Relationships between personality disorders and anthropometry, hormones and metabolism in women

R. Rosmond, F. Baghei, G. Holm, and P. Björntorp

Department of Heart and Lung Diseases, Göteborg University, Sahlgrenska University Hospital, Göteborg, Sweden

ABSTRACT. This study sought to examine the potential influence of personality disorders (PD) on anthropometry, hormones and metabolism in women. In a population sample of women born in 1956 (no.=270), estimates of PD:s by Structured Clinical Interview for DSM-III-R, Axis II, were correlated with anthropometric, endocrine, and metabolic factors. The PD:s were grouped into three thematic clusters: cluster A (characterized by oddness or eccentricity), cluster B (characterized by self-centeredness, emotionality, and erratic behavior) and cluster C (characterized by anxiety and fear). Subjects with cluster A PD:s had significantly increased body mass index (BMI, kg/m^2) and abdominal sagittal diameter (cm) as well as lower salivary cortisol after dexamethasone (DEX) compared to controls. Subjects with cluster B also had a significantly higher abdominal sagittal diameter and significantly lower salivary cortisol levels after DEX than controls. In addition, subjects with cluster B PD:s had decreased levels of ACTH, and significantly higher concentrations of lactate and triglycerides, while high-density lipoprotein (HDL) cholesterol was significantly lower compared to controls. A significantly higher waist/hip ratio was seen among subjects with cluster C PD:s. In addition, these subjects had higher levels of insulin, glucose, lactate, triglycerides, total cholesterol and low-density lipoprotein (LDL) cholesterol than controls. Moreover, IGF-I and HDL cholesterol were significantly decreased in the former group. These results suggest that PD:s are involved in the development of obesity and abdominal fat accumulation in women, with different endocrine and metabolic profiles depending on the type of PD. (J. Endocrinol. Invest. *24:* 159-165, 2001)

INTRODUCTION

Psychiatric research recognizes 11 distinctive personality disorders (PD) (1). These are grouped into three thematic clusters: cluster A (paranoid, schizoid, schizotypal), cluster B (antisocial, borderline, histrionic, narcissistic) and cluster C (avoidant, dependent, obsessive-compulsive, passive-aggressive). Cluster A PD:s are characterized by oddness or eccentricity. Cluster B PD:s share a dramatic presentation along with self-centeredness, emotionality, and erratic behavior. Anxiety and fear underlie cluster C PD:s.

Cortisol escape from dexamethasone (DEX) suppression is associated with abdominal obesity and its related metabolic abnormalities (2-4). Such relationships have recently been observed in cluster B and cluster C PD in middle-aged men (5), suggesting a link between personality and disturbances in metabolism via the hypothalamic-pituitary-adrenal (HPA) axis.

We have repeatedly observed relationships between centralization of body fat and traits of depression and anxiety as well as psychosocial impairments in both men and women (6-10). This is in accordance with Kretschmer's original observation that depression is more frequent in the pychnic type of body build (11). Personality characteristics have, however, to our knowledge not been studied systematically in relation to anthropometry, hormones and metabolism in women.

We have recently come to recognize that perceived stress from environmental pressure is closely related to disease-generating factors, probably via activation of the HPA axis (3). As soon as a stressor is perceived, the HPA axis secretes cortisol into the bloodstream to assist in meeting the demands of stressors (12). However, each person's unique combination of

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Correspondence: Dr. Roland Rosmond, Department of Heart and Lung Diseases, Sahlgrenska University Hospital, S-413 45 Göteborg, Sweden.

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heredity, life experience, personality, and ability to cope are all involved in the perception of an event and the meaning attached to it. Moreover, most diseases have multiple causes, including factors such as genetics and personality. Interest in the potential influence of personality disorders on anthropometry, hormones and metabolism promoted the present study in a population sample of women.

METHODS

Study population

In the present study, we recruited the subjects from an ongoing cohort study of women (no.=1137) born in 1956 on uneven days of the month, and living in Göteborg (8-10, 13). The study was initiated in 1996. Based on self-reported waist/hip ratio (WHR) the following three subgroups, each of 150 women, were selected for further studies: the lowest (≤0.738) and the highest values (≥0.895) as well as women around the arithmetic mean (0.798-0.822). From November 1997 to December 1998, they were invited to a health examination at the laboratory, and 270 (60%) volunteered to participate. Women were excluded from the study if they had Cushing's syndrome, Addison disease, or were post-menopausal. All women gave written informed consent before participating in the study, which was approved by the Göteborg University Ethics Committee.

Clinical examination

Two physicians (R.R., F.B.) subjected all participants to a clinical examination by obtaining a restricted history supplemented by a physical examination that emphasized detailed investigation of potential key organ systems. The clinical examination aimed to disclose the following earlier or current diseases: myocardial infarction, angina pectoris, stroke, hyperlipidemias, hypertension, endocrine disorders, Type 1 and Type 2 diabetes mellitus.

Study design

The same research technician performed all anthropometric and biochemical examinations in the morning after the participants had been fasting overnight.

Personality disorders. The Structured Clinical Interview for DSM-III-R, Axis II (SCID II) Screen Questionnaire was used to evaluate PD. This questionnaire includes 124 questions to be answered "yes" or "no". The questions concerning antisocial behavior (no.=23) were excluded due to their offensive character in order to avoid defect or hostile responses. Moreover, the original SCID II Screen questionnaire (14) does not include the questions regarding antisocial personality disorders compared to the translated and modified version used in this study. One additional criterion was added as described in Ekselius *et al.* (14).

Anthropometry. Body weight was measured to the nearest 0.1 kg, and height was measured to the nearest 0.01 m. The body mass index (BMI) was calculated as the weight in kilograms divided by the square of height in meters. The waist (W) circumference was measured half-way between the lower rib and iliac crest, and the hip (H) circumference over the widest part in the gluteal region, and the WHR calculated. The abdominal sagittal diameter was determined as the distance between the examination table and the highest point of the abdomen in a recumbent position (15).

Hormones, glucose, and lipids. The assessment of cortisol was done by a sampling device called Salivette (Sarstedt, Landskrona, Sweden), where salivary cortisol concentrations were determined (Orion Corporation, Espoo, Finland). We gave the participants one tablet of DEX (Decadron, MSD, Sollentuna, Sweden) of 0.5 mg to be taken at 10:00 h on a random working day. The following morning the participants collected saliva using the Salivette. ACTH (Nichols Institute Diagnostics, San Juan Capistrano, CA, U.S.A.), corticosteroid-binding globulin (Medgenix Diagnostics, Fleurus, Belgium), prolactin and follicle-stimulating hormone (Diagnostic Products Corporation, Los Angeles, CA, U.S.A.), IGF-I (Nichols Institute Diagnostics, San Juan Capistrano, CA, U.S.A.), thyroid-stimulating hormone and free thyroxine (Abbott Laboratories, Abbott Park, IL, USA) were analyzed by commercially available RIA kits. Serum insulin was measured by RIA (Pharmacia & Upjohn Diagnostics, Uppsala, Sweden), and glucose in whole blood was determined by the automated glucose analyzer ESAT 6660 from Eppendorf (16). Triglycerides, total- and high-density lipoprotein (HDL) cholesterol were measured with an enzymatic procedure in a Boehringer Mannheim Cobas Fara II (Boehringer Mannheim, Germany). Low-density lipoprotein (LDL) cholesterol was determined using the Friedewald formula.

Statistical methods. Test if the distribution of a variable has the same location parameter across two groups was performed with Pitman permutation test (17, 18). The significance level was based on the exact distribution of the test statistic, and a $p<0.05$ was considered to be significant. We ex-

Table 1 - *The distribution of personality disorders within clusters A, B and C.*

	Cluster A No. of subjects/total no. (%)	Cluster B No. of subjects/total no. (%)	Cluster C No. of subjects/total no. (%)
Paranoid	18/(6.7)		
Schizotypal	1/(0.4)		
Schizoid	3/(1.1)		
Borderline		10/(3.7)	
Histrionic		4/(1.5)	
Narcissistic		11/(4.1)	
Avoidant			12/(4.4)
Dependent			8/(3.0)
Obsessive-compulsive			9/(3.3)
Passive-aggressive			9/(3.3)

cluded cases with missing values from the analysis. All data analysis was performed using StatXact-3 Version 3.1 (Cytel Software Corporation, Cambridge, MA, U.S.A.).

RESULTS

Frequent use psychopharmacological drugs was a consistent correlate of non-response. In the group with the lowest WHR, non-response was associated with poor working conditions and current smoking. In the group with mean WHR, non-response was associated with low educational level. However, no pattern of association between health status and non-response was observed in any of the subgroups.

The clinical examination revealed that one (0.4%) subject suffered from angina pectoris and myocardial infarction. One subject had stroke, 21 (7.8%) subjects had hypertension and 17 (6.3%) subjects had hyperlipidemias. Moreover, 12 (4.4%) subjects had a dysfunctional thyroid gland.

Table 1 presents the distribution of PD:s within each cluster. The most prevalent PD were paranoid (cluster A), narcissistic (cluster B) and avoidant (cluster C); 17.4% of the subjects received at least one PD diagnosis according to the SCID II Screen questionnaire, and 18 (6.7%) received at least two PD diagnoses (not shown in Table 1).

The differences in anthropometry between controls and subjects with cluster A, B and C PD:s are presented in Table 2. Subjects with cluster A PD:s had significantly increased BMI and abdominal sagittal diameter compared with controls. Subjects with cluster B PD:s had also higher BMI (borderline) and abdominal sagittal diameter than controls. WHR and abdominal sagittal diameter (borderline) were higher among subjects with cluster C PD:s.

The endocrine profile of controls and subjects with PD:s are given in Table 3. Subjects with cluster A and cluster B PD:s had significantly lower salivary cortisol levels after dexamethasone (DEX) compared to controls. In addition, subjects with cluster B PD:s had a borderline significant decreased level of ACTH. IGF-I was borderline significantly lower in subjects with cluster C PD:s compared to controls.

Table 4 shows the differences in metabolic variables between controls and subjects with PD:s of cluster A, B and C. In subjects with cluster B PD:s, lactate and triglycerides were significantly higher, while HDL

Table 2 - *Differences (mean±SD) in anthropometry between subjects without personality disorders (controls) and subjects with personality disorders of clusters A, B and C.*

	Controls (no.=223)	Cluster A (no.=22)	Cluster B (no.=25)	Cluster C (no.=38)
Body mass index (kg/m^2)	24.5±4.1	26.9±5.4*	26.4±3.9(*)	25.5±4.8
Waist-to-hip ratio	0.80±0.07	0.82±0.07	0.83±0.07	0.83±0.09*
Abdominal sagittal diameter (cm)	19.4±2.6	21.2±3.3**	21.0±2.4*	20.5±3.7(*)

(*)0.05<p<0.10, *p<0.05, **p<0.01.

Table 3 - *Differences (mean±SD) in hormones between subjects without personality disorders (controls) and subjects with personality disorders of clusters A, B and C.*

	Controls (no.=223)	Cluster A (no.=22)	Cluster B (no.=25)	Cluster C (no.=38)
Salivary cortisol level after dexamethasone (nmol/l)	41.1±11.5	1.6±0.7**	1.8±1.2*	2.9±3.7
Adrenocorticotropic hormone (ng/l)	25.7±16.0	21.7±10.6	20.1±9.7(*)	21.7±9.9
Corticosteroid-binding globulin (mg/l)	52.5±10.5	51.4±6.4	53.9±10.0	52.8±12.6
Prolactin (µg/l)	11.5±9.7	12.9±13.1	14.4±15.9	11.2±6.0
Follicle-stimulating hormone (IU/l)	14.0±10.9	12.2±10.4	14.3±11.2	14.0±9.4
Insulin-like growth factor I (µg/l)	204.5±54.6	195.1±54.3	210.1±52.9	185.0±69.1(*)
Thyroid-stimulating hormone (µIU/l)	1.2±1.0	1.3±0.7	1.2±0.6	1.3±1.0
Thyroxine (pmol/l)	15.8±4.4	15.2±2.6	14.2±2.0	15.3±2.7

(*)0.05<*p*<0.10, *p<0.05, **p<0.01.

cholesterol was lower compared to controls. In subjects with cluster C PD:s, insulin (borderline), glucose, lactate (borderline), triglycerides, total cholesterol (borderline) and LDL cholesterol (borderline) were lower than in controls. Moreover, HDL cholesterol was significantly decreased in the former group.

DISCUSSION

In this study, PD:s were registered by the SCID II Screen questionnaire, which is self-rated (13). It has been shown that this questionnaire is sufficiently reliable and valid compared to SCID II interview when adding one more criteria to satisfy for each PD (14); 17.4% of the subjects obtained at least one PD. The PD:s identified were examined in relation to estimates of obesity (BMI), abdominal fat distribution (WHR) and visceral fat mass (abdominal sagittal diameter) as well as their associated endocrine and metabolic perturbations (19). The endocrine measurements included demonstrating a relative resistance to negative feedback of the HPA axis by using a modified low-dose DEX suppression test that is simple enough to be applied under ordinary everyday life in a large-scale population. DEX resistance or non-suppression is common in men with abdominal obesity (2, 20). Some but not all studies have shown that borderline and obsessive-compulsive personalities are also associated with cortisol escape from DEX suppression (21-24).

Table 4 - *Differences (mean±SD) in metabolic variables between subjects without personality disorders (controls) and subjects with personality disorders of clusters A, B and C.*

	Controls (no.=223)	Cluster A (no.=22)	Cluster B (no.=25)	Cluster C (no.=38)
Insulin (mU/l)	7.8±3.5	9.0±3.1	9.0±3.4	9.1±3.6(*)
Glucose (mmol/l)	4.4±0.4	4.4±0.4	4.4±0.4	4.6±1.7*
Lactate (mmol/l)	0.6±0.3	0.7±0.3	0.8±0.5*	0.7±0.3(*)
Triglycerides (mmol/l)	1.0±0.5	1.1±0.5	1.3±0.7*	1.4±0.7**
Total cholesterol (mmol/l)	5.1±0.8	5.2±0.8	5.3±1.0	5.4±0.8(*)
High-density lipoprotein cholesterol (mmol/l)	1.6±0.4	1.5±0.4	1.4±0.2*	1.4±0.3*
Low-density lipoprotein cholesterol (mmol/l)	3.0±0.7	3.2±0.7	3.2±0.7	3.3±0.7(*)

(*)0.05<*p*<0.10, *p<0.05, **p<0.01.

The response to stressful events has two major components: an emotional response and a physiological response (25). Emotional and physiological responses may be of two kinds. The first kind is anxiety and fear, with autonomic arousal that leads to tachycardia, increased blood pressure and muscle tension; the second kind is depression with increased cortisol secretion. Anxiety responses are generally associated with events that pose a threat, whilst depression is usually associated with events that involve separation or loss (12, 26). Features of personality can make some individuals more vulnerable to physiological responses when experiencing stressful events. Thus, difficult circumstances are more likely to induce an increase in blood pressure in a person who always worried about minor problems than in a person who has been less prone to worry. In worry-prone subjects, abnormal behavior occurs only in response to stressful events. In subjects with abnormal personalities, unusual behavior occurs even in the absence of stressful events (1).

Before interpreting the results, potential biases should be cautiously examined. The non-responders showed a structure, different to the responders in such a way that selection bias probably is not negligible. High consumption of sedative-hypnotic drugs was the most prominent and consistent correlate of non-response. Although we cannot quantify the precise extent of the bias, the observed differences may appear in the sample to be weaker than it really is in the target population (27).

Subjects with cluster A PD:s were characterized by obesity and elevated visceral fat mass (Fig. 1), and lower salivary cortisol levels after DEX (Fig. 2). This suggests that the associations between cluster A PD:s and visceral centralized (abdominal sagittal diameter) obesity (BMI) in women is not obligatory coupled to a dysfunctional HPA axis. This has also been shown in men previously (5). Despite elevated visceral adipose tissue depots (Fig. 1), women with cluster A PD:s showed no associated abnormalities in glucose, insulin and lipid metabolism. Subjects with cluster B PD:s had elevated visceral fat mass and lower salivary cortisol levels after dexamethasone than controls along with a tendency to lower ACTH values. Furthermore, lactate and triglycerides were significantly increased, while HDL cholesterol was significantly decreased. In men, cluster B PD:s was found to be associated with elevated visceral fat mass only among subjects who were dexamethasone suppression test non-suppressors (5). In subjects with cluster C PD:s, both WHR and abdominal sagittal diameter (borderline) were elevated. These women had also somewhat low IGF-I and HDL cholesterol values, accompanied by high levels of insulin (borderline), glucose, lactate (borderline), triglycerides, total and LDL cholesterol (borderline). Since anxiety and fear underlies cluster C PD:s, an autonomic arousal in the sympathetic nervous system may explain the unfavourable lipid profile. Lipid mobilization is highly influenced by catecholamines (28), and in turn, the end product, circulating free fatty acids, influences the glucose metabolism (29).

In relation to the previous study on middle-aged men (5), in which we used the same methodology as in the present study, the following aspect is noteworthy. In contrast to the findings in men, none of

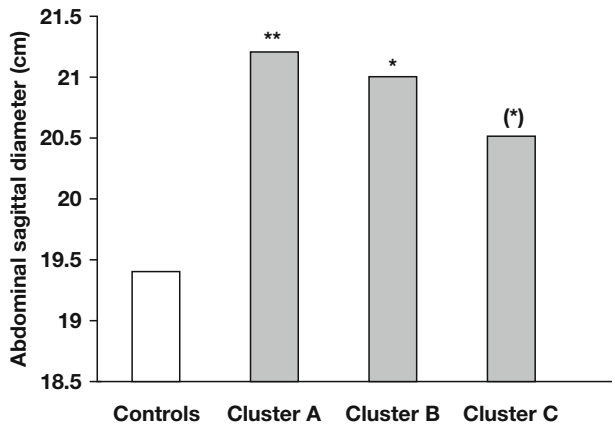

Fig. 1 - *Mean differences in abdominal sagittal diameter (cm) between controls and subjects with personality disorders of clusters A, B and C. (*) 0.05<p<0.10, *p<0.05, **p<0.01.*

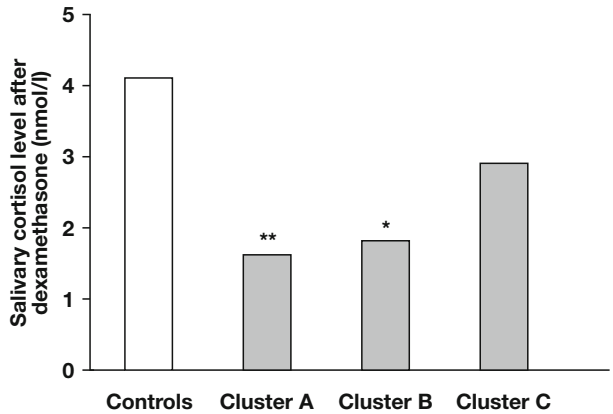

Fig. 2 - *Mean differences in salivary cortisol level after dexamethasone (nmol/l) between controls and subjects with personality disorders of clusters A, B and C. *p<0.05, **p<0.01.*

the PD:s in women were associated with a diminished DEX suppression, suggesting that the involvement of the HPA axis is more pronounced in men than in women. While sex differences of the central nervous system and the HPA axis activity are widely documented (30), little is known about sexual divergence of central nervous system control of the HPA axis. Cluster A PD showed consistent association to visceral obesity in both studies. Recently, a PET-scan study revealed that such personality characteristics are associated with a low density of dopamine D_2 receptor (31). We have recently found that a polymorphism in exon 6 of the dopamine D_2 receptor gene, identified with the restriction enzyme NcoI, is significantly associated with cluster A PD:s (Rosmond *et al.* submitted for publication). These findings provide empirical biologic correlates of cluster A PD:s and schizophrenia, suggesting that environmental factors such as stressful events may be of less etiological significance.

Although the current cross-sectional design limits our ability to determine the temporal sequence of cause and effect, the design yields useful information to generate new etiologic hypotheses that might be tested. The relationship between PD:s and visceral fat mass might be due to a change of personality as a secondary consequence of the obese state, because of the psychosocial handicaps of being obese (32). This seems, however, rather unlikely because the BMI values of the examined women with PD:s on an average were 25.9 kg/m^2, suggesting moderate overweight and few individuals were overtly obese.

In summary, the results presented herein suggest that PD:s are involved in the syndrome of abdominal obesity in women. The direction of causal influence, however, can not be stated. Nevertheless, the association between PD:s and abdominal obesity indicates that the problem of abdominal obesity and cardiovascular disease in women (33-35) need targeted prevention and intervention strategies.

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