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Depressive symptoms and poorer performance on the Stroop Task are associated with weight gain

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Abstract

Objective: Executive function impairments and depression are associated with obesity but whether they predict weight gain is unclear.

Methods: Forty-six individuals (35 m, 37 ± 10 y) completed the Stroop Task, Iowa Gambling Task (IGT), Wisconsin Card Sorting Task (WCST), Inventory for Depressive Symptomatology (IDS-SR), Physical Anhedonia Scale (PAS), and Perceived Stress Scale (PSS). Body composition (DXA) and fasting glucose were also measured. Data from return visits were used to assess changes in weight.

Results: Poorer Stroop and WCST performance associated with higher BMI whereas poorer IGT and WCST performance associated with higher body fat (%; all p 's < 0.05). Stroop interference ($p = 0.04$; $p = 0.05$) and IDS-SR ($p = 0.06$; $p = 0.02$) associated with increased BMI and weight gain (%/yr). In a multivariate linear model Stroop interference ($\beta = 0.40$, $p < 0.01$; $\beta = 0.35$, $p < 0.01$) and IDS-SR ($\beta = 0.38$, $p < 0.01$; $\beta = 0.37$, $p < 0.01$) independently predicted increased BMI and weight gain (%/yr) even after controlling for baseline weight and glucose levels.

Conclusions: Poorer response inhibition and depressive symptoms, but not glucose levels, predicted weight gain. Evaluating neurocognitive and mood deficits could improve current treatment strategies for weight loss.

Keywords

Cognitive function; Obesity; Mood disorders; Depression; Psychopathology

1. Introduction

Obesity is associated with cardiovascular risk factors including impaired glucose regulation, and more recently, with increased risk of Alzheimer's disease and cognitive decline later in life [6]. The link between obesity and neurocognitive impairment has been most strongly

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Conflict of interest

No conflict.

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inferred by adiposity associated alterations within the prefrontal cortex (PFC) [38]. Studies from our lab [25,34] and others have demonstrated that elevated BMI is associated with decreased activity [25,39] and lower gray matter volume [29] in the PFC.

A growing body of literature indicates a link between obesity and impairments in executive functions [20,36], higher order brain processes that regulate cognitive processes such as planning and reasoning, flexibility, inhibition, problem solving, and decision making [13]. Neuroimaging studies indicate that the PFC is activated during executive function related tasks [39]. Impairments in executive functions are associated with maladaptive eating behaviors, inability to resist impulses, impaired decision making and self-control, and generalized poor weight loss attempts ([20]; X [41]). The ability to make healthy choices in the face of biological drives relies heavily on executive functioning [5].

Executive functions represent several different neurocognitive domains and the literature is unclear as to specific areas of executive functioning that associate with obesity. For instance, compared to normal BMI adolescents those with higher BMI performed significantly worse on measures of inhibition, flexibility, and decision-making [37], but no differences were seen in measures of working memory, planning, or reasoning tasks. In contrast, one study found that overweight and obese individuals displayed significant impairments in working memory compared to lean individuals [9]. Furthermore, individuals with obesity, anorexia-nervosa, or bulimia nervosa displayed poorer decision making abilities on the IGT task [4] compared to controls. The lack of consensus as to which domains of executive function are impaired the most with increasing adiposity and therefore which measurements to use highlights a critical need for studies with longitudinal follow-up to fully uncover which measures are important.

Psychological variables are also related to impairments in executive functions, obesity and glucose regulation. A recent review suggests a neuropsychological model of obesity and obesity related behaviors [21] with interactions between psychological and cognitive factors [27]. Neuroimaging studies have reported that the altered brain areas seen in obesity and executive dysfunction are also associated with psychological factors such as stress and depression [22]. In healthy adults, decreased gray and white matter volumes within the dorsolateral and ventrolateral PFC were associated with greater perceived stress [30]. Compared to healthy controls, individuals with major depressive disorder (MDD) had decreased gray matter volume in the PFC [32]. However, independent of MDD, higher BMI across both groups was also associated with decreased gray matter volume in the PFC. Other studies looking at the impact of psychological factors on executive functioning found that nonsomatic (i.e. anhedonia and self-esteem) depression symptom clusters were predictive of executive functioning impairment [17] and our group recently showed that perceived stress, anhedonia and depression were associated with weight gain [19].

There is a known association between depression and impaired glucose regulation and many studies also link impaired glucose regulation [18] to lower levels of cognitive function on neuropsychological tests [23]. There is evidence that even mildly increased glucose concentrations in the non-diabetic range are associated with impaired cognition [18].

Moreover, a recent study from our lab found an association between impaired glucose regulation and poorer performance on the Stroop Task, a measure of selective attention [36].

Despite the identified associations between psychological factors, glucose regulation, executive function and obesity, few studies [6,21,32] have examined all 4 variables simultaneously and none with longitudinal data on weight change. We hypothesized that measures of executive functioning would be associated with adiposity measures at baseline and that impaired executive functioning and fasting glucose levels at baseline would predict increased body adiposity over time. Lastly, we hypothesized that the association between executive functioning and change in adiposity may be moderated by psychological variables (Depression, Anhedonia, Stress).

2. Methods

2.1. Participants

Forty-six individuals were recruited from the greater Phoenix area by advertisement to participate in one of three studies on our clinical unit (Clinical Trial Identifier: NCT00523627, NCT00342732, NCT01224704). All were observational studies of the effects of overconsumption and different diets on energy expenditure or food intake preference as risk factors for obesity. The studies did not include any medications or weight loss interventions. All measures were collected on the Clinical Research Unit of the National Institute of Diabetes and Digestive and Kidney Diseases – Phoenix (NIDDK). Inclusion criteria for all studies consisted of healthy adults, between the ages of 18–55, with no evidence of illness by history, physical or basic laboratory measures. No participants were taking medication. Exclusion criteria included substance abuse (positive urine test), nicotine use, or reported excess alcohol use (> 3 drinks/day). Prior to participation, all participants were informed of the nature, purpose and risks of the study and written informed consent was obtained. The decision to combine data from these studies to assess the impact of psychosocial measures on body weight was pre-planned as these studies were all relatively small. The experimental protocols were approved by the Institutional Review Board of the NIDDK.

2.2. Study design

The first four days of each study was identical. Upon admission to the Unit, participants were placed on a standard weight maintaining diet (WMEN) for three days (20% protein, 50% carbohydrate, and 30% fat), calculated for each individual based on weight [12]. Participants were instructed to consume the entirety of every weight maintaining meal. Within the first 2 days after admission, while participants were weight stable, percent body fat was measured using Dual-Energy X-Ray Absorptiometry (DPX-L; Lunar Radiation, Madison, WI). Further, all individuals completed the neuropsychological performance tests and questionnaires (described below) approximately 1-h after eating breakfast. After three days of the WMEN, a 75 g oral glucose tolerance test was done to exclude individuals with diabetes mellitus [1]. Plasma samples that were drawn at – 10 and 0 min were averaged as a measure of fasting glucose.

2.3. Participants completed 6 neuropsychological measures

1. Iowa Gambling Task [2]: Participants were presented with four decks of cards (A, B, C, D) from which they could choose 100 cards, one card at a time, resulting in either gain or loss of money. Two decks (A & B) were disadvantageous, giving large rewards (\$100) in addition to large penalties that resulted in an overall net loss, while the remaining two decks (C & D) were advantageous, giving small rewards (\$50) but also small penalties resulting in an overall net gain. Cards were presented in 5 blocks of 20 cards each. Instructed to win as much money as possible, participants had to resist the immediate payoff of disadvantageous decks to achieve the long-term rewards of the advantageous decks. The net IGT score is determined by the number of cards chosen from disadvantageous decks subtracted from the number of cards chosen from advantageous decks. A positive score indicates overall advantageous choices and a negative score indicates overall disadvantageous choices (e.g. poorer performance).
2. Wisconsin Card Sorting Task [28]: is a computer based test to measure cognitive flexibility or set shifting, the ability to shift cognitive strategies flexibly in the face of changing environmental contingencies. It is a measure of executive function and provides an assessment of prefrontal cortical function, specifically dorsolateral prefrontal function [16]. The task consists of four stimulus cards and 128 response cards which depict figures (crosses, circles, triangles, or stars), colors (red, blue, green, or yellow), and numbers (1, 2, 3, or 4). The participant is instructed to match response cards to one of the four stimulus cards according to the three possible dimensions (figures, colors, or numbers). The participant receives feedback as to if they were right or wrong. After achieving 10 consecutive correct matches, the matching principle is changed without warning. The test ends once the participant matches 6 categories (10 correct card matches per category) or all 128 trials have been attempted. One main outcome variable is perseverative errors, the inability to shift or modify the response to a stimulus despite negative reinforcement. Perseverative errors occur when the participant continues to apply the previous matching paradigm even though a new matching paradigm has been identified. Higher scores represent poorer performance and more perseverative errors.
3. Stroop Word Color Task [11]: is a computer-based test used to measure response inhibition, the ability to attend to certain environmental stimuli while inhibiting others. The task consists of three separate timed trials and participants are instructed to respond as quickly as possible. The stimulus appears in the center of the computer screen and participants are instructed to provide a verbal response prior to pressing the space bar, which displays the next stimulus. During the first trial the participant is instructed to identify the color-words on the screen in black ink (RED, GREEN, BLUE). In the second trial the participant is instructed to identify the color strings of "XXXX" which are written in red, blue, or green ink, and in the final trial participants are instructed to identify the color of the ink in which the color word is written. In this final condition, all the

stimuli were incongruent (e.g., the word “BLUE” printed in red letters). The “Stroop effect” which is a measure of interference, is calculated based on the difference in speed and accuracy (e.g., reaction time) when the color and word are incongruent. It takes more time and participants are more prone to errors when the color does not match the color of the color-word. Stroop interference scores were calculated by subtracting the number of trials achieved in the third condition from the number of trials achieved in the second condition (color-word naming) and dividing the difference by the number of trials achieved in the second condition (color-word naming), multiplied by 100 $[(CN-SW/CN) * 100]$. Higher Stroop interference scores represent poorer performance (decreased processing speed) and suggests impaired executive functioning [15].

4. The Inventory for Depressive Symptomatology – Self-Report [35]: is a 30-item self-report measure of depressive signs and symptoms. Questions are multiple choice scaled from 0 to 3 with 0 being least severe and 3 most severe. Total scores are derived from the sum of all 30 questions with scores ranging from 0 to 90. Scores less than or equal to 14 are indicative of the normal range (i.e. not depressed), 15–25 represent mild depressive symptoms, 26–38 represent moderate depressive symptoms and > 39 are indicative of moderate to severe depression.
5. Physical Anhedonia Scale [7]: is a 61-item scale, which assesses sensitivity to reward (i.e. the degree to which individuals take pleasure from, and are motivated to engage in rewarding behaviors). Items are provided as true and false statements, with each anhedonic response given a score of 1. Higher scores indicate the presence of more anhedonic symptoms (lack of interest/pleasure).
6. Perceived Stress Scale [8]: is a 14-item scale, which assesses different facets related to stress such as unpredictability, lack of control, and stressful life circumstances in the last month. Items are rated on a 5-point Likert scale from 0 (never) to 5 (very often). Scores range from 0 to 56, with higher scores indicating more of perceived stress.

2.4. Follow-up visits

Data from return visits or from re-enrollment into other studies on our research unit was used to assess changes in body weight. In one study (NCT00523627) this was done as scheduled visits requested at six months and one year, and annually up to 5 years. For those who participated in more than one study on our research unit, data from baseline visits to enroll into these other studies were also used. Participants were not prescribed a weight loss intervention during this time nor were they monitored between follow-up visits or enrollment into new studies. Participants' last available return or re-enrollment visit was used as follow-up. Mean follow-up time was 32 ± 25 months (range = 6 months to 3 years) and the mean change in weight at follow-up was 2 ± 3 kg (range = – 7 kg to 9kg).

2.5. Statistical analysis

All statistical analyses were performed using SAS software, version 9.2 (SAS Institute, Cary, NC). Given the defined hypothesis at the outset, alpha was set at 0.05 for all analyses. Normally distributed data are presented as mean $\pm$ standard deviation, while non-parametric data are presented as median (interquartile range (IGR)). Student's *t*-tests were utilized to assess differences in baseline characteristics for normally distributed data and Wilcoxon rank Sum Test for non-parametric data. Pearson correlation coefficients were used to assess relationships between normally distributed variables and Spearman correlation coefficients were used for non-parametric data. Change in adiposity measures were assessed by calculating the percent change per year in order to control for baseline measures as well as variation in follow-up time. Thus, a negative percent change in adiposity is representative of adiposity loss (or weight loss) and a positive percent change in adiposity is representative of adiposity or weight gain. If correlations demonstrated a potential relationship with change in percent change in adiposity measures per year, the variable was entered into a multivariate linear regression model (GLM) controlling for the baseline adiposity measure, age, gender, and race.

3. Results

Participant characteristics are shown in Table 1. Participants ranged in age from 18 to 54 years and in BMI from 18 to 43 (kg/m²). Based on established BMI criteria, 33% (*n* = 15) were classified as lean (BMI < 25), 37% (*n* = 17) as overweight (BMI $\geq$ 25 and < 30) and 30% (*n* = 14) as obese (BMI $\geq$ 30). Consistent with a physically and psychologically healthy cohort, the scores of psychological measures were not within clinical range. Men had a lower percent body fat (*p* = 0.0001) and lower levels of fasting plasma glucose (*p* = 0.024) compared to women, but there were no significant differences between men and women on measures of cognitive function or psychological measures. Caucasians had significantly higher IGT scores compared to Hispanics (*F* = 3.41, *p* = 0.02) but no other significant differences were found between races for adiposity, cognitive functioning, or psychological variables. There were no differences between levels of education on any observed variable.

At baseline, higher BMIs were associated with higher Stroop interference scores (*r* = 0.29, *p* = 0.05; Fig. 1) and more total (*p* = 0.37, *p* = 0.01) and perseverative errors (*p* = 0.36, *p* = 0.01) on the WCST. Higher percent body fat was associated with poorer IGT scores (*r* = -0.37, *p* = 0.01; Fig. 1B), greater total (*p* = 0.38, *p* = 0.008) and perseverative errors (*p* = 0.34, *p* = 0.02) on the WCST and trended with higher glucose levels (*r* = 0.27, *p* = 0.07). Stroop interference scores were not associated with higher percent body fat (*r* = 0.19, *p* = 0.21) although the relationship was in the same direction.

There were no other associations between baseline measures of adiposity, glucose levels or psychological variables with the cognitive performance tests. Furthermore, there were no significant associations between psychological variables and baseline measures of adiposity or glucose levels.

Higher baseline Stroop interference scores (*r* = 0.29, *p* = 0.05; Fig. 2A) and higher depressive symptomatology (*r* = 0.33, *p* = 0.04; Fig. 2B) were positively associated with

percent weight change per year. Similarly, percent BMI change per year was positively associated with higher baseline Stroop interference scores ($r = 0.30$, $p = 0.04$; Fig. 3A) and trended towards significance with higher depressive symptomatology ($r = 0.29$, $p = 0.06$; Fig. 3B). Fasting glucose levels and scores on the IGT, WCST, PAS, and PSS were not associated with percent weight change or BMI change per year or any other adiposity measure (NS).

In a GLM, after controlling for age, sex, race and glucose (all p values > 0.05), both depressive symptomatology ($\beta = 0.35$, $p = 0.01$) and Stroop interference ($\beta = 0.40$, $p = 0.004$) remained positive predictors of percent BMI change per year, explaining 37% of the variance ($F = 3.88$, $p = 0.004$; Table 2). Similarly, in an analysis of percent weight change per year as the dependent variable, both depressive symptomatology ($\beta = 0.37$, $p = 0.06$) and Stroop interference ($\beta = 0.38$, $p = 0.006$) also remained significant predictors even after adjusting for age, sex, race and glucose (all p values > 0.05). The full model explained 38% of the variance ($F = 3.94$, $p = 0.004$; Table 2). Thus, Stroop interference and depressive symptomatology are associated with percent BMI and weight change per year above and beyond fasting plasma glucose levels.

4. Discussion

We found that individuals with greater adiposity performed significantly worse on various measures of executive function. Specifically, those with higher BMI performed worse on the Stroop and WCST and those with greater percent body fat performed worse on the IGT and WCST. In a longitudinal analysis, we found that greater depressive symptomatology and higher Stroop interference scores (i.e. poorer performance) were significant predictors of increases in weight and BMI, even after controlling for baseline weight, age, gender, race and fasting glucose levels. Whereas those with slower processing speeds or greater interference on the Stroop task gained weight, those with faster processing speeds (less interference) lost weight.

Numerous cross-sectional studies suggest a link between obesity and impairments in executive functions and decreased activity in the PFC [20,36]. These studies indicate that the PFC is activated during executive function related tasks. Despite these associations there is a lack of consensus across the literature as to which domains of executive function are the most critical to studying obesity and weight gain. In the current study, we found that worse performance on the Stroop task, a measures of response inhibition, was associated with weight gain over time. Neuroimaging studies have indicated that the Stroop response inhibition phase, known as the Stroop effect, is associated with greater activation of the PFC (X [41].). Furthermore, studies estimating energy intake have reported that poorer performance on measures of response inhibition result in greater consumption of energy intake and greater intake from candy during laboratory eating tests [24,36]. Thus, it appears that less activity in the PFC results in poorer response inhibition, indicated here by poorer Stroop interference scores, which may lead to overeating and subsequent weight gain.

There is a known connection between impaired glucose tolerance and obesity [10] and past research from our lab reported that higher fasting plasma glucose was associated with poor

performance on the Stroop task [15]. In the current study, we found that Stroop interference and depressive symptomatology were predictors of weight gain, even after adjusting for fasting plasma glucose values. To our knowledge, this is the first study to show that neuropsychological variables (i.e. Stroop interference and depressive symptomatology) predict weight gain over and above biological factors (i.e. baseline weight and fasting plasma glucose). These findings highlight the importance of including biological assessments in studies looking at eating behavior and cognition.

This is one of the first studies to examine the effect of executive function across a variety of executive function tasks on changes in weight longitudinally. Decision making abilities (IGT) and cognitive flexibility (WCST) were not associated with weight change, possibly because both of these tests are less reproducible measures of cognitive function [14,31] compared to the Stroop. While the Stroop task has relatively high reliability over time, the IGT and WCST have poor test-retest reliability [36] which may be explained by learning effects such that healthy individuals are expected to improve after taking the tests more than once (S [40].). In contrast, the Stroop task is not impacted by learning effects, as evidenced by its high test-retest reliability [36]. Previous literature [24,26,36] has also indicated that the stop signal task and the go-no go task both measure response inhibition and have been associated with weight change as well as food intake. However, further research is needed to evaluate which measure of response inhibition (Stroop, stop signal, go-no go task) is most strongly associated with weight gain.

We found that higher scores on a measure of depressive symptomatology, but not perceived stress or anhedonia, predicted increased changes in weight. This finding was consistent with previous studies showing that individuals with higher levels of depressive symptoms tended to gain more weight [19] compared to those without depressive symptoms [33]. Ibrahim et al. [19] found higher depression and higher anhedonia scores predicted long-term weight gain but higher depression and lower anhedonia scores led to weight loss. In this smaller cohort however, we did not find an interaction between anhedonia and depression.

Our study has several strengths and limitations. Notably, we observed discordant associations between different adiposity measures and cognitive task performance. BMI is an imperfect surrogate for percent body fat, a more exact measure, which may help explain this discrepancy. Indeed, our main variable of interest, Stroop performance, was not significantly associated with percent body fat. However, the relationship did demonstrate the same directionality which is in line with our finding that Stroop performance was associated with higher BMI. Although our sample size was relatively small, we had the ability to assess longitudinal not just cross-sectional associations with weight and adiposity. We did not have a direct measure of food intake, which could further elucidate the relationship between the impairments in executive function and weight gain. While a small proportion of our study volunteers scored in the mild to moderate range of depression, none were severely depressed and we were therefore unable to assess a true range of clinical depression. Lastly, we did not utilize neuroimaging techniques to confirm the link between Stroop, decreased brain activity within the PFC, and weight gain. Our sample consisted of individuals from an urban public hospital who could participate in inpatient studies and thus might not be typical of the general population. However, our studies included a diverse racial population which allows

for greater generalizability. We included glucose, a biological measurement with a known association with adiposity and cognitive performance. A final strength of the study is that measures were conducted under highly controlled conditions, including 3-days of WMEN and testing that took place within 1-h of a WMEN breakfast.

5. Conclusion

In summary, we demonstrated that higher levels of adiposity were associated with poorer performance on measures of executive function. Furthermore, we found that higher Stroop interference scores and depressive symptoms predicted weight/BMI gain. Our results indicate that response inhibition and mood may play an important role in long-term weight change. Response inhibition is a key feature of self-control and is important in inhibiting impulsive responses to specific stimuli such as overconsumption of high calorie food. Future studies should examine whether such improvements in response inhibition (e.g. using neuromodulation techniques [20]) or cognitive retraining [3] could improve weight loss outcomes. Inhibitory control deficits and depressive symptoms appear to be important underlying risk factors for weight gain, and thus evaluating neurocognitive and mood deficits may improve current treatment strategies for weight loss.

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Fig. 1.

A) Associations between BMI (kg/m²) and Stroop interference scores ($r = 0.29$; $p = 0.05$), B) between percent body fat (%) and IGT scores ($r = -0.37$; $p = 0.01$) Unadjusted Pearson correlation coefficients are shown.

Fig. 2. Associations between percent weight change per year (A) Stroop interference scores ($r = 0.29$; $p = 0.05$) and (B) depressive symptomatology ($r = 0.34$; $p = 0.02$). Unadjusted Pearson correlation coefficients are shown.

Fig. 3.

Associations between percent BMI change per year and (A) Stroop interference scores ($r = 0.30$; $p = 0.04$) and (B) depressive symptomatology ($r = 0.29$; $p = 0.06$). Unadjusted Pearson correlation coefficients are shown.

Table 1

Participant demographic characteristics.

Variable	All	Men	Women
N	46	35	11
Race	6 AA, 8H, 15 W, 17 NA	6 AA, 6H, 11 W, 12 NA	2H, 4 W, 5 NA
Age (yrs)	37.2 ± 10.2	36.3 ± 10.0	40 ± 10.6
Education	12 (12, 14)	12 (12, 14)	14 (12, 16)
Weight (kg)	82.5 ± 19.2	84.7 ± 19.7	75.5 ± 16.1
% Weight change (per year)	1.0 ± 3.7	1.1 ± 3.9	0.5 ± 3.2
BMI (kg/m ²)	28.3 ± 6.7	27.7 ± 6.5	30.2 ± 7.4
% BMI change (per year)	0.36 ± 5.5	1.1 (5.1)	0.68 (2.6)
Fasting glucose (mg/dL)	92.7 ± 6.5 ^a	91.5 ± 5.0	96.0 ± 9.0
Percent body fat (%)	31 ± 12 ^a	26 ± 10	42 ± 10
Follow-up time (months)	32.4 ± 24.7	34.4 ± 23.9	26.2 ± 27.5
Stroop interference	24.7 ± 10.2	23.9 ± 10.5	27.1 ± 9.4
IGT raw	5.8 ± 24.9	7.4 ± 25.3	1.0 ± 23.9
Perseverative ER raw	16.5 (8, 31)	15 (7, 29)	23 (9, 40)
Total correct	70.1 ± 13.1	71.0 ± 11.8	67 ± 17.11
Total error	34 (19, 61)	32 (16, 61)	43 (21, 71)
IDS	12.3 ± 4.1	12.2 ± 9.3	12.6 ± 10.1
PAS	5.1 ± 1.1	5.2 ± 1.0	4.9 ± 1.4
PSS	4.2 ± 2.2	3.9 ± 1.1	5.3 ± 4.1

AA = African American; H = Hispanic; C = Caucasian; A = Asian; NA = Native American + + + +.

IGT = Iowa Gambling Task; Total Correct and Total Error = Wisconsin Card Sorting Task; PAS = Physical Anhedonia Scale; IDS = Inventory for Depressive Symptomatology; PAS = Physical Anhedonia Scale; PSS = Perceived Stress Scale.

Mean ± Standard Deviation (Minimum, Maximum) or Median (25%IQR, 75%IQR).

^aSex differences with $p < 0.05$.

Table 2

Multivariate linear models.

Variables	% BMI change per year			% Weight change per year		
	B	SE B	β	B	SE B	β
Intercept	-4.1	10.8	-	5.8	11.4	-
Age (years)	-0.14	0.07	-0.26	-0.16	0.08	-0.28
Gender (male)	-3.4	1.8	-0.27	-3.1	1.9	-0.23
Race (white)	-0.83	0.50	-0.22	-0.48	0.52	-0.12
Fasting plasma glucose	0.09	0.12	0.11	-0.01	0.13	-0.02
Depressive symptomatology	0.22 [*]	0.08	0.35	0.22 [*]	0.08	0.37
Stroop interference	0.22 [*]	0.07	0.40	0.24 [*]	0.08	0.38
$R^2 = 0.37$; $F = 3.88$; $p = 0.004$			$R^2 = 0.38$; $F = 3.94$; $p = 0.004$			

* $p < 0.01$.