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- Consider whether sleep problems contribute toward poor control of type 2 diabetes in patients with provisional threshold or subthreshold depression

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Association Between Specific Depression Symptoms and Glycemic Control Among Patients With Comorbid Type 2 Diabetes and Provisional Depression

Stephanie J. Czech, PhD[a,*]; Susan M. Orsillo, PhD[b]; Paul A. Pirraglia, MD, MPH[c]; Thomas M. English, PhD[d]; and Alexa J. Connell, PhD[e]

ABSTRACT

Objective: To determine whether specific depression symptoms are associated with glycemic control independent of potential demographic and clinical covariates among primary care patients with comorbid type 2 diabetes and provisional threshold or subthreshold depression.

Method: We examined a convenience sample of patients diagnosed with type 2 diabetes and provisional threshold or subthreshold depression (N = 82) at 2 family health centers. Cases were identified using a population-based registry of patients diagnosed with type 2 diabetes (*ICD-9* codes 250.00 for controlled type 2 diabetes and 250.02 for uncontrolled type 2 diabetes). Data from patients with a primary care provider appointment from the beginning of April 2011 through the end of June 2012 and with at least one 9-item Patient Health Questionnaire (PHQ-9) depression screener and a glycated hemoglobin A_{1c} (HbA_{1c}) laboratory test between 2 weeks before and 10 weeks after PHQ-9 screening were eligible for inclusion. We defined provisional threshold or subthreshold depression using PHQ-9 scoring criteria, which were designed to yield provisional diagnostic information about major depressive disorder based on *DSM-5* diagnostic criteria.

Results: Patients reporting higher severity of sleep problems on the PHQ-9 had significantly higher HbA_{1c} levels (mean = 8.48, SD = 2.17) compared to patients reporting lower severity or absence of this symptom (mean = 7.19, SD = 1.34, $t_{48.88} = -3.13$, $P = .003$). Problems with sleep contributed unique variance on glycemic control ($\beta = 0.27$, $P = .02$) when controlling for potential clinical and demographic covariates, with those reporting more sleep difficulties having higher HbA_{1c} levels.

Conclusions: For patients with type 2 diabetes and provisional threshold or subthreshold depression, it may be prudent to aggressively address sleep problems as a potential mechanism toward improving diabetes control.

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[a]Department of Psychiatry, Rhode Island Hospital, Providence
[b]Department of Psychology, Suffolk University, Boston, Massachusetts
[c]Primary Care, Providence VA Medical Center and Alpert Medical School, Brown University, Providence, Rhode Island
[d]Department of Quantitative Health Sciences, University of Massachusetts Medical School, Worcester
[e]Department of Family Medicine and Community Health, University of Massachusetts Medical School, Worcester
**Corresponding author:* Stephanie J. Czech, PhD, Department of Psychiatry, Rhode Island Hospital, 146 West River St, Ste 11B, Providence, RI 02904 (stephanie_czech@brown.edu).

Clinical Points

- For patients with type 2 diabetes and depression, sleep problems may negatively impact diabetes control.
- There may be clinical benefit of deliberate assessment and intervention for sleep problems early in the treatment process for patients with type 2 diabetes and depression.

The prevalence rates of both type 2 diabetes and depression, disorders associated with debilitating functional impairment, have notably increased in recent decades.[1,2] Moreover, both disorders commonly co-occur, as it is estimated that clinically relevant depression is nearly 2 times more likely in patients with type 2 diabetes than in those without.[3] The comorbidity of the 2 illnesses profoundly impacts disease self-management, daily functioning, and quality of life[4,5] and has been linked with diabetic complications, sexual dysfunction, and increased mortality,[6] in addition to increased medical costs across medical and mental health settings.[4]

Comorbid depression may negatively impact the course and severity of diabetes via its influence on glycemic control.[6–11] While mechanisms have not yet been determined,[12,13] psychological/behavioral, sociocultural, and biological pathways have been proposed. For example, depression has been posited to interfere with good glycemic control via behavioral mechanisms, including inability to adhere to diabetes self-management recommendations.[4,8] Biologically, research suggests that depression affects glycemic control via metabolically relevant pathways, including alterations in neuroendocrine or immune functioning and glucose metabolism.[9,11] Likewise, poor glycemic control has been proposed to adversely affect mood, reinforcing this relationship.[8] For example, high blood glucose levels in patients with diabetes have been found to be associated with negative mood states such as anger and sadness,[14] increased worry about glucose status,[15] and poor glycemic control with impaired sense of well-being and perceived health.[16] On the biological side, there is some evidence to suggest that uncontrolled glycemia may impact mood through the effects of blood glucose fluctuations on cortical function in the brain.[8,14]

Several studies have examined the relationship between depression and glycemic control, and both cross-sectional and longitudinal studies have yielded inconsistent findings.[10,13] While methodological issues may account for mixed findings, studies that include large populations in primary care and allow for the inclusion of confounding variables and potentially important moderators,[4,17–19] have continued to exhibit inconsistent results.

To date, research examining the relationship between depression and glycemic control has treated depression as a homogenous construct. Yet, there is considerable evidence that depression is heterogeneous.[20] For example, the *DSM-5*[21] criteria for depression allow for differences in not only the severity of symptoms (ie, mild, moderate, severe), but also the nature of symptoms (eg, negative emotions, anhedonia). Importantly, while heterogeneity of symptom patterns is not specific to depression, depression is unique in that somatic symptom criteria related to weight/appetite, sleep, and motor activity are bidirectional. In other words, individuals can meet these criteria for a depressive episode with either an increase or a decrease in these symptoms. Thus, examining relationships between depression symptoms and glycemic control may provide insight into specific aspects of depression most deleterious toward glycemic control in patients with type 2 diabetes.

We have identified only 1 previous study examining the relationship between the 9 individual depression symptoms consistent with the *DSM-5* diagnostic criteria and glycemic control. Bot and colleagues[22] examined cross-sectional and longitudinal associations between 9-item Patient Health Questionnaire (PHQ-9) depressive symptoms and glycated hemoglobin A_{1c} (HbA_{1c}) in 343 and 291 patients with type 2 diabetes, respectively, finding a significant association with HbA_{1c} for sleep problems, appetite problems, and suicidal ideation when adjusting for relevant controls at baseline and no association at 1-year follow-up. In that study,[22] the sample was not limited to patients with probable clinically relevant depression, but instead included all diabetic patients who completed a PHQ-9 screener. In contrast, we utilized the PHQ-9 diagnostic algorithm, based on *DSM-5* diagnostic criteria, to limit the sample to patients with provisional threshold and subthreshold depression. We believe this methodology provided an opportunity to detect significant predictors of HbA_{1c} in a more clinically relevant sample of type 2 diabetes patients endorsing depression symptoms.

We aimed to determine whether specific depression symptoms are associated with glycemic control in primary care patients with comorbid type 2 diabetes and provisional threshold or subthreshold depression. We propose that identifying the most relevant depression symptoms in this population will serve to guide and focus interventions toward improving both depression and type 2 diabetes.

METHOD

Participants

Participants were a convenience sample of patients at 2 family health centers of the University of Massachusetts Memorial Medical Center in Worcester, Massachusetts, an industrial city, and Barre, Massachusetts, a rural town, both located in Central Massachusetts. The health centers serve as academic group practice model residency sites, emphasizing patient-centered full-spectrum family medicine care and family medicine residency education. The centers serve an ethnically and socioeconomically diverse population, consistent with the diversity of Worcester and the surrounding communities.

We identified cases using an established hospital-based registry of patients with a diagnosis of type 2 diabetes (*ICD-9* codes 250.00 for controlled type 2 diabetes and 250.02 for

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uncontrolled type 2 diabetes). We included data from all patients who attended an appointment with their primary care provider from the beginning of April 2011 through the end of June 2012 and had a record of at least 1 PHQ-9 screen and an HbA_{1c} laboratory test that occurred between 2 weeks before and 10 weeks after PHQ-9 screening. This time gap was intended to maximize the opportunity for temporal overlap between these measurements, given that HbA_{1c} is a clinically useful index of mean blood glucose during the preceding 60 to 120 days[23] and the PHQ-9 measures depression symptoms within the previous 2 weeks.[24] Informed consent was waived by the University of Massachusetts Medical School Institutional Review Board according to the code of federal regulations that govern the protection of human subjects (45 CFR 46. 116 [d]) given that research was minimal risk, the waiver did not adversely affect the subject, and the research could not have practically been carried out without the waiver.

Procedure

As part of routine care, all patients with a diagnosis of type 2 diabetes were advised to have HbA_{1c} tests and depression screening every 6 months. Chart alerts signaled the delivery of depression screeners to all patients with a diabetes diagnosis at the time of appointment check in. Medical assistants were trained to assist patients in screener completion if necessary, and screeners were collected by primary care physicians during patient visits. Screener data were subsequently scanned into the electronic health record, and individual item responses were entered into a database that supported patient care for patients with type 2 diabetes and other chronic health problems, including major depressive disorder.

Demographic, health behavior, and clinical data were entered into the electronic health record through the registration process via the registration department within the University of Massachusetts Memorial Medical Center system (ie, race, ethnicity, age, gender, employment status, marital status), by primary care physicians during the medical visit (ie, diabetes complications, medical comorbidity, medication regimen, body mass index [BMI], smoking status), and by the office staff at time of check in for the medical visit (ie, health insurance information).

Measures

Demographic characteristics. Information on demographic characteristics was collected by patient self-report and included race (ie, white, African American/black, other, unknown), ethnicity (ie, Hispanic, other, unknown), age (in years), gender (ie, male, female), socioeconomic status (as defined by health insurance information, ie, "low insurance status" = Mass Health, Commonwealth Care, Health Safety Net; "moderate to high insurance status" = private health insurance, Commonwealth Choice, Medicare; unknown = unknown), employment status (ie, employed, student, unemployed, retired, unknown), and marital status (ie, married, divorced, single, widowed, other, unknown).

Provisional depression. We assessed depressive symptoms with the PHQ-9, a 9-item self-report depression module taken from the full PHQ,[25] which covers 8 diagnostic categories and was developed to correspond to specific *DSM-IV-R*[26] diagnoses. The PHQ-9 items are consistent with the recently revised *DSM-5*, and the American Psychiatric Association has recommended continued use of the measure to enhance clinical decision-making.[21] The PHQ-9 is a dual-purpose instrument that yields provisional diagnostic information about threshold and subthreshold MDD diagnoses and can be used as a severity measure to grade depressive symptom severity and monitor patient status over time.[24,25] The PHQ-9 has been found to have strong psychometric properties,[24,25,27] and health care guidelines strongly support its use with patients diagnosed with chronic diseases including diabetes.[28,29] The Cronbach α coefficient on the PHQ-9 for the current study was found to be 0.75.

Items on the PHQ-9 reflect the 9 diagnostic criteria of depression and are scored from 0 ("not at all") to 3 ("nearly every day"). Scores range from 0 to 27, with higher scores reflecting greater depression severity. Cutpoints of 5, 10, 15, and 20 represent thresholds for mild, moderate, moderately severe, and severe depression symptoms, respectively. When used as a diagnostic measure, 8 of the 9 symptoms count toward a provisional diagnosis of MDD if they are endorsed as present for at least "more than half the days" (score ≥ 2) in the past 2 weeks. The item assessing suicidal ideation counts toward a provisional diagnosis if it is present at all during the previous 2 weeks (score ≥ 1). A provisional diagnosis of MDD requires the presence of ≥ 5 symptoms (which must include either depressed mood or anhedonia). A provisional diagnosis of subthreshold depression requires ≥ 2 symptoms (including depressed mood or anhedonia) to be endorsed.

Health behavior risk factors. We collected BMI (kg/m^2) measurements and patient self-report of status as a smoker from the medical chart.

Glycemic control. We obtained HbA_{1c} levels and dates of each measurement from laboratory test results as reported in the medical chart.

Medical comorbidity. We measured medical comorbidity with the Charlson Comorbidity Index,[30] a weighted index reflecting the extent of comorbidity considering 19 medical conditions, which has been used in previous research on the association between depression and glycemic control in primary care.[19] This empirically derived measure was developed from a cohort of 604 patients admitted during a 1-month period to assess the risk of death from comorbid disease. Adjusted relative risks were used to derive weights (0 to 6) for 19 medical conditions. The total score for each patient is equal to the total sum of the weights of each condition listed as diagnoses (and identified by *ICD-9* codes) in the electronic health record for that patient (ie, higher total score suggests a higher risk of death from comorbid disease). We employed an updated *ICD-9-Clinical Modification* coding algorithm consistent with Quan et al[31] and excluded 2 conditions—diabetes and diabetes with end organ damage—from this measure (given that study inclusion criteria already required

Table 1. Demographic Characteristics of 82 Patients Diagnosed With Type 2 Diabetes and PHQ-9–Defined Provisional Depression or Subthreshold Depression

Variable	Patients
Age, mean ± SD (median), y	56.27 ± 14.21 (55.39)
Gender, female, n (%)	44 (53.7)
Race, n (%)	
White	55 (67.1)
Black	10 (12.2)
Other	12 (14.6)
Unknown	5 (6.1)
Ethnicity, n (%)	
Hispanic	12 (14.6)
Other	56 (68.3)
Unknown	14 (17.1)
Marital status, n (%)	
Married	33 (40.2)
Divorced	14 (17.1)
Single	25 (30.5)
Widowed	7 (8.5)
Other	2 (2.4)
Unknown	1 (1.2)
Employment, n (%)	
Employed	18 (22.0)
Retired	23 (28.0)
Unemployed	41 (50.0)
Socioeconomic status, n (%)	
Low insurance status	28 (34.1)
Moderate to high	54 (65.9)

Abbreviation: PHQ-9 = 9-item Patient Health Questionnaire.

a diagnosis of diabetes, and a separate measure, as described below, captured the presence of diabetes complications such as end organ damage). Thus, for the present study, the item count was reduced to 17 items, with a possible total score of 32.

Severity of diabetes complications. We modeled diabetes complications with the Diabetes Complications Severity Index (DCSI),[32] a scale used in previous research,[18] which integrates data from diagnostic codes (*ICD-9*), pharmacy, and laboratory results to model the severity of diabetes complications from 7 categories (ie, retinopathy, nephropathy, neuropathy, cerebrovascular disease, cardiovascular disease, peripheral vascular disease, and metabolic syndrome) at any 1 point in time. Each category of complication is associated with a list of *ICD-9* diagnoses, and each diagnosis was given a severity rating score based on previous models. Complication scores range from 0 to 80 and depend on the presence of specific *ICD-9* codes in the electronic health record for a given patient.

Complexity of medication regimen. We extracted data concerning medications from the electronic health record to measure complexity of the medication regimen for diabetes (ie, diet only, oral hypoglycemic only, insulin only, or oral hypoglycemic plus insulin). The electronic health record medication list was reconciled by the primary care physician at every patient visit. If a patient was not prescribed any medications, then the classification "diet and exercise only" was assigned for this variable.

Analyses

We conducted 9 independent sample *t* tests to explore potential differences in HbA_{1c} between patients indicating higher severity (ie, score of 2 or 3) versus absent or lower severity (ie, score of 0 or 1, respectively) of each of the original 9 PHQ-9 items. For each item that yielded a significant group difference, we employed Pearson product-moment correlations to examine the magnitude of the relationship between PHQ-9 items and HbA_{1c}. Pearson product-moment correlations were also conducted between demographic and clinical variables and HbA_{1c}. We conducted multiple regression to examine unique contributions of specific PHQ-9 items to HbA_{1c}. In all analyses, model assumptions were reasonably upheld for all statistical tests, thus no data transformations were necessary.

RESULTS

Data from 82 adults (44 females) meeting the inclusion criteria for the current study were analyzed. Demographic and clinical characteristics for the total sample are described in Tables 1 and 2.

We conducted 9 independent-sample *t* tests using the Bonferroni correction comparing HbA_{1c} levels between groups of patients reporting high severity (ie, score of 2 or 3) versus low severity (ie, score of 0 or 1) of symptoms on each of the 9 PHQ-9 items. For PHQ-9 item 3 related to sleep problems (ie, "Trouble falling or staying asleep, or sleeping too much?"), patients in the high symptom severity group were found to have significantly higher HbA_{1c} levels (mean = 8.48, SD = 2.17) compared to those in the low symptom severity group (mean = 7.19, SD = 1.34, $t_{48.88} = -3.13$, $P = .003$). The magnitude of the difference was large ($\eta^2 = 0.11$). No other significant differences emerged.

To further explore the potential relationship between sleep problems and HbA_{1c}, we conducted Pearson product moment correlation. A small but significant association was found between sleep problems and HbA_{1c} ($r = 0.23$, n = 82, $P = .04$).

To examine the potentially unique contribution of sleep problems to HbA_{1c}, we conducted multiple regression analysis (Table 3). Demographic and clinical variables were employed that have been identified in the literature as moderators of the relationship between depression and HbA_{1c} as covariates (ie, comorbid cardiovascular disease, age, and gender). While other covariates were considered, we did not include them in the model given that they were not significantly correlated with HbA_{1c}. The overall equation was significant ($R^2 = 0.14$, $F_{4,77} = 3.05$, $P = .02$), and sleep problems contributed unique variance on HbA_{1c} ($\beta = 0.27$, $P = .02$), with those reporting more sleep difficulties having higher HbA_{1c} levels.

CONCLUSION

We sought to determine whether specific symptoms of depression are associated with glycemic control in a population of primary care patients with comorbid type 2 diabetes and provisional threshold or subthreshold depression. We found glycemic control varied with severity

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Table 2. Clinical Characteristics of 82 Patients Diagnosed With Type 2 Diabetes and PHQ-9–Defined Provisional Depression or Subthreshold Depression[a]

Variable	Patients
Body mass index[a]	
Mean ± SD (median), kg/m^2	34.31 ± 8.91 (31.82)
Normal, n (%)	8 (9.8)
Overweight, n (%)	19 (23.2)
Obese, n (%)	55 (67.1)
Medication regimen, n (%)	
Diet only	16 (19.5)
Oral only	39 (47.6)
Insulin only	7 (8.5)
Insulin plus oral	20 (24.4)
Tobacco use (smoker), n (%)	16 (19.5)
Cardiovascular disease, n (%)	16 (19.5)
Charlson Comorbidity Index score, mean ± SD (median)	0.89 ± 1.49 (0.00)
Diabetes Complications Severity Index score, mean ± SD (median)	1.15 ± 1.67 (0.50)
HbA_{1c}, mean ± SD (median), %	8.18 ± 2.07 (7.85)
Glycemic control, n (%)[b]	
HbA_{1c} < 7.0%	31 (37.8)
HbA_{1c} ≥ 7.0%	51 (62.2)
PHQ-9 total score, mean ± SD (median)	15.32 ± 5.23 (15.00)
Depression status, n (%)[c]	
Depression	72 (87.8)
Subthreshold depression	10 (12.2)

[a]Normal BMI = < 25.0 kg/m^2, obese = ≥ 30.0 kg/m^2, overweight = 25–29.9 kg/m^2.
[b]Glycemic control categories are based on American Diabetes Association criteria for controlled diabetes (HbA_{1c} < 7.0%) and uncontrolled diabetes (HbA_{1c} ≥ 7.0%).[33]
[c]Depression diagnostic categories are based on PHQ-9 provisional diagnostic scoring criteria.
Abbreviations: HbA_{1c} = hemoglobin A_{1c}, PHQ-9 = 9-item Patient Health Questionnaire.

Table 3. Multiple Regression Analysis Examining Unique Contribution of Sleep Problems to HbA_{1c}

Predictors	β	*t*	*P*
Comorbid cardiovascular disease	−0.21	−1.68	.10
Age	−0.08	−0.72	.48
Gender	0.12	1.02	.31
Sleep problems[a]	0.27	2.44	.02

[a]Sleep problems = score for item 3 (ie, "Trouble falling or staying sleep, or sleeping too much" on the 9-item Patient Health Questionnaire self-report depression measure).
Abbreviation: HbA_{1c} = hemoglobin A_{1c}.

of PHQ-9 symptom only for the item related to sleep problems. Further exploration of this relationship revealed a unique contribution of sleep problems to HbA_{1c}, with patients reporting more sleep difficulties having significantly higher HbA_{1C} levels.

Strengths of our study include limiting the sample to patients with provisional threshold and subthreshold depression, allowing for a clinically relevant population. Additional strengths include data collection in the primary care setting wherein most patients with type 2 diabetes present, considering potentially confounding clinical and demographic variables for analyses, and specifying the timing of depression screening and HbA_{1c} measurement in order to maximize the opportunity for temporal overlap between these measurements. Limitations include the cross-sectional nature of the study design, small sample size, inability to stratify our data based on meeting criteria for provisional threshold versus subthreshold depression due to inadequate power, utilization of a convenience sample of primarily white patients with moderate to high socioeconomic status, and lack of clinical interview, which would more accurately diagnose depression and its subtypes. Further, the false-positive rate for PHQ-9 measurement of threshold depression (sensitivity = 0.80, 95% CI, 0.71–0.87)[27] may have obscured the presence of depression in our sample.

Research to date on the relationship between depression and glycemic control in patients with type 2 diabetes has been inconsistent.[10,13] A possible explanation is that studies have not accounted for the heterogeneity of depression symptoms. In terms of individual symptoms of depression, our study suggests that, specifically, sleep problems are related to elevated HbA_{1c} levels in type 2 diabetic patients with provisional subthreshold or threshold depression. We were unable to assess the bidirectional somatic symptoms of sleep, appetite, and motor activity in this study, and, thus, the question of whether specific sleep problems (ie, insomnia vs hypersomnia) may differentially relate to glycemic control in this population suggests a further line of investigation. Of note, 2 relatively common *DSM-5* depression subtypes, atypical depression and melancholic depression, have been well validated in the literature and may be particularly relevant in patients with type 2 diabetes given their opposite patterns of somatic symptoms pertaining to sleep and appetite.[34,35] Thus, future research might investigate the relationship between depression subtypes and HbA_{1c} in patients with type 2 diabetes.

Our findings have clinical implications concerning depressed patients with type 2 diabetes presenting in the primary care clinic. Sleep problems are commonly reported in patients with diabetes, and studies have associated diabetes with poor sleep quality, sleep duration (due to either insomnia or sleep-disordered breathing), and increased daytime sleepiness.[36–39] Further, there is accumulating evidence supporting associations between poor sleep quality and duration (including due to obstructive sleep apnea) and poor glycemic control in patients with type 2 diabetes when controlling for important potential confounds.[40,41] Research examining the impact of improved sleep on glycemic control is sparse. While studies addressing the impact of continuous positive airway pressure therapy on glycemic control in diabetic patients with sleep apnea are accumulating, findings are mixed and long-term treatment studies are needed.[42]

With respect to depression, poor sleep is a well-known risk factor and maintaining factor.[43] Ample empirical evidence supports the use of behavioral therapies and medications in treating insomnia in the context of depression.[44] Randomized clinical trials[45–47] further indicate that treating both insomnia and depression simultaneously leads to higher depression remission rates than treating depression alone.

Clinically, a question of interest is whether and how improving sleep may impact both depression severity and

diabetes control in patients with this comorbidity. To answer this question, it will be important to understand mechanisms by which sleep disturbances potentially contribute to poor glycemic control in depressed patients. It appears that from a pathophysiologic standpoint, uncontrolled diabetes, depression, and insufficient sleep duration and quality may have commonalities (ie, concerning activation of the sympathetic nervous system, hypothalamic-pituitary-adrenal axis, and inflammatory processes).[9,11,48] Thus, it is possible that these processes exacerbate and influence the effect of these comorbidities on each other. Importantly, sleep disorders (including short sleep duration and obstructive sleep apnea) and depression are both associated with obesity,[49,50] which is a well-studied risk factor for impaired metabolic control with potential behavioral (eg, daytime fatigue and physical inactivity, increased appetite and eating behavior), psychological (eg, increase in emotional eating), and pathophysiologic mechanisms.[48] Finally, behaviorally there are several ways in which sleep problems may negatively impact one's ability to obtain optimal targets for glycemic control above and beyond the potentially disrupting effects of depressed mood and anhedonia. For example, severe fatigue and excess daytime sleep would most likely negatively affect one's ability to effectively comply with diabetes self-management recommendations.

There has been recent attention toward tailoring interventions for depression in patients with diabetes. While cognitive-behavioral therapy, antidepressant medication, and collaborative care interventions have been found to be effective for improving depression in this population, findings are mixed in terms of their effectiveness toward improving glycemic control.[51] Our findings suggest that for patients with type 2 diabetes and provisional depression or subthreshold depression, there may be clinical benefit toward deliberate assessment and intervention for sleep problems early in the treatment process. For patients in this population, sleep may be a mechanism toward improving diabetes control.

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POSTTEST

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1. **When using the 9-item Patient Health Questionnaire (PHQ-9) to provide provisional diagnostic information, a diagnosis of provisional major depressive disorder (MDD) requires all of the following *except*:**
   a. The presence of ≥ 5 symptoms
   b. The presence of ≥ 1 somatic symptom
   c. The presence of either depressed mood or anhedonia
   d. That symptoms be present for "more than half the days"

2. **Mrs A has achieved good control of her diabetes during medical visits over the past 5 years. During her physical today, her HbA_{1c} is 8.6%. She reports having lost her son 6 months ago, and her PHQ-9 score is positive for a diagnosis of provisional MDD. Mrs A reports that, during the weeks after her son's death, she would lie in bed thinking about how she could have prevented it. Her PHQ-9 indicates sleep problems "nearly every day," and she states that, although her worries and guilt are improving, she continues to lie awake for hours each night. Based on the information in this article, it would make most sense to refer Mrs A to a behavioral health provider who:**
   a. Specializes in bereavement counseling
   b. Specializes in behavioral treatment for insomnia
   c. Can prescribe medication
   d. Treats depression and grief and provides behavioral treatment for insomnia and diabetes management