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Preliminary analysis of age of illness onset effects on symptom profiles in major depressive disorder

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Abstract

Objective—Major depressive disorder (MDD) is prevalent across the lifespan, but relatively little is known about how age of illness onset impacts the cognitive and affective presentation of MDD.

Method—We explore depressive symptoms and cognition in 70 adults (30–89 years old) with MDD. Participants were divided into three groups based on age-of-MDD-onset: early (<30 years), midlife (30–49.9 years) and late (>50 years). Symptoms were assessed using the Hamilton Depression Rating Scale (HDRS); principle component analysis was used to create symptom component scores. Cognitive functions were measured.

Results—The late-onset group were significantly older than the early and midlife-onset groups. Analysis controlled for age and haemoglobin A1c levels as some participants had diabetes. The late-onset group demonstrated greater Weight Loss & Gastrointestinal symptoms compared to the early-onset group. Suicidal Thoughts & Sleep disturbance were higher in both the early and late-onset groups compared to the midlife group. Correlations between symptom components and cognitive domains varied by age-of-onset group.

Discussion—This preliminary analysis demonstrates cognitive and affective profiles that are both unique to age-of-onset and common across MDD. Symptoms profiles may assist in identifying factors influencing depression and enhance the clinical evaluation and care of individuals struggling with the effects of depression across the lifespan.

Keywords

Major depressive disorder; Symptom profile; Cognition; Aging

Introduction

Major depressive disorder (MDD) is the most common psychiatric disorder, with an annual prevalence of 6.6%, a lifetime prevalence of 16.2% and a significant impact on mortality (Kessler et al. 2003). There is increasing awareness of MDD developing across the lifespan

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(Weiss and Garber 2003; Blazer 2003), with studies beginning to examine symptom profiles and factors influencing onset and course of disease (Alexopoulos et al. 2002; Baldwin and Tomenson 1995; Gottfries 1998; Iacoviello et al. 2010). Among adults with MDD, several studies have suggested that symptom profiles may differ depending upon whether age-of-onset occurs in early, midlife or late-life (Alexopoulos et al. 2002; Baldwin and Tomenson 1995; Gottfries 1998). A better understanding of the effect of age-of-onset on MDD symptom profile and associations with cognitive difficulties may clarify why results differ across studies and enhance MDD clinical evaluations and care.

Among adults, studies have generally investigated symptom profiles in young/midlife compared to late-life depression (LLD), with the suggestion that LLD is a more heterogeneous group, including individuals with onset throughout the lifespan. Moreover, it has been suggested that the mechanism for MDD differs with age; biochemical changes being notable in young MDD compared to white matter changes in LLD (Blazer 2003; Kalia 2005). Among LLD, loss of interest in activities, psychomotor retardation and cognitive dysfunction are more common than among young/midlife MDD (Alexopoulos et al. 2002; Korten et al. 2012). Furthermore irritability and severity of symptoms are reduced in LLD compared to young/midlife MDD (Gottfries 1998; Gallo and Rabins 1999), co-morbid anxiety symptoms have also been shown to be lower in LLD although results are mixed (Gottfries 1998; Baldwin and Tomenson 1995). A recent study found significant differences in symptoms endorsed by young/midlife versus older adults with MDD on two depression scales (Hybels et al. 2011). The LLD group more strongly endorsed items associated with appetite/weight loss and reduced libido; whereas the young/midlife group scored more highly on sadness, irritability, feelings of failure, pessimism and thoughts of death/suicide (Hybels et al. 2011). Latent cluster analysis assigned individuals to groups based on symptom profile similarities however groups did not differ on current age or age-of-onset (Hybels et al. 2011). Notably, depression severity was the factor that best explained group membership. Studies have also identified differences between LLD groups with early versus late-life illness onset. Late-onset has been associated with fewer thoughts that life is worthless, less guilt and less anxiety (Baldwin and Tomenson 1995; Gallagher et al. 2010). Taken together these results may suggest that age-of-onset may be as important as current chronological age when examining depression symptom profiles.

Unlike symptom profiles, cognitive difficulties in depression have been extensively explored (Han et al. 2006; McClintock et al. 2010). In both young MDD and LLD, depression has been associated with impairments particularly in executive function and episodic memory (Stordal et al. 2004; Elderkin-Thompson et al. 2006), although it is important to note that not all patients show deficits (McClintock et al. 2010). In LLD, deficits in memory function are often mediated by executive abilities (Elderkin-Thompson et al. 2006; Elderkin-Thompson et al. 2004; Lamar et al. 2012) and executive dysfunction has been shown to persist even after alleviation of depressive symptoms and normalization of other cognitive (e.g. memory) deficits (Kalayam and Alexopoulos 1999).

The aim of the current study was two-fold. First, to determine if depression age-of-onset influenced depressive symptomatology profiles on a commonly used clinical measure of depression –the Hamilton Depression Rating Scale (HDRS). Based on previous findings

investigating the component structure of the HDRS (Hammond, 1998; Pancheri, Picardi, Pasquini, Gaetano, & Biondi, 2002; Shafer, 2006), we predict that symptoms will differ across groups divided by MDD age-of-onset with the late onset group demonstrating greater psychomotor slowing and less irritability than the early onset group. Second, to explore associations between HDRS components and cognitive abilities; we hypothesize that the pattern of correlations between these variables will differ across age-of-onset groups with cognitive function being more strongly associated with HDRS components in the late age-of-onset group.

Methods

Participants

Data were collected as part of a larger program of research investigating depression and diabetes across the lifespan within the Department of Psychiatry, University of Illinois at Chicago. This research was approved by the University of Illinois at Chicago Institutional Review Board and conducted in accordance with the Declaration of Helsinki. All participants gave informed consent.

Data presented here include adults with a clinical diagnosis of MDD aged over 30, recruited via community outreach (newspaper, radio and television advertisements), allowing investigation of depressive symptoms across the lifespan. Participants underwent a preliminary telephone screen. Exclusion criteria were current or past history of neurological disorders (i.e. dementia, stroke, seizure, etc.), history of head injury or loss of consciousness, present or past history of substance abuse or dependence, present or past history of an Axis I psychiatric diagnosis other than MDD and psychotropic medication use including anti-depressant medication. All study participants were free of anti-depressant medication for at least two weeks in order to study depressed mood in an untreated state (no individual was taken off medication to participate).

After passing the telephone screen, participants completed a detailed evaluation including cognitive, i.e. Mini-Mental State Examination (MMSE; Folstein et al. 1975) and affective, i.e. Structured Clinical Interview for DSM-IV (SCID; Spitzer et al. 1992) screens. Measures were administered by a trained research assistant and followed by an evaluation by a board certified (AK) or board eligible (OA) psychiatrist who completed the HDRS (Hamilton 1960). Raters were blind to telephone screen information. Age-of-onset of depressive disorder was recorded during the SCID. Final inclusion criteria was a score of ≥15 on the HDRS. All participants had an MMSE score >24 indicating that cognitive impairment was unlikely and were native English speakers. Participants received an assessment of vascular risk using the Framingham Stroke Risk Profile (FSRP; Wolf et al. 1991). History of stable (e.g. diabetes) or remitted medical illness (e.g. cancer) were not exclusionary factors. Haemoglobin A1c (hA1c) was measured for all participants.

One hundred and eighteen individuals with a history of MDD attended initial screening. Forty-eight individuals were excluded: 15 -past substance or alcohol abuse/dependence; two -English as a second language; three -contra-indicative medication; one -non-compliant with thyroid medication; 21 -psychiatric diagnoses other than depression; one -history of memory

loss; one -abnormalities on MRI; four -sleep apnea. Of the 70 individuals who passed screening, 19 (27%) had diabetes. This is only marginally higher than the state of Illinois average (23.8%; Danaei et al. 2009) and is likely due to active recruitment of individuals with diabetes for one of our studies. To assure that results were not driven by individuals with diabetes, analyses were performed controlling for hA1c levels. See Table 1 for details of participants.

Neuropsychological Assessment

Qualifying participants completed a comprehensive neuropsychological assessment, administered by a trained research assistant. The cognitive domains of Learning and Memory (L-M), Attention and Information Processing (AIP), Executive Function (EF) and Semantic Language (SL) were assessed using the following measures. L-M: California Verbal Learning Test-II (Delis et al. 2000) short and long delay free recall, free recall measures from the Wechsler Memory Scale-III (Wechsler et al. 1998) Logical Memory I and II and Visual Reproduction I and II. AIP: Stroop Color and Word trial raw scores (Golden 1978), Trail Making Test A time to completion (Army Individual Test Battery 1944) and Digit-Symbol Coding raw score from the Wechsler Adult Intelligence Scale-III (Wechsler 1997). EF: Category Switching total correct from the Delis-Kaplan Executive Function System battery (Delis et al. 2001), Trail Making Test B time to completion (Army Individual Test Battery 1944), Stroop Interference Score (Golden 1978), Backwards Digit Span raw score from Wechsler Adult Intelligence Scale-III (Wechsler 1997) and Self-Ordered Pointing Task Total Errors (Petrides and Alivistatos 2002). SL: total correct responses from Animal Fluency (Spreen and Strauss 1998) as well as a (semantic) Association Index and Percent in Cluster (Carew et al. 1997).

Raw scores for each of the above mentioned variables were transformed into z-scores using the mean and standard deviation of 87 healthy control subjects recruited as participants in the studies described above. Control participants were recruited across the same age range as the depressed patients (30–89), but were older (62.18 years) and had higher educational level (15.41) than patients. Z-scores were coded so high scores reflected good performance across all variables and were collated to produce a mean score for each cognitive domain, L-M, AIP, EF and SL. For each cognitive domain, Cronbach's alpha was computed to assess how well the variables measured each latent construct across MDD participants. All values were considered good, indicating that each variable measured a unidimensional latent construct (L-M, α=.860; AIP, α=.780; EF, α=.628; SL, α=.730).

Statistical Analyses

Analysis was performed using PASW Statistics 18 (SPSS). Principle component analysis (PCA) with varimax rotation was performed to identify the individual components for the HDRS across the entire sample of depressed individuals. PCA included all 17 items from the HDRS with components extracted for eignevalues ≥1. Varimax rotation was performed with the maximum iterations for conversion set to 25. The highest component loadings for each item on the HDRS were selected, two items for each component (except Component 2 which included 3 items), the scores for these items were used to calculate the component scores for subsequent analysis. Raw data from the HDRS was converted into z-scores and

was recoded as negative for those items with a negative loading; scores were averaged to create the variable for each component.

In order to examine differences associated with age-of-onset, individuals were separated into three groups based on: 1) onset before age 30; 2) onset in midlife -between 30–49.9 years of age; 3) onset in later life –after 50 years of age. Although many studies use a cut-off of 60 years to distinguish LLD, others have utilised younger cut-offs at 40, 50 or 55 years (Korten et al. 2012; Aizenstein et al. 2011; Beekman et al. 2002). We employ a cut-off at 50 years to represent a median of these options and to ensure a larger sample size at this grouping level. Individuals with an earlier onset were divided based on onset before or after age 30, in order to examine potential differences between those who develop MDD during the critical risk period or later.

Differences between age-of-onset groups on demographic variables, HDRS components and cognitive function were assessed using Chi-square, individual analyses of variance (ANOVAs) and ANCOVAs controlling for current age and hA1c values. Post-hoc ANCOVAs were performed to determine individual group differences. Partial correlations between the HDRS components that had demonstrated group difference and cognitive variables were performed for each age-of-onset group controlling for current age and hA1c levels. These variables were controlled for due to group differences in current age and number of diabetics in each group. Due to the exploratory nature of the analysis we have not imposed multiple comparison corrections but have reported all correlations for reader consideration.

Results

Age-of-onset

Twenty-five (36%) individuals had early onset, i.e. first episode before age 30; 31 (44%) had midlife onset (30–49.9 years); and 14 (20%) had late onset (after age 50).

Demographic Variables

As expected the groups differed significantly on current age ($F(2,65)=7.85$, $p=.001$), where the late onset group (mean age=64.14 years) were significantly older than early (mean age=53.75 years) and midlife (mean age=49.87 years) onset groups. This result remained significant after controlling for hA1c levels ($F(3,65)=6.53$, $p=.003$). There were significantly more individuals with diabetes in the midlife group compared to the early onset group ($X^2=7.20$, $p=.009$). No significant difference in diabetes diagnosis were observed between the early and late onset groups ($X^2=.042$, $p=1$) but comparison of midlife and late onset groups demonstrated a non-significant trend ($X^2=4.01$, $p=.09$). Distribution of patients was not sufficient to incorporate this variable into analyses for all groups therefore we hA1c levels were included as a control variable for all analyses. Groups did not differ on highest education level, sex, FSRP or HDRS scores even after controlling for age and hA1c levels. See Table 1 for demographic information. Further analysis will include current age and hA1c levels as covariates.

Principle Components Analysis

PCA resulted in a seven component solution explaining 66% of the variance, see Table 2 for the component structure.

HDRS Components

After controlling for current age and hA1c values group differences were noted on two Components. For Component 1 –composed of weight loss and gastrointestinal symptoms, scores were higher in the late onset group ($F(4,60)=4.57$, $p=.014$) compared to the early ($p=.01$) and midlife onset groups (trend towards significance, $p=.061$). Component 6 comprised of high endorsement of suicidal thoughts and early sleep insomnia, but low endorsement of mid-night insomnia ($F(4,60)=3.19$, $p=.048$); for Component 6 the midlife onset group demonstrated significantly lower scores compared to the early and late onset groups ($p<.05$). A non-significant trend was noted for Component 7 (Guilt and Hypochondria; $F(4,60)=3.02$, $p=.056$), with higher scores in the early versus late onset group ($p<.05$). No group differences were noted for the other Components.

Correlations between HDRS Components and Cognitive variables by age-of-onset group

Correlations were performed controlling for age and hA1c, only where significant group differences had been observed, see Table 3. In the midlife onset group, Component 1-Weight loss and Gastrointestinal symptoms correlated with L-M and EF with lower cognitive function associated with higher symptom scores. A similar pattern was observed with EF in the late onset group but did not reach significance in the early onset group. A significant correlation was also observed between Component 6-Suicidal Thoughts and Early Sleep Insomnia and L-M for the early onset group only, where higher L-M scores were associated with more endorsement of symptoms.

Discussion

In this sample we describe profiles of MDD in 70 individuals grouped based on age-of-onset: before 30 years (36%), between 31–49.9 years (44%) or after age 50 (20%). The community based data presented here is complementary to that provided by larger-scale epidemiology studies (Kessler et al. 2005; Kessler et al. 2003) and provides an exploratory analysis of the symptom and cognitive profile associated with MDD age-of-onset. All patients were community dwelling, medication free and met criteria for MDD, diagnosed by an experienced Psychiatrist.

PCA identified seven components for the HDRS. Previous Factor Analyses have identified between 3–6 factors on the HDRS and the scale has been criticised for not representing a single construct (Cole et al. 2004). Prior studies of LLD and MDD demonstrate similarities with the components we identified where items cluster into a factor across a number of studies i.e. Weight Loss with Gastrointestinal symptoms; Low mood with Slowing; and Agitation with Psychological Anxiety (Hammond 1998; Pancheri et al. 2002; Shafer 2006). Differences also exist, with more items being included within each factor in previous studies. It is worth noting, that the study with the greatest similarity to the components described here investigated LLD rather than MDD in youth/midlife (Hammond 1998).

Moreover, many studies performed factor analysis whereas we performed principle component analysis (to include all of the observed variance), which may also lead to differences in results.

In accordance with our first study aim, we found that MDD age-of-onset influenced depressive symptomatology profiles on the HDRS. Late onset individuals demonstrated more Weight Loss and Gastrointestinal symptoms than the other two groups (only non-significant trend for midlife) and less Guilt and Hypochondria (trend towards significance only) than the early-onset group. Previous studies have suggested that weight loss is common in late-life depression, often due to a lack of self-care (Korten et al. 2012; Gallo and Rabins 1999; Koster et al. 2010), although it is worth noting that both weight loss and gain are common symptoms of MDD (Koster et al. 2010). Those with both early and late illness onset endorsed more suicidal thoughts and insomnia compared to those with midlife illness onset. It has been suggested that early insomnia is common (Gottfries 1998) and suicide rates particularly high in late-life depression (Conwell et al. 2002).

Many symptoms were not in keeping with our hypothesized profile differences, demonstrating a similar symptom pattern regardless of age of first depressive episode. In particular, similar profiles were noted on anxiety and agitation, where we hypothesized less anxiety among late-onset adults (Gottfries 1998). Individuals were excluded from the study if they had co-morbid diagnoses including anxiety disorders, therefore we may have excluded patients who demonstrated this pattern. In addition, the late-onset group was relatively small, which may limit our power to detect group differences.

Few correlations were observed between the HDRS components and cognitive domains; significant results differed by age-of-onset group partly supporting our second study aim and hypothesis. In midlife and late-onset groups, more symptoms of weight loss and gastrointestinal problems were associated with poorer cognitive abilities, but no association was observed for the early onset group. This may reflect severity of illness with more symptoms and greater cognitive impairment being strongly associated (Han et al. 2006). These symptoms may be especially impactful among older adults where weight loss is associated with declining health and additional complications (McDougall et al. 2007; Yohannes et al. 2003). Seemingly paradoxically, suicidal thoughts and sleep disturbances in the early onset MDD group are associated with better performance in L-M. Although sleep disturbances are common in depression (Armitage 2007), some speculate that individuals with better memory are prone to rumination leading to sleep disruption (Guastella and Moulds 2007; Moulds et al. 2007), alternatively this pattern may reflect some form of adaptation due to disease duration. Other work has demonstrated better performance by individuals with current depression compared to healthy controls on a complex decision making tasks, leading to speculation that depression may promote systematic processing due to an increased desire for control (von Helversen et al. 2011). These differences in correlations regardless of no significant group differences in either HDRS components or cognitive domains may reflect subtle differences associated with the mechanisms relating to age-of-onset or illness duration. A larger, lifespan study is required to elucidate these potential associations.

We acknowledge several limitations. The primary limitation is the modest study sample size and the small number of participants within the late-onset group. For the PCA, the sample size is within the criteria for pilot analysis (minimum pilot ratio ≥3:1; minimum general analysis ratio 5:1; current sample ratio 4:1) and all components meet Kaiser's criteria of eigenvalues ≥1 (Osborne and Costello 2004; Neill 2012). So although the sample is modest, PCA is appropriate for this exploratory study; future work should include a larger cohort of individuals across the lifespan. Given the modest sample sizes across the individual groups particularly the late-onset group, results should be interpreted with caution. Due to the exploratory nature of the analysis we have not imposed multiple comparison corrections but have reported all correlations for reader consideration. Using the HDRS may also be seen as a limitation as it does not include symptoms of hypersomnia, increased appetite or weight gain; however, the same criticism could be levelled against any measure of depressive symptomatology many of which are not all-inclusive. The benefit of the HDRS is that it relies on rating by a clinical expert rather than self-report, where cognitive abilities and motivation may influence questionnaire completion. It may be suggested that being unmedicated, this sample has a “less severe” depression than is typical. While this may be case, all individuals met criteria for MDD (HDRS range 15–27) and thus may reflect a “typical” community sample.

Conclusion

In summary, we have demonstrated both differences and similarities between adults based on age-of-onset of depressive illness that may assist clinicians in identifying components influencing depression at various ages and promote earlier detection of possible MDD in vulnerable individuals. Knowledge of these differences may not only enhance clinical evaluations for MDD but also individual care for patients (Bierman and Statland 2010). In the long term, clusters of symptoms may differentiate between patients, perhaps predicting neurobiological brain changes which may inform treatment selection. Studies may examine whether symptom profiles predict response to different treatments or are associated with specific brain changes, although further research is required before interventions can be implemented. It has been suggested that better understanding of symptom type and aetiology could also informing depression risk associated with physical health problems such as acute coronary syndrome and metabolic syndrome, although such disorders often co-occur with age (Davidson et al. 2005; Luppino et al. 2011). Future large-scale studies exploring symptom profiles in MDD across the lifespan should include additional younger age-of-onset groups (i.e. adolescence) as well as minor and subclinical depression to further investigate risk factors including health status, stress and other vulnerabilities specific to age-of-onset in MDD. Epidemiological samples may also allow exploration of both age and illness onset effects by comparing young MDD individuals directly with older early-onset LLD patients. Disentangling these influences may only be possible with large-scale community samples.

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Key points

- Individuals with late-onset depression (>50 years) reported more symptoms of Weight Loss & Gastrointestinal difficulties than those with early-onset depression (<30 years).
- Suicidal Thoughts & Sleep disturbance were greater in groups with early and late-onset depression, compared to onset in midlife (30–50 years).
- Weight Loss & Gastrointestinal symptoms were correlated with cognitive function in groups with depression onset in mid- or late-life

Table 1

Mean and Standard Deviation for Demographic data and Depressive Scales

	Whole sample (n=70)	Early Onset <30 yrs (n=25)	Midlife Onset 30–49.9 yrs (n=31)	Late Onset ≥50 yrs (n=14)
Current Age*	54.10 (12.18)	53.72 (13.75)	49.87 (9.81)	64.14 (8.04)
	Range 31–88	Range 31–86	Range 32–64	Range 55–88
Sex (m:f)	31:39	9:16	17:14	5:9
Diabetic (n:y)	51:19	22:3	17:14	12:2
FSRP score	7.27 (4.68)	7.95 (5.48)	6.04 (3.72)	9.07 (4.68)
Highest Degree	14.13 (2.62)	13.64 (2.33)	14.42 (2.81)	14.36 (2.71)
Age-of-onset	35.72 (16.68)	17.52 (7.15)	38.77 (6.05)	58.86 (8.86)
	Range 31–86±			
Years since first diagnosis	18.40 (17.01)	36.22 (15.35)	11.10 (9.19)	5.29 (4.94)
	Range 0–71±	Range 2–71	Range 0–38	Range 0–14
HDRS score	18.9 (14.13)	19.04 (2.95)	18.63 (2.59)	19.21 (3.89)
HDRS Range	15–27	15–25	15–26	15–27

FSRP= Framingham Stroke Risk Profile score; HDRS=Hamilton Depression Rating Scale.

*Significant difference between groups;

±two individuals described themselves as “always having been depressed” and are not included in this group mean;

Table 2

Rotated components structure

Components	1	2	3	4	5	6	7
Depressed Mood	.193	.390	.080	.042	**.604**	.114	−.202
Feelings of Guilt	.070	−.004	.156	−.162	.173	−.249	**.692**
Suicidal thoughts	−.034	.171	−.075	−.066	.140	**.595**	−.239
Insomnia - Early	.158	−.239	.162	−.026	−.104	**.785**	−.042
Insomnia - Middle	.136	.042	.259	−.370	−.290	**−.517**	−.255
Insomnia - Late	.033	−.329	.354	**−.524**	−.092	−.231	−.254
Reduced Work and Activities	.066	**−.478**	.420	.409	−.165	−.025	.036
Psychomotor Retardation	−.061	−.029	−.009	.001	**.882**	.028	.010
Agitation	−.018	**.754**	−.026	−.021	.004	−.019	.060
Anxiety - Psychological	−.151	**.672**	.467	.009	.109	−.156	−.027
Anxiety – Somatic	−.180	.073	**.783**	−.141	−.148	−.058	.091
Gastrointestinal Somatic Symptoms	**.837**	−.044	−.095	.027	−.019	.134	.093
General Somatic Symptoms	−.346	−.374	−.190	**.519**	.076	−.152	−.061
Genital Symptoms	.062	−.034	−.009	**.865**	−.024	−.028	−.243
Hypochondria	−.115	.066	.049	−.026	−.365	.030	**.678**
Loss of Weight	**.852**	−.050	.062	−.057	.074	−.099	−.149
Insight	−.216	−.045	**−.649**	.052	−.286	−.122	−.132
Rotated Sum of Squares Eigenvalue	1.76	1.76	1.74	1.66	1.59	1.46	1.29
Rotated Sum of Squares % Variance	10.36	10.33	10.21	9.75	9.32	8.61	7.61
% Variance explained by orthogonal factors	13.97	12.47	11.17	8.11	7.48	6.79	6.19
Total Variance explained by orthogonal factors				66.18%			

Item included in each component presented in bold. Component descriptors: 1) Weight loss & Gastrointestinal symptoms; 2) Agitation/Anxiety & Reduced activities; 3) Lack of insight & Somatic anxiety; 4) Somatic Symptoms & sleep disturbance; 5) Low mood & slowing; 6) Sleep disturbance & suicidal thoughts; 7) Guilt & Hypochondria.

Table 3

Partial correlations between HDRS components showing group differences and cognitive variables, by age-of-onset group controlling for current age and hA1c levels.

	1 Weight loss & GI	6 Sleep disturbance & suicidal thought
Learning & Memory		
<30	−.378	**.478** *
dfs=20	.101	**.033**
30 – 49.9	**−.404** *	−.143
dfs=24	**.040**	.485
≥ 50	−.192	.022
dfs=10	.550	.946
Attention & Information Processing		
<30	−.025	−.200
dfs=20	.915	.398
30 – 49.9	−.235	.091
dfs=24	.248	.658
≥ 50	−.222	.064
	.487	.842
Executive Function		
<30	−.363	.157
dfs=20	.115	.510
30 – 49.9	**−.541** **	.003
dfs=24	**.004**	.990
≥ 50	**−.669** *	−.025
dfs=10	**.017**	.939
Semantic Language		
<30	−.102	−.135
dfs=20	.667	.571
30 – 49.9	−.296	.149
dfs=24	.142	.468
≥ 50	−.107	−.354
dfs=10	.740	.259

dfs = degrees of freedom

* Significant correlation p<.05;

** Significant correlation p<.01;