that it engaged participants in the topic of interest using the same means of communication, allowing a level and means of interaction that was directly comparable to their familiar interactions. However, this means of recruitment also created a bias in recruiting people who were existing and ongoing members of the community, who were therefore more likely to report positive experiences. There may have been individuals who suffered through accessing the group but who would not have remained present in order to respond to the advert, and these voices need consideration if possible through other means.

## 5. Conclusions

To conclude, we believe that this study represents an important addition to the social identity and recovery literature. Our findings illustrate the importance of ensuring groups that potentially play a key role in recovery (and positive long-term health outcomes) should be formally incorporated into the treatment process. This requires clinical interventions to address the "relational dimension" of eating disorders rather than solely focusing on eradicating disordered behaviours (Leonidas & dos Santos, 2014, p. 926).

## Contributors

Authors 1, 2 and 3 designed the study and development of data collection materials. Author 1 collected the data, conducted literature searches, and provided summaries of previous research studies. Authors 1, 2 and 4 conducted and refined the qualitative analysis. All authors contributed to and have approved the final manuscript.

## Conflict of interest

Author 3 is the former Director and current Research and Training Manager for the support organisation that assisted with participant recruitment and hosted the secure chat room where the online focus groups were conducted.

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**Appendix C: Macdonald, P., Kan, C., Stadler, M., De Bernier, G. L., Hadjimichalis, A., Le Coguic, A. Allan, J. Ismail, K. & Treasure, J. (2018). Eating Disorders in people with Type 1 Diabetes: experiential perspectives of both clients and healthcare professionals.** *Diabetic Medicine*, *35*(2), 223-231

## Research: Educational and Psychological Aspects

# Eating disorders in people with Type 1 diabetes: experiential perspectives of both clients and healthcare professionals

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## Abstract

Aims To explore the experiential perspective of people with Type 1 diabetes mellitus and eating disorders and that of the healthcare professionals treating them, and to understand the experience of both sides to inform future development of healthcare services.

Methods Participants were recruited from Diabetics with Eating Disorders (a national UK charity), and through professional networks. Nine partially/fully recovered individuals with Type 1 diabetes and eating disorders and eight healthcare professionals participated in semi-structured interviews carried out by medically trained researchers. Data were transcribed and coded using a six-stage framework of thematic analysis.

Results Four superordinate themes and several subordinate themes emerged from the Type 1 diabetes and eating disorders dataset: (1) perceptions surrounding service provision; (2) reflections on the recovery process; (3) the experiential perspective of living with Type 1 diabetes and an eating disorder; and (4) support mechanisms. Healthcare professional data elicited three superordinate themes and several subordinate themes: (1) service provision; (2) personal insight and reflection of professional role; and (3) challenges of working with dual diagnoses.

Conclusion People with Type 1 diabetes and eating disorders and their healthcare professionals provided insight into healthcare services from the patient and care delivery perspectives. There was general agreement from both groups that a multidisciplinary, collaborative (family inclusive), clinical approach to treatment is important, as well as adequate training opportunities for service providers. These findings may help to inform development strategies for multidisciplinary care approaches to Type 1 diabetes complicated by eating disorders.

Diabet. Med. 35, 223–231 (2018)

## Introduction

### Risk and problems

Eating problems and eating disorders are twice as common in people with Type 1 diabetes mellitus as in people without diabetes, with a prevalence ranging from 8% to 36% [1–6]. A specific feature of eating disorders in Type 1 diabetes is omission/reduction of insulin to control weight and compensate for eating [7,8]. Interestingly, girls/women are 10 times more likely have an eating disorder than boys/men, and the same applies to the Type 1 diabetes population [9]. Although

some people with Type 1 diabetes and eating disorders fulfil the standard criteria for an eating disorder, not all of the symptoms and behaviours related to the Type 1 diabetes comorbidity are currently appropriately reflected in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition; for example, the omission/reduction of insulin to control weight and compensate for eating [7,8]. Eating disorders in the context of Type 1 diabetes are associated with poorer glycaemic control and accelerated development of late complications of diabetes (retinopathy, neuropathy, cardio- and cerebrovascular events, limb amputation) and increased rates of acute complications (diabetic ketoacidosis and severe hypoglycaemia), resulting in a threefold increase in mortality compared with people with Type 1 diabetes without eating disorders [5,7,10–13].

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### Models of illness

The Medical Research Council recommend that the model of illness framework is a good starting point for developing complex interventions. There are many contributory risk or maintaining factors [14,15], and a variety of models for Type 1 diabetes and eating disorders have been constructed. Most include poor adjustment and difficulties in coping with diabetes, body image concerns, emotional dysregulation and social problems [15–18]. The treatment skills require integration of expertise in the management of Type 1 diabetes, including its technologies, and knowledge about the relative importance of the macronutrients, eliciting and challenging unhelpful psychological beliefs and effects as well as any underlying developmental effects, such as insecure attachments.

### Treatment/services

In general, there is considerable uncertainty about how treatment and services for people with Type 1 diabetes and an eating disorder should be provided because poor outcomes have been achieved with standard eating disorder treatments, with individuals often dropping out early [19]. The standard evidence-based self-management strategies for Type 1 diabetes are complex and require a major focus on food macronutrient content (carbohydrate counting) in order to be able to match the multiple daily insulin injections, eating and exercise patterns, and current blood sugar levels with the target of keeping blood glucose levels in near-normoglycaemic range to avoid late and acute complications [20]. By contrast, eating disorder teams encourage people to stop calorie counting, be flexible in eating behaviour and moderate their high standards and perfectionism [21,22]. It can therefore be difficult to build a model of care that fits within the remit of both groups.

The aim of the present study was to explore experiences of treatment and service provision from the perspectives of people with Type 1 diabetes and eating disorders and of healthcare professionals (HCPs) in both specialist diabetes and eating disorder settings.

## Methods

### Recruitment and sample

People with Type 1 diabetes with eating disorders were recruited from a national charity, Diabetics with Eating Disorders, and through professional networking. Adult women with Type 1 diabetes (aged ≥18 years), who had a current or previous diagnosis of eating disorders as defined by Diagnostic and Statistical Manual of Mental Disorders, 5th edition, and who had either fully recovered or were in a stable medical condition were enrolled. Participants were purposively selected to reflect the experiential perspective at different ages, duration of illness and stages of recovery. Ethics approval was granted by UK National Health Service Health Research Authority (REC ref 14/LO/0423).

The HCPs were purposively selected from a range of disciplines/professions at King's College Hospital London and South London and the Maudsley NHS Foundation Trusts and large tertiary care centres in South London. Inclusion criteria for the professional group required nurses, therapists and psychologists to have had at least 1 year's experience of working with adults with Type 1 diabetes and eating disorders. Participants of both groups had to be fluent in English.

### Data collection

Face-to-face semi-structured interviews were carried out by three researchers (A.L.C., G.L.B. and A.H.) over the course of 1 year and lasted for ~1 h. Topic guides were developed from clinical experience and included open-ended questions pertaining to the research questions (Appendix 1). Having gained appropriate consent, all interviews were audio-recorded and transcribed verbatim.

### Data analysis

During transcription, the typed transcripts were anonymized using ID numbers for all participants. A thematic analysis approach was then adopted, which is a qualitative method for identifying, analysing and reporting patterns or themes within a dataset. We based our analysis on Braun and Clarke's six-stage framework of analysis [23].

Two researchers worked on the coding at each stage of the analysis (P.M./G.B. and P.M./A.H. on the thematic framework for the data obtained from people with Type 1 diabetes and eating disorders, and P.M./A.L.C. for the data from HCPs). The transcripts were transcribed and read several

times (phase 1), with initial codes generated before the transcripts were collated into potential themes (phase 2). Researchers worked independently in the identification of themes, and several face-to-face meetings and Skype calls took place to discuss emerging themes, an example of which included whether 'Disagreements between profes- sionals' should be merged into the sub-theme 'Splitting'. This was changed, after discussion, to 'Disagreements and splitting within the team'. Thus, after discussion, themes were reviewed and refined, sub-themes consolidated or merged into existing themes, and descriptive labels altered to better reflect the subject matter or deleted if deemed irrelevant to research aims (phase 3). Each finalized theme represented an idea acquired from the data, representing all the contained sub-themes pertinent to the research aim. The computer software programme, NVIVO, was used to manage the data [24]. In both frameworks, it was mutually agreed between the researchers that data saturation had been achieved, i.e. that no new themes were emerging from the narratives.

## Results

### People with Type 1 diabetes with eating disorders

Participants' characteristics are described in Table 1. The final codebook for thematic analysis is shown in Table 3.

#### Personal accounts of living with eating disorders and Type 1 diabetes

Participants offered detailed accounts of living with both an eating disorder and diabetes. There was a deep awareness of both physical and psychological states that influenced behaviours and thoughts deemed to be prob- lematic in recovery, some of which included accounts of not injecting, denial, perfectionism and rigid thinking patterns.

> I can get into these vicious circles where you feel bad so you don't do anything then you feel bad for not doing anything .. . That's hard to get out of.
>
> (Participant 1)

Perceived connections or incompatibilities between the eat- ing disorders and diabetes were also raised in terms of interactions or links between the two conditions. One participant, for example, said that having been always self-conscious and focused on weight, the diabetes had come as an extra burden.

> I think I would have had an eating disorder anyway but I don't think the diabetes helped at all because obviously, the focus on food and having to be very aware of what you're eating and numbers .. .
>
> (Participant 1)

There were several reports of potential triggers that may have contributed to the development of an eating disorder or even exacerbated it. These ranged from being in denial of having diabetes, coupled with a desire to be 'normal', to develop- mental issues surrounding puberty, such as fad diets, body image and the consequences of restricting insulin. Links between having hypoglycaemic events and binge-eating were also reported.

> Having diabetes, one had hypos and it was actually a hypo that spurred my first binge. I had this really bad hypo and ate some fudge – and I ended up eating the whole lot. Then the binge–starve cycle started.
>
> (Participant 6)

Four women spoke of the benefits of having Type 1 diabetes and an eating disorder, citing feelings of familiarity, focus, control and gaining a sense of identity as positive factors. There was, however, also a sense of empathy and advice to others still in recovery, e.g. the importance of seeking help, perseverance, developing confidence, examining core values and developing a keen sense of awareness.

Table 1 Patient characteristics

| Participant ID | Age | Type of eating disorder (time of diagnosis) | Duration of eating disorders illness, years | Duration of diabetes, years | Perceived stage of change* |
|---|---|---|---|---|---|
| 1 | 26 | Anorexia nervosa | 13 | 8 | Acute |
| 2 | 21 | Bulimia nervosa | 2.5 | 10 | Partially recovered |
| 3 | 31 | Anorexia nervosa (initially)/Bulimia nervosa (later) | 14 | 23 | Recovered |
| 4 | 22 | Anorexia nervosa (initially)/Bulimia nervosa (later) | 3 | 11 | Partial recovery |
| 5 | 18 | Anorexia nervosa (initially)/Bulimia nervosa (later) | 4 | 8 | Partial recovery |
| 6 | 34 | Eating disorder not specified | 6 | 7 | Partial recovery |
| 7 | 26 | NA | 1.5 | 20 | Recovered |
| 8 | 27 | Bulimia nervosa | 7 | 13 | Partial recovery |
| 9 | 62 | Bulimia nervosa (initially)/Binge-eating disorder (later) | 42 | 49 | Recovered |

NA, not available.
*Acute: the eating disorder has substantial and immediate impact on a participant's ability to live a meaningful life; Partial recovery: the eating disorder has some impact on a participant's ability to live a meaningful life. Recovered: the eating disorder has limited/no impact on a participant's ability to live a meaningful life.

Recognizing the signs, definitely ... Also, to help that person make the link. I think for a long time I was like 'Great, I've got the pressure of diabetes and now I've got an eating disorder that has come from nowhere.

(Participant 8)

### Recovery coping mechanisms and strategies

All participants spoke of behavioural techniques and coping strategies used during recovery. These included the acquisition of psycho-educational materials, participation in normal lifestyle activities and the importance of accessing help and support. The triggers that led to increased motivation to adopt a more adaptive approach were less clear. Neverthe- less, it was useful to hear goal-oriented actions and talk of staged recovery, the achievement of which often resulted in higher self-esteem and confidence.

... every injection you make they are all small victories and keeping reminding yourself that, reminding yourself of the good things that have happened in your recovery.

(Participant 4)

Similarly, reports of cognitive re-framing and changing perceptions suggested a growing understanding of self. One participant spoke about unhelpful contributory factors such as perfectionism and obsessive thoughts, whilst another spoke about the need to 're-programme the brain to a different pattern'. One woman reported a changing perspective after retinal screening and the realization of how close she had come to losing her vision. Several other women spoke of an acceptance and realization of the severity of illness that then prompted changing perceptions to a healthier mindset and increased self-belief. Participants, in general, attributed vari- ous reasons to changing healthier perceptions from 'eureka moments' to a realization of what they were doing to their bodies as well a growing sense of self compassion.

... being able to be a bit kinder to myself, which I had never done before. Definitely when I stopped the cruci- fying, that's when things started to get a little better. So, strength-wise, I learned to be kinder to myself, and I never ever done that before.

(Participant 3)

Awareness of the benefits and desire for a healthier lifestyle also appeared conducive to making positive changes, such as the desire for a greater sense of well-being, renewed social life, re-engaging in normal life, as well as signs of 'bigger picture' thinking in terms of career plans, health, marriage and children. There was a sense of regret at lost opportunity and several references made to the emptiness of living with Type 1 diabetes and an eating disorder, in contrast to the richness of a fuller and healthier life. The recovery process was not linear in nature; responses indicated that there were steps forwards and backwards and ongoing challenges to address.

When I'm binging, that's the problem, ... that's when it gets out of control, and my weight does start going up and up. And that's when I get scared, and then I do whatever I can to try and stop that.

(Participant 7)

### Thoughts and suggestions for appropriate medical care provision

Participants provided a unique experiential insight into issues surrounding aspects of service provision. Although data reflected positive reports and experience of clinical care, there appeared to be greater occurrences of negative accounts. These mainly focused on a lack of understanding about the links between the two conditions. There were also several reflections on the potentially detrimental effects of unhelpful guidance, inadequate care or even neglect.

Then there were incidents with things like them forgetting to give me my insulin, forgetting to do blood tests ... I was in there to be treated for my eating disorder and part of that is struggling with taking it so for me to have to remind them that I needed to do it or that they needed to test my blood, was just ridiculous.

(Participant 1)

Most interviewees expressed the need for the medical profession to have a greater knowledge and understanding of the illnesses in the context of a multidisciplinary, collaborative approach. Responses highlighted the impor- tance of greater awareness and understanding, appropriate training and adequate knowledge of both illnesses, along with early recognition in terms of referrals.

I think you really have got to have the multidisciplinary approach, you have to have different places on board. You have to be willing to liaise with the diabetes team, and you really have to work together to pull all your knowledge together.

(Participant 5)

Participants offered suggestions of how future service plans should be developed and implemented. It was important for them to be regarded as a valued member, to have emotional support and be listened to without judgment, for there to be mutual trust and for professionals to recognize their unique- ness and individuality in terms of differing needs. It was also important for them to be able to trust that their care team possessed the appropriate education, training and under- standing to treat them and that their families were also given adequate support.

I think it is really important to surround yourself with people who you can trust, and I think that particularly when you are diabetic and you have an eating disorder, you just expect that people won't get it, so you've already got this attitude of they're not going to understand me, so

when you do have the right health professionals it just makes everything so much easier.

(Participant 3)

### Importance of and need for social support

Support and trust from family, friends, university counsellors and work colleagues and mostly from parents and partners, was particularly valued. One woman spoke about the detrimental effects of problematic interactions with her mum. Participants also referred to the need for family education and interventions. In some cases, there was concern that their family was not equipped to address the issues in hand.

I think advice for loved ones is crucial to that person's recovery because I think loved ones panic and they feel anxious and they go 'Oh my God, my darling's starving themselves' .. . and they act in a way they might think is supportive but it might not be, it might hinder .. .

(Participant 8)

Peer support was also greatly valued. The women regularly referred to the benefits derived from talking to other people experiencing similar problems, particularly those people already recovered. Online forums and both face- to-face and online support groups were valued, in particular.

I could go on there 24 hours and there would be somebody else up who would also be in DKA (online forum) or freaking out or you know, wondering why they developed thrush again, or something like that. So yeah that was really, really massively important.

(Participant 3)

### Healthcare professionals

The characteristics of the HCPs are described in Table 2. The final codebook for thematic analysis is presented in Table 3.

### Reflections on role requirements from a professional perspective

The clinicians in the study provided in-depth knowledge and understanding of their professional role. Responses suggested a strong awareness of role differentiation, as well as specialist knowledge in their individual fields; i.e. recognizing the need to refer to various specialists. HCPs also provided personal opinions on specific aspects of care and how these should be addressed.

.. . by comparison to the rest of the eating disordered population, I guess it's just like dealing with another comorbidity, whether we would with depression, in this case it is a physical comorbidity, which is diabetes. HCP 7

(Eating disorders)

There was considerable empathy from the HCPs. They recognized the difficulties and conflict between fear of gaining weight, necessary focus on food intake and consequent temptation to omit insulin. There was a keen sense of awareness and beliefs about their own role in how best to support this group.

It takes a great deal of understanding. As much as they are experts, we do have to keep reinforcing that we can stabilize the diabetes with them and stabilize their eating pattern, their weight does not have to be erratic; it does not have to constantly go up, but we do have to get them to a relatively healthy weight range .. . HCP 5

(Eating disorders)

In general, HCPs showed great awareness of the interactions and relationships between the two conditions and knowledge required to provide gold standard care.

It'd be helpful to be educated in both spheres so that the diabetic consultant has some idea of the impact on triggering eating disorder or maintaining eating disorder .. . and likewise, the eating disorder therapist should understand the clinical implications of diabetes. HCP 7

(Eating disorders)

### Challenges of working with dual diagnoses

By far, the greatest challenge was working with problematic thoughts and behaviours; i.e. insulin omission, difficulties in engaging, high anxiety, unrealistic recovery goals, diagnostic ambiguities, transient nature of barriers put up and threats of self-harm and suicide. Other concerns included responsibility of clinical risk and the anxiety inducing effects on their own team members.

Prepare yourself for the challenge because it's not easy! Because your patient is resistant to both: resistant to having diabetes and resistant to having an eating disorder .. . so it's a double whammy. HCP 7

(Eating disorders)

Limitations and constraints were also a concern. For some HCPs, there was concern about proximity to adequate service provision for their clients, whilst others spoke about the difficulties time constraints imposed with regard to juggling different aspects of the job. One HCP referred to the bureaucracy involved in working within the National Health Service (NHS) framework. There were also confidence issues arising from perceived gaps in training and knowledge.

*I definitely need some assistance most of the time. If I feel like that, then I guess some novice is going to feel even worse.* HCP 3

(Diabetes)

Finally, just under half of the HCPs made reference to clients consciously (or otherwise) causing disagreements within the team. Splitting occurred when clients played specialists

**Table 2** Characteristics of healthcare professionals

| HCP ID | Speciality | Training in other field | Occupation | Length of experience | Specialist area |
|---|---|---|---|---|---|
| 1 | Eating disorders | Cognitive analytical therapy | Psychologist | NA | Couple counsellor Counsellor and Hypnotherapist in private practice. |
| 2 | Eating disorders | Motivational interviewing trainer | Psychiatrist consultant and professor in psychiatry | 20 years | Community treatments Eating disorders; deliberate self-harm; suicide prevention; personality disorders; aetiological factors; brief psychological treatments; new technologies in treatment; health services research. |
| 3 | Diabetes | Motivational interviewing training DAFNE | Specialist diabetes nurse | 20 years | Type 1 diabetes focusing on pregnancy and hypoglycaemia. |
| 4 | Diabetes | Cognitive behavioural therapy, motivational interviewing, eye movement desensitization and reprocessing | Clinical psychologist | 3 years | Fear of hypo/hyperglycaemia; eating disorders (e.g. insulin manipulation for weight loss/ management), adherence difficulties Interests: abnormal psychology, clinical psychology, neuropsychology. |
| 5 | Eating disorders | -Motivational interviewing training, - -DAFNE (observation) -Diabetes training -Carers skill workshops -Supported eating training on unit | Nurse (eating disorders outpatients) | 14 years | Specialist physical health care nurse/Charge nurse |
| 6 | Diabetes | Undertaken courses in motivational interviewing and cognitive behavioural therapy strategies | Registered Diabetes Dietician | 6 years | Transition DAFNE Educator and Peer reviewer |
| 7 | Eating disorders | NA | Consultant Clinical Psychologist (eating disorders outpatients) | NA | NA |
| 8 | Diabetes | DAFNE patient education programme | Dietician | 12 years | Diabetes specialist Dietician, Clinical lead dietician |

DAFNE, Dose Adjustment for Normal Eating; HCP, healthcare professional; KCL, King's College London; NA, not available.

against each other by using lies or deceit to achieve individual desires and goals. This also included disagreements between team members and/or their approach to care.

> Because often the patients know a lot about diabetes, so they can blind the psychiatrist or eating disorders person with the diabetes .. . . They will cheat the nurse. HCP 3
>
> (Diabetes)

> I do get quite frustrated when a professional says, "from now on, instead of taking no insulin, you're going to take 20 units, three times a day". In what world is that ever going to happen?! I think it's a discussion with the patient. HCP 6
>
> (Diabetes)

*Insights into current and future service provision*
All HCPs offered in-depth insight into aspects of care, both current and future, with suggestions for service development.

Clinicians offered examples of strategies and approaches that worked well, including specific aids, information, education, behavioural and emotional strategies. Other tools included didactic information and encouraging bigger picture thinking, coaching, goal setting, use of motivational interviewing and written work as well as aids to help with diabetes, for example, glucose sensors.

> It is a bit like with anything really, in terms of therapy, CBT [cognitive behavioural therapy] particularly. It's about getting them to work gradually towards a goal. Obviously, the fear is about putting on weight. .. .. It's about making small steps to try and increase the level of insulin rather than taking no insulin at all. HCP 4
>
> (Diabetes)

All participants emphasized the importance of team collaboration, support and being part of a multidisciplinary team. This gave them the opportunity to bridge any gaps in

**Table 3** Final codebook used for thematic analysis

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People with Type 1 diabetes and an eating disorder

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Personal accounts of living with Type 1 diabetes and an
  eating disorder

- Descriptions of the mental and physical adverse effects
- Links and interactions between the illnesses
- Illness development and perceived triggers
- Advice and empathy for others
- Perceived benefits to illnesses

Recovery coping mechanisms and strategies

- Improved self-efficacy and coping strategies
- Cognitive reframing and changing perceptions
- Benefits and desire for a healthier lifestyle
- Ongoing challenges and awareness of relapse triggers

Thoughts and suggestions for appropriate medical
  care provision

- Positive experiences and reports of care from
  clinical service providers
- Problematic clinical care including advice and guidance
- The need for a holistic, multi-disciplinary approach
  to treatment
- Suggestions on how services can be improved

Importance and need for social support

- Positive impact of social support
- Need for family interventions
- Peer support
- Problematic family dynamics and interactions

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Healthcare professionals

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Reflections on role requirements from a
  professional perspective

- Role perception, beliefs and understanding
- Empathy and understanding for patient perspective
- Required knowledge for diabetes and eating disorders
  specialists
- Interactions between the illnesses

Challenges of working with dual diagnoses

- Working with challenging thoughts and behaviours
- Financial, manpower and service proximity constraints
- Gaps in training, knowledge and support
- Disagreements and splitting within the team

Insights into current and future service provision

- Effective therapeutic strategies and approaches
- Importance of working in a multidisciplinary team
- Family influences, education and inclusion]
- Suggestions for service development
- Importance of therapeutic alliance

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knowledge. Most HCPs emphasized the fact that they could
not provide service provision in isolation.

*I couldn't imagine just doing it on my own. There is no
way I'd be able to. I don't have the skills. You need*

*support ... When I sit down with a psychiatrist, I see them
tackling things much more head on or much more directly
than I would, and moving patients more than I can in these
situations.* HCP 3

(Diabetes)

All HCPs made some reference to the influence of family and
importance of family inclusion. The usefulness of psycho-
educational skills programmes and the development of
family skills interventions was frequently raised. There was
also mention of potential complications in family involve-
ment because of either problematic family dynamics or
resistance of patients to having their family involved in their
care package.

I think that particularly within the adolescent group
working with family is absolutely vital. Because some-
body growing up with diabetes, there tends to be a greater
sense of dependency on the family. That can become a kind
of conflict as an adolescent control that can be quite
difficult in terms of family dynamics. You can get a lot of
family conflict arising. A young person trying to take more
control in terms of diabetes or rebelling about the diabetes.
Working with family so that they can support their child
with diabetes … For example, if they get a lot of cravings
and are really struggling with binge-drinking, if there's
loads of chocolate and temptation in the house, that can
potentially undermine the efforts of the young person. It is
a family effort. HCP 4

(Diabetes)

Seven clinicians provided suggestions on service development
including more training, supervision and improved team
collaboration. There were several references to professional
training programmes, e.g. motivational interviewing training
for the Diabetes team was mentioned and courses run by
Dose Adjustment for Normal Eating (DAFNE) for those
individuals working in Eating Disorders.

… DAFNE has helped us to understand as a team that, as
much as they can evoke these feelings of real anxiety in us,
we can quite easily contain and manage that. HCP 5

(Eating disorders)

Finally, six HCPs raised the need for establishing a warm,
trusting alliance with those individuals with Type 1 diabetes
and an eating disorder, emphasizing the importance of
building rapport and a strong bond/relationship.

## Discussion

The aim of the present paper was to carry out an exploratory
qualitative study to examine clients' and HCPs' viewpoints
about treatment and service provision. The overriding theme
was the need to adopt a model of care based on shared
expertise across diabetes and mental health and training,
encouraging relevant specialist teams to become involved

early in care planning and management. There was widely shared agreement between the two samples of the need for a multidisciplinary, collaborative approach to treatment provision that offers expertise in diabetes, eating disorders and other psychological problems. There were examples of poor practice where this failed to occur.

In terms of the specific psychological/behavioural treatments, it is important to note that there is often other psychiatric comorbidity in addition to the eating disorders in people with Type 1 diabetes. In a study by Allan [15], over three-quarters of the sample (77.5%) were formally diagnosed with another psychiatric condition, highlighting the need for professionals to develop transdiagnostic skills to provide adequate care for this complex group. In the present study, clients described cognitive re-framing, changing perceptions and bigger picture thinking, often outlined within a goal-oriented process, as important tools for recovery. Psychological therapies specifically designed for people with Type 1 diabetes and eating disorders are, therefore, likely to be beneficial [15].

The majority of participants valued support from family, friends and peers, and many HCPs commented on the importance of family inclusion. These findings support previous work [25,26]. For example, an analysis of feedback from the DEPICTED study suggested that, for young people, carers may need training in patient-centred communication skills and emotional needs also need to be considered [27]. Some services discourage family involvement following the transition from adolescence into adult services. Hostility, criticism or over-involvement (expressed emotions) are common and natural responses from carers, and expressed emotion has been found not only to be a maintaining factor of eating disorders [28] but also to have negative implications for diabetes control [29,30]. Equipping carers with skills, such as motivational interviewing with an emphasis on affirmations and avoiding conflict, may be of particular value for people with Type 1 diabetes [31].

We believe that this is the first study to examine the experiential perspectives of both people with diabetes and eating disorders and HCPs on aspects of both living with and working with Type 1 diabetes and eating disorders. The study has several limitations. Firstly, most of the HCPs interviewed were recruited from a large research establish- ment with a strong emphasis on psychological support, therefore, this sample of HCPs may have had access to more resources, in terms of training and support. Secondly, most of the people with Type 1 diabetes and eating disorders were members of a national charity and may have been more vocal in expressing their needs. Thirdly, there were a few leading questions in the topic guides and, finally, no men with Type 1 diabetes and eating disorders, participated in the study.

In conclusion, patients and HCPs agree on the importance of an integrated approach to treatment. In terms of the HCPs' needs, appropriate training opportunities, supervision and monitoring are vital prerequisites to adequate service provision. Both clients and HCPs offered useful therapeutic strategies that could help inform future interventions. Consequently, it is essential that future research uses a model of a multidisciplinary, collaborative clinical approach that could be tested in clinical trials, providing an evidence-based treatment service to people with Type 1 diabetes and eating disorders.

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## Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix 1. Topic guides.

Article type    : Research Article

# Research: Educational and Psychological Aspects

# 'Diabulima' through the lens of social media: a qualitative review and analysis of online blogs by people with Type 1 Diabetes mellitus and Eating Disorders

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What's new?

- People with Type 1 Diabetes who also have an Eating Disorder are difficult to treat because the underlying factors are not well understood.

- Multiple factors that trigger and maintain intentional insulin restriction have been identified in this thematic analysis.

- Taking small steps at a time is a key recovery strategy described by people with diabulimia.

- Triggers for insulin omission behaviour, as well as the recovery strategies identified in the present study, may contribute to the future development of a clinical intervention for people with Type 1 Diabetes and an Eating Disorder.

**Aims** To perform a qualitative review of online blogs authored by people self-identifying as having Type 1 Diabetes and an Eating Disorder or 'diabulimia', a term used by people with

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Type 1 Diabetes to describe an Eating Disorder that is characterized by deliberate restriction of insulin to control weight.

**Methods** We conducted a structured qualitative review of online blogs published between 2012 and 2017 authored by people who report having Type 1 Diabetes and an Eating Disorder or diabulimia. The subsequent thematic analysis followed a six-phase process and was conducted by two independent researchers.

**Results** From 147 000 search results, 11 blogs (304 posts) matched criteria for further analyses. Three key themes and 18 subthemes emerged: 1) different aspects of bloggers' relationship with insulin, including motives for omitting insulin, secrecy of insulin omission and perception of control; 2) bloggers' experiences of Diabetes complications, and Diabetes ketoacidosis in particular, as well as their worries about future complications; 3) strategies for recovery and triggers for relapse, which involved Diabetes self-management and setting up a support system.

**Conclusions** Qualitative analyses of blogs authored by people with Type 1 Diabetes and an Eating Disorder or diabulimia have identified high levels of Diabetes distress and provided insight into different motives for insulin omission and strategies for recovery. Considering the limited evidence for effective interventions, these findings may help the development of complex interventions to improve biomedical and psychological outcomes in this group.

**Introduction**

A diagnosis of Type 1 Diabetes mellitus is a life event, and living with the condition involves multiple significant practical challenges. Self-managing Type 1 Diabetes entails frequent checking of blood glucose levels, calculating the carbohydrate content of meals, considering the effects of exercise, and self-injecting insulin in adjusted doses. These burdens and role

transitions are predisposing factors for mental disorders, such as depression and Eating Disorders, and for Diabetes-specific distress, such as fears of insulin-related weight gain and fear of acute and chronic Diabetes complications [1,2].

Eating Disorders are amongst the most common mental comorbidities of Type 1 Diabetes.

Eating Disorder prevalence is twice as high in young people with Type 1 Diabetes compared with the background population [3]. These include, but are not limited to, bulimia nervosa, binge Eating Disorder and anorexia nervosa [4].

'Diabulimia' is not currently a diagnosis separate from generic Eating Disorders in standard psychiatric classifications [International Classification of Diseases, tenth revision (ICD-10) and Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5)]. Diabulimia is a term often used by people with Diabetes to refer to their condition because it is only possible in Type 1 Diabetes. Diabulimia is characterized by the fear that insulin causes weight gain and by the deliberate restriction of insulin to control weight. Insulin restriction is associated with a threefold greater risk of mortality in people who practise this, compared with people with Type 1 Diabetes who do not [5].

Mortality is secondary to acceleration of microvascular and macrovascular complications [6,7] and to acute complications such as diabetic ketoacidosis (DKA) or severe hypoglycaemia [8,9]. There is currently no effective intervention that improves Diabetes control and mental health in people with Type 1 Diabetes and an Eating Disorder [10].

A greater understanding of the thoughts, beliefs and experiences associated with intentional insulin omission, Eating Disorder behaviour, and recovery from Eating Disorder in people with Type 1 Diabetes and Eating Disorder is needed to inform the development of effective interventions.

Blogs are personal diary-type posts published online by an individual. Structured analyses of blogs represent a novel method of qualitative research as they give access to a range of ready-made narratives [11–13].

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The aim of the present study was to perform a qualitative review of online blogs authored by people self-identifying as having Type 1 Diabetes and an Eating Disorder, or diabulimia.

## Participants and Methods

### Data collection

We applied methods previously developed for a structured approach for qualitative reviewing of blogs [11–13]. Data for thematic analysis consisted of written content from personal online blogs, obtained through the search engine Google. Data collection began in February 2017 by identifying blog sites using the search terms 'Type 1 Diabetes' AND 'Eating Disorder' OR 'bulimia' OR 'anorexia' OR 'binge eating' OR 'diabulimia' AND 'blog'. The results were ranked in order from the most to least relevant, based on keyword occurrence. Each of the websites were independently reviewed by two researchers (E.S. and M.S.) to assess if they met the following inclusion criteria: content located on a publicly accessible and personal blog site or forum; blog published within the previous 5-year period; author self-identifying as having Type 1 Diabetes and an Eating Disorder (including the term diabulimia); and blog written in English.

Blogs were excluded if the author mentioned other existing chronic conditions unrelated to their Diabetes or Eating Disorder (e.g. cancer, neurological disease). We excluded blogs authored by third parties (medical professionals, care givers, journalists) and blogs published on sites maintained by service providers. Links to related blogs that were found within blogs were also included if they met the criteria (*n*=2).

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All posts within each blog were fed into the primary analysis. Both analysts read these posts in full and identified those that were related to personal experience of either Diabetes or Eating Disorder or diabulimia. These were included in the next step for the coding process of the thematic analysis. In contrast to a semi-structured interview study or a focus group study with a clearly defined interview or topic guide, blogs also included posts referring to some everyday life topics that were not related to our research question and posts citing or linking to other sources (newspaper and research

rticles) rather than personal experience. We excluded posts that were not related to the research topic. For each blog a maximum of 15 posts (the most recent) were included in the analyses.

Informed consent was not required as this research focuses on the blog rather than the blogger and only public blogs were used [14,15]. All data were password protected and blog authors were given a non-identifiable ID.

**Data analysis**

A thematic analysis approach was adopted, which is a qualitative method for identifying, analysing and reporting patterns within a dataset. We applied Braun and Clarke's six-phase framework of analysis [16]. Firstly, the study authors familiarized themselves with the data and read all blog posts in full (primary analysis). Blog posts related to the research topic were then input into Nvivo software for data management, where initial codes were created by two researchers independently and then jointly. Both worked on the coding at each stage of the analysis. The blog transcripts were read several times and initial codes were generated before being collated into potential themes. A blog extract could be un-coded, coded once or coded multiple times. Researchers worked independently in the identification of themes, and several

face-to-face meetings took place to discuss emerging themes. Both analysts agreed that data saturation had been reached (i.e. no new codes could be created).

Themes and subthemes were agreed through a collaborative process between the two authors (one diabetologist, one psychologist) conducting the analysis, and discussed within the larger multidisciplinary group of co-authors (psychiatrists, psychologists, people living with Diabetes). This multidisciplinary approach was taken to allow a broad approach to the qualitative analysis because of the different professional backgrounds and experiences. Themes were reviewed and refined, subthemes consolidated or merged into existing themes, and descriptive labels altered to best reflect the subject matter or deleted if deemed irrelevant. Lastly, theme names were defined, which ensured blog content was fully captured, and a report produced.

## Results

### Systematic search

Figure 1 is a flowchart illustrating how the blogs were systematically reviewed. The search terms produced 147 000 results. The first 100 results produced 11 relevant blogs (304 posts). We had demographic information for seven bloggers, whose age ranged between 25 and 34 years. All bloggers identified as female. Bloggers came from the UK (*n*=4), the USA (*n*=4), or did not provide information on their location (*n*=3). The number of posts per blog varied between three and 124. Of the 11 bloggers, 10 referred to their condition as 'diabulimia'. Bloggers also mentioned 'anorexia', 'Eating Disorder' and 'Type 1 Diabetes with an Eating Disorder'. One blogger talks about professionals referring to her as a 'SEED (severe and enduring Eating Disorder) patient'.

**Thematic analysis**

Three main themes emerged: bloggers' relationship with insulin; bloggers' experience of Diabetes complications; and strategies for recovery and triggers for relapse. The themes and subthemes are presented in Fig. 2.

**Relationship with insulin**

The majority of bloggers reflected on their use (and omission) of insulin in an emotionally charged and multi-layered way, as if they were reflecting on a relationship that was difficult and complex. This theme was therefore labelled 'relationship with insulin'. This theme was rich and multifaceted because there was a broad spectrum of perceptions, thinking patterns, emotions and behaviours related to insulin administration and insulin omission, respectively. For example, a wide variety of factors that trigger and maintain the deliberate manipulation of insulin, which is the key behaviour driving diabulimia, was described.

Bloggers' perceptions of the influence of insulin on body weight was a subtheme that occurred frequently in the blogs. They reported that giving the correct dose of insulin immediately led to sudden weight gain. Bloggers expressed their worry and fear of gaining weight as a consequence of injecting insulin. Some described that they began to associate insulin with fat. They also described the struggle they had when reintroducing insulin, because it was associated with weight gain.

> *The weight gain was psychologically crippling.* (ID 113)

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> *My weight ballooned and I gained about two stone - the teasing at school began and thus I*
>
> *began to associate insulin with fat.* (ID 111)

Experimenting with insulin was driven by a broad spectrum of triggers that ranged from fear of gaining weight to a perception of control and intentional manipulation. One blogger compared manipulating insulin with a game. Several bloggers appeared to have gone through a process of conscious decision-making, almost a quasi-experimental approach to their own body, when they first started intentionally omitting insulin.

> *At sixteen years old, feeling the pressures of being around other girls in sixth form, I first made*
>
> *the conscious decision to cut out my insulin in order to lose weight.* (ID 111)

> *This was when the 'experiment' started. I decided to experiment with my insulin; taking less*
>
> *ad less, until I was taking none at all.* (ID 113)

Some bloggers attributed gambling features to their pattern of omitting insulin. Others described how features of addiction (in their own words) became associated with insulin omission.

> *For years I played the game of omission, only intermittently having spurts of inspiration to*
>
> *become 'a better diabetic'.* (ID 121)

> *[…] alike to a stupor I could only imagine would compare to what heroin addicts find so*
>
> *addictive.* (ID 113)

The secrecy of insulin omission for weight loss, as well as a perception of control over body weight by omitting insulin, were described and associated with a sense of empowerment.

*With the secret eating and insulin omission, I began to lose weight.* (ID 111)

*I felt as if it was my secret tool that nobody knew about and nobody else could do.* (ID 111)

The chronicity and cyclical nature of insulin omission behaviour was a theme that emerged from four blogs. The initial experimental approach gradually converted to chronic intentional omission or restriction behaviour, with the intention to manipulate body weight. Other common cognitions included the ambivalence of fearing weight gain but at the same time also wanting good Diabetes control. These cognitions became discordant when the blogger had a binge as a response to negative thoughts and feelings, but also wanted to be a 'good diabetic' by giving the correct amount of insulin that matched carbohydrate intake of the binge, in order to not worsen Diabetes control.

*I would omit my insulin to get the extra few pounds off and then would stop. Anybody with Diabetes and an Eating Disorder will know that this is never the case. Once becomes twice, twice becomes three times and so the cycle continues.* (ID 111)

*I thought if I couldn't stop the binging, I could at least stop the weight gain. So anytime I binged, I wouldn't take insulin.* (ID 119a)

**Experience of Diabetes complications**

Almost all blogs described the experience of acute and/or chronic Diabetes complications. The language used by the bloggers was full of medical terms to describe their acute complications leading to hospital admission, the chronic complications they experienced and how they coped with these in their daily lives. In spite of the medical terminology they used, this was a theme

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where the bloggers linked strong emotions to their experience, and described how they coped and their thoughts about their future health.

The most described Diabetes complication in the blogs was DKA. It was common for most bloggers to describe hospital care for the treatment of DKA. Some blogs read like rational and distanced descriptions of the DKA experience. The bloggers also described the opposite experience of an excessive insulin dose leading to severe hypoglycaemia, which in turn triggered compensatory overeating behaviours.

*I was in such severe DKA that I needed to be in their ICU for the first 4 days.* (ID 112a)

*I must have overshot with the insulin, sent myself crashing through the roof and the floor.* (ID 115)

Although DKA and severe hypoglycaemia are medically classified as acute Diabetes complications, there were descriptions of frequent and repetitive episodes of acute Diabetes complications in the context of diabulimia, which represented a chronic illness type burden to the participants. Some reported how they continued their daily life whilst experiencing clinical symptoms of acute Diabetes complications, suggesting they had gloomily accepted DKA and severe hypoglycaemia as part of their life and had adjusted to it.

*[…] Was walking to and from work every day in severe ketoacidosis but I kept at it.* (ID 111)

*Concentrating at school was extremely difficult. My eyes would blur due to the raised blood sugar levels, making simply reading the set work a challenge.* (ID 113)

Diabetes long-term complications impacting on the bloggers' everyday life was a recurrent theme of most blogs; the reports vary depending on the complications experienced by the individual. For example, some bloggers described the symptoms of severe neuropathy

causing pain and loss of autonomic function. The impact of Diabetes late complications on their quality of life was conveyed through more emotive comments, some of which were gloomy and cynical in nature.

*I have been told I need a permanent catheter [...] I will have to live the rest of my life with a piss bag strapped to my leg.* (ID 110)

*Thanks to the decade of damage I did to my body, I am now also the proud recipient of peripheral neuropathy, vasovagal syncope, and gastroparesis – which caused three ulcers and cyclical vomiting syndrome.* (ID 112a)

Fears of future Diabetes complications were drastically and explicitly described by the bloggers, including their own and future family's wellbeing. They described the threat their condition posed to their physical integrity and their life.

*Diabetes always complicates things. It's even possible the pregnancy could go horribly wrong. I could die; the baby could die; the baby could have serious developmental issues.* (ID 119b)

*I know that I wanted a family one day, with my limbs, eyes, heart, kidneys, and myself intact.*

(ID 117a)

Some bloggers described their fear of future complications from a personal and insightful perspective, acknowledging the self-harming component of insulin omission behaviour, ranging from not caring for one's health to the extreme of referring to insulin restriction as 'slow suicide'.

*I put my body in harm out of pure desperation to lose weight. I soon realized how damaging and harmful this was. I was doing no justice to myself.* (ID 117b)

**Strategies for recovery and triggers for relapse**

All blogs discussed recovery, and across all blogs this theme was characterized by a strong willingness to share one's individual experience of recovery in order to help others. These included describing rational strategies for recovery that involved emotional and Diabetes self-management strategies. Some blogs discussed new relationships appearing to be instrumental in recovery. Feelings of empowerment, optimism and hopefulness were often mentioned in the context of the recovery process.

*'m empowered that I can control this disease and not let it control me.* (ID 117b)

*I am happy to say that over the past few years, I have taken my chance at recovery, and I have run with it.* (ID 121)

Many of the recovery strategies described by bloggers were focused on experimenting with structured dietary approaches and prioritizing healthy lifestyle over weight loss. Some tried to develop strategies to help manage weight and to be able to reduce insulin doses for meals. Others were instead focused on a relaxed attitude towards body weight and prioritizing being healthy over being thin.

*These carbs provide me with the nutrients that I need. They also help sustain my blood sugar throughout the day.* (ID 117b)

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*There's no undereating. There's no starving myself or throwing up after a binge. This has nothing to do with my body image. It has to do with my health and how I feel about myself.* (ID 119c)

Improving Diabetes management and recovering by taking small steps at a time and being patient was described as a key strategy. Even seemingly small changes made a difference on the route to recovery.

*I'm just concentrating on keeping my blood sugar levels stable, eating right, and staying active. The results will come with time.* (ID 117b)

*You don't need to make the decision to change the rest of your life right now. Recovery is all about micro-decisions - an infinite list of small choices you make every day. ID 112b*

Resources and triggers for recovery included new relationships and new roles in life (e.g. a new romantic relationship or motherhood), but also the experience of life-threatening complications. Surviving a severe DKA episode was described as a wake-up call for recovery by one blogger.

*I'd like to take a moment to thank my boyfriend. I thank you dearly, for looking after me when I could not look after myself [...]* (ID 113)

*Becoming a mother was what helped me see my life in a different perspective. That there is a reson that I'm here.* (ID 117a)

*[...]I had to share a room with 3 other women once I was released into a regular ward. They all died, and I realized that by some miracle I had been given a second chance.* (ID 112a)

Bloggers highlighted the importance of surrounding themselves with a support system in recovery, which included healthcare professionals as well as friends and family.

*The first and most important step is to set up a treatment team.* (ID 112b)

> *A good tactic to evade this trap of secrecy is to choose at least one friend or family member*
>
> *that you trust - someone that you make a pact with - no lies.* (ID 112b)

Bloggers' intentions to help others have been an important part of recovery. Previous peer support motivated some bloggers to continue helping others in order to reciprocate the help they received.

> *They are women who, not only help encourage me to live my best life in harmony with my*
>
> *Diabetes, but also provide me with valuable wisdom, guidance, and most importantly, a*
>
> *supportive shoulder to lean on when needed.* (ID 121)

> *To help others, to educate, to inspire, to empower, and show compassion.* (ID 117a)

Triggers for relapse, such as peer pressure for thinness and stressful life events wre commonly described.

> *Even friends who don't mean to trigger are suddenly opting for skinny lattes. Comments*
>
> *overheard all around 'oh I have to get back on the treadmill!'.* (ID 114b)

> *When I moved to a new city a year after my diagnosis, the troubling signs returned.* (ID 119a)

## Discussion

This structured qualitative review of internet blogs written by people with Type 1 Diabetes and an Eating Disorder, or diabulima, used thematic analysis of blog content to obtain insight into the experiences, thoughts and feelings of this group of people.

Although wider search terms describing various subsets of Eating Disorders were used, the majority of blog authors used the term 'diabulimia' to refer to their condition, which confirms the term has face validity in the patient community [17]. The 11 diabulimia blogs were all authored by women in their 20s and 30s, which parallels the observations of female gender bias in Eating Disorders [18].

Research suggests boys/men are less likely to come forward because they face greater stigma and as a result of traditional male gender roles [19]. Typically, the age of onset of an Eating Disorder is mid to late adolescence, either soon after the onset of Type 1 Diabetes or onset of puberty [5,20,21]. One could speculate that these adult bloggers are further along in their recovery and therefore able to talk (blog) about their strategies, resources and triggers.

The subsequent thematic analysis identified three main themes: bloggers' relationship with insulin; experience of medical complications; and strategies for recovery and triggers for relapse. Interestingly, the 'relationship with insulin' theme gave deep insight into a multifaceted problem with a wide variety of factors that triggered and maintained the deliberate manipulation of insulin, which is the key behaviour driving diabulimia.

The pattern of fear of weight gain leading to insulin omission, as well as insulin overdose after binge-eating episodes are patterns which have been previously described [22], but the deeper- seated motives behind the insulin manipulation that bloggers described, such as the 'thrill', 'addictive', 'experimental' or 'secretive' aspects of insulin omission were novel observations. Preliminary evidence has found highly processed foods share pharmacokinetic properties with addictive drugs [23].

Additionally, animal models of bulimia suggest bingeing on food releases dopamine [24], similarly to human addiction processes. The acknowledgement of insulin omission as self-harming behaviour expressed in the blogs highlights the inherent ambivalence of these behaviours.

It is not surprising that the blogs discussed hospitalization after DKA, as insulin omission attributable to diabulimia increases DKA risk significantly [25]. Although DKA and severe hypoglycaemia are acute Diabetes complications, we observed that bloggers with diabulimia experienced repeated and cyclical patterns of these complications. Their description of how they adjusted to living with recurrent DKA and severe hypoglycaemia contradicts the

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medical categorization of these as acute complications. In the context of Type 1 Diabetes with an Eating Disorder, recurrent Diabetes acute complications are being experienced as a chronic illness burden rather than as an acute event, which is a novel observation in this group. Interestingly, the language in which the bloggers reported their acute and late Diabetes complications was medicalized. This may be a result of living with Diabetes for a long time, which results in many interactions with healthcare rofessionals so that people with Diabetes adopt their use of language when talking about their condition [26].

Previous research suggests that people with Diabetes and an Eating Disorder may have unhelpful beliefs about insulin and lack coping strategies to manage recovery [21,27,28]. By contrast, we found that some of these bloggers were very insightful about the consequences of insulin omission and the self-harming nature of the Eating Disorder behaviour. Their accounts of the burden of having severe Diabetes late complications (including the fear of experiencing late complications in the future) indicate that most of the bloggers were fully aware of the consequences of their insulin restriction.

This observation is confirmed by the self-reflection of some bloggers who, from a personal and insightful perspective, referred to their behaviour as self-harming or even 'slow suicide'. Some were even able to share their strategies, resources and triggers for recovery, which often included helping others.

We found a mixture of healthy and unhealthy attitudes towards food in their approaches to recovery. Some bloggers recognized the need for carbohydrates, whereas others decided to use restrictive diets. The concept of approaching the recovery process in small steps was a common subtheme with various aspects of recovery, including insulin injections, accepting body shape and keeping blood glucose levels stable. Some blogs discussed peer pressure and social relationships, with new relationships appearing to be instrumental in recovery.

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The present study has some limitations. Blogs are written for many reasons [29] and are not free from self-presentational bias, although there is also evidence that blogs can be considered to provide trustworthy [30] and rich data [13]. It is difficult to collect participant demographic data for blogs as they allow complete anonymity. Another limitation of blogs is that they do not capture the views of all individuals with Type 1 Diabetes and an Eating Disorder (i.e. the majority who do not blog). As with all qualitative studies, researchers have an influence on the interpretation of their findings [13]. We hoped to reduce this bias by taking a multidisciplinary approach.

Qualitative analyses of blogs authored by people with Type 1 Diabetes and an Eating Disorder, or diabulimia, have identified high levels of Diabetes distress and provided insight into different motives for insulin omission and strategies for recovery. Considering the limited evidence for effective interventions, these findings may help the development of complex interventions to improve biomedical and psychological outcomes in this group of people.

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**Competing interests**

None declared.

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FIGURE 1 Flowchart illustrating how blogs were systematically reviewed.


FIGURE 2 Thematic map illustrating themes and subthemes.

Figure 1. Flowchart illustrating how blogs were systematically reviewed





Figure 2
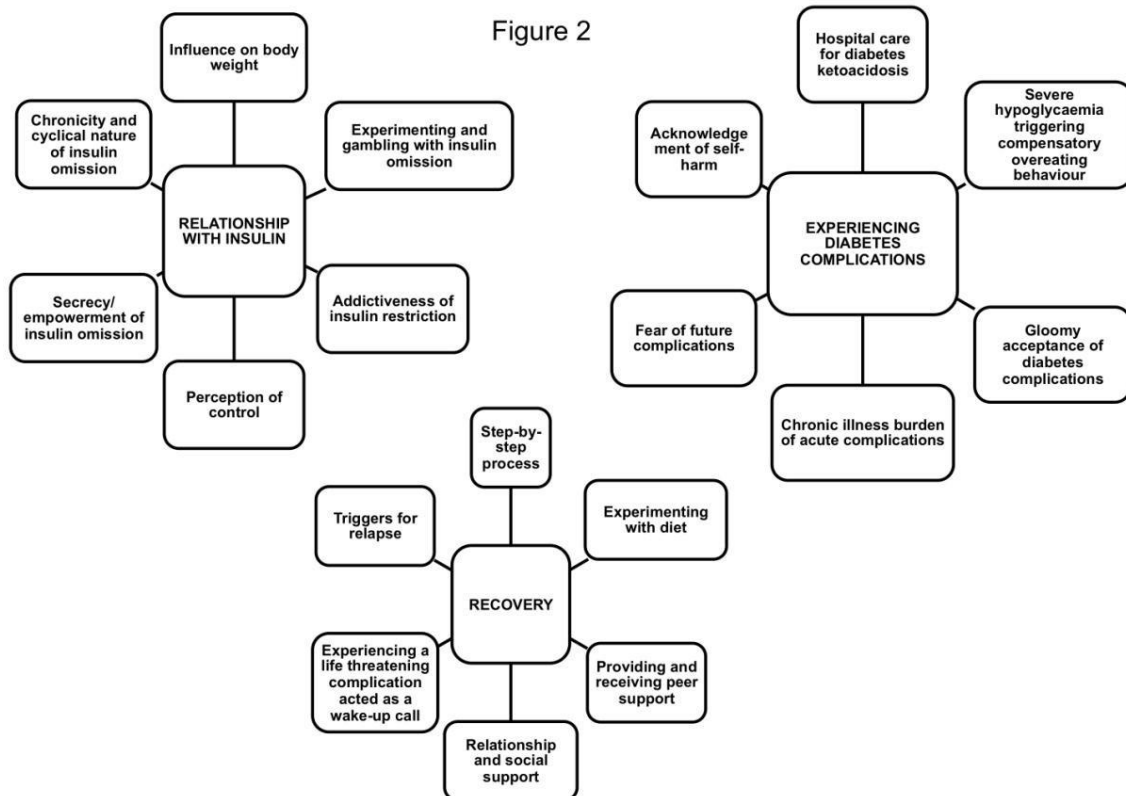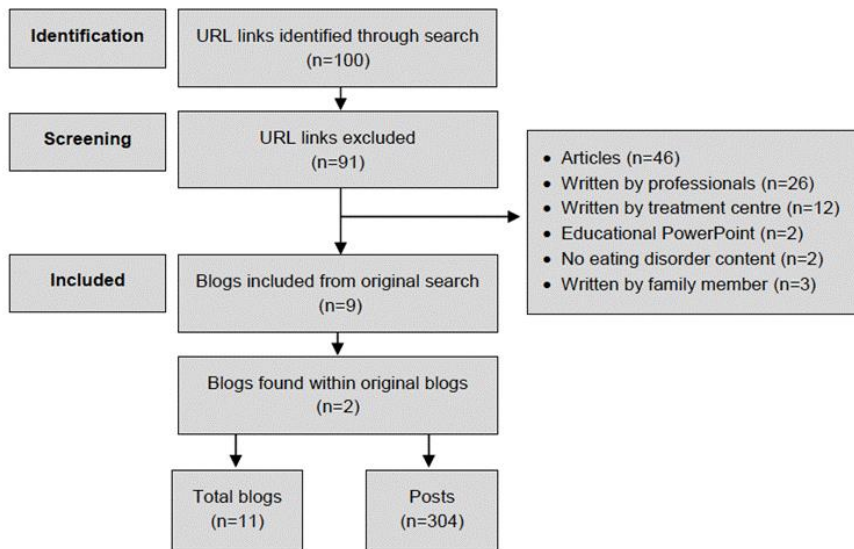
# Understanding poor outcomes in women with type 1 diabetes and eating disorders

*Jacqueline Anne Allan*

Although it has been debated for many years, there is now a general consensus that there is an increased incidence of eating disorders in people with type 1 diabetes. With the addition of insulin omission as a clinical symptom in both anorexia nervosa and bulimia nervosa in the DSM-V (*Diagnostic and Statistical Manual of Mental Disorders, fifth edition*), incidence rates may increase even further. People with eating disorders and diabetes develop debilitating complications at a younger age, show a higher rate of disengagement with healthcare teams, are harder to treat and have a significantly higher mortality rate. Little is known, however, about why eating disorders are more common in this demographic or why people with eating disorders are much more difficult to treat. Using an online questionnaire, 98 people with type 1 diabetes and an eating disorder were surveyed in order to determine if they have comorbid psychiatric diagnoses and which terminology they use to describe their eating disorder. This article describes the findings of this survey and discusses the importance of having correct diagnostic terms for eating disorders in people with diabetes.

**Article points**

1. The comorbidity of eating disorders with type 1 diabetes represents a notoriously difficult combination to treat effectively, which may be further complicated by the presence of other psychiatric diagnoses.

2. This article describes a study that was carried out to ascertain the psychiatric characteristics of those with type 1 diabetes who are recovering from an eating disorder and to investigate their attributions in regard to diagnosis.

3. This small study shows that those with type 1 diabetes and an eating disorder (or those who deliberately omit insulin for weight control) are likely to have multiple psychiatric morbidities that can further complicate treatment.

**Key words**

- Diabulimia
- Eating disorders
- Insulin omission

**Authors**

Jacqueline Anne Allan is studying for her PhD at Birkbeck, University of London and is Founder and Managing Director of Diabetics with Eating Disorders.

The onset of physical illness is a well-known risk factor for the development of mental conditions. There are reportedly higher rates of depression (Anderson et al, 2001), anxiety (Grigsby et al, 2002) and emotional instability (Rassart et al, 2014) among people with type 1 diabetes. Eating disorders are also associated with high rates of comorbid mental pathology and substance abuse (Striegel-Moore and Bulik, 2007).

The comorbidity of eating disorders with type 1 diabetes represents a notoriously difficult combination to treat effectively, which may be further complicated by the presence of other psychiatric diagnoses. It has been reported

that this demographic does not respond well to standard treatment for eating disorders, and even when there appears to be an improvement in psychological well-being, this does not relate to an improvement in glucose management (Olmsted et al, 2002). Any treatment model that does not allow for comorbidities, as well as diabetes-specific factors, may be ineffective.

An additional barrier to effective treatment may be the way these individuals categorise themselves. The colloquial nomenclature for using insulin omission for weight control is "diabulimia", although this is not an academic or clinical term. Regardless, it has been adopted by the diabetes community and it is how they refer to this practice. The issue of

different terminology may partially account for treatment difficulties within this demographic. People have reported approaching healthcare professionals with this self-diagnosis only to be told that diabulimia "doesn't exist" or was "made up on the Internet" (www.dwed.org.uk). The term may be problematic but the action of insulin omission for weight loss purposes is very common, with researchers reporting an incidence of up to 40% of 15–30-year-old females with type 1 diabetes using this mechanism to facilitate weight loss (Fairburn et al, 1991). Regardless of the diagnostic term, or lack thereof, the practice is incredibly dangerous. Those who omit insulin have significantly higher rates of diabetes complications, such as retinopathy and nephropathy (Rydall et al, 1997) and, therefore, cost the NHS considerably more than people with diabetes but without eating disorders. Furthermore they have a much higher mortality rate (Goebel-Fabbri et al, 2008).

This article describes a study that was carried out to ascertain the psychiatric characteristics of those with type 1 diabetes who are recovering from an eating disorder and to investigate their attributions in regard to diagnosis.

## Method

Ethical approval was obtained from Birkbeck College (University of London). Participants were recruited for the study via the registered charity, Diabetics with Eating Disorders (DWED), with use of their social media pages. The study recruited 95 females and 3 respondents who did not wish to state their gender. All participants had been in recovery for at least 2 years, which is a recommendation of the eating disorder charity, B-eat, for inclusion in research.

Participants were asked to follow a link to the questionnaire, read a study brief and provide their consent to the briefing. They were then advised to allow 10–20 minutes in a quiet environment to fully complete an online questionnaire. This questionnaire was developed using an online survey tool (www.questionpro.com) and consisted of 17 items, which were designed specifically for this survey.

For each question, the participants were able to select a "prefer not to say" option. Participants were asked to respond to the following open-ended questions:

- How old are you?
- What gender are you?
- At what age were you diagnosed with type 1 diabetes?
- What nationality are you?
- Do any other family members have type 1 diabetes?
- If so, what is their relationship to you?
- Have you ever received an official eating disorder diagnosis?
- If so, what was the diagnosis?
- Have you ever received another mental health diagnosis?
- If so, what was the diagnosis?
- How long after your diabetes diagnosis did you develop an eating disorder?
- Did you manipulate or omit insulin?
- Do you think that you had any of the following?:
  - Anorexia.
  - Bulimia.
  - Diabulimia.
  - A combination of bulimia and diabulimia.
  - A combination of anorexia and diabulimia.
  - A combination of anorexia and bulimia.
  - A combination of all three conditions.
  - Eating disorder not otherwise specified (EDNOS).
- In the time that you had your eating disorder, did you seek professional help?
- If so, which of the following did you approach?:
  - GP.
  - DSN.
  - Diabetic consultant.
  - Dietitian.
  - Eating disorder specialist.
  - Other health professional (please state).
- Have you experienced bullying because of your weight?
- Have you experienced bullying because of your diabetes?

## Results
### Demographics

In total, 98 participants responded to the advert and completed the questionnaire. As

mentioned, three of the respondents did not wish to state their gender and 95 were female. Respondent age ranged from 18 to 65 years, with a mean age of 28.49 (standard deviation [SD]=9.66).

### Age of onset

The age at which respondents were diagnosed with type 1 diabetes ranged from 0–45 years old, with a mean age of 11.9 (SD=8.98). The age at which they felt they were developing an eating disorder ranged from 7–45 years, with a mean age of 16.96 (SD=7.06). The average time between diagnosis of type 1 diabetes and onset of an eating disorder was 4.76 years (SD=4.7), but ranged from 0–19 years.

### Insulin manipulation, diabulimia and eating disorder diagnosis

Ninety-four of the 98 respondents (95.9%) had omitted or manipulated insulin during their eating disorder. Four believed they had anorexia (4.1%); 27 believed they had diabulimia (27.5%); 19 believed they had a combination of bulimia and diabulimia (19.4%); 18 believed they had a combination of anorexia and diabulimia (18.4%); and 28 believed that they had a combination of all three conditions (28.6%). None reported a diagnosis of bulimia or "eating disorder not otherwise specified" (EDNOS). Two participants did not respond (2.04%). Data are shown in *Figure 1*.

Despite all of the respondents feeling that they did have an eating disorder, 38 participants had never been diagnosed with one (38.8%). Of those who had been diagnosed, 22 had been given a diagnosis of bulimia nervosa (22.4%), nine had been given an diagnosis of anorexia nervosa (9.2%), 13 had received an EDNOS diagnosis (13.3%), 10 had been given dual anorexia and bulimia nervosa diagnoses (10.2%), four had dual diagnoses of bulimia and EDNOS (4.1%) and two had been given a triple diagnosis of bulimia, anorexia and EDNOS (2%).

### Other diagnoses

A total of 76 (77.6%) of the participants had been diagnosed with a mental health illness.
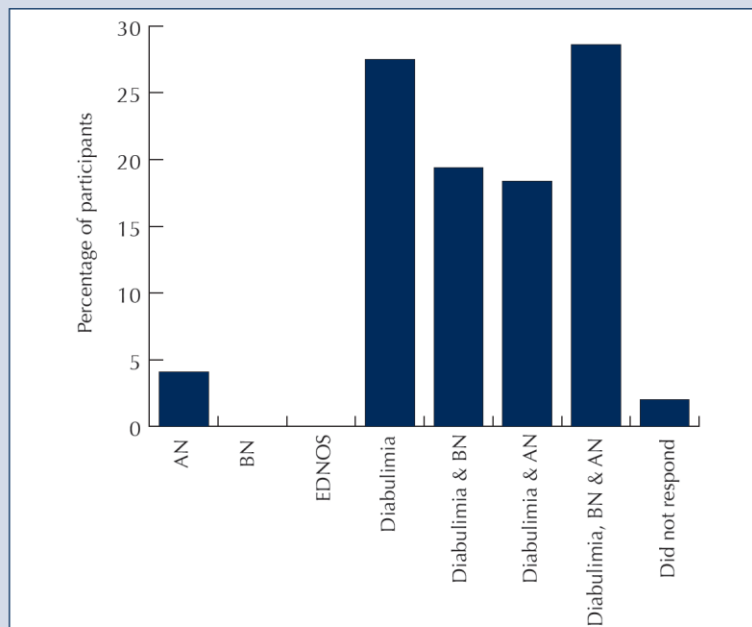


Figure 1. Participant reported beliefs about eating disorder (AN=anorexia nervosa; BN=bulimia nervosa; EDNOS=eating disorder not otherwise specified).
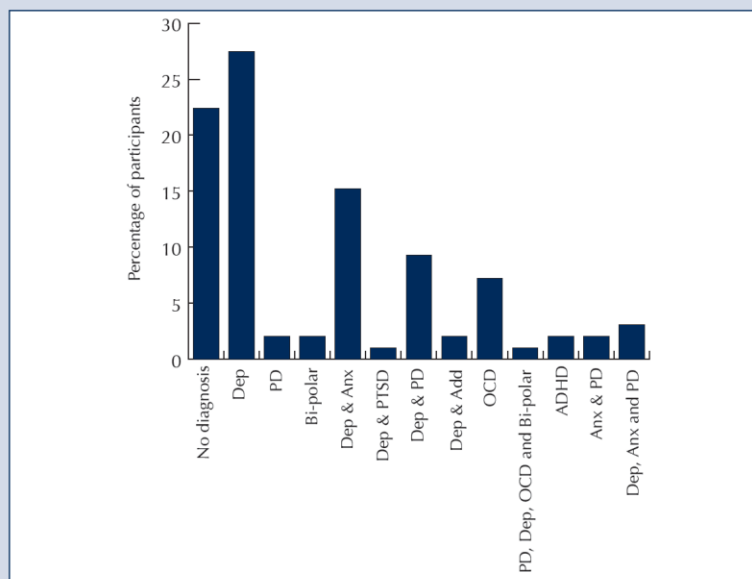


Figure 2. Percentage of participants diagnosed with mental health disorder (Dep=depression; PD=personality disorder; Anx=anxiety; PTSD=post-traumatic stress disorder; Add=addiction; OCD=obsessive compulsive disorder; ADHD=attention deficit and hyperactivity disorder).

Of these, 58 (59.18%) had been diagnosed with depression; 20 (20.34%) with anxiety; 19 (19.5%) with personality disorders; three (3.06%) with bipolar disorder; one (1.02%) with

post-traumatic stress disorder; two (2.04%) with an addiction; eight (8.30%) with obsessive compulsive disorder and two (2.04%) with attention deficit hyperactivity disorder.

Further analysis showed that 27 were diagnosed with depression only (27.5%), two had personality disorder only (2.04%) and two had bipolar disorder only (2.04%). Fifteen participants had both depression and anxiety (15.2%); one had depression and post-traumatic stress disorder (1.02%); nine had depression and personality disorder (9.3%); two had depression and addiction (2.04%); seven had obsessive compulsive disorder alone (7.2%) and one had personality disorder, depression, obsessive compulsive disorder and bipolar disorder (1.02%). Two people had attention deficit hyperactivity disorder (2.04%), two had anxiety and personality disorder (2.04%) and three had depression, anxiety and personality disorder (3.1%). See *Figure 2* (previous page).

## Discussion

As found in the non-diabetic population of those with eating disorders (Hudson et al, 2007), this study showed there were comorbidities with most of the core DSM-V (*Diagnostic and Statistical Manual of Mental Disorders, fifth edition*) mood, anxiety and personality disorders. The results showed that the majority (77.6%) were formally diagnosed with another psychiatric condition and the majority of the sample had been diagnosed with multiple disorders. This may make treatment for this demographic even more difficult. It has long been understood that emotional issues mediate blood glucose control (Simonds et al, 1981). For the person with an eating disorder and borderline personality disorder, for example, insulin misuse not only facilitates weight loss but is also a self-destructive behaviour. Combine that with the tendency to form unstable relationships and clinically these individuals represent a very challenging treatment prospect that diabetes teams may not be skilled in (Leichter and Dreelin, 2005). Furthermore, there is the issue of practitioner engagement; clinicians find those with type 1 diabetes and an eating disorder harder to manage and feel

that they lack training in the area (Tierney et al, 2009). Individuals with many comorbidities present an even more significant challenge.

A very interesting finding of this small study is that, when asked to indicate which eating disorder they had, all but four of the participants replied "diabulimia". What may be even more important is that a number of participants thought they had a combination of eating disorders including diabulimia, showing that there is a distinction between the act of insulin omission and that of dietary restriction or classical binge/purging. None of the participants felt that they had EDNOS or bulimia, which are terms that have been used to describe the act of insulin omission frequently in academic research (Nielsen 2002; Takii 2002; Young-Hyman and Davis, 2010). There is an obvious disconnect between what an individual feels they have and what they are actually being diagnosed with, which may further explain why these individuals are resistant to standard eating disorder treatment.

It is also notable that all participants who identified their gender were female. It may be that the other three participants were, in fact, male and that this is further illustrative of the increased stigma around men with eating disorders. Alternatively it could be because recruitment was largely from online social networks that are predominantly female occupied. Further research focusing on males should be a priority.

Binge-eating disorder has its own classification in the DSM-V and it could be argued that, on this basis, diabulimia, or another formal diagnostic term relating to the deliberate manipulation of insulin for weight control purposes, may also warrant its own diagnostic criteria.

Diabulimia may have diabetes-specific roots separating it etiologically from anorexia and bulimia, and this may explain some of the problems in treating it within those paradigms. To judge severity using weight or episodic frequency criterion rather than $HbA_{1c}$ is problematic (Allan and Nash, 2014), further justifying the need for a distinct classification. To treat insulin omission in the same way as

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other clinical features is potentially dangerous.

## Conclusion

Those with type 1 diabetes and an eating disorder (or those who deliberately omit insulin for weight control) are likely to have multiple psychiatric morbidities that can further complicate treatment. These individuals also attribute their illness, or at least the practice of insulin omission, to "diabulimia". These aspects must be taken into consideration when both diagnosing and treating eating disorders in people with type 1 diabetes.

NICE clinical guidelines state that healthcare professionals dealing with those with type 1 diabetes should maintain a high index of suspicion for eating disorders. Furthermore, they recommend immediate referral to psychological services. In practice, there seems to be an issue as to where to refer these people. Although resources exist via charities such as DWED, there appears to be no consensus on how to treat these individuals, which is made even more complicated by the high rates of multiple comorbidities.

The charity DWED recommends that DSNs become part of a multidisciplinary team and liaise with psychological services frequently. It may be that their patients are attending programmes that do not understand the mechanisms of insulin omission. Good communication between everyone involved may lessen the risk of these people being treated inappropriately.

Healthcare professionals working with people with diabetes and eating disorders can direct patients to the DWED website (www.dwed.org. uk). Both healthcare professionals and people with eating disorders may benefit from reading the book, *Skills-based Learning for Caring for a Loved One with an Eating Disorder* (Treasure et al, 2007). ■

*"NICE clinical guidelines state that healthcare professionals dealing with those with type 1 diabetes should maintain a high index of suspicion for eating disorders."*

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**Appendix F: Pilot Project**

Type 1 Diabetes and Eating Disorders: An Exploratory Study

Jacqueline Anne Allan

12408688

Project Report Submitted as

Partial fulfilment for the

Degree of Bachelor of Science,

Birkbeck, University of London

2013

ABSTRACT

1) INTRODUCTION

2) METHOD

3) RESULTS

4) DISCUSSION

5) CONCLUSION

6) REFERENCES

7) APPENDIX

*Abstract*

Current academic research demonstrates that women suffering with Type 1 Diabetes are at twice the risk of developing a clinically significant Eating Disorder. Further study has also shown that up to 40% of women aged 15 – 30 with Type 1 Diabetes regularly omit insulin in order to induce hyperglycaemia and rapidly lose weight. This practice is popularly known as Diabulimia and the chronic omission of insulin for weight control will appear in the upcoming DSM-V. Eating Disorders with Type 1 Diabetes and Insulin Omission are extremely dangerous and the health implications for this demographic are much increased. Despite this, the majority of the literature has focused on prevalence rather than the factors influencing its development or the biopsychosocial aspects of the lives of those suffering. The current study aims to use an internet-based survey informed by an exhaustive literature review, together with demographic information and factor analysis, to explore which aspects the sufferers themselves feel are the most important in the development of their Eating Disorder and to explore their biopsychosocial environment.

*Introduction*

*What is Type 1 Diabetes?*

Type 1 Diabetes occurs when the immune system mistakenly attacks the islet of Langerhans cells in the pancreas. These cells are endocrine, responsible for producing the hormone insulin which regulates the amount of glucose in the blood. Insulin processes carbohydrates and transports the resulting glycogen to where it is needed for energy around the body. There is no unifying theory as to why this immune response occurs but as Type 1 Diabetes is increasing in prevalence in the Western world (Gale2002) current popular theories include: overactive virus response, genetic mutation, and environmental factors (Peng & Hagopian 2006). Following the destruction of these cells the body is incapable of producing insulin, meaning that it must be injected synthetically. There is currently no cure for Type 1 Diabetes which is a lifelong chronic condition that needs micromanagement to ensure good health. In order to mimic the body's natural processes there are two main ways of controlling Diabetes. The first is to use long and short acting insulin. The long acting insulin is typically injected once a day to synthesise a natural baseline level of insulin in the blood; these insulins tend to have a lifespan of 18 – 24 hours. The short acting form is injected when eating, to synthesise the body's natural reaction to incoming energy, and these are generally active for 3 – 5 hours. The second way of controlling Type 1 Diabetes is to use an insulin pump, which is a small machine attached to the body that drips in a constant stream of rapid acting insulin, using what is called a basal/ bolus regime. The basal units replicate background insulin at a rate per hour and the bolus units are used in response to food.

In the UK it is estimated that approximately 300,000 people have Type 1 Diabetes (Diabetes UK 2012). Type 1 Diabetes develops rapidly, normally over a period of weeks, and onset commonly occurs in adolescence with the peak age of diagnosis between 10 -14 (Diabetes UK 2012) It is a highly individual illness and patients

must ascertain how much background insulin they need each day and how much insulin they need to cover the carbohydrate content of their food. The actual amount is dependent on many factors such as: duration of Diabetes, sex, weight, glycaemic load of food, activity level, menstrual cycle and age. External factors like stress, temperature and season can also affect the blood sugar. It can be extremely challenging to control blood sugar, but it is of vital importance that it is kept between 4 – 8 millimoles (mmol/L) per litre. There are serious consequences of blood sugar being out of range. If an error occurs and there is too much insulin in the blood, then the Type 1 diabetic[17] will experience an episode of hypoglycaemia, whereby the lack of energy leads to extreme disorientation, a burst of adrenaline, fight or flight responses, spasms, fits and, if left untreated, coma and death. If there is too little insulin in the blood, then blood sugar rises causing hyperglycaemia, and in the short term this causes extreme fatigue, thirst, breathing difficulties and glycosuria. If the hyperglycaemia is not treated, Diabetic Ketoacidosis (DKA) will develop. DKA is a state whereby in the absence of insulin the body is not getting enough energy from food and as a result resorts to burning protein for energy from fat, muscle and organ tissue and excreting it through the urine. This process induces massive weight loss and creates by-products known as Ketones. Ketones are highly acidic and extremely toxic to the body. There is no treatment other than insulin for DKA and if left untreated it is always fatal. Many complications develop from prolonged hyperglycaemia including Neuropathy (damage to the nerves particularly in the extremities, commonly resulting in amputations), Gastroparesis (damage to the nerves in the stomach), Retinopathy (damage to the eyes, with Diabetes currently being the leading cause of blindness)[18] and Nephropathy (damage to the kidneys).

Despite the threat of these complications, it has been shown that many Type 1 Diabetics, particularly females between the age of 15 – 30, regularly omit or manipulate their insulin to induce hyperglycaemia and DKA in order to control their weight. As mentioned, this practice is commonly known as Diabulimia. Type 1 males have also been shown to have a higher drive for thinness that their non- diabetic peers (Svensson, Engström & Aman 1992). Estimates on prevalence of insulin omission and manipulation vary widely (Rodin 1991, 2000, 2004/ Peveler & Fairburn 1991/ Striegel-Moore 1992/ Polonsky1994) but are as high as 40% among women between the ages of 15 – 30. Further research has shown that having Type 1 Diabetes increases two fold the

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[17] There is much debate on the appropriate term to describe someone who has been diagnosed with Type 1 Diabetes. Charities such as JDRF and Diabetes UK advocate using the term Person with Type 1 Diabetes to avoid stigma. As the current study focuses on Eating Disorders within Type 1 Diabetes the charity 'Diabetics with Eating Disorders' was approached to ascertain the correct label for their members. They have stated that over 400 Members voted to call themselves Diabetics; as such the current study will use that convention.

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chances of developing a clinically diagnosable Eating Disorder such as anorexia nervosa, bulimia nervosa or EDNOS (Eating Disorder not Otherwise Specified) (Jones, Lawson, Daneman , Olmsted & Rodin 2000)

*Type 1 Diabetes and Anorexia Nervosa*

Anorexia Nervosa is characterised by a pathological need to be excessively thin, obsessiveness around the consumption of calories and caloric expenditure, avoidance of certain food groups, moral judgements on food, fear of eating, body dysmorphia and often compulsive exercise. There are certain behaviours that are indicative of Anorexia such as keeping a diary of food and exercise and being extremely rigid around diet and exercise. As a Type 1 diabetic you are taught that all food must be monitored, all exercise must be monitored, routine is emphasised, many hospitals actively encourage the keeping of a food diary, and carbohydrate counting is routinely taught as part of treatment.  In Type 1 Diabetes then, Anorexic behaviours may be inadvertently encouraged (Rodin et al 2002; Meltzer, Johnson, Prine, Banks, Desrosiers & Silverstein 2001; Smith, Latchford, Hall, & Dickson 2008, Diabetes Australia 2008) which may account for the increased risk. There are Diabetes-specific aspects that may also be important. In order to avoid hypoglycaemic attacks, some Type 1 Diabetics cut down on their food intake to avoid having to inject (Ishmail & Treasure 2010; Rodin et al 2009; Goebel-Fabbri, Fikkan, Franko, Pearson, Anderson & Weinger 2008). Often numbers are accompanied with a moral judgement, i.e. good blood sugar number which can in turn lead to moral judgements on food which would be more indicative of an Eating Disorder (Goebel-Fabbri, Uplinger, Gerken & Mangham 2009; Tierney, Deaton, & Whitehead 2009, Diabetes Australia). Weight management also plays an important part and it is standard practice to weigh patients and discuss this in clinic. Type 1 Diabetes by its very nature, then, ensures that the patient is focused on weight, consumption and numbers and, as it is typically occurring in adolescence, from a young age. This may indeed explain the increased risk for Anorexia.

*Type 1 Diabetes and Bulimia Nervosa*

This moralising of food, creating 'forbidden' items and the constant need for control may also explain the increased incidence of bulimia among Type 1 Diabetics. Bulimia Nervosa is characterised by eating excessive amounts of food (bingeing) and then using inappropriate compensatory behaviours to rid the body of this consumption, by self-induced vomiting (purging), using laxatives and over exercising. Bulimics report a loss of control during a binge and a sense of release at the purge. In Type 1 Diabetes, when hypoglycaemic, the patient *is* out of control due to the lack of energy to the brain, and this lack of control combined with the bodily response of craving sugar to redress the balance can lead to serious binges. These binges in turn can lead to rocketing blood sugar which needs massive amounts of insulin to control. This can lead the patient to a vicious circle. Guilt at the overconsumption is also common and can foster these inappropriate compensatory behaviours (Criego, Crow & Goebel-Fabbri 2009; Rodin et al 2002). These are aspects that could help explain the increased risk for bulimia in those with Type 1 Diabetes.

*Type 1 Diabetes and Insulin Omission*

According to the office of national statistics, last year in England and Wales there were 8000 accident and emergency admissions for DKA[19], and it is reasonable to assume that many of these were due to the onset of Type 1 Diabetes. One of the major symptoms of undiagnosed Type 1 is extreme and rapid weight loss. Thus at diagnosis, most Type 1 Diabetics will have experienced weight reduction. Unfortunately, this can give rise to praise from peers or the family and thus create a conflict within the Diabetic as they are being complimented for their changing shape but this is due to a life threatening health condition (Ishmail &Treasure 2010; Rodin et al 2002; Crow, Keel & Kendall 1998). On commencement of insulin therapy, this weight may return which may cause further disturbance at what is recognised to be a sensitive age, particularly for females (Rodin et al 2002, 2009; Jack 2003). This initial weight loss means that most Type 1 Diabetics know that lack of insulin leads to extreme weight loss immediately from diagnosis.

There are other aspects of Type 1 Diabetes and Diabetes care that may explain the increased risk factor for the development of an Eating Disorder. Denial and Resentment of Diabetes can lead to the patient not looking after themselves appropriately as they can fear testing and injecting. They may fall into the hyperglycaemic process which induces weight loss. 'Burnout', whereby the constant micromanagement of the condition leads the patient to lapse in self-care (Ishmail & Treasure 2010, Teirney et al, 2009; Rodin 2002) may also be contributory as this can result in the same weight loss. Although this weight reduction is at times an unintentional by-product and symptom of another psychological problem, it can none the less become problematic and an initiating factor of the onset of an Eating Disorder.

Healthcare providers can also unwittingly encourage disturbed attitudes in regard to Diabetes. The constant warnings of complications such as blindness and amputation may desensitise patients to the seriousness of high blood sugar or they may feel that regardless of how 'good' they are, they are somewhat doomed to illness anyway (Goebel Fabbri, Teirney 2009). Also the message may be construed as something that will happen at some distant point in the future or to 'someone' else. Diabetes Clinic can be a source of frustration for many Type 1 Diabetics, health professionals may come across as judgemental and patronising, and this can lead to disengagement with them and further resentment of their condition.

*Type 1 Diabetes, Societal Pressure & Family Environment*

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[19] National Diabetes Audit 2012

Type 1 Diabetes is typically diagnosed in adolescence which may cause many problems for the psychological development of the child. At a time where typically one is trying to become more autonomous and assert independence, the onset of a life-threating illness can lead to increased parental involvement and this can increase family conflict (Rodin 2002). The patient may disagree with parental management of the Diabetes or feel that just as they were gaining control over their life, this is then taken away by the illness (Rodin, Diabetes Australia). Adolescence is a time of increased sensitivity regarding body image and self-esteem, and the development of Type 1 Diabetes may make them feel different, they may be embarrassed about testing or injecting in front of their peers and may fear peer rejection. Recent research from the Juvenile Diabetes Research Foundation has shown that children with Type 1 Diabetes are more likely to be bullied than their non-Diabetic peers[20]. It has been suggested that the readily available mechanism for weight loss is alone to blame for the increased risk factors as teenagers are desperate to fit in (Ishmail & Treasure 2010).

Type 2 Diabetes has seen a massive increase in media coverage. It is typically caused by diet and lifestyle, with obesity and inactivity being the main risk factors, and is around 10 times more common that Type 1 Diabetes, with 3.5 million estimated sufferers in the UK (Diabetes UK). It is typically diagnosed in adults but societies' increasingly poor diet and the 'obesity crisis' has seen it becoming more prevalent in younger people (Diabetes UK). The increased media coverage and the common failure of the media to distinguish between the two types of Diabetes have led to increased ignorance around Type 1. Many people do not know that there are two Types and as such many Type 1s are misunderstood, having to field ignorant questions from people who believe that they 'ate too much sugar as a child', that they have 'done this to themselves' or that their disease is 'the same thing that my gran died of'. This can then lead to a weight complex and the need to separate oneself from the stereotype of the 'fat lazy' Diabetic[21].

*Other Eating Disorder Risk Factors:*

Outwith Type1 Diabetes and those previously noted, there are many risk factors for development of an Eating Disorder. Family factors seem to be important, with family dysfunction, family conflict, family rigidity, maternal concerns with weight and shape, maternal mental health problems, lack of autonomy within the family system and family history of Eating Disorders all being significant. Personality factors such as perfectionistic tendencies, low self-esteem and distorted body image are also important, as are external factors

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[20] www.jdrf.org.uk

[21] www.dwed.org.uk

such as academic pressure, involvement in sport, peer group conflict and being bullied (Fairburn 2001,2008; Schmidt 2001)

*The Current Study*

The current study aims to explore which of the preceding aspects accounts for the increased risk factor among those with Type 1 Diabetes for developing an Eating Disorder. While most of the literature to date has focussed on the prevalence of Eating Disorders or the medical implications of them, very little has been done to ascertain the opinions of the sufferers themselves or their environment. This study uses a questionnaire informed by an extensive literature review and designed to analyse which of the suggested aspects are most important. Current sufferers report that there are many barriers to effective treatment. Eating Disorders consultants tend to be woefully inadequate at dealing with Diabetes, often diagnosing anorexia in those who are underweight and treating them as such. There are many problems with this approach, as a Type 1 Diabetic food must be tightly controlled and treatment for anorexia often focusses on relaxing around food, so not only are patients treated for an illness that they don't have, but treatment may be in direct contradiction to good Diabetes practice. There have also been cases of patients being diagnosed with bulimia as they are sick after eating but this vomiting is due to gastroparesis (whereby the nerves in the stomach are so damaged by high blood sugar that they are no longer able to push food into the intestine, resulting in food being regurgitated). They are therefore deemed non-compliant in Eating Disorder treatment which can in turn lead to total disengagement. Often treatment is focused solely on food and completely ignores any issues related to the psychological implications of injecting or insulin. Moreover, if these are not addressed, then treatment is likely to fail. Diabetes specialists often have no training in Eating Disorders and this can lead to a misunderstanding of the patient who is often chastised for having high blood sugar, is repeatedly weighed at appointments and increasingly frustrated with the lack of empathy. This in turn can lead to further disengagement. [22] The ignorance of health professionals can be potentially life threatening to the patient. If it transpires that 'classic' Eating Disorder factors account for the increased risk, then serious questions about the Diabetic environment must be asked to inform future policy. If the results of the current study show that Diabetes related factors account for the risk, then that also has serious implications for the treatment of those suffering with Eating Disorders. Demographic information has also been collated to further understand the issues surrounding treatment and the biopsychosocial environment of the patient. The results of this study may then have important ramifications for all of those involved in treating Type 1 Diabetics who also have Eating Disorders.

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[22] www.dwed.org.uk/resources

*Method*

*Participants*: Participants were recruited via the Registered Charity, Diabetics with Eating Disorders', Social Media Pages. The Social Media Sites used were Twitter, Facebook, Tumblr, TuDiabetes, YouTube and Google +. Participants were 95 females and 3 respondents who did not wish to state their gender (ages of 18 – 65) with more than 2 years recovery as recommended by the National Eating Disorder Association (b-eat). [23]

*Procedure*: Participants were asked to follow a link to the questionnaire and read and consent to the briefing. They were then instructed that they should have 10 – 20 minutes in a quiet environment available to participate. Participation in the research involved completing an online questionnaire in two parts. Part 1 asked questions about the participant (but not anything identifying). Part 2 asked 50 questions to be rated on a likert scale. They were asked to rate the importance of factors in the development of Eating Disorders in Type 1 Diabetics. Participants were reminded at the top of the online questionnaire that should they feel uncomfortable answering any of the questions they could select the 'prefer not to say option' or could exit the questionnaire completely by closing the relevant tab on their browser.

*Materials*: Online questionnaire by http://www.questionpro.com/

http://Jacqs.questionpro.com

*Questionnaire & Literature Review*: The questionnaire created was informed by an extensive literature review collected by the researcher over 6 years using Google Scholar. The terms providing notification were [Insulin non compliance "Type 1 Diabetes"], [Eating Disorder intitle: "Type 1 Diabetes"] [intitle: Diabulimia] [intitle: ED-DMT1] [Psychological intitle "Type 1 Diabetes"]. One hundred and fifty academic papers were reviewed and suggested reasons for the development of Eating Disorders among Type 1 Diabetics extracted and put into a questionnaire. There were a total of 40 items on the questionnaire to be answered using a 5 point likert scale. Questions were randomised using a random number generator and, to avoid response bias, randomly selected questions were reverse scored i.e.

39) My family was very rigid and this contributed to the development of my Eating Disorder

40) A disturbed attitude to food <u>did not</u> contribute to the development of my Eating Disorder

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[23] For Advert please see Appendix

*Design:*   A Principal Component Factor Analysis was run using SPSS ver 20.

## Results

*Demographics*:

98 participants responded to the advert and completed the questionnaire.  Of these 98, 66 were from the UK, 3 were from Australia, 13 were from the United States, 6 were from Eire, 2 were from Iceland, 4 were from Canada, 1 was from Italy and 1 was from Brazil.

Of the respondents 3 did not wish to state their gender and 95 were female. Respondent age ranged from 18 – 65 with a mean age of 28.49 (SD = 9.66). The age at which respondents were diagnosed with Type 1 Diabetes ranged from 0 – 45 years old with a mean age  of 11.9 (SD = 8.98), and the age at which they felt they were developing an Eating Disorder ranged from 7 – 45 years old with the mean age being 16.96 (SD = 7.06). The average time between diagnosis of Type 1 Diabetes and onset of an Eating Disorder was 4.76 years (SD = 4.7) but ranged from 0 - 19 years. However, it may be assumed that of those people who have said 0 years some were already suffering from an Eating Disorder prior to the diagnosis of Type 1 Diabetes.

Of the 98 participants 38 had another family member with Type 1 Diabetes, the most common of which was the brother (n = 14) followed by father (n = 13), grandparent (n= 11) cousin (n =8), sister (n = 6), aunt (n = 4), niece (n = 3), mother (n= 2), son (n = 1).

Ninety-four of the 98 respondents (95.9%) had omitted or manipulated insulin during their Eating Disorder. Four believed they had anorexia (4.1%), 27 believed they had Diabulimia (27.5%), 19 believed they had a combination of bulimia and Diabulimia (19.4%), 18 believed they had a combination of Anorexia and Diabulimia (18.4%), and 28 believed that they had a combination of all 3 conditions (28.6%). Two selected the prefer not to say option.

Demographic Chart 1: Do you think you had?

## Do you think that you had:
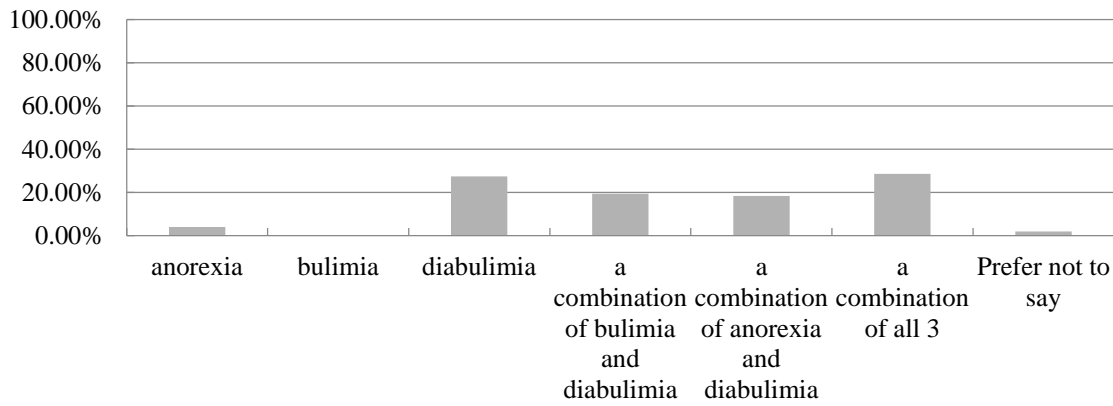


Despite all of the respondents feeling that they did have an Eating Disorder, 38 participants had never been diagnosed with one (38.8%). Of those who had been diagnosed, 22 had been given a diagnosis of Bulimia Nervosa (22.4%), 9 had been given an diagnosis of Anorexia Nervosa (9.2%), 13 had received an Eating Disorder not Otherwise Specified (EDNOS) diagnosis (13.3%), 10 had been given dual Anorexia and Bulimia Nervosa diagnoses (10.2%), 4 had dual diagnoses of Bulimia and EDNOS (4.1%) and 2 had been given a triple diagnosis of Bulimia, Anorexia and EDNOS (2%)

Demographic Chart 2: What were you diagnosed with?

## Have you had an Eating Disorder Diagnosis:
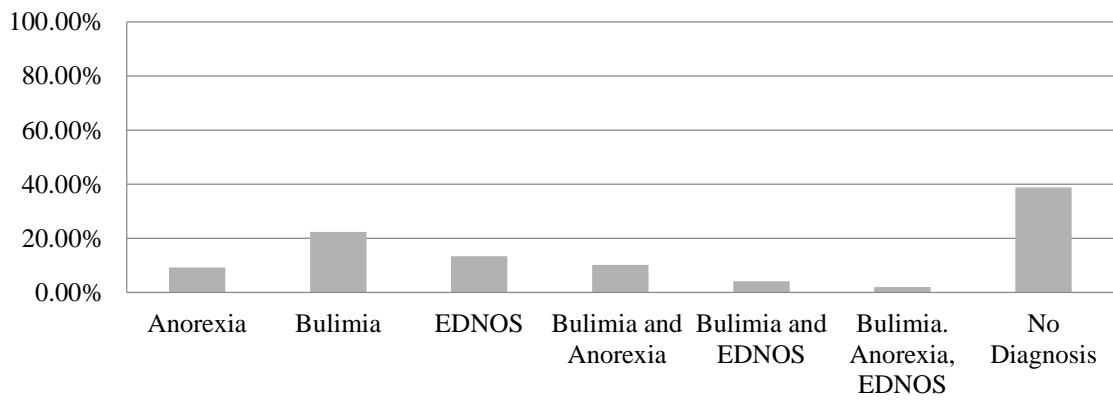


Seventy-six of the 98 respondents had approached a health professional about their Eating Disorder (77.6%), 50 approached their GP (51%), 24 approached their Diabetic Specialist Nurse (DSN) (24.5%), 46 approached their Diabetic Consultant (46.9%), 29 approached a Dietician (29.6%, 13 approached an Eating Disorder Nurse (13.3%), 24 approached an Eating Disorder Consultant (24.5%), 29 approached a psychologist (29.6%), 25 approached a Psychiatrist (25.5%), 17 approached a councillor (17.4%), 3 approached a social worker

(3.1%), 22 approached a mental health worker (22.4%), and 10 approached another health professional (10.2%).

Demographic Chart 3: Whom did you approach for help?



**If so who did you approach? Please select all that apply**
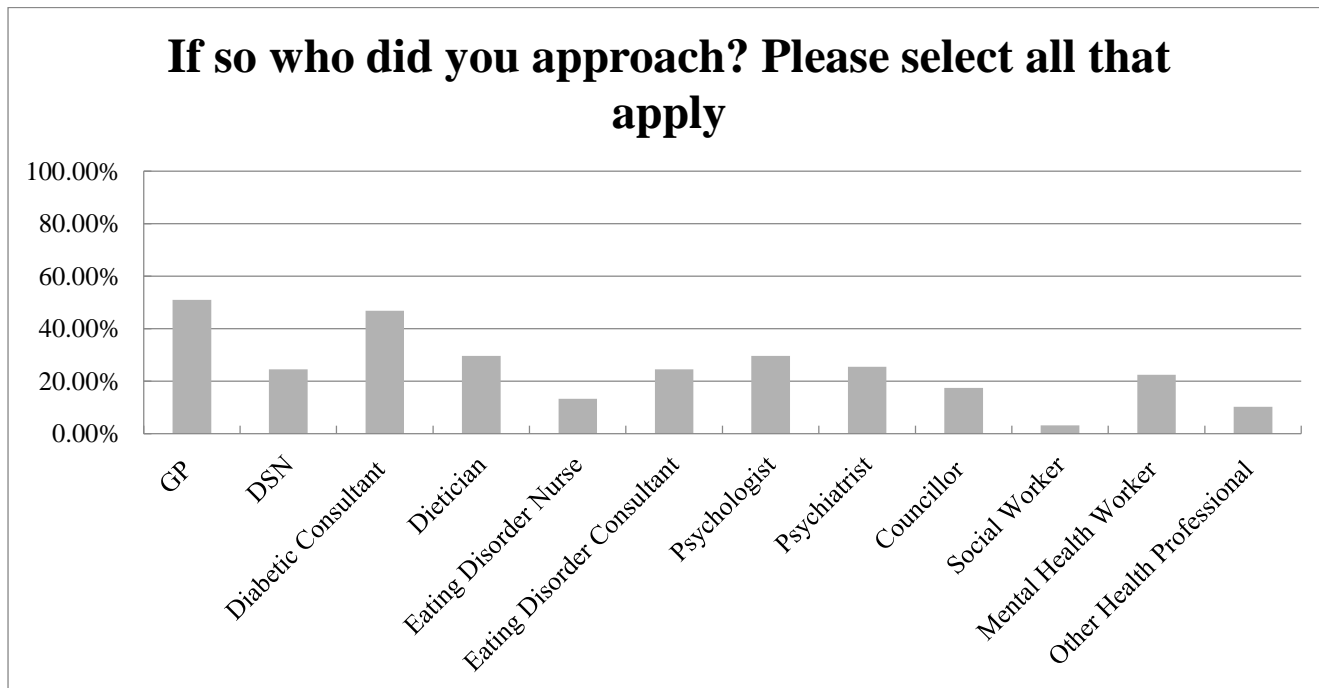
Only 22 of the 98 respondents did not have another mental health diagnosis (22.4%). Of those who did, 27 were diagnosed with depressions (27.5%), 2 had a Personality Disorder (2%), 2 had Bi-Polar (2%), 15 had depression and anxiety (15.2%), 1 had Depression and PTSD (1%), 9 had Depression and Personality disorder (9.3%), 2 had Depression and Addiction (2%), 7 had Obsessive compulsive disorder (7.2%), 1 had Personality Disorder, Depression, OCD and Bi-Polar (1%), 2 had Attention Deficit Hyperactivity Disorder (2%), 2 had Anxiety and Personality Disorder (2%), 3 had Depression, Anxiety and Personality Disorder (3.1%)

Demographic Chart 4: Do you have any other Mental Illnesses?

**Have you been given another mental health diagnosis:**
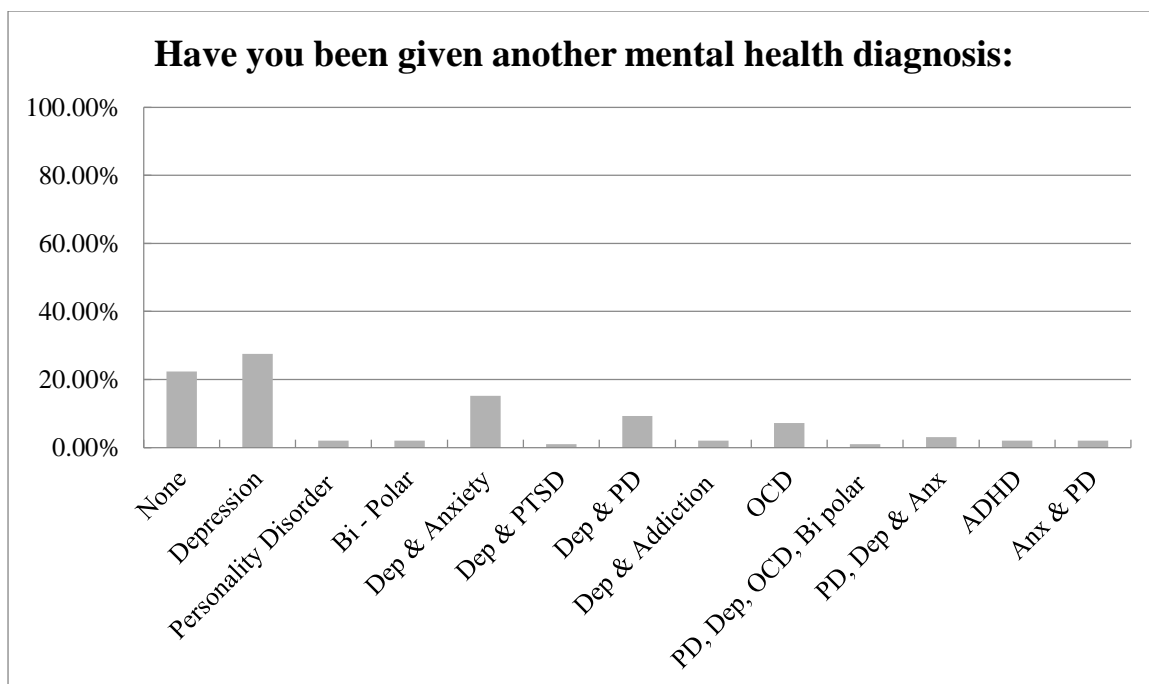
44 respondents were bullied because of their weight (45.8%) and 24 were bullied because of Type 1 Diabetes (24.5%).

*Factor Analysis:*

*Data Screening:* Following data screening, several variables were removed from the analysis as they yielded few correlations > 0.3. (Field 2009). The following items were removed: disturbed attitude to food (item 40), lack of autonomy within the family unit (item 36), family dysfunction (item 35), realisation of quick weight loss (item 11), societal pressure (item 25), distorted body image (item 30), a sense of achievement at losing weight (item 18), academic pressure (item 17), conflict amongst peer group (Item 19), fear of injecting and testing (item 24) and adherence to timed meals (item 10). No outliers were identified and there were no missing cases.

*Principal Component Analysis (PCA):* PCA was chosen as the method of extraction as the aim of the study was to provide an exploratory analysis. Although the Kaiser-Meyer-Olkin measure of sampling adequacy was just short of the recommended value of .5  (KMO = .451) and the diagonals of the anti-image correlation matrix were between .391 and .688, Bartlett's test of sphericity was significant $x^2 = (378) = 1703.112$ p < 0.001 and communalities were all above .3 demonstrating common variance and justifying a Factor Analysis.

*Output:* The initial analysis extracted 8 factors using Kaiser's Criterion of Eigenvalue > 1. Using the scree plot it was shown that the point of inflexion occurred at the 6th component and as such a 5-factor solution was appropriate.

Factor Plot 1

Scree Plot
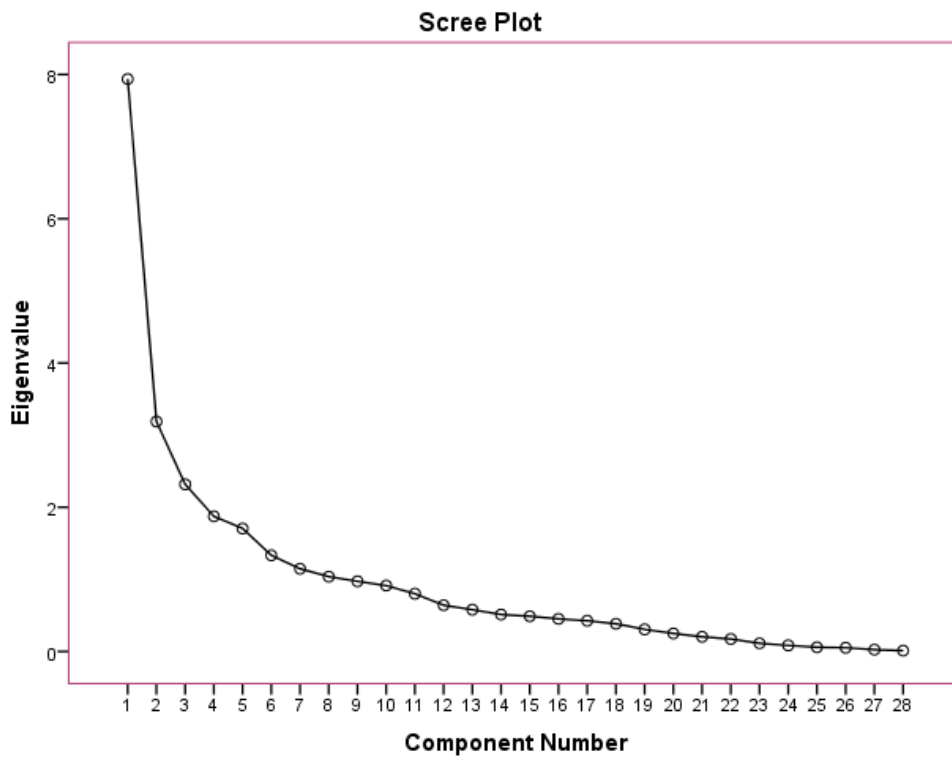
Orthogonal rotation (Varimax) was used to increase the explanatory power of the analysis. After rotation, Factor 1 explained the largest amount of variance (eigenvalue = 5.317, variance = 18.991) and clustered items relating to Diabetes specific distress. Factor 2 clustered highest on Diabetes Diet related items and explained 11.5 % of the variance (Eigenvalue = 3.21). Factor 3 items clustered on more classic risk factors out with Diabetes and explained 10.95% of the variance (Eigenvalue 3.06). Factor 4 explained 10.8% of the variance (Eigen value 3.02) and items related to communication and self-esteem problems. Factor 5 related to issues with Diabetes treatment and explained 8.56% of variance (Eigen value 2.37). In total, then, the 5 Factors extracted explained 60.8% of the total variance in the data.

| Table 1: Rotated Component Matrix and Communalities | Factors Diabetes Distress | Diabetes Diet | Classic Risk Factors | Self Esteem | Diabetes Treatment | Communalities |
|---|---|---|---|---|---|---|
| General Diabetes Stress | 0.887 | | | | | 0.652 |
| Resentment of Diabetes | 0.868 | | | | | 0.706 |
| Denial of Diabetes | 0.734 | | | | | 0.643 |
| embarrassment of testing and or injecting | 0.73 | | | | | 0.779 |
| Diabetes Burn out | 0.676 | 0.463 | | | | 0.438 |
| Fear of Hypos | 0.616 | | | | | 0.551 |
| Needed to feel in control | 0.58 | | | 0.437 | | 0.537 |
| Over - Emphasis of complication | 0.555 | 0.476 | | | | 0.745 |

349

| | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|
| Restrained Diet | | 0.767 | | | | 0.699 |
| Binging after Hypos | | 0.666 | | | | 0.534 |
| Perfectionist Attitude | 0.504 | 0.531 | | | | 0.867 |
| Early Puberty | | 0.49 | | | | 0.455 |
| ED prior to Dx | | | 0.768 | | | 0.593 |
| Initial Weightloss | | | 0.698 | | | 0.656 |
| Weight Gain After Dx | | | 0.698 | | 0.517 | 0.462 |
| Fear of weight gain | | | 0.563 | | | 0.551 |
| Examples of disturbed eating in the family | | | 0.532 | | | 0.719 |
| Pre Existing Mental Health problems | | | 0.514 | | | 0.553 |
| Unhappy gaining weight regardless of DX | | | | 0.784 | | 0.52 |
| Didn't like the way my body looked | | | | 0.753 | | 0.634 |
| Low self esteem | | | | 0.62 | | 0.801 |
| Couldn't talk to parents | | | | 0.514 | | 0.522 |
| Conflict at home with family | | | | 0.476 | | 0.84 |
| Disagreement with parental Diabetes treatment | | 0.403 | | 0.451 | | 0.618 |
| Attention to weight from med staff | | | | | 0.835 | 0.424 |
| Pressure from Health Professionals | 0.427 | | | | 0.759 | 0.491 |
| Maternal Concern with weight and shape | | 0.433 | | | 0.519 | 0.38 |
| Too much Dietary focus | | | | | 0.405 | 0.652 |

*Extraction Method: Principal Component Analysis.*

*Rotation Method: Varimax with Kaiser Normalization.*

*a. Rotation converged in 10 iterations.*
*Please note loadings <0.4 excluded*

## *Discussion*

### *Demographic analysis implications*

Although participants were recruited from predominantly UK-based sources and as such the majority were UK nationals (67%), there were also a relatively large number of international respondents, demonstrating that this issue is one of global importance. While there is increasing awareness of this issue in the UK, a concentrated global effort may be required. As would possibly be expected, the vast majority of the participants were female, corresponding to current literature suggesting that women are more likely to develop Eating Disorders. (Fairburn, Treasure, Schmidt date). There were, however, 3 respondents who selected the 'prefer not to say' option which could be indicative of the stigma surrounding male Eating Disorders (Peate 2001). Furthermore, the participants were recruited from predominantly female-occupied support groups, which could also explain the lack of male respondents.

There was a wide range of ages both for onset of Type 1 Diabetes and onset of an Eating Disorder, with respondents showing deviation from the popular misunderstanding that Eating Disorders only affect teenagers,

the youngest being just 7 at the time of onset and the oldest being 45. However, the mean age of Eating Disorder onset was 16.96 years corresponding with research into Eating Disorders among the general population and demonstrating that this is a critical period of which clinicians should be aware. Noteworthy is that it is during this time that Diabetic teenagers under NHS care undergo 'transition' whereby they are discharged from paediatric care and assigned a new adult clinic. Given the outcome of this study, this transition period may be an ideal time to screen for Eating Disturbances. Professor Jane Morris from the Eden Unit in Aberdeen has undertaken to produce a questionnaire for Type 1 Diabetics who may be at risk of developing an Eating Disorder[24]. Previous attempts at screening have used insufficient measures, for example, the EAT-26[25] which does not measure any levels of Diabetes-related distress. The results of this study show that a thorough screening process at this time may be warranted. However, the range between the time of Type 1 diagnosis and the development of an Eating Disorder (0 – 19 years) also shows that many people may already be showing signs of disturbed eating at diagnosis and as such screening measures as part of the initial treatment and training plan are also warranted. If these high-risk patients can be identified at this time, they may be able to not only get support for the issues they currently have but also may avoid developing Diabulimia which is much more physically dangerous. The range identified, however, suggests that regular screening for an Eating Disorder is also warranted as it can appear long after diagnosis. This may be problematic in that it may appear that yet more focus is on food and weight or worse inform vulnerable patients about the hyperglycaemic process. However, criteria such as HbA1c level could avoid exposure to those deemed not at risk. The HbA1c is a measure of average blood sugar over 3 months and is the measure of how well blood sugar is controlled. Thus if HbA1c is within the normal range, screening at least for insulin manipulation is not necessary.

Data were collected on whether or not the participant had relatives who also had Type 1 Diabetes. It is a common theory in research into Eating Disorders that disturbances may arise from modelling eating habits of the mother (Polivy & Herman 2002; Agras, Hammer, & McNicholas 1999). If the same were to hold for Type 1 Diabetes, then a family-based intervention could provide a basis for treatment. However, the current results show that of the sizeable minority of those who have relatives with Type 1 Diabetes, the brother is the most likely and only 2 of the 98 respondents had mothers with the diagnosis.

A significant finding of this study is that of the 98 respondents, all but 4 stated that they had manipulated or omitted insulin, the remaining 4 having selected the 'prefer not to say' option. Ninety-six percent of the

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[24] Contact: janemorris1@nhs.net

[25] Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological medicine*, 9(2), 273-279.

respondents, then, have had a form of what is commonly termed Diabulimia. This has massive implications as to how we conceive of Eating Disorders. Currently, the manipulation or omission of insulin has no psychiatric diagnosis attached to it and, although it will appear in the DSM V, it is to be categorised as an inappropriate compensatory behaviour in bulimia[26]. The present study has shown that this is clearly problematic. Although 22.4% of the participants had received an official diagnosis of bulimia and this was most common, not one of them identified with that diagnosis. Several stated that they believed they had a mixture of conditions including bulimia, but none identified with that term alone. Thus to diagnose this as bulimia could cause confusion not only among sufferers but also professionals. The physical mechanics of Diabulimia alone are so different from that of bulimia that it is inevitable that for this demographic, this diagnosis may be detrimental. The diagnostic criteria are also inadequate; if the criteria hold from the DSM IV, then periods of manipulation or omission must have recurred over 3 months[27]. As previously discussed, DKA is always fatal if left untreated regardless if this is a single incident or one that has happened twice a week for 3 months. To expect a sufferer to wait 3 months for a diagnosis is potentially life threatening. Also, insulin omission need not occur after a binge or episodic loss of control, because the sufferer may eat a normal amount and withhold the necessary insulin. The categorisation of bulimia for this demographic, then, appears to be somewhat arbitrary and could promote the wrong message to those charged with diagnosing it. However, it may be that additional criteria for those who use this as an 'inappropriate compensatory' measure will be included; it certainly warrants further explanation than that currently given.

Of those who reported approaching a health professional, GP's were the most likely choice (51%). This is significant as in terms of specialism they are arguably the least equipped to deal with patients reporting these problems. As Diabulimia is not an official diagnosis, getting access to any kind of support can prove difficult. Diabetes Consultants were also likely to be approached (46.9%). Again, this is problematic as the main job of a Diabetes consultant is to ensure that blood sugars are stable and in range; they are not trained to deal with psychological aspects of Type 1 Diabetes. The same applies to Diabetic Specialist Nurses who were also likely to be approached (24.5%). Similarly, Diabetes Dieticians (29.6% approached) are trained to provide information about what foods are best for blood sugar, so it may be difficult when a patient presents with an Eating Disorder - particularly insulin omission - and if the patient is binging, the situation is further

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[26] This was confirmed via email with Stephen Wonderlich, Ph.D.Chester Fritz Distinguished Professor

Associate Chairman, Department of Clinical Neuroscience, University of North Dakota School of Medicine & Health Sciences who is charged with the structure of the wording for DSM V.

[27] American Psychiatric Association. (2000). Diagnostic and statistical manual of mental disorders (4th ed., text rev.). Washington, DC: Author.

complicated. Eating Disorder Specialists were also approached: 24.5 % saw an Eating Disorder Consultant and 14.5 % an Eating Disorder Nurse. Conversely, the problem is that Eating Disorder Specialists are not trained in Diabetes, with the separation of mental health nursing into its own specialism whereby trainees are only trained in rudimentary physical medicine, then this becomes even more apparent. Patients who have been in treatment have stated that they felt that most of their sessions are about educating the therapist and that this fosters mistrust and a lack of respect for the treatment provider.[28] Out with the aforementioned specialisms the respondents also approached psychologists (29.6%), psychiatrists (25.5%), social workers (3.1%) mental health workers (22.4%) and other health professionals (10.2%). Many of the respondents approached multiple professionals and as such a system-wide education programme and protocol are warranted.

It has been an important finding of the current study that of the 98 participants only 22.4% did not have co-morbid mental health diagnoses. The most common concurrent diagnosis was depression (27.5%), but this is an expected finding as it has previously been shown that those with Type 1 Diabetes are twice as likely to suffer from the condition. Within females it is more prevalent (Anderson, Freedland, Clouse & Lustman2001), a further 15.9% having co-morbid anxiety and depression and many having further diagnoses such as OCD, ADHD and PTSD. There were also many instances of Personality Disorder. These findings have significant implications for the treatment of this demographic. With such a high incidence of dual, triple and sometimes quadruple diagnoses, it may be worth investigating if the Eating Disorder is a symptom of a broader mental health syndrome. At the very least, other issues should be investigated concurrently with the Eating Disorder. It may also be relevant that bullying was common among the respondents, with 44.9% being victimised because of their weight and 24.5% experiencing bullying due to Type 1 Diabetes.

*Factor analysis and implications*

The first and largest factor extracted named Diabetes Distress demonstrates how important the Diabetes Environment is to the development of an Eating Disorder. The items most highly correlated were: General Diabetes Stress, Resentment of Diabetes, Denial of Diabetes, Embarrassment of Injecting and/or testing, Diabetes Burn Out, Fear of Hypos, Over-Emphasis of Complications and Pressure from health care professionals combined with a Perfectionist Attitude and a Need to feel in control. This demonstrates why health care providers need to be fully aware of the vulnerabilities of their patients. The combination of Need for Control and a Perfectionist attitude with the Diabetes Aspects suggests that clinicians, by pushing for perfect control of blood sugars and trying to scare their patients with the threat of complications and

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[28] www.dwed.org.uk

hypoglycaemia, may be inadvertently pushing the patient away from optimal control and encouraging a stressful Diabetes environment that precipitates Eating Disorder development.

The 2nd factor extracted represents labelled Diabetes Diet the relationship between some Diabetes diet aspects, Binging after Episodes of Hypoglycaemia, Diabetes Burn Out and Over Emphasis of Complications and what would be considered more 'classic' Eating Disorder predictors, Restrained Diet, Perfectionist Attitude, Early Puberty and to a lesser extent, family issues, disagreement with Parents on Diabetes treatment and Maternal Concern with Weight and Shape. This shows a complex interplay of environments but demonstrates that education about diet could be beneficial. Currently most clinics in the UK offer a programme called DAFNE, (Dose Adjustment for Normal Eating) and early screening for eating disturbances could identify patients who could benefit from an early intervention, if this were coupled with information for families and mothers about the risk factors for Eating Disorders, then perhaps development could be averted.

Factor 3, Classic Risk was composed of items that described those already highly at risk at diagnosis and included already having an Eating Disorder and a Previous Mental Health Diagnosis with Initial Weight Loss, Weight Gain after Diagnosis, Fear of Weight Gain and Examples of Disturbed Eating in the Family. The identification of this Factor justifies screening for an Eating Disorder at the time of diagnosis so that patients can get help before Diabulimia has a chance to develop.

The 4th Factor, Self Esteem, identified shows the importance of Self Esteem, Body Image and Family Cohesion. The highest loadings were on Didn't like the way my body looked, Unhappy with Weight and low self-esteem as well as lesser loadings on Couldn't Talk to Parents, Disagreement with Family over Diabetes Treatment and Conflict with Family at Home. Self Esteem and Body image are as important to this demographic as they are to the general population, but as the results show that family communication are also important in concurrence; this is suggestive of a larger environmental problem. Further questions arise about whether body image is affected by having Type 1 Diabetes and if this is exacerbated by communication problems in the family. Intervention at the family level could then provide the basis for treatment.

Factor 5, Diabetes Treatment, had the highest loading on Attention to Weight from Medical Staff, Pressure from Health Professionals and Weight Gain on Diagnosis, with lower loadings on Maternal Concern with Weight and Shape and Too Much Dietary Focus. This may be representative of sensitivity to weight concerns that are then exacerbated by health care professionals. As such, training for health care professionals that explains potential vulnerability of those with Type 1 Diabetes could be beneficial. For example, being weighed is part of the quarterly checks that a Diabetic undertakes in clinic, this could be eradicated for at least those in transition services or within the critical age period who have no visually obvious weight issues. This could potentially avoid health professionals inadvertently triggering the development of Eating Disorders.

*Limitations*

There are obvious limitations with the current study. Firstly as an undergraduate project, work with clinical populations is forbidden and as such the pool of participants in greatly reduced. This may partly explain the small sample size and where the study's main limitations lie. A larger sample size may have produced higher correlations and factor loadings also more of the variables may have reached the significance criterion to be included.

## Conclusion

### Suggestions for Further Research

It has been confirmed that in the upcoming DSM-V compulsive overeating will be included in the Eating Disorders section as Binge Eating Disorder[29]. A future study may take this into consideration and investigate whether there is also a higher incidence of BED among this population, as that would also have significant ramifications for the treatment of Type 1 Diabetes. Given the limited scope of the current study as work undertaken as part of an undergraduate degree, a larger study focussing on a global scale would certainly be warranted. Thirty-three percent of the respondents were not of UK nationality and an investigation into differences between countries and cultures could yield interesting results and help inform a global policy on Eating Disorders in Type 1 Diabetes. It has also been widely reported that incidences of males developing Eating Disorders is rising. A future study could concentrate on aiming to recruit more male participants.

### Summary

The current study has demonstrated that Type 1 Diabetes and Eating Disorders are extremely complicated. Many issues are highly inter related. The demographic information shows that those suffering with Eating Disorders also often have multiple other mental health diagnoses, have been bullied and will approach a multitude of health professionals. The Factor Analysis has demonstrated that several aspects of the Diabetes environment diet, distress or treatment are incredibly important in the development of Eating Disorders but that individual differences may also play an important role. There are obvious problems with the status quo and the current study has hopefully illuminated some possible suggestions on how best to change the current situation. What is certain is that currently this demographic are misunderstood, under represented and under treated.

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Appendix

Appendix A: Original Advert

**Are you female and over the age of 18 with at least two years solid recovery from a Type 1 Diabetes related Eating Disorder? Jacqueline Allan from Birkbeck, University who many of you will know from the support forums is carrying out research into why Eating Disorders develop among women with type 1 Diabetes as compared to those without. If you would like to participate in this research by answering a brief questionnaire that will look at feelings towards the development of your illness, please follow the below link. Please note that no identifying information is required so answers are completely confidential.**

Appendix B: Questionnaire

Questionnaire

Please ensure that you have 10 - 20 free minutes to answer this questionnaire in a quiet environment with no distractions. This questionnaire has 2 parts. In part 1 you will be asked some basic questions about yourself, if at any point you do not want to give an answer please select the rather not say option. If at any point you would like to withdraw from the study you can just close your browser.

Please only proceed if you have more than two years in recovery and are over the age of 16

Part 1:

How old are you

At what age were you diagnosed with Type 1 Diabetes?

What nationality are you

Do any other family members have type 1 Diabetes?

What is their relationship to you?

How long after diagnosis did you develop an Eating Disorder?

Did you manipulate or omit insulin

Do you think that you had anorexia, bulimia, Diabulimia, or a combination of bulimia and Diabulimia, a combination of anorexia and Diabulimia, a combination of anorexia and bulimia, a combination of all 3?

In the time that you were suffering from your Eating Disorder did you seek professional help?

If so who did you approach, GP, DSN, Diabetic Consultant, Dietician, Eating Disorder Specialist, Other health professional please state……

Have you ever received an official Eating Disorder Diagnosis? If so what

Have you ever received another mental health diagnosis? If so What

Where you the victim of bullying because of weight?

Were you the victim of bullying because of Type 1 Diabetes?

Part 2

Directions: The following statements concern your perception of the environment you were in at the time of developing an Eating Disorder. Please indicate how strongly you agree with each statement by using the below judgements. In the boxes after each statement, click a number from 1 to 5 from the following scale:

1) Strongly disagree

2) Disagree

3) Neither disagree nor agree

4) Agree

5) Strongly agree

Please note that there are no right or wrong answers. Take your time and consider each statement carefully. Once you have completed all questions please select submit at the bottom of the page. If at any point you do not wish to continue please just close your browser.

In the second section you are asked to rate if you feel certain statements and factors contributed to the development of your Eating Disorder, or if they did not.  In some questions you are asked if a particular factor **did** contribute to the development of your Eating Disorder, for example:

    a)  Pressure to be good at sports **contributed** to the development of my Eating Disorder

If you don't agree with this statement then you would select 1) Strongly Disagree or 2) Disagree, if you feel neutral about this particular issue you would select 3) Neither Agree or Disagree, if however you felt that it did contribute you would select 4) Agree and if you felt that this played a significant part in the development you would select 5) Strongly Agree

You will notice that some of the questions are worded differently to this, the reason they are is to avoid a phenomenon called response bias, which is where a participant may get into a habit of selecting responses. For this reason you should consider these questions carefully before responding, for example

   b)  Sibling Rivalry **did not contribute** to the development of my Eating Disorder

In this case you may feel that in fact sibling rivalry **did** play a part in the development of your Eating Disorder so in this case you would 2) disagree or, 1) strongly disagree. You may feel neutral about sibling rivalry so in that case you would select 3) Neither agree or disagree or you may feel like it did not contribute, in which case you would either 4) agree or 5) strongly agree with the statement

1) Strongly Disagree   2) Disagree      3)Neither Disagree or Agree  4) Agree         5)Strongly  Agree

1) I needed to feel in control of something and this contributed to the development of my Eating Disorder

2) Initial weight loss at diagnosis did not contribute to the development of my Eating Disorder

3) A restrained diet did not contribute to the development of my Eating Disorder

4) Weight gain after I started on Insulin did not contribute to the development of my Eating Disorder

5) Early Puberty contributed to the development of my Eating Disorder

6) Low self-esteem contributed to the development of my Eating Disorder

7) Too much focus on my diet contributed to the development of my Eating Disorder

8) Attention to my weight from Medical Staff did not contribute to the development of my Eating Disorder

9) Regardless of my Diabetes I was unhappy I was gaining weight and this contributed to the development of my Eating Disorder

10) Adherence to timed meals contributed to the development of my Eating Disorder

11) Conflict at home within my family did not contribute to the development of my Eating Disorder

12) The realisation I could lose weight quickly contributed to the development of my Eating Disorder

13) General Diabetes stress contributed to the development of my Eating Disorder

14) Fear of weight gain did not contribute to the development of my Eating Disorder

15) A perfectionist attitude towards my blood sugar did not contribute to the development of my Eating Disorder

16) Denial that I had Diabetes did not contribute to the development of my Eating Disorder

17) Academic Pressure did not contribute to the development of my Eating Disorder

18) A sense of achievement at losing weight contributed to the development of my Eating Disorder

19) Conflict among my peer group contributed to the development of my Eating Disorder

20) Fear of hypoglycaemia contributed to the development of my Eating Disorder

21) Embarrassment at injecting or testing in front of anyone contributed to the development of my Eating Disorder

22) Diabetes Burn Out did not contribute to the development of my Eating Disorder

23) An over – emphasis on what would happen if I didn't look after myself did not contribute to the development of my Eating Disorder

24) Fear of injecting and or self-testing contributed to the development of my Eating Disorder

25) Societal pressure to be thinner contributed to the development of my Eating Disorder

26) Examples of disturbed eating in my family contributed to the development of my Eating Disorder

27) Overeating/ binging, following episodes of hypoglycaemia contributed to the development of my Eating Disorder

28) Pressure from my health care professionals contributed to the development of my Eating Disorder

29) Maternal concern with weight and shape did not contribute to the development of my Eating Disorder

30) A distorted body image did not contribute to the development of my Eating Disorder

31) Resentment of having Diabetes did not contribute to the development of my Eating Disorder

32) An Eating Disorder prior to developing Type 1 Diabetes contributed to the development of my Eating Disorder

33) Disagreement with the way my parents dealt with my Diabetes contributed to the development of my Eating Disorder

34) I felt I couldn't talk to my parents and this contributed to the development of my Eating Disorder

35) Family dysfunction contributed to the development of my Eating Disorder

36) Feeling that I was not an individual within my family did not contribute to the development of my Eating Disorder

37) I had pre-existing Mental Health issues and this contributed to the development of my Eating Disorder

38) I didn't like the way my body looked and this contributed to the development of my Eating Disorder

39) My family was very rigid and this contributed to the development of my Eating Disorder

40) A disturbed attitude to food did not contribute to the development of my Eating Disorder


Appendix C: Ethics Approval

**Departmental Ethics Committee**
**DEPARTMENT OF PSYCHOLOGICAL SCIENCES**
**BIRKBECK COLLEGE UNIVERSITY OF LONDON**

**CLASSIFICATION OF RESEARCH PROPOSAL**

**Date of Submission:** December 2012
**Investigator:** Jacqueline Anne Allan
**Reference Number:** 121318
**Title of project:** The development of eating disorders in females with Type 1 diabetes: A factor analysis

Dear Jacqueline
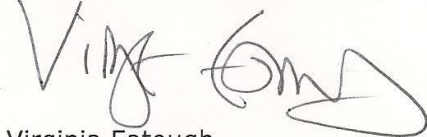
The above application has been given ethical approval by the departmental ethics committee.

You should be aware that it is your responsibility to report any unexpected problems or events arising from the research which might have adverse consequences for you and/or your participants. In the first instance, please discuss with your supervisor who will advise you as to whether the problem causes a change to the planned research and needs further ethical approval from the committee. If so, please submit a revised application giving details of why this is necessary.

Approval for this study expires December 2015. If the study is still ongoing at this time please submit a renewal of ethical approval form which can be found on the departmental webpage.

Please retain this certificate for your records.

Good luck with the research.

Virginia Eatough
Chair of the departmental ethics committee

Date: 07-12-2012

# Changing diabetes®

# Diabetes and eating disorders: Insulin omission and the DSM-5

*Jacqueline Allan, Diabetics with Eating Disorders and Birkbeck University, London; Dr Jen Nash, Diabetes Wellbeing Service, Hillingdon Hospital and Positive Diabetes*

The fact that eating disorders are more prevalent in young women with type 1 diabetes has been well documented (Affenito et al, 1997; Rodin et al, 1986; Rydall et al, 1997; Pinar, 2005). The omission of insulin in order to promote weight loss is much less understood and researched, however. This article describes the current clinical picture of diabetes-associated eating disorders and outlines the work of the charity Diabetics with Eating Disorders (DWED), which was established to support individuals with the condition.

## Defining insulin omission

The practice of insulin omission for weight loss purposes is commonly named diabulimia; however, it should be noted that this is a label given by the media. Among some academics, the nomenclature eating disorders in diabetes mellitus type 1 (ED-DMT1) is used to denote the spectrum of disturbed eating behaviour found within this specific demographic.

## Insulin omission as a DSM diagnostic category

Unlike anorexia, bulimia and binge eating disorder, insulin omission is not named as a mental health condition in its own right in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). Instead, insulin omission appeared in the DSM-IV subsumed under the criteria for bulimia (American Psychiatric Association, 2000):

*"Individuals with diabetes mellitus and bulimia nervosa may omit or reduce insulin doses in order to reduce the metabolism of food consumed during eating binges."*

This reference has been built upon only slightly in the recently published DSM-5 by the additional inclusion of insulin omission under the criteria for anorexia nervosa (American Psychiatric Association, 2013):

*"Individuals with anorexia nervosa may misuse medications, such as by manipulating dosage, in order to achieve weight loss or avoid weight gain. Individuals with diabetes mellitus may omit or reduce insulin doses in order to minimize carbohydrate metabolism."*

Although another mention of insulin omission as clinically relevant is a welcome addition to the DSM-5, the position of DWED is that the failure to identify chronic insulin omission as a mental health condition in its own right is problematic. Under these diagnostic criteria, one may ask: "what is the difference between people with diabetes and anorexia and those with diabetes and bulimia?" Simply put, the answer is weight; however, determining eating disorder severity by weight is not relevant to people with type 1 diabetes who omit insulin. The measure of severity for this demographic would more accurately be HbA$_{1c}$. Furthermore, these diagnostic criteria propagate the idea that one simply has anorexia or bulimia with diabetes as a footnote. We know that there are diabetes-specific environmental factors that contribute to the development of diabulimia and, perhaps more importantly, that eating disorder treatment programmes that do not address the diabetes-related factors fail abjectly (Rodin et al, 1991; Smith et al, 2008; Ismail et al, 2010).

## A treatment model that works

Currently, individuals who are identified as omitting insulin are usually referred to their local eating disorder service. The difficulty is that eating disorder professionals are not experts in diabetes or the psychological implications of diabulimia, often seeing the problem as one of food alone rather than one of food, insulin and all the other stresses of the diabetes regimen. This leads to inappropriate use of NHS resources and, therefore, increased costs, not only in the initial ineffective treatment, but also in the costs of dealing with people with seriously uncontrolled diabetes over the long term. There is also an impact on the individuals themselves, which include failure to maintain employment, reliance on benefits, deterioration in mental wellbeing and relationships and, at its worst, death.

A person with type 1 diabetes who has an eating disorder, particularly insulin omission, cannot be dealt with in isolation by an eating disorder team. What DWED has observed to be effective is the patients' DSNs being proactive in collaborating with both the individuals and their eating disorder teams to guide and educate them as to how diabetes can be managed whilst the eating disorder is being treated. A multidisciplinary approach is the only effective way to

*Eating disorders in diabetes mellitus type 1 awareness poster (Diabetics with Eating Disorders)*

following the death of a close friend. The organisation has grown from a grassroots support service to a well-established body involved in campaigning, training, raising awareness and advocacy. The trustee board consists of recognisable names in the fields of diabetes (Dr Stephen Thomas, Dr Miranda Rosenthal, Dr Jen Nash and Nicola Allen, DSN) and eating disorders (Prof Janet Treasure, OBE), as well as former patients and carers. DWED regularly advises and trains healthcare bodies (such as the Institute of Psychiatry, Royal College of Physicians and Royal College of Nursing), the UK Parliament, the Scottish Parliament, charities (such as Diabetes UK, the Juvenile Diabetes Research Foundation and Beat) and private clinics on eating disorders in type 1 diabetes. They have also been involved in the development of several intervention programmes, have delivered lectures internationally and are currently involved with bodies such as Healthwatch, NICE, Strategic Clinical Networks and NHS England and NHS London. To find out more about DWED, or to discuss how they can work with your team, please visit www.dwed.org.uk or email info@dwed.org.uk. ■

Affenito SG, Backstrand JR, Welch GW et al (1997) Subclinical and clinical eating disorders in IDDM negatively affect metabolic control. *Diabetes Care* **20**: 182–4

American Psychiatric Association (2000) *Diagnostic and Statistical Manual of Mental Disorders* (4th edition, revised). APA, Arlington, VA, USA: 546

American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders* (5th edition). APA, Arlington, VA, USA: 376

Ismail K, Maissi E, Thomas S et al (2010) A randomised controlled trial of cognitive behaviour therapy and motivational interviewing for people with type 1 diabetes mellitus with persistent sub-optimal glycaemic control: a Diabetes and Psychological Therapies (ADaPT) study. *Health Technol Assess* **14**: 1–101, iii–iv

Pinar R (2005) Disordered eating behaviors among Turkish adolescents with and without type 1 diabetes. *J Pediatr Nurs* **20**: 383–8

Rodin GM, Johnson LE, Garfinkel PE et al (1986) Eating disorders in female adolescents with insulin dependent diabetes mellitus. *Int J Psychiatry Med* **16**: 49–57

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Rydall AC, Rodin GM, Olmsted MP et al (1997) Disordered eating behavior and microvascular complications in young women with insulin-dependent diabetes mellitus. *N Engl J Med* **336**: 1849–54

Smith FM, Latchford GJ, Hall RM, Dickson RA (2008) Do chronic medical conditions increase the risk of eating disorder? A cross-sectional investigation of eating pathology in adolescent females with scoliosis and diabetes. *J Adolesc Health* **42**: 58–63

treat a person with type 1 diabetes and an eating disorder. Hopefully, the next revision of the DSM will differentiate diabulimia and reflect the need for this approach; certainly, DWED is working hard to raise awareness of the condition and establish treatment protocols that are effective. A large part of the work that DWED is undertaking involves training both diabetes teams and eating disorder teams in the best ways to support their patients with eating disorders.

## Further information

Diabetics with Eating Disorders (DWED) is a Registered Charity in Scotland that was set up in April 2009 by Jacqueline Allan

**Appendix H: Recruitment Advert**

Do you have some time to contribute to research on Type 1 Diabetes? My name is Jacqueline Allan and I am a Type 1 Diabetic currently undertaking a phd in research and my dissertation is on psychological aspects of Type 1 Diabetes and Blood Sugar Control. This questionnaire will discuss potentially sensitive subjects, such as relationship to food, Diabetes distress, family relations and psychological state. All you need to do is answer a questionnaire that you can find here. (www.typeoneandpsychology.org.uk) This is quite a long questionnaire and you can save your progress and complete it in your own time (it shouldn't take any more than 45 mins) For the duration of the study you will receive a log in based on your email address if you wish to complete it in more than one session. This system is entirely computerised however so no-one including the researcher will know or keep your email after the study is completed. There is absolutely no identifying information required so your responses will remain completely anonymous. You are also free to withdraw your participation at any time.

**Appendix I: First Page of the Website & Ethics Screening**

The aim of the (Psychological Aspects in Type 1 Diabetes (PA1D) project is to understand how Type 1 Diabetes affects YOU and therefore what you and those who treat you, need to know to help manage your blood sugar. The hope is that this will be a large scale study with wide reaching implications across the healthcare system. We have designed the study to be as easy to complete as possible but as it may take longer than 10 minutes you can log in and out as and when you see fit. Please also share this website with your Type 1 friends and colleagues so that we can get as many responses as possible. The bigger the response, the more we can do with the data. Thank you so much for getting involved in this ground breaking research. Hopefully together we can improve treatment and outcomes for Type 1 Diabetes everywhere.

Kind Regards

The (pancreatically challenged) Researcher

Share (facebook, twitter, linkedin, tumblr)

I would like to take part

Click **YES**

**DEPARTMENT OF PSYCHOLOGICAL SCIENCES**
**BIRKBECK UNIVERSITY OF LONDON**

**Title of Study:**                 Psychological Factors effecting Blood Sugar Control
**Name of researcher:** *Jacqueline Anne Allan*

Dear Participant

The study is being undertaken as part of a Doctoral degree in the Department of Psychological Sciences, Birkbeck University of London. The study has received ethical approval and the researcher has a DBS certificate

This is a study of how psychosocial factors affect variations in blood sugar. If you agree to participate you will take part in a series of questionnaires. These questionnaires will discuss potentially sensitive subjects, such as relationship to food, Diabetes distress, family relations and psychological state.

This is quite a long questionnaire and you can save your progress and complete it in your own time (it shouldn't take any more than 45 mins). For the duration of the study you will receive a log in based on your email address if you wish to complete it in more than one session. The system for providing this is entirely computerised so no-one including the researcher will know or keep your email after the study is completed unless you request this. There is absolutely no identifying information required so your responses will remain completely anonymous. You are also free to withdraw your participation at any time.     There will be opportunity for involvement in a future project at the ned of this questionnaire for which you can leave your email address if you wish to participate. This information will be stored securely and separately from your responses to the questionnaire so there will be no way of identifying your responses to the questionnaire.

All Data will remain totally anonymous and all information will be treated with the utmost confidentiality. A dedicated secure server has been purchased for this purpose. No identifying information will be recorded unless you wish to participate in the future research

The results of the study will be written up in a report of the study for my Masters degree. You will not be identifiable in the write up or any publication which might ensue.

If you have any questions at all regarding this study please feel free to contact the researcher

Jacqueline Allan
j.allan@bbk.ac.uk
07869 116 832

Please confirm the below before starting the questionnaire

I have had the details of the study explained to me and willingly consent to take part.

YES/NO

My questions have been answered to my satisfaction and I understand that I may ask further questions at any time.

YES/NO

I understand that I will remain anonymous and that all the information I give will be used for this study only.

YES/NO

I understand that I may withdraw my consent for the study at any time and to decline to answer any particular questions.

YES/NO

I confirm that I am over 16 years of age.

YES/NO

The study is supervised by Dr Anne Miles If you wish to contact the supervisor, contact details are:

Dr Anne Miles
ae.miles@bbk.ac.uk
020 7079 0868

Departmental address: Department of Psychological Sciences, Birkbeck University of London, Malet St, London WC1E 7HX

If you have any questions at all you may also contact the researcher

Jacqueline Allan
j.allan@bbk.ac.uk
07869 116 832

**Appendix J: Debrief Screen**

Dear Participant

Thank you so much for participating in this research. Your input will help develop the canon of research about psychosocial factors relating to blood sugar. If you would like to see the dissertation that results from this research, announcements will be available through the website you have answered the questionnaire on.

There are a number of resources to help you if you feel that you have been affected by any issues raised by this survey. Please see the below links for further support

http://www.Diabetessupport.co.uk/

www.dwed.org.uk

http://www.diabulimiahelpline.org/

www.Diabetes.org.uk

http://www.bpdworld.org/

http://www.mind.org.uk/

http://www.sane.org.uk/

http://www.rethink.org/

http://www.Diabetes.co.uk/forum/

You can also contact the researcher at any time

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And don't forget to share this study fb/tw/tb/ln/G+

## Appendix K: Full Sample Characteristics

| Nationality | n |
|---|---|
| American | 278 |
| British | 257 |
| Australian | 31 |
| Canadian | 30 |
| Irish | 13 |
| Danish | 13 |
| New Zealander | 9 |
| European/ American | 9 |
| South African | 5 |
| Swedish | 4 |
| Greek | 3 |
| Prefer not to say | 3 |
| German | 2 |
| Barbadian | 2 |
| Norwegian | 2 |
| Polish | 2 |
| Latvian | 2 |
| Mexican/ American | 2 |
| More than dual national | 2 |
| Finish | 1 |
| Dutch | 1 |
| Jordanian | 1 |
| Czech | 1 |
| Indian | 1 |
| Italian | 1 |
| Filipino | 1 |
| Singaporean | 1 |
| Bulgarian | 1 |
| Austrian | 1 |
| Croatian | 1 |
| Malaysian | 1 |
| Israeli | 1 |
| French | 1 |
| Lithuanian | 1 |
| Russian/ European | 1 |

| Country of Residence | n |
|---|---|
| America | 293 |
| UK | 274 |
| Canada | 34 |
| Australia | 32 |
| Denmark | 17 |
| New Zeland | 8 |
| Eire | 4 |
| South Africa | 3 |
| Sweden | 3 |
| Germany | 2 |
| Greece | 2 |
| Latvia | 2 |
| Jordan | 1 |
| Czech Republic | 1 |
| India | 1 |
| Barbados | 1 |
| Norway | 1 |
| The Philippines | 1 |
| Poland | 1 |
| Bulgaria | 1 |
| Austria | 1 |
| Malaysia | 1 |
| Israel | 1 |
| France | 1 |
| China | 1 |

| Ethnicity | n |
|---|---|
| Caucasian | 559 |
| Prefer not to say | 81 |
| Mixed Race | 18 |
| Hispanic | 8 |
| Black | 6 |
| Jewish | 4 |
| Asian | 4 |
| Chinese | 2 |
| Arabic | 2 |
| Native American | 1 |
| Aborigine | 1 |
| South East Asian | 1 |

| Diabetes Related Complication | n |
|---|---|
| Retinopathy | 135 |
| Neuropathy | 101 |
| Gastroparesis and other stomach related issues | 47 |
| Nephropathy | 36 |
| High Blood Pressure | 19 |

| Diabetes Related Complication | n |
|---|---|
| Tendon issues | 15 |
| Cardiovascular Disease | 13 |
| Macular Oedema | 13 |
| Autonomic Neuropathy | 9 |
| High Cholesterol | 8 |
| Dental Issues | 7 |
| Cataracts | 7 |
| Glaucoma | 6 |
| Necrobiosis Lipoidica Diabeticorum | 6 |
| Ulcers | 6 |
| Charcot Foot/ neuroarthropathy | 5 |
| Erectile Dysfunction | 4 |
| Osteoporosis/ penia | 4 |
| Hyperlipidaemia | 3 |
| Diabetic mastopathy | 3 |
| Vitamin Deficiencies | 2 |
| Amputation | 2 |
| Liver Damage | 2 |
| myofascial pain syndrome | 1 |
| Papilledema | 1 |
| Nodular perigo | 1 |

| Other Diagnosed Health Condition | n |
|---|---|
| Hypothyroidism | 95 |
| Asthma | 56 |
| Coeliac | 23 |
| Allergies | 19 |
| Hypertension | 16 |
| PCOS | 12 |
| Arthritis | 12 |
| IBS | 9 |
| Vitamin Deficiencies | 9 |
| Rheumatoid Arthritis | 8 |
| Epilepsy | 7 |
| Hyperthyroid | 7 |
| Migraines | 7 |
| Fibromyalgia | 7 |
| Carpal Tunnel/ Raynaud's | 6 |
| Endometriosis | 6 |
| Psoriasis | 5 |
| Chronic Fatigue | 5 |
| Hyper Mobility | 4 |
| Eczema | 4 |
| Gastritis | 4 |
| Cancer | 4 |
| Gastro -Oesophageal Reflux Disease | 3 |
| Duputrens Contracture | 3 |
| Ulcerative colitis | 3 |

| Other Diagnosed Health Condition | n |
|---|---|
| Familial Hypercholesterolemia | 3 |
| Heart Disease | 3 |
| Addison's | 2 |
| Cystic Fibrosis | 2 |
| Vertigo | 2 |
| Chron's Disease | 2 |
| Dry Eye | 2 |
| Amenorrhea | 2 |
| Meniere's | 2 |
| Incontinence | 2 |
| Morton's Neuroma | 2 |
| Pancreatitis | 2 |
| Sinus Tachycardia | 2 |
| Psoriatic Arthritis | 2 |
| Temporomandibular Joint Disorder | 2 |
| Coronary Artery Disease | 1 |
| Pernicious Anaemia | 1 |
| Antiphospholid Syndrome | 1 |
| Parkinson's | 1 |
| Polymorphic Light Eruptions | 1 |
| Emphysema | 1 |
| Ehlers Dan - los Syndrome | 1 |
| Barret's Oesophagus | 1 |
| Sciatica | 1 |
| Oral Lichen Planus | 1 |
| Restless Legs Syndrome | 1 |
| Peritonitis | 1 |
| Congenital Scoliosis | 1 |
| Early Menopause | 1 |
| Faecal Incontinence | 1 |
| Sleep Apnoea | 1 |
| Spondyloestesis | 1 |
| Chronic Idiopathic Cough | 1 |
| Syndrome of Inappropriate Antidiuretic Hormone | 1 |
| Sarcoidosis | 1 |
| Supraventricular Tachycardia | 1 |
| Chronic Obstructive Pulmonary Disease | 1 |
| Gilberts Syndrome | 1 |
| Uveitis | 1 |
| Pituitary Adenomas | 1 |
| Ankylosing Spondylitis | 1 |
| Postural Tachycardia Syndrome | 1 |
| Plantar Fasciitis | 1 |
| Vitiligo | 1 |
| Interstitial Cystitis | 1 |
| Congestive Heart Failure | 1 |
| Thalassemia | 1 |
| Hirschsprung's Disease | 1 |
| Spherocytosis | 1 |
| Sickle Cell | 1 |

| Other Diagnosed Health Condition | n |
| --- | --- |
| Lupus | 1 |
| Sjogren's | 1 |
| Myasthenia Gravis | 1 |
| Goitre | 1 |
| Microplactinoma | 1 |
| Glaucoma | 1 |
| Multiple Sclerosis | 1 |
| Primary Biliary Cirrhosis | 1 |
| haemochromatosis | 1 |

| Mental Health Diagnoses | n |
| --- | --- |
| Depression | 155 |
| Anxiety | 68 |
| Borderline | 14 |
| Generalised Anxiety Disorder | 11 |
| Bipolar Disorder | 10 |
| Post-Traumatic Stress Disorder | 10 |
| Obsessive Compulsive Disorder | 7 |
| Attention deficit disorders | 5 |
| Social Phobia | 4 |
| Panic Disorder | 4 |
| Asperger's | 3 |
| Specific Phobia | 2 |
| Adjustment Disorder | 1 |
| Agoraphobia | 1 |
| Disassociation | 1 |
| Seasonal Affective Disorder | 1 |
| Avoidant Personality Disorder | 1 |
| Dependent Personality Disorder | 1 |

| | | DEPS1 Losing weight is an important goal to me | DEPS2 I skip meals and/or snacks | DEPS3 Other people have told me that my eating is out of control | DEPS4 When I overeat, I don't take enough insulin to cover the food | DEPS5 I eat more when I am alone than when I am with others | DEPS6 I feel that it's difficult to lose weight and control my Diabetes at the same time | DEPS7 I avoid checking my blood sugar when I feel like it is out of range | DEPS8 I make myself vomit | DEPS9 I try to keep my blood sugar high | DEPS10 I try to eat to the point of spilling ketones in my urine | DEPS11 I feel fat when I take all of my insulin | DEPS12 Other people tell me to take better care of my Diabetes | DEPS13 After I overeat, I skip my next insulin dose | DEPS14 I feel that my eating is out of control | DEPS15 I alternate between eating very little and eating huge amounts | DEPS16 I would rather be thin than to have good control of my Diabetes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| DEPS1 Losing weight is an important goal to me | Pearson Correlation | 1 | 0.319 | 0.331 | 0.297 | 0.454 | 0.606 | 0.322 | 0.232 | 0.322 | 0.282 | 0.438 | 0.239 | 0.309 | 0.523 | 0.445 | 0.448 |
| | Sig. (2-tailed) | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 509 | 509 | 508 | 507 | 507 | 504 | 509 | 508 | 508 | 505 | 508 | 508 | 509 | 509 | 508 | 502 |
| DEPS2 I skip meals and/or snacks | Pearson Correlation | 0.319 | 1 | 0.370 | 0.349 | 0.300 | 0.294 | 0.370 | 0.336 | 0.448 | 0.391 | 0.420 | 0.347 | 0.417 | 0.347 | 0.536 | 0.471 |
| | Sig. (2-tailed) | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 509 | 510 | 509 | 508 | 507 | 504 | 510 | 509 | 509 | 506 | 509 | 509 | 510 | 509 | 509 | 502 |
| DEPS3 Other people have told me that my eating is out of control | Pearson Correlation | 0.331 | 0.370 | 1 | 0.443 | 0.432 | 0.340 | 0.436 | 0.404 | 0.492 | 0.449 | 0.486 | 0.515 | 0.489 | 0.603 | 0.507 | 0.527 |
| | Sig. (2-tailed) | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 508 | 509 | 509 | 508 | 507 | 503 | 509 | 508 | 508 | 505 | 508 | 509 | 509 | 508 | 509 | 501 |
| DEPS4 When I overeat, I don't take enough insulin to | Pearson Correlation | 0.297 | 0.349 | 0.443 | 1 | 0.531 | 0.399 | 0.618 | 0.323 | 0.665 | 0.569 | 0.577 | 0.580 | 0.679 | 0.600 | 0.548 | 0.606 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 507 | 508 | 508 | 508 | 506 | 502 | 508 | 507 | 507 | 504 | 507 | 508 | 508 | 507 | 508 | 501 |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| cover the food | | | | | | | | | | | | | | | | | |
| DEPS5 I eat more when I am alone than when I am with others | Pearson Correlation | 0.454 | 0.300 | 0.432 | 0.531 | 1 | 0.483 | 0.454 | 0.301 | 0.473 | 0.369 | 0.495 | 0.400 | 0.464 | 0.627 | 0.593 | 0.511 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 507 | 507 | 507 | 506 | 507 | 502 | 507 | 506 | 506 | 503 | 506 | 507 | 507 | 507 | 507 | 500 |
| DEPS6 I feel that it's difficult to lose weight and control my Diabetes at the same time | Pearson Correlation | 0.606 | 0.294 | 0.340 | 0.399 | 0.483 | 1 | 0.433 | 0.202 | 0.368 | 0.282 | 0.490 | 0.352 | 0.345 | 0.538 | 0.487 | 0.456 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 504 | 504 | 503 | 502 | 502 | 504 | 504 | 503 | 503 | 500 | 503 | 503 | 504 | 504 | 503 | 498 |
| DEPS7 I avoid checking my blood sugar when I feel like it is out of range | Pearson Correlation | 0.322 | 0.370 | 0.436 | 0.618 | 0.454 | 0.433 | 1 | 0.361 | 0.605 | 0.529 | 0.520 | 0.562 | 0.651 | 0.570 | 0.591 | 0.582 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 509 | 510 | 509 | 508 | 507 | 504 | 510 | 509 | 509 | 506 | 509 | 509 | 510 | 509 | 509 | 502 |
| DEPS8 I make myself vomit | Pearson Correlation | 0.232 | 0.336 | 0.404 | 0.323 | 0.301 | 0.202 | 0.361 | 1 | 0.470 | 0.434 | 0.419 | 0.294 | 0.412 | 0.406 | 0.393 | 0.454 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 508 | 509 | 508 | 507 | 506 | 503 | 509 | 509 | 508 | 505 | 508 | 508 | 509 | 508 | 508 | 501 |
| DEPS9 I try to keep my blood sugar high | Pearson Correlation | 0.322 | 0.448 | 0.492 | 0.665 | 0.473 | 0.368 | 0.605 | 0.470 | 1 | 0.820 | 0.661 | 0.582 | 0.861 | 0.606 | 0.585 | 0.737 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 508 | 509 | 508 | 507 | 506 | 503 | 509 | 508 | 509 | 505 | 508 | 508 | 509 | 508 | 508 | 502 |
| DEPS10 I try to eat | Pearson Correlation | 0.282 | 0.391 | 0.449 | 0.569 | 0.369 | 0.282 | 0.529 | 0.434 | 0.820 | 1 | 0.569 | 0.479 | 0.831 | 0.539 | 0.481 | 0.613 |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| to the point of spilling ketones in my urine | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 505 | 506 | 505 | 504 | 503 | 500 | 506 | 505 | 505 | 506 | 505 | 505 | 506 | 505 | 505 | 498 |
| DEPS11 I feel fat when I take all of my insulin | Pearson Correlation | 0.438 | 0.420 | 0.486 | 0.577 | 0.495 | 0.490 | 0.520 | 0.419 | 0.661 | 0.569 | 1 | 0.528 | 0.655 | 0.625 | 0.607 | 0.663 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 508 | 509 | 508 | 507 | 506 | 503 | 509 | 508 | 508 | 505 | 509 | 508 | 509 | 508 | 508 | 502 |
| DEPS12 Other people tell me to take better care of my Diabetes | Pearson Correlation | 0.239 | 0.347 | 0.515 | 0.580 | 0.400 | 0.352 | 0.562 | 0.294 | 0.582 | 0.479 | 0.528 | 1 | 0.596 | 0.515 | 0.535 | 0.564 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 508 | 509 | 509 | 508 | 507 | 503 | 509 | 508 | 508 | 505 | 508 | 509 | 509 | 508 | 509 | 501 |
| DEPS13 After I overeat, I skip my next insulin dose | Pearson Correlation | 0.309 | 0.417 | 0.489 | 0.679 | 0.464 | 0.345 | 0.651 | 0.412 | 0.861 | 0.831 | 0.655 | 0.596 | 1 | 0.623 | 0.577 | 0.722 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 |
| | N | 509 | 510 | 509 | 508 | 507 | 504 | 510 | 509 | 509 | 506 | 509 | 509 | 510 | 509 | 509 | 502 |
| DEPS14 I feel that my eating is out of control | Pearson Correlation | 0.523 | 0.347 | 0.603 | 0.600 | 0.627 | 0.538 | 0.570 | 0.406 | 0.606 | 0.539 | 0.625 | 0.515 | 0.623 | 1 | 0.698 | 0.639 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 |
| | N | 509 | 509 | 508 | 507 | 507 | 504 | 509 | 508 | 508 | 505 | 508 | 508 | 509 | 509 | 508 | 502 |
| DEPS15 I alternate between eating very little and eating huge amounts | Pearson Correlation | 0.445 | 0.536 | 0.507 | 0.548 | 0.593 | 0.487 | 0.591 | 0.393 | 0.585 | 0.481 | 0.607 | 0.535 | 0.577 | 0.698 | 1 | 0.631 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 |
| | N | 508 | 509 | 509 | 508 | 507 | 503 | 509 | 508 | 508 | 505 | 508 | 509 | 509 | 508 | 509 | 501 |
| DEPS16 I would rather be thin than | Pearson Correlation | 0.448 | 0.471 | 0.527 | 0.606 | 0.511 | 0.456 | 0.582 | 0.454 | 0.737 | 0.613 | 0.663 | 0.564 | 0.722 | 0.639 | 0.631 | 1 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | |

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| to have good control of my Diabetes | N | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 502 | 502 | 501 | 501 | 500 | 498 | 502 | 501 | 502 | 498 | 502 | 501 | 502 | 502 | 501 | 502 | | | |

**Appendix M: Full Correlation Matrix for SEM**

| | | Age | Age at Dx | DiabetesDuration | HbA1c% | DEPS-R | GADT | FAMCH | FAMAT | REST | DDSEBT | DDSPRDT | DDSRRDT | DDSIDT | DDSTOTAL | APSDISC | CFC | ZPDT | APSSTD | APSORD | CEDST |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Age | Pearson Correlation | 1 | 0.485 | 0.534 | -0.180 | -0.318 | -0.290 | 0.025 | 0.075 | 0.286 | -0.307 | -0.056 | -0.347 | -0.194 | -0.288 | -0.206 | 0.143 | 0.312 | -0.003 | 0.105 | -0.241 |
| | Sig. (2-tailed) | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.593 | 0.128 | 0.000 | 0.000 | 0.215 | 0.000 | 0.000 | 0.000 | 0.000 | 0.002 | 0.000 | 0.941 | 0.019 | 0.000 |
| | N | 686 | 686 | 686 | 658 | 490 | 512 | 471 | 411 | 488 | 504 | 498 | 504 | 503 | 494 | 495 | 480 | 468 | 496 | 500 | 505 |
| Age at Dx | Pearson Correlation | 0.485 | 1 | -0.480 | -0.058 | -0.158 | -0.131 | 0.024 | 0.079 | 0.138 | -0.132 | -0.038 | -0.157 | -0.083 | -0.122 | -0.117 | 0.056 | -0.207 | -0.054 | -0.003 | -0.150 |
| | Sig. (2-tailed) | 0.000 | | 0.000 | 0.136 | 0.000 | 0.003 | 0.610 | 0.111 | 0.002 | 0.003 | 0.400 | 0.000 | 0.064 | 0.007 | 0.009 | 0.219 | 0.000 | 0.227 | 0.949 | 0.001 |
| | N | 686 | 686 | 686 | 658 | 490 | 512 | 471 | 411 | 488 | 504 | 498 | 504 | 503 | 494 | 495 | 480 | 468 | 496 | 500 | 505 |
| DiabetesDuration | Pearson Correlation | 0.534 | -0.480 | 1 | -0.125 | -0.166 | -0.167 | 0.002 | 0.001 | 0.153 | -0.181 | -0.020 | -0.197 | -0.115 | -0.171 | -0.092 | 0.088 | -0.109 | 0.049 | 0.107 | -0.097 |
| | Sig. (2-tailed) | 0.000 | 0.000 | | 0.001 | 0.000 | 0.000 | 0.962 | 0.988 | 0.001 | 0.000 | 0.658 | 0.000 | 0.010 | 0.000 | 0.041 | 0.054 | 0.018 | 0.278 | 0.017 | 0.030 |
| | N | 686 | 686 | 686 | 658 | 490 | 512 | 471 | 411 | 488 | 504 | 498 | 504 | 503 | 494 | 495 | 480 | 468 | 496 | 500 | 505 |
| HbA1c% | Pearson Correlation | -0.180 | -0.058 | -0.125 | 1 | 0.543 | 0.322 | -0.082 | 0.067 | -0.353 | 0.376 | 0.213 | 0.494 | 0.263 | 0.435 | 0.262 | -0.285 | 0.350 | -0.106 | -0.018 | 0.327 |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sig. (2-tailed) | 0.000 | 0.136 | 0.001 | | 0.000 | 0.000 | 0.080 | 0.183 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.020 | 0.690 | 0.000 |
| | N | 658 | 658 | 658 | 659 | 476 | 495 | 455 | 399 | 469 | 484 | 479 | 485 | 483 | 476 | 478 | 464 | 452 | 479 | 483 | 487 |
| DEPS-R | Pearson Correlation | -0.318 | -0.158 | -0.166 | 0.543 | 1 | 0.485 | -0.205 | 0.059 | -0.604 | 0.559 | 0.291 | 0.737 | 0.418 | 0.650 | 0.508 | -0.445 | 0.562 | -0.030 | -0.056 | 0.513 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.260 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.519 | 0.239 | 0.000 |
| | N | 490 | 490 | 490 | 476 | 491 | 461 | 425 | 371 | 437 | 460 | 453 | 459 | 459 | 451 | 450 | 435 | 421 | 452 | 452 | 460 |
| GADT | Pearson Correlation | -0.290 | -0.131 | -0.167 | 0.322 | 0.485 | 1 | -0.139 | 0.027 | -0.615 | 0.617 | 0.390 | 0.527 | 0.456 | 0.630 | 0.587 | -0.224 | 0.618 | -0.017 | -0.015 | 0.714 |
| | Sig. (2-tailed) | 0.000 | 0.003 | 0.000 | 0.000 | 0.000 | | 0.004 | 0.609 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.716 | 0.753 | 0.000 |
| | N | 512 | 512 | 512 | 495 | 461 | 512 | 431 | 375 | 450 | 471 | 464 | 470 | 470 | 462 | 462 | 449 | 434 | 465 | 466 | 480 |
| FAMCH | Pearson Correlation | 0.025 | 0.024 | 0.002 | -0.082 | -0.205 | -0.139 | 1 | 0.412 | 0.291 | -0.039 | -0.145 | -0.137 | -0.166 | -0.143 | -0.167 | 0.243 | -0.253 | 0.083 | 0.129 | -0.260 |
| | Sig. (2-tailed) | 0.593 | 0.610 | 0.962 | 0.080 | 0.000 | 0.004 | | 0.000 | 0.000 | 0.415 | 0.003 | 0.004 | 0.000 | 0.003 | 0.000 | 0.000 | 0.000 | 0.087 | 0.007 | 0.000 |
| | N | 471 | 471 | 471 | 455 | 425 | 431 | 471 | 405 | 422 | 436 | 431 | 436 | 435 | 428 | 430 | 419 | 406 | 431 | 434 | 429 |
| FAMAT | Pearson Correlation | 0.075 | 0.079 | 0.001 | 0.067 | -0.059 | 0.027 | 0.412 | 1 | 0.057 | -0.043 | 0.079 | -0.015 | -0.070 | -0.030 | -0.070 | 0.130 | -0.081 | 0.034 | 0.006 | -0.096 |
| | Sig. (2-tailed) | 0.128 | 0.111 | 0.988 | 0.183 | 0.260 | 0.609 | 0.000 | | 0.274 | 0.411 | 0.128 | 0.766 | 0.179 | 0.559 | 0.180 | 0.013 | 0.131 | 0.517 | 0.906 | 0.064 |
| | N | 411 | 411 | 411 | 399 | 371 | 375 | 405 | 411 | 365 | 376 | 373 | 376 | 375 | 370 | 372 | 365 | 352 | 373 | 375 | 371 |
| REST | Pearson Correlation | 0.286 | 0.138 | 0.153 | -0.353 | -0.604 | -0.615 | 0.291 | 0.057 | 1 | -0.584 | -0.310 | -0.581 | -0.434 | -0.615 | -0.729 | 0.325 | -0.644 | 0.146 | 0.103 | -0.754 |
| | Sig. (2-tailed) | 0.000 | 0.002 | 0.001 | 0.000 | 0.000 | 0.000 | 0.000 | 0.274 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.002 | 0.029 | 0.000 |
| | N | 488 | 488 | 488 | 469 | 437 | 450 | 422 | 365 | 488 | 447 | 442 | 447 | 447 | 440 | 443 | 428 | 415 | 445 | 447 | 446 |
| DDSEBT | Pearson Correlation | -0.307 | -0.132 | -0.181 | 0.376 | 0.559 | 0.617 | -0.039 | 0.043 | -0.584 | 1 | 0.448 | 0.711 | 0.618 | 0.888 | 0.545 | -0.201 | 0.545 | -0.026 | 0.006 | 0.629 |
| | Sig. (2-tailed) | 0.000 | 0.003 | 0.000 | 0.000 | 0.000 | 0.000 | 0.415 | 0.411 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.573 | 0.899 | 0.000 |
| | N | 504 | 504 | 504 | 484 | 460 | 471 | 436 | 376 | 447 | 504 | 496 | 503 | 503 | 494 | 460 | 445 | 433 | 463 | 465 | 467 |
| DDSPRDT | Pearson Correlation | -0.056 | -0.038 | -0.020 | 0.213 | 0.291 | 0.390 | -0.145 | 0.079 | -0.310 | 0.448 | 1 | 0.436 | 0.419 | 0.686 | 0.344 | -0.094 | 0.375 | 0.026 | 0.025 | 0.360 |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sig. (2-tailed) | 0.215 | 0.400 | 0.658 | 0.000 | 0.000 | 0.000 | 0.003 | 0.128 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.050 | 0.000 | 0.586 | 0.589 | 0.000 |
| | N | 498 | 498 | 498 | 479 | 453 | 464 | 431 | 373 | 442 | 496 | 498 | 496 | 495 | 494 | 454 | 439 | 427 | 456 | 458 | 460 |
| DDSRRDT | Pearson Correlation | -0.347 | -0.157 | -0.197 | 0.494 | 0.737 | 0.527 | 0.137 | 0.015 | -0.581 | 0.711 | 0.436 | 1 | 0.524 | 0.862 | 0.510 | -0.381 | 0.569 | -0.092 | -0.040 | 0.522 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.004 | 0.766 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.048 | 0.389 | 0.000 |
| | N | 504 | 504 | 504 | 485 | 459 | 470 | 436 | 376 | 447 | 503 | 496 | 504 | 502 | 494 | 459 | 445 | 432 | 462 | 464 | 466 |
| DDSIDT | Pearson Correlation | -0.194 | -0.083 | -0.115 | 0.263 | 0.418 | 0.456 | -0.166 | -0.070 | -0.434 | 0.618 | 0.419 | 0.524 | 1 | 0.756 | 0.385 | -0.189 | 0.473 | 0.017 | 0.017 | 0.497 |
| | Sig. (2-tailed) | 0.000 | 0.064 | 0.010 | 0.000 | 0.000 | 0.000 | 0.000 | 0.179 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 | 0.721 | 0.721 | 0.000 |
| | N | 503 | 503 | 503 | 483 | 459 | 470 | 435 | 375 | 447 | 503 | 495 | 502 | 503 | 494 | 459 | 444 | 433 | 462 | 464 | 466 |
| DDSTOTAL | Pearson Correlation | -0.288 | -0.122 | -0.171 | 0.435 | 0.650 | 0.630 | -0.143 | -0.030 | -0.615 | 0.888 | 0.686 | 0.862 | 0.756 | 1 | 0.567 | -0.290 | 0.617 | -0.035 | -0.001 | 0.632 |
| | Sig. (2-tailed) | 0.000 | 0.007 | 0.000 | 0.000 | 0.000 | 0.000 | 0.003 | 0.559 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.463 | 0.979 | 0.000 |
| | N | 494 | 494 | 494 | 476 | 451 | 462 | 428 | 370 | 440 | 494 | 494 | 494 | 494 | 494 | 452 | 438 | 426 | 454 | 456 | 458 |
| APSDISC | Pearson Correlation | -0.206 | -0.117 | -0.092 | 0.262 | 0.508 | 0.587 | -0.167 | -0.070 | -0.729 | 0.545 | 0.344 | 0.510 | 0.385 | 0.567 | 1 | -0.247 | 0.524 | 0.130 | 0.077 | 0.617 |
| | Sig. (2-tailed) | 0.000 | 0.009 | 0.041 | 0.000 | 0.000 | 0.000 | 0.000 | 0.180 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.004 | 0.087 | 0.000 |
| | N | 495 | 495 | 495 | 478 | 450 | 462 | 430 | 372 | 443 | 460 | 454 | 459 | 459 | 452 | 496 | 438 | 429 | 491 | 494 | 465 |
| CFC | Pearson Correlation | 0.143 | 0.056 | 0.088 | -0.285 | -0.445 | -0.224 | 0.243 | 0.130 | 0.325 | -0.201 | -0.094 | -0.381 | -0.189 | -0.290 | -0.247 | 1 | -0.346 | 0.239 | 0.189 | -0.252 |
| | Sig. (2-tailed) | 0.002 | 0.219 | 0.054 | 0.000 | 0.000 | 0.000 | 0.000 | 0.013 | 0.000 | 0.000 | 0.050 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 |
| | N | 480 | 480 | 480 | 464 | 435 | 449 | 419 | 365 | 428 | 445 | 439 | 445 | 444 | 438 | 438 | 480 | 413 | 440 | 440 | 448 |
| ZPDT | Pearson Correlation | -0.312 | -0.207 | -0.109 | 0.350 | 0.562 | 0.618 | -0.253 | 0.081 | -0.644 | 0.545 | 0.375 | 0.569 | 0.473 | 0.617 | 0.524 | -0.346 | 1 | -0.072 | -0.069 | 0.628 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.018 | 0.000 | 0.000 | 0.000 | 0.000 | 0.131 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | | 0.134 | 0.149 | 0.000 |
| | N | 468 | 468 | 468 | 452 | 421 | 434 | 406 | 352 | 415 | 433 | 427 | 432 | 433 | 426 | 429 | 413 | 468 | 432 | 433 | 436 |
| APSSTD | Pearson Correlation | -0.003 | -0.054 | 0.049 | -0.106 | -0.030 | -0.017 | 0.083 | 0.034 | 0.146 | -0.026 | 0.026 | -0.092 | 0.017 | -0.035 | 0.130 | 0.239 | -0.072 | 1 | 0.500 | -0.073 |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sig. (2-tailed) | 0.941 | 0.227 | 0.278 | 0.020 | 0.519 | 0.716 | 0.087 | 0.517 | 0.002 | 0.573 | 0.586 | 0.048 | 0.721 | 0.463 | 0.004 | 0.000 | 0.134 | | 0.000 | 0.117 |
| | N | 496 | 496 | 496 | 479 | 452 | 465 | 431 | 373 | 445 | 463 | 456 | 462 | 462 | 454 | 491 | 440 | 432 | 497 | 494 | 465 |
| APSORD | Pearson Correlation | 0.105 | -0.003 | 0.107 | -0.018 | -0.056 | -0.015 | 0.129 | 0.006 | 0.103 | 0.006 | 0.025 | -0.040 | 0.017 | -0.001 | 0.077 | 0.189 | -0.069 | 0.500 | 1 | -0.108 |
| | Sig. (2-tailed) | 0.019 | 0.949 | 0.017 | 0.690 | 0.239 | 0.753 | 0.007 | 0.906 | 0.029 | 0.899 | 0.589 | 0.389 | 0.721 | 0.979 | 0.087 | 0.000 | 0.149 | 0.000 | | 0.020 |
| | N | 500 | 500 | 500 | 483 | 452 | 466 | 434 | 375 | 447 | 465 | 458 | 464 | 464 | 456 | 494 | 440 | 433 | 494 | 501 | 466 |
| CEDST | Pearson Correlation | -0.241 | -0.150 | -0.097 | 0.327 | 0.513 | 0.714 | -0.260 | 0.096 | -0.754 | 0.629 | 0.360 | 0.522 | 0.497 | 0.632 | 0.617 | -0.252 | 0.628 | -0.073 | -0.108 | 1 |
| | Sig. (2-tailed) | 0.000 | 0.001 | 0.030 | 0.000 | 0.000 | 0.000 | 0.000 | 0.064 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.117 | 0.020 | |
| | N | 505 | 505 | 505 | 487 | 460 | 480 | 429 | 371 | 446 | 467 | 460 | 466 | 466 | 458 | 465 | 448 | 436 | 465 | 466 | 505 |

**Appendix N: The Factors in Eating Disorder Questionnaire**

*Factors in Eating Disorders questionnaire*

This questionnaire has 2 parts. In part 1 you will be asked some basic questions about yourself, if at any point you do not want to give an answer please select the rather not say option. If at any point you would like to withdraw from the study you can just close your browser.

Part 1:

How old are you?

What is you Gender?

What nationality are you?

What ethnicity are you?

What age were you when you were diagnosed with Type 1 Diabetes?

What was your last HbA1c%?

How long after your Type 1 Diagnosis did you develop an Eating Disorder?

Have you ever been formally diagnosed with an Eating Disorder by a Health Care Professional?

What Eating Disorder(s) were you diagnosed with?

What Eating Disorder(s) do you think you had/ have?

Have you ever omitted insulin for weight loss purposes?

Have you sought help from an Health Care Professional regarding your Eating Disorder?

What Health Care Professionals did you approach?

Have you been diagnosed by a Health Care Professional with any other mental illness?

Have you been the victim of weight related bullying?

Have you been the victim of T1D related bullying?

Part 2

Directions: The following statements concern your perception of the environment you were in at the time of developing an Eating Disorder. Please indicate how strongly you agree with each statement by using the below judgments. In the boxes after each statement, click a number from 1 to 5 from the following scale:

1) Strongly disagree

2) Disagree

3) Neither disagree nor agree

4) Agree

5) Strongly agree

Please note that there are no right or wrong answers. Take your time and consider each statement carefully. Once you have completed all questions please select submit at the bottom of the page. If at any point you do not wish to continue please just close your browser.

In the second section you are asked to rate if you feel certain statements and factors contributed to the development of your Eating Disorder, or if they did not. In some questions you are asked if a particular factor **did** contribute to the development of your Eating Disorder, for example:

   a)   Pressure to be good at sports **contributed** to the development of my Eating Disorder

If you don't agree with this statement then you would select 1) Strongly Disagree or 2) Disagree, if you feel neutral about this particular issue you would select 3) Neither Agree or Disagree, if however you felt that it did contribute you would select 4) Agree and if you felt that this played a significant part in the development you would select 5) Strongly Agree

You will notice that some of the questions are worded differently to this, the reason they are is to avoid a phenomenon called response bias, which is where a participant may get into a habit of selecting responses. For this reason you should consider these questions carefully before responding, for example

   b)   Sibling Rivalry **did not contribute** to the development of my Eating Disorder

In this case you may feel that in fact sibling rivalry **did** play a part in the development of your Eating Disorder so in this case you would 2) disagree or, 1) strongly disagree. You may feel neutral about sibling rivalry so in that case you would select 3) Neither agree or disagree or you may feel like it did not contribute, in which case you would either 4) agree or 5) strongly agree with the statement

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1) Strongly Disagree  2) Disagree     3)Neither Disagree or Agree  4) Agree        5)Strongly  Agree

1) I needed to feel in control of something and this contributed to the development of my Eating Disorder

2) Initial weight loss at diagnosis contributed to the development of my Eating Disorder

3) A restrained diet contributed to the development of my Eating Disorder

4) Weight gain after I started on Insulin contributed to the development of my Eating Disorder

5) Early Puberty contributed to the development of my Eating Disorder

6) Low self-esteem contributed to the development of my Eating Disorder

7) Too much focus on my diet contributed to the development of my Eating Disorder

8) Attention to my weight from Medical Staff did not contribute to the development of my Eating Disorder

9) Regardless of my Diabetes I was unhappy I was gaining weight and this contributed to the development of my Eating Disorder

10) Adherence to timed meals contributed to the development of my Eating Disorder

11) Conflict at home within my family contributed to the development of my Eating Disorder

12) The realisation I could lose weight quickly contributed to the development of my Eating Disorder

13) General Diabetes stress contributed to the development of my Eating Disorder

14)  Fear of weight gain contribute to the development of my Eating Disorder

15) A perfectionist attitude towards my blood sugar did not contribute to the development of my Eating Disorder

16) Denial that I had Diabetes contributed to the development of my Eating Disorder

17)   Academic Pressure did not contribute to the development of my Eating Disorder

18) A sense of achievement at losing weight contributed to the development of my Eating Disorder

19) Conflict among my peer group contributed to the development of my Eating Disorder

20) Fear of hypoglycaemia contributed to the development of my Eating Disorder

21) Embarrassment at injecting or testing in front of anyone contributed to the development of my Eating Disorder

22) Diabetes Burn Out contributed to the development of my Eating Disorder

23) An over – emphasis on what would happen if I didn't look after myself contributed to the development of my Eating Disorder

24)  Fear of injecting and or self-testing contributed to the development of my Eating Disorder

25) Societal pressure to be thinner contributed to the development of my Eating Disorder

26) Examples of disturbed eating in my family contributed to the development of my Eating Disorder

27) Overeating/ binging, following episodes of hypoglycaemia contributed to the development of my Eating Disorder

28) Pressure from my health care professionals contributed to the development of my Eating Disorder

29) Maternal concern with weight and shape did not contribute to the development of my Eating Disorder

30) A distorted body image contributed to the development of my Eating Disorder

31) Resentment of having Diabetes contributed to the development of my Eating Disorder

32) An Eating Disorder prior to developing Type 1 Diabetes contributed to the development of my Eating Disorder

33) Disagreement with the way my parents dealt with my Diabetes contributed to the development of my Eating Disorder

34) I felt I couldn't talk to my parents and this contributed to the development of my Eating Disorder

35) Family dysfunction contributed to the development of my Eating Disorder

36) Feeling that I was not an individual within my family contributed to the development of my Eating Disorder

37) I had pre-existing Mental Health issues and this contributed to the development of my Eating Disorder

38) I didn't like the way my body looked and this contributed to the development of my Eating Disorder

39) My family was very rigid and this contributed to the development of my Eating Disorder

40) A disturbed attitude to food did not contribute to the development of my Eating Disorder

**Appendix O: Recruitment Advert for the Confirmatory Sample**

Do you have Type 1 Diabetes? Have you ever experienced and Eating Disorder or Diabulimia? Do you have some time to contribute to research on Type 1 Diabetes? My name is Jacqueline Allan and I am a Type 1 Diabetic currently undertaking a phd in research and my dissertation is on Eating Disorders and Diabulimia in Type 1 Diabetes. This questionnaire will discuss potentially sensitive subjects, such as relationship to food, Diabetes distress, family relations and psychological state. All you need to do is answer a questionnaire that you can find here. (www.typeoneandpsychology.org.uk) This system is entirely computerised however so no-one including the researcher will know or keep your email after the study is completed. There is absolutely no identifying information required so your responses will remain completely anonymous. You are also free to withdraw your participation at any time.

# Appendix P: Correlation Matrix – FEDS questionnaire

| | 1 | 3 | 4 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 18 | 20 | 21 | 22 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 33 | 34 | 35 | 36 | 38 | 39 | 40 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1) needed to feel in control | 1.000 | .172* | 0.132 | .471* | .310* | 0.072 | .305* | 0.127 | .321* | .330* | .352* | .240* | .243* | 0.157 | .538* | 0.124 | .204* | .204* | 0.059 | .445* | .296* | .184* | .340* | .306* | .271* | 0.087 | 0.151 | .369* | .334* | .239* | .515* | .219* | 0.093 |
| 3) restrained diet | .172* | 1.000 | -0.049 | .301* | .641* | .318* | .338* | .532* | .249* | .191* | .370* | 0.082 | .375* | .199* | .203* | .347* | .197* | .404* | .235* | .294* | .349* | .410* | .502* | .297* | 0.032 | .211* | .343* | .243* | .306* | .222* | .292* | .416* | .271* |
| 4) Weight gain after I started on Insulin | 0.132 | -0.049 | 1.000 | 0.095 | 0.040 | .222* | 0.142 | 0.101 | 0.119 | .333* | .333* | .395* | 0.105 | .259* | .306* | 0.161 | 0.114 | 0.123 | 0.102 | .256* | 0.098 | .227* | 0.170 | 0.064 | .294* | .286* | 0.108 | 0.085 | 0.149 | .176* | .307* | 0.121 | .246* |
| 6) Low self-esteem | .471* | .301* | 0.095 | 1.000 | .538* | 0.110 | .556* | .214* | .341* | .352* | .394* | .279* | .228* | .290* | .567* | 0.152 | .183* | .269* | 0.139 | .621* | .266* | .294* | .372* | .306* | .224* | 0.101 | 0.120 | .284* | .317* | .293* | .682* | 0.129 | 0.157 |
| 7) Too much focus on my diet | .310* | .641* | 0.040 | .538* | 1.000 | .354* | .395* | .541* | .261* | .174* | .531* | .185* | .359* | .268* | .312* | .315* | .199* | .501* | 0.092 | .427* | .393* | .490* | .618* | .302* | -0.016 | .215* | .296* | .347* | .243* | .324* | .470* | .322* | .262* |
| 8) Attention to my weight from Medical Staff | 0.072 | .318* | .222* | 0.110 | .354* | 1.000 | .188* | .229* | .180* | 0.014 | .290* | .270* | .322* | 0.130 | -0.005 | 0.100 | .241* | .282* | 0.036 | 0.143 | .233* | .264* | .527* | .198* | 0.130 | .285* | 0.167 | .279* | .211* | 0.111 | 0.140 | .286* | .373* |
| 9) Regardless of my Diabetes I was unhappy I was gaining weight | .305* | .338* | 0.142 | .556* | .395* | .188* | 1.000 | .227* | .245* | .342* | .402* | .320* | .209* | .206* | .537* | .274* | 0.129 | .394* | 0.141 | .632* | 0.166 | .329* | .290* | 0.170 | 0.153 | .181* | 0.096 | 0.162 | .183* | 0.165 | .665* | .189* | 0.151 |
| 10) Adherence to timed meals | 0.127 | .532* | 0.101 | .214* | .541* | .229* | .227* | 1.000 | .338* | 0.069 | .383* | 0.063 | .401* | 0.158 | .173* | .324* | .274* | .293* | .237* | 0.149 | .285* | .338* | .429* | 0.111 | -0.043 | .236* | .431* | .270* | .337* | .343* | .228* | .375* | .238* |
| 11) Conflict at home within my family | .321* | .249* | 0.119 | .341* | .261* | .180* | .245* | .338* | 1.000 | .194* | .284* | 0.078 | .252* | .318* | .284* | .259* | .335* | .272* | .236* | .319* | .493* | .212* | .369* | .427* | 0.120 | .185* | .472* | .588* | .844* | .577* | .268* | .478* | 0.029 |
| 12) realisation I could lose weight quickly | .330* | .191* | .333* | .352* | .174* | 0.014 | .342* | 0.069 | .194* | 1.000 | .397* | .211* | 0.074 | .376* | .570* | .273* | .278* | .308* | .224* | .408* | 0.067 | 0.152 | 0.146 | .186* | .297* | .253* | .176* | 0.087 | .236* | 0.092 | .510* | 0.146 | -0.042 |
| 13) General Diabetes stress | .352* | .370* | .333* | .394* | .531* | .290* | .402* | .383* | .284* | .397* | 1.000 | .209* | .320* | .525* | .432* | .412* | .361* | .633* | .246* | .439* | .287* | .569* | .529* | .185* | 0.083 | .524* | .351* | .184* | .289* | .204* | .453* | .304* | .355* |
| 14) Fear of weight gain | .240* | 0.082 | .395* | .279* | .185* | .270* | .320* | 0.063 | 0.078 | .211* | .209* | 1.000 | -0.052 | 0.040 | .349* | 0.017 | 0.055 | 0.141 | 0.009 | .468* | 0.171 | 0.108 | .197* | .200* | .413* | .189* | 0.080 | 0.094 | 0.079 | 0.144 | .434* | 0.067 | .429* |
| 15) perfectionist attitude towards my blood sugar | .243* | .375* | 0.105 | .228* | .359* | .322* | .209* | .401* | .252* | 0.074 | .320* | -0.052 | 1.000 | 0.150 | 0.113 | .268* | 0.134 | .281* | 0.089 | .190* | .326* | .328* | .430* | .191* | -0.020 | 0.168 | .186* | .263* | .285* | .182* | .240* | .330* | 0.130 |
| 16) Denial that I had Diabetes | 0.157 | .199* | .259* | .290* | .268* | 0.130 | .206* | 0.158 | .318* | .376* | .525* | 0.040 | 0.150 | 1.000 | .320* | .436* | .559* | .379* | .482* | .240* | .192* | .329* | .208* | 0.152 | 0.103 | .451* | .311* | .245* | .347* | .267* | .265* | .275* | 0.118 |
| 18) A sense of achievement at losing weight | .538* | .203* | .306* | .567* | .312* | -0.005 | .537* | .173* | .284* | .570* | .432* | .349* | 0.113 | .320* | 1.000 | .208* | .200* | .239* | .190* | .641* | .189* | .176* | .247* | .313* | .341* | 0.101 | .218* | .199* | .268* | .253* | .772* | .179* | 0.130 |
| 20) Fear of hypoglycaemia | 0.124 | .347* | 0.161 | 0.152 | .315* | 0.100 | .274* | .324* | .259* | .273* | .412* | 0.017 | .268* | .436* | .208* | 1.000 | .460* | .353* | .379* | .239* | .302* | .468* | .331* | 0.133 | 0.028 | .380* | .311* | .196* | .314* | .223* | .212* | .223* | 0.123 |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 21) Embarrassment at injecting or testing | .204* | .197* | 0.114 | .183* | .199* | .241* | 0.129 | .274* | .335* | .278* | .361* | 0.055 | 0.134 | .559* | .200* | .460* | 1.000 | .381* | .576* | .189* | .237* | .280* | .293* | .247* | 0.030 | .328* | .336* | .359* | .389* | .360* | .173* | .279* | 0.038 |
| 22) Diabetes Burn Out | .204* | .404* | 0.123 | .269* | .501* | .282* | .394* | .293* | .272* | .308* | .633* | 0.141 | .281* | .379* | .239* | .353* | .381* | 1.000 | .243* | .325* | .339* | .516* | .458* | .306* | -0.063 | .524* | .271* | .255* | .252* | 0.166 | .316* | .316* | .239* |
| 24) Fear of injecting and or self-testing | 0.059 | .235* | 0.102 | 0.139 | 0.092 | 0.036 | 0.141 | .237* | .236* | .224* | .246* | 0.009 | 0.089 | .482* | .190* | .379* | .576* | .243* | 1.000 | 0.154 | .189* | 0.146 | 0.112 | 0.146 | -0.158 | .256* | .297* | .182* | .250* | .194* | 0.127 | .176* | 0.084 |
| 25) Societal pressure to be thinner | .445* | .294* | .256* | .621* | .427* | 0.143 | .632* | 0.149 | .319* | .408* | .439* | .468* | .190* | .240* | .641* | .239* | .189* | .325* | 0.154 | 1.000 | .310* | .290* | .382* | .392* | .245* | 0.136 | 0.151 | .220* | .230* | .206* | .778* | .207* | 0.163 |
| 26) Examples of disturbed eating in my family | .296* | .349* | 0.098 | .266* | .393* | .233* | 0.166 | .285* | .493* | 0.067 | .287* | 0.171 | .326* | .192* | .189* | .302* | .237* | .339* | .189* | .310* | 1.000 | .297* | .353* | .544* | 0.010 | 0.138 | .333* | .368* | .474* | .446* | .259* | .487* | .212* |
| 27) Overeating/ binging, following episodes of hypoglycaemia | .184* | .410* | .227* | .294* | .490* | .264* | .329* | .338* | .212* | 0.152 | .569* | 0.108 | .328* | .329* | .176* | .468* | .280* | .516* | 0.146 | .290* | .297* | 1.000 | .472* | .193* | -0.088 | .416* | .291* | .188* | .222* | .220* | .293* | .236* | .265* |
| 28) Pressure from my health care professionals | .340* | .502* | 0.170 | .372* | .618* | .527* | .290* | .429* | .369* | 0.146 | .529* | .197* | .430* | .208* | .247* | .331* | .293* | .458* | 0.112 | .382* | .353* | .472* | 1.000 | .287* | 0.081 | .305* | .314* | .387* | .360* | .305* | .381* | .344* | .298* |
| 29) Maternal concern with weight and shape | .306* | .297* | 0.064 | .306* | .302* | .198* | 0.170 | 0.111 | .427* | .186* | .185* | .200* | .191* | 0.152 | .313* | 0.133 | .247* | .306* | 0.146 | .392* | .544* | .193* | .287* | 1.000 | 0.086 | 0.010 | .285* | .501* | .438* | .452* | .295* | .555* | 0.154 |
| 30) A distorted body image | .271* | 0.032 | .294* | .224* | -0.016 | 0.130 | 0.153 | -0.043 | 0.120 | .297* | 0.083 | .413* | -0.020 | 0.103 | .341* | 0.028 | 0.030 | -0.063 | -0.158 | .245* | 0.010 | -0.088 | 0.081 | 0.086 | 1.000 | 0.091 | -0.002 | 0.061 | .195* | 0.036 | .361* | -0.008 | .201* |
| 31) Resentment of having Diabetes | 0.087 | .211* | .286* | 0.101 | .215* | .285* | .181* | .236* | .185* | .253* | .524* | .189* | 0.168 | .451* | 0.101 | .380* | .328* | .524* | .256* | 0.136 | 0.138 | .416* | .305* | 0.010 | 0.091 | 1.000 | .227* | 0.041 | .213* | 0.086 | 0.157 | 0.167 | .401* |
| 33) Disagreement with the way my parents dealt with my Diabetes | 0.151 | .343* | 0.108 | 0.120 | .296* | 0.167 | 0.096 | .431* | .472* | .176* | .351* | 0.080 | .186* | .311* | .218* | .311* | .336* | .271* | .297* | 0.151 | .333* | .291* | .314* | .285* | -0.002 | .227* | 1.000 | .562* | .546* | .506* | .173* | .586* | 0.133 |
| 34) I felt I couldn't talk to my parents | .369* | .243* | 0.085 | .284* | .347* | .279* | 0.162 | .270* | .588* | 0.087 | .184* | 0.094 | .263* | .245* | .199* | .196* | .359* | .255* | .182* | .220* | .368* | .188* | .387* | .501* | 0.061 | 0.041 | .562* | 1.000 | .699* | .693* | .223* | .586* | 0.094 |
| 35) Family dysfunction | .334* | .306* | 0.149 | .317* | .243* | .211* | .183* | .337* | .844* | .236* | .289* | 0.079 | .285* | .347* | .268* | .314* | .389* | .252* | .250* | .230* | .474* | .222* | .360* | .438* | .195* | .213* | .546* | .699* | 1.000 | .660* | .267* | .552* | 0.102 |
| 36) Feeling that I was not an individual within my family | .239* | .222* | .176* | .293* | .324* | 0.111 | 0.165 | .343* | .577* | 0.092 | .204* | 0.144 | .182* | .267* | .253* | .223* | .360* | 0.166 | .194* | .206* | .446* | .220* | .305* | .452* | 0.036 | 0.086 | .506* | .693* | .660* | 1.000 | .204* | .639* | 0.121 |
| 38 I didn't like the way my body looked | .515* | .292* | .307* | .682* | .470* | 0.140 | .665* | .228* | .268* | .510* | .453* | .434* | .240* | .265* | .772* | .212* | .173* | .316* | 0.127 | .778* | .259* | .293* | .381* | .295* | .361* | 0.157 | .173* | .223* | .267* | .204* | 1.000 | 0.131 | .212* |
| 39) My family was very rigid | .219* | .416* | 0.121 | 0.129 | .322* | .286* | .189* | .375* | .478* | 0.146 | .304* | 0.067 | .330* | .275* | .179* | .223* | .279* | .316* | .176* | .207* | .487* | .236* | .344* | .555* | -0.008 | 0.167 | .586* | .586* | .552* | .639* | 0.131 | 1.000 | .187* |
| 40) A disturbed attitude to food | 0.093 | .271* | .246* | 0.157 | .262* | .373* | 0.151 | .238* | 0.029 | -0.042 | .355* | .429* | 0.130 | 0.118 | 0.130 | 0.123 | 0.038 | .239* | 0.084 | 0.163 | .212* | .265* | .298* | 0.154 | .201* | .401* | 0.133 | 0.094 | 0.102 | 0.121 | .212* | .187* | 1.000 |

Pearson Correlation is significant at the 0.05 level

** Pearson Correlation is significant at the 0.01 level (2-tailed).