

## Psychological Conditions in Adults With Diabetes

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Type 1 (T1D) and Type 2 diabetes (T2D) represent a demanding set of biopsychosocial challenges for patients and their families, whether the age of disease onset occurs in childhood, adolescence, or adulthood. Psychological conditions, defined as syndromes, disorders, and diabetes-specific psychological issues affect a larger proportion of individuals with T1D and T2D compared to the general population. In this review, we summarize the prevalence, impact and psychological treatments associated with the primary categories of psychological conditions that affect adults with T1D and T2D: depressive symptoms and syndromes, anxiety disorders, eating behaviors and disorders and serious mental illness. The implications of the literature for psychologists are discussed, and priorities for future research to advance the science of psychological conditions for adults with T1D and T2D are identified.

*Keywords:* Type 1 and Type 2 diabetes, depression, anxiety, obesity

Type 1 (T1D) and Type 2 diabetes mellitus (T2D) represent a demanding set of biopsychosocial challenges for patients and their families regardless of the age of disease onset. Whether the etiology of diabetes is an autoimmune disorder that depletes the pancreas of insulin-producing (beta) cells, as in T1D, or the development of insulin-resistance in conjunction with lipo- and glucotoxicity, as in T2D (American Diabetes Association [ADA], 2015), the behavioral implications for patients are similar. In both disorders, landmark studies have shown that maintaining glycemic control close to nondiabetes levels reduces risk of long-term complications (The Diabetes Control and Complications Trial Research Group, 1993; U.K. Prospective Diabetes Study Group, 1998). Health care providers, therefore, recommend that patients use medications and multiple self-care behaviors to augment or replace insulin and

achieve glycemic control. This requires maintaining a balance between dietary intake, energy exertion through physical activity and oral and/or injectable medications (e.g., insulin) for the entire life span following diagnosis (ADA, 2015). See Hunter (2016) for more information on T1D and T2D and their treatments.

Self-care requirements, as well as social and environmental factors, place a considerable burden on patients and their families (Polonsky et al., 1995). Populations at highest risk for diabetes, such as racial and ethnic minorities and persons from low socioeconomic backgrounds, may experience environmental exposures (e.g., poverty, stress) that increase risk for mental illness, interfere with self-care and compromise medical management (Mueser & McGurk, 2004). All of these factors set the stage for psychological conditions that affect adults with diabetes. In this article, we use the word *conditions* as an inclusive term to describe symptoms, syndromes and diagnoses that precede or develop subsequent to the onset of T1D or T2D. These conditions differ from broader psychological processes that may be important for diabetes self-management (e.g., self-efficacy, knowledge, adherence) which are described by Gonzalez, Tanenbaum, and Commissariat (2016). Tools to assess psychological conditions in diabetes have been developed (e.g., Antisdel, Laffel, & Anderson, 2001) and a relatively small but growing treatment literature shows that intervening on psychological problems in persons with diabetes improves psychological well-being and may improve metabolic control of diabetes (e.g., Cox et al., 2001; Katon et al., 2010).

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*Editor's note.* This article is one in a collection of articles published in a special issue of *American Psychologist* titled "Diabetes and Psychology" (October 2016). Deborah J. Wiebe, Vicki S. Helgeson, and Christine M. Hunter provided scholarly lead for the special issue.

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In this article, we first summarize the prevalence and treatment literature on psychological conditions in T1D and T2D adults with a particular focus on depression, anxiety disorders, eating disorders and other severe mental illnesses (SMIs). These individual conditions have similar effects on diabetes outcomes which are discussed in the next section. Finally, the limitations of the existing literature and future directions for research and clinical practice that can be achieved by psychologists are discussed.

### Depression

The definition of *depression* varies markedly across studies in patients with diabetes ranging from high levels of self-reported depressive symptoms to formal psychiatric diagnoses such as major depressive disorder, dysthymia or adjustment disorder with depressed mood (Holt, de Groot, Lucki et al., 2014). Variable definitions have resulted in a heterogeneous literature with mixed findings for prevalence, impact and treatment. In this article, we use the term *depressive symptoms* when referring to self-reported symptoms inventories and *depression* when referring to a formal psychiatric diagnosis.

### Prevalence of Depression and Depressive Symptoms in Diabetes

In a meta-analysis of cross-sectional studies of diagnosed depression and depressive symptoms, Anderson, Freedland, Clouse, and Lustman (2001) found the point prevalence rates for elevated depressive symptoms were 21.3% for adults with T1D and 27% in studies of adults with T2D.

Recent meta-analyses of longitudinal studies identified a 24–38% increased risk for T2D in those with depressive symptoms with higher risk among studies using psychiatric diagnostic interviews to diagnose depression (29%; Nouwen et al., 2010; Rotella & Mannucci, 2013). Rates of depressive disorders, as assessed by psychiatric interview, ranged from 8% to 15% in adults with T1D and T2D (Anderson et al., 2001), with no studies examining rates of diagnosed depression in T1D samples exclusively. These rates are elevated compared to the adjusted global point prevalence (4.7%; 95% confidence interval [CI] [4.4%, 5.0%]) of depressive disorders and elevated depressive symptoms found in the general population from pooled prevalence studies ( $k = 116$ ) conducted predominantly in North America and European countries (Ferrari et al., 2013).

Few longitudinal studies have examined duration and recurrence of depressive disorders. Lustman, Griffith, and Clouse (1988) conducted a 5-year longitudinal evaluation of patients diagnosed with major depressive disorder and found a 79% relapse rate. Cross-sectional studies of elevated depressive symptoms suggest that depressive symptoms appear to persist for prolonged periods (e.g., 12–18 months), but no longitudinal studies have documented duration of diagnosed depression episodes in T1D or T2D samples to date (de Groot et al., 2007; Holt, de Groot, & Golden, 2014; Peyrot & Rubin, 1999).

Data from the Multiethnic Study of Atherosclerosis, as well as a subsequent meta-analysis, have shown a bidirectional longitudinal association between depressive symptoms and T2D mellitus in adults (Golden et al., 2008; Golden et al., 2004). Antidepressant medications have been shown to be a risk factor for T2D (Barnard, Peveler, & Holt, 2013; Rotella & Mannucci, 2013). Conversely, having diabetes requires significant lifestyle changes and self-management behaviors that impose a significant burden on the patient, which may lead to depression (Nouwen et al., 2011). In individuals with T2D, the rates of depression are higher among those prescribed insulin compared to those using noninsulin medications or dietary and lifestyle interventions alone (Hermanns, Kulzer, Krichbaum, Kubiak, & Haak, 2005). Although insulin itself is not a causative agent, the use of insulin requires more intense disease self-management burden for the patient (Holt, de Groot, & Golden, 2014). Other diabetes specific risk factors for depression include recurrent hypoglycemia and poor glycemic control (Holt, de Groot, & Golden, 2014).

### Screening and Treatment

Screening for depression in patients with diabetes is encouraged in primary care settings and can be performed using self-report inventories developed for the general population (e.g., Patient Health Questionnaire, Kroenke,



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Spitzer, Williams, & Löwe, 2010; Centre for Epidemiologic Studies Depression Scale, Radloff, 1977; Beck Depression Inventory, Beck, Ward, Mendelson, Mock, & Erbaugh, 1961 and the Hospital Anxiety and Depression Scale Zigmond & Snaith, 1983). A review of the literature designed to evaluate screening for depressive symptoms and depression found that the majority of criteria are either fully or partially fulfilled (e.g., definitions of depression, linkages to diabetes outcomes, screening measures, acceptability of measures, availability of effective treatment options, screening effectiveness in changing outcomes, cost-effectiveness, quality assurance, adequate staffing). However, most screening efforts are opportunistic rather than systematic thereby limiting the implementation of depression treatment options. Screening alone is insufficient to improve clinical outcomes and needs to be integrated into defined depression management protocols (Holt & van der Feltz-Cornelis, 2012; Pouwer et al., 2011).

An important consideration in depression screening is the extent to which somatic symptoms assessed in standard depression screening inventories should be attributed to depression or diabetes. For example, a substantial subset of T1D or T2D patients develop a deficit in small intestinal incretin production that impairs satiety (ADA, 2015). This deficit results in persistent hunger sensation and increased caloric intake that could be mistakenly attributed to increased appetite associated with depressed mood. Self-reported screening measures cannot correctly attribute such symptoms to diabetes or depression. Follow-up diagnostic evaluation is needed.

Psychotherapy approaches for depression in diabetes have predominantly used cognitive behavior therapy delivered

individually by mental health providers or trained nurse case managers. Other common psychological interventions used in people with diabetes include problem solving, interpersonal therapy, motivational interviewing, counseling and psychodynamic therapy (van der Feltz-Cornelis et al., 2010). Few randomized trials have been conducted and they have focused on predominantly T2D samples. No trials have been conducted exclusively in adults with T1D. The greatest effects on depression and diabetes outcomes have been seen in psychotherapeutic interventions combined with diabetes self-management (van der Feltz-Cornelis et al., 2010), suggesting the benefits of integrated psychological and medical care to address depressive symptoms and the maladaptive behavior changes (i.e., sedentary lifestyle, poor diet) that often accompany depression. Despite common concerns among health professionals about stigma and rejection of psychotherapeutic interventions, patients with diabetes who have received psychological interventions have shown high rates of satisfaction with treatment (de Groot, Pinkerman, Wagner, & Hockman, 2006).

A significant limitation of this literature is the examination of depression screening and treatment interventions in older adult and ethnically diverse samples. No data have specifically examined prevalence rates of depression in older adult samples with either T1D or T2D. Cultural and contextual factors should be carefully considered when assessing depressive symptoms among minorities in light of evidence that rates of reported depression may vary across cultural or racial groups (Wagner, Perkins, Piette, Lipton, & Aikens, 2009) and the experience of racial discrimination increases depression but decreases reporting of symptoms and treatment (Wagner & Abbott, 2007).

## Anxiety

### Prevalence of Anxiety in Adults With Diabetes

Adults with diabetes have a 20% increased prevalence of anxiety disorders compared to those without diabetes (Smith et al., 2013), with the highest rates for generalized anxiety disorder. Women, younger individuals, those with longer diabetes duration, and those with additional medical conditions are at highest risk. Rates of subsyndromal anxiety symptoms are also increased. The prevalence of elevated symptoms is similar in T1D and T2D (Grigsby, Anderson, Freedland, Clouse, & Lustman, 2002). Evidence suggests that anxiety disorders are highly persistent in persons with diabetes (Fisher et al., 2008).

Among anxiety disorders, only posttraumatic stress disorder (PTSD) has been shown to predict the onset of T2D (Atlantis, Vogelzangs, Cashman, & Penninx, 2012), with odds ratios ranging from 1.3 to 2.1 (Miller-Archie et al., 2014). Cross-sectional studies document that PTSD increases the odds of T2D in a dose-response manner with



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more trauma events increasing the likelihood of diabetes (Husarewycz, El-Gabalawy, Logsetty, & Sareen, 2014).

### **Anxiety Unique to Diabetes**

There are also anxieties unique to diabetes, including fear of complications, hypoglycemia, and invasive procedures. Worry about the possibility of serious, long-term complications is often rated as the most distressing aspect of both T1D and T2D (Snoek, Pouwer, Welch, & Polonsky, 2000). Anticipatory fear of hypoglycemia is also a common concern. Hypoglycemia, defined as blood glucose levels significantly below the normal range (i.e., <70 mg/dl) is physically unpleasant as it involves a counter regulatory hormonal response (e.g., adrenalin) and temporary cognitive impairments. Insufficiently treated hypoglycemia may result in diabetic coma or death. Hypoglycemic episodes may be unpredictable or unexplained by the person with T1D or T2D and may be experienced as aversive or embarrassing due to changes in short-term functioning and the social appraisals of others. Fear of the potentially life threatening nature of this acute and recurrent complication may result in changes in self-care behaviors designed to raise overall blood glucose levels beyond recommended levels to prevent future hypoglycemic episodes (Shepard, Vajda, Nyer, Clarke, & Gonder-Frederick, 2014).

A specific phobia that is particularly problematic for patients with T1D and T2D is fear of invasive self-care behaviors such as fear of injections, self-monitoring of blood glucose, and insertion of subcutaneous insulin infusion devices (i.e., insulin pumps) and continuous glucose monitors. Although the lifetime prevalence of blood/injury/

injection specific phobia is not more common in the setting of diabetes, this disorder, or subclinical symptoms of it, can compromise diabetes self-care in adults and children (Cemeroglu et al., 2015) and can be a barrier to successful transition from oral agents to injections in older adults with T2D patients (Bahrmann et al., 2014). These fears are also associated with higher glycosylated hemoglobin (A1c; Cemeroglu et al., 2014).

A related problem is fear of invasive self-care behaviors in a *new location* on the body (Patton, Eder, Schwab, & Sisson, 2010). Individuals with T1D or T2D who are treated with injectable insulin are recommended to routinely rotate their injection sites to reduce scarring of subcutaneous tissue which may impede insulin absorption (ADA, 2015). Fear of invasive self-care behaviors may impede site rotation and result in suboptimal use of insulin and overall glycemic control.

### **Screening and Treatment**

There has been no evaluation of routine screening for anxiety in health care settings among patients with diabetes (Pouwer, 2009). When assessing anxiety in persons with diabetes, care must be taken to distinguish symptoms of anxiety from those of hypoglycemia. The adrenergic, affective, and cognitive symptoms of anxiety and hypoglycemia can be remarkably similar. Individuals with diabetes who report symptoms of anxiety and/or panic should be encouraged to self-monitor blood glucose while symptomatic. Symptoms of anxiety during euglycemia would be suggestive of an anxiety disorder.

Because of the lack of side effects, nonpharmacological therapies may be preferable as initial therapy for anxiety in diabetes with most studies showing small but statistically significant effects. Small, uncontrolled studies suggest that systematic desensitization can decrease specific fears and phobias such as of self-monitoring of blood glucose and injections. Small, controlled trials of mindfulness-based cognitive therapy (van Son et al., 2013), as well as diabetes education, self-management training, and psychoeducation have all shown benefits for anxiety in patients with T1D (Hopkins et al., 2012) and T2D (Penckofer et al., 2012).

Blood glucose awareness training (Cox et al., 2001) is an empirically validated cognitive-behavioral therapeutic intervention that teaches patients to identify their idiosyncratic symptoms of hypo- and hyperglycemia as an adjunct to self-monitoring of blood glucose. Blood glucose awareness training has been shown to decrease anxiety in patients with T1D. There is also preliminary evidence for the anxiety lowering effects of exercise in T2D (Sardar, Boghrabadi, Sohrabi, Aminzadeh, & Jalalian, 2014). Stepped care for depression (Ell et al., 2010), including problem-solving therapy with or without antidepressant medication, has also been shown to decrease symptoms of anxiety.

## Disordered Eating Behaviors and Eating Disorders

Disordered eating behaviors (DEBs) and eating disorders represent a spectrum of symptoms that may include restricted caloric intake, distorted body image, binge eating and/or purging behaviors such as excessive exercise, vomiting, and the use of laxatives to lose weight. Eating disorder diagnostic categories include Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder and Other Specified Feeding or Eating Disorder (American Psychiatric Association, 2013). DEBs have been defined as eating behaviors and cognitions that occur at lower frequency than eating disorders and pose difficulty vis a vis recommendations for the behavioral management of medical diagnoses (Young-Hyman, 2012).

The management of T1D and T2D involves multiple components that raise the risk of DEBs and eating disorders in patients including: higher rates of overweight and obesity, close monitoring, and/or restrictions of dietary choices in the service of medically recommended weight loss or blood glucose control, prescribed physical activity, loss of autonomy in the selection of food, and the ability to manipulate weight through insulin underadministration or omission. Eating disorder diagnostic criteria, developed for use with the general population, have evolved to include some symptoms specific to diabetes, notably intentional insulin restriction as a purging behavior.

### Prevalence

The prevalence of DEBs and eating disorders in patients with T1D and T2D vary by methodology of assessment (e.g., self-report questionnaires vs. diagnostic interviews) and whether measures designed to assess eating disorders have been tailored to account for diabetes-specific behaviors and treatment contingencies that either contribute to or potentially inflate prevalence rates. The majority of studies have focused on adolescents and women with T1D (Young-Hyman & Davis, 2010). Young and colleagues (2013) found the prevalence of DEBs to be as high as 51.8% in adolescent T1D samples compared to 48.1% in nondiabetes samples using diabetes-adapted measures. The prevalence of eating disorders (as defined by *Diagnostic and Statistical Manual of Mental Disorders* criteria) was 6.4% in T1D samples compared to 3.0% in nondiabetes samples using diabetes-adapted measures with the vast majority of presentations involving bulimia or binge eating disorder (Rodin et al., 2002). Use of generic measures resulted in much higher prevalence rates for both DEBs (24.4% diabetes vs. 10.1% nondiabetes) and eating disorders (10.1% vs. 2.3%, respectively) in T1D samples. Among DEB symptoms, bulimic symptoms and insulin omission are more common in T1D than T2D patients (Young-Hyman, 2012).

Few studies have examined prevalence rates of DEBs and eating disorders in adults with T2D despite the fact that T2D is highly correlated with overweight and obesity. Prevalence rates for eating disorders and binge eating disorder range from 5.3% to 14% in samples of T2D adults (Çelik et al., 2015; Nicolau et al., 2015). Although not well studied, the point prevalence of night eating syndrome has been found to be 7% in a sample of  $N = 194$  T2D patients (Hood, Reutrakul, & Crowley, 2014). T2D patients show increased scores on “drive for thinness” and body dissatisfaction from baseline to follow-up assessment (Herpertz et al., 2001). Caloric restriction/restraint and binge eating are more common DEB symptoms in patients with T2D compared to T1D. There are few longitudinal studies of eating disorders but those that exist have noted the persistence of DEBs and eating disorders in T1D and T2D patients over time (Rodin et al., 2002). No studies have examined differential rates of eating disorders or DEBs in older adults or communities of color.

Evaluation of psychiatric conditions has shown that the presence of DEBs and eating disorders are associated with comorbid anxiety disorders (Papelbaum et al., 2005) and elevated levels of depressive symptoms (Herpertz et al., 2001) in T1D patients and depressive symptoms and obesity in T2D patients (Nicolau et al., 2015; Çelik et al., 2015; Hood et al., 2014).

### Screening and Treatment

No studies have evaluated systematic screening for DEBs and eating disorders in primary care or other medical practices. Screening should utilize diabetes-specific measures (e.g., Diabetes Eating Problems Survey, Antisdel et al., 2001) that distinguish medical nutrition treatment recommendations from DEB; account for physiologic cues that may be attributable to the diabetes disease process rather than a psychological symptom; and assess patient’s expectations and goals for glycemic control (e.g., intensive glycemic control in preparation for pregnancy; Young-Hyman, 2012). Psychiatric interview protocols have been adapted for the assessment of symptoms in the context of diabetes treatment including the Eating Disorders Examination (Cooper, Cooper, & Fairburn, 1989). To establish a diagnosis of an eating disorder or DEB, evaluation should include adjustment to diagnosis/illness, weight and shape concerns, medical recommendations for diet (e.g., weight loss recommendations; treatment of hypoglycemia), the use of insulin and medications to control blood glucose and incretin production that may affect satiety, and comorbid psychopathology (Young-Hyman, 2012).

Treatment approaches such as cognitive-behavioral therapy, interpersonal therapy and integrative cognitive therapy with adjunctive pharmacotherapy to address comorbid psychiatric symptoms have been established as the standard of

care in the general population and can be adapted to address diabetes self-management behaviors in collaboration with the diabetes care team (Young-Hyman, 2012). Use of existing treatment approaches in patients with T1D and T2D should consider the incorporation of a multidisciplinary approach that involves the patient, family member(s), diabetes educator, dietitian with diabetes training, diabetologist, or endocrinologist in addition to the mental health provider to provide support and cross-disciplinary collaboration of recommendations to the patient (Goebel-Fabbri, 2009). As with all psychological interventions, physical safety is paramount to all other treatment considerations as eating disorders and DEB may result in metabolic dysregulation (e.g., extreme high blood glucose values or diabetic ketoacidosis) which should be addressed medically. Similarly, intensive glycemic management is not recommended for patients with eating disorders until the symptoms of DEBs and eating disorders can be significantly reduced. Flexible food choices and incremental advances in insulin administration combined with emotional coping and problem-solving skills are recommended (Goebel-Fabbri, 2009).

### Severe Mental Illness

There is increasing evidence to document the relationship between SMIs and diabetes. The majority of research in SMI includes patients with schizophrenia and depression; however, some studies also include individuals with schizoaffective, bipolar, and other debilitating mental disorders (Druss, Zhao, Von Esenwein, Morrato, & Marcus, 2011; Allison et al., 2009). Obesity is highly prevalent among individuals with SMI and is an important contributor to their higher risk of T2D, cardiovascular disease, and premature mortality. Compared to the general population, the prevalence of obesity in those with SMI is significantly higher (29% vs. 17.7% for men and 60% vs. 28.5% for women; Daumit et al., 2003). Similarly, individuals with bipolar disorders have a higher prevalence of overweight, obesity, and metabolic syndrome compared to the general population (Fiedorowicz et al., 2008). Diabetes is 1.5 to 2 times more common in individuals with schizophrenia compared to the general population (Allison et al., 2009) independent of age, race, sex, use of atypical antipsychotic medications, and BMI (Regenold et al., 2002). Individuals with SMI have a higher rate of death and die at younger ages compared to the general population, primarily due to cardiovascular disease for which T1D and T2D are risk factors (Allison et al., 2009).

The higher prevalence of obesity, one of the strongest T2D risk factors, in those with depression and other SMI is related to three broad categories of exposures—medications, unhealthy lifestyle, and environmental factors. In the case of SMI, much of the work examining the risk of weight gain

and obesity with medications used to treat SMI has focused on the second-generation antipsychotics; however, mood stabilizers, such as lithium and valproate, have also been associated with weight gain (Allison et al., 2009). Tricyclic antidepressants and selective serotonin reuptake inhibitors have been implicated in weight gain and the magnitude of effects vary across medications within each class (Zimmermann, Kraus, Himmerich, Schuld, & Pollmächer, 2003).

Individuals with diabetes and depression or SMI may also have low socioeconomic status, stigma, discrimination, lack of social support, and poor health behaviors (Mueser & McGurk, 2004). For example, those with SMI are less likely to exercise (Daumit, Goldberg, Anthony, & Dixon, 2004) and more likely to consume diets higher in calories and fat and low in fruits and vegetables (Amani, 2007) compared to the general population. Individuals with SMI are more likely to be unemployed, live with low income, and have less educational attainment (Draine, Salzer, Culhane, & Hadley, 2002), which may disrupt health care maintenance and prevention, increasing the risk for metabolic disorders (Draine et al., 2002).

### Screening and Treatment

The American Diabetes Association (ADA) recommends screening patients for cardiometabolic risk factors prior to or just after initiation of any antipsychotic medication (ADA, The American Psychiatric Association, The American Association of Clinical Endocrinologists, & The North American Association for the Study of Obesity, 2004). If metabolic disorders are identified, providers should initiate appropriate treatment or refer the patient to the appropriate medical specialist (e.g., general internist, endocrinologist). Medical therapies for diabetes and its conditions are the same for those with SMI as they are for the general population (ADA et al., 2004). Individuals who are overweight or obese should receive physical activity and nutrition counseling and be referred to a weight management program (ADA et al., 2004). Patients who develop diabetes should be referred to an ADA-recognized diabetes self-management education program (ADA et al., 2004). The best evidence suggests that behavioral interventions are beneficial in lowering BMI and weight in individuals with SMI (McGinty & Daumit, 2011) and are most effective when behavioral weight loss programs are adapted to accommodate the high prevalence of psychiatric symptoms and cognitive impairment in those with SMI (Daumit et al., 2013).

### Psychological Conditions and Disease Outcomes

The impact of each of the psychological conditions described above can be assessed in terms of diabetes outcomes with particular emphasis on average blood glucose control, diabetes complications, and adherence to self-care behaviors.

## Psychological Conditions and Glycemic Control

A1c has been established as the benchmark physiologic outcome variable for T1D and T2D due to the linear association of sustained elevated A1c values (defined as >7.0%) and the development of diabetes complications in T1D (The Diabetes Control and Complications Trial Research Group, 1993) and T2D (U.K. Prospective Diabetes Study Group, 1998). Most psychological conditions have been found to be associated with poorer glycemic control or elevated A1c.

Evaluation of the association of diagnosed depression and depressive symptoms with glycemic control has indicated a small effect size for T1D (Cohen's  $r = .19$ ) and T2D (Cohen's  $r = .14$ ) in a meta-analysis of predominantly cross-sectional studies of adults (Lustman et al., 2000). A stronger association between depressive symptoms and a range of micro- and macrovascular diabetes complications has been found for both T1D (e.g., Cohen's  $r = .21$ ; de Groot, Anderson, Freedland, Clouse, & Lustman, 2001) and T2D (Cohen's  $r = .27$ ; de Groot et al., 2001; Pouwer, Nefs, & Nouwen, 2013). Additional studies of T2D women have demonstrated that a lifetime history of major depressive disorder—even in full remission—continues to confer risk for worse outcomes years and even decades later including worse glycemic control, impaired cardiovascular functioning, and dysregulated physiological reactivity to acute stressors (e.g., Wagner, Finan, Tennen, White, & Burg, 2011).

Depression in the context of T1D and T2D is also associated with increased health care costs (Egede, Zheng, & Simpson, 2002), greater disability (Egede, 2004), and early mortality (Gallo et al., 2005; Katon et al., 2008; Molosankwe, Patel, Gagliardino, Knapp, & McDaid, 2012). Early mortality associated with T1D and T2D and depression is attributable to a variety of medical causes (Park, Katon, & Wolf, 2013).

The association of anxiety symptoms and T1D and T2D adults are less clear. An early meta-analysis showed that the presence of an anxiety disorder was associated with worse glycemic control while elevated anxiety symptoms were not (Anderson et al., 2002). A recent study of Hispanics with diabetes found that the presence of anxiety symptoms was associated with poorer A1c (Kendzor et al., 2014). Further studies in adults are warranted with careful attention to ascertainment of anxiety and sociocultural factors. Although there are limited data regarding co-occurring T2D and PTSD, some studies show that people with both conditions have worse glycemic control (Miller et al., 2011) and a worse metabolic and anthropometric profile (Trief, Ouimette, Wade, Shanahan, & Weinstock, 2006) than people with T2D who do not have PTSD.

In cross-sectional and longitudinal investigations of T1D samples, DEBs and eating disorders have been found to be associated with hyperglycemia (Young et al., 2013) and microvascular complications such as diabetic retinopathy,

microalbuminuria (antecedent to renal disease), proteinuria, peripheral neuropathy, as well as short-term lipid abnormalities, and hospitalizations for diabetic ketoacidosis (Young-Hyman & Davis, 2010). In the limited literature on eating disorders and night eating behaviors in T2D, there are mixed findings on the association of these disorders on glycemic control (Çelik et al., 2015; Hood et al., 2014; Nicolau et al., 2015). Little is known about the relationship of DEBs and eating disorders with long-term diabetes complications in T2D samples.

## Psychological Conditions and Self-Care Behaviors

All of the psychological conditions in this review have been found to be associated with decreased diabetes self-care behaviors in adults. Depression and depressive symptoms are associated with poor adherence to therapeutic recommendations such as medical appointments, diet, exercise, medication use, glucose monitoring, and foot care (Gonzalez et al., 2008) as well as impaired problem solving skills, a core component in the self-management of diabetes. Poor problem solving skills are independently associated with poor metabolic control in persons with diabetes (Hill-Briggs & Gemmell, 2007).

Anxiety worsens diabetes self-care (Janzen Claude, Hadjistavropoulos, & Friesen, 2014), including adherence to dietary recommendations (Talbot, Maguen, Epel, Metzler, & Neylan, 2013). Similarly, DEBs and eating disorders are highly correlated with decreased adherence to self-care behaviors, particularly insulin administration. Intentional lowered insulin doses or insulin omission (i.e., skipped doses) to avoid weight gain can result in diabetic ketoacidosis leading to coma and death.

## Conclusion

T1D and T2D are emotionally and cognitively demanding diseases that place patients at risk for a variety of psychological conditions. The accumulated literature describes a reciprocal set of relationships in which some psychological conditions increase the risk for incident diabetes and some conditions develop in the context diabetes diagnosis or exacerbation of complications and have the potential to worsen diabetes outcomes. The treatment literature shows that intervening on psychological disorders can be effective on psychological outcomes and *may* improve glycemic control.

As rates of T1D and T2D continue to rise (ADA, 2014) and the burden of depression and other psychological conditions increases globally, there is an acute need to better understand and intervene on the interplay between psychological conditions and diabetes. This requires innovative research and integration of medical and psychological treatment that crosses traditional academic silos. For example,

clear and comprehensive biopsychosocial models of diabetes and its common conditions have yet to be validated and much remains to be learned about the biological pathways that initiate and maintain psychological conditions before and after diagnosis of T1D and T2D.

Second, the current literature is limited by relatively small samples drawn from predominantly White mainstream cultural groups, cross-sectional research methods that typically do not utilize control groups, variability in the use of measures designed to capture psychological and disease constructs that limit comparison across trials, variability in methodological rigor across trials and lack of replication trials. Greater precision and methodological rigor is required to further refine many of the relationships between variables. For example, much of the accumulated literature on 'depression' is highly heterogeneous in terms of depression assessment methods (e.g., self-report questionnaires vs. psychiatric diagnostic interview protocols). Similarly, there are no studies that have examined prevalence rates or treatment of depression in T1D adult samples. Some studies have combined T1D and T2D patient samples with sample sizes for T1D that are too small to conduct meaningful subgroup analyses. Other studies have elected to omit T1D from data collection. This has resulted in a paucity of literature on a physiologically distinct and important segment of the population with diabetes.

Greater precision is also needed in understanding the co-occurrence and interaction among psychological conditions affecting individuals with T1D and T2D. For example, the multifactorial construct of diabetes-related distress has been found to be correlated with poor glycemic control and shares some variance with depressive symptoms (Fisher et al., 2007). Measures have been developed to capture this disease-specific phenomenon in T1D and T2D patients, family members, and providers (Welch, Jacobson, & Polonsky, 1997; Polonsky et al., 2005; Franks et al., 2012). Much remains to be learned about the ways that diabetes distress may interact, exacerbate or result from depressive symptoms in T1D and T2D patients and the further development of empirically validated interventions for those with diabetes distress alone, diabetes distress with depressive symptomatology, and those with depressive symptomatology alone. Robust study designs that yield greater understanding beyond simple associations are needed to better understand the overlapping and unique contributions of diabetes and co-occurring conditions exists for all of the conditions described in this review.

In terms of interventions, it remains unknown whether intervening on psychological risk factors such as depression and PTSD reduces the risk for incident diabetes, and if so, which treatments are most effective. Greater research is needed to establish empirically validated interventions to treat psychological conditions in T1D and T2D populations across the life span, especially among older adults and

cultural subgroups. Among the current empirically validated interventions, there is a need to identify which interventional elements (e.g., cognitive-behavioral therapy) are most effective, and under which conditions, for patients with T1D and T2D. Psychologists in clinical practice will be called upon to maximize mental health and fiscal outcomes for these patients making psychologists critical providers of care.

In sum, psychological science and practice have much to offer to patients and families with T1D and T2D but much work remains to be done. Psychologists are well-positioned with the tools and expertise needed to make advances in models of diagnosis, prevention, and treatment of psychological conditions in patients with diabetes. Our engagement in this area will continue to be vital to improving outcomes for individuals and families with diabetes.

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Received March 12, 2015

Revision received December 22, 2015

Accepted March 22, 2016 ■