



# Cognitive function in adults with type 2 diabetes and major depression

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#### **Abstract**

The aim of this study was to identify characteristics of neuropsychological functioning among type 2 diabetic adults with and without major depression. Twenty type 2 diabetics with major depression, 20 non-depressed type 2 diabetics and 34 controls without diabetes or depression were compared. A mixed effects repeated measures analysis of covariance indicated significant differences in overall cognitive functioning between diagnostic groups, specifically depressed diabetics demonstrated greater cognitive dysfunction than controls. Further comparisons indicated that depressed diabetics performed significantly worse than non-depressed diabetics in attention/information processing speed. Relative to controls, depressed diabetics performed significantly worse in attention/information processing speed and executive functioning, while there was a trend for non-depressed diabetics to perform worse in executive functioning. These findings suggest that depression negatively impacts cognitive performance among adults with type 2 diabetes, which may have implications for neural circuitry underlying cognitive and mood changes in diabetic patients.

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The prevalence of depressive symptoms is higher in adults with diabetes relative to the general population, ranging from 8.5 to 31%, with 11% meeting criteria for major depression (Anderson, Clouse, Freedland, & Lustman, 2001; Gavard, Lustman, & Clouse, 1993). The presence of depression and diabetes tends to worsen the course of both, particularly through diabetic complications, worsened glucose control, and a greater number of depressive relapses (Blazer, Moody-Ayers, Craft-Morgan, & Bunchett, 2002; Goodnick, 1997). Additionally, depression may increase vulnerability to and/or exacerbate existing cognitive deficits. While the precise neurobiological mechanisms underlying depression and cognitive abnormalities in type 2 diabetes are unknown, microvascular disease underlies many complications in type 2 diabetes (including damage to the eyes, kidneys and peripheral nerves), and vascular disease

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(measured as high-intensity lesions) has been observed among older adults with major depression (Kumar, Mintz, Bilker, & Gobblieb, 2002). Thus, studying the relationship of depression and cognitive functioning in type 2 diabetes can lead to better understanding of the pathophysiology of depression and vascular disease.

A recent review suggests that there are few studies examining the interactive effects of depression on cognition in type 2 diabetes (Awad, Gagnon, & Messier, 2004). In contrast, there has been considerable research on the relationship between type 2 diabetes and cognitive functioning. Overall, type 2 diabetes has been related to mild cognitive deficits, likely in the domains of verbal memory, processing speed and to a less degree, executive functioning (see review by Awad et al., 2004). However, methodological and study design differences, such as variations in sampling, assessment instruments, degree of diabetes severity and the presence of comorbid illnesses have resulted in tentative conclusions (Hassing et al., 2004; Stewart & Liolitsa, 1999; Strachan, Deary, Ewing, & Frier, 1997).

Similar to the diabetes literature, research examining the relationship between depression and cognitive functioning is mixed. There is evidence suggesting that the pattern of cognitive impairment varies by depression subgroup or severity (e.g., major versus minor depression) (Airaksinen, Larsson, Lundberg, & Forsell, 2004; Elderkin-Thompson et al., 2003). In general, depression has been linked to decline in a range of cognitive domains, including memory, executive functioning, attention and psychomotor speed (Alexopoulos, Kiosses, Klimstra, Kalayam, & Bruce, 2002; Butters et al., 2004).

The purpose of this study was to examine whether type 2 diabetics with major depression were characterized by more severe cognitive dysfunction relative to type 2 diabetics with no current or history of depression (diabetic controls) and a healthy non-depressed, non-diabetic control group. We predicted that depressed diabetics would have poorer overall cognitive functioning than diabetic and healthy controls, with the greatest impairments in verbal memory, attention/processing speed and executive functioning. Further, diabetic controls would perform more poorly than healthy controls in verbal memory, attention/processing speed and executive functioning.

#### 1. Method

#### 1.1. Subjects

Data for the present study were from a larger research project examining the cerebrovascular basis of depression in type 2 diabetes at the UCLA Medical Center (A. Kumar, primary investigator). Patients were recruited between October 2002 and September 2004. Fifty-four adults (between the ages of 30 and 80) diagnosed with type 2 diabetes by their primary care physicians were recruited from three outpatient clinical sites: Gonda Diabetes Center at the University of California, Los Angeles (UCLA; Westwood, CA), UCLA Division of Endocrinology (Santa Monica, CA) and a satellite diabetes clinic (Alhambra, CA). Of these, 27 met DSM-IV criteria for major depressive disorder, received a score of at least 15 on the 17-item Hamilton Rating Scale for Depression (HAM-D), and denied a history of depression prior to diabetes onset. The remaining 24 diabetics denied a history of or current depression and did not meet diagnostic criteria (diabetic controls). Three other patients were diagnosed with minor depression and excluded from the present analyses. Twenty-six healthy controls were recruited through community efforts, specifically senior centers, senior newsletters and newspaper advertisements circulating in Los Angeles.

Exclusion criteria included: dementia, central nervous system diseases, unstable medical illnesses, other Axis I disorders (including bipolar disorder), drug or alcohol dependence, or head trauma. No participant scored below 24 on the Mini Mental State Exam (MMSE; Folstein, Folstein, & McHugh, 1975), and no subject was admitted if they were taking anti-depressant or psychotropic medications. Depressed patients were referred for treatment following the study assessments; however, no patients were deemed as severely depressed or suicidal. All diabetic patients were on varying combinations of oral hypoglycemic agents and insulin for blood sugar control.

Additional exclusion criteria were implemented to address language and cultural biases that might interfere with neuropsychological assessment interpretation, including: monolingual in any language other than English (i.e., Spanish) and if bilingual, a preference to test in any language other than English, which excluded seven patients from the depressed diabetic group and four from the diabetic control group. The final sample included 20 depressed diabetics and 20 diabetic controls. Two subjects were excluded from the healthy control group due to language biases, and one subject refused testing. An additional 11 healthy controls were included with the present sample from a related late-life depression study administering the same neuropsychological battery, which totaled 34 healthy controls.

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