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Research Letter

Body composition in subtypes of depression – a population-based survey

Introduction

Obesity and mood disorders are major public health problems, which seem to share pathophysiological pathways (Björntorp, 2001; Anisman, 2009). In longitudinal studies obesity has been shown to predict depression (Roberts *et al*. 2003; Kasen *et al*. 2007) and weight gain to be associated with more severe depression (Noppa & Hallstrom, 1981; Murphy *et al*. 2009). But also contradictory observations are reported (Friedman & Brownell, 1995). Regardless of the extensive research in the field, the data on depression-related alterations in detailed body composition are scarce.

The symptom profile of depression modifies the biological correlates of depression. The typical features of melancholic depression are persistent depressive mood, worse symptoms in the morning, early morning awakenings, significant weight loss, psychomotor symptoms and excessive guilt. These features are more often seen in association with dexamethasone non-suppression and elevated cortisol levels (APA, 1994; Gold & Chrousos, 2002), which in turn may lead to insulin resistance and deposition of visceral fat even in subjects without psychiatric disorders (Björntorp & Rosmond, 1999). Some clinical studies have reported elevated visceral fat deposits in depressed patients with hypercortisolaemia (Weber-Hamann *et al*. 2002) as well as in patients with melancholic depression (Thakore *et al*. 1997). Dysthymic disorder features long-lasting, chronically (minimum 2 years) depressed mood with changes in appetite, sleep, concentration, accompanied with fatigue and hopelessness, without fulfilling the diagnostic criteria of major depressive disorder (MDD). Subjects with dysthymia have been observed to have a less dysfunctional hypothalamic–pituitary–adrenal (HPA) axis compared with subjects with MDD (Oshima *et al*. 2000), and therefore they could be considered less likely to develop depression-related adverse metabolic effects including central obesity.

The association between obesity and depression still remains highly controversial (McElroy *et al*. 2004). Large-scale epidemiological studies with careful anthropometric measures and psychiatric diagnostics are warranted in order to estimate the association between obesity and subtypes of depressive disorders in unselected populations (Hach *et al*. 2007). Therefore, we examined differences in detailed body composition in subjects without a depressive disorder, MDD with or without melancholic features, or dysthymia in a large unselected population-based sample.

Method

Health 2000 survey

The study data are derived from the Health 2000 survey, which comprehensively represents the Finnish population aged over 29 years ($n=8028$). The methods and basic results have been published elsewhere (Aromaa & Koskinen, 2004; Heistaro, 2008; available at www.terveys2000.fi). The survey consisted of a health interview, a thorough health examination with measurements, laboratory tests, a structured mental health interview and several self-report questionnaires. Data were collected during 2000 and 2001.

Psychiatric diagnostics

A Munich-Composite International Diagnostic Interview (M-CIDI; Wittchen *et al*. 1998) was performed on those attending the health examination, assessing the 12-month prevalence of major depressive episodes and dysthymia with DSM-IV criteria (APA, 1994; Pirkola *et al*. 2005; Saarni *et al*. 2007). Based on the CIDI, three depressive disorder classes were formed: MDD with melancholic features ($n=76$), MDD without melancholic features ($n=169$) and dysthymic disorder ($n=147$) (including 53 with double depression, of which 23 had MDD with melancholic features).

Body composition

Weight and body composition were measured using the Inbody 3.0 segmental multi-frequency bioimpedance analyser (SMFBIA, Biospace Co. Ltd, South Korea) yielding measures of total body fat-free mass and fat percentage. In comparison with other body composition measurement methods such as dual X-ray analysis, underwater weighing (Malavolti *et al*. 2003; Salmi, 2003; Salmi & Pekkarinen, 2004) and $2H_2O$/Br dilution (Sartorio *et al*. 2005), the InBody 3.0 has been shown to be a reliable and accurate method for measuring body composition. Height was

measured using a wall-mounted stadiometer. Waist circumference was measured standing, half way between the iliac crest and the lowest rib, at the end of light expiration; hip circumference was measured at the point of maximum girth. For obesity [body mass index (BMI) $\geqslant 30\ kg/m^2$] and abdominal obesity (waist circumference $\geqslant 88$ cm for women and $\geqslant 102$ cm for men) classifications we used the World Health Organization cut-off points (World Health Organization, 1995; Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001).

Response rates

The final sample consisted of 7977 individuals alive at the time of the health interview. The M-CIDI was performed with 6038 subjects (95% of those attending the comprehensive health examination). Of these, 33 subjects were excluded due to unreliable reporting, leaving 6005 subjects, which is 75.3% of the original sample. Compared with participants in the M-CIDI, those who only attended the home interview were found to score significantly more symptoms in the Beck Depression Inventory (BDI) and General Health Questionnaire (GHQ-12) (8.34 *v.* 7.00, $p<0.001$; 2.17 *v.* 1.80, $p<0.001$) They also had a slightly lower BMI (26.44 *v.* 27.0 kg/m^2, $p<0.001$). There were no significant differences in waist circumference or waist-to-hip ratio between M-CIDI participants and non-participants. Bioimpedance was yielded for 5831 (73.1%) and BMI for 7208 (90.4%) participants.

Statistical methods

Analyses were conducted using the statistical software Stata 8.2 for Windows (StataCorp LP, USA). All analyses accounted for the two-stage sampling design. Post-stratification weights were used to correct for non-response and oversampling of people aged over 80 years (Aromaa & Koskinen, 2004; Heistaro, 2008). The confidence intervals (CI) for proportions were constructed using a logit transformation.

We used logistic regression for survey data to analyse the association between different diagnoses, obesity and abdominal obesity. Linear regression for survey data was used to analyse continuous variables. Subjects without MDD or dysthymia were used as the reference category, i.e. the controls could have had some other psychiatric disorder. Regression analyses were conducted in a step-wise manner. All covariates except BMI were entered as dummy variables. Separate models were created for each diagnostic group. No statistically significant gender interaction was found for any of the outcome measures and therefore gender-adjusted models were used.

Results

Subjects with dysthymia were more often abdominally obese (54.9 *v.* 40.6%, $p<0.005$), and had greater fat percentage (30.4 *v.* 27.4%, $p<0.005$) and fat mass (23.8 *v.* 21.5 kg, $p<0.005$) than the reference group. The reference group had greater fat-free mass (53.0 *v.* 51.1–43.5 kg, $p<0.005$) than all the depressive disorder groups (data not shown).

In the regression models (Table 1) subjects with dysthymia had an increased likelihood of being abdominally obese (waist circumference $>88/102$ cm; odds ratio 1.71, 95% CI 1.21–2.41), and an increased fat percentage ($\beta = 1.56$%, 95% CI 0.33–2.80) and waist-to-hip ratio ($\beta = 0.02$, 95% CI 0.01–0.03) compared with referents. Adjustment for BMI, education, diet, smoking, antidepressive or antipsychotic medication and income did not change this result. Differences in fat mass became statistically significant after BMI was added to the model. Differences in mean BMI ($\beta = 0.32\ kg/m^2$, 95% CI -0.61 to 1.24) or waist circumference ($\beta = 1.54$ cm, -0.69 to -3.78) were not statistically significant, nor were the differences in fat-free mass, or leg or arm muscle (data not shown).

Subjects with melancholic or non-melancholic depression did not differ from the population or from each other on any of the measures.

Discussion

This is the first population-based study comparing detailed body composition between different subtypes of depression. People with depressive disorders did not have increased BMI, but people with dysthymia had increased waist-to-hip ratio, increased fat percentage and were more often abdominally obese than the controls. This was also apparent after adjusting for BMI and for fat mass only after adjusting for BMI, indicating that dysthymia was associated with increased visceral and total body fat rather than body weight. Contrary to our expectations, MDD with melancholic features did not show any tendency for metabolically unfavourable changes in body composition.

Previous studies report contradictory results on the association between obesity and depression (Noppa & Hallstrom, 1981; Friedman & Brownell, 1995; Roberts *et al*. 2003; Murphy *et al*. 2009). Our results open a new interpretation by finding that some forms of depression are associated not with overweight as such, but with abdominal obesity. This view is supported by clinical studies examining cortisone metabolism or visceral fat deposits in subtypes of depression

Table 1. *Results from the regression models; body composition in different subtypes of depression*

	No MDD	MDD without melancholia	MDD with melancholic features	Dysthymia
Model I[a]				
Subjects, *n*	5613	169	76	147
BMI, kg/m^2	0.00 (ref)	0.28 (−0.43 to 0.99)	−0.60 (−1.62 to 0.41)	0.32 (−0.61 to 1.24)
Waist circumference, cm	0.00 (ref)	0.65 (−1.14 to 2.24)	−1.63 (−4.59 to 1.33)	1.54 (−0.69 to 3.78)
Waist-to-hip ratio	0.00 (ref)	0.00 (−0.01 to 0.01)	0.01 (−0.01 to 0.03)	0.02 (0.01–0.03)**
Fat percentage	0.00 (ref)	0.27 (−0.81 to 1.34)	−0.34 (−1.93 to 1.25)	1.56 (0.33–2.80)*
Abdominal obesity, OR (95% CI)	1.00 (ref)	1.15 (0.84 to 1.58)	0.92 (0.56–1.53)	1.71 (1.21–2.41)**
Fat mass, kg	0.00 (ref)	0.66 (−0.73 to 2.05)	−1.06 (−2.98 to 0.85)	1.67 (−0.21 to 3.55)
Fat-free mass, kg	0.00 (ref)	0.62 (−0.30 to 1.54)	−1.98 (−3.54 to −0.41)*	−1.01 (−2.64 to 0.61)
Model II[b]				
Waist circumference, cm	0.00 (ref)	0.10 (−0.64 to 0.85)	−0.21 (−1.57 to 1.15)	1.00 (0.07–1.93)*
Waist-to-hip ratio	0.00 (ref)	0.00 (−0.01 to 0.01)	0.01 (−0.01 to 0.03)	0.02 (0.01–0.03)**
Fat percentage	0.00 (ref)	−0.10 (−0.63 to 0.42)	0.49 (−0.28 to 1.27)	1.16 (0.48–1.84)**
Abdominal obesity, OR (95% CI)	1.00 (ref)	1.09 (0.62–1.90)	1.22 (0.51–2.95)	2.16 (1.20–3.90)**
Fat mass, kg	0.00 (ref)	0.11 (−0.34 to 0.56)	0.16 (−0.44 to 0.76)	1.08 (0.56–1.59)**
Fat-free mass, kg	0.00 (ref)	0.34 (−0.44 to 1.11)	−1.34 (−2.63 to −0.05)*	−1.32 (−2.36 to −0.28)*
Model III[c]				
BMI, not adjusted for BMI, kg/m^2	0.00 (ref)	0.22 (−0.51 to 0.95)	−0.62 (−1.68 to 0.45)	0.19 (−0.80 to 1.18)
Waist circumference, cm	0.00 (ref)	0.45 (−1.39 to 2.26)	−1.93 (−5.00 to 1.15)	0.74 (−1.64 to 3.12)
Waist-to-hip ratio	0.00 (ref)	0.00 (−0.01 to 0.01)	0.01 (−0.01 to 0.03)	0.01 (0.00–0.02)*
Fat percentage	0.00 (ref)	−0.24 (−0.78 to 0.30)	0.30 (−0.50 to 1.09)	0.73 (0.03–1.43)*
Abdominal obesity, OR (95% CI)	1.00 (ref)	1.08 (0.61–1.91)	1.09 (0.46–2.57)	2.03 (1.14–3.59)*
Fat mass, kg	0.00 (ref)	−0.01 (−0.48 to 0.46)	−0.03 (−0.63 to 0.56)	0.73 (0.18–1.27)**
Fat-free mass, kg	0.00	0.39 (−0.40 to 1.18)	−1.07 (−2.40 to 0.26)	−0.51 (−1.55 to 0.53)

Values are given as β coefficient (95% CI).

MDD, Major depressive disorder; BMI, body mass index; ref, reference; OR, odds ratio; CI, confidence interval.

[a] Adjusted for age and gender.

[b] Adjusted for age, gender and BMI.

[c] Adjusted for: age, gender, BMI, education, income, marital status, diet, smoking, antidepressive and antipsychotic medication.

* $p \leqslant 0.05$, ** $p \leqslant 0.01$.

(Thakore *et al*. 1997; Oshima *et al*. 2000; Gold & Chrousos, 2002; Weber-Hamann *et al*. 2002). The finding of increased abdominal obesity has public health importance, as abdominal obesity is especially harmful due to its promotion of insulin resistance, elevated triglycerides, diabetes and hypertension, all of which increase the risk of cardiovascular disease (Reaven, 1988).

Based on previous studies (Oshima *et al*. 2000), we expected that subjects with MDD would have a greater degree of adverse changes in body composition compared with those with dysthymia or those without any psychiatric disorders. Nevertheless, some previous studies suggest that the duration of depressive symptoms may be more relevant with regard to depression-related biological alterations, than symptom severity (Lehto *et al*. 2008). Thus, being exposed to depression-related physiological changes such as HPA hyperactivity for an extended period of time could explain the observed adverse changes in body composition in dysthymia.

Compared with participants in the comprehensive health examination, those who did not attend the M-CIDI had somewhat higher GHQ-12 and BDI symptom scores and lower BMI without significant differences in waist circumference or waist-to-hip ratio. Based on this, it is possible that our findings slightly overestimate the effect of dysthymia on abdominal obesity. On the other hand, as the subjects with MDD or dysthymia were compared with the rest

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of the population, some control subjects had other psychiatric disorders (Pirkola *et al*. 2005). This may have weakened our findings, since schizophrenia and schizo-affective disorder are also associated with abdominal obesity (Saarni *et al*. 2009).

A particular strength of our study is that both a structured psychiatric interview and detailed body composition measurement were carried out for a large population-based sample. We were also able to adjust for a large set of possible confounders. A weakness in the current study was conducting several statistical tests without correction. However, consistent results from different body composition measures that individuals with dysthymia have greater abdominal obesity but not greater BMI than controls reduces the risk of false positives due to multiple testing. The examination of melancholic and non-melancholic depression and dysthymia as separate subgroups made it possible to test the hypothesis of different body composition profiles among depressed subjects. However, the cross-sectional design of our study does not allow conclusions about possible causal pathways between abdominal obesity and dysthymia. Longitudinal studies with comprehensive psychiatric interviews and body composition measures are needed to further examine the possible interplay between body composition and depressive disorders.

Conclusions

Our results indicate that subtypes of depression, especially dysthymia, should be taken into account and studied separately when investigating associations between depressive disorders, obesity and metabolic adversities. BMI might not capture all metabolically unfavourable changes in body composition in these patient groups.

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Declaration of Interest

None.

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Letter to the Editor

The need for drug-naive research in first-episode psychosis: a response to Moncrieff & Leo (2010)

Moncrieff & Leo (2010) provide a thorough overview of the literature on the association between use of antipsychotic medication and global brain volume changes. One of the central arguments of the piece is that some of the brain abnormalities observed in schizophrenia patients are not a consequence of the illness itself but in fact result from antipsychotic medication. The mechanisms by which the structure of the brain is influenced by antipsychotic medication is currently not well understood. The authors argue that there is an urgent need for studies that randomize first-episode psychosis patients to either treatment with antipsychotic medication or to withhold antipsychotic treatment for a few weeks while studying the effect of antipsychotics on brain structure. Such studies would inform the issue of the relative role of antipsychotic medication and the progression of psychosis in brain changes associated with psychotic disorders.

Our group at the Early Psychosis Prevention and Intervention Centre (EPPIC) in Melbourne, Australia, is currently conducting a trial with such a design (Francey *et al*. 2010). The study involves randomizing