The weight of subclinical vascular disease & neuroticism in late-life depression
Marijnissen, Radboud

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Depressive symptom profiles are differentially associated with general and visceral obesity

Published

Depressive symptom clusters are differentially associated with general and visceral obesity

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Abstract

*Background* – Depressive symptoms and obesity, both risk factors for unfavourable health outcomes, are mutually related although mixed results have been reported. We examined the relationship between obesity and depressive symptoms taking into account different measures for obesity, i.e. Body Mass Index (BMI), Waist circumference (WC) and Waist-to-Hip Ratio (WHR), as well as different depressive symptom clusters. 

*Methods* – Cross-sectional population-based survey. Baseline data of the Nijmegen Biomedical Study. Participants: 1284 persons aged 50 through 70 years. Obesity (BMI, WC and WHR) and depressive symptoms were measured, the latter using the Beck Depression Inventory (BDI). Principal components analysis of the BDI-items yielded two factors, representing a cognitive-affective symptom cluster and a somatic-affective symptom cluster. Multiple regression analyses corrected for confounders were conducted for each measure of obesity, with separate models testing the BDI sum score and both depression symptom clusters, respectively.

*Results* – The BMI was significantly associated with both the BDI sum score ($\beta=.12$, $p<.001$) as well as cognitive and somatic-affective symptom clusters ($\beta=.08$, $p=.008$ and $\beta=.10$, $p=.001$, respectively). The WC and WHR, however, were specifically associated with the somatic-affective symptom cluster ($\beta=.11$, $p<.001$ and $\beta=.07$, $p=.004$, respectively). 

*Conclusions* – Visceral obesity, which is more indicative of vascular risk than BMI, is specifically associated with somatic-affective depressive symptom cluster, which might suggest that these symptoms are primarily due to a (subclinical) somatic condition.

**Keywords**

Depression, Obesity, Aged, Body Mass Index (BMI), Waist Circumference (WC)


**Introduction**

Depression and obesity are risk factors for unfavourable health outcomes, especially cardiovascular disease (CVD) (Faith et al, 2002; Barry et al, 2008; Allison et al, 2009; Everson-Rose et al, 2009). The prevalence of clinically relevant depressive symptoms is estimated at 13.5% of elderly people in the community (Beekman et al, 1999). According to the World Health Organization during the past 20 years, the prevalence of obesity (body mass index (BMI) ≥ 30 kg/m²) in all age groups has more than doubled in Western countries, leading to a prevalence rate of more than 20% in the United States and over 15% in European countries. Most studies report a positive association between obesity and more-severe depressive symptoms (Stunkard et al, 2003; Sachs-Ericsson et al, 2007; De Wit et al, 2009; Luppino et al, 2010), although an absent or even an inverse relationship has also been reported (Carpenter et al, 2000; Ho et al, 2008). Longitudinal studies suggest that depression predicts the onset of obesity and vice versa (Sachs-Ericsson et al, 2007; De Wit et al, 2009; Luppino et al, 2010). Current hypotheses about the predisposition of depressive people to obesity include lifestyle factors and direct effects of psychotropic drugs (Stunkard et al, 2003; Schwartz et al, 2004). Factors that are hypothesized to predispose to depression in the obese include stigma, a negative body image and disappointment about failing diets, as well as activity limitations (Dixon et al, 2003).

In the present chapter, two important limitations of previous studies will be examined more in depth: different measures of obesity and the subtyping of depressive symptoms. In most studies obesity is defined based on the BMI. With increasing age, BMI is less accurate because it does not account for changes in body composition and loss of height, which may lead to an underestimation or overestimation of fatness (Villareal et al, 2005). This is especially relevant because increases in visceral adipose tissue primarily determine the risk of obesity leading to cardiovascular disease (Nicklas et al, 2004). The most convenient anthropometric indices of visceral adipose tissue are the waist-to-hip ratio (WHR) and waist circumference (WC).

Another issue is the subtyping of depression. In patients with depression and CVD, the somatic-affective symptom cluster of depression, but not the cognitive-affective symptom cluster, was related to subsequent vascular events (de Jonge et al, 2006). Similarly, one may hypothesise, that the somatic-affective symptoms of depression are more strongly related to the WHR and WC than the cognitive-affective symptoms.

The objective of the present study was to explore the relationship between depressive
symptoms and obesity in a community-based sample aged 50 through 70 years, taking into account different measures of obesity (BMI, WHR, and WC) as well as the different symptom clusters within the concept of depression. It was hypothesized first that depressive symptoms in later life are associated with obesity and second that visceral measures of obesity are more strongly related to somatic-affective depressive symptoms than to cognitive-affective depressive symptoms.

**Methods**

**Sample**

The present sample was drawn from the Nijmegen Biomedical Study (NBS), a population-based survey conducted in the eastern part of The Netherlands of people aged 20 through 90 years (Hoogendoorn et al, 2006). In 2004 and 2005, a questionnaire was sent to all participants (n=2807) aged 50 to 70; 1517 (54%) of these gave additional informed consent to participate in a study on atherosclerosis. These participants visited the hospital for a detailed assessment of atherosclerotic disease, its risk factors and consequences (Holewijn et al, 2010). This latter group was considered eligible for the present study. The Medical Ethics Committee of the Radboud University Nijmegen Medical Centre approved the study protocol.

**Variables of interest**

**Obesity** - A trained staff member measured obesity according to a predefined protocol. Weight and height were measured and Body Mass Index (BMI) was calculated as body weight (kg) divided by the square of height (m²). Waist circumference was measured at the level of the umbilicus, hip circumference was measured at the level of the greater trochanter and WHR was calculated.

**Depressive symptoms** - Depressive symptoms were measured using the Beck Depression Inventory I (BDI-I). The BDI-I is a 21-item self-report questionnaire with excellent psychometric characteristics (Beck et al, 1987). Each item is rated on a 0 to 3 scale, with 0 representing ‘absence’ and 1-3 representing increasing levels of severity of the symptom. The BDI-I yields a total score ranging from 0 to 63. Based on previous research in this area, a sum score of 10 or greater is indicative of clinically significant depressive symptoms (Pizzi et al, 2008).

**Covariates**

In addition to age and sex, the following potential confounders were a priori considered...
based on their relationship with depressive symptoms and obesity. 
The first set of confounders included lifestyle factors such as smoking, the use of alcohol, physical activity and use of psychotropic drugs known to affect body weight (Simon et al, 2008). Smoking was based on self-reported information and defined as current smoking (yes/no) and formerly smoking (yes/no). Use of alcohol was based on the number of standardized units per week. Excessive use (> 21 drinks/week for men, >14 drinks/week for women) was corrected for. Physical activity was based on the number of exercise sessions per week of more than 30 minutes moderate to vigorous activity (Stampfer et al, 2000) and dichotomized as 0 or 1 moment versus 2 more sessions. The use of psychotropic drugs was based on self-report data regarding the previous month. People were instructed to collect medication containers first before filling in this questionnaire. We considered the use of antidepressants, lithium and antipsychotic drugs as psychotropic drugs influencing body weight (Schwartz et al, 2004).

The second set of confounders included somatic co-morbidity. Based on its relationship with obesity, diabetes mellitus and cardiovascular disease were evaluated separately. Diabetes mellitus (DM) was defined as a glucose level of 7.0 mmol/litre or higher after an overnight fast or previously diagnosed and treated DM. Cardiovascular disease (CVD) status was assessed during the research visit at the hospital and defined as a self-reported history of myocardial infarction, transient ischemic attack, stroke, peripheral arterial disease, coronary artery bypass /angioplasty, or treated angina pectoris. Other somatic co-morbidity was lumped together and coded as present/absent.

**Statistical methods**

Because the BDI sum score had a skewed distribution in the sample, a log-transformation was applied to obtain a normal distribution. All further analyses were conducted using the log-transformed sum score. The BMI, WHR and WC were normally distributed. Principal components analysis (PCA) was conducted on the 21 individual BDI items to obtain fewer factors or components while retaining the original item information. PCA was selected rather than factor analysis for 2 reasons: (1) its ultimate goal is to reduce data into components useful for other purposes; and (2) its superior ability to remedy multicolinearity between factors, should it exist (Costello et al, 2005). Varimax rotation was selected because it forces factors to be uncorrelated. Factor scores were calculated on the basis of unstandardized item factor loadings and transformed into standardized z scores (using the Anderson-Rubin method) to increase their interpretability.

The scree plot of eigenvalues and the number of complex items revealed a 2-factor solution as the optimal solution, comparable to the traditional 2-factor structure of the BDI (cognitive-affective vs somatic-affective symptoms) as well as a previous factor analyses of the BDI in a Dutch cardiac population (Kaiser-Meyer-Olkin measure of
Because a U-shape relationship between obesity and depressive symptoms can explain some inconsistencies between previous studies (de Wit et al, 2009), whether such a relationship existed in this sample was first checked by comparing the depressive symptom scores for four categories of BMI (underweight (BMI ≤20 kg/m²), normal weight (>20.0-≤25 kg/m²), overweight (>25.0-≤30 kg/m²), and obesity (>30 kg/m²). This resulted in the exclusion of 26 patients with underweight (see Results).

Linear regression analyses were conducted with obesity indices as the dependent variables. For each index of obesity (BMI, WHR, and WC), two different models were evaluated. Depressive symptoms were evaluated in the first model by including the log-transformed sum score of the BDI and in the second model by including the two factor scores yielded by the PCA together. All models were corrected for sex, age, life-style factors and somatic co-morbidity (as described above). Sex differences were explored by post-hoc analyses based on studies showing an association of obesity and depression in women only (Carpenter et al, 2000) or abdominal obesity and depression in men only (Herva et al, 2006).

All analyses were performed in SPSS version 17.0 (SPSS, Inc. Chicago, IL).

Results

Of the 1517 subjects who consented to participation in the study of non-invasive measurements of atherosclerosis, 233 participants were excluded, leaving a final study sample of 1284 people. Reasons for exclusion were not responding to the postal questionnaire containing the BDI (n=185); having 3 or more missing items on the BDI (n=37); violating the rules for a reliable measurement of atherosclerotic disease or its risk factors (having smoked before coming to the hospital, n=4; not obeying the fasting rule, n=2; not stopping their lipid lowering medication, n=5). See Table 2 for the clinical characteristics of this sample.

Included subjects (n=1284) did not differ from excluded subjects and subjects not giving additional informed consent (n=1523) with respect to age and sex, but had significantly less severe depressive symptoms (median BDI sum score 4 (Interquartile range (IQR): 2 – 8) vs 5 (IQR: 2 – 9); Z=-2.4, p=.018).
Table 1  Factor loadings of depressive symptom dimensions and relation to Beck Depression Inventory items and previous dimensional constructs.

<table>
<thead>
<tr>
<th>Items of the Beck Depression Inventory</th>
<th>Original Beck &amp; Steer dimensional structure</th>
<th>Dimensional structure De Jonge et al (2006)</th>
<th>Dimensional structure present study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cognitive - Affective factor</td>
<td>Somatic - Affective factor</td>
<td>Appetitive factor</td>
</tr>
<tr>
<td>Sadness</td>
<td>Cognitive</td>
<td>0.45</td>
<td>0.64</td>
</tr>
<tr>
<td>Pessimism</td>
<td>Cognitive</td>
<td>0.58</td>
<td>0.56</td>
</tr>
<tr>
<td>Sense of failure</td>
<td>Cognitive</td>
<td>0.66</td>
<td></td>
</tr>
<tr>
<td>Dissatisfaction</td>
<td>Cognitive</td>
<td>0.49</td>
<td>0.69</td>
</tr>
<tr>
<td>Guilt</td>
<td>Cognitive</td>
<td>0.70</td>
<td></td>
</tr>
<tr>
<td>Punishment</td>
<td>Cognitive</td>
<td>0.59</td>
<td></td>
</tr>
<tr>
<td>Self-dislike</td>
<td>Cognitive</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>Self-accusinations</td>
<td>Cognitive</td>
<td>0.71</td>
<td></td>
</tr>
<tr>
<td>Suicidal ideas</td>
<td>Cognitive</td>
<td>0.49</td>
<td></td>
</tr>
<tr>
<td>Crying</td>
<td>Cognitive</td>
<td></td>
<td>0.52</td>
</tr>
<tr>
<td>Irritability</td>
<td>Cognitive</td>
<td></td>
<td>0.45</td>
</tr>
<tr>
<td>Social withdrawal</td>
<td>Cognitive</td>
<td>0.51</td>
<td>0.42</td>
</tr>
<tr>
<td>Indecisiveness</td>
<td>Cognitive</td>
<td>0.40</td>
<td>0.68</td>
</tr>
<tr>
<td>Body image change</td>
<td>Somatic</td>
<td></td>
<td>0.57</td>
</tr>
<tr>
<td>Work difficulty</td>
<td>Somatic</td>
<td></td>
<td>0.69</td>
</tr>
<tr>
<td>Insomnia</td>
<td>Somatic</td>
<td></td>
<td>0.55</td>
</tr>
<tr>
<td>Fatigability</td>
<td>Somatic</td>
<td></td>
<td>0.58</td>
</tr>
<tr>
<td>Loss of appetite</td>
<td>Somatic</td>
<td></td>
<td>0.42</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Somatic</td>
<td></td>
<td>0.66</td>
</tr>
<tr>
<td>Somatic preoccupation</td>
<td>Somatic</td>
<td></td>
<td>0.67</td>
</tr>
<tr>
<td>Loss of libido</td>
<td>Somatic</td>
<td></td>
<td>0.50</td>
</tr>
</tbody>
</table>

First, the existence of a U-shaped relationship between obesity and depressive symptoms was examined by comparing the severity of depressive symptoms within four categories of weight. The analysis of covariance using the log-transformed BDI score and corrected for all potential confounders yielded significant overall effect (F=3.8, df=3, p=.01), showing significantly higher depressive symptoms in the obese category than in the normal weight (LSD post-hoc, p=.001) and overweight category (LSD post-hoc, p=.040), but not the underweight category (LSD post-hoc, p=.96). Similar results were found with respect with the somatic-affective depressive symptom cluster, whereas no significant relationship was found with the cognitive-affective symptom cluster. Given this U-shaped relationship between depression and BMI, and the interest
in examining the relationship between depression and obesity, the 26 underweight patients were removed from further analyses.

The Pearson correlation coefficient (r) of the BDI sum score (after log-transformation) was 0.42 with the cognitive-affective symptom cluster (p<.001) and 0.81 with the somatic-affective symptoms cluster (p<.001). The different measures of obesity were

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**Table 3**  Fully adjusted multivariate regression analyses of depressive symptoms on different measures of obesity

<table>
<thead>
<tr>
<th></th>
<th>Obesity</th>
<th>Visceral obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Body Mass Index</td>
<td>Waist circumference</td>
</tr>
<tr>
<td></td>
<td>β</td>
<td>P value</td>
</tr>
<tr>
<td><strong>Whole sample (n=1258)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Continuous BDI score (log10)</td>
<td>.12</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive BDI score</td>
<td>.08</td>
<td>.008</td>
</tr>
<tr>
<td>Somatic BDI score</td>
<td>.10</td>
<td>.001</td>
</tr>
<tr>
<td><strong>Men (n=625)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Continuous BDI score (log10)</td>
<td>.09</td>
<td>.027</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive BDI score</td>
<td>.06</td>
<td>.15</td>
</tr>
<tr>
<td>Somatic BDI score</td>
<td>.08</td>
<td>.063</td>
</tr>
<tr>
<td><strong>Women (n=633)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Continuous BDI score (log10)</td>
<td>.14</td>
<td>.001</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive BDI score</td>
<td>.09</td>
<td>.023</td>
</tr>
<tr>
<td>Somatic BDI score</td>
<td>.12</td>
<td>.004</td>
</tr>
</tbody>
</table>

* Linear regression analyses adjusted for age, smoking (yes/no), alcohol (excessive usage yes/no), sports (yes/no), and usage of psychotropic drugs (yes/no), diabetes mellitus (DM), cardiovascular disease (CVD), chronic co-morbidity other than DM/CVD (yes/no). BDI= Beck Depression Inventory
strongly correlated with each other; Pearson correlations for BMI and WC 0.80 (p<.001), BMI and WHR 0.38 (p<.001) and WC and WHR 0.78 (p>.001).

As shown in Table 3, the BMI was significantly associated with the BDI sum score (Model 1) and with both the cognitive-affective and the somatic-affective symptom clusters (Model 2). With regard to the measures representing visceral obesity, significant associations were found between WC and WHR and the BDI sum score (Model 1). WC and WHR were specifically associated with the somatic-affective symptom cluster, but not the cognitive-affective symptom cluster (Model 2).

Including interaction terms between all measures of depressive symptoms with sex did not yield significant interaction terms (all p-values > .23). Although the differences between men and women were not statistically different, post hoc analyses showed larger effect sizes for women than for men in all depressive symptoms measures in relation to the BMI and WC, but not the WHR (see Table 3).

Discussion

Main findings
In line with previous findings (de Wit et al, 2009) a U-shaped relationship was found between BMI and depressive symptoms in a population-based sample of people aged 50 to 70. Excluding underweight patients, a positive correlation was found between all different measures of depressive symptoms and BMI, although measures of visceral obesity (WC and WHR), were specifically associated with the somatic-affective symptom cluster. This relationship may suggest an organic etiology of these symptoms of depression.

Relation between obesity (BMI) and depressive symptoms
Recent studies also reported a positive association between depressive symptoms and body mass index (Stunkard et al, 2003; Sachs-Ericsson et al, 2007; de Wit et al, 2010), whereas older studies and studies in specific subgroups defined by ethnicity, sex, age or chronic illness, did not find a relationship or even reported a negative association (Carpenter et al, 2000; Ho et al, 2008). Several explanations can be put forward to explain these discrepancies. First, ignoring the U-shaped association between obesity and depression may easily lead to the conclusion that there is an absent or inverse relationship between depression and obesity. The U-shaped association seems particularly important in the oldest old people because frailty and co-morbidity are associated with depressive symptoms as well as weight loss (Andrew et al, 2007). Second, cultural
aspects and gender differences may account for the psychological sequelae of being overweight. In (some) Eastern cultures obesity is regarded a positive characteristic. In Chinese elderly people, for example, a higher BMI was associated with fewer depressive symptoms (Ho et al, 2008). The opposite seems true for Western countries. Furthermore, in Western societies sex differences may also contribute to sex-specific findings because women are more likely to be stigmatized for being overweight or obese than are men.

Relation between obesity (BMI) and depressive symptom clusters
Separating depressive symptoms into a cognitive-affective and somatic-affective symptom cluster shows that the cognitive symptoms and the somatic symptoms of depression contribute to the relationship between obesity and depressive symptoms. The somatic symptoms accompanying obesity (Dixon et al, 2003) may easily be mixed up with the somatic-affective criteria (fatigue, lack of energy, disturbances in sleep or appetite) of the diagnosis of depression. In the current sample, the somatic-affective symptoms did not entirely explain the relationship between the BMI and general depressive symptoms, because the cognitive-affective symptoms have a significant and independent effect. This is consistent with a recent survey about the association between obesity and depression in middle-aged women that found a strong relationship between obesity and more psychological symptoms such as depressed mood, feelings of guilt or suicidal ideation (Simon et al, 2008). The relationship between BMI and cognitive-affective symptoms may be expected to differ according to gender. In Western cultures, an ideal of slimness in women is associated with elegance, attractiveness, self-control and youth. Studies of self-ratings of attractiveness show that overweight men and women rate themselves as less attractive than to men and women with normal BMI, although men in higher BMI ranges still rate themselves more attractive than do women in these BMI ranges (Weeden et al, 2005). In the current study, the relationship between BMI and cognitive-affective symptoms reached significance only in women.

Relation between visceral obesity and different measures of depression
Depressive symptoms were positively associated with visceral obesity, a more specific risk factor for cardiovascular disease than BMI (Weber-Hamann et al, 2006). Therefore, visceral obesity might be a pathway relating depression to greater incidence rates of cardiovascular disease (Everson-Rose et al, 2009). This is consistent with recent studies showing that obesity and dyslipidemia components of the metabolic syndrome in particular are predictive of depressive symptoms (Akbaraly et al, 2009). Because metabolic disorders and depression are both associated with low-grade systemic inflammation, inflammation may be the mediating mechanism. Inflammation is associated
with the features of sickness behavior, including general weakness, painful symptoms, inability to concentrate and fatigue (Dantzer et al, 2008), that are similar to somatic symptoms in depression.

The somatic-affective symptom cluster rather than the cognitive-affective symptom cluster was related to visceral obesity. Several explanations for this can be put forward. From a psychological perspective, one may hypothesize that feeling guilty about uncontrolled eating habits or inability to keep to a strict diet may be more strongly associated with higher weight (BMI) than with having a “pot-belly” (WC). From a somatic perspective, it may be hypothesized that somatic-affective symptoms are primarily due to a (subclinical) somatic condition or that these symptoms reflect somatic health rather than mental health. The diagnostic criteria of depression would then at least partly confound the prospective role of depression as risk factor for cardiovascular disease (Nicholson et al, 2006). A final potential explanation is that persons with somatic depressive symptoms represent a different subtype of depression. Nevertheless, a recent study showed that depressive symptoms in older people are associated with an increase of visceral fat deposits over time (Vogelzangs et al, 2008). Because these authors made no distinction between specific depressive symptom clusters, it is unknown whether this finding was specifically related to the somatic-affective symptoms of depression and may have been caused by underlying illnesses (e.g. diabetes mellitus).

Methodological considerations
Some limitations of this study have to be acknowledged for proper interpretation. First, the cross-sectional design limits causal interpretation of our findings. In addition to a causal relationship between obesity and depressive symptoms, an underlying latent variable, such as childhood abuse or genetics, may explain both variables (Stunkard et al, 2003), although the first studies examining a common genetic basis have yielded negative results (Choy et al, 2009).

Second, being a population-based survey some selection might have occurred towards the healthier part of the population. This may account for the relatively low severity of depressive symptoms. Therefore, the findings apply to persons with low depressive symptoms but might not be generalizable to persons with major depressive disorder. Nonetheless, 16.7% of the sample scored on BDI above the cut-off of 10 (Pizzi et al, 2008), whereas meta-analyses show that 13.5% of the population suffers from clinically relevant depressive symptoms, compared to only 9.8% population frequency of minor depression and 1.8% frequency of major depression based on DSM-IV research (Beekman et al, 1999). Although these data suggest a continuum from depressive symptoms to depressive disorders, it is not clear whether this assumption holds true (Beekman et al, 1999). Finally, Computer Tomography was not used at the level of the fourth lumbar

**Clinical implications and future research**
Although correlations were of limited size, further research should explore the clinical implications of obesity in patients with depression. Because depression is associated with poorer adherence, interventions for medical problems in obese patients may benefit from effective concurrent treatment of depression (Carpenter et al, 2000). Furthermore, the treatment of obesity itself, including gastric bypass surgery, leads to a decrease in depression (Stunkard et al, 2003). Alternatively the treatment of depression with pharmacotherapeutic agents is often associated with weight gain, whereas cognitive-behavioral therapy is not (Stunkard et al, 2006). In case of co-morbid obesity and depressive symptoms, treatment programs could include a physical exercise program, because this enhances muscle and skeletal strength, decreases obesity and positive affects depression.
References


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