Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

HHS Public Access

Author manuscript

J Diabetes Complications. Author manuscript; available in PMC 2017 March 01.

Published in final edited form as:

J Diabetes Complications. 2016 March ; 30(2): 312–317. doi:10.1016/j.jdiacomp.2015.11.010.

Depressive Symptoms, Serious Psychological Distress, Diabetes Distress and Cardiovascular Risk Factor Control in Patients with Type 2 Diabetes

Rhonda J. Winchester, MD[a], **Joni S. Williams, MD, MPH**[a,b], **Tamara E. Wolfman, MD**[b], and **Leonard E. Egede, MD, MS**[a,b,c]

Rhonda J. Winchester: runwithjane@gmail.com; Joni S. Williams: stromjl@musc.edu; Tamara E. Wolfman: wolfmant@musc.edu

[a]Center for Health Disparities Research, Medical University of South Carolina, 135 Rutledge Avenue, P.O. Box 250593, Charleston, South Carolina USA 29425

[b]Department of Medicine, Division of General Internal Medicine and Geriatrics, Medical University of South Carolina, 96 Jonathan Lucas Street, Suite 803, MSC 623, Charleston, South Carolina USA 29425

[c]Health Equity and Rural Outreach Innovation Center, Ralph H. Johnson VA Medical Center, 109 Beet Street, Charleston, South Carolina USA 29401

Abstract

OBJECTIVE—This study examined the association between cardiovascular disease (CVD) risk factor control and elevated depressive symptoms (EDS), serious psychological distress (SPD), and diabetes distress (DD) in patients with type 2 diabetes (T2DM).

METHODS—Cross-sectional study of adults seen at an academic medical center and Veterans Affairs Medical Center in the southeastern US. Linear regression models were computed using CVD risk factors as clinically meaningful outcomes (glycosylated hemoglobin A1c (HbA1c); systolic (SBP) and diastolic (DBP) blood pressure; and low-density lipoprotein cholesterol (LDL-C)); EDS, SPD, and DD were primary independent variables. Covariates included sociodemographics and comorbidities.

Corresponding Author: Leonard E Egede, MD, MS, Center for Health Disparities Research, Health Equity and Rural Outreach Innovation Center, Medical University of South Carolina, 135 Rutledge Avenue, Room 280G, P.O. Box 250593, Charleston, SC 29425-0593, Phone: 843-792-2969, Fax: 843-876-1201, egedel@musc.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Conflict of Interest: The authors report no potential conflicts of interest relevant to this article.

Author Contributions: LEE obtained funding for the study. RJW, JSW and LEE designed the study, acquired, analyzed and interpreted the data. LEE, RJW, JSW and TW developed the analysis, contributed to interpretation and critically revised the manuscript for important intellectual content. All authors approved the final manuscript.

Guarantors: LEE and JSW are the guarantors of the study and take full responsibility for the work as a whole, including the study design, access to data, and the decision to submit and publish the manuscript.

Disclaimer: This article represents the views of the authors and not those of NIH, MUSC, VHA or HSR&D.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

RESULTS—The sample consisted of 361 adults. Correlation analyses showed significant relationships between DD and HbA1c, DBP, and LDL-C. Adjusted linear regression models showed DD to be significantly associated with HbA1c and LDL-C, and SPD to be significantly associated only with LDL-C. In the fully adjusted model, DD remained significantly associated with HbA1c (β=4.349; 95% CI (−0.649, 2.222)).

CONCLUSIONS—In this sample of adults with T2DM, DD and SPD were significantly associated with CVD risk factors; however, after controlling for covariates, only DD was shown to be significantly associated with poor glycemic control.

PRACTICE IMPLICATIONS—Strategies are warranted to examine the relationship between DD and CVD risk factor control in patients with T2DM.

Keywords

diabetes; diabetes distress; depressive symptoms; serious psychological distress; cardiovascular disease risk factor

1.INTRODUCTION

Many patients with type 2 diabetes (T2DM) experience comorbid psychological states, such as elevated depressive symptoms (EDS), serious psychological distress (SPD), and diabetes distress (DD). Depressive symptoms are two times more common in individuals with diabetes compared to those without diabetes [1]. Serious Psychological Distress occurs in 7.6% of U.S. adults with diabetes, more than twice the prevalence among the population without diabetes [2,3]. Approximately 18–35% of individuals with diabetes experience DD, which distinctly influences disease management and glycemic control, such that as DD increases, glycemic control worsens and vice versa [4–8].

In addition to a higher prevalence of affective changes, adults with diabetes have heart disease death rates two to four times higher than adults without diabetes [1]. Compared to adults without diabetes in the general population, adults with comorbid diabetes and EDS have a 2.4 to 3.5 times higher cardiovascular disease (CVD) mortality rate [9]. Furthermore, adults with diabetes and psychological distress have a 1.7 times higher rate of CVD events [10], and adults with diabetes and DD have a 1.8 times higher rate of ischemic heart disease compared to adults without diabetes [11].

For many years, it has been hypothesized that major (clinical) depression is the predominant psychological factor influencing behaviors and motivation in adults with T2DM, leading to chronic hyperglycemia and adverse complications such as those related to cardiovascular health [12]. More recently, however, newer studies have shown that the majority of individuals with T2DM and EDS are not clinically depressed and do not require a diagnosis of clinical depression or a major depressive disorder; rather, their emotional symptoms are more reflective of DD [13]. Furthermore, the presence of EDS, SPD, or DD has differing independent effects on diabetes and CVD associated risk factors such as hemoglobin A1c (HbA1c), systolic blood pressure (SBP), diastolic blood pressure (DBP), and low-density lipoprotein cholesterol (LDL-C). For example, evidence shows the prevalence of coronary heart disease (CHD) and T2DM is significantly higher among adults with SPD than those

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

without SPD [14]. Additionally, hypertension (HTN), a manifestation of CVD, is more common in individuals with SPD than those without SPD [15] and has been found to occur in almost 56% of individuals with T2DM and major (clinical) depression [16]. Finally, HbA1c is higher among individuals with psychological distress, such as anxiety, social dysfunction, and loss of confidence than those without a comorbid psychological condition [17].

Adverse outcomes and high rates of morbidity and mortality associated with T2DM and CVD necessitate better understanding of CVD risk factor control in patients with comorbid affective changes such as EDS, SPD, and DD. Understanding these associations will aid in the developments of targeted health interventions and the ability to reduce overall CVD morbidity and mortality in adult patients with T2DM. Previous research has documented an association between poor glycemic control and either EDS or DD among individuals with T2DM [4–6,8,11,12]; however, evidence supporting associations between other CVD risk factors and DD, SPD, and EDS, both separately and collectively, is sparse. Therefore, we hypothesize there is an association between poor risk factor control and DD independent of the effect of EDS and SPD in patients with T2DM.

2. MATERIAL AND METHODS

2.1. Research Design

This study was conducted as part of a larger study funded by the National Institutes of Health/National Institute of Diabetes & Digestive & Kidney Diseases (NIH/NIDDK). A self-report survey was used to recruit participants and yield data on demographics, EDS, SPD, and DD. A records review supplied patient data on HbA1c, SBP, DBP, and LDL.

2.2. Sample selection and setting

A random sample of patients was recruited from two sites: a general internal medicine clinic at an academic institution (university clinic) and a primary care clinic of a Veterans Affairs Medical Center (VAMC). Eligible patients were clinic patients, 18 years of age and older with a diagnosis of T2DM in their medical record, and a clinic appointment between May 2013 and August 2013. Patients were ineligible if they did not speak English, or if the research assistants determined (by interaction or chart documentation) they were cognitively impaired or too ill to participate. Over a three-month period, participants with a clinical diagnosis of T2DM were approached in the waiting areas of the two recruitment clinics. After relocation to a private area, research assistants administered affective status scales to the participants to assess EDS, SPD, and DD. The demographic characteristics collected and reported here were based on self-report. The response rate was approximately 90%. We did not capture data on nonparticipants, so we are unable to describe differences between participants and non-participants.

2.3. Study Variables

2.3.1. Demographic Characteristics—Demographic variables collected for this study included age, gender, race/ethnicity, marital status, educational level, employment status, annual income level, and health insurance. Age was assessed as a continuous variable, but

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

then categorized into four age categories (18–49, 50–64, 65–74, and 75–89 years old). Gender was dichotomized into two groups: female or male. Marital status was categorized into five groups: 1) never married, 2) married, 3) separated, 4) divorced, or 5) widowed. Ethnicity was based on self-report as either 1) Hispanic/Asian/American Indian, 2) non-Hispanic White (NHW), or 3) non-Hispanic Black (NHB). Years of education was categorized into four groups: 1) <high school, 2) high school, 3) college, or 4) graduate level education. Eight income levels were defined: 1) $0 – $9,999; 2) $10,000 – $14,999; 3) $15,000 – $19,999; 4) $20,000 – $24,999; 5) $25,000 – $34,999; 6) $35,000 – $49,999; 7) $50,000 – $74,999; 8) >$75,000. Insurance status was divided into six groups: 1) no insurance, 2) private insurance, 3) Medicare, 4) Medicaid, 5) VA/military insurance, or 6) other insurance. Comorbid conditions were first counted (maximum possible comorbid conditions was 11) and then divided into four groups: 1) 0–1, 2) 2–3, 3) 4–5, or 4) 6–11. These conditions included 1) myocardial infarction; 2) coronary heart disease; 3) stroke; 4) asthma; 5) cancer; 6) chronic obstructive pulmonary disease, emphysema, or chronic bronchitis; 7) arthritis, gout, lupus, or fibromyalgia; 8) kidney disease (excluding kidney stones, bladder infection or incontinence); 9) vision impairment; 10) kidney failure requiring dialysis or transplant; or 11) toe, foot or leg amputation.

2.4. Definitions of Psychological Complications and Study Instruments

2.4.1. Patient Health Questionnaire-8 (PHQ-8)—The Patient Health Questionnaire-8 (PHQ-8) scale is a validated screening instrument used to identify persons experiencing elevated depressive symptoms [18,19]. This survey includes eight of the nine DSM-IV (Diagnostic and Statistical Manual of Mental Disorders) major depression disorder criteria: anhedonia, depressed mood, trouble sleeping, loss of energy, changes in appetite, trouble concentrating, and psychomotor retardation or agitation experienced over the last two weeks [20]. The questions are scored on a scale of 0–3, with 0 indicating “not at all,” 1 indicating “several days,” 2 indicating “more than half the days,” and 3 indicating “nearly every day”. The score for each item is summed to produce a total score between 0 and 24. A PHQ-8 score ≥10 suggests the presence of elevated depressive symptoms; however, this score cannot be definitively equated to a clinical diagnosis of major (clinical) depression (i.e., major depressive disorder) without confirmation from a clinician. Therefore, for the purposes of this study, PHQ-8 scores are indicative of EDS.

2.4.2. Serious Psychological Distress Scale (K6)—SPD involves experiencing distress related to having a severe mental illness such as schizophrenia, bipolar disorders, major depressive disorders, schizoaffective disorders, and post-traumatic stress disorders lasting twelve or more months [3]. The Kessler Psychological Distress Scale (K6) is a six-item screening tool used to identify the presence and severity of psychological distress during the past 30 days [21–25]. Each question is scored from 0–4, with 0 indicating “none of the time”, 1 indicating “a little of the time,” 2 indicating “some of the time,” 3 indicating “most of the time,” and 4 indicating “all of the time”. These scores are then summed to yield a total score between 0–24. A score of 13 or more indicates serious psychological distress [21,25].

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

2.4.3. Diabetes Distress Scale-17 (DDS17)—DD specifically refers to individual patient concerns about disease management, support, emotional burden, and access to care [3]. The Diabetes Distress Scale-17 (DDS17) is a 17-item internally consistent (α=0.93) questionnaire that assesses diabetes-related emotional distress in research and clinical practice [26,27]. The responses to each item are rated between 1 (no distress) and 6 (serious distress) concerning distress experienced over the last month [26 Mean item scores are then calculated with scores 1) <2.0 indicative of little or no distress, 2) 2.0–2.9 indicative of moderate distress, and 3) ≥3.0 indicative of high distress.

2.5. Statistical Analyses

We performed four sets of analyses. First, we calculated sample percentages for each demographic variable using aX^2 test. Second, we used Pearson's correlation to assess the relationship between HbA1c, SBP, DBP, LDL-C, PHQ-8, K6, and DDS17. Third, multiple linear regression models were computed using variables for CVD risk factor control (HbA1c, SBP, DBP, and LDL-C) as the outcomes and each affective status scale (PHQ-8, K6, and DDS17) as primary independent variables adjusting for covariates, which included race/ethnicity, gender, education, income, site, age, marital status, insurance, comorbid conditions. For each outcome in this model, the independent relationship with each individual affective status scale was analyzed. Finally, we ran a fully adjusted linear regression model in which the CVD risk factor control outcomes (HbA1c, SBP, DBP, and LDL-C) were used as the dependent variables; PHQ-8, K6, and DDS17 were all added as primary independent variables; and race/ethnicity, gender, education, income, site, age, marital status, insurance, and comorbidity burden were included as covariates. For each outcome in this model, all three affective status scales were added simultaneously to assess the collective relationship between the independent predictors and outcomes. Statistical significance was based on the two-tailed p-value of 0.05. Stata software version 13 was used for statistical analyses [28].

3. RESULTS

Table 1 shows demographic characteristics of the total sample population of adults with T2DM. There were a total of 361 patients with T2DM in the study, with a nearly equivalent percentage of patients from each site (51% from the academic medical center and 49% recruited from the VAMC). The majority of patients was male (66%), between the ages of 50–74 years old (72%), NHB (60%), and married (47%). Approximately 50% of the sample had a college degree or higher. More than half of the sample (54%) had an annual household income <$25,000, with the majority of the sample having some form of health insurance (96%). Nearly 70% had four or more comorbid conditions.

Table 2 shows the unadjusted correlation between EDS, SPD, and DD, as measured by PHQ-8, K6, and DDS17 respectively, and cardiovascular risk factors (HbA1c, SBP, DBP, and LDL-C). There was a significant correlation between DD and HbA1c, DBP, and LDL-C control. EDS and SPD were not significantly associated with any CVD risk factors. Correlation between the affective status scales was also performed. The correlations between DDS17 and PHQ8 and K6 were 0.43 and 0.45, respectively. The correlation

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

between PHQ8 and K6 was 0.61. Based on the correlations between DDS17, PHQ8 and K6, it appears different constructs are being measured despite the relatedness of the affective variants.

Table 3 shows the adjusted linear regression model for each affective status scale (EDS, SPD, and DD) and multiple CVD risk factors. DD was significantly associated with HbA1c (β coefficient = 0.322, 95% CI = 0.1231, 0.5201) and LDL-C (β coefficient = 5.7118, 95% CI = 0.4116, 11.0119). SPD was significantly associated with LDL-C (β coefficient = 1.0039, 95% CI = 0.0235, 1.9843). EDS was not associated with any of the CVD risk factors.

Table 4 shows the fully adjusted model in which the three affective status scales were included in the same model simultaneously, adjusting for covariates. DD was significantly associated with HbA1c (β coefficient = 0.3944, 95% CI = 0.1764, 0.6125) independent of the other affective status indicators. The other CVD risk factors—SBP, DBP, and LDL—were not significantly associated with DD in the final adjusted model. Additionally, there were no significant associations between SPD and EDS and CVD risk factors in the final model when adjusting for sociodemographic characteristics and comorbidity burden.

4. DISCUSSION AND CONCLUSIONS

4.1. Discussion

In this sample of adult patients with T2DM, DD was significantly associated with HbA1c in adjusted models, independent of influences from EDS and SPD. In the unadjusted correlation, DD was significantly associated with HbA1c, DBP, and LDL-C; however, after adjusting for relevant confounders, DD was only significantly associated with HbA1c and LDL-C. Neither EDS nor SPD were significantly correlated with any of the CVD risk factors; however, SPD was significantly associated with LDL-C in the unadjusted analyses. When adjusting for covariates, DD was significantly associated with HbA1c and LDL-C, while SPD was significantly associated with LDL-C. However, when all three affective status scales (DD, SPD, EDS) were included in the same model, only DD remained significantly associated with HbA1c, while controlling for relevant confounding factors. This finding suggests that DD is more strongly associated with CVD risk factor control than either EDS or SPD, and may require further attention when caring for adult patients with T2DM. Composite control of risk factors in patients with DD should be a goal in management for patients with T2DM to decrease risk of poor CVD outcomes.

Our findings are supported by evidence from previous studies evaluating the relationship between psychological complications and glycemic control, where DD has been shown to present a more significant risk for poorer HbA1c and outcomes compared to EDS or SPD. In this sample, we found DD (and not EDS and SPD) to be significantly associated with HbA1c when adjusting for relevant confounding factors. This is similar to the findings demonstrated by Fisher and colleagues, who found a direct relationship between DD and HbA1c control in individuals with T2DM [4–8,11]. In a study of 234 patients with T2DM to examine the effects of changes in depressive symptoms and DD on glycemic control, Zagarins et al [8] found changes in DD (and not in depressive symptoms) to be associated

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

with a significant change in HbA1c. In final adjusted models assessing the independent effects of socioeconomic and psychological social determinants of health on diabetes outcomes including self-care behaviors and quality of life, Walker and colleagues found significant association between DD and HbA1c and self-care behaviors such as medication adherence [29]. Additionally, although previous research once attributed major (clinical) depression to be more related to poorer HbA1c control, our study findings align with newer research indicating DD to be a stronger associating factor, not clinical depression, in glycemic control [6,12,13]. Finally, with regard to psychological distress and HbA1c in individuals with T2DM, prior evidence suggests similar findings to our research demonstrating no association between psychological distress and level of HbA1c [10,17].

Given individuals with T2DM have elevated rates of ischemic heart disease and are 2 to 4 times more likely to have heart disease, our research demonstrates the importance of managing CVD risk factors such as blood pressure [1,11]. Specifically, more evidence is needed to understand the relationship between blood pressure control and affective changes such as DD, EDS, and SPD. The effects of elevated systolic or diastolic blood pressures have commonly been grouped together as HTN, with higher rates of HTN in individuals with both SPD and T2DM [14,15]. In a study aimed to estimate the prevalence of psychological distress in individuals with and without CVD risks, individuals with ischemic heart disease (adjusted odds ratio (AOR), 2.3) were more likely to have SPD compared to those without, and those with SPD and HTN (AOR, 2.6) were more likely to have higher out-of-pocket burdens compared to patients with only CVD conditions [30]. Decreased rates of HTN were found by Khuwaja and colleagues among those with depression and T2DM [16]. However, our research findings indicate no direct association between SBP or DBP and EDS, DD, or SPD in individuals with T2DM. Subsequent studies should focus on the relationship between varying affective states and blood pressure control.

Prior research has differed on how psychological changes may be associated with LDL-C control in individuals with T2DM. In our study, we demonstrated DD and SPD to be significantly associated with LCL-C; however, when all affective constructs were included in the same model, these associations did not persist as LDL-C was not significantly associated with any of the affective status scales. Similarly, previous studies have not shown statistically significant higher rates of LDL-C in individuals with psychological distress and T2DM [10,13], while higher cholesterol levels have been seen in those with SPD and T2DM in other research [14,15]. Additionally, research with respect to the effects of EDS and DD on LDL-C in individuals with T2DM is limited. As with blood pressure control, subsequent studies are needed to examine the relationship between affective changes and LDL-C control.

This study contains limitations that must be mentioned. First, the cross-sectional design of the study does not allow for cause-effect inferences. It is also important to consider that as this data is cross-sectional, the opposite direction is possible, whereby increased difficulty in self-managing T2DM and CVD risk factors could influence the presence and development of DD. It will be important to include DD (in addition to other psychological constructs) in longitudinal analyses to understand direction and causality. Second, there are potential confounders that were not controlled for, including diabetes knowledge, self-management

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

practices, medication adherence, and social support. Additionally, we did not have information on the duration of diabetes or the medications used to treat diabetes or hypertension. Third, the generalizability of the study findings may be limited given the study population. These results are representative of a sample comprised of individuals from health care facilities in the Southeast and may not be reflective of other U.S. patient populations with T2DM. Finally, it is noteworthy to mention that while SPD, EDS, and DD were assessed as separate and distinct constructs in these analyses, it is possible SPD and EDS are alternative measures of non-specific distress, while DD is specific to diabetes. Thus, it may be that the real finding is that diabetes-specific distress is a stronger driver of poor outcomes than non-specific distress.

4.2. Conclusions

Despite the aforementioned study limitations, the results of our study are important and provide new information about the relationship between affective status variants and CVD risk factor control in patients with T2DM. In this study of patients with T2DM, DD and SPD each had a significant independent association with CVD risk factor control when controlling for covariates; however, only DD was significantly associated with CVD risk factor control, specifically glycemic control, when multiple affective status scales were present in the model. These results signify the need for additional research to understand the impact of compromised mental health on CVD risk factor control in adults with T2DM. When treating EDS in patients diagnosed with T2DM, it is imperative to also pay attention to DD, as it may be more strongly associated with CVD risk factor control compared to other psychological constructs. Strategies such as resource utilization, patient education, and counseling are needed to minimize the impact of mental health disorders on CVD risk control in T2DM.

4.3. Practice Implications

According to the American Diabetes Association, DD is a difficult complication to identify as it can be confused with other mental disorders such as clinical depression, anxiety, and stress. Often times, individuals diagnosed with T2DM endorse psychological symptomology that does not meet the criteria for Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV or DSM-V or International Classification of Diseases (ICD) Ninth Edition (ICD-9) or ICD-10 diagnoses such as major depressive disorder. This inability to distinguish between different psychological constructs makes it challenging to identify patients with T2DM who have comorbid mental health conditions. As such, DD is often times recognized as a complication that occurs secondary to the worry, frustration, burden, and burnout associated with managing a chronic disease such as diabetes. Fisher and colleagues found patients diagnosed with DD demonstrated poorer glycemic control and worse outcomes. They were less likely to perform self-management behaviors such as blood sugar monitoring and medication adherence. In our study, we found DD was significantly associated with poorer glycemic control and should be addressed when caring for patients with T2DM.

Several strategies for reducing DD in patients with T2DM can be suggested. First, patients with T2DM are often cared for by primary care providers where current clinical guidelines encourage screening for affective changes such as EDS and depression, especially in

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

patients with chronic diseases. DD is a complication that should be monitored at both the patient and provider level; therefore, general practitioners caring for patients with T2DM and comorbid DD should receive training in the identification and management of DD, as well as be motivated to share care with the patients and collaborate with interdisciplinary teams to treat the symptoms. Additionally, routine screening for DD, for example, can be feasible and assessed by administering validated, self-report screening instruments such as the DDS17 and DDS2 to patients with T2DM at each visit. Symptoms can be measured that approximate clinical levels of DD and other psychological states such as EDS and treated appropriately. Given the association between psychological complications and poorer outcomes, there is a need for improved recognition and prompt treatment of DD in patients with T2DM. Future strategies should be considered that include broader screening for mental health problems associated with T2DM to improve overall quality of life and outcomes. Second, it is important to help patients with T2DM recognize that comorbid psychological conditions and complications are prevalent and can impede their abilities to properly care for themselves. Patients must be reassured that although self-managing T2DM can be a daunting task, control can be accomplished through slow progression, which will ultimately assist with the reduction of DD. For example, four important behaviors needed to manage diabetes include adhering to medical regimens, adopting healthier eating habits, improving physical activity levels, and monitoring daily blood sugar readings. Providers can explain that a manageable approach to managing T2DM may not include addressing all four skills simultaneously (which is associated with higher levels of DD), but may require patients to identify the area of greatest weakness (i.e., the behavior that causes the most distress) as an area of initial focus. As progress is made towards achieving the initial goals, efforts must be rewarded, so the patients remain empowered and motivated to achieve subsequent goals, thereby limiting the associated DD that was once associated with poorer outcomes. Third, sources of social support should be identified for all patients with T2DM, whether personal contacts or a group of colleagues sharing the same diagnoses. Evidence suggests that higher levels of social support are associated with improved clinical outcomes, reduced psychosocial symptomatology such as DD, and the adaptation of beneficial lifestyle activities. It is imperative that providers identify personal support networks and involve them in management plans for patients with T2DM. Having a supportive and more relatable person (aside from the provider) with whom different aspects of diabetes management can be discussed will also help to reduce the distress associated with caring for a chronic disease such as diabetes. Additionally, patients with T2DM might also benefit from attending group sessions to address other aspects of diabetes care such as educational, nutritional, and spiritual growth and development. By doing so, patients may establish coping mechanisms for dealing with the added stress associated with managing T2DM such as keeping a journal, developing relaxation techniques, getting organized, and setting goals. Fourth, because self-managing T2DM can lead to the regular recurrence of DD, periodic assessment and monitoring of DD (and other comorbid psychological and medical conditions and complications) by clinicians is required in the care of patients with T2DM. This is necessary to maximize wellbeing, empower patients, and remove obstacles preventing more appropriate management. Furthermore, it is important to better understand the biologic mechanisms that exist between DD and glycemic control. More evidence is needed to

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

understand how DD relates to the likelihood of having poorer clinical and behavioral outcomes.

Having the availability of multiple strategies such as skill development and a coordinated approach to clinical care between the patient, support systems, and clinicians will serve as targeted approaches to reducing DD, and ultimately improving CVD risk factor control in adults with T2DM.

ACKNOWLEDGEMENTS

Funding Source: This study was supported by Grant K24DK093699 from The National Institute of Diabetes and Digestive and Kidney Disease (PI: Leonard Egede). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

REFERENCES

1. Centers for Disease Control and Prevention. [8 January 2014] National Diabetes Fact Sheet: National Estimates and General Information on Diabetes and Prediabetes in the United States, 2011. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. 2011. Web. <http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2011.pdf>.
2. Li C, Ford E, Zhao G, Balluz L, Berry J, Mokdad A. Undertreatment of mental health problems in adults with diagnosed diabetes and serious psychological distress. Diabetes Care. 2010; 33:1061–1064. [PubMed: 20185747]
3. Egede LE, Dismuke CE. Serious psychological distress and diabetes: a review of the literature. Curr Psychiatry Rep. 2012; 14:15–22. [PubMed: 22002804]
4. Fisher L, Hessler D, Polonsky W, Mullan J. When is diabetes distress clinically meaningful? Diabetes Care. 2012; 35:259–264. [PubMed: 22228744]
5. Fisher L, Glasgow RE, Strycker LA. The relationship between diabetes distress and clinical depression with glycemic control among patients with type 2 diabetes. Diabetes Care. 2010; 33:1034–1036. [PubMed: 20150291]
6. Fisher L, Mullan JT, Arean P, Glasgow RE, Hessler D, Masharani U. Diabetes distress but not clinical depression or depressive symptoms is associated with glycemic control in both cross-sectional and longitudinal analyses. Diabetes Care. 2010; 33:23–28. [PubMed: 19837786]
7. Tol A, Baghbanian A, Sharifirad G, Shojaeizadeh D, Eslami A, Alhani F, Tehrani MM. Assessment of diabetic distress and disease-related factors in patients with type 2 diabetes in Isfahan: a way to tailor an effective intervention planning in Isfahan-Iran. J Diabetes Metab Disord. 2012; 11(20)
8. Zagarins S, Allen NA, Garb JL, Welch G. Improvement in glycemic control following a diabetes education intervention is associated with change in diabetes distress but not change in depressive symptoms. J Behav Med. 2012; 35:299–304. [PubMed: 21691844]
9. Egede L, Nietert P, Zheng D. Depression and all-cause and coronary heart disease mortality among adults with and without diabetes. Diabetes Care. 2005; 28:1339–1345. [PubMed: 15920049]
10. Dalsgaard E, Vestergaard M, Skriver M, Maindal H, Lauritzen T, Borch-Johnsen K, Witte D, Sandbaek A. Psychological distress, cardiovascular complications and mortality among people with screen-detected type 2 diabetes: follow-up of the ADDITION-Denmark Trial. Diabetologia. 2014; 57:710–717. [PubMed: 24442448]
11. Adriaanse MC, Pouwer F, Dekker JM, Nijpels G, Stehouwer CD, Heine RJ, Snoek FJ. Diabetes-related symptom distress in association with glucose metabolism and comorbidity. Diabetes Care. 2008; 31:2268–2270. [PubMed: 18728236]
12. Gonzalez JS, Fisher L, Polonsky WH. Depression in diabetes: have we been missing something important? Diabetes Care. 2011; 34:236–239. [PubMed: 21193621]
13. Fisher L, Skaff MM, Mullan JT, Arean P, Mohr D, Masharani U, Glasgow R, Laurencin G. Clinical depression versus distress among patients with type 2 diabetes: not just a question of semantics. Diabetes Care. 2007; 30:542–548. [PubMed: 17327318]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

14. New Hampshire Department of Health and Human Services. [Last accessed: December 29, 2014] Serious psychological distress among NH adultsm, 2007—Issue Brief. New Hampshire Behavioral Risk Factor Surveillance System. 2009. Available at: http://www.dhhs.nh.gov/dphs/hsdm/brfss/documents/pyschdistress.pdf.
15. Murphy T. Effects of chronic medical conditions and serious psychological distress on health and access to care. New Mexico Epidemiology Report. 2010 Available at: http://www.astho.org/accreditation/new-mexico-state-health-assessment/.
16. Khuwaja AK, Lalani S, Dhanani R, Azam IS, Rafique G, White F. Anxiety and depression among outpatients with type 2 diabetes: a multi-centre study of prevalence and associated factors. Diabetol Metab Syndr. 2010; 2:1–7. [PubMed: 20180954]
17. Hamer M, Stamatakis E, Kivimaki M, Pascal Kengne A, Batty GD. Psychological distress, glycated hemoglobin, and mortality in adults with and without diabetes. Psychosom Med. 2010; 72:882–886. [PubMed: 20884891]
18. Egede L, Ellis C. Diabetes and depression: global perspectives. Diabetes Res Clin Pract. 2010; 87:302–312. [PubMed: 20181405]
19. Kroenke K, Strine TW, Spitzer RL, Williams JB, Berry JT, Mokdad AH. The PHQ-8 as a measure of current depression in the general population. J Affect Disord. 2009; 114:163–173. [PubMed: 18752852]
20. Hudson DL, Karter AJ, Fernandez A, Parker M, Adams AS, Schillinger D, Moffet HH, Zhou J, Adler NE. Differences in the clinical recognition of depression in diabetes patients: The Diabetes Study of Northern California (DISTANCE). Am J Manag Care. 2013; 19:344–352. [PubMed: 23781889]
21. Kessler RC, Green JG, Gruber MJ, Sampson NA, Bromet E, Cuitan M, Furukawa TA, Gureje O, Hinkov H, Hu CY, Lara C, Lee S, Mneimneh Z, Myer L, Oakley-Browne M, Posada-Villa J, Sagar R, Viana MC, Zaslavsky AM. Screening for serious mental illness in the general population with the K6 screening scale: results from the WHO World Mental Health (WMH) Survey Initiative. Int J Methods Psychiatr Res. 2010; 19:4–22. [PubMed: 20527002]
22. Kessler RC, Andrews G, Colpe LJ, Hiripi E, Mroczek DK, Normand SL, Walters EE, Zaslavsky AM. Short screening scales to monitor population prevalences and trends in non-specific psychological distress. Psychol Med. 2002; 32:959–976. [PubMed: 12214795]
23. Kessler RC, Barker PR, Colpe LJ, Epstein JF, Gfroerer JC, Hiripi E, Howes MJ, Normand SL, Manderscheid RW, Walters EE, Zaslavsky AM. Screening for serious mental illness in the general population. Arch Gen Psychiatry. 2003; 60:184–189. [PubMed: 12578436]
24. Mitchell CM, Beals J. The utility of the Kessler Screening Scale for Psychological Distress (K6) in two American Indian communities. Psychol Assess. 2011; 23:752–761. [PubMed: 21534694]
25. Centers for Disease Control and Prevention. Serious psychological distress. [accessed February 2, 2007] National Center for Health Statistics: Survey questionnaire, National Health Interview Survey, 2002. 2004. <http://www.cdc.gov/pub/Health_Statistics/NCHS/Survey_Questionnaires/NHIS/2002/>
26. Fisher L, Glasgow RE, Mullan JT, Skaff MM, Polonsky WH. Development of a brief diabetes distress screening instrument. Ann Fam Med. 2008; 6:246–252. [PubMed: 18474888]
27. Polonsky WH, Fisher L, Earles J, Dudl RJ, Lees J, Mullan J, Jackson RA. Assessing psychological distress in diabetes: development of the DDS. Diabetes Care. 2005; 28.3:626–631. [PubMed: 15735199]
28. STATA version 12.0 [computer program]. College Station, TX: STATA Press; 2011.
29. Walker RJ, Gebregziabher M, Martin-Harris B, Egede LE. Independent effects of socioeconomic and psychological social determinants of health on self-care and outcomes in type 2 diabetes. Gen Hosp Psychiatry. 2014; 36:662–668. [PubMed: 25103544]
30. Okumura Y, Ito H. Out-of-pocket expenditure burdens in patients with cardiovascular conditions and psychological distress: a nationwide cross-sectional study. Gen Hosp Psychiatry. 2013; 35:233–238. [PubMed: 23391611]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

HIGHLIGHTS

- Examined association between CVD risk factors and psychological factors in T2DM
- Primary outcome was CVD risk factor control (A1c, SBP, DBP and LDL)
- Psychological factors included depression, SPD, and diabetes distress (DD)
- DD was associated with A1c and LDL, and SPD with LDL in partially adjusted models
- In the fully adjusted model, only DD remained significantly associated with A1c

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 1

Sample Demographics (n=361)

Age	
18–49 years	10.6
50–64 years	43.0
65–74 years	29.1
75–89 years	17.3
Gender/Sex	
Female	33.8
Male	66.2
Race/Ethnicity	
Non-Hispanic White	36.7
Non-Hispanic Black	60.0
Hispanic/Asian/American Indian	3.3
Marital status	
Never married	13.1
Married	47.2
Separated	8.3
Divorced	18.1
Widowed	13.3
Educational level	
Less than HS graduate	15.8
HS graduate	33.2
College	40.4
Grad education	10.5
Annual income level	
$0–$9,999	18.8
$10,000–$14,999	13.6
$15,000–$19,999	11.6
$20,000–$24,999	9.7
$25,000–$34,999	15.0
$35,000–$49,999	10.3
$50,000–$74,999	9.4
>$75,000	11.6
Health insurance	
None	3.6
Private	10.5
Medicare	16.9
Medicaid	17.7
VA	40.6
Other	10.8
Comorbidities	

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

0–1 condition	5.3
2–3 conditions	25.7
4–5 conditions	34.5
6–11	34.5
Site	
Academic Medical Center	51.0
Veterans Affairs Medical Center	49.0
Psychological ConditionsComplications	
Diabetes Distress	1.4 +/− 0.6
Serious Psychological Distress	4.6 +/− 5.4
Elevated Depressive Symptoms	5.9 +/− 5.1

All numbers represent percentages or mean +/− SD.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 2

Unadjusted Correlation between DDS17, PHQ8, and K6 and CVD Risk Factor Outcomes

	HbA1c	Systolic BP	Diastolic BP	LDL-C
DDS17	0.179[a]	0.084	0.150[a]	0.133[a]
PHQ8	−0.024	−0.040	0.046	0.080
K6	−0.006	0.024	0.071	0.081

[a]Statistically significant difference at p <0.05.

DDS17=Diabetes Distress Scale-17; PHQ8=Patient Health Questionnaire-8; and K6=Serious Psychological Distress Scale

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 3

Adjusted Linear Regression Model for the Relationship between DDS17, PHQ8, and K6 and Outcomes

	HbA1c	Systolic BP	Diastolic BP	LDL-C
	β Coefficient (95% CI)	β Coefficient (95% CI)	β Coefficient (95% CI)	β Coefficient (95% CI)
DDS17	0.476[a] (0.161, 0.791)	−1.24 (−4.407, 1.919)	−0.685 (−2.751, 1.380)	8.198[a] (−0.169, 16.565)
PHQ8	−0.000 (−0.043, 0.042)	−0.213 (−0.634, 0.208)	0.045 (−0.230, 0.321)	0.737 (−0.341, 1.815)
K6	−0.003 (−0.042, 0.036)	−0.069 (−0.453, 0.315)	0.138 (−0.112, 0.388)	1.004[a] (0.024, 1.984)

[a]Statistically significant difference, $p<0.05$.

β=Beta coefficient. DDS17=Diabetes Distress Scale-17; PHQ8=Patient Health Questionnaire-8; K6=Serious Psychological Distress Scale. Adjusted for race/ethnicity, gender, education, income, site, age, marital status, insurance, and comorbidity burden

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 4

Final Adjusted Linear Regression Model of DDS17, PHQ8, and K6 and CVD Risk Factor Control

	HbA1c	Systolic BP	Diastolic BP	LDL-C
	β Coefficient (95%CI)	β Coefficient (95%CI)	β Coefficient (95%CI)	β Coefficient (95%CI)
DDS17	0.611[a] (0.258, 0.963)	−0.894 (−4.447, 2.658)	−1.336 (−3.650, 0.979)	5.823 (−3.541, 15.186)
PHQ8	−0.018 (−0.071, 0.036)	−0.228 (−0.761, 0.304)	−0.030 (−0.376, 0.317)	−0.071 (−1.444, 1.302)
K6	−0.025 (−0.074, 0.025)	0.095 (−0.391, 0.582	0.219 (−0.099, 0.536)	0.769 (−0.476, 2.014)

[a] Statistically significant difference, p<0.05.

β=Beta coefficient. DDS17=Diabetes Distress Scale-17; PHQ8=Patient Health Questionnaire-8; K6=Serious Psychological Distress Scale. Adjusted for race/ethnicity, gender, education, income, site, age, marital status, insurance, and comorbidity burden