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Abstract

Aim: To provide an overview of the existing literature pertaining to insulin restriction as a disordered eating behaviour in individuals with type 1 diabetes and present a novel maintenance model: The Transdiagnostic Model of Disordered Eating in Type 1 Diabetes.

Method: A systematic review was conducted of the current literature relevant to insulin omission and/or restriction in the context of disordered eating in type 1 diabetes. A new maintenance model was then developed by incorporating diabetes-specific factors into existing eating disorder models.

Results: Type 1 diabetes may complicate the development and maintenance of disordered eating behaviour. Diabetes-specific circumstances, including disease diagnosis, insulin management, insulin restriction, and diabetes-related complications, contribute to the maintenance of disordered eating cognitions and behaviours.

Discussion: The proposed model offers a comprehensive representation of insulin restriction as a disordered eating behaviour in type 1 diabetes. Future research should test the model to further understand the mechanisms underlying disordered eating in type 1 diabetes and inform treatments for this at-risk population.

Keywords: type 1 diabetes, diabetes, eating disorder, disordered eating, insulin restriction, insulin omission
Disordered eating and insulin restriction in type 1 diabetes: A systematic review and testable model

Type 1 diabetes (T1D), or insulin-dependent diabetes, accounts for 10% of all diabetes cases, with most cases being diagnosed in childhood. Compared to individuals without diabetes, those with diabetes are at heightened risk for developing comorbid eating disorders (EDs) (Young et al., 2013), with approximately 30% of women and 20% of men with T1D diabetes reporting comorbid disordered eating behaviour (Doyle et al., 2017). This high prevalence is of grave concern given that EDs have the highest mortality rate of any mental illness (Arcelus, Mitchell, Wales, & Nelson, 2011). Insulin restriction is one particular disordered eating behaviour that is unique to individuals with T1D, however to date, there have been no comprehensive reviews in this area. The current paper reviews the literature pertaining to insulin restriction as a disordered eating behaviour in T1D and, building on previous models (Fairburn, Cooper, & Shafran, 2003; Goebel-Fabbri, Fikkan, Connell, Vangsness, & Anderson, 2002; Treasure et al., 2015), offers an explanatory model for the development and maintenance of disordered eating in T1D.

Mechanism of action of insulin restriction in T1D

Blood glucose, or blood sugar, comes from the food we eat, and is carried to all the cells in the body to supply energy. Insulin is the hormone that is responsible for transporting sugar in the bloodstream to our cells. In individuals with T1D, the body does not produce enough insulin, causing impaired glucose utilisation, increased blood glucose concentration, and excretion of glucose in the urine. As the body’s capacity both to store glucose and to use it as an energy source is reduced, the body must obtain energy from elsewhere and begins to break down stores
of fat and protein, causing weight loss (Frayn, 2010). Individuals with T1D are at risk of complications such as hypo- or hyper-glycaemia, conditions in which the glucose level in bloodstream is either dangerously low or dangerously high. Chronic poor glycaemic control is also associated with poor health outcomes including macro-vascular (e.g. cardiovascular disease), and micro-vascular (e.g. eye, kidney, and nerve damage) complications. As such, treatment for T1D typically requires the administration of insulin, and as glycemic control improves, weight gain is a common side effect (Jacob, Salinas, Adams-Huet, & Raskin, 2006). Weight concerns may then develop (Grilo, 2014).

Insulin restriction is the practice of purposefully underdosing or complete omission of the required insulin for secondary gain (Snyder et al., 2016), including for underdosing in order to purge calories via glucosuria (glucose excreted through the urine). Within the literature this phenomenon has been coined “insulin mismanagement”, “insulin omission” or “diabulimia” (Callum & Lewis, 2014). The term diabulimia can be inaccurate, as it only encompasses bulimic symptomatology, and is not sufficient in capturing individuals who have purging patterns without bingeing episodes. Furthermore, this tendency to conceptualise insulin misuse only in the context of BN, is reflected in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), which considers “other medication” in its definition of purging in BN, whereas this consideration of medication misuse is omitted from the definition of purging in AN binge-purge subtype (AN-BP) diagnostic criteria.

**Prevalence and course of disordered eating in T1D**

Disordered eating behaviours, defined as problematic eating behaviours and cognitions that are not practiced at a high enough frequency or severity to merit the formal diagnosis of an ED, are
an increasing problem for more than a quarter of people with T1D (Doyle et al., 2017; Treasure et al., 2015). Research has shown that adolescents with T1D experience more clinical EDs (Jones, Lawson, Daneman, Olmsted, & Rodin, 2000; Young et al., 2013) and higher rates of binge eating, excessive exercise, and subthreshold EDs (Jones et al., 2000; Young et al., 2013) compared to their peers. Furthermore, disordered eating has been associated with poorer glycaemic control in adolescents (Young et al., 2013) and in young adults (Doyle et al., 2017) with T1D.

EDs have been found to develop after diabetes diagnoses in participants with T1D but are thought to develop prior to diabetes diagnosis in participants with type 2 diabetes (Gagnon, Aimé, & Bélanger, 2017). Colton et al. (2015) conducted a longitudinal study to examine the prevalence of disordered eating and EDs in a sample of females with T1D. Mean age was 11.8 years at time 1 and 23.7 years at time 7. At time 7, 32.4% met the criteria for a current ED, and an additional 8.5% had a subthreshold ED. Mean age at ED onset was 22.6 years. With regard to ED subtypes in T1D, research (Mannucci et al., 2005) suggests that there is no increased prevalence for anorexia nervosa (AN) compared to controls, however there is an increase in prevalence for bulimia nervosa (BN) and eating disorder not otherwise specified (EDNOS) for individuals with T1D.

The current study

Despite the growing empirical evidence surrounding the phenomenon of insulin restriction as a disordered eating behaviour in T1D, a systematic review in the area is still lacking. The current study therefore aimed to systematically evaluate the current literature concerning insulin restriction as a disordered eating behaviour in individuals with type 1 or insulin-dependent
diabetes, in order to develop an empirically-based maintenance model of disordered eating in these populations.

**METHOD**

A search for relevant literature was conducted in May 2017 using the following electronic databases: PsycInfo, PsycArticles, and Medline (Ovid). The search strategy utilised the following search terms: (‘type 1 diabetes’ OR ‘insulin-dependent diabetes’) AND (‘eating disorder*’ OR ‘disordered eating’ OR ‘anorexi*’ OR ‘bulimi*’ OR ‘binge eating disorder’ OR ‘diabulimia’ OR ‘insulin mismanagement’ OR ‘insulin omission’). The initial database search identified 459 articles that were then screened for eligibility via their titles (see Figure 1). This resulted in the identification of 211 articles that were further screened for inclusion based on their abstract. From the abstract screening, 52 potentially relevant articles were further considered. These full-text articles were assessed for eligibility using the following inclusion criteria: (1) the study examined and measured insulin misuse (e.g. insulin restriction, insulin omission, insulin overdose) in the context of disordered eating (e.g. shape/weight concerns, drive dieting, binge eating) in a T1D or insulin-dependent diabetes population; (2) was a quantitative study (3) was in English language; and (4) the study was published in a peer-reviewed journal. Studies were excluded if they: (1) only measured disordered eating and not insulin restriction/omission or (2) only measured insulin restriction/omission without disordered eating pathology. The reference lists of all full-text articles were screened, however no other relevant studies were found. A total of 31 articles met all of the selection criteria for the current review.

---- Insert Figure 1 about here ----
RESULTS

Systematic evaluation of the empirical literature

**Insulin restriction/omission.** Of the papers included in the literature review, 22 studies investigated insulin restriction/omission for weight control, weight loss, and/or weight concerns; eight studies (Bachle et al., 2016; Baechle et al., 2014; Goebel-Fabbri et al., 2008; Goebel-Fabbri et al., 2011; Merwin et al., 2015; Schober et al., 2011; Snyder et al., 2016; Wisting et al., 2015) measured frequency of restriction/omission in the context of disordered eating; and one study (Wisting et al., 2013) focused specifically on insulin restriction/omission after overeating. Insulin restriction/omission was reported in all studies except in one female sample (Kichler et al., 2008) and two male samples (Bryden et al., 1999; Fairburn, Peveler, Davies, Mann, & Mayou, 1991). Prevalence of insulin restriction/omission ranged from to 4.1% (Herpertz et al., 1998) to 58% (Pinhas-Hamiel et al., 2013) in T1D populations (i.e. without comorbid ED), and from 47.9% (Powers et al., 2012) to 90% (Custal et al., 2014) in comorbid T1D/ED populations. Prevalence of insulin restriction/omission in T1D females without EDs ranged from 5.6% (Herpertz et al., 1998) to 58% (Pinhas-Hamiel et al., 2013) and males without EDs from 1.4% (Ackard et al., 2008) to 9.4% (Wisting et al., 2013).

**Prevalence of disordered eating.** The findings regarding disordered eating prevalence in the current review are mixed. Studies suggest that individuals with T1D are 2.4 times more likely to have an ED (Jones et al., 2000) and up to 4 times more likely than controls to present with disordered eating (Pinar, 2005). However, other studies reported no difference in disordered eating prevalence relative to the general population (Bachle et al., 2016; Birk & Spencer, 1989; Fairburn et al., 1991), and one study reported less disordered eating prevalence than controls.
(Ackard et al., 2008). Of the studies that looked at gender differences, there was higher prevalence of disordered eating in T1D females compared to T1D males (Bachle et al., 2016, Fairburn et al., 1991), however one study found the difference to be not statistically significant (Pinar, 2005).

**The relationship between insulin restriction and other disordered eating behaviours.**

The results from the systematic review suggest that insulin restriction/omission does not occur exclusively in T1D individuals with disordered eating (Fairburn et al., 1991), with the reverse also being true, that ED or disordered eating status does not guarantee insulin restriction/omission as a purging behavior. Rates of insulin restriction/omission in those presenting with disordered eating behaviours ranged from 2.7% (Bachle et al., 2016) to 42% (Jones et al., 2000) in females, and from 1.9% (Bachle et al., 2016) to 11.7% (Baechle et al., 2014) in males. Individuals with disordered eating were significantly more likely to restrict/omit insulin compared to those with low eating pathology (Cantwell & Steel, 1996; Pinar 2005; Goebel-Fabbri et al., 2008), however one study (Baechle et al., 2014) found this difference was only significant in males. Conversely, two studies (Wisting et al., 2015; Wisting et al., 2017) found no significant association between disordered eating and insulin restriction/omission in males.

Of the studies that differentiated between disordered eating symptoms, Powers et al. (2012) found that of those reporting insulin restriction/omission for weight loss purposes, 26% solely withheld insulin as their ED symptom while 74% reported other disordered eating behaviours in addition to insulin restriction/omission. These findings were supported by Takii et al. (1999) who found that 75% of participants with comorbid T1D/ED used insulin omission alone, 19% used insulin omission with self-induced vomiting, and 6% used insulin omission with
excessive exercise. The results also suggest that while insulin restriction/omission is frequently reported, binge eating (d'Emden et al., 2013), driven exercise (d'Emden et al., 2013), and dieting (d'Emden et al., 2013; Jones et al., 2000), are more commonly reported disordered eating behaviours. Insulin restriction is also common after periods of overeating (Wisting et al., 2013).

**Emotions in insulin restriction and theories of action.** There exist few studies that have investigated the mechanisms underlying disordered eating behaviour in T1D. Merwin et al. (2014) assessed eating behaviours and insulin dosing in individuals with T1D and found that the majority of individuals endorsed some degree of disinhibited eating when they think their blood glucose is low and accompanied by negative affect (e.g. guilt/shame). The frequency of disinhibited eating was also positively associated with weight-related insulin restriction (Merwin et al., 2014). In 2015, Merwin and colleagues furthered their research and assessed emotions, eating, and insulin dosing in adults with T1D using ecological momentary assessment. They found that individuals who reported higher negative affect were more likely to restrict insulin. Insulin restriction was also more likely if an individual experienced increased anxiety/nervousness and guilt/disgust with self before eating, or when individuals reported that they broke a dietary rule (e.g. “no sweets”).

**The transdiagnostic model of disordered eating in T1D**

Within the literature, few models (Goebel-Fabbri et al., 2002; Treasure et al., 2015) have been developed to encompass the development and maintenance of disordered eating in T1D. One model by Treasure et al. (2015), suggests that individuals with vulnerability factors such as low self-esteem and perfectionism personality traits may find diabetes management frustrating. This coupled with the focus on weight and eating in diabetes management, and environmental exposures in the form of “fat talk”, is posited to further increase weight and shape concerns. This
in turn, triggers dieting behaviours, such as dietary restriction and/or insulin misuse to aid weight loss, which can lead to wide fluctuations in plasma glucose. These fluctuations may then lead to neuroadaptive changes and an associated addictive pattern of desire for high sugar/fat foods, thus leading to the development of a vicious circle of disordered eating. While Treasure et al.’s (2015) model has strengths from drawing from the ED literature (Fairburn et al., 2003; Stice, Shaw, & Nemeroff, 1998), it also uses literature on food addiction to develop assumptions for the causation and maintenance model. However, inclusion of food addiction is problematic, as food addiction largely does not explain excessive eating, and the notion that excessive eating can cause neuroadaptive changes akin to those observed in drug addiction is highly contentious (Rogers, 2017). It is therefore suggested that food addiction be removed from ED models as it may neither explain nor significantly aid recovery, especially in clinical settings. As such we propose a new model for disordered eating in T1D, which excludes food addiction and encompasses the various types of disordered eating behaviours, thus providing a comprehensive assessment of disordered eating in T1D.

The proposed model for disordered eating in T1D is outlined in Figure 2. The model can be seen to draw from both Fairburn et al.’s (2003) transdiagnostic model of disordered eating and Treasure et al.’s (2015) maintenance model. The current model also provides support for Goebel-Fabbri et al.’s (2002) model by including constructs such as perfectionism, affect regulation, and diabetes-related complications, discussed within their model. The current model however has added strengths by including three disordered eating behaviours: dietary restriction, bingeing, and purging, in order to better capture transdiagnostic presentations in T1D populations, as evidenced by the results of the systematic review. The model has also removed food addiction as a maintenance mechanism of action.
The proposed model suggests that, consistent with Fairburn et al. (2003), low self-esteem and perfectionism predispose an individual to dysfunctional self-evaluation, including an overconcern with eating, weight, and shape. The pressure of diabetes management and the learned importance of eating, exercise, and effects of insulin (including weight gain) further exacerbates an individual’s concern about their body. An individual may then cope with the uncertainties and frustration with diabetes with strict behaviours (e.g. dieting), which may then develop into disordered eating behaviours such as bingeing, purging, and/or restricting. Dietary restriction itself is associated with bingeing in non-diabetic individuals (Fairburn et al., 2003).

In line with the findings from the current literature review, the current model suggests that insulin restriction/omission is maintained by disinhibited eating, poor affect regulation, and perceived low blood glucose (Merwin et al., 2014; Merwin et al., 2015). Further complicating the effects of disordered eating, individuals with T1D have additional health effects such as those arising from hypo- or hyper-glycaemia. Problematically, individuals with T1D have a propensity towards disinhibited eating (e.g. eating foods they do not typically allow) when their plasma glucose is low (Goebel-Fabbri, 2009; Merwin et al., 2014), as hypoglycaemia itself increases hunger, and especially desire for sugary foods (Strachan, Ewing, Frier, Harper, & Deary, 2004) which will add to pressure to eat foods perceived as “naughty” and thereby increase the likelihood of catastrophic disinhibition of restraint. Hypoglycaemia has also been suggested to be a justification for bingeing on restricted foods (Merwin et al., 2014; Treasure et al., 2015) and two studies (Schober et al., 2011; Snyder et al., 2016) included in the current literature review.
found that overdosing of insulin was associated with the desire for uncontrolled binge eating or to eat more than one should. Importantly, fluctuations in blood glucose, particularly when caused by disordered eating, expose the individual not only to ED-related complications, but also to other harmful diabetes-related complications. The reality of these short- and medium-term diabetes-related complications, including the fear of weight gain and poor glycaemic control, then feeds back into the over-evaluation of eating, shape, and weight, and the cycle continues.

**DISCUSSION**

The current study aimed to systematically evaluate the empirical literature concerning insulin misuse as a disordered eating behaviour in individuals with type 1 or insulin-dependent diabetes, in order to develop an empirically-based maintenance model of disordered eating in these populations. The results from the systematic review demonstrate that disordered eating status (Jones et al., 2000) and female gender (Bryden et al., 1999; Fairburn et al., 1991) in particular are implicated in insulin restriction.

**Clinical implications**

Insulin misuse is a dangerous method of controlling weight, as those who misuse insulin are at high risk for diabetes-related complications and premature death. When an ED is comorbid with T1D, the risk of complications increase, as well as a threefold increase in mortality (Goebel-Fabbri et al., 2008; Nielsen, Emborg, & Mølbak, 2002). Comorbidity of AN in T1D for example has been associated with premature death (Nielsen et al., 2002). The duration of severe insulin omission and duration of T1D has also been found to be significantly associated with retinopathy and nephropathy in those with comorbid EDs (Takii et al., 2008). Treatment outcomes for
individuals with T1D and comorbid EDs are also unfavourable. Custal et al. (2014) assessed treatment outcomes, dropouts, ED pathology and personality characteristics for individuals with comorbid T1D and ED, compared to ED patients without diabetes. They found higher dropout rates from therapy in individuals with T1D and worse treatment outcomes in spite of having no significant differences in ED psychopathology (except insulin misuse). Insulin misuse should therefore be considered as a form of purging behaviour, and should not be investigated solely in the context of BN, to ensure that individuals with diabetes are screened for all EDs such as AN, binge-eating disorder (BED), or other specified feeding or eating disorder (OSFED).

**Potential research directions**

Although there have been recent reviews published in the area of disordered eating in T1D (e.g. Larrañaga, Docet, & García-Mayor, 2011), a development and maintenance model for disordered eating in T1D has yet to be empirically established. Future research directions should therefore test the proposed model in order to validate and further understand the mechanisms underlying insulin misuse in T1D. Furthermore, the majority of research has been conducted in adolescent populations, particularly with females, with the main focus given to insulin restriction/omission. Preliminary evidence (Schober et al., 2011; Snyder et al., 2016), suggests that insulin overdosing may serve a different function to insulin restriction or omission, such that insulin underdosing is used to promote weight loss, while insulin overdosing may be used to facilitate episodes of disinhibited eating or binge eating. More research is therefore needed in this area. Moreover, given that the onset of EDs in T1D has been found to emerge well into early adulthood (Colton et al., 2015), there is need for future research to focus on disordered eating in adults with T1D.
Finally, the current literature review also sheds light on researchers’ use of custom questionnaires to measure insulin misuse (see Table 1). Future work should also include the use and further development of sensitive measures of disordered eating pathology and interventions specific to those with T1D. Notably, given the increase in ED prevalence rates in the general population (Micali, Hagberg, Petersen, & Treasure, 2013) and the life-threatening nature of this comorbid phenomenon, the area of insulin misuse in T1D is an area worthy of further study.
References


Investigation, 28(7), 417-419. doi: 10.1007/BF03347221


Figure 1. Flow chart of the study selection process.
# DISORDERED EATING AND INSULIN RESTRICTION

## Table 1. Studies included in systematic review.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Participants</th>
<th>Measures</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ackard et al. (2008)</td>
<td>70 female and 73 male adolescents (age $M = 15.3$ years) with T1D, 4746 (2357 female, 2377 male, 12 gender missing, age $M = 14.9$ years) controls.</td>
<td>Custom insulin restriction or omission question</td>
<td>11 out of 143 reported insulin omission (1.4% males and 10.3% females) and dosage reduction (1.4% males and 7.4% females) as means of weight control. T1D reported less weight dissatisfaction and were less likely to use any unhealthy weight control behaviors and more likely to report regular meal consumption than controls.</td>
</tr>
<tr>
<td>Affenito, Rodriguez, Backstrand, Welch, and Adams (1998)</td>
<td>90 female T1D (aged 18-46 years)</td>
<td>DSM-III-R, EDE, BTR, custom insulin restriction or omission question</td>
<td>27 of 90 (30%) reported disordered eating, and 12 of 27 (44%) reported insulin misuse for weight control.</td>
</tr>
<tr>
<td>Bachle, Stahl-Pehe, and Rosenbauer (2016)</td>
<td>819 adolescents (51% male, aged 11–21 years) with T1D</td>
<td>SCOFF, custom frequency of insulin restriction question</td>
<td>28.2% of the female and 9.2% of the male patients were SCOFF-positive but denied insulin restriction. 4.2% of the female and 5.3% of the male patients reported frequent insulin restriction without disordered eating. 2.7% of the female and 1.9% of the male patients reported both disordered eating and insulin restriction.</td>
</tr>
<tr>
<td>Baechle et al. (2014)</td>
<td>629 patients (54.1% male, age $M = 15.3$ years) with T1D, 6,813 comparison participants (51.3% male, age $M = 14.6$ years)</td>
<td>SCOFF, custom insulin restriction or omission question</td>
<td>No significant difference between groups for SCOFF. 20.5% of female and 18.5% of male diabetic patients reported insulin restriction at least three times per week. SCOFF-positive patients (31.2% of female, 11.7% of male) reported more insulin restriction than SCOFF-negative patients, however, the differences were only significant in male patients.</td>
</tr>
<tr>
<td>Birk and Spencer (1989)</td>
<td>385 females (age $M = 28.2$) with IDDM</td>
<td>Diabetes-adapted Pyle Eating Behavior Survey</td>
<td>Prevalence of bulimia and anorexia was within the range identified in the general population. More than 70 (&gt;18%) individuals reported reducing or omitting insulin to control weight.</td>
</tr>
<tr>
<td>Bryden et al. (1999)</td>
<td>33 females and 43 males (baseline age $M = 15$ years, follow-up age $M = 23$ years) with T1D</td>
<td>EDE-Q, custom insulin reduction or omission questions</td>
<td>Over the course of 10 years, 10 (30%) of females reported underusing insulin to control weight. None of the males reported insulin misuse.</td>
</tr>
<tr>
<td>Cantwell and Steel (1996)</td>
<td>48 females (aged 17-30 years) with T1D</td>
<td>EAT and semi-structured interview with custom insulin purging questions</td>
<td>10 of 48 (20.8%) participants reported insulin misuse. High EAT scorers were more likely to misuse insulin than low scorers (36% vs. 8%).</td>
</tr>
<tr>
<td>Custal et al. (2014)</td>
<td>20 females (age $M = 25.3$ years) with comorbid T1D/ED and 20 females (age $M = 28$ years) with ED</td>
<td>Semi-structured interview with custom insulin purging questions</td>
<td>18 of out 20 (90%) T1D reported skipping or reducing insulin dose as a purging method.</td>
</tr>
<tr>
<td>d'Emden et al. (2013)</td>
<td>124 adolescents (53% female, age $M = 15.4$ years) with T1D</td>
<td>Diabetes-adapted Youth-EDE and EDI</td>
<td>Disordered eating behaviour was reported by 32.3% (37.9% of females, 25.9% of males), binge eating (17.7%), driven exercise (13.0%), dietary restraint (8.9%), insulin manipulation/omission (5.6%), vomiting (3.3%), laxative (0.8%) or diuretic use (0.8%).</td>
</tr>
<tr>
<td>Fairburn, Peveler, Davies, Mann, and Mayou (1991)</td>
<td>54 females and 46 males (aged 17-25 years) with IDDM and 67 female age matched controls</td>
<td>EDE, EAT, custom insulin underuse or omission questions</td>
<td>20 of 54 (37%) T1D females had omitted or underused insulin to influence their weight. This behaviour was not restricted to those with a clinical ED. None of the men reported disordered eating, and none had misused insulin to influence their weight.</td>
</tr>
<tr>
<td>Study</td>
<td>Sample Description</td>
<td>Measures</td>
<td>Findings/Comments</td>
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<tr>
<td>Goebel-Fabbri et al. (2008)</td>
<td>234 females (age $M = 45$ years) with T1D</td>
<td>BTR, EDI, custom insulin restriction questions</td>
<td>71 of 234 (30%) reported insulin restriction at baseline. Insulin restrictors scored higher on baseline measures of bulimia and other disordered eating symptoms than non-restrictors.</td>
</tr>
<tr>
<td>Goebel-Fabbri et al. (2011)</td>
<td>207 females (age $M = 44$ years) with T1D</td>
<td>PAID, BTR, custom insulin restriction questions</td>
<td>34 women (23%) reported new restriction at 11-year follow-up from baseline, and endorsed greater fear of weight gain with improved blood glucose relative to non-restrictors.</td>
</tr>
<tr>
<td>Herpertz et al. (1998)</td>
<td>355 females (age $M = 44.3$) and 307 males (age $M = 45.9$) with IDDM</td>
<td>Modified Questionnaire for the Diagnosis of Eating Disorders (FSE)</td>
<td>4.1% of the whole sample (5.6% females, 2.6% males) reported intentional insulin undertreatment or omission for weight regulation.</td>
</tr>
<tr>
<td>Jones, Lawson, Daneman, Olmsted and Rodin (2000)</td>
<td>356 females (aged 12-19 years) with T1D and 1098 age matched controls.</td>
<td>EDI, EAT, EDE, diabetes-adapted adapted DSED</td>
<td>41 of 356 (11%) T1D participants reported insulin restriction to lose weight. 36 of 356 met diagnostic criteria for ED, and of these, 15 (42%) reported insulin misuse. Deliberate insulin omission was most common weight loss behavior after dieting.</td>
</tr>
<tr>
<td>Kichler, Foster, and Opipari-Arrigan (2008)</td>
<td>75 females (aged 11–17 years) with T1DM and their mothers.</td>
<td>DSMP, EDI, EAT, EDE-Diabetes</td>
<td>None of the participants endorsed omitting insulin for the purposes of weight management.</td>
</tr>
<tr>
<td>Merwin et al. (2015)</td>
<td>83 adults (88% female, age $M = 41.89$ years) with T1D</td>
<td>Ecological momentary assessment with custom questions</td>
<td>Negative affect was associated with increased likelihood to restrict insulin. Increases in anxiety/nervousness and guilt/disgust with self before eating further increased the odds of restricting insulin at the upcoming meal. Insulin restriction was more likely when individuals reported that they broke a dietary rule.</td>
</tr>
<tr>
<td>Merwin et al. (2014)</td>
<td>276 adults (68.5% female, age $M = 43.5$ years) with T1D</td>
<td>DEPS, custom disinhibited eating questions</td>
<td>The frequency of disinhibited eating was positively associated with weight-related insulin mismanagement.</td>
</tr>
<tr>
<td>Neumark-Sztainer et al. (2002)</td>
<td>70 female and 73 male adolescents (aged 12-21 years) with T1D</td>
<td>DEPS</td>
<td>10.3% of females reported skipping insulin and 7.4% reported taking less insulin to control their weight. Only one male reported doing either of these behaviors.</td>
</tr>
<tr>
<td>Olmsted, Colton, Daneman, Rydall and Rodin (2008)</td>
<td>126 females (baseline age $M = 11.9$ years) with T1D. Follow-up annually for 5 years.</td>
<td>ChEDE</td>
<td>At baseline 19 of 126 (18%) participants reported disordered eating. Of these 19, 3 (16%) reported insulin manipulation at baseline. 38 participants developed disordered eating during follow-up, and 2 (5%) of these reported insulin omission after disordered eating onset.</td>
</tr>
<tr>
<td>Peveler et al. (2005)</td>
<td>87 patients baseline (gender not reported, aged 11-25 years) and 63 follow-up (aged 20-38 years) with T1D</td>
<td>EDE</td>
<td>Insulin misuse for weight control reported by 31 (35.6%) of subjects.</td>
</tr>
<tr>
<td>Pinar (2005)</td>
<td>45 adolescents (50% female, age $M = 15.49$ years) with T1D and 55 (50% female, age $M = 15.49$ years) control adolescents</td>
<td>EAT, BIS, custom insulin misuse questions</td>
<td>Disordered eating almost four times as common in T1D than controls. 18 of 45 (40%) T1D reported insulin misuse. EAT score higher in T1D who did strict diet restrictions and misused insulin to lose weight.</td>
</tr>
<tr>
<td>Pinhas-Hamiel et al. (2013)</td>
<td>26 females (age $M = 21.5$ years) with T1D</td>
<td>Not reported</td>
<td>15 of 26 (58%) reported omitting insulin for weight loss.</td>
</tr>
<tr>
<td>Pollock-BarZiv SM &amp; Davis C. (2005).</td>
<td>51 females (age $M = 21.5$ years) with T1D</td>
<td>EDI and diabetes-adapted DSED</td>
<td>27.5% of total participants reported insulin manipulation or omission to promote weight loss.</td>
</tr>
<tr>
<td>Powers et al. (2012)</td>
<td>48 patients (97.8% female, age $M = 26.2$ years) with comorbid T1D/ED</td>
<td>EDE-Q, EDI-3, medical record abstraction</td>
<td>27 of 48 (47.9%) reported withholding insulin for weight loss purposes. Of these 27, 7 (14.6%) solely withheld insulin as their ED symptom and 20 (41.7%) had additional ED symptoms.</td>
</tr>
</tbody>
</table>
25 Rydall, Rodin, Olmsted, Devenyi, and Daneman (1997).
91 females (baseline age $M = 15$ years, follow-up age $M = 19$ years) with IDDM
Diabetes-adapted DSED
12 of 88 (14%) reported omission or underdosing of insulin to lose weight at baseline and 30 of 88 (34%) at follow-up.

26 Schober et al. (2011)
241 patients (42.5% male, age $M = 14$ years) with T1D
DSMP, phone interview, custom insulin manipulation questions
71 of 241 (29.5%) reported intentional over and/or underdosing of insulin. Most reported reason for intentional overdosing was the wish for uncontrolled binge eating (49%). 15.5% reported insulin omission with the intention of weight reduction.

27 Snyder, Truong, and Law (2016)
58 adolescents (48.3% male, age $M = 16.1$ years) with T1D
DSMP, custom insulin overdosing and underdosing questions
3 of 58 (5.25%) reported insulin misuse by underdosing and 9 of 58 (15.5%) by overdosing. Most reported reason for insulin misuse by overdosing was the desire to eat more than one should (88.9%, $n = 8$), and underdosing to lose weight (66.7%, $n = 2$).

28 Takii et al. (1999)
33 females (aged 16–36 years) with comorbid T1D/ED (22 BN, 11 BED) and 33 control females (aged 15-35 years) with T1D.
SCID, EDI, custom interview
16 of 22 (72.7%) comorbid T1D/ED reported severe insulin omission. Of these 16, 12 (54.5%) used insulin omission alone, 3 (13.6%) used insulin omission with self-induced vomiting, and 1 (4.5%) used insulin omission with excessive exercise. EDI scores Controls < T1D/BED < T1D/BN

105 adolescents (42% male, aged 12-20 years) with T1D
Diabetes-adapted ChEDE
Disordered eating was significantly associated with insulin restriction in females. No significant association between disordered eating and insulin restriction in males.

30 Wisting, Froisland, Skriverhaug, Dahl-Jorgensen, and Ro (2013)
770 adolescents (50.6% female, aged 11-19 years) with T1D
DEPS-R
31.6% of total participants (36.8% of females, 9.4% of males) reported insulin restriction and 6.9% (26.2% of females, 4.5% of males) reported skipping their insulin dose entirely after overeating. 41.1% of the insulin restrictors scored above the cutoff on the DEPS-R.

31 Wisting et al. (2017)
104 adolescents (58.6% female, age $M = 15.7$ years) with child-onset T1D
Diabetes-adapted ChEDE
Skipping meals was significantly associated with higher insulin omission due to shape/weight concerns in females. No significant association between disordered eating and insulin omission in males.

Note. $M =$ mean, T1D = type 1 diabetes, IDDM = insulin-dependent diabetes mellitus, ED = eating disorder, BTR = Bulimia Test Revised, DSED = Diagnostic Survey for Eating Disorders, EAT = Eating Attitudes Test, BIS = Body Image Scales, ChEDE = Child Eating Disorder Examination, DEPS-R = Diabetes Eating Problem Survey, EDE = Eating Disorders Examination, EDI = Eating Disorder Inventory, PAID = Problem Areas in Diabetes, SCID = Structured Clinical Interview for DSM-III-R, DSMP = Diabetes Self-Management Profile
Figure 2. The transdiagnostic model of disordered eating in T1D. Diabetes-specific mechanisms are indicated in grey. Figure adapted from Fairburn et al.’s (2003) transdiagnostic maintenance model of disordered eating and Treasure et al.’s (2015) maintenance model for disordered eating in T1D.