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Is Ignorance Bliss? Depression, Antidepressants, and the Diagnosis of Prediabetes and Type 2 Diabetes

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Abstract

Objective—To examine the association between clinically identified and undiagnosed prediabetes and Type 2 diabetes with depression and antidepressant medication use.

Methods—Data come from the National Health and Nutrition Examination Study (2005 and 2007), a population-based cross-sectional survey. Analysis is limited to adults aged 30 and older (n = 3,183, Mean age = 52.1 year). Depression syndrome was measured by the Patient Health Questionnaire-9. Participants were categorized using fasting glucose levels as normoglycemic (glucose <100 mg/dL), undiagnosed prediabetes (glucose 100–125.9), clinically identified prediabetes (glucose 100–125.9 plus clinician diagnosis), undiagnosed Type 2 diabetes (glucose >126), and clinically identified Type 2 diabetes (glucose >126 plus clinician diagnosis or use of antidiabetic medications). Health behaviors included smoking, poor diet, excessive alcohol use, and obesity. Health promotion behaviors included efforts to change diet, lose weight, and increase physical activity.

Results—Clinically identified diabetes was associated with 4.3-fold greater odds of depression, but undiagnosed diabetes was not significantly associated with depression. This relationship was more pronounced for prediabetes. Clinically identified diabetes was associated with 1.8-fold greater odds of antidepressant use, but undiagnosed diabetes was not significantly associated with antidepressant use. Health behaviors were not consistently related to depression syndrome.

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Conclusion—The relationship between diabetes status and depression and antidepressant use depends on whether the diabetes has been clinically identified. Findings are consistent with the hypothesis that the relationship between diabetes and depression may be attributable to factors related to disease management. Previous reports linking antidepressants and diabetes may be attributable to clinical ascertainment bias.

Keywords

depression; diabetes; comorbidity; antidepressants; coping

Prospective, population-based studies have consistently indicated a bidirectional relationship between depression and Type 2 diabetes. A recent meta-analysis reported that depression is associated with approximately 60% elevated risk of incident Type 2 diabetes, and that Type 2 diabetes is only associated with a modest elevation in risk of new depression (Mezuk, Eaton, Albrecht, & Golden, 2008). More recent examinations have reported that Type 2 diabetes is associated with approximately 30% increased risk of developing depression (Golden et al., 2008; Pan et al., 2010). Among those with established Type 2 diabetes, depression is associated with poorer disease management, inadequate glycemic control, and risk of diabetes complications (de Groot, Anderson, Freedland, Clouse & Lustman, 2001).

There is evidence that the association between diabetes and risk of depression is strongest among those using more intense methods of diabetes control (e.g., insulin as opposed to oral medications) (Golden et al., 2008; Pan et al., 2010). In a longitudinal study Golden and colleagues (2008) reported that while diagnosed Type 2 diabetes was predictive of elevated depressive symptoms, undiagnosed diabetes was not related to higher levels of depressive symptomology and was potentially associated with lower risk of depression (Golden et al., 2008). This finding—combined with evidence that more intense diabetes management may be associated with greater risk of depression— have led some to ask whether “ignorance is bliss” (Golden & Mezuk, 2009). That is, is receiving a diagnosis of diabetes and the accompanying clinical recommendations to substantially change lifestyle (i.e., diet, exercise, alcohol use), rather than the state of chronic hyperglycemia, the “depressogenic” element in this comorbidity. A recent meta-analysis found that the prevalence of elevated depressive symptoms among persons with undiagnosed diabetes or prediabetes is lower than the prevalence among those with diagnosed diabetes (Nouwen et al., 2011), although the majority of these studies did not account for key confounders (e.g., race/ethnicity, health behaviors, comorbidity), which previous reports have demonstrated can substantially influence this association (Mäntyselkä et al., 2011). This limitation is important because recent evidence from epidemiologic studies has indicated that in the context of high levels of stress, engaging in poor health behaviors (i.e., alcohol use, smoking, poor diet) is associated with lower risk of depression for some groups (Jackson, Rafferty, & Knight, 2010; Mezuk et al., 2010), suggesting that efforts to modify health behaviors in stressful, socially disadvantaged contexts without feasible alternative coping behaviors may have the perverse consequence of increasing risk of depression.

Parallel to this evidence of diabetes subsequent to depression, several recent studies have suggested a link between antidepressant medication use and risk of Type 2 diabetes. However, the degree to which this association reflects confounding by indication is unresolved (Pan et al., 2010; Kivimäki et al., 2010a; Rubin et al., 2008; Rubin et al., 2010; Pan et al., 2011). It has been argued that the weight gain associated with some antidepressants may be a mediating mechanism linking depression and diabetes (Kivimäki et al., 2010b), but randomized controlled studies have demonstrated that antidepressant use is associated with improved glycemic control among patients with both depression and diabetes (Lustman et al., 2000; Lustman et al., 2006). It is also possible that persons with

clinically identified diabetes have more contact with health care providers as part of disease management, and this may increase the likelihood that depressive symptoms are identified by a clinician and treated with antidepressant medications; this process would introduce a spurious association between diabetes and antidepressant use through clinical ascertainment bias (Berkson, 1946).

The relationship between depression and Type 2 diabetes has implications for diabetes prevention and treatment practice. For example, if some diabetes care regimens are "depressogenic" in nature, this suggests that health care providers need to both routinely screen for depression among patients and integrate effective coping and problem-solving strategies into diabetes case counseling to reduce risk of subsequent depression (Katon et al., 2010). Similarly, if both depression and antidepressant medication use are associated with risk of diabetes, this indicates that providers need to focus on nonpharmacologic depression treatment strategies, particularly for patients with elevated diabetes risk (e.g., patients with a positive family history of diabetes, gestational diabetes, or overweight) (Rubin et al., 2010).

The purpose of this study was to evaluate the association between current depressive symptoms and antidepressant medication use with clinically identified and undiagnosed Type 2 diabetes. The secondary aim was to examine the associations of health behaviors and diabetes care regimens to depressive symptomology. If the association between Type 2 diabetes and depression arises as a result of the behavioral changes and associated psychological stress that result from receiving a diagnosis of diabetes, rather than through biological pathways via hyperglycemia, we expect that clinically identified diabetes, but not undiagnosed diabetes, will be associated with depressive symptomology. Similarly, if the association between Type 2 diabetes and antidepressant medications arises as a result of ascertainment bias through increased connection with the health care system or as a result of treating diabetes care-related distress, we expect that only clinically identified diabetes will be associated with antidepressant medication use.

Two main hypotheses were evaluated:

Hypothesis 1: Clinically identified, but not undiagnosed, diabetes is associated with elevated depressive symptoms

Hypothesis 2: Clinically identified, but not undiagnosed, diabetes is associated with greater use of antidepressant medications.

Finally, this study also examined whether the association between clinically identified diabetes and depression is partly attributable to the behavioral changes required to manage this condition. Specifically, we were interested in whether the relationship between diabetes and depression varies as a function of engaging in poor health behaviors (e.g., smoking, high-fat diet, excessive alcohol intake), health promotion behaviors (e.g., eating a low calorie diet, increasing physical activity), and diabetes care regimens (e.g., insulin and use of other medications).

Method

Participants and Procedures

Data are from the 2005 and 2007 cross-sections of the National Health and Nutrition Examination Survey (NHANES). NHANES is a nationally representative cross-sectional survey conducted by the National Center for Health Statistics (7Centers for Disease Control, 2004). Analysis were limited to non-Hispanic White, non-Hispanic Black, and Hispanic adults aged 30 or older (M_{age} = 52.08, SE = 0.49) who provided blood samples for assessment of fasting plasma glucose as part of the Mobile Examination Center (MEC)

component of the NHANES interview (1,501 in 2005 and 1,940 in 2007, for a total of 3,441 participants). After excluding participants with incomplete data on the Patient Health Questionniare-9 (PHQ-9) (n = 186), and those with clinically identified diabetes who reported an age of onset younger than age 30 or for whom age of onset was missing (n = 72), the final analytic sample size was 3,183. The two waves were combined for analysis, and weights were used to account for the stratified multistage probability sampling approach. Those excluded for missing data on the PHQ-9 (n = 186) were less likely to be non-Hispanic White (77.5% in the retained sample vs. 65.4% in the excluded sample, chi-square test of independence $\chi^2(2, n = 3369) = 9.13$, $p = .01$, $\phi_c = .04$). The age of those excluded from analysis ($M = 52.61$, $SE = 1.54$) did not significantly differ from those included ($M = 52.08$, $SE = 0.49$), $t(3369) = -.34$, $p = .740$. Chi-square tests of independence indicated that those excluded from analysis also did not differ in terms of diabetes status, $\chi^2(4, n = 3369) = 7.43$, $p = .11$, *ns*, education, $\chi^2(1, n = 3369) = 1.67$, $p = .20$, $\phi = .02$, *ns*, poverty-to-income ratio, $\chi^2(1, n = 3369) = 2.46$, $p = .12$, $\phi = .03$, *ns*, or antidepressant medication use, $\chi^2(1, n = 3369) = 1.92$, $p = .17$, $\phi = .02$, *ns*.

Measures

Type 2 diabetes—Type 2 diabetes status (normoglycemia, prediabetes, and diabetes) was defined according to American Diabetes Association (ADA) guidelines using fasting (for at least 8 hours) plasma glucose (American Diabetes Association, 2010). Participants were asked if they had ever been told by a physician that they had diabetes or high sugar, and their current medications were recorded. Diabetes and prediabetes were considered *clinically identified* if participants reported either that a physician had told them they had diabetes (indicated by a positive response to the question, "[Other than during pregnancy] Have you ever been told by a doctor or health professional that you have diabetes or sugar diabetes?" for diabetes and "Have you ever been told by a doctor or other health professional that you have any of the following: prediabetes, impaired fasting glucose, impaired glucose tolerance, borderline diabetes or that your blood sugar is higher than normal but not high enough to be called diabetes or sugar diabetes?"). Persons currently using hypoglycemic agents were classified as having clinically identified diabetes, not prediabetes, regardless of their response to these self-report items. Normoglycemia was defined as fasting plasma glucose <100 mg/dL and no use of hypoglycemic agents or physician diagnosis. Undiagnosed prediabetes was defined as fasting plasma glucose between 100 and 125.9 mg/dL and no report of physician diagnosis. Clinically identified prediabetes was defined as fasting plasma glucose between 100 and 125.9 mg/dL plus report of physician diagnosis. Undiagnosed Type 2 diabetes was defined as fasting plasma glucose >126 mg/dL and no report of physician diagnosis. Clinically identified Type 2 diabetes was defined as plasma glucose >126 mg/dL plus report of physician diagnosis or use of hypoglycemic agents.

Depression syndrome—Depression syndrome was assessed using the Patient Health Questionniare-9 (PHQ-9) (Kroenke, Spitzer & Williams, 2001). Individuals were categorized as likely being a case of major depression syndrome if they endorsed either dysphoria or anhedonia, plus four or more remaining items "more than half the days" in the past two weeks. Individuals were categorized as likely being a case of minor depression if they endorsed either dysphoria or anhedonia, and at least two, but less than four, of the remaining symptoms for at least "more than half the days." For both minor and major depression syndrome the item on suicidal ideation was counted if it was endorsed regardless of duration, consistent with the PHQ-9 diagnostic algorithm. The reliability and validity of the PHQ-9 has been assessed both in the general population and in clinical samples (Kroenke, Spitzer, Williams, Lowe, 2010), and this measure has moderate concordance with clinical diagnosis of major depressive disorder (Kappa ranging from 0.50–0.69) (Eaton, Hall, MacDonald & McKibben, 2007).

Medication use—Current prescription drug use was assessed by visual inspection of medication bottles by interviewers. Medications were classified using Lexicon Plus, a comprehensive database of all prescription and some nonprescription drug products available in the United States. Insulin or other medications with indications as antidiabetic agents (e.g., metformin) were categorized as diabetic medications, and any individual who reported taking these agents were categorized as having diagnosed Type 2 diabetes, as described above. Current use of all classes of antidepressant medications (e.g., selective serotonin reuptake inhibitors [SSRI], monoamine oxidase inhibitors [MAOI], or tricyclic antidepressants [TCA]) was collapsed into a single variable indicating antidepressant use. The majority (61%) of antidepressant agents used were SSRIs.

Poor health behaviors—Three types of poor health behaviors were assessed: smoking, excessive alcohol use, and poor eating habits. Cigarette smoking was dichotomized as current versus former/never smoker. Heavy alcohol consumption was defined as both being a current drinker and having an average of more than two drinks on days when alcohol was consumed (nondrinkers and those consuming two drinks or fewer were the reference group). Two proxy indicators of eating habits were derived from body mass index (BMI) and a 24-hr dietary recall. BMI was calculated from measured weight and height while wearing light clothing and dichotomized as obese ($\geq$30 kg/m^2) versus not obese (BMI <30 kg/m^2). Based on previous studies of diet and stress coping (Dallman et al., 2003), poor diet was defined as having low adherence to recommended levels of total fat, saturated fat, carbohydrates, and cholesterol consumption, based on the United States Department of Agriculture (USDA) guidelines (USDA, 2005). The measure of dietary adherence was created by assessing the number met of a possible four USDA guidelines using a single 24-hr dietary recall questionnaire. On average, participants endorsed one of the USDA recommended levels, and as a result having a poor diet was defined as being below the sample mean (e.g., meeting less than one dietary guideline). These four poor health behaviors were summed to create a count with scores ranging from 0 to 4 (M = 1.05, SE = 0.03), which was recategorized for analysis (range: 0–3+, M = 1.05, SE = 0.03) because of small cell sizes.

Health promotion behaviors—Three types of health promotion behaviors were assessed: increasing physical activity, controlling weight, and managing diet. Participants were asked both whether they had been *told* by a physician to engage in these behaviors, and whether they were *trying* to engage in the behaviors. A count of the number of physician recommendations was created by summing these behaviors (range: 0–3, M = 0.93, SE = 0.03). A second count of the number of positive health behaviors the respondent was attempting was also created (range: 0–3, M = 1.58, SE = 0.04). Respondents with clinically identified diabetes were also asked the number of times in an average day they check their blood sugar (range: 0–6, M = 1.30, SE = 0.06).

Demographic variables and other covariates—Demographic characteristics (age, sex, race/ethnicity, education) were assessed by self-report. Race/ethnicity was categorized as non-Hispanic White, non-Hispanic Black, or Hispanic. Education was measured in years, and dichotomized as high school completion or less versus more than high school. Household poverty-to-income ratio (PIR), a measure of a family's poverty threshold accounting for family size and composition, was used to index socioeconomic status; poverty status was defined as a PIR $\leq$1.85, based on the eligibility cutpoint for USDA food assistance programs (supplemental nutrition program for women, infants, and children; food stamps; etc.). Health insurance was dichotomized as yes if participants reported having health insurance or being covered by a health care plan, including Medicaid or Medicare. Number of health care visits in the past year, categorized as none, one, 2 to 3, 4 to 9, 10 to 12, and 13 or more, was also assessed. The presence of three medical comorbidities common

among diabetes patients, kidney disease, hypertension, and heart disease, which included congestive heart failure, myocardial infarction, coronary heart disease, or angina, was indexed by summing the number of conditions present (range: 0–3). Presence of each condition was based on self-report of physician diagnosis for kidney disease and heart disease, and self-report of physician diagnosis, antihypertension medication use, or elevated blood pressure (average systolic blood pressure ≥140 mm|Hg or diastolic blood pressure ≥90 mm|Hg) for hypertension.

Statistical Analysis

Initial comparisons of covariates by diabetes status were assessed using chi-squared tests for categorical variables and *F* tests for continuous variables. To address the first hypothesis, logistic regression was used to test the association between Type 2 diabetes status (e.g., normoglycemic, undiagnosed, clinically identified) as the independent variable and major depression syndrome as the dependent variable. Four nested regression models were fit: unadjusted, adjusted for demographic characteristics, additional adjustment for poor health behaviors, and additional adjustment for adherence to health promotion behaviors and health insurance. To address the second hypothesis, logistic regression was used to assess the association between diabetes status as the independent variable and antidepressant medication use as the dependent variable. For both the depression and antidepressant analyses, the reference group of the primary independent variable was always normoglycemia (that is, the odds ratios reflect the expected difference between diagnosed diabetes/prediabetes vs. normoglycemia, and undiagnosed diabetes/prediabetes vs. normoglycemia). Finally, analyses were conducted to assess the relationship between health behaviors, diabetes care regimens, and depression syndrome. To address whether the relationship between poor health behaviors, health promotion behaviors, and likelihood of depression varied by clinical characteristics of Type 2 diabetes, models were stratified by diabetes status. Among those with clinically identified diabetes, the influence of diabetes care regimens on likelihood of depression was also assessed.

A series of sensitivity analyses were conducted to assess the robustness of these results. Models were refit excluding participants with prediabetes (undiagnosed and clinically identified). Proportional odds models were fit using a three-level measure of depression syndrome (major, minor, and none) as the outcome. Models were also fit with additional adjustment for medical comorbidities to account for potential diabetes-related complications. Finally, models were fit using the individual health behaviors rather than counts. The results of these analyses were consistent with those presented here, indicating that the findings are robust to these variations in model specification. Survey weights were used in all analyses to account for the sampling design, and as a result all descriptive statistics report standard errors rather than standard deviations. NHANES is approved by the National Center for Health Statistics Research Ethics Review Board and all participants provided informed consent. All analyses were conducted using SAS (v9.2) and all *p* values refer to two-tailed tests.

Results

As shown by Table 1, 8.8% ($n = 419$) of the sample had clinically identified Type 2 diabetes, 3.5% ($n = 126$) had clinically identified prediabetes, 3.1% ($n = 131$) had undiagnosed Type 2 diabetes, 38.7% ($n = 1,213$) had undiagnosed prediabetes, and the remaining 45.8% ($n = 1,294$) were normoglycemic. Among those with clinically identified Type 2 diabetes, the average age of onset was 51.6 years ($SE = 0.8$ years). Consistent with previous research, persons with Type 2 diabetes were older, more likely to be Black or Hispanic, and of lower socioeconomic status. Persons with clinically identified diabetes were most likely to report being told by their physician to engage in positive health

behaviors and report attempting those behaviors. The mean fasting plasma glucose levels of undiagnosed Type 2 diabetes and clinically identified Type 2 diabetes were identical (153.5 mg/dL), but there was more variability in this measure among the undiagnosed group. As expected, the clinically identified Type 2 diabetes group reported greater frequency of health care visits in the past year (an average of 4–9 visits) than the other groups (which each reported an average of 2–3 visits).

As shown by Table 2, clinically identified Type 2 diabetes and prediabetes were significantly associated with major depression syndrome even after accounting for health behaviors (Odds ratio [OR] = 4.26; 95% confidence interval [CI]: 2.00–9.07, $p < .001$). As hypothesized, undiagnosed diabetes was not associated with depression syndrome in either crude (OR = 1.06; 95% CI [0.59–1.89], $p = .850$) or adjusted (OR = 1.35; 95% CI [0.70–2.59], $p = .375$) analyses. When the prediabetes cases were excluded from the analysis, the association between undiagnosed Type 2 diabetes and depression was still not statistically significant (adjusted OR = 2.65, 95% CI [0.75–9.33], $p = .130$), but the point estimate was more similar to that for clinically identified diabetes (adjusted OR = 3.21, 95% CI [1.42–7.22], $p = .005$) (Supplemental Table 1). Analyses with a three-level indicator of depressive symptom severity (e.g., major, minor, and no depression) using proportional odds regression were consistent with these findings, indicating a much stronger relationship between diagnosed diabetes and higher depressive symptoms (adjusted OR = 2.57, 95% CI [1.72–3.84], $p < .001$) than between undiagnosed diabetes and depressive symptoms (adjusted OR = 1.51, 95% CI [1.03–2.21], $p = .034$). As shown by Table 3, clinically identified Type 2 diabetes and prediabetes were significantly associated with antidepressant use (OR = 1.75; 95% CI [1.20–2.54], $p = .004$). As hypothesized, undiagnosed diabetes was not associated with antidepressant use in either crude (OR = 0.78; 95% CI [0.59–1.04], $p = .094$) or adjusted analyses (OR = 0.86; 95% CI [0.66–1.13], $p = .278$). These results persisted when prediabetes was excluded from the analysis (adjusted OR for undiagnosed Type 2 diabetes = 0.77, 95% CI [0.27–2.16], $p = .616$) (Supplemental Table 2). Additional adjustment for presence of medical comorbidities did not substantially influence the results (data not shown).

Finally, Tables 4 and 5 examined the relationship between health promotion and poor health behaviors and major depression syndrome, respectively. Among the normoglycemic and undiagnosed diabetes groups there was no significant association between number of health promotion behaviors and depression. In contrast, among those with clinically identified diabetes there was suggestive evidence that engaging in health promotion behaviors was associated with lower relative odds of depression as compared to abstaining from these behaviors, $\chi^2(3, n = 545) = 9.61$, $p = .022$, As shown by Table 5, there were no statistically significant associations between poor health behaviors and depression syndrome for any group. The point estimates in the undiagnosed diabetes group were all less than unity, consistent with the hypothesis that engaging in more of these behaviors was associated with lower likelihood of depression relative to abstaining, but this association was not statistically significant, $\chi^2(3, n = 2638) = 4.02$, $p = .259$. Models including each health behavior individually rather than as a count did not reveal any particular type of health promotion or poor health behavior consistently associated with depression syndrome (data not shown). Among those with clinically identified diabetes, type of care regimen was not significantly associated with likelihood of depression syndrome.

Discussion

The primary finding of this study is that clinically identified diabetes is associated with depression syndrome, whereas un-diagnosed diabetes is not. This relationship was most evident among the prediabetes cases. Similarly, clinically identified, but not undiagnosed,

diabetes is associated with antidepressant medication use. These findings are consistent with the hypothesis that formal clinical diagnosis of diabetes is predictive of depression (Nouwen et al., 2011). A recent study on hypertension reported parallel findings; that is, clinically identified, but not undiagnosed, hypertension was associated with elevated psychological distress (Hamer, Batty, Stamatakis, & Kivimaki, 2010). Together, these findings support the hypothesis that factors related to the clinical identification and self-management of diabetes, rather than a state of chronic hyperglycemia in and of itself, underlies the risk of depression (and treatment of depression with pharmacologic agents) associated with Type 2 diabetes. However, these findings do not preclude the possibility that other biological mechanisms not examined here (e.g., inflammation, sympathetic nervous system activation) contribute to the relationship between diabetes and depression (Champaneri, Wand, Malhotra, Casagrande & Golden, 2010). It is also important to note that subsequent sensitivity analyses demonstrated a positive but nonsignificant association of undiagnosed Type 2 diabetes with depression syndrome.

These findings raise two overarching clinical questions: What is inherently distressing about receiving a diagnosis of diabetes, and does this distress contribute to patient nonadherence to diabetes care regimens? Diabetes care is generally characterized by problem-focused coping, in which a person changes their behavior and learns new skills to manage the stressor, in this case diabetes (Macrodimitris & Endler, 2001). The development and prognosis of Type 2 diabetes is influenced by both uncontrollable (e.g., genetic liability) and controllable (e.g., health behaviors) factors; therefore, exclusively using a problem-focused approach may not be an effective coping strategy. The process of repeatedly “failing” to control this stressor (i.e., achieving adequate glycemic control, adhering to behavioral changes) may affect self-esteem and sense of mastery or induce a sense of learned helplessness, subsequently increasing risk of depression (Duangdao & Roesch, 2008; Bennett & Elliot, 2005). Consistent with this hypothesis, research suggests that maintaining a sense of perceived control over Type 2 diabetes is negatively correlated with developing depression (Macrodimitris & Endler, 2001). Similarly, a recent qualitative study identified multiple stressors associated with diabetes diagnosis that influence ability to engage in self-care (Penckofer, Ferrans, Velsor-Friedrich, & Savoy, 2007). For instance, patients found it difficult to adjust their lives to comply with new meal plans and medication protocols to keep their blood sugar under control, which created additional time pressure in their lives and limited their ability to travel and make plans. To deal with this psychosocial stress, patients reported sometimes engaging in behaviors such as eating high fat and sugar foods, which they referred to as “taking a diabetes break” (Penckofer, Ferrans, Velsor-Friedrich, & Savoy, 2007, p. 686)

The null findings regarding antidepressant medication use and Type 2 diabetes risk confirm those of a recent study (Kivimäki et al., 2011) and contrast with those reported by Rubin and colleagues in the Diabetes Prevention Program (DPP) (Rubin et al., 2008; Rubin et al., 2010). The DPP required a high degree of participant involvement (e.g., clinical ascertainment every 6 months, interviews every 3 months) (Diabetes Prevention Program, 1999), and it is conceivable that involvement in the trial may have influenced attitudes about health care use in general (e.g., participants may have become more willing to discuss depressive symptoms with their physician). Indeed, Rubin and colleagues have previously reported that the use of antidepressants among DPP participants marginally increased over the follow-up period, although they report that this increase paralleled increases in the general population over that same period (Rubin et al., 2005). If participants with mild or moderate depressive symptoms, which have also been associated with diabetes risk (Campayo et al., 2010), were more likely to either seek clinical care or otherwise come to the attention of a clinician to address psychological distress, this would produce a spurious association between anti-depressant use and Type 2 diabetes; specifically, that

antidepressant use predicts onset of Type 2 diabetes, even after controlling for more severe depressive symptomology due to residual confounding by indication (Kivimäki et al., 2010a). Randomized controlled trials of antidepressant use among patients with both Type 2 diabetes and depression have shown that these medications are associated with improvements in glycemic control (Lustman et al., 2000; Lustman et al., 2006) rather than worsening prognosis of diabetes. Together, these findings indicate that antidepressant medications in and of themselves do not increase the risk of Type 2 diabetes, and should remain a component of effective management of depression among patients with Type 2 diabetes.

In this study health behaviors were not consistently associated with depression syndrome. Among those with diagnosed diabetes the point estimates indicated that engaging in more health promotion behaviors was associated with lower likelihood of depression, although these estimates were not always statistically significant. Poor health behaviors were not significantly associated with depression syndrome after accounting for diabetes status; this finding is unexpected given the correlation between behavioral disorders such as nicotine and alcohol dependence and psychopathology (John, Meyer, Rumpf & Hapke, 2004; Hasin, Stinson, Ogburn & Grant, 2007). However, the robustness of the associations between less severe depressive symptomology and more normative health behaviors such as adhering to a healthy diet (Meier, Berchtold, Akre, Michaud & Suris, 2010), regular smoking (Benjet, Wagner, Borges, & Medina-Mora, 2004), alcohol use (Golding, Burnam, Wells & Benjamin, 1993; Goodman & Huang, 2002), and overall (as opposed to visceral) obesity (Sachs-Ericsson et al., 2007) are less clear and vary by characteristics such as age, gender, socioeconomic status, and race/ethnicity. There was also no evidence that type of diabetes care-regimen was associated with depression syndrome, in contrast with previous reports (Pan et al., 2010); one reason for this difference may be the lack of information on duration of diabetes care regimen.

Implications for Clinical Care

The results of this study have implications for clinical care. The Collaborative Care Model (CCM), in which nonphysician personnel enhance patient self-care with diabetes education, encouragement to engage in enjoyable physical activities, goal setting, and problem-solving skills through regular contact with patients, has demonstrated effectiveness in improving clinical outcomes for patients with depression and Type 2 diabetes (Katon et al., 2010). This model is successful, at least partially, because it is focused on open communication between patients and health care providers about health goals and it provides a social resource (in the form of a care manager) to help patients achieve those goals. Other approaches to behavior change in health care settings, such as motivational interviewing and brief counseling sessions in primary care (Spanou et al., 2010; Smith, Heckemeyer, Kratt, & Mason, 1997; West, Gore, DiLillo, Greene & Bursac, 2007), as well as incorporation of patient care decision aids (Breslin, Millan, Montori, 2008), may also be helpful for patients with diabetes.

Strengths and Limitations

These findings should be interpreted in light of study limitations. This is a cross-sectional study, and thus the direction of the relationship between depression syndrome and Type 2 diabetes cannot be inferred. Depression syndrome was determined using the PHQ-9, a self-report instrument that has moderate concordance with clinical diagnosis, but may have missed less severe cases of depression (Kroenke, Spitzer, & Williams, 2001). However, the findings from the sensitivity analysis examining major and minor depression were similar, suggesting this possible misclassification did not substantially influence the results. Health behaviors, including diabetes care regimens, were assessed by self-report and may be

subject to reporting bias. Although the fasting plasma glucose levels of those with clinically identified and undiagnosed Type 2 diabetes were similar, without additional clinical data we cannot exclude the possibility that the clinically identified cases were more severe in other ways than the undiagnosed cases (e.g., longer duration, earlier age of onset); thus, it is possible that main predictor of depression risk was diabetes severity rather than factors related to disease management or behavior change. Future studies should explicitly test this hypothesis using a prospective design. However, the findings persisted after additional adjustment for medical comorbidities, suggesting that that this limitation did not substantially influence the findings. Also, while BMI is influenced by diet and physical activity, it is not a direct measure of food intake or other health behaviors. Finally, although only cases of clinically identified diabetes among adults aged 30 and older were included in the analysis, without further information it cannot be determined that all the diabetes cases examined here were definitively Type 2 rather than Type 1 diabetes. However, the average age of diabetes cases in the sample was approximately 50 years, and more than 90% of diabetes cases in the general adult population are Type 2.

This study also has a number of strengths. The large, population-based sample reduces the risk of selection bias. Also, clinically identified and undiagnosed prediabetes and Type 2 diabetes were assessed using fasting plasma glucose samples in accordance with ADA standards. Medication use was assessed by direct inspection of pill bottles and categorized according to a national database, which mitigates the risk of underreporting of medication use and misclassification of medications. This study also accounted for key confounders, including socioeconomic status, race/ethnicity, and health behaviors, that many previous reports did not (Nouwen et al., 2011).

Additional prospective studies are needed to identify the causal mechanisms that contribute to comorbid depression and Type 2 diabetes, particularly concerning the mediating processes linking diabetes diagnosis, depression, health behaviors and treatment regimen (Markowitz, Gonzalez, Wilkinson, & Safren, 2011). Studies that investigate both severe and subsyndromal depressive symptomology among individuals at high risk for developing Type 2 diabetes may be particularly informative (Holt et al., 2009). Longitudinal studies are also needed to better understand the relationship between health behavior change and depression in the context of diabetes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Descriptive Characteristics Stratified by Diabetes Status: NHANES 2005/6 and 2007/8

		Undiagnosed		Clinically identified		
	Normoglycemic *n* (wt %)	**Prediabetes *n* (wt %)**	**Diabetes *n* (wt %)**	**Prediabetes *n* (wt %)**	**Diabetes *n* (wt %)**	**χ^2 or *F*, *p* value**
N	1294	1213	131	126	419	
Age (Mean, *SE*)	48.2 (0.5)	53.7 (0.5)	59.4 (1.5)	56.1 (1.5)	61.2 (0.9)	75.9, <0.001
Women	762 (62.3)	498 (42.2)	48 (35.1)	70 (56.2)	213 (54.8)	98.7, <0.001
Race/ethnicity						
Non-Hispanic White	704 (77.9)	705 (80.2)	67 (72.8)	71 (76.1)	176 (66.1)	55.4, <0.001
Non-Hispanic Black	284 (11.5)	191 (8.4)	23 (10.7)	23 (11.7)	126 (19.2)	
Hispanic	306 (10.6)	317 (11.4)	41 (16.5)	32 (12.2)	117 (14.7)	
High school education or less	606 (38.2)	652 (45.5)	93 (65.6)	59 (38.4)	280 (58.6)	55.8, <0.001
PIR ≥ 1.85	867 (78.7)	772 (76.4)	70 (61.6)	83 (73.3)	243 (65.5)	38.4, 0.001
Depression syndrome						
None	1217 (95.0)	1117 (93.3)	117 (87.2)	110 (89.2)	364 (87.9)	48.3, <0.001
Minor	50 (2.7)	62 (4.5)	8 (7.1)	7 (2.7)	27 (5.5)	
Major	27 (2.3)	34 (2.2)	6 (5.7)	9 (8.0)	28 (6.6)	
Any depression syndrome	77 (5.0)	96 (6.7)	14 (12.8)	16 (10.8)	55 (12.1)	28.6, <0.001
Antidepressant medications	136 (13.4)	115 (10.8)	9 (10.0)	22 (20.9)	68 (22.8)	24.9, <0.001
Health promotion behaviors (*n*, %)						
Told by doctor to lose weight	233 (17.1)	327 (28.8)	44 (43.0)	62 (46.6)	271 (64.1)	312.4, <0.001
Told by doctor to increase activity	348 (25.9)	411 (34.6)	55 (50.5)	69 (51.7)	300 (70.7)	210.9, <0.001
Told by doctor to change diet	286 (19.8)	353 (30.4)	45 (43.9)	71 (56.3)	306 (72.4)	480.2, <0.001
Sum of doctor recommendations (*M, SE*)	0.6 (0.1)	0.9 (0.1)	1.4 (0.1)	1.5 (0.1)	2.1 (0.1)	127.3, <0.001
Trying to lose weight	604 (47.8)	571 (47.2)	54 (45.7)	66 (51.4)	314 (75.6)	68.1, <0.001
Trying to increase activity	657 (50.8)	628 (51.8)	61 (50.0)	86 (67.3)	253 (61.5)	26.9, <0.001
Trying to change diet	642 (51.6)	634 (53.7)	73 (61.4)	79 (63.0)	323 (76.6)	96.6, <0.001
Sum of attempted health behaviors (*MSE*)	1.5 (0.1)	1.5 (0.1)	1.6 (0.1)	1.8 (0.1)	2.1 (0.1)	20.6, < 0.001
Diabetes medications (*n*, %)						
None	1294 (100.0)	1213 (100.0)	131 (100.0)	126 (100.0)	75 (18.5)	
Oral only					266 (60.1)	

		Undiagnosed		Clinically identified		
	Normoglycemic *n* (wt %)	**Prediabetes *n* (wt %)**	**Diabetes *n* (wt %)**	**Prediabetes *n* (wt %)**	**Diabetes *n* (wt %)**	**χ^2 or *F*, *p* value**
Insulin or insulin + oral					78 (21.4)	
Fasting glucose mg/dL (*M, SE*)	92.0 (0.2)	107.6 (0.2)	153.5 (6.5)	110.5 (2.3)	153.6 (4.3)	921.0, <0.001
No. times check sugar/day (*M, SE*)					1.3 (0.1)	
Has health insurance (*n*, %)	1032 (84.9)	966 (85.1)	104 (84.0)	107 (88.5)	362 (89.7)	7.1, 0.210
Poor health behaviors (*n*, %)						
Current smoker	276 (22.9)	241 (20.9)	28 (24.6)	20 (18.8)	64 (16.7)	35.5, <0.001
Heavy alcohol use	269 (22.1)	272 (22.9)	26 (19.8)	22 (20.2)	45 (9.6)	24.5, <0.001
Poor diet	327 (25.8)	316 (27.9)	45 (39.0)	34 (31.2)	126 (28.4)	10.8, 0.056
BMI ≥ 30	353 (25.0)	462 (39.4)	69 (57.7)	51 (38.0)	243 (60.8)	190.2, <0.001
Sum of poor health behaviors (*M, SE*)	0.96 (0.03)	1.11 (0.04)	1.41 (0.10)	1.08 (0.08)	1.18 (0.04)	10.4, <0.001

Note. Values are unweighted *n* (weighted percentage) except where noted. *p* value derived from Rao-Scott chi-squared tests for categorical variables and *F* tests for continuous variables.

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Table 2

Association Between Depression Syndrome and Clinically-Identified and Undiagnosed Diabetes

	Model 1 OR [95% CI]	Model 2 OR [95% CI]	Model 3 OR [95% CI]	Model 4 OR [95% CI]
Diabetes status (ref. normoglycemic)				
Undiagnosed prediabetes/diabetes	1.06 [0.59–1.89]	1.35 [0.73–2.49]	1.28 [0.69–2.39]	1.35 [0.70–2.59]
Clinically identified prediabetes/diabetes	3.22 [1.80–5.75]*	4.14 [2.11–8.12]*	3.91 [1.98–7.72]*	4.26 [2.00–9.07]*
Age		0.97 [0.95–0.99]*	0.97 [0.95–0.99]*	0.97 [0.95–1.00]
Gender (ref. men)		2.03 [1.20–3.43]*	2.09 [1.24–3.50]*	2.25 [1.34–3.78]*
Race/ethnicity (ref. non-Hispanic White)				
Non-Hispanic Black		0.79 [0.42–1.49]	0.79 [0.42–1.49]	0.74 [0.38–1.42]
Hispanic		0.47 [0.25–0.90]*	0.51 [0.26–1.01]	0.41 [0.19–0.90]*
Low education		1.48 [0.71–3.09]	1.41 [0.67–2.97]	1.36 [0.67–2.77]
High PIR		0.35 [0.17–0.71]*	0.36 [0.18–0.71]*	0.45 [0.22–0.94]*
Count of poor health behaviors (ref. none)				
One			1.31 [0.68–2.52]	1.28 [0.68–2.42]
Two			1.60 [0.79–3.25]	1.43 [0.70–2.90]
Three or four			1.67 [0.72–3.88]	1.39 [0.58–3.34]
Count of trying to engage in health promotion behaviors (ref. none)				
One				1.08 [0.51–2.29]
Two				2.28 [1.13–4.58]*
Three				0.72 [0.33–1.61]
Health insurance (ref. none)				0.42 [0.22–0.78]*
Total *n*	3183	3183	3183	3183
−2 Log-likelihood	39298549	36858791	36734841	35424898
AIC	39298555	36858809	36734865	35424930

Note. Estimates are adjusted for all covariates in the model.

* $p < .05$.

Table 3

Association Between Antidepressant Medication Use and Clinically-Identified and Undiagnosed Diabetes

	Model 1 OR [95% CI]	Model 2 OR [95% CI]	Model 3 OR [95% CI]	Model 4 OR [95% CI]
Diabetes status (ref. normoglycemic)				
Undiagnosed prediabetes/diabetes	0.78 [0.59–1.04]	0.91 [0.69–1.20]	0.88 [0.68–1.15]	0.86 [0.66–1.13]
Clinically identified prediabetes/diabetes	1.86 [1.27–2.72]*	2.13 [1.47–3.10]*	2.00 [1.38–2.89]*	1.75 [1.20–2.54]*
Age		1.00 [1.00–1.01]	1.00 [1.00–1.01]	1.00 [0.99–1.01]
Gender (ref. men)		3.11 [2.44–3.96]*	3.17 [2.46–4.07]*	3.03 [2.33–3.92]*
Race/ethnicity (ref. non-Hispanic White)				
Non-Hispanic Black		0.24 [0.15–0.37]*	0.23 [0.15–0.36]*	0.23 [0.14–0.37]*
Hispanic		0.25 [0.17–0.36]*	0.26 [0.18–0.37]*	0.31 [0.22–0.44]*
Low education		1.22 [0.92–1.64]	1.20 [0.89–1.60]	1.24 [0.93–1.66]
High PIR		0.71 [0.59–0.86]*	0.73 [0.60–0.90]*	0.66 [0.51–0.85]*
Poor health behaviors (ref. none)				
One			1.47 [0.96–2.25]	1.46 [0.94–2.26]
Two			1.33 [0.89–2.01]	1.37 [0.90–2.10]
Three or four			1.28 [0.73–2.27]	1.21 [0.65–2.24]
Depression syndrome (ref. minor/not depressed)				6.20 [3.74–10.29]*
Health insurance (ref. none)				3.86 [2.31–6.43]*
Total *n*	3183	3183	3183	3183
−2 Log-likelihood	118379871	109526167	109123358	105293701
AIC	118379877	109526185	109123382	105293729

Note. Estimates are adjusted for all covariates in the model.

*$p < .05$.

Table 4

Association Between Depression Syndrome and Health Promotion Behaviors Stratified by Diabetes Status

	Normoglycemic OR [95% CI]	Clinically identified prediabetes/ diabetes OR [95% CI]	Undiagnosed prediabetes/ diabetes OR [95% CI]
Age	0.99 [0.96–1.03]	0.95 [0.92–0.99]*	0.95 [0.93–0.97]*
Gender (ref. men)	3.29 [0.97–11.10]	2.08 [0.92–4.69]	1.55 [0.60–3.97]
Race/ethnicity (ref. White)			
Black	0.77 [0.28–2.12]	0.69 [0.28–1.69]	1.06 [0.32–3.52]
Hispanic	0.49 [0.17–1.41]	0.57 [0.20–1.60]	0.39 [0.13–1.20]
Low education	1.32 [0.45–3.87]	2.33 [0.74–7.28]	1.37 [0.58–3.27]
High PIR	0.44 [0.14–1.41]	0.25 [0.09–0.65]*	0.35 [0.14–0.89]*
Count of health promotion behaviors (ref. none)			
One	0.39 [0.07–2.07]	0.21 [0.06–0.82]*	2.36 [0.86–6.46]
Two	3.38 [0.96–11.88]	0.73 [0.22–2.45]*	2.30 [0.67–7.93]
Three	1.38 [0.39–4.84]	0.20 [0.05–0.76]*	0.40 [0.16–1.05]
Diabetes management behaviors			
Using oral medications	—	0.93 [0.32–2.73]	—
Using insulin	—	1.07 [0.23–4.88]	—
Total *n*	1294	545	1344
−2 Log-likelihood	14100852	7778244	12809775
AIC	14100872	7778268	12809795

Note. Estimates are adjusted for all covariates in the model.

*$p < .05$.

Table 5

Association Between Depression Syndrome and Poor Health Behaviors Stratified by Diabetes Status

	Normoglycemic OR [95% CI]	Clinically identified prediabetes/ diabetes OR [95% CI]	Undiagnosed prediabetes/ diabetes OR [95% CI]
Age	1.00 [0.97–1.03]	0.97 [0.93–1.00]	0.96 [0.93–0.98] *
Gender (ref. men)	3.47 [0.91–13.21]	2.09 [0.93–4.71]	1.58 [0.66–3.78]
Race/ethnicity (ref. White)			
Black	0.75 [0.28–2.02]	0.58 [0.22–1.51]	1.28 [0.39–4.14]
Hispanic	0.51 [0.18–1.41]	0.59 [0.21–1.70]	0.44 [0.15–1.32]
Low education	1.20 [0.44–3.23]	2.37 [0.78–7.19]	1.24 [0.48–3.19]
High PIR	0.48 [0.14–1.61]	0.26 [0.10–0.69] *	0.31 [0.14–0.67] *
Count of poor health behaviors (ref. none)			
One	2.61 [0.82–8.31]	2.96 [0.97–9.00]	0.43 [0.12–1.52]
Two	2.21 [0.45–10.95]	4.78 [1.00–22.93]	0.84 [0.25–2.91]
Three or Four	1.08 [0.15–7.72]	6.31 [1.16–34.40] *	0.98 [0.28–3.47]
Diabetes management behaviors			
Using oral medications	—	0.75 [0.24–2.33]	—
Using insulin	—	1.08 [0.27–4.38]	—
n	1294	545	1344
−2 Log-likelihood	14350207	8035457	13264913
AIC	14350227	8035481	13264933

Note. Estimates are adjusted for all covariates in the model.

* $p < .05$.