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Improvement in glycemic control following a diabetes education intervention is associated with change in diabetes distress but not change in depressive symptoms

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Abstract In diabetes patients, depression is correlated with diabetes-specific emotional distress, and observational studies have suggested that diabetes distress may have a greater impact on diabetes outcomes than depression itself. To examine the relative effects of change in depressive symptoms and change in diabetes distress on change in glycemic control, we conducted a diabetes self-management education intervention in 234 type 2 diabetes (T2DM) patients, and measured glycemic control (HbA1c), depressive symptoms (CES-D), and diabetes distress (PAID) at baseline and 6 months. In multiple linear regression, change in depressive symptoms was not associated with change in HbA1c ($P = 0.23$). Change in diabetes distress was significantly associated with change in HbA1c ($P < 0.01$), such that a 10-point decrease in diabetes distress (which corresponds to the average change in distress in this study population) was associated with a 0.25% reduction in HbA1c. Change in diabetes distress, and not change in depressive symptoms, was associated with both short- and long-term change in glycemic control for patients with poorly controlled T2DM.

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Introduction

Depression is two to three times more prevalent in diabetes patients compared to the general population (Ali et al. 2006; Fisher et al. 2008), and the combination of diabetes and depression has been associated with diabetes-related morbidity and mortality (Egede and Ellis 2010; Lin et al. 2010). The relationship between depression and poor glycemic control in diabetes patients has been well-studied and was supported in a large meta-analysis published in 2000 (Lustman et al. 2000), as well as in a recent meta analysis by van der Feltz-Cornelis et al. (van der Feltz-Cornelis et al. 2010). This more recent meta analysis analyzed different types of depression interventions, and while no effect was found for collaborative care and only a small effect was found for pharmacological interventions, a moderate effect was found for psychotherapeutic interventions on glycemic control. However, three of the five psychotherapeutic interventions included a diabetes education component, and it is unclear whether the psychotherapy or this education was driving the change in glycemic control.

Contrary to these positive findings, recent reviews of pharmacologic and non-pharmacologic treatments for depression found that these treatments did not improve diabetes outcomes (Petrak and Herpertz 2009; Wang et al. 2008), and recent prospective studies, including one cognitive behavioral therapy intervention in depressed patients (Georgiades et al. 2007), found no association between depression and glycemic control (Aikens et al. 2009; Fisher

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et al. 2010; Georgiades et al. 2007). These recent studies have raised concerns with the earlier positive findings, including the use of populations which included both type 1 and type 2 diabetes patients (Cohen et al. 1997; de Groot et al. 1999; Eaton et al. 1992; Konen et al. 1996; Lustman et al. 1986; Mazze et al. 1984; Van Tilburg et al. 2001), cross-sectional study designs (Cohen et al. 1997; de Groot et al. 1999; Konen et al. 1996; Van der Does et al. 1996; Van Tilburg et al. 2001), or less rigorous methods of measuring depression (Konen et al. 1996; Van der Does et al. 1996).

The early positive findings for the relationship between depression and glycemic control may be explained in part by the fact that depression is correlated with diabetes-specific emotional distress in diabetes patients (Kokoszka et al. 2009; Pouwer et al. 2005), and this distress, rather than depression itself, may be driving the positive association with glycemic control. This hypothesis is supported by a recent cross-sectional study which reported that diabetes-specific emotional distress mediated the association between depressive symptoms and glycemic control (van Bastelaar et al. 2010). A second recent observational study reported that diabetes distress, and not depression or depressive symptoms, was associated with glycemic control (Fisher et al. 2010). However, no analyses related to the relative impact of distress versus depression on glycemic control have been conducted in the context of an intervention aimed at improving these outcomes.

Given the high prevalence of depression in diabetes patients, understanding the relative importance of depression and diabetes-specific emotional distress on glycemic control is critical for formulating effective strategies to help diabetes patients maintain glycemic control and minimize diabetes-related morbidity and mortality. This is the first analysis to examine the relative effects of change in depressive symptoms and change in diabetes distress on change in glycemic control following a behavioral (i.e., diabetes self-management education [DSME]) intervention.

Methods

Study design and participants

Participants were recruited from the adult type 2 diabetes (T2DM) patient population of a large medical center following chart review and physician approval for patient participation, as previously described (Welch et al. 2010). Participants were aged 30–80 years, had poorly controlled blood glucose (HbA1c $\geq$ 7.5%), and were able to speak and write in English. Exclusion criteria included the presence of major diabetes complications (i.e., proliferative retinopathy, cardiovascular conditions including stoke or myocardial infarction within the past 12 months, congestive heart failure, renal disease [microalbumin $>$ 300 ug/mg], severe autonomic neuropathy, lower limb amputations), pregnancy, any severe psychiatric disorders such as schizophrenia or mental retardation, or visual, literacy, or comprehension barriers that would prevent completion of study questionnaires.

All participants received four sessions of DSME within the 6-month intervention period. Participants were randomized to receive DSME from one of four certified diabetes educators (CDE; certified by the American Association of Diabetes Educators Diabetes Education Accreditation Program) who were part of the hospital diabetes program. Two of the study CDEs delivered standard DSME, and two delivered DSME using motivational interviewing (MI), a patient-centered counseling style that stresses the importance of the interaction between patient and clinician to influence patient motivation and improve target health behaviors. All participants received an initial DSME session for 1 h followed by three 30 min sessions at 1, 3, and 6 months. Participants completed a baseline research visit to assess demographic (e.g., age, gender, education level), clinical (e.g., glycemic control, BMI), and behavioral factors (e.g., depression, diabetes distress). Participants completed a second research visit following the final (6 month) DSME session during which the baseline assessments were repeated. The study was approved by the hospital's Internal Review Board committee.

Study measures

Glycosylated hemoglobin (HbA1c) was measured in random blood samples analyzed at the Baystate Medical Center reference laboratory. The Baystate Medical Center laboratory uses the HPLC ion capture method (Tosh Medics Inc., San Francisco, CA).

Data on behavioral factors (i.e., diabetes distress, depressive symptoms, diabetes self-care behaviors, treatment satisfaction) were collected by self-report using validated, reliable questionnaires. Diabetes distress was measured using the Problem Areas in Diabetes (PAID) scale (Polonsky et al. 1995), a 20-item scale which provides an overall measure of diabetes-related emotional distress, such that higher scores denote greater distress. The PAID has high internal reliability ($\alpha > 0.90$), sound concurrent validity as determined by moderate to strong correlations with a range of theoretically-related measures (Polonsky et al. 1995; Welch et al. 1997), and responsiveness to change with brief psychosocial and educational interventions (Welch et al. 2003). Depressive symptoms were measured using the Center for Epidemiologic Studies Depression Scale (CES-D), a reliable, widely used 20-item assessment of depressive feelings and behaviors during the past week (Radloff 1977). The standard cutoff score

of ≥16 on the CES-D has a sensitivity of 0.95 and a specificity of 0.70 for predicting major depression (Thomas et al. 2001). Diabetes self-care behaviors were measured using the Self Care Inventory-Revised (SCI-R; Weinger et al. 2005), which assesses patient perceptions of diet, exercise, medication adherence, and self-management of blood glucose. The Diabetes Treatment Satisfaction Questionnaire Change version (DTSQ-C; Bradley 1999) was used to assess change in patient satisfaction at the end of a treatment intervention.

Statistical analyses

Given that all study participants received a similar, four-session DSME intervention, all analyses were conducted in the population as a whole rather than within treatment groups. Treatment effect was adjusted for if groups differed by independent or dependent variables.

Means, standard deviations, and ranges were reported for continuous covariates (i.e., age, BMI, diabetes duration, HbA1c, questionnaire scores) and categorical covariates were described as the number and percent of participants in each category (i.e., gender, race/ethnicity, education level, medication use). Associations of baseline depressive symptoms and baseline diabetes distress with baseline HbA1c were assessed using multiple linear regression. Covariates associated with HbA1c and/or dependent variables at $P < 0.2$ were tested in regression models, and covariates associated with at least a 10% change in the beta coefficient for HbA1c were retained in final models.

Change scores for HbA1c and questionnaire scores were calculated as follow-up minus baseline values, such that positive values indicated an increase over time. Additional change scores were calculated for HbA1c, reflecting change in HbA1c from 6 to 12 months and from baseline to 12 months. Multiple linear regression was used to estimate associations between the HbA1c change variables and changes in depressive symptoms and diabetes distress. Appropriate covariates were adjusted for as described above. Analyses were conducted in the group as a whole, and then in groups defined by depression status (CES-D ≥ 16 vs. <16). We also conducted these analyses in groups defined by gender, as previous research has reported that the association between depression and HbA1c differs by gender (Cherrington et al. 2010). All analyses were conducted using SAS software version 9.1 (SAS Institute, Cary, NC).

Results

Of 545 patients who contacted study personnel and were screened for eligibility, 234 patients were eligible and enrolled in the study (Table 1) and 147 (62.8%) completed the second research visit (range of drop-out rates for study groups: 32.8–43.1%; $P = 0.61$). Treatment groups differed at baseline in terms of education status ($P = 0.02$) and insulin use ($P = 0.04$). Although patients who completed the 6-month follow-up visit and those who withdrew from the study differed in terms of age ($P = 0.01$), this difference was not clinically significant (mean age [SD]: 56.9 [9.5] vs. 53.6 [11.1], respectively).

Table 1 Selected baseline characteristics of study participants

	Mean (SD)	Range
Age (years)	55.7 (10.2)	31–80
Duration of diabetes (years)	8.2 (7.0)	0.5–38.0
BMI (kg/m^2)	34.5 (6.4)	20.6–57.4
Female	59.0%	–
White race	84.1%	–
Hispanic ethnicity	12.1%	–
Education beyond high school	63.6%	–
Use insulin	35.2%	–
Use oral agents	82.8%	–
HbA1c (%)	8.9 (1.2)	7.5–14.7
PAID score	42.1 (23.4)	0–96.3
CES-D score	16.3 (11.8)	0–58.0
SCI-R score	57.3 (16.4)	7.7–100.0

PAID Problem Areas in Diabetes, range: 0 (low distress)–100 (high distress); *CES-D* Center for Epidemiologic Studies—Depression scale, range: 0 (no depression)–60 (high depression), with ≥16 classified as major depression; *SCI-R* Self-Care Inventory—Revised, range: 0 (low self-care behaviors)–100 (high self-care behaviors)

In multiple linear regression analysis, depressive symptoms were associated with HbA1c at baseline, such that each 1-point reduction in depressive symptoms was associated with a 0.018% reduction in HbA1c (Table 2, Model 1). However, this effect for depressive symptoms was removed with further adjustment for diabetes distress and self-care behaviors (Table 2, Model 2 and Model 3).

Mean change in HbA1c from baseline to 6-month follow-up was $-0.58 \pm 1.33\%$ ($P < 0.01$) indicating a statistically and clinically significant improvement in blood glucose control during the intervention period (Table 3). Change in HbA1c following the 6-month intervention period (i.e., from 6 to 12 months) was not significant ($P = 0.92$), while change from baseline to 12 months was significant ($-0.49 \pm 1.56\%$; $P < 0.01$). Change in HbA1c did differ by treatment group, such that the MI + DSME group had a mean change in HbA1c that was significantly lower (i.e., less improved) than the group receiving DSME alone ($\beta \pm$ SE: 0.41 ± 0.19; $P = 0.037$).

Diabetes distress was reduced (i.e., improved), as shown by a clinically significant mean change in PAID score of -9.6 ± 16.5 ($P < 0.01$; Table 3). Self-care behaviors (SCI-R) were also improved, as was treatment satisfaction.

Table 2 Baseline association of depressive symptoms and diabetes distress with HbA1c estimated using multiple linear regression

	β (SE)	Model R^2 (adjusted)	*P*-value
Model 1			
Depressive symptoms (CES-D)	0.018 (0.007)	0.03	<0.01
Model 2			
Depressive symptoms (CES-D)	−0.003 (0.009)		0.75
Diabetes distress (PAID)	0.029 (0.007)	0.07	<0.01
Model 3			
Depressive symptoms (CES-D)	−0.002 (0.009)		0.85
Diabetes distress (PAID)	0.013 (0.005)		<0.01
Self-care behaviors (SCI-R)	−0.014 (0.005)	0.10	<0.01

See Table 1 note

Table 3 Change in covariates from baseline to 6 months

	Mean difference (SD)	*P*-value
BMI (kg/m^2)	−0.3 (2.3)	0.07
HbA1c (%): Change from baseline to 6 months	−0.58 (1.33)	<0.01
HbA1c (%): Change from 6 to 12 months	−0.01 (1.43)	0.92
HbA1c (%): Change from baseline to 12 months	−0.49 (1.56)	<0.01
Diabetes distress (PAID)	−9.6 (16.5)	<0.01
Depression (CES-D)	−1.2 (8.4)	0.08
Self-care behaviors (SCI-R)	9.1 (13.8)	<0.01
Treatment satisfaction	9.9 (6.0)	<0.01

See Table 1 note

Depressive symptoms tended to decrease over the course of follow-up ($P < 0.08$).

In multiple regression analyses adjusting for treatment group (MI vs. non-MI) and change in self-care behaviors, we found that change in diabetes distress was significantly associated with change in HbA1c from baseline to 6 month follow-up, such that a 10-point reduction on the PAID scale of diabetes distress (which corresponds to the average change in distress in the study population) was associated with a significant reduction in HbA1c of 0.25% ($P < 0.01$; Table 4). Change in depressive symptoms was not associated with change in HbA1c from baseline to 6 months ($P = 0.23$). Results were similar when change in HbA1c was defined as change from baseline to 12 months. When change in HbA1c was defined as change from 6 to 12 months, there was no significant association between change in HbA1c and change in diabetes distress or change in depressive symptoms. Findings did not differ in groups defined by depression status (i.e., CES-D $\geq$ 16 vs. <16; data not shown) or gender (data not shown).

Table 4 Association of change in depressive symptoms and change in diabetes distress with change in HbA1c estimated using multiple linear regression

	β (SE)	Model R^2 (adjusted)	*P*-value
Change in HbA1c: baseline to 6 months[a,b]			
Change in diabetes distress	0.025 (0.006)	0.15	<0.01
Change in depressive symptoms	0.015 (0.012)	0.06	0.23
Change in HbA1c: 6 to 12 months[b]			
Change in diabetes distress	−0.007 (0.007)	0.01	0.35
Change in depressive symptoms	−0.001 (0.014)	0.00	0.96
Change in HbA1c: baseline to 12 months[a,b]			
Change in diabetes distress	0.018 (0.007)	0.05	0.04
Change in depressive symptoms	0.016 (0.017)	0.02	0.35

Change from baseline to 6 months

[a] Adjusted change in self-care behaviors

[b] Adjusted for treatment group

Discussion

In this analysis of T2DM patients before and after a DSME intervention, change in HbA1c over the course of the 6-month intervention period was associated with change in diabetes-specific emotional distress during this same period, but not with change in depressive symptoms. Similar results were observed when change in HbA1c was defined as change from baseline to 12 months, suggesting that short-term change in diabetes distress is associated with long-term change in HbA1c. While previous observational studies have reported on the associations between glycemic control and depression (Aikens et al. 2009; Ali et al. 2006; Egede and Ellis 2010; Fisher et al. 2010; Georgiades et al. 2007; Lin et al. 2010; Lustman et al. 2000) as well as diabetes distress (Fisher et al. 2010; Kokoszka et al. 2009; van Bastelaar et al. 2010), this is the first analysis to examine the relative effects of change in depressive symptoms and change in diabetes-specific emotional distress on change in glycemic control before and after a behavioral intervention.

Early studies of the association between depressive symptoms and glycemic control yielded conflicting results, and many studies reporting a positive association between depression and glycemic control were based on cross-

sectional study designs (Cohen et al. 1997; de Groot et al. 1999; Konen et al. 1996; Van der Does et al. 1996; Van Tilburg et al. 2001). In a cross-sectional analysis of our baseline data, we did find a positive association between depressive symptoms and HbA1c, but this association was removed after adjustment for diabetes distress.

Our ability to detect an effect for change in depressive symptoms on change in HbA1c was limited by the relatively small change in depressive symptoms observed over the course of this 6-month intervention. Depression is a complicated condition affected by multiple factors, and longer-term interventions may be needed to affect depression rates. Furthermore, the DSME-based intervention did not address depression specifically, and an intervention tailored to treating depression may be necessary to observe an association with change in glycemic control. Finally, while there was a high level of patient engagement in the DSME intervention sessions, the drop-out rate for the 6-month research visits was relatively high (37%). This is likely attributable to patients feeling that they had gained what they needed from their participation in the four DSME sessions and that the final research visit wasn't important for their diabetes care. However, it is also possible that patients who did not return for the final research visit differed from those who did return in terms of depression status, and were in fact experiencing higher rates of depression. If this were the case, the ability of the study to detect an effect for change in depression status would have been affected (i.e., reduced).

Although the results of the current analysis do support the hypothesis that change in diabetes distress may affect change in HbA1c, changes in these measures happened concurrently during the 6-month intervention, and it is also possible that change in HbA1c was itself bringing about a change in diabetes distress. To try and determine whether the change in diabetes distress did in fact influence change in HbA1c (and not vice versa) we analyzed change in HbA1c following the intervention (i.e., from 6 to 12 months) and from baseline to 12 months. However, the most dramatic change in HbA1c occurred during the 6-month intervention, and so although change in diabetes distress from baseline to 6 months was associated with change in HbA1c from baseline to 12 months, most of this longer-term change in HbA1c was driven by change during the first 6 months (i.e., HbA1c tended to decrease from baseline to 6 months and then remain at this decreased level from 6 to 12 months). While it therefore cannot be concluded that change in diabetes distress is driving the observed change in HbA1c, we can conclude that a short-term reduction in diabetes distress is associated with a sustained reduction in HbA1c.

While clinical management of diabetes generally does not include evaluation and/or treatment for diabetes distress (Fisher et al. 2010; Pouwer 2009), there are a variety of ways in which DSME may improve both diabetes distress and glycemic control. One mechanism may involve development of coping skills that promote acceptance of the disease and management of anger, anxiety, and fears related to diabetes (Solowiejczyk 2010). Another possibility is that distress is reduced by the collaborative nature of the DSME program. DSME also involves processes related to problem-solving, which in turn have been shown to be associated with a reduction in diabetes distress (Glasgow et al. 2004).

The mounting evidence regarding the importance of diabetes-specific emotional distress on glycemic control lends itself to further investigation of interventions directed at reducing diabetes distress. In their recent observational study, Fisher et al. (2010) suggest that T2DM patients with "depression" may actually be experiencing major depressive disorder (MDD) and/or diabetes-specific emotional distress, and that it is the diabetes distress and not the MDD that is affecting glycemic control over time (Fisher et al. 2010). Our results from a DSME-based intervention support this insight, and highlight the need for greater awareness of the important role of diabetes distress in diabetes care. Standardized strategies for the effective treatment of diabetes distress, such as DSME, may be useful for the evaluation and treatment of diabetes distress in clinical settings.

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Conflict of interest The authors declare that they have no conflict of interest.

References

Aikens, J. E., Perkins, D. W., Lipton, B., & Piette, J. D. (2009). Longitudinal analysis of depressive symptoms and glycemic control in type 2 diabetes. *Diabetes Care, 32*, 1177–1181.

Ali, S., Stone, M. A., Peters, J. L., Davies, M. J., & Khunti, K. (2006). The prevalence of co-morbid depression in adults with type 2 diabetes: A systematic review and meta-analysis. *Diabetic Medicine: A Journal of the British Diabetic Association, 23*, 1165–1173.

Bradley, C. (1999). Diabetes treatment satisfaction questionnaire. Change version for use alongside status version provides appropriate solution where ceiling effects occur. *Diabetes Care, 22*, 530–532.

Cherrington, A., Wallston, K. A., & Rothman, R. L. (2010). Exploring the relationship between diabetes self-efficacy, depressive symptoms, and glycemic control among men and women with type 2 diabetes. *Journal of Behavioral Medicine, 33*, 81–89.

Cohen, S. T., Welch, G., Jacobson, A. M., De Groot, M., & Samson, J. (1997). The association of lifetime psychiatric illness and increased retinopathy in patients with type I diabetes mellitus. *Psychosomatics, 38*, 98–108.

de Groot, M., Jacobson, A. M., Samson, J. A., & Welch, G. (1999). Glycemic control and major depression in patients with type 1

and type 2 diabetes mellitus. *Journal of Psychosomatic Research, 46*, 425–435.

Eaton, W. W., Mengel, M., Mengel, L., Larson, D., Campbell, R., & Montague, R. B. (1992). Psychosocial and psychopathologic influences on management and control of insulin-dependent diabetes. *International Journal of Psychiatry in Medicine, 22*, 105–117.

Egede, L. E., & Ellis, C. (2010). Diabetes and depression: Global perspectives. *Diabetes Research and Clinical Practice, 87*, 302–312.

Fisher, L., Mullan, J. T., Arean, P., Glasgow, R. E., Hessler, D., & Masharani, U. (2010). Diabetes distress but not clinical depression or depressive symptoms is associated with glycemic control in both cross-sectional and longitudinal analyses. *Diabetes Care, 33*, 23–28.

Fisher, L., Skaff, M. M., Mullan, J. T., Arean, P., Glasgow, R., & Masharani, U. (2008). A longitudinal study of affective and anxiety disorders, depressive affect and diabetes distress in adults with type 2 diabetes. *Diabetic Medicine: A Journal of the British Diabetic Association, 25*, 1096–1101.

Georgiades, A., Zucker, N., Friedman, K. E., Mosunic, C. J., Applegate, K., Lane, J. D., et al. (2007). Changes in depressive symptoms and glycemic control in diabetes mellitus. *Psychosomatic Medicine, 69*, 235–241.

Glasgow, R. E., Toobert, D. J., Barrera, M., Jr, & Strycker, L. A. (2004). Assessment of problem-solving: A key to successful diabetes self-management. *Journal of Behavioral Medicine, 27*, 477–490.

Kokoszka, A., Pouwer, F., Jodko, A., Radzio, R., Mucko, P., Bienkowska, J., et al. (2009). Serious diabetes-specific emotional problems in patients with type 2 diabetes who have different levels of comorbid depression: A Polish study from the European depression in diabetes (EDID) research consortium. *European Psychiatry: The Journal of the Association of European Psychiatrists, 24*, 425–430.

Konen, J. C., Curtis, L. G., & Summerson, J. H. (1996). Symptoms and complications of adult diabetic patients in a family practice. *Archives of Family Medicine, 5*, 135–145.

Lin, E. H., Rutter, C. M., Katon, W., Heckbert, S. R., Ciechanowski, P., Oliver, M. M., et al. (2010). Depression and advanced complications of diabetes: A prospective cohort study. *Diabetes Care, 33*, 264–269.

Lustman, P. J., Anderson, R. J., Freedland, K. E., de Groot, M., Carney, R. M., & Clouse, R. E. (2000). Depression and poor glycemic control: A meta-analytic review of the literature. *Diabetes Care, 23*, 934–942.

Lustman, P. J., Griffith, L. S., Clouse, R. E., & Cryer, P. E. (1986). Psychiatric illness in diabetes mellitus. Relationship to symptoms and glucose control. *The Journal of Nervous and Mental Disease, 174*, 736–742.

Mazze, R. S., Lucido, D., & Shamoon, H. (1984). Psychological and social correlates of glycemic control. *Diabetes Care, 7*, 360–366.

Petrak, F., & Herpertz, S. (2009). Treatment of depression in diabetes: An update. *Current Opinion in Psychiatry, 22*, 211–217.

Polonsky, W. H., Anderson, B. J., Lohrer, P. A., Welch, G., Jacobson, A. M., Aponte, J. E., et al. (1995). Assessment of diabetes-related distress. *Diabetes Care, 18*, 754–760.

Pouwer, F. (2009). Should we screen for emotional distress in type 2 diabetes mellitus? *Nature Reviews Endocrinology, 5*, 665–671.

Pouwer, F., Skinner, T. C., Pibernik-Okanovic, M., Beekman, A. T., Cradock, S., Szabo, S., et al. (2005). Serious diabetes-specific emotional problems and depression in a Croatian-Dutch-English survey from the European depression in diabetes [EDID] research consortium. *Diabetes Research and Clinical Practice, 70*, 166–173.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385–401.

Solowiejczyk, J. (2010). Diabetes and depression: Some thoughts to think about. *Diabetes Spectrum, 23*, 11–15.

Thomas, J. L., Jones, G. N., Scarinci, I. C., Mehan, D. J., & Brantley, P. J. (2001). The utility of the CES-D as a depression screening measure among low-income women attending primary care clinics. The center for epidemiologic studies-depression. *International Journal of Psychiatry in Medicine, 31*, 25–40.

van Bastelaar, K. M., Pouwer, F., Geelhoed-Duijvestijn, P. H., Tack, C. J., Bazelmans, E., Beekman, A. T., et al. (2010). Diabetes-specific emotional distress mediates the association between depressive symptoms and glycaemic control in type 1 and type 2 diabetes. *Diabetic Medicine: A Journal of the British Diabetic Association, 27*, 798–803.

Van der Does, F. E., De Neeling, J. N., Snoek, F. J., Kostense, P. J., Grootenhuis, P. A., Bouter, L. M., et al. (1996). Symptoms and well-being in relation to glycemic control in type II diabetes. *Diabetes Care, 19*, 204–210.

van der Feltz-Cornelis, C. M., Nuyen, J., Stoop, C., Chan, J., Jacobson, A. M., Katon, W., et al. (2010). Effect of interventions for major depressive disorder and significant depressive symptoms in patients with diabetes mellitus: A systematic review and meta-analysis. *Annals of General Hospital Psychiatry, 32*, 380–395.

Van Tilburg, M. A., McCaskill, C. C., Lane, J. D., Edwards, C. L., Bethel, A., Feinglos, M. N., et al. (2001). Depressed mood is a factor in glycemic control in type 1 diabetes. *Psychosomatic Medicine, 63*, 551–555.

Wang, M. Y., Tsai, P. S., Chou, K. R., & Chen, C. M. (2008). A systematic review of the efficacy of non-pharmacological treatments for depression on glycaemic control in type 2 diabetics. *Journal of Clinical Nursing, 17*, 2524–2530.

Weinger, K., Butler, H. A., Welch, G. W., & La Greca, A. M. (2005). Measuring diabetes self-care: A psychometric analysis of the self-care inventory-revised with adults. *Diabetes Care, 28*, 1346–1352.

Welch, G. W., Jacobson, A. M., & Polonsky, W. H. (1997). The problem areas in diabetes scale. An evaluation of its clinical utility. *Diabetes Care, 20*, 760–766.

Welch, G., Weinger, K., Anderson, B., & Polonsky, W. H. (2003). Responsiveness of the problem areas in diabetes (PAID) questionnaire. *Diabetic Medicine: A Journal of the British Diabetic Association, 20*, 69–72.

Welch, G., Zagarins, S. E., Feinberg, R. G., & Garb, J. L. (2010). Motivational interviewing delivered by diabetes educators: Does it improve blood glucose control among poorly controlled type 2 diabetes patients? *Diabetes Research and Clinical Practice* (in press).