Prevalence and Correlates of Depression in Individuals With and Without Type 1 Diabetes

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OBJECTIVE — Depression is associated with poor glycemic control and complications in people with type 1 diabetes. We assessed the prevalence of depression and antidepressant medication use among adults with and without type 1 diabetes and the association between depression and diabetes complications.

RESEARCH DESIGN AND METHODS — In 2006–2008, the Coronary Artery Calcification in Type 1 Diabetes Study applied the Beck Depression Inventory II (BDI-II) to 458 participants with type 1 diabetes (47% male, aged 44 ± 9 years, type 1 diabetes duration 29 ± 9 years) and 546 participants without diabetes (nondiabetic group) (51% male, aged 47 ± 9 years). Use of antidepressant medication was self-reported. Depression was defined as a BDI-II score >14 and/or use of antidepressant medication. Occurrence of diabetes complications (retinopathy, blindness, neuropathy, diabetes-related amputation, and kidney or pancreas transplantation) was self-reported.

RESULTS — Mean BDI-II score, adjusted for age and sex, was significantly higher in participants with type 1 diabetes than in nondiabetic participants (least-squares mean ± SE: 7.4 ± 0.3 vs. 5.0 ± 0.3; $P < 0.0001$). Type 1 diabetic participants reported using more antidepressant medications (20.7 vs. 12.1%, $P = 0.0003$). More type 1 diabetic than nondiabetic participants were classified as depressed by BDI-II cut score (17.5 vs. 5.7%, $P < 0.0001$) or by either BDI-II cut score or antidepressant use (32.1 vs. 16.0%, $P < 0.0001$). Participants reporting diabetes complications ($n = 209$) had higher mean BDI-II scores than those without complications (10.7 ± 9.3 vs. 6.4 ± 6.3, $P < 0.0001$).

CONCLUSIONS — Compared with nondiabetic participants, adults with type 1 diabetes report more symptoms of depression and more antidepressant medication usage. Depression is highly prevalent in type 1 diabetes and requires further study on assessment and treatment.

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Type 1 diabetes is a chronic illness that requires continuing medical care, education, and diligent patient self-management to prevent acute complications and to reduce the risk of long-term complications. Yet many patients do not achieve glycosylated hemoglobin (GHb) levels <7.0%, the American Diabetes Association goal to prevent complications (1). Depression is a modifiable risk factor whose treatment could improve glycemic control and health outcomes in patients with type 1 diabetes.

In people with diabetes, depression has been associated with hyperglycemia (2,3); lower levels of diabetes self-care (2); complications, including coronary/cardiovascular disease (4–7), neuropathy (6,8), and retinopathy (3,6); and increased mortality (9). However, few data are available regarding prevalence of depression in individuals with type 1 diabetes compared with the general population. A meta-analysis carried out by Anderson et al. (10) led to the conclusion that the prevalence of depression in adults with any type of diabetes is double that of individuals without diabetes. Since then, results from the 2006 Behavioral Risk Factor Surveillance System (11) have found the age-adjusted prevalence of major depression in individuals with diabetes to be 8.3%, while the estimated prevalence of major depression in the general U.S. population is 5.3% (12). Hislop et al. (13) found that over one-third of young adults with diabetes experience psychological distress.

These findings must be interpreted with caution, however. Many studies on the subject of depression and diabetes have methodological limitations such as lack of control group, small sample size, and failure to distinguish between type 1 and type 2 diabetes (10,14). Where there is no control group, recruitment bias can limit generalizability. Since type 1 and type 2 diabetes differ considerably in terms of age of onset, duration, day-to-day management, presence of comorbid conditions, and nature and onset of complications, depression may affect the two conditions differently and different processes may be involved in the development of depression in individuals with type 1 and type 2 diabetes. Therefore, inferences from combined groups may not represent diabetes type-specific prevalence of depression. As an example, of 42 studies analyzed in the Anderson meta-analysis, 22 (52%) did not use a control group, and of 20 controlled studies, only 3 reported separate results for type 1 and type 2 diabetes (10).

A recent review of the literature (14) states that it is not yet possible to conclude that depression is more prevalent in individuals with type 1 diabetes than in age-matched control subjects due to widely varying diagnostic techniques, small sample sizes, inadequate control groups, and failure to distinguish between types of diabetes in previous studies. In this study, we aimed to assess the prevalence of depression defined by self-reported questionnaire and/or antidepressant drug use in a cohort of adults with and without type 1 diabetes. We also aimed to confirm previous findings suggesting that depression is associated with

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elevated GHb, coronary artery calcification (CAC), and diabetes complications in adults with type 1 diabetes.

RESEARCH DESIGN AND METHODS

— The data presented in this report were collected as part of the third study visit in the Coronary Artery Calcification in Type 1 Diabetes (CACTI) Study from 2006 to 2008. At the time of this analysis, 1,130 CACTI participants had completed the third CACTI study visit. Participants without diabetes were recruited from the community and include spouses, neighbors, and friends of type 1 diabetic participants to reduce potential differences in socioeconomic and educational factors. Questionnaires were completed by 1,004 participants (88.9% of third-visit participants), including 458 type 1 diabetic participants (47% male, aged 44 ± 9 years, type 1 diabetes duration 29 ± 9 years) and 546 nondiabetic participants (51% male, aged 47 ± 9 years). One-hundred and twenty-six subjects who did not complete the BDI-II tended to be younger (42.0 ± 8.5 vs. 45.5 ± 9.0; $P < 0.0001$) than those who were included in the study but were not different in terms of BMI, A1C, duration of diabetes, or CAC.

The Beck Depression Inventory II (BDI-II) is a 21-item self-report instrument that assesses the severity of depressive symptomatology in adolescents and adults. It is a revised version of the original BDI (15), updated to correspond to criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (16). Each of the 21 items measures the presence and severity of a somatic or cognitive symptom of depression, rated on a 4-point scale ranging from 0 to 3. The ratings are summed, yielding a total score that can range from 0 to 63. The BDI-II has been validated as a sensitive, specific, and predictive tool for measuring depression (17). Despite the fact that the somatic symptoms of preexisting medical conditions such as diabetes may overlap those of depression, studies have shown that the somatic items do not interfere with the discriminative capacity of the BDI-II in primary-care settings (18) or in participants with diabetes (19). The BDI-II has also been shown to be sensitive and specific at any phase of a depressive disorder (20).

Use of antidepressant medication was self-reported (participants brought their medications to the study visit for verification) and included use of selective serotonin reuptake inhibitors, norepinephrine/dopamine reuptake inhibitors, selective serotonin/norepinephrine reuptake inhibitors, selective norepinephrine reuptake inhibitors (SNRIs), tetracyclic antidepressants, and tricyclic antidepressants. Depression was defined as use of at least one antidepressant medication and/or, as in previous studies (3,5), a BDI-II score >14. Occurrence of diabetes complications (retinopathy, blindness, neuropathy, diabetes-related amputation, and kidney or pancreas transplantation) was self-reported.

To assess CAC, all patients underwent two electron-beam computed tomography scans within 5 min without contrast at baseline and two scans at follow-up. Images were obtained of the entire epicardial system using an Imatron C-150 Ultrafast CT scanner (Imatron, South San Francisco, CA), with a 100-ms exposure. The standard acquisition protocol was used (21). Scanning started from near the lower margin of the bifurcation of the main pulmonary artery. Images were electrocardiographically triggered at 80% of the R-R interval, and 30–40 contiguous 3-mm slices were acquired. Calcified coronary artery areas were identified as those with a minimum density of 130 Hounsfeld units (HU) and a minimum area of three pixels (1.03 mm^2). A calcium score for each region was calculated by multiplying the area by the density score (one for 130–199, two for 200–299, three for 300–399, and 4 for >399 HU). A total CAC score in Agatston units (AU) was calculated by adding up scores for all slices separately for left main, left anterior descending, circumflex, and right coronary arteries. The volume scores were calculated using the volumetric method, which is based on isotropic interpolation (22).

Statistical methods

Descriptive statistics were reported as means and frequencies with *P* values for differences between groups. ANCOVA, independent-sample *T* tests, and Pearson correlations were used for hypothesis testing for continuous outcomes, and the χ^2 test of independence and logistic regression were used for hypothesis testing for categorical outcomes. Wilcoxon's rank-sum tests were used to compare CAC scores among depressed and nondepressed participants, type 1 diabetic participants versus nondiabetic control subjects, and depressed versus nondepressed within diabetes type. Due to the nonnormal distribution of CAC scores with an abundance of scores of zero, logistic regression was used to model the probability of a CAC score >0 for type 1 diabetes status (yes or no), depression (yes or no), and depression within diabetes category (yes or no), adjusted for age and sex. A *P* value of <0.05 was considered statistically significant. SAS 9.1 was used for all statistical analysis.

RESULTS

— Characteristics of the 1,004 participants in this study were as follows: 458 type 1 diabetic patients (47% male, aged 44 ± 9 years, type 1 diabetes duration 29 ± 9 years) and 546 nondiabetic participants (51% male, aged 47 ± 9 years) (Table 1). In our sample, the prevalence of depression (as defined by BDI-II >14 and/or antidepressant use) in participants with type 1 diabetes was significantly higher than that of age- and sex-adjusted nondiabetic participants (32.1 vs. 16.0%, $P < 0.0001$). A BDI-II score >14 was seen in 17.5% of type 1 diabetic participants compared with 5.7% in nondiabetic participants ($P < 0.0001$). Antidepressant use was reported in 20.7% of type 1 diabetic participants compared with 12.1% of nondiabetic participants ($P = 0.0003$). The length of antidepressant use was 3.65 ± 3.37 years among those with diabetes and 2.88 ± 3.08 years for control subjects ($P = 0.15$). Among those being treated for depression, 69.9% of type 1 diabetic participants and 84.6% of nondiabetic participants had BDI-II scores ≤14 at the time of the study ($P = 0.03$). Participants with type 1 diabetes were 3.52 (2.28–5.43) times more likely to have a BDI-II score >14, were 2.48 (1.83–3.36) times more likely to have a history of depression than nondiabetic participants ($P < 0.0001$ for each), and were 1.89 (1.34–2.67) times more likely to be on antidepressant medications than nondiabetic participants ($P = 0.0003$) in unadjusted analyses. In logistic regression adjusted for age and sex, participants with type 1 diabetes were 3.66 (2.35–5.71) times more likely to have a BDI-II score >14, were 2.60 (1.90–3.56) times more likely to have a history of depression than nondiabetic participants ($P < 0.0001$ for each), and were 1.99 (1.39–2.84) times more likely to be on antidepressant medications than nondiabetic participants ($P = 0.0002$). Participants with diabetes were also more likely to have moderate depression (BDI-II ≥20) than those with-

Table 1—*Participant characteristics*

	Type 1 diabetic subjects	Nondiabetic subjects
n	458	546
Age (years)	43.6 ± 8.9	47.0 ± 8.7
Sex (men)	214 (46.7)	277 (50.8)
Diabetes duration (years)	29.4 ± 8.7	
GHb (%)	7.9 ± 1.2	5.6 ± 0.5
Race/ethnicity (% non-Hispanic white)	94.8	86.6
BMI (kg/m^2)	26.8 ± 4.8	26.6 ± 4.8
BDI-II score	7.4 ± 7.3	5.0 ± 5.4
Antidepressant medication use (yes)	93 (20.7)	65 (12.1)
Length of time on antidepressant medication (years)	3.65 ± 3.37	2.88 ± 3.08
Complications (total)	209 (45.7)	
Retinopathy	166 (36.5)	
Blindness	27 (6.0)	
Neuropathy	97 (21.4)	
Amputation	6 (1.3)	
Transplantation	6 (1.3)	
Presence of CAC (yes)	249/419 (59.4)	233/493 (47.3)
Square root of CAC volume	5.96 ± 9.27	3.26 ± 6.56

Data are means ± SD, *n* (%), or proportion in unadjusted analysis unless indicated otherwise.

out diabetes (6.1 vs. 2.4%, $P = 0.003$; odds ratio 2.65 [95% CI 1.34–5.23], $P = 0.005$.)

Mean BDI-II score, adjusted for age and sex, was significantly higher in participants with type 1 diabetes than in nondiabetic participants (least-squares means ± SE: 7.4 ± 0.3 vs. 5.0 ± 0.3; $P < 0.0001$). Depression by BDI-II cut score and/or antidepressant use was more prevalent in women with type 1 diabetes (37.9%) than in nondiabetic women (20.5%, $P < 0.0001$) and in men with type 1 diabetes than in nondiabetic men (25.5 vs. 11.6%, $P < 0.0001$) (Table 2). Within type 1 diabetes participants, there was still a difference between men (25.5%) and women (37.9%) in the frequency of depression by BDI-II cut score and/or antidepressant use ($P = 0.005$) but not when depression was defined only by a BDI-II score (14.5% for men vs. 20.1% for women, $P = 0.12$). Depression defined by BDI-II score alone was more prevalent in women with type 1 diabetes than in nondiabetic women (20.1 vs. 7.8%, $P < 0.0001$) and in men with type 1 diabetes than in nondiabetic men (14.5 vs. 3.6%, $P < 0.0001$). Women with type 1 diabetes had higher BDI-II scores than nondiabetic women (7.6 ± 7.3 vs. 5.7 ± 5.9, $P = 0.0012$), and men with type 1 diabetes had higher BDI-II scores compared with nondiabetic men (7.1 ± 7.3 vs. 4.3 ± 4.8, $P < 0.0001$). However, BDI scores were similar among men and women with type 1 diabetes (7.1 ± 7.3 vs. 7.6 ± 7.3, $P = 0.50$).

Table 2—*Prevalence of depression by sex and diabetes*

	Men	Women	*P*	All
Type 1 diabetes				
BDI-II >14	14.5*	20.1†	0.12	17.5‡
Antidepressant use	13.7	26.8§	0.0007	20.7‡
BDI-II > 14 or medications	25.5*	37.9†	0.005	32.1‖
No diabetes				
BDI-II >14	3.6	7.8	0.032	5.7
Antidepressant use	8.4	16.0	0.007	12.1
BDI-II >14 or medications	11.6	20.5	0.005	16.0

Data are percent, unless otherwise indicated. *$P < 0.0001$ for men with type 1 diabetes vs. men without. †$P < 0.0001$ for women with type 1 diabetes vs. women without. ‡$P < 0.0001$ for all with type 1 diabetes compared with all control subjects. §$P = 0.003$ for women with type 1 diabetes vs. women without. ‖$P = 0.0003$ for all with type 1 diabetes compared with all control subjects.

Current GHb was not correlated with BDI-II score in type 1 diabetic participants ($r = 0.07$, $P = 0.14$); however, type 1 diabetic participants with a history of depression had slightly higher GHb than those without depression (8.1 ± 1.2 vs. 7.8 ± 1.1%, $P = 0.013$). Type 1 diabetic participants reporting the presence of at least one complication ($n = 209$) had significantly higher BDI-II scores than those without complications (8.8 ± 8.2 vs. 6.1 ± 6.1, $P < 0.0001$) and were more likely to be depressed by BDI-II cut score (23.4%) than type 1 diabetic participants without complications (12.1%, $P = 0.002$). Both those with (8.8 ± 8.2) and those without (6.1 ± 6.1) complications had significantly higher BDI-II scores than control subjects (5.0 ± 5.4, $P < 0.05$ both), had higher prevalence of depression by BDI-II scores, BDI-II or antidepressant use, and antidepressant use alone (online appendix Table A1 [available in the online appendix at http://care.diabetesjournals.org/cgi/content/full/dc08-1835/DC1]). Among those with diabetes, the number of complications was positively correlated with BDI-II total scores ($R = 0.25$, $P < 0.0001$).

Of 1,004 participants with BDI-II scores, 421 patients with type 1 diabetes and 494 nondiabetic participants had information available on CAC. Due to the highly skewed distribution of CAC scores, we performed logistic regression modeling with the probability of CAC >0. More participants with type 1 diabetes had CAC compared with nondiabetic participants (59.6 vs. 47.2%, $P = 0.0002$). Also, type 1 diabetic participants who were depressed (BDI-II score >14) were more likely to have CAC than those who were not depressed (74.7 vs. 56.6%, $P = 0.005$) (see online appendix Table A2). However, this finding was not the same among nondiabetic participants who were depressed compared with those who were not depressed (44.4 vs. 47.3%, $P = 0.77$). Defining depression by either BDI-II score or antidepressant use, type 1 diabetic participants were more likely to have CAC than those who were not depressed (67.4 vs. 56.1%, $P = 0.029$), and participants with type 1 diabetes who were depressed by a BDI >14 were 2.35 (95% CI 1.25–4.39, $P = 0.01$) times more likely to have a CAC score >0 than participants with type 1 diabetes without depression in adjusted analysis (Table 3). However, there was no relationship between depression by BDI-II or medication

Table 3—*Adjusted odds ratios of CAC score >0 by depression and type 1 diabetes status

	Type 1 diabetes		No diabetes	
Classification	CAC score >0	*P*	CAC score >0	*P*
Depressed by BDI-II >14	2.35 (1.25–4.39)	0.01	1.02 (0.44–2.36)	0.96
Antidepressant use	1.35 (0.78–2.34)	0.37	1.29 (0.70–2.37)	0.41
BDI or medications	1.80 (1.11–2.92)	0.017	1.12 (0.65–1.91)	0.69

Data are odds ratio (95% CI), unless otherwise indicated. *Adjusted for age, sex, and duration of diabetes in type 1 diabetic subjects and age and sex for control subjects.

use and a CAC score >0 among nondiabetic participants (data not shown).

Of the control subjects, 172 had a partner in the study with diabetes, 179 had a partner in the study without diabetes, and 195 did not have a partner in the study. There was no difference in prevalence of depression by the BDI-II (5.2 vs. 5.0 vs. 6.7%, $P = 0.76$), use of antidepressant therapy (12.8 vs. 10.6 vs. 12.3%, $P = 0.90$) or history of depression by BDI-II or antidepressant use (15.7 vs. 14.0 vs. 17.4%, $P = 0.76$) among those with a partner living with diabetes, those with a nondiabetic partner, and those without a partner in the study, respectively. There was also no difference in BDI-II scores among the three types of control subjects (partner with type 1 diabetes [4.67 ± 4.84], partner without type 1 diabetes [5.11 ± 5.43], and no partner in the study [5.16 ± 5.75]; $P = 0.65$).

CONCLUSIONS — Despite published data on depression and diabetes, these results are some of the first to specifically demonstrate an increased prevalence of depression among adults with type 1 diabetes compared with age- and sex-matched control subjects. We have shown that adults with type 1 diabetes are more than twice as likely as adults without diabetes to have depression as assessed by BDI-II >14 and/or current antidepressant use. In this sample, those with type 1 diabetes were more than three times as likely to have a clinically significant score on the BDI-II and almost twice as likely to be on antidepressant medication as nondiabetic adults.

Previous literature has estimated the rate of depression in diabetes to be between 3.8 and 27.3% (11). In this large cohort, the prevalence of depression in type 1 diabetes was 32.1% defined by either BDI-II score >14 or antidepressant medication use. Previous studies have used a wide variety of measures and criteria for diagnosing depression, and BDI-II scores of >14 are considered indicative of mild depression. Another possible explanation is that depression may be more common in type 1 than in type 2 diabetes, since previous studies have mainly reported on a mixed sample of type 1 and type 2 diabetes. In addition, the inclusion of current antidepressant medication use as an indicator of depression in this study may capture individuals successfully treated for depression who may no longer score high on depression assessments.

Findings regarding the association between depression and hyperglycemia have been inconsistent in the literature. Although GHb was not significantly correlated with BDI-II score in all participants with type 1 diabetes, we found a significant relationship between GHb and a history of depression among participants with type 1 diabetes. This could be due in part to the data being cross-sectional. This lack of significant correlation has been seen in previous studies (23), although a significant correlation has been found elsewhere (2,13).

Our data confirm previous findings regarding the association of depression with complications of diabetes. Type 1 diabetic participants reporting the presence of at least one diabetes complication scored significantly higher on the BDI-II than participants with type 1 diabetes with no complications. Coronary artery disease (CAD) is the leading cause of mortality in people with type 1 diabetes (7), and even mild manifestations of depression (BDI scores >10) are related to carotid plaque formation (24), making depression an important risk factor to evaluate in patients with type 1 diabetes. In the Pittsburgh Epidemiology of Diabetes Complications Study, both CAC and BDI-II score were independently correlated with clinical CAD, and presence of CAC was predictive of clinical CAD 84 and 71% of the time in men and women, respectively (4). We report that CACTI study participants with type 1 diabetes who were depressed were two times more likely to have a CAC score >0.

The etiology of the relationship between type 1 diabetes and depression is likely to be multifactorial. First, depression may be a response to the psychosocial stress caused by living with the demands and constraints imposed by type 1 diabetes. Second, biological processes specific to diabetes, such as insulin resistance, changes in brain structures such as the hippocampus, and inflammatory processes, may be related to psychological symptoms (7). Third, because both conditions are prevalent, they may coexist coincidentally. Further research into the mechanisms involved in the observed relationship is needed.

There are several important strengths of this study. As previously discussed, this is the first large-scale assessment of depression in a type 1 diabetes–specific population that uses a control group composed of friends, neighbors, and spouses to reduce the potential for selection bias in socioeconomic status and related factors. Also, the use of both a well-validated depression assessment and antidepressant medication use as indicators of depression improves ascertainment of cases to include both currently treated and untreated depression.

This study has several limitations. First, the use of a self-report questionnaire is cost- and time-effective, but the preferred method of psychological diagnosis is generally by diagnostic interview with a psychologist. It has been suggested that self-report questionnaires may overestimate prevalence of depression compared with diagnostic interview (10,14), and it may be more appropriate to use the term "clinically significant levels of depressive symptoms" in the context of this study. However, diagnostic interviews identify major depressive disorder but may exclude other clinically relevant presentations. The presence of depressive symptomatology in general may be better assessed with a tool such as the BDI-II. Another limitation of this study is its cross-sectional nature, meaning that cause-effect relationships cannot be determined. Since symptoms and therefore measurements related to both diabetes and depression can fluctuate significantly over time, a longitudinal design may give a more accurate picture of this relationship. In type 1 diabetic participants, for example, GHb captured at a specific point in time may reflect a number of mediating circumstances and is not as informative as

GHb pattern over long periods of time. It is possible that a stronger correlation between glycemic control and depression in type 1 diabetes would be found with a longitudinal study design.

Depression can severely compromise day-to-day functioning and, in the absence of treatment, tends to follow a chronic or relapsing course (8). In addition to the detrimental effects on essential daily self-care behaviors, there are significant health care costs associated with depression in patients with diabetes (25). Screening patients with type 1 diabetes for depressive symptoms is vital. Our data also suggest that appropriate intervention is especially important in patients with complications of diabetes, as they are especially likely to suffer from depressive symptoms according to our data. Treatment of depression should be accompanied by prospective assessment of its efficacy in improving mental health symptoms as well as diabetes health outcomes.

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