Depression: an important comorbidity with metabolic syndrome in a general population

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Objective: There is a recognised association between depression, diabetes and cardiovascular disease. The aim of this study was to examine in a sample representative of the general population whether depression, anxiety and psychological distress are associated with metabolic syndrome and its components.

Research Design and Methods: Three cross-sectional surveys including clinical health measures were completed in rural regions of Australia during 2004-06. A stratified random sample (n=1690, response rate 48%) of men and women, aged 25-84 years, was selected from the electoral roll. Metabolic syndrome was defined by the Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, Adult Treatment Panel III (NCEP ATP III) and International Diabetes Federation (IDF) criteria. Anxiety and depression were assessed by the Hospital Anxiety and Depression Scale and psychological distress by the Kessler 10 measure.

Results: Metabolic syndrome was associated with depression but not psychological distress or anxiety. Participants with metabolic syndrome had higher scores for depression (n=409, mean score 3.41, 95% CI 3.12-3.70) than those without metabolic syndrome (n=936, mean 2.95, 95% CI 2.76-3.13). This association was also present in 338 participants with metabolic syndrome and without diabetes (mean score 3.37, 95%CI 3.06-3.68). Large waist circumference and low HDL-cholesterol showed significant and independent associations with depression.

Conclusions: Our results show an association between metabolic syndrome and depression in a heterogeneous sample. The presence of depression in individuals with metabolic syndrome has implications for clinical management.
Recent definitions of metabolic syndrome (1,2) specify the following quantitative criteria: large waist circumference, high blood pressure, dyslipidaemia (high triglycerides and low HDL-cholesterol) and fasting hyperglycaemia with underlying insulin resistance as the likely mechanism. The combination of these components is a strong predictor of cardiovascular disease and type 2 diabetes. Understanding the mechanisms involved and factors associated with metabolic syndrome is of great interest given the pandemic of obesity and increasing prevalence of metabolic syndrome (32% in the U.S. adult population in 1999-2000 (3) using the Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, Adult Treatment Panel III (NCEP ATP III) (1) criteria and 28% in our region(4)).

There is an increasing interest in the association between metabolic syndrome and depression, and whether causal relationships are involved. The proposed link is consistent with reports that depression is associated with development of diabetes and with poor glycaemic control in established diabetes (5). For instance, Björntorp has hypothesised that psychological problems are associated with metabolic disorders via visceral fat accumulation (6). The postulated role of the hypothalamic-pituitary-adrenal (HPA) axis in the pathogenesis of central adiposity and metabolic syndrome has led to the conceptualisation of metabolic syndrome as a neuroendocrine disorder (7).

To investigate the link between metabolic syndrome and depression, several studies have been conducted with results generally supporting an association of metabolic syndrome with depression. However, the groups studied were not representative samples from the general population with metabolic syndrome, being either relatively young (8,9), men only (10), pre-menopausal women only (11,12), or a clinically targeted population (13)(see Table 1). Other important variations such as study design, psychological measures used, and definition of metabolic syndrome used, have led to inconsistent results.

To the best of our knowledge, this is the first study to assess evidence from a randomised sample of a heterogeneous population with a high prevalence of metabolic syndrome (4) to determine whether depression has an important association with metabolic syndrome.

**RESEARCH DESIGN AND METHODS**

**Participants**—Three cross-sectional surveys of cardiovascular disease risk factors and related health behaviour were carried out in south-eastern Australia (14) to obtain rural data for comparison with the existing urban data. The first survey was conducted in August–October 2004 in Limestone Coast (LC - South Australia), the second in February–March 2005 in Corangamite Shire (CO - Victoria), and the third in May-October 2006 in the Wimmera region (WI - Victoria). These regions are predominantly rural farming areas.

Each survey utilized a stratified random sample of the population aged 25 to 74 years drawn from the electoral roll. Stratification was by gender and ten-year age-groups with the exception of the combined 25–44 age-group considered as one stratum. The original samples consisted of 1,120 individuals in LC, 1,000 in CO and 1,500 in WI. After excluding individuals who had died or had left the region, a total of 552 persons in LC (participation rate 51%), 415 persons in CO (42%) and 596 persons in WI (53%) participated in the study. The WI sample included an additional 127 subjects.
(participation rate 44%) from the age-group 75-84 years.

The survey methodology, as previously described (4), comprised self-administered questionnaires, physical measurements and laboratory tests. A comparison of the socioeconomic background with population statistics available indicated that the participants closely resembled the true populations of the areas surveyed (4).

**Measures**—The questionnaire, which included questions on health behaviour, symptoms and diseases, medical history, socioeconomic background, and psychosocial factors, together with the invitation to attend the health check, was sent by mail to all selected participants. Health checks were carried out in local health centres or other survey sites by specially trained nurses.

In the health check, weight, height, waist and hip circumference, systolic and diastolic blood pressure as well as fasting lipids and glucose were ascertained, and body mass index (BMI) was computed as described in more detail elsewhere (4). The venous blood samples were drawn after an overnight fast of at least 10 hours and analysed at the Flinders Medical Centre Clinical Trials Laboratory which is internationally accredited for lipid measurement under the Centres for Disease Control Lipid Standardisation Program (Atlanta, Georgia, USA) (4).

**Metabolic syndrome definition**—The following definitions of metabolic syndrome were used:

a) The most recent NCEP ATP III (1) criteria require three or more of the following: waist circumference ≥102 cm for men and ≥88 cm for women; fasting glucose ≥5.6 mmol/L or on medication for high blood glucose; systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg or antihypertensive medication; triglycerides ≥1.7 mmol/L; HDL-cholesterol <1.03 mmol/L for men and <1.30 mmol/L for women.

b) The International Diabetes Federation (IDF) (2) criteria specify central obesity with waist circumference ≥94 cm for men and ≥80 cm for women of Europid origin, plus two or more of the following: fasting plasma glucose ≥5.6 mmol/L or previously diagnosed type 2 diabetes; systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg or antihypertensive medication; plasma triglycerides ≥1.7 mmol/L; plasma HDL-cholesterol <1.03 mmol/L for men and <1.29 mmol/L for women.

**Psychological measures**—Depression and anxiety were measured by the Hospital Anxiety and Depression Scale (HADS) (15). The instrument consists of seven items for anxiety (HADS-A) and seven for depression (HADS-D), reported by respondents over the last week. Responses are scored on items from 0 to 3: separate summed scores for anxiety and depression range from 0 to 21, where normal is 0 to 7, mild is 8-10, moderate is 11-14 and severe is 15-21. In the present study, we defined anxiety and depression as having a score in the mild to severe range (≥8).

Psychological distress was assessed by the Kessler-10 measure (K10) (16), a 10 item measure of the anxiety and depression symptoms experienced in the most recent four-week period. Responses are recorded on a five-point scale and the score is the sum of the responses. Total scores are categorised into two levels of psychological distress: low (10-15) and moderate-high (16-50). The internal consistency coefficients for the HADS-A, HADS-D and K-10 in this study were α=0.82, 0.79 and 0.87 respectively.

Ethics approval for this study was obtained from the Flinders University Clinical Research Ethics Committee. Informed consent in writing was obtained from
participants when they attended the health check component of the survey.

Statistical analyses—Statistical analyses were undertaken using SPSS version 14.0. Internal consistency was determined by using Cronbach’s alpha. Pearson correlation coefficients were used to assess the intercorrelations between depression, anxiety, and psychological distress. Pearson chi-square test was used to test the associations of depression, anxiety, and psychological distress with the presence of metabolic syndrome. Independent t-tests were used to compare mean age and alcohol consumption for patients with metabolic syndrome and for healthy subjects. Multivariate analysis of covariance was used to test differences between those with metabolic syndrome and those without metabolic syndrome for psychological distress, anxiety, and depression. Analysis of covariance was used to examine the association between depression and the five components of metabolic syndrome simultaneously. Analyses were adjusted for age, gender, smoking status, alcohol intake, physical activity, marital status and education. Non-significant covariates were not included in the final models.

RESULTS

For 1,345 men and women aged 25-84 information was available for metabolic syndrome, HADS-D, HADS-A, K-10, smoking status, alcohol intake, and physical activity (Table 2). A total of 409 (30.4%) participants met the NCEP ATPIII criteria (1) for metabolic syndrome. Ninety participants (6.7%) had diabetes (based on self-reported diabetes or fasting glucose greater than or equal to 7.0 mmol/L): 71 (5.3%) with and 19 (1.4%) without metabolic syndrome.

The characteristics of the 409 participants with and 936 without metabolic syndrome are presented in Table 2 which also shows the characteristics of the 338 subjects without diabetes who had metabolic syndrome.

In comparing all 409 metabolic syndrome participants with those without metabolic syndrome, no gender based prevalence differences were found. Participants with metabolic syndrome were older (mean 60.5 vs. 55.0 years, p<0.001). The correlations between depression (HADS-D) and anxiety (HADS-A), depression and psychological distress (K-10) and anxiety and psychological distress were 0.59 (95% CI 0.56-0.63), 0.66 (95% CI 0.63-0.69) and 0.72 (95% CI 0.69-0.74) respectively, and were all significant (p<0.001). Participants with metabolic syndrome were more likely to have moderate to severe depression (10% vs. 6.9%, p=0.069); but the two groups were not significantly different in psychological distress (30.1% vs. 25.7%, p=0.115) or anxiety (9.8% vs. 10.4%, p=0.820).

Multivariate analysis showed that participants with metabolic syndrome by NCEP ATP III criteria (1) had higher scores for depression compared with those without metabolic syndrome (mean scores 3.41 vs. 2.95, p=0.013, Table 3) after adjusting for gender, smoking status, alcohol intake and physical activity.

When each of the components of metabolic syndrome was considered for all participants, both the HDL-cholesterol and waist circumference components were independently associated with depression (Table 3). Participants with lower HDL-cholesterol had higher scores for depression compared with those with higher HDL-cholesterol (mean scores 3.75 vs. 2.93, p=0.003). Participants with a larger waist circumference had higher scores for depression than those with smaller waist circumference (mean scores 3.38 vs. 2.86, p=0.002).

The 338 participants with metabolic syndrome but without diabetes were similarly more likely to have moderate to severe
depression (10.1% vs. 6.9%, p=0.086, table 2). Again no significant differences between
groups were found in psychological distress 
(30.5% vs. 25.7%, p=0.108) or anxiety (9.2% 
vs. 10.4%, p=0.604). Association of low HDL 
cholesterol (mean depression scores 3.68 vs. 2.92, p=0.004) and large waist circumference 
(mean depression scores 3.36 vs. 2.86, 
p=0.003) with depression were also found 
(Table 3).

Similar results were obtained when 
using IDF criteria (2). Participants (n=409) 
with metabolic syndrome had higher scores 
for depression than those without metabolic 
syndrome (mean scores 3.30 vs. 2.95, 
p=0.035, data not shown) after controlling for 
covariates. In the group of 338 participants 
with metabolic syndrome but without 
diabetes, we obtained the same association 
when using the IDF criteria (mean scores 3.27 
vs. 2.95, p=0.070, data not shown). Metabolic 
syndrome was associated with depression, 
anxiety, and psychological distress in the 409 
participants with metabolic syndrome 
(p=0.009) as well as the 338 participants 
without diabetes (p=0.032) when combined in 
multivariate analyses and adjusted for the 
same covariates.

CONCLUSIONS

In the present study, we have 
demonstrated an association between 
metabolic syndrome and depression. 
Although the association is modest, it is 
important because of the increasing 
prevalence of metabolic syndrome, and the 
effect that depression can have on the ability 
of patients to successfully make lifestyle 
changes and comply with medication required 
for hypertension and dyslipidemia.

The association is demonstrated here 
in a general population to our knowledge for 
the first time, whereas earlier studies (Table 
1) used subgroups of populations (8-13, 17). 
This association between metabolic syndrome 
and depression was present regardless of the 
diabetes status. This distinction is important 
because many individuals with metabolic 
syndrome have diabetes, which itself is 
known to be associated with depression (5).

Metabolic syndrome has been defined 
in several ways that involve quantitative 
anthropometric, clinical and laboratory 
measurements (1, 2). For the primary 
assessment, we chose NCEP ATP III (1) 
criteria, as these were used in most of the 
previously reported studies (8,9,11-13,17). In 
addition, we used the more recently described 
IDF criteria (2), with a lower cut-off point for 
waist circumference, and showed that the 
association was consistent across both 
definitions.

Of the components of metabolic 
syndrome, increased waist circumference was 
associated with depression as reported in 
another study using a restricted population 
sample (9). This association is present 
regardless of the diabetes status and remained 
even when adjusted for significant covariates. 
We also found a significant independent 
relationship between low HDL-cholesterol 
level and depression (Table 3). This finding 
appears to be consistent with other recent 
research (17) but the underlying mechanism 
remains unknown.

There has been little consistency in the 
psychological tests used in previous studies to 
measure depression (Table 1). Assessment 
varyes from individual interview instruments 
(e.g. The Structured Clinical Interview for 
DSM Disorders or the Hopkins Symptom 
Checklist) to self-reported epidemiological 
measures (e.g. Centre for Epidemiological 
Studies-Depression Scale) (Table 1). Most 
measures include somatic components of 
depression, and the length of recall ranges 
from present symptoms to past months. 
Symptoms are recorded in either intensity or 
presence. In this study, we used the HADS, 
which has been designed for and validated in 
medical patients. It measures the presence of 
cognitive and affective components of
depression and, unlike other instruments, excludes many of the somatic symptoms of depression (fatigue, loss of appetite and weight, sleep disturbance, psychomotor changes), that may overlap with physical problems. In addition, the length of recall is limited to the past week. The HADS is widely used in population health studies and screening in primary care, although it has only been used in one reported study (13) linking metabolic syndrome and depression.

The patho-physiological basis for the association between metabolic syndrome and depression is likely to be complex and to involve the inflammatory state that has been described as a consequence of central obesity (18). Björntorp (7) has postulated that psychosocial factors, including depression, can activate the hypothalamic-pituitary-adrenal (HPA) axis producing hypersecretion of corticotrophin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH) and cortisol. This dysregulation of the HPA axis promotes deposition of visceral adipose tissue (6), which secretes inflammatory cytokines such as Interleukins-1 and -6 (IL-1, IL-6) and tumor necrosis factor-alpha (TNF-α) (19,20). Both IL-6 and TNF-α have been implicated in insulin resistance, which is considered to be the key factor in the metabolic abnormalities (21) of the metabolic syndrome. The pro-inflammatory response associated with depression may also have a direct effect on dyslipidaemia (22). An alternative construct of the link between metabolic syndrome and depression places development of central obesity and activation of inflammatory processes as the initiating step. Depression is seen as being a consequence of this immune activation (23). In this model, development of depression is analogous to “sickness behaviour” that can be associated with viral infection or other causes of immune activation. Dysregulation of the HPA axis can occur via cytokine-induced stimulation of the central noradrenergic stress system (24).

The main limitation of our study is the cross-sectional design, which does not allow for the demonstration of the existence of HPA axis activation and an inflammatory state in participants with central obesity. Establishment of this link requires both longitudinal investigation and further analysis of blood samples which would allow direct examination of the link between depression, inflammatory state and metabolic syndrome. Another limitation of this study is the rural population; the association still needs to be demonstrated in urban groups as well as those that have greater cultural and socio-economic diversity. While the characteristics of participants in our study closely resembled those of the local populations surveyed (4) it is possible that depressed individuals may have been less likely to participate. If this is the case the present findings could over- or under-state the association.

In summary, our data show an association between metabolic syndrome and the cognitive and affective components of depression in a general population, where the prevalence of depression in those with metabolic syndrome is 50% higher. The importance of our study lies in the heterogeneity of the sample used. Contrasting other studies, conclusions we have made are unlikely to be attributed to idiosyncrasies of the sample. Based on the findings in this study, awareness of depressive symptoms as part of metabolic syndrome could be important in clinical management as in other chronic diseases. Acknowledgement of depressive symptoms by the practitioner and the patient should improve ability to undertake lifestyle changes with adjustment of physical activity and food intake, as well as adherence to medication which are likely to be compromised by depression. Identification and management of depression should therefore precede or accompany other measures in the management of metabolic syndrome. It is also possible that treatment of
metabolic syndrome with lifestyle changes will ameliorate depression through reduction of visceral adiposity and inflammation. Intervention studies to address this hypothesis could provide further insight into the relationship between depression, central obesity and inflammation.

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Professor Kerin O’Dea, Ms Anna Chapman, Ms Anna Kao-Philpot, Dr Andrew Baird, the nurses carrying out the survey and regional hospitals providing facilities for the study.

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REFERENCES


Table 1. Studies of depression and metabolic syndrome

<table>
<thead>
<tr>
<th>Authors</th>
<th>n</th>
<th>gender</th>
<th>Age range (years)</th>
<th>Depression measure</th>
<th>Metabolic syndrome measure</th>
<th>Main outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herva et al.(9)</td>
<td>5698</td>
<td>men and women</td>
<td>31 mean</td>
<td>HSCL-25</td>
<td>ATP III</td>
<td>No clear association between metabolic syndrome and psychological distress.</td>
</tr>
<tr>
<td>Kinder et al.(8)</td>
<td>6189</td>
<td>men and women</td>
<td>17-39</td>
<td>SCID</td>
<td>ATP III</td>
<td>Association between metabolic syndrome and depression in women only. High BP and high TG associated with depression.</td>
</tr>
<tr>
<td>McCaffery et al.(10)</td>
<td>173</td>
<td>Twin men</td>
<td>≥45</td>
<td>CES-D</td>
<td>#</td>
<td>Small association between metabolic syndrome and depression (participants with self-reported diabetes excluded).</td>
</tr>
<tr>
<td>Miller et al.(25)</td>
<td>100</td>
<td>men and women</td>
<td>18-45</td>
<td>HAM-D; BDI</td>
<td>#</td>
<td>Evidence linking depressive symptoms with inflammatory processes as part of the mechanism for cardiovascular morbidity and mortality.</td>
</tr>
<tr>
<td>Raikkonen et al.(11)</td>
<td>425</td>
<td>women</td>
<td>42-50 (at study entry)</td>
<td>BDI</td>
<td>ATP III</td>
<td>Depression, anxiety, tension and anger are associated concurrently with and/or predict the risk for developing metabolic syndrome.</td>
</tr>
<tr>
<td>Raikkonen et al.(12)</td>
<td>432</td>
<td>women</td>
<td>middle-aged</td>
<td>BDI</td>
<td>WHO; ATP III; IDF</td>
<td>Depressive symptoms associated with the cumulative prevalence and risk for developing metabolic syndrome for all criteria used.</td>
</tr>
<tr>
<td>Vozelgang et al.(17)</td>
<td>867</td>
<td>men and women</td>
<td>≥65</td>
<td>CES-D</td>
<td>ATP III</td>
<td>Synergistic relationship between depression, cortisol and metabolic syndrome.</td>
</tr>
<tr>
<td>Skilton et al.(13)</td>
<td>1598</td>
<td>men and women</td>
<td>30-80</td>
<td>HADS-D</td>
<td>ATP III; IDF</td>
<td>Association between metabolic syndrome and depression in a cohort of subjects at an increased risk of cardiovascular disease.</td>
</tr>
</tbody>
</table>

#: Authors were not using any of the defined criteria of metabolic syndrome but were analysing clusters of metabolic factors

1 Longitudinal in design

ATP III: Third Report of the National Cholesterol Education Program (Adult Treatment Panel); IDF: International Diabetes Federation; WHO: World Health Organisation; BP: Blood pressure; TG: Triglycerides
### Table 2. Sample characteristics of participants aged 25-84

<table>
<thead>
<tr>
<th>Overall Metabolic Syndrome</th>
<th>n (%)</th>
<th>n (%)</th>
<th>p-value&lt;sup&gt;3&lt;/sup&gt;</th>
<th>n (%)</th>
<th>p-value&lt;sup&gt;3&lt;/sup&gt;</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>A vs. B</td>
<td>C</td>
<td>A vs. C</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>Yes – Diabetes included</td>
<td></td>
<td>Yes – Diabetes excluded</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>936 (100.00)</td>
<td>409 (100.0)</td>
<td>0.306</td>
<td>338 (100.0)</td>
<td>0.338</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>446 (47.6)</td>
<td>208 (50.9)</td>
<td>172 (50.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>490 (52.4)</td>
<td>201 (49.1)</td>
<td>166 (49.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes (self-reported or fasting glucose ≥ 7.0 mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>19 (2.0)</td>
<td>71 (17.4)</td>
<td>&lt;0.001</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Age (years)</td>
<td>55.0 (13.1)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>60.5 (10.8)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>&lt;0.001</td>
<td>59.8 (10.9)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>132 (14.1)</td>
<td>49 (12.0)</td>
<td>34 (10.1)</td>
<td></td>
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<tr>
<td></td>
<td>303 (32.4)</td>
<td>150 (36.7)</td>
<td>111 (32.8)</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>501 (53.5)</td>
<td>210 (51.3)</td>
<td>193 (57.1)</td>
<td></td>
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</tr>
<tr>
<td>Alcohol (g/week)</td>
<td>7.4 (11.6)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>6.6 (10.6)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.196</td>
<td>6.9 (10.9)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.442</td>
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<tr>
<td>Daily Physical Activity (&gt;30 minutes)</td>
<td>767 (81.9)</td>
<td>314 (76.8)</td>
<td>0.034</td>
<td>261 (77.2)</td>
<td>0.071</td>
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<tr>
<td>Depression</td>
<td>65 (6.9)</td>
<td>41 (10.0)</td>
<td>0.069</td>
<td>34 (10.1)</td>
<td>0.086</td>
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<tr>
<td>Anxiety</td>
<td>97 (10.4)</td>
<td>40 (9.8)</td>
<td>0.820</td>
<td>31 (9.2)</td>
<td>0.604</td>
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<td>Psychological Distress</td>
<td>241 (25.7)</td>
<td>123 (30.1)</td>
<td>0.115</td>
<td>103 (30.5)</td>
<td>0.108</td>
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</table>

### Metabolic Syndrome Components

<table>
<thead>
<tr>
<th></th>
<th>n (%)</th>
<th>n (%)</th>
<th>p-value&lt;sup&gt;3&lt;/sup&gt;</th>
<th>n (%)</th>
<th>p-value&lt;sup&gt;3&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated Fasting Glucose&lt;sup&gt;2&lt;/sup&gt;</td>
<td>112 (12.1)</td>
<td>247 (60.1)</td>
<td>&lt;0.001</td>
<td>176 (52.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypertension&lt;sup&gt;2&lt;/sup&gt;</td>
<td>441 (47.1)</td>
<td>374 (91.4)</td>
<td>&lt;0.001</td>
<td>306 (40.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Elevated Triglycerides&lt;sup&gt;2&lt;/sup&gt;</td>
<td>129 (13.9)</td>
<td>281 (71.1)</td>
<td>&lt;0.001</td>
<td>239 (72.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low HDL-cholesterol&lt;sup&gt;2&lt;/sup&gt;</td>
<td>67 (7.2)</td>
<td>194 (47.8)</td>
<td>&lt;0.001</td>
<td>158 (46.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Central Obesity&lt;sup&gt;2&lt;/sup&gt;</td>
<td>226 (24.3)</td>
<td>351 (86.2)</td>
<td>&lt;0.001</td>
<td>288 (85.5)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<sup>1</sup> Mean (SD)

<sup>2</sup> Cut-offs are for NCEP ATP III criteria as defined in the methods section

<sup>3</sup> p-values are obtained by chi-square or independent t-test as appropriate

A: participants without metabolic syndrome; B: participants with metabolic syndrome including those with T2DM; C: participants with metabolic syndrome excluding those with T2DM. n/a: not available
Table 3. Results of analysis of variance for mean HADS-Depression scores by metabolic syndrome (NCEP ATP III) and its components for participants aged 25-84

<table>
<thead>
<tr>
<th></th>
<th>Including participants with both metabolic syndrome and diabetes (n=1345)</th>
<th>Excluding participants with both metabolic syndrome and diabetes (n=1274)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (95% CI)</td>
<td>Mean (95% CI)</td>
</tr>
<tr>
<td><strong>Overall Metabolic Syndrome</strong></td>
<td>Yes 3.41 (3.12-3.70)</td>
<td>No 2.95 (2.76-3.13)</td>
</tr>
<tr>
<td><strong>Components:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting Glucose</td>
<td>Abnormal 3.14 (2.84-3.44)</td>
<td>Normal 3.06 (2.88-3.24)</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Abnormal 3.18 (2.98-3.38)</td>
<td>Normal 2.94 (2.70-3.19)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>Abnormal 3.17 (2.89-3.45)</td>
<td>Normal 3.04 (2.85-3.22)</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>Abnormal 3.75 (3.37-4.13)</td>
<td>Normal 2.93 (2.76-3.10)</td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>Abnormal 3.38 (3.14-3.63)</td>
<td>Normal 2.86 (2.66-3.06)</td>
</tr>
</tbody>
</table>