Published in final edited form as:
J Diabetes Complications. 2014 ; 28(6): 798–804. doi:10.1016/j.jdiacomp.2014.06.014.

Depression is not associated with diabetes control in minority elderly

Priya Palta[a,b], **Sherita Hill Golden**[a,c,d], **Jeanne A. Teresi**[e,f], **Walter Palmas**[g], **Paula Trief**[h,i], **Ruth S. Weinstock**[i], **Steven Shea**[g,j], **Jennifer J. Manly**[k,l], and **Jose A. Luchsinger**[g,j,*]

[a] Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA

[b] Department of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

[c] Welch Center for Prevention, Epidemiology, and Clinical Research, Johns Hopkins University, Baltimore, MD, USA

[d] Department of Medicine, Division of Endocrinology and Metabolism, Johns Hopkins University School of Medicine, Baltimore, MD, USA

[e] Research Division, Hebrew Home at Riverdale, Bronx, NY, USA

[f] Morris W. Stroud, III, Center for Studies on Quality of Life, and New York State Psychiatric Institute, Columbia University, New York, NY, USA

[g] Department of Medicine, Division of General Medicine, Columbia University School of Medicine, New York, NY, USA

[h] Department of Psychiatry and Behavioral Sciences, SUNY Upstate Medical University, Syracuse, NY, USA

[i] Department of Medicine, SUNY Upstate Medical University, Syracuse, NY, USA

[j] Department of Epidemiology, Columbia University Mailman School of Public Health, New York, NY, USA

[k] Department of Neurology and Taub Institute, Division of Cognitive Neuroscience, Columbia University College of Physician and Surgeons, NY, USA

[l] Gertrude H. Sergievksy Center, Columbia University Medical Center, New York, NY

Abstract

Aims—We investigated the longitudinal association of depression, with and without cognitive dysfunction, with hemoglobin A1c (HbA1c), systolic blood pressure (SBP), and low-density lipoprotein (LDL) in a predominantly minority cohort.

[*] Corresponding author at: Division of General Medicine, PH9 Center, room 210, 630 West 168th street, New York, NY 10032. Tel.: +1 212 305 4730; fax: +1 212 305 9349. jal94@columbia.edu (J.A. Luchsinger)..

Conflict of Interest: Dr. Weinstock has received research support for multisite diabetes clinical trials from Medtronic, Eli Lilly, Sanofi, Biodel, Novo Nordisk, Intarcia, Astra-Zeneca, Ultradian. Otherwise, none of the authors report conflicts of interest.

Methods—There were 613 participants. Presence of depression was defined by a score ≥ 7 on the Short-CARE depression scale. We tested participants for executive dysfunction using the Color Trails Test (CTT), part 2, and for memory dysfunction using the total recall task of the Selective Reminding Test (TR-SRT). We classified performance in these tests as abnormal based on standardized score cutoffs (<16th percentile and one standard deviation below the sample mean). Random effects models were used to compare repeated measures of the diabetes control measures between those with depression versus those without depression and ever versus never cognitively impaired.

Results—Baseline depression was present in 36% of participants. Over a median follow-up of 2 years, depression was not related to worse HbA1c, SBP, or LDL. The presence of (1) abnormal performance on a test of executive function and depression (n = 57) or (2) abnormal performance on a test of verbal recall and depression (n = 43) was also not associated with clinically significant worse change in diabetes control.

Conclusions—Depression, with or without low performance in tests of executive function and memory, may not affect clinically significant measures of diabetes control in the elderly.

Keywords

Diabetes; Depression; Diabetes control; Cognitive dysfunction; Older adults

1. Introduction

The prevalence of depression in communities of older adults with diabetes is approximately 33% (Anderson, Freedland, Clouse, & Lustman, 2001). Diabetes self-management is complex and time-intensive, requiring patients to be meticulous and motivated. Individuals with depression are overwhelmed by feelings of sadness, negativity, loss of interest in activities, and fatigue, all of which combined may result in ineffective disease self-management and medication non-adherence (Gonzalez et al., 2008). However, appropriate self-management of diabetes is important for the prevention of diabetes-related complications and other adverse outcomes (Haas et al., 2012). Poor self-management on the part of individuals with diabetes can lead to an increased incidence of related complications, such as, micro- and macro-vascular disease, and death (American Diabetes Association, 2013).

Depression has been found to be highly prevalent in persons with diabetes, but few longitudinal studies have examined the impact of depression on diabetes control (Trief et al., 2006). As people live longer with diabetes, depression has been shown to play a role in the adequacy of disease self-management and subsequent risk for diabetes-related complications. Moreover, depression is often accompanied by cognitive dysfunction (Richard et al., 2012), which may further affect the ability of a patient with diabetes to control their condition appropriately. Recent data from the ACCORD-MIND study showed that depression accelerated cognitive decline in type 2 diabetes (Sullivan et al., 2013). It is possible that cognitive decline accompanying depression could impact the ability of diabetes patients to adequately self-manage their disease. This problem may be more salient in

minority elders, who have a high prevalence of comorbid diabetes, cognitive dysfunction, and depression (Noble, Manly, Schupf, Tang, & Luchsinger, 2012).

We hypothesized that depression, with and without cognitive dysfunction, would be associated with worse control in the diabetes parameters usually followed by clinicians: glycemia, lipids, and blood pressure. We analyzed longitudinal data from a sample of minority elders with detailed longitudinal data on depression, cognitive performance, and parameters of diabetes control.

2. Subjects

The Informatics for Diabetes Education and Telemedicine (IDEA-Tel) project was initially designed to assess the feasibility and effectiveness of home-based telemedicine for management of diabetes in a sample of older adults residing in the state of New York (Shea et al., 2002). Participants were Medicare beneficiaries and resided in a federally designated medically underserved area. The exclusion criterion included the following: moderately or severely cognitively impaired; a severe visual, mobility or motor impairment; a severe comorbid condition; communication impairment; no electrical outlet for the telemedicine unit; or planned to reside in another location for more than 3 months. Adults >55 years of age (n =1,655), with type 2 diabetes, were randomized to receive the intervention (a home-based interactive telemedicine unit used for televisits with a diabetes nurse educator, transmission of self-measured blood glucose and blood pressure data and access to the web in addition to usual care) or usual care alone. Changes in hemoglobin A1c (HbA1c), blood pressure and LDL cholesterol levels were the primary endpoints of IDEATel. Details of the study (inclusion/exclusion, randomization scheme, intervention and evaluation of primary study outcomes) have been previously described (Shea, 2007; Shea et al., 2002, 2009).

IDEATel had 2 study sites in New York State, Upstate, and Downstate (New York City). An ancillary cognition study was started in 2005 at the New York City (Columbia University) IDEATel site and was the source for the study sample reported in these analyses. IDEATel was carried out from 2000 to 2008 (Phase 1: 2000–2004, Phase 2: 2004–2008); 600 participants in New York City (Columbia University site) were recruited in phase 1, and 150 participated in phase 2. These 750 participants were all randomized to the telemedicine intervention or usual care. We recruited 613 of the 750 participants from phases 1 (n = 476) and 2 (n = 137) at the New York City site, for this cognition ancillary study between 2004 and 2008 and participants were followed yearly until 2012. In addition to the baseline visit, 538 (87.7%) participants had one follow-up visit, 437 (71.3%) had 2 follow-up visits, 350 (57.1%) had 3 follow-up visits, 231 (37.4%) had 4 follow-up visits, and 90 (14.7%) had 5 follow-up visits. Measures of memory and executive function were administered in this ancillary study in addition to the assessment of depression. The sole exclusion criterion for the ancillary study was non-willingness or inability to begin or complete the cognitive assessments. Columbia University's Institutional Review Board approved all protocols for this study. The baseline for the current analyses was the time of recruitment into the cognition ancillary study.

3. Materials and methods

3.1. Assessment of depression

Presence of depression was measured using the SHORT-CARE Depression questionnaire (Gurland, Golden, Teresi, & Challop, 1984), a shortened version of the longer CARE depression scale (Gurland et al., 1977). The CARE questionnaire is based on the Geriatric Mental State Schedule (GMS) Depression scale (Copeland et al., 1976), but is shorter, easily administered by non-clinical personnel, and has been widely administered in ethnically diverse populations. The internal consistency reliability of the CARE depression measure in the development sample was 0.87; in the IDEATel sample the estimates ranged from 0.86 to 0.89 across administrations. The interrater reliability estimate was 0.94 (Teresi, Golden, & Gurland, 1984). The measure evidenced high concurrent validity (0.75) with a clinical diagnosis of depression (Gurland et al., 1988). Depression was assessed in two ways. For the primary analysis, participants with a CARE score ≥ 7 at baseline were categorized as having depression; otherwise, they were categorized as not depressed. In a secondary analysis using all available follow-up data, participants with a CARE score ≥ 7 at any one visit were categorized as ever having depression; otherwise, they were categorized as never having depression. There is literature to suggest that a cutoff of 7 on the CARE questionnaire evidenced a high sensitivity and specificity for clinical depression in an elderly population (Mann, Graham, & Ashby, 1984).

3.2. Comorbid depression and low performance in cognitive tests

A secondary analysis was conducted examining the association of comorbid depression and low performance in cognitive tests, with diabetes control. Executive cognitive function can be broadly defined as the ability to plan, initiate and complete the execution of complex tasks (Royall et al., 2002) (e.g. planning and completing diabetes treatment). Memory can be defined as the ability to recall in general (e.g. remembering to take diabetes medications) (Small & Mayeux, 1999). Executive function was examined using the Color Trails Test (CTT), part 2 (D'Elia, Satz, Uchiyama, & White, 1996), and memory was assessed using the total recall (TR) task of the Selective Reminding Test (SRT) (Buschke & Fuld, 1974). In the CTT, part 2, the participant connects consecutively numbered dots that alternate between the colors pink and yellow. The main measure in this test is the time necessary to complete the task. In the SRT, the participant is asked to recall as many words as possible from a list of 12 words in 6 trials. The total recall is the number of words remembered across the 6 trials. Low performance on the CTT and the TR-SRT were defined as a standardized score, <16th percentile or one standard deviation (SD) below the sample mean at baseline. This cutoff is typically used in clinical settings to categorize patients as “abnormal” on cognitive functioning (Binder, Iverson, & Brooks, 2009; Duara et al., 2011; Heaton, Grant, & Matthews, 1991; Heaton, Miller, Taylor, & Grant, 2004) as compared to the 1.5 SD cutoff used to classify mild cognitive impairment (MCI) (Petersen, 2004).

3.3. Outcomes

The primary outcomes were changes in measures of diabetes control, including, hemoglobin A1c (HbA1c), systolic blood pressure (SBP), and low-density lipoprotein (LDL) cholesterol. Both HbA1c and lipids were collected using 12-hr fasting blood samples. HbA1c was

analyzed by boronate affinity chromatography with Primus CLC 385 (Primus, Kansas City, MO). Lipid levels were analyzed using enzymatic colorimetric methods (Vitros; Johnson & Johnson, New Brunswick, NJ). The Friedewald equation was used to calculate LDL cholesterol (Friedewald, Levy, & Fredrickson, 1972). SBP was measured by averaging the second and third of three readings, taken 1 minute apart, using the Dinemap PRO 100 automated device (Perloff et al., 1993).

3.4. Statistical analysis

Chi-square tests for categorical variables and the Kruskal–Wallis Test for continuous variables were used to test for significant differences in participant characteristics and diabetes control measures between participants with and without baseline depression.

Random effects models (Diggle, Heagerty, Liang, & Zeger, 2002), incorporating random effects for intercepts (i.e., individuals) and clustering within primary care provider (PCP), were used to examine the longitudinal relationship between baseline depression and changes in HbA1c, SBP, and LDL cholesterol. An interaction term in the random effects model was incorporated between depression and time to estimate the effect of depression on rates of change in measures of diabetes control across follow-up. Assessments of nonlinearity were performed by inclusion of quadratic (group × $time^2$) and exponential terms (group × e^{-time}) for time and evaluating goodness of fit statistics (i.e., Akaike Information Criterion (AIC) (Akaike, 1974) and Schwarz's Bayesian Information Criterion (BIC) (Schwarz, 1978)). Model fit did not improve significantly to warrant the use of these non-linear terms for time in the final models. In addition to age and education, demographic characteristics that were significantly different between exposure groups at baseline (sex and race/ethnicity) were adjusted for in the analyses. IDEATel randomization group assignment was also included in the final model to account for differences in the group due to the intervention. To account for possible confounding by diabetes severity, insulin, metformin, sulfonylurea or thiazolidinedione medication use was further adjusted in models of HbA1c outcomes. We also conducted sensitivity analyses examining (1) longitudinal assessment of depression (ever/never depressed across follow-up), (2) examining the outcomes in non-linear models, and (3) redefining cognitive dysfunction by a threshold of 1.5 SD rather than 1.0 SD.

A subsidiary analysis was performed to examine the association of comorbid depression and low performance in cognitive tests with changes in measures of diabetes control, compared to neither low performance nor depression. All analyses were performed using STATA 13.0 (Stata Corp, College Station, TX).

4. Results

Among the 613 participants included in this analysis, the overall mean age of participants was 73 years, and 70% were female. Participants had an average of 7.5 years of formal education. Most participants were either Hispanic (82.5%) or Black (15.5%), with <1.0% of participants reporting White (non-Hispanic) race.

Amongst the analytic sample, 218 (36%) were classified with depression at baseline (Table 1). Those with depression were more frequently women ($p < 0.001$) and Hispanic ($p =$

0.018). Mean (standard error) values of the diabetes control measures, accounting for clustering within PCP, are presented across each study visit and by baseline depression status in Table 2. No differences in baseline measures or rates of change in diabetes control measures were observed between participants with and without depression (Table 3). The inferences were unchanged after adjusting for low performance on the CTT and TR-SRT. In the subsidiary analysis, 9% (n = 57) of the total sample was classified as having both low performance in the CTT and depression at baseline (Table 4). Seven percent (n = 43) were classified as having both low performance in the TR-SRT and depression at baseline (Table 5). No baseline differences were observed for HbA1c (Tables 4 and 5). Differences at baseline were observed in systolic blood pressure when comparing participants with executive dysfunction to those with neither depression nor executive dysfunction ($\beta = 5.1$, 95% confidence interval: 0.3, 9.9). Differences at baseline were also observed in LDL cholesterol when comparing participants with only depression to those with neither depression nor memory dysfunction ($\beta = -0.2$, 95% confidence interval: −0.3, −0.01); however, these differences are likely not clinically significant. No differences in rates of change were observed for any of the diabetes control measures (Tables 4 and 5). In the secondary analyses, using a longitudinal assessment of depression (ever/never depressed across follow-up), the overall inference for the significance of the associations were unchanged (Tables 3, 4, and 5).

We conducted sensitivity analyses examining non-linear models and using different threshold levels for defining low performance in the CTT and TR-SRT, and the results were unchanged. We also examined effect modification by IDEATel randomization arm and time of study recruitment (phase 1 or 2) and found no evidence of effect modification.

5. Discussion

In this sample of older minority adults with type 2 diabetes, we found that the presence of depression was not independently associated with changes in the usual measures of diabetes control, glycemia, lipids, and blood pressure (American Diabetes Association, 2013). Depression with low performance in tests of executive function and memory was also not associated with changes in diabetes control compared to individuals with neither low cognitive performance nor depression.

The link between depression and poor glycemic control has been previously studied with some limitations. In a meta-analysis of 24 studies, researchers found that depression was significantly associated with hyperglycemia, a common indicator of diabetes control (Lustman et al., 2002). Cross-sectional studies have shown that depressive symptoms were significantly associated with poorer levels of total cholesterol, HbA1c, diastolic blood pressure and LDL after adjustment (Gary, Crum, Cooper-Patrick, Ford, & Brancati, 2000). However, a follow-up to this cross-sectional analysis with longitudinal data found no statistically significant associations between baseline depressive symptoms or change in depressive symptoms and diabetes control over a three-year period (Gary et al., 2005). A cross-sectional study of older adult African Americans also showed no association between depression and diabetes control (Nguyen et al., 2002). Our findings confirm those from another prospective study that showed no association between baseline depressive

symptoms or change in depressive symptoms and diabetes control over a three-year period (Gary et al., 2005). Compared to that study, most studies to date on depression and diabetes control have been cross-sectional and limited by small sample sizes (Gary et al., 2000; Lustman et al., 2002).

A previous analysis performed in the parent IDEATel cohort evaluated the effect of depressive symptoms on glycemic control and found no association cross-sectionally or longitudinally (Trief et al., 2006). Compared to this study, our study focused on the New York City sub-cohort that was predominantly composed of ethnic minorities and had cognitive data not available for the previous analysis. In addition, our analysis also focused on lipids and blood pressure as measures of diabetes control as compared to focusing solely on glycemia.

Several issues should be considered in the interpretation of the results. First, the parent IDEATel study was a randomized controlled trial (RCT), and the potential for selection bias in RCTs is high with the healthiest individuals most likely to enroll. Persons with moderate to severe cognitive impairment were excluded in the first phase of IDEATel, but they were not excluded from our recruitment during the second phase, when they may have developed cognitive impairment. These factors may limit the generalizability of our findings and the power to find a relationship between depression and diabetes control. Second, we tried to capture clinical depression using the CARE depression questionnaire. However, we did not have a clinical definition of depression nor information on the use of anti-depressant medication, and misclassification of depression could have led to our null findings. Third, we did not have a measure of diabetes process of care such as adherence, which have been reported to be affected by depression (Gonzalez et al., 2008). However, we believe that the examination of depression in relation to the most important clinical measures of diabetes management provides clinically significant information that most diabetes practitioners can relate to. Finally, this study population had a relatively well-controlled HbA1c at baseline and this may impact the ability to detect significant longitudinal changes in parameters of metabolic control relative to cognitive dysfunction and depressive symptoms.

In conclusion, this study showed that depression using the CARE questionnaire, which correlates with clinical depression, was not independently associated with poorer diabetes control in elderly patients with type 2 diabetes. Depression accompanied by low performance in cognitive tests also did not result in clinically significant poorer diabetes control compared to individuals with normal cognitive performance and no depression. The main clinical implication from our findings is that the presence of depression, found commonly in persons with diabetes, does not necessarily impact their diabetes control. However, depression is an important determinant of quality of life, and it should be screened for and treated appropriately among persons with diabetes. More studies are needed examining whether depression affects diabetes control in clinical cohorts.

Acknowledgments

This study was supported by NIMHD grant P60 MD 000206, Alzheimer's Association grant IIRG-05-15053, the Fidelity Foundation, and Cooperative Agreement 95-C-90998 from the Centers for Medicare and Medicaid Services, ClinicalTrials.gov Identifier: NCT00271739; National Institutes of Health/National Institute of Diabetes

and Digestive and Kidney Diseases T32 Training Grant in Clinical Research and Epidemiology in Diabetes and Endocrinology (T32 DK062707) awarded to P.P., National Heart, Lung, and Blood Institute Training Grant in Cardiovascular Epidemiology, Biostatistics and Preventive Medicine (T32 HL007055) awarded to P.P.

References

Akaike H. A new look at the statistical model identification. IEEE Transactions on Automatic Control. 1974; 19:716–723.

American Diabetes Association. Standards of medical care in diabetes–2013. Diabetes Care. 2013; 36:S11–S66. [PubMed: 23264422]

Anderson RJ, Freedland KE, Clouse RE, Lustman PJ. The prevalence of comorbid depression in adults with diabetes: a meta-analysis. Diabetes Care. 2001; 24:1069–1078. [PubMed: 11375373]

Binder LM, Iverson GL, Brooks BL. To err is human: “abnormal” neuropsychological scores and variability are common in healthy adults. Archives of Clinical Neuropsychology. 2009; 24:31–46. [PubMed: 19395355]

Buschke H, Fuld PA. Evaluating storage, retention, and retrieval in disordered memory and learning. Neurology. 1974; 24:1019–1025. [PubMed: 4473151]

Copeland JR, Kelleher MJ, Kellett JM, Gourlay AJ, Gurland BJ, Fleiss JL, et al. A semi- structured clinical interview for the assessment of diagnosis and mental state in the elderly: the Geriatric Mental State Schedule. I. Development and reliability. Psychological Medicine. 1976; 6:439–449. [PubMed: 996204]

D'Elia, LF.; Satz, P.; Uchiyama, CL.; White, T. Color Trails Test. Psychological Assessment Resources; Odessa, FL: 1996.

Diggle, PJ.; Heagerty, P.; Liang, K.; Zeger, SL. Analysis of longitudinal data. Oxford University Press; New York: 2002.

Duara R, Loewenstein DA, Greig MT, Potter E, Barker W, Raj A, et al. Pre-MCI and MCI: neuropsychological, clinical, and imaging features and progression rates. The American Journal of Geriatric Psychiatry. 2011; 19:951–960. [PubMed: 21422909]

Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low- density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clinical Chemistry. 1972; 18:499–502. [PubMed: 4337382]

Gary TL, Baptiste-Roberts K, Crum RM, Cooper LA, Ford DE, Brancati FL. Changes in depressive symptoms and metabolic control over 3 years among African Americans with type 2 diabetes. International journal of psychiatry in medicine. 2005; 35:377–382. [PubMed: 16673837]

Gary TL, Crum RM, Cooper-Patrick L, Ford D, Brancati FL. Depressive symptoms and metabolic control in African-Americans with type 2 diabetes. Diabetes Care. 2000; 23:23–29. [PubMed: 10857963]

Gonzalez JS, Peyrot M, McCarl LA, Collins EM, Serpa L, Mimiaga, et al. Depression and diabetes treatment nonadherence: a meta-analysis. Diabetes Care. 2008; 31:2398–2403. [PubMed: 19033420]

Gurland B, Golden RR, Teresi JA, Challop J. The SHORT-CARE: an efficient instrument for the assessment of depression, dementia and disability. Journal of Gerontology. 1984; 39:166–169. [PubMed: 6699370]

Gurland B, Kuriansky J, Sharpe L, Simon R, Stiller P, Birkett P. The Comprehensive assessment and Referral Evaluation (CARE)–rationale, development and reliability. International Journal of Aging and Human Development. 1977; 8:9–42. [PubMed: 873639]

Gurland BJ, Teresi J, Smith WM, Black D, Hughes G, Edlavitch S. Effects of treatment for isolated systolic hypertension on cognitive status and depression in the elderly. Journal of American Geriatrics Society. 1988; 36:1015–1022.

Haas L, Maryniuk M, Beck J, Cox CE, Duker P, Edwards L, et al. National standards for diabetes self-management education and support. Diabetes Care. 2012; 35:2393–2401. [PubMed: 22995096]

Heaton, RK.; Grant, I.; Matthews, CG. Comprehensive norms for an extended Halstead– Reitan Battery: demographic corrections, research findings, and clinical applications. Psychological Assessment Resources; Odessa, FL: 1991.

Heaton, RK.; Miller, SW.; Taylor, MJ.; Grant, I. Revised comprehensive norms for an expanded Halstead–Reitan Battery: demographically adjusted neuropsychological norms for African American and Caucasian adults professional manual. Psychological Assessment Resources; Lutz, FL: 2004.

Lustman PJ, Anderson RJ, Freedland KE, de Groot M, Carney RM, Clouse RE. Depression and poor glycemic control: a meta-analytic review of the literature. Diabetes Care. 2002; 23:934–942. [PubMed: 10895843]

Mann AH, Graham N, Ashby D. Psychiatric illness in residential homes for the elderly: a survey in one London borough. Age and Ageing. 1984; 13:257–265. [PubMed: 6496236]

Nguyen HT, Arcury TA, Grzywacz JG, Saldana SJ, Ip EH, Kirk JK, et al. The association of mental conditions with blood glucose levels in older adults with diabetes. Aging & Mental Health. 2002; 16:950–957. [PubMed: 22640032]

Noble JM, Manly JJ, Schupf N, Tang MX, Luchsinger JA. Type 2 diabetes and ethnic disparities in cognitive impairment. Ethnicity and Disease. 2012; 22:38–44. [PubMed: 22774307]

Perloff D, Grim C, Flack J, Frohlich ED, Hill M, McDonald M, et al. Human blood pressure determination by sphygmomanometry. Circulation. 1993; 88:2460–2470. [PubMed: 8222141]

Petersen RC. Mild cognitive impairment as a diagnostic entity. Journal of Internal Medicine. 2004; 256:183–194. [PubMed: 15324362]

Richard E, Reitz C, Honig LH, Schupf N, Tang MX, Manly JJ, et al. Late-life depression, mild cognitive impairment, and dementia. JAMA: the journal of the American Medical Association. 2012; 70(3):374–382.

Royall DR, Lauterbach EC, Cummings JL, Reeve A, Rummans TA, Kaufer DI, et al. Executive control function: a review of its promise and challenges for clinical research. A report from the Committee on Research of the American Neuropsychiatric Association. Journal of Neuropsychiatry and Clinical Neurosciences. 2002; 14:377–405. [PubMed: 12426407]

Schwarz G. Estimating the dimension of a model. Annual Statistic. 1978; 6:461–464.

Shea S. The Informatics for Diabetes and Education Telemedicine (IDEATel) project. Transactions of the American Clinical and Climatological Association. 2007; 118:289–304. [PubMed: 18528511]

Shea S, Starren J, Weinstock RS, Knudson PE, Teresi J, Holmes D, et al. Columbia University's Informatics for Diabetes Education and Telemedicine (IDEATel) Project: rationale and design. Journal of the American Medical Informatics Association. 2002; 9:49–62. [PubMed: 11751803]

Shea S, Weinstock RS, Teresi JA, Palmas W, Starren J, Cimino JJ, et al. A randomized trial comparing telemedicine case management with usual care in older, ethnically diverse, medically underserved patients with diabetes mellitus: 5 year results of the IDEATel study. Journal of the American Medical Informatics Association. 2009; 16:446–456. [PubMed: 19390093]

Small SA, Mayeux R. A clinical approach to memory decline. Journal of practical psychiatry and behavioral health. 1999; 5:87–94.

Sullivan MD, Katon WJ, Lovato LC, Miller ME, Murray AM, Horowitz KR, et al. Association of depression with accelerated cognitive decline among patients with type 2 diabetes in the ACCORD-MIND trial. JAMA Psychiatry. 2013; 70(10):1041–1047. [PubMed: 23945905]

Teresi JA, Golden RR, Gurland BJ. Concurrent and predictive validity of indicator scales developed for the comprehensive assessment and referral evaluation interview schedule. Journal of Gerontology. 1984; 39:158–165. [PubMed: 6699369]

Trief PM, Morin PC, Izquierdo R, Teresi J, Eimicke JP, Goland R, et al. Depression and glycemic control in elderly ethnically diverse patients with diabetes: the IDEATel project. Diabetes Care. 2006; 29:830–835. [PubMed: 16567823]

Table 1

Characteristics of study population (n = 613), by baseline depression status, at the baseline of IDEATel cognition ancillary study.

Characteristic	Total sample N = 613	No depression CARE-Dep score < 7 n = 346	Depression CARE-Dep score ≥ 7 n = 218	*p* value
Age, years, mean (SD)	73.0 (6.5)	73.0 (6.6)	72.5 (6.3)	0.282
Female sex, n (%)	426 (69.5)	215 (62.1)	176 (80.7)	<0.001
Education, years, mean (SD)	7.5 (4.1)	7.4 (4.1)	7.3 (4.1)	0.691
Race/Ethnicity, n(%)				0.018
Hispanic	506 (82.5)	286 (82.7)	192 (88.1)	
African-American (non-Hispanic)	95 (15.5)	57 (16.5)	21 (9.6)	
White (non-Hispanic)	4 (0.007)	0 (0)	3 (0.01)	
Insulin medication use, n (%)	164 (26.8)	99 (28.6)	65 (29.8)	0.759
Metformin medication use, n (%)	291 (47.5)	183 (52.9)	108 (49.5)	0.438
Sulfonylurea medication use, n (%)	228 (37.2)	152 (43.9)	76 (34.9)	0.033
Thiazolidinedione, n (%)	153 (25.0)	94 (27.2)	59 (27.1)	0.979
IDEATel intervention group	309 (50.4)	183 (52.9)	106 (48.6)	0.324
Hemoglobin A1c, % (mmol/mol), mean	7.4(57)	7.5 (58)	7.4(57)	0.460
Systolic blood pressure, mmHg, mean (SD)	140.2 (21.1)	140.4 (20.7)	139.9 (21.8)	0.614
Diastolic blood pressure, mmHg, mean (SD)	69.6 (10.9)	69.9 (10.7)	69.2 (11.1)	0.353
Total cholesterol, mmol/l, mean (SD)	4.4 (1.1)	4.4 (1.0)	4.4 (1.1)	0.916
HDL cholesterol, mmol/l, mean (SD)	1.3 (0.4)	1.2 (0.4)	1.3 (0.4)	0.140
LDL cholesterol, mmol/l, mean (SD)	2.5 (1.0)	2.5 (0.9)	2.5 (1.0)	0.650

Table 2

Adjusted mean (standard error) values of diabetes control measures, by baseline depression status.

	Hemoglobin A1c, % (mmol/mol)				Systolic blood pressure, mmHg				LDL cholesterol, mmol/l			
	No depression		Depression		No depression		Depression		No depression		Depression	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Visit 1	7.5 (58)	0.1	7.3 (56)	0.1	140.4	1.1	139.9	1.4	2.5	0.1	2.5	0.1
Visit 2	7.4 (57)	0.1	7.4 (57)	0.1	139.1	1.4	139.1	1.7	2.5	0.1	2.5	0.1
Visit 3	7.3 (56)	0.1	7.4 (57)	0.2	140.9	2.3	138.2	2.8	2.4	0.1	2.3	0.1
Visit 4	7.9 (63)	0.2	8.1 (65)	0.3	144.2	2.2	142.9	3.0	2.3	0.1	2.2	0.2
Visit 5	8.0 (64)	0.1	7.8 (62)	0.2	139.5	1.7	140.8	2.2	2.2	0.1	2.3	0.1
Visit 6	7.3 (56)	0.2	7.3 (56)	0.2	135.5	2.1	139.0	2.6	2.2	0.1	2.2	0.1

SE = standard error; estimates account for clustering by primary care provider.

Table 3

Random effects models for the associations between depression status and differences in diabetes control measures at baseline and rates of change. Depression is defined both at baseline only (top rows) and as ever having depression.

Metabolic measure	Adjusted baseline values of diabetes control measures[a,b]				Annual rate of change (slopes) in diabetes control measures over 5-year follow-up[a,c]			
	No depression	Depression	Difference	*p* value	No depression	Depression	Difference	*p* value
Analyses with depression defined at baseline only								
Hemoglobin A1c (HbA1c) (% (mmol/mol))[d]	8.2 (66) (6.2, 10.2)	8.2 (66) (6.2, 10.1)	–0.05 (–0.3, 0.2)	0.661	0.08 (0.04, 0.1)	0.08 (0.03, 0.1)	–0.004 (–0.06, 0.06)	0.909
Systolic blood pressure (mmHg)[e]	132.9 (105.5, 160.3)	132.2 (104.9, 159.4)	–0.7 (–3.9, 2.5)	0.671	–0.1 (–0.7, 0.5)	0.01 (– 0.7, 0.8)	0.2 (–0.8, 1.1)	0.750
LDL cholesterol (mmol/l)[e]	4.3 (3.1, 5.6)	4.3 (3.0, 5.5)	–0.07 (–0.2, 0.1)	0.320	–0.05 (–0.1, –0.02)	–0.04 (–0.1, 0.0005)	0.02 (–0.03, 0.06)	0.492
Analyses with depression defined as ever vs. never during follow-up								
Hemoglobin A1c (HbA1c) (% (mmol/mol))[d]	8.1 (65) (6.3, 9.9)	8.1 (65) (6.3, 9.9)	–0.05 (–0.3, 0.2)	0.670	0.09 (0.04, 0.1)	0.07 (0.03, 0.1)	–0.02 (–0.08, 0.04)	0.545
Systolic blood pressure (mmHg)[e]	122.8 (97.3, 148.3)	122.8 (97.5, 148.0)	–0.06 (–3.2, 3.0)	0.967	–0.2 (–0.8, 0.5)	–0.2 (–0.8, 0.5)	–0.002 (–0.9, 0.9)	0.996
LDL cholesterol (mmol/l)[e]	4.4 (3.2, 5.6)	4.3 (3.2, 5.5)	–0.07 (–0.2, 0.07)	0.318	–0.06 (–0.09, –0.03)	–0.03 (–0.06, –0.003)	0.02 (–0.02, 0.07)	0.250

Data are estimates (95% confidence interval); SDS = significant depressive symptoms.

[a] Model includes random intercepts and clustering by primary care provider.

[b] Depression group variable in model.

[c] Interaction term of depression group by time.

[d] Models are adjusted for age, race, sex, education, insulin medication use, metformin medication use, sulfonylurea medication use, thiazolidinedione use, and IDEATel treatmentgroup.

[e] Models are adjusted for age, race, sex, education, and IDEATel treatment group.

Table 4

Random effects models for the associations between baseline depression and executive dysfunction and differences in baseline measures and rates of change in diabetes control measures.

Diabetes control measure	Adjusted baseline values of diabetes control measures[a]				Annual rate of change (slopes) in diabetes control measures over 5-year follow-up[a]			
	No depression - No executive Dysfunction[d]	Depression - No executive Dysfunction	No depression - Executive Dysfunction	Depression - Executive Dysfunction	No depression - No executive Dysfunction[d]	Depression - No executive Dysfunction	No depression - Executive Dysfunction	Depression - Executive dysfunction
Analyses with depression defined at baseline only								
HbA1c (% (mmol/mol))[b]	8.7 (72) (6.0, 11.5)	8.8 (73) (6.0, 11.5)	8.9 (74) (6.1, 11.6)	8.5 (69) (5.8, 11.2)	0.1 (0.05, 0.2)	0.06 (–0.02, 0.2)	0.03 (–0.04, 0.09)	0.1 (0.01, 0.2)
Systolic blood pressure (mmHg)[c]	144.4 (107.7, 181.1)	142.3 (105.8, 178.7)	149.5[e] (112.8, 186.2)	144.1 (107.8, 180.4)	–0.07 (–1.0, 0.9)	0.6 (–0.8, 1.9)	–0.7 (–1.7, 0.3)	0.4 (–1.0, 1.8)
LDL cholesterol (mmol/l)[c]	4.2 (2.4, 6.0)	4.1 (2.3, 5.8)	4.2 (2.5, 6.0)	4.1 (2.3, 5.8)	–0.05 (–0.09, –0.003)	–0.02 (–0.08, 0.04)	–0.07 (–0.1, –0.02)	–0.03 (–0.1, 0.03)
Analyses with depression defined as ever vs. never during follow-up								
HbA1c (% (mmol/mol))[b]	8.1 (65) (6.2, 9.9)	8.2 (66) (6.3, 10.0)	8.1 (65) (6.3, 9.9)	8.1 (65) (6.3, 9.9)	0.1 (0.03, 0.2)	0.08 (0.02, 0.1)	0.08 (0.02, 0.1)	0.07 (0.02, 0.1)
Systolic blood pressure (mmHg)[c]	120.1 (94.5, 145.7)	123.5 (98.0, 148.9)	122.0 (96.7, 147.4)	121.7 (96.4, 146.9)	–0.4 (–1.5, 0.7)	–0.09 (–1.0, 0.8)	–0.2 (–1.2, 0.8)	–0.2 (–1.0, 0.6)
LDL cholesterol (mmol/l)[c]	4.4 (3.2, 5.5)	4.4 (3.3, 5.6)	4.4 (3.2, 5.5)	4.3 (3.1, 5.4)	–0.05 (–0.1, –0.001)	–0.06 (–0.1, –0.02)	–0.04 (–0.1, 0.01)	–0.03 (–0.1, 0.01)

Data are estimates (95% confidence interval).

Depression is defined both at baseline only (top rows) and as ever having depression.

[a] Model includes random intercepts and clustering by primary care provider.

[b] Models are adjusted for age, race, sex, education, insulin medication use, metformin medication use, sulfonylurea medication use, thiazolidinedione use, and IDEATel treatment group.

[c] Models are adjusted for age, race, sex, education, and IDEATel treatment group.

[d] Reference group.

[e] $p < 0.05$ indicates a significant difference from the reference group (no depression/no executive dysfunction).

Table 5

Random effects models for the associations between baseline depression and executive dysfunction and differences in baseline measures and rates of change in diabetes control measures.

Diabetes control measure	Adjusted baseline values of diabetes control measures[a]				Annual rate of change (slopes) in diabetes control measures over 5-year follow-up[a]			
	No depression - No memory dysfunction[d]	Depression - No memory dysfunction	No depression - Memory dysfunction	Depression - Memory dysfunction	No depression - No memory dysfunction[d]	Depression - No memory dysfunction	No depression - Memory dysfunction	Depression - Memory dysfunction
Analyses with depression defined at baseline only								
HbA1c (% (mmol/mol))[b]	8.2 (66) (6.2, 10.1)	8.1 (65) (6.2, 10.1)	8.3 (67) (6.3, 10.2)	8.1 (65) (6.1, 10.1)	0.09 (0.04, 0.1)	0.09 (0.04, 0.1)	0.04 (–0.08, 0.2)	0.01 (–0.1, 0.1)
Systolic blood pressure (mmHg)[c]	133.0 (105.4, 160.5)	132.3 (105.0, 159.7)	132.8 (105.2, 160.4)	132.1 (104.3, 159.8)	–0.1 (–0.8, 0.5)	0.1 (–0.7, 1.0)	–0.02 (–1.9, 1.9)	–0.6 (–2.5, 1.2)
LDL cholesterol (mmol/l)[c]	4.4 (3.1, 5.6)	4.2[e] (3.0, 5.5)	4.2 (3.0, 5.5)	4.6 (3.3, 5.8)	–0.06 (–0.09, –0.03)	–0.03 (–0.1, 0.01)	–0.01 (–0.1, –0.08)	–0.04 (–0.1, 0.05)
Analyses with depression defined as ever vs. never during follow-up								
HbA1c (% (mmol/mol))[b]	7.9 (63) (6.1, 9.7)	8.2 (66) (6.4, 10.0)	7.9 (63) (6.1, 9.7)	8.0 (64) (6.2, 9.8)	0.08 (0.03, 0.1)	0.1 (0.02, 0.2)	0.1 (0.05, 0.2)	0.02 (–0.05, 0.09)
Systolic blood pressure (mmHg)[c]	121.6 (95.9, 147.3)	125.2 (99.6, 151.0)	122.0 (96.7, 147.3)	123.7 (98.0, 149.3)	–0.3 (–0.5, 1.1)	–1.3[e] (–2.5, 0.05)	–0.1 (–0.7, 0.9)	–0.6 (–1.6, 0.4)
LDL cholesterol (mmol/l)[c]	4.4 (3.2, 5.6)	4.4 (3.2, 5.6)	4.3 (3.2, 5.5)	4.4 (3.2, 5.6)	–0.06 (–0.1, –0.03)	–0.04 (–0.1, 0.02)	–0.02 (–0.06, 0.01)	–0.05 (–0.1, –0.001)

Data are estimates (95% confidence interval).

Depression is defined both at baseline only (top rows) and as ever having depression.

[a] Model includes random intercepts and clustering by primary care provider.

[b] Models are adjusted for age, race, sex, education, insulin medication use, metformin medication use, sulfonylurea medication use, thiazolidinedione use, and IDEATel treatment group.

[c] Models are adjusted for age, race, sex, education, and IDEATel treatment group.

[d] Reference group.

[e] $p < 0.05$ indicates a significant difference from the reference group (no depression/no memory dysfunction).