Disordered Eating Behavior in Individuals With Diabetes

Importance of context, evaluation, and classification

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This review was conducted to examine disordered eating behavior (DEB), including diagnosable eating disorders, in the context of diabetes. The use of criteria and assessment methods standardized on the healthy population is examined. Also considered is the need for modified assessment methods and classification of this behavior when evaluating patients with diabetes. Future directions for research are suggested.

Literature published from 1980 to present was examined using "eating disorders and diabetes" as search terms. Over 100 peer-reviewed articles were identified via PubMed, Cochrane Reviews, PsycInfo, etc. Bibliographies from articles were reviewed to ascertain additional publications. Cited articles include reviews and individual studies indicating experimental design (self as control, healthy control, or population estimate), assessment methods (self-report, questionnaires, and structured/clinical interviews), and use of standard diagnostic criteria. Not all relevant articles could be included. However, some older references (published before 2000) are included because they provide foundation literature from which our understanding of DEB in the population of patients with diabetes derives and/or are validation studies for measurement methods. Additional references pertinent to hypothesized mechanisms are cited.

Most studies, including those in a 2005 meta-analysis (N = 8 case-controlled studies), tend to focus on young women with type 1 diabetes, usually between ages 15–35 years, when weight concerns, DEB, and eating disorders are at a high prevalence (1). Recent studies have included type 2 diabetic patients, minorities, and male patients (2–9). The diagnosis of diabetes has been associated with elevated rates of DEB and eating disorders, particularly when insulin omission is considered purging (1,8–11).

Diagnosed eating disorders and subclinical DEB have been associated with poorer health in individuals with type 1 diabetes. Early reports found prevalence rates of the co-occurrence of diabetes and DEB to be low and accompanied by psychiatric comorbidity and weight loss, but diabetes control was not compromised (12–14). More recent cross-sectional studies have demonstrated a positive association between elevated A1C and diagnosable eating disorders (2), subclinical DEB (8), and intentional insulin omission (1). The presence of diagnosable eating disorders and behavior categorized as subclinical DEB has been associated with increases in retinopathy (15), neuropathy (16), transient lipid abnormalities (17), hospitalizations for diabetic ketoacidosis (6), and poor short-term metabolic control (1,6,8,18). Studies assessing the association of DEB and eating disorders with long-term metabolic control have produced mixed results (6,19–22). A prospective 5-year study did not find a significant relationship between DEB or eating disorders and poorer glycemic control (22). Less is known about the relationships between DEB and health status in individuals with type 2 diabetes (4,7,9).

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PREVALENCE OF DIAGNOSABLE EATING DISORDERS AND DEB IN PATIENTS WITH DIABETES

— Controversy exists regarding whether there is increased prevalence of diagnosable eating disorders and DEB in individuals with type 1 diabetes than referent populations (1,8,19). Rates similar to (1,8,23–25) and higher than (1,6,23,24,26) healthy same-aged peers (19,27) have been found. Some studies found equivalent or lower rates of diagnosable eating disorders but higher rates of DEB, particularly bulimia symptoms. Estimates of diagnosable eating disorders and DEB in adolescent and young adult females with type 1 diabetes range from 3.8% (12)–27.5% for patients classified as bulimic or having binge eating disorder (BED), based upon evaluation with the Eating Disorders Examination (EDE), (28) and 38–40%when insulin omission is considered purging (21,29). Individuals on insulin therapy may find insulin omission or dose reduction an easy method for weight management via glycosuria. This method is frequently reported by adolescent and young adult females with type 1 diabetes (2,29,30). Rates of both subclinical DEB and diagnosable eating disorders are variable based on the criteria used.

When establishing the relative prevalence of DEB in the diabetic population, most studies have not matched control samples for weight (1,19,21,26). Weight status is a strong predictor of eating disorders and DEB among overweight women attempting weight loss (31). Type 1 diabetic cohorts studied have been significantly heavier than comparison groups, with the average BMI above the normal range (1,19). To compare an age- and sex- (but not BMI-) matched control sample to one with type 1 diabetic patients, the EDE (32) was administered. Similar rates of eating pathology were identified. However, using the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) criteria, which include insulin omission (33), higher rates were identified in the diabetic sam-

ple. Average BMI *Z* score was higher ($P = 0.05$) in the cohort with diabetes, and higher BMI was associated with diagnosable eating disorders (26). In the 5-year follow-up study of type 1 diabetic patients (22), BMI was the strongest predictor of eating disorders. Mannuci et al. (5) compared overweight and obese type 2 diabetic patients with obese nondiabetic patients seeking weight loss, and an obese non–treatment seeking sample. Among all three samples, low levels of BED were diagnosed (<5%). Obese diabetic patients had the lowest scores on the EDE overall but the highest Restraint Scale scores (5). Only one study was found that stratified the diabetes cohort (type 1 and 2 diabetic, male and female, ages 18–65 years) by weight status; 3% of under- and normal-weight women had a current eating disorder, whereas 7% of overweight and 10% of obese women had diagnosable eating disorders (27). These rates are similar to those for nondiabetic samples with similar BMI levels seeking weight loss (31).

Subclinical DEB using DSM-IV Text Revision (TR) (33) criteria such as binge eating, self-induced vomiting, insulin omission, excessive caloric restriction, and intense exercise for weight control are commonly reported by female patients with type 1 diabetes (22,23). In the 5-year prospective study by Colton et al. (22), patients reporting subclinical DEB ranged from 3–26% depending on the behavior. Fifty-one percent of the teens assessed multiple times over a 5-year period reported DEB at least once, and early subclinical DEB was found to be highly persistent over time. Rates documented for male patients are lower but appear to be changing. Svensson et al. (34) studied adolescent males with type 1 diabetes compared with healthy control subjects and found no diagnosable eating disorders in either group. Males with type 1 diabetes had significantly higher BMIs and scores on the Drive for Thinness Scale on the Eating Disorder Inventory (EDI), suggesting higher risk for the development of DEB. Bulimia scores were, however, higher in control subjects than in patients with diabetes (34). Studies using questionnaires and structured interviews that compare occurrence in type 1 versus type 2 diabetic patients show similar rates (9,35). However, types of reported cognitions and behaviors differ. "Drive for thinness" and "body dissatisfaction" were more often reported by type 2 diabetic patients, whereas insulin omission was more frequently reported by type 1 diabetic patients (9,35).

INSULIN AND WEIGHT GAIN

— As glycemic control improves, weight gain is a common side effect of successful treatment with insulin (36). Weight concerns may develop when intensive treatment results in weight gain as well as improved metabolic control (1,6,8,37). Increasing weight can require increasing doses of insulin or secretagogue treatment to control blood glucose, which can lead to increased hunger, hypoglycemia, and dietary intake (38). Comparing rates of DEB between type 1 diabetic adolescent females and healthy control subjects, using both questionnaire and interview data, type 1 diabetic females were significantly heavier (6.8 kg) and had higher rates of DEB (19). Engström et al. suggest that eating more might be a response to overinsulinization and episodes of hypoglycemia because both "strongly activate appetite regulation in the hypothalamus" (19). The evaluation of supraphysiological levels of insulin as contributing to overeating and insulin reduction as a compensatory strategy to reduce appetite has not been explored. Compared with injections, insulin pump therapy can provide more physiological insulin dosing and lower the need for insulin (37,39). To compare rates of DEB and attitudes between adolescent girls with type 1 diabetes using pumps versus injections, assessments included the Drive for Thinness Scale, Bulimia and Body Dissatisfaction subscales of the EDI-2, the Dietary Restraint subscale of the Eating Attitudes Test (EAT-26) (40), and questions regarding efforts to control weight by taking less insulin than prescribed. There were no differences in rates of DEB or attitudes between adolescent girls with type 1 diabetes who were using insulin pumps and those using multiple daily injections. Average BMI was within normal limits for both groups and did not predict DEB (37). However, insulin dose per kilogram body weight, which is often up to 15% lower in pump users than those using injections (37,39), was not evaluated as a possible contributor to weight, satiety, overeating, or insulin omission.

DYSREGULATION OF SATIETY

— Destruction of β-cells results in the inability to secrete both insulin and amylin, contributing to dysregulation of appetite and satiety. Amylin mediates several satiety mechanisms via its effects on the area postrema, an area of the brainstem that integrates hormonal and metabolic signals to regulate food intake. Acute anorectic effects of amylin include slowed gastric emptying, reduced glucagon secretion, and lateral hypothalamic activity. Amylin is also thought to play a role in long-term weight regulation (41,42). Amylin, insulin, leptin, and glucagon act synergistically with cholecystokinin to reduce appetite (42). These actions are opposed by ghrelin, concentrations of which are lower in obese and type 2 diabetic patients and higher than would be expected in patients with anorexia nervosa and type 1 diabetes (42,43). Amylin analogs and other hormones affecting the gut-brain axis are considered potential therapies for conditions related to obesity and DEB (44). Pramlintide, an analog of amylin, enhances feelings of satiety, reducing food intake and causing weight loss in patients with diabetes (45).

In a study of other hormonal satiety mechanisms in lean and obese nondiabetic men, levels of postprandial insulin and glucose-dependent insulinotropic polypeptide (GIP) were found to be key determinants of short-term appetite regulation (46). Miglitol, a second generation α-glucosidase inhibitor, was tested to evaluate its effect on glucagon-like peptide I (GLP-1) GIP, insulin-glucose dynamics, and satiety in obese type 2 diabetic women. After a solid meal challenge, type 2 diabetic women taking miglitol had increased GLP-1 response, suppressed GIP, increased satiety, and decreased hunger and food intake versus those taking placebo (47). A review of studies of incretins in type 1 diabetic patients during the remission phase of the disease suggested that the use of long-acting GLP-1 agonists, which modulate insulin dynamics, could help patients stick to their dietary program and maintain a normal between-meal interval (48). No studies were identified that assessed DEB in the context of physiological mechanisms that are known to be associated with dysregulation of appetite and satiety and/or potentiate weight gain.

The current DSM-IV-TR definition of DEB does not address hormonal alterations due to diabetes pathophysiology or treatment that may affect eating behaviors. The DSM-IV-TR classification system for DEB defines binging as a loss of control over behavior and insulin omission as a maladaptive strategy to either

prevent weight gain or reduce weight via glycosuria without an accompanying binge. DSM-IV-TR criteria state: "Individuals with diabetes and bulimia nervosa may omit or reduce insulin doses to reduce the metabolism of food consumed during eating binges" (33). Refinement of criteria to include the effects of treatment has been suggested by the working group convened during the International Conference on Eating Disorders and Diabetes Mellitus, which met in Minneapolis, Minnesota, in 2008 (49). Consideration of physiological mechanisms that affect appetite and satiety in the assessment of DEB in patients with diabetes may also be clinically relevant.

CLASSIFICATION: ADHERENCE NONCOMPLIANCE, OR DISTRESS

— Behaviors considered triggers and consequences of DEB are embedded in the diabetes treatment regimen (49). Behaviors and attitudes such as dietary restraint, food preoccupation (carbohydrate monitoring and restriction, portion control, and control of blood sugars through selective food intake), and programmed exercise are prescribed components of diabetes care (39,50). These same behaviors, excepting control of blood glucose, characterize successful weight loss treatment (51). Behaviors become dysfunctional, DEB or eating disorders, when they are used inappropriately for rapid weight loss, are carried to excess, interfere with activities of daily living, or become a health risk (33). However, in the context of diabetes care, strict adherence to these behaviors provides tools by which glycemic control may be achieved (39). Thus, endorsement of items on screening measures such as the EAT-26, EDI-III, and Diagnostic Survey for Eating Disorders (52) indicating food preoccupation and restriction could initially be identified as DEB in patients with diabetes. When using measures standardized in the general population, clinical evaluation by an individual familiar with the diabetes self-care regimen may be needed to further elucidate the intent and relationships between endorsed behaviors and weight concerns.

Feeling out of control of food intake is a DSM-IV-TR diagnostic criterion for bulimia, BED, and EDNOS (see Table 1: DSM-IV-TR criteria) (33). Attempts to follow a prescribed dietary plan have been documented to result in patients experiencing loss of control of eating behavior (53). The essential criterion for BED is repeatedly eating amounts of food in a short period of time that are "larger than most individuals would eat under similar circumstances." Because DEB is rarely quantified by direct observation, this criterion is usually self-reported. The determination of an objective versus a subjective binge is an established challenge in the evaluation of eating disorders. Illustrating the relationship between report of food intake, weight status, and diabetes, a study of black women with type 2 diabetes found the prevalence of underreporting of dietary intake to be 47%. Underreporting of intake was significantly associated with BMI and waist circumference in the expected direction:

Table 1—*Adapted from the DSM-IV-TR: necessary diagnostic criteria for eating disorders (axis-1)*

- Anorexia nervosa: rare in individuals with diabetes.
  - Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight <85% of that expected or failure to make expected weight gain during period of growth, leading to body weight <85% of that expected).
  - Intense fear of gaining weight or becoming fat, despite being underweight.
  - Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of seriousness of the current low body weight.
- Bulimia nervosa: low prevalence rate of diagnosis but commonly reported behavior in individuals with diabetes.
  - Recurrent episodes of binge eating, characterized by:
    - Eating, in a discrete period of time (e.g., within any 2-h period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
    - A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).
  - Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications (insulin omission); and fasting or excessive exercise.
  - Binge eating and inappropriate compensatory behaviors both occur, on average, ≥2 episodes per week for ≥3 months.
  - Self-evaluation is unduly influenced by body shape and weight concerns.
- Eating disorder not otherwise specified (EDNOS): more commonly diagnosed in individuals with diabetes and includes disorders of eating that do not meet the criteria for any specific eating disorder.
  - All of the criteria for bulimia nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of <2 episodes per week or for duration <3 months.
  - The regular use of inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food (e.g., self-induced vomiting after the consumption of two cookies or insulin omission or reduction after consumption of normal amount of food).
- Binge eating disorder (BED): most prevalent diagnosis in individuals with diabetes.
  - Recurrent episodes of binge eating. An episode is characterized by eating a larger amount of food than normal during a short period of time (within any 2-h period) and lack of control over eating during the binge episode (i.e., the feeling that one cannot stop eating).
  - Binge eating episodes are associated with three or more of the following:
    - Eating until feeling uncomfortably full.
    - Eating large amounts of food when not physically hungry.
    - Eating much more rapidly than normal.
    - Eating alone because you are embarrassed by how much you are eating.
    - Feeling disgusted, depressed, or guilty after overeating.
      - Marked distress regarding binge eating is present.
      - Binge eating occurs, on average, ≥2 days per week for ≥6 months.
      - The binge eating is not associated with the regular use of inappropriate compensatory behavior (i.e., purging, excessive exercise, etc.) and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

larger body size (54). Thus, overweight patients with type 2 diabetes may underreport dietary intake. Studies that evaluated whether type 1 diabetic patients overreport or underreport dietary intake associated with their weight status could not be identified using our search strategies.

Difficulty with diabetes self-management could appear to be DEB. Treatment-related behaviors that may alter energy intake include eating too much or too little relative to glycemic status and insulin dose, overconsumption of food to treat or prevent hypoglycemia, and treatment of presumed hypoglycemic symptoms without checking blood glucose, resulting in unnecessary caloric intake and the need to take compensatory insulin (55). Intentional insulin reduction or omission relative to food intake has been assessed in various studies (2,21). However, the definitions of what constitutes insulin omission and reduction have not been established in the context of diabetes self-management behaviors, e.g., dose adjustment.

Ongoing diabetes treatment exposes patients to situations and emotions known to be associated with the development of DEB. Feelings of loss of autonomy and control are associated with the development of eating disorders in nondiabetic individuals seeking weight loss (31). The relationships are presumed to be similar in patients with diabetes (53). Issues for consideration include but are not limited to: feelings of loss of control because of required monitoring and reporting of food intake, physical activity and blood glucose, feeling loss of autonomy because of parental/spousal/familial concern/vigilance regarding health status (53), increased perfectionism due to accountability to health care providers regarding self-care behaviors and glycemic status (8), lower self-esteem and body image secondary to diagnosis and treatment (56), and weight gain due to the initiation of treatment (36).

Recent studies have examined relationships between personality characteristics and DEB (57–59). Characteristics studied include coping styles (60), unmitigated communion (putting others' needs and opinions before one's own), harm avoidance (avoidance of conflict), and self-directedness (57). Women in particular are prone to unmitigated communion, harm avoidance, and lower self-directedness, characteristics which are hypothesized to place them at risk for DEB. It has been speculated that difficulty adjusting to the diabetes self-care regimen and consequent weight gain may predispose to adoption of maladaptive weight management behavior to avoid disruption in relationships with care providers and family members. Thus, certain personality characteristics in the context of the self-management regimen may make patients susceptible to DEB, independent of other psychological, familial, or societal influences (11).

Evaluating new-onset type 1 diabetic girls and boys, ages 8–13 years, from diagnosis up to 14 years of age, Pollock et al. (12) concurrently evaluated DEB and psychiatric symptomatology. Although the authors found low rates of DSM-III diagnosable eating disorders (3.8%), "youths with eating problems were nine times more likely to have had a psychiatric disorder than the rest of the patients." A diagnosed eating disorder was distinguished from problematic eating behavior specific to the diabetes care regimen. Regimen-specific problems appeared to be part of a constellation of pervasive noncompliance associated with higher psychiatric morbidity (12). The association between psychiatric morbidity and diagnosable eating disorders (assessed using the Structured Clinical Interview and the Binge Eating Scale) was also studied in type 2 diabetic patients. Overweight and obese patients were found to have more diagnosable eating disorders than patients with a normal weight. Those patients with eating disorders had significantly more anxiety disorders and trended toward being more depressed (4). Distress, particularly depression, may accompany onset of weight concerns associated with weight gain, consequent to the difficulty of adhering to the diabetes care regimen (8). Herpertz et al. (7) conducted a cross-sectional study of the relationship between weight DEB, and psychopathology in patients with type 2 diabetes, 80% of whom could be expected to be overweight. Weight had limited association with weight-related distress but no direct association with generalized psychopathology. Eating disorder pathology was, however, strongly associated with depression and low self-esteem (7).

Studies in populations seeking weight loss have shown that the onset of DEB can precede or follow psychological distress (58). The pathway whereby increased weight precedes weight concerns is illustrated in a study of type 1 diabetic patients. BMI was tracked concurrently with weight and shape concerns. As both male and female patients became overweight, DEB increased (23). Given the known comorbidity between increased psychological distress and diabetes (61) and symptoms of psychological maladjustment and DEB (58), findings suggest that assessment of psychological adjustment and regimen compliance should be included when symptoms of DEB are considered (12).

Treatment-based behavior that could be identified as DEB might not be driven by weight concerns. For example, deviation from the prescribed insulin treatment regimen could be noncompliance or poor adjustment to the illness. DEB criteria and evaluation methods developed in the general population may need to be modified to assess the range of self-treatment behaviors relevant to patients with diabetes as well as concurrent psychological adjustment. Adding insulin omission as a purging behavior has increased the potential validity of screening measures and DSM-IV-TR criteria, but other behaviors central to diabetes treatment may also be important. Taking into account treatment-related and physiological phenomena has the potential to improve the accuracy of our assessments.

MEASUREMENT OF DEB AND EATING DISORDERS

A review of representative literature indicated that questionnaires (9,34,37,49,62), interviews (13,19,28,29), and both methods combined (4,12,15,19,26) have been used to identify presence of DEB in studies of individuals with diabetes. All studies reviewed, except that by Markowitz et al. (62), have used instruments and interview techniques developed and/or standardized in the nondiabetic population to establish the presence of DEB and eating disorders. Questionnaires included but are not limited to the Eating Attitudes Test (EAT)-26 (40), Eating Disorders Inventory (EDI)-2/3 (63), the Bulimia Test-Revised (BULIT-R) (64), the Diagnostic Survey for Eating Disorders (DSED) (49), and the Binge Eating Scale (4). Information is gathered via clinical interview for the EDE (32) and the DSM-IV (33), and each provides symptom criteria by which to categorize behaviors as eating disorders and DEB. One study reported modification of the EDE for diabetes (15). The DSED can be self-administered or conducted by interview.

Questionnaires are generally acknowledged to be appropriate for screening but not for diagnosis. This issue is

underscored for individuals with diabetes by the wording of questions that ask about prescribed diabetes management behaviors. The BULIT-R DSED, and EDI-3 were examined by the authors, a psychologist diabetes educator, and a psychologist with type 1 diabetes to identify questions that could be answered affirmatively in the context of diabetes self-management behaviors. "Do you feel you have control over the amount of food you consume?" and "I eat a lot of food when I'm not even hungry" are BULIT-R questions (used in studies by Affenito et al. [17,18]) that directly relate to the diabetes care regimen: the former by prescription of dietary restraint and the latter by use of exogenous insulin. A later question, "I feel that food controls my life," could be endorsed by any individual with diabetes. Similarly, questions on the EDI-3 Drive for Thinness Scale include, "I eat sweets and carbohydrates without feeling nervous," and "I feel extremely guilty after overeating." Three of seven questions on the Drive for Thinness subscale (used by Engstrom et al. [19] to evaluate females, and Svensson et al., [34] to evaluate males) of the EDI regarding eating and dieting that could be answered in the context of having and treating diabetes were identified. An additional three that address the importance of weight status could potentially be endorsed in the context of health care provider recommendations. Using EDI-3 adult and adolescent norms, endorsement of these questions would likely meet criteria for the presence of subclinical DEB. Similarly, the DSED (used by Rydall et al., [15] and Jones et al., [29]) asks about dieting behaviors that could be treatment based. Thus, when using questionnaires standardized in healthy populations, scores may be elevated in both type 1 and type 2 diabetic patients due to items that reflect the diabetes self-care regimen. Conversely, patients who are hesitant to reveal treatment-related weight concerns to health care providers may under-endorse items that relate to their regimen. It has been suggested that refinement of questions and criteria to include the effects of diabetes treatment is needed (49).

Intentional omission or reduction of insulin dose for weight management purposes is included in DSM-IV-TR criteria (see Table 1) under the category of purging behavior (27). However, this behavior could be an aspect of generalized noncompliance (12) or an attempt to reduce hunger, improve feelings of satiety, or reduce the cycle of overeating consequent to hypoglycemia. If the question is posed whether insulin omission is intended to control weight, the answer is frequently affirmative (6,8,10). Further refinement of the question querying the intent of and motivation for insulin reduction in the context of insulin dosing and hunger, as well as weight concerns, would help to establish whether the behavior could be considered adaptive rather than a purge of calories via glycosuria (10). The EDE asks after behavioral intent, associated mood, and thoughts. However, the contribution of the diabetes care regimen, diabetes self-management education, and health care provider directives regarding weight are not routinely assessed to determine their contribution to weight-driven behaviors or concerns. Further refinement and standardization of questionnaires and interview formats to determine the motivation and intent of behaviors in the diabetes population will increase diagnostic accuracy in the context of diabetes.

The Diabetes Eating Problems Survey (DEPS), created by Markowitz et al. (62), includes questions regarding insulin adjustment specifically for the purposes of weight reduction and couches questions in terms of diabetes care. Issues related to glycemic control and iatrogenic weight gain are specifically identified and questioned. A questionnaire that asks about satiety and fullness in the context of insulin dosing, blood glucose levels, and eating in response to hypoglycemia is currently being tested and standardized (D.L.Y.-H. and C.L.D., personal communication). Validity studies that compare the use of well-established DEB criteria, interview formats, and instruments, versus diabetes-specific questionnaires and interview formats assessing treatment prescription, adjustment to illness, hunger, satiety, and unintended outcomes of treatment, are suggested to enhance the accuracy of diagnosis and prevalence of eating disorders and DEB in this population.

SUMMARY

Limitations of current research findings

Gaps in understanding the association of DEB and diabetes include: lack of weight-matched control subjects when comparing the prevalence of eating disorders or subclinical DEB; evaluation of the contributions of an insulin dosing schedule and overinsulinization (19), loss of satiety mechanisms via hormonal dysregulation, and dietary prescriptions as potential causes perceived as loss of control over food intake; the intent of behavior in those seeking to prevent weight gain secondary to treatment; incomplete psychological characterization of samples, including psychological constructs such as loss of control, autonomy, and self-efficacy over blood glucose and weight; the potential for misclassification of behaviors and attitudes as reflecting DEB when they possibly reflect skills and attitudes learned as part of the diabetes care/self-management regimen; and the need for refinement of existing measurement tools and development of assessment methods that address diabetes-specific attitudes, concerns, and behaviors that are prescribed as part of treatment; as well as physiological mechanisms that are beyond the control of the patient.

Most studied cohorts have consisted of subjects that were white, heavier than control samples, recruited from tertiary care centers, and often monitored more frequently and thoroughly than patients receiving care in the community. Sample selection bias may be operating to eliminate well-controlled well-adjusted individuals from clinical studies, potentially selecting individuals most vulnerable to the development of DEB. No studies were identified that monitored patients from the time of diagnosis to establish the temporal sequence of the onset of behavior considered maladaptive and whether weight gain occurs first or the care regimen is manipulated to prevent weight gain. Few studies could be found wherein the comorbidity of depression and other forms of psychological distress and DEB were evaluated.

Directions for future research

Evaluation, characterization, and classification of DEB in individuals with diabetes have clinical importance. However, classification of these behaviors is less clinically informative if population-specific criteria and taxonomy are not established. Further, focusing on identified gaps in future investigations of DEB in this population could improve clinical care for this serious comorbid condition. Studies that chronicle the development of DEB prospectively from diagnosis will allow us to assess the contributions of the many factors that predispose individuals to the development of DEB, potentially identifying approaches to diabetes treatment with a lower risk of iatrogenic complications. It

is clinically important to be able to identify those individuals who are at risk for this comorbid condition in association with and independent of the burden of diabetes care. In order to distinguish whether insulin reduction or omission is maladaptive, evaluation of the intent and context of this behavior is needed. Is it a means to regain control over excessive eating by using self-management skills or, in contrast, is it intended as a short-cut weight management strategy (purging via glycosuria)? Physiological mechanisms such as an insulin dose in excess of physiological requirement, hypoglycemia, and a hormonally driven imbalance in hunger, food intake, and experience of satiety appear to be critical factors in establishing diabetes-specific criteria that discriminate between maladaptive manipulations of the diabetes care regimen to control weight and potentially adaptive regimen modifications. Studies are needed that address these distinctions.

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