Disordered eating behaviour is associated with blunted cortisol and cardiovascular reactions to acute psychological stress

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Summary

Research suggests a potential dysregulation of the stress response in individuals with bulimia nervosa. This study measured both cardiovascular and cortisol reactions to a standardised laboratory stress task in individuals identified as showing disordered eating behaviour to determine whether dysregulation of the stress response is characteristic of the two branches of the stress response system. Female students (N = 455) were screened using two validated eating disorder questionnaires. Twelve women with disordered eating, including self-induced vomiting, and 12 healthy controls were selected for laboratory stress testing. Salivary cortisol and cardiovascular activity, via Doppler imaging and semi-automatic blood pressure monitoring, were measured at resting baseline and during and after exposure to a 10-min mental arithmetic stress task. Compared to controls the disordered eating group showed blunted cortisol, cardiac output, heart rate, and stroke volume reactions to the acute stress, as well as an attenuated vasodilatory reaction. These effects could not be accounted for in terms of group differences in stress task performance, subjective task impact/engagement, age, BMI, neuroticism, cardiorespiratory fitness, or co-morbid exercise dependence. Our findings suggest that disordered eating is characterised by a dysregulation of the autonomic stress-response system. As such, they add further weight to the general contention that blunted stress reactivity is characteristic of a number of maladaptive behaviours and states.

1. Introduction

Stress plays a substantial role in disordered eating (Crowther and Chernyk, 1986; Lacey et al., 1986; Cattanach and Rodin, 1988; Koo-Loeb et al., 2000). Disordered eating, particularly bulimia nervosa, is associated with increased negative perceptions of daily life stress and decreased coping skills (Crowther and Chernyk, 1986; Cattanach and Rodin, 1988; Lo Sauro et al., 2008). Increased perceptions of stress and negative affect can precede binges and ultimately contribute to the etiology and maintenance of bulimia nervosa (Abraham and Joseph, 1987; Cattanach et al., 1988; Cattanach and Rodin, 1988; Lingswiler et al., 1989; Troop et al., 1994). These behaviours suggest a potential dysregulation of the stress response in individuals with bulimia nervosa, which could contribute to the stress—illness relationship (Cattanach and Rodin, 1988; Koo-Loeb et al., 1998). Research measuring the neuroendocrine and cardiovascular responses to a standardised stress task in disordered eating individuals and healthy controls has an important part to play in determining whether bulimia is characterised by a general dysregulation of the autonomic stress response.

Few previous studies have used standardised laboratory procedures to investigate cardiovascular and neuroendocrine responses to stress in individuals with disordered eating; those that have produced conflicting results. In a study of hospitalised women with bulimia nervosa versus controls, women with bulimia had significantly blunted cortisol and norepinephrine stress reactions (Pirke et al., 1992). A broadly similar result emerged from a study examining reactions to two stress tasks in non-hospitalised women with bulimia and controls (Koo-Loeb et al., 1998); women with bulimia had blunted sympathetic activation in response to mental stress indicated by blunted
systolic blood pressure, heart rate, and epinephrine reactions, and attenuated pre-ejection period reactions. Two additional studies screened female students for disordered eating and stress-tested those with extreme high and low scores. Whereas one study observed no differences between those with disordered eating symptoms and controls in cardiovascular reactions to stress (Cattanach et al., 1988), the other reported that those high in disordered eating symptoms, but not meeting a clinical diagnosis for disordered eating, displayed higher heart rate and blood pressure reactivity compared to a group low in disordered eating symptoms (Koo-Loeb et al., 2000). A more recent study compared the stress reactivity of patients with bulimia nervosa, binge eating disorder, and obesity; although the groups did not vary on most measures of stress reactivity, those with bulimia were reported to show higher heart rate reactivity than the other groups (Messerli-Burgy et al., 2010).

There is no clear consensus emerging from these studies. Differences in previous findings may partially be attributed to the different populations of participants tested: in-patient hospitalised (Pirke et al., 1992), enrolled in an out-patient hospitalisation program (Messerli-Burgy et al., 2010), clinically confirmed diagnosis (Koo-Loeb et al., 1998), and high scores on an eating disorder questionnaire, but no official diagnosis (Cattanach et al., 1988; Koo-Loeb et al., 2000). It should be noted that in the three studies which tested confirmed bulimics, only two had a healthy control group and both showed blunted responses (Pirke et al., 1992; Koo-Loeb et al., 1998); the other study compared bulimics and people with binge eating disorder to an obese population (Messerli-Burgy et al., 2010). The aim of the present study was to compare neuroendocrine, measured by salivary cortisol, and cardiovascular reactions to an acute psychological stress task in a group of participants who reported disordered eating behaviours versus healthy controls. On balance, it was hypothesised that those with disordered eating would show blunted reactions to mental stress relative to controls, since blunted stress reactivity has been observed in two previous studies comparing bulimics to a healthy control group (Pirke et al., 1992; Koo-Loeb et al., 1998). To our knowledge, this is the first study to measure both cortisol and cardiovascular reactions to the same mental stress task in individuals with disordered eating behaviour. It is important to determine whether dysregulation of the stress response is characteristic of both branches, of the stress effector system: hypothalamic—pituitary—adrenal (HPA) axis and sympathetic nervous system. The one study, to date, to examine this issue measured cortisol and a-amylase, considered a marker of sympathetic nervous system activation (Chatterton et al., 1996), to a standard laboratory stress exposure (Monteleone et al., 2011) in women with anorexia nervosa and bulimia. Relative to those with anorexia nervosa, women with bulimia showed blunted cortisol reactivity, whereas women with anorexia nervosa showed blunted a-amylase reactivity. In the present study, disordered eating and control groups were confirmed by responses to two questionnaires, one of which, the Eating Disorder Examination Questionnaire (Fairburn and Beglin, 1994), is regarded as an appropriate substitute for a clinical interview to diagnose eating disorders.

2. Methods

2.1. Participants

Questionnaires measuring problematic eating behaviour were administered to 455 female students (age, M = 19.3, SD = 2.08 years) recruited from University of Birmingham. Only women were targeted as problematic eating behaviour is reasonably well characterised in women and is rarer in men (Anderson, 1995; Woodside et al., 2001). The 12 participants who scored highest on the problematic eating criteria (disordered eating group; DE) and 12 who scored the lowest (healthy control group) were selected to attend a laboratory session to measure cardiovascular and cortisol reactions to an acute psychological stress task. None smoked, and none had a history of cardiovascular disease, a current endocrine or immune disorder, an acute infection or another
chronic illness, nor were any of the participants taking medication related to such disorders. However, one participant in the DE group was taking the antidepressant, fluoxetine. The outcome of the analyses with and without this participant were virtually identical and so it was decided to include them in the study. Means and standard deviations for age, body mass index (BMI), and estimated cardio-respiratory fitness for the two groups are presented in Table 1 and scores on eating disorder questionnaires are presented in Table 2. Participants were paid £10 for completing the laboratory session; all gave informed consent and the study was approved by the appropriate Ethics Committee.

2.2. Eating questionnaires

2.2.1. SCOFF

The SCOFF questionnaire contains 5 items which screen for the existence of an eating disorder. The questions focus on (Insert Table 1 Here) the core features of anorexia and bulimia and positive answers to ≥2 questions indicate possible caseness. In a study of a clinical population and matched controls the SCOFF demonstrated 100% sensitivity and 87.5% specificity (Morgan et al., 1999). A more recent study conducted in a primary care setting identified the SCOFF as having a sensitivity of 84.6% and specificity of 89.6% (Mond et al., 2008).

2.2.2. EDE-Q

The EDE-Q is a 36-item questionnaire version of the Eating Disorders Examination, screening device for symptoms of eating disorders (Fairburn and Beglin, 1994). The EDE-Q assesses frequency of eating disorder related behaviours over the past 28 days and is scored on a 7-point Likert scale. Participants are considered to be displaying symptoms of an eating disorder if they register total scores ≥60 on items 1—15 and scores ≥32 on items 29—36 or if they reported self-induced vomiting. The EDE-Q sub-scales have shown good internal consistency, Cronbach’s α = .84, and a test—retest reliability of .86 (Stice and Bearman, 2001). In the present survey data, Cronbach’s α = .92 for items 1—15 and .93 for items 29—36.

2.3. Control and confounding variables

2.3.1. Exercise dependence

The Exercise Attitudes and Beliefs Questionnaire (EABQ) was administered to control for the possible co-morbidity of exercise dependence and disordered eating (Davis et al., 1995, 1997; Epling and Pierce, 1988; Bamber et al., 2000). The EABQ is a 12-item questionnaire generated from the diagnostic criteria of exercise dependence in women proposed by Bamber and her colleagues on the basis of a qualitative study of exercise attitudes and behaviour in 56 female exercisers (Bamber et al., 2003). The items are scored on a 7-poing Likert-type scale and the highest score is 72. The EABQ has been shown to show acceptable internal consistency, Cronbach’s α = .88 (Heaney et al., 2011).

2.3.2. Neuroticism

Neuroticism was measured by the Eysenck Personality Questionnaire Revised-Abbreviated (EPQR-A) (Francis et al., 1992). The subscale measure consists of six Yes/No questions in which each “Yes” scores one points, points are then added for a total score. The neuroticism subscale has demonstrated satisfactory levels of internal consistency Cronbach’s α = 70—.77 (Francis et al., 1992).

2.3.3. Physical activity and estimated cardiorespiratory fitness
Participants were asked to categorise their physical activity levels 1—5, where 1 signified inactivity and 5 indicated participation in a brisk exercise for over 3 h per week. These physical activity levels, 1, 2, 3, 4, and 5 were then assigned scores of .00, .32, 1.06, and 3.03, respectively (Jurca et al., 2005). Cardio-respiratory fitness in METS was estimated using the following formula, \((\text{age} \times 0.10) - ((\text{BMI}) \times 0.17) - ((\text{resting heart rate}) \times 0.03) + (\text{physical activity score}) + 18.07\) (Jurca et al., 2005).

2.3.4. Psychological stress task questionnaire

To test for potential group differences in perception of the stress task, participants were administered a 3-item questionnaire after repeating the stress task and asked to rate how they found the task in terms of whether the task was difficult, stressful, and engaging. Responses were made on Likert scale with 0 representing “not at all” and 6 representing “extremely”.

2.4. Salivary cortisol measurements

Two stimulated saliva samples were obtained using salivettes. Samples were obtained seven minutes into the baseline period and 20 min after the stress task exposure. Participants placed the salivette dental swab in their mouths and gently chewed for 2 min to collect saliva. The swab was returned to the salivette tube and stored in the fridge until the end of the laboratory session. Salivettes were then centrifuged at 400 rpm for 5 min and the saliva was pipetted into eppendorfs which were stored at -20°C until assay. Salivary cortisol samples were analysed all in the same day in duplicate by ELISA (IBL International, Germany). The mean intra-assay coefficient of variation was 10% and the inter-assay coefficient was <10%. Salivary cortisol assays were only analysed in 22 of the participants (11 in each group), due to insufficient saliva from the other two participants.

(Insert Table 2 Here)

2.5. Cardiovascular measurements

The laboratory session consisted of four periods: 10 min adaptation, 10 min baseline, 10 min of stress task exposure, and 20 min of recovery. During the latter three periods, systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured discontinuously using a semi-automatic sphygmomanometer (Critgon Inc., Tampa, FL), and heart rate (HR) was measured continuously by electrocardiography (ECG) with spot electrodes placed on the lower left rib and the right and left clavicle. Echocardiograph measurements were performed using a Philips Sonos 7500 ultrasound machine with an S3 two-dimensional transducer (1—3 MHz) and digital images of spectral waveforms were recorded continuously for later analysis. For each measurement point, averages were obtained from three or more spectral waveforms recorded; measurements for aortic blood flow to be averaged across 60-s intervals. An apical five-chamber view of the heart was used with Doppler mode to identify flow through the aortic valve during systole. The velocity profile of the aortic flow was obtained using pulse-wave spectral mode at a screen sweep speed of 100 mm s⁻¹, Doppler sampling of the flow was taken immediately below the orifice of the aortic valve. The flow was quantified automatically using the velocity time integral, which is the mean distance through which blood travels in the outflow tract during ventricular contraction. Each velocity time integral was made from at least three velocity profiles taken towards the end of expiration. The diameter of the aortic valve was measured from a parasternal long axis view and the aortic valve area was calculated. Stroke volume (SV) was calculated from velocity time integral x the aortic valve area; cardiac output (CO) was calculated as HR x SV. Total peripheral resistance (TPR) was calculated as MAP/CO x 80.

2.6. Acute psychological stress task
The psychological stress task was the paced auditory serial addition test (PASAT) (Grownwall, 1977), which demonstrates good test retest reliability (Willemsen et al., 1998) and reliably perturbs both the cardiovascular system and salivary cortisol (Ring et al., 2002; Phillips et al., 2005, 2006, 2009b). The PASAT was presented via a compact disk player. Participants were presented with a series of single digit numbers and required, in each case, to add any given number to the number previously presented and call out the answer. Thus, the task involves attention and memory as well as simple addition. The intervals between the numbers were 4.5 s for the first 2 min and shortened by .5 s every subsequent 2 min. The task also involved elements of competition and social evaluation. A leader board was displayed prominently and participants were informed that they were in direct competition with their peers. They were awarded 1000 points at the start of the task but lost five points for every wrong answer or omission. The final points total served as a performance score. They received a brief burst of loud, aversive noise at a point during the last five numbers of every block of 10 numbers, largely contingent on an error or a hesitation; all participants received the same number of noise bursts. Participants were also videotaped throughout and informed that the tape would be assessed by “body language experts”.

2.7. Procedure

The DE group was formed from the 12 highest scorers on the SCOFF and Eating Disorder Examination Questionnaire (EDEQ) and the control group was formed from the 12 lowest scorers. All participants in the DE group reported selfinduced vomiting. These 24 participants attended a laboratory session starting after 1430 h. They were required to abstain: from alcohol 12 h, vigorous exercise 12 h, caffeine 2 h, and food and drink other than water 1 h before testing. On entering the laboratory participants had their height and weight measured, and BMI was calculated. During the adaptation phase participants were asked to recline and had electrocardiograph electrodes and a blood pressure cuff attached, and a Doppler echocardiography probe positioned; they then sat quietly for 10 min. This was followed by a further formal 10-min resting baseline period, after which participants were read instructions regarding the mental stress task and completed a brief practice to ensure they understood the task. Participants then completed the 10 min of stress task exposure, and immediately afterwards rated the task in terms of subjective impact. This was followed by a 20-min recovery period. After the recovery period participants completed further questionnaires measuring potential confounding variables.

2.8. Data reduction and analysis

For the cardiovascular data, averages of each period (baseline, task, and recovery) were computed. Group differences in physical, personality, stress task performance, and post task ratings were explored using univariate ANOVAs. Analyses of group differences in cardiovascular activity were by means of repeated measures ANOVAs, using the Greenhouse-Geisser correction. Repeated measures ANCOVAs were used to test for group differences in salivary cortisol; assay batch and time of awakening served as covariates. It is important to adjust for time of awakening since the cortisol awakening response is strongly related to time of awakening (Stalder et al., 2010) and may be related to DE (Therrien et al., 2008). ANCOVAs were also used to determine if any group differences in reactivity withstood adjustment for potential confounders: i.e., other variables that differed between groups.

3. Results

3.1. Validating allocation to control and disordered eating groups

There were no differences between groups in age, height, weight, BMI, or estimated cardiorespiratory fitness. As indicated, the means and standard deviations of these characteristics are displayed in Table 1. As would be expected, the disordered eating group had significantly higher scores than the control group on the SCOFF and EDE-Q. The relevant statistics are presented in Table 2.

3.2. Personality and co-morbidity characteristics

The disordered eating group registered higher scores on the questionnaire measure of exercise dependence (Control, M = 20.0, SD = 11.58; DE, M = 36.3, SD = 16.5), F (1, 23) = 7.87, p = .01, ² = .263, which is often co-morbid with disordered eating (Bamber et al., 2000). There were no significant differences between groups for neuroticism (p = .13; Control, M = 5.3, SD = 3.34; DE = 7.7, SD = 1.10).

3.3. Menstrual cycle phase and oral contraceptives

Forty-two percent of participants reported taking the contraceptive pill (42% of the disordered eating group and 42% of the control group). Four women reported being in the follicular phase of their menstrual cycle in the disordered eating group and four women reported being in the follicular phase of their menstrual cycle in the control group. There were no differences between groups in either contraceptive pill use or menstrual cycle phase.

3.4. Stress task performance and post task ratings

There was no significant difference between performance on the PASAT (p = .78). Groups did not differ on their ratings of how difficult (p = .31) or stressful (p = .49) the task was, nor in the extent to which it engaged them (p = .29). Means and standard deviations for groups on stress task performance and post task ratings are displayed in Table 3.

3.5. Cardiovascular reactions to acute psychological stress

A repeated measures ANOVA on CO revealed a significant main effect for time, F (2, 44) = 73.5, p < .001, ² = .770. There was no significant main effect for group, but there was a significant group x time interaction, F (2, 44) = 10.35, p = .002, ² = .320. This interaction is illustrated in Fig. 1a, which reveals that the ED group showed a blunted CO response to the stress task. Repeated measures ANOVA for HR yielded a significant main effect for time, F (2, 44) = 103.78, p < .001, and a significant main effect of group,

(Insert Table 3 and Figure 1 Here)

F (1, 22) = 7.56, p = .01, ² = .256. There was also a significant group x time interaction, F (2, 44) = 5.13, p = .03, ² = .189. As shown in Fig. 1b, the ED group was less responsive to the stress task than the control group. For SV, there were no main effects but there was a significant group x time interaction effect, F (2, 44) = 8.15, p = .001, ² = .270, which is illustrated in Fig. 1c. SV increased to the stress task only in the control group. For SBP, F (2, 44) = 64.21, p < .001, ² = .745 and DBP, F (2, 44) = 40.30, p < .001, ² = .647,

(Insert Figure 2 Here)

there was only a main effect of time. These summary data are presented in Fig. 2a and b, respectively. For TPR, there a significant effect for time, F (2, 44) = 18.90, p < .001, ² = .462, and a

significant group x time interaction for, $F (2, 44) = 6.430, p = .004$, $\text{R}^2 = .226$. As can be seen in Fig. 2c, the ED group shown a blunted vasodilatory response was.

(Insert Figure 3 Here)

3.6. Salivary cortisol reactions to acute psychological stress

Repeated measures ANCOVA, adjusting for assay batch and awakening time, revealed a significant main effect of time, $F (1, 18) = 7.6, p = .01$, $\text{R}^2 = .297$, and a significant group x time interaction, $F (1, 18) = 5.03, p = .04$, $\text{R}^2 = .218$. As shown in Fig. 3, cortisol increased following the stress task only in the control group.

3.7. Covariance analyses

Given that exercise dependence has recently been found to be negatively associated with cardiac and cortisol reactivity (Heaney et al., 2011) and that the ED and control groups differed on their EABQ scores, the above analyses were revisited adjusting for EABQ score in ANCOVA. The CO, $F (1, 44) = 7.66, p = .007$, $\text{R}^2 = .167$, and HR, $F (1, 44) = 4.08, p = .05$, $\text{R}^2 = .163$, group x time interaction effects remained statistically significant, as did the interactions for SV, $F (1, 44) = 5.21, p = .01$, $\text{R}^2 = .199$, and TPR, $F (1, 44) = 3.73, p = .04$, $\text{R}^2 = .151$. Finally, the group x time interaction effect for cortisol also withstood adjustment for exercise dependence score, $F (1, 44) = 4.61, p = .05$, $\text{R}^2 = .213$.

4. Discussion

The present study compared neuroendocrine and cardiovascular reactions to an acute psychological stress task in a group of participants with disordered eating behaviours and healthy controls. The results were in line with our original hypothesis; individuals with disordered eating exhibited blunted salivary cortisol, CO, HR, and SV reactions and an attenuated vasodilatory response to acute psychological stress compared to healthy controls. There were no differences between groups in SBP and DBP reactivity. Given that exercise dependence has also been associated with blunted cardiovascular and cortisol reactivity (Heaney et al., 2011) and is often co-morbid with eating disorders (Davis et al., 1995, 1997; Bamber et al., 2000), it was important to adjust for EABQ scores as a potential confounder. All group x time interactions remained statistically significant when adjusting for EABQ scores. In addition, the present effects cannot be readily attributed to group differences in stress task performance and subjective task impact or task engagement, menstrual cycle phase, oral contraceptive use, neuroticism, age, BMI, or estimated cardio-respiratory fitness.

This is the first study we know of to measure both neuroendocrine and cardiovascular responses to the same mental stress task in individuals with disordered eating behaviour. Our results confirm previous findings of blunted cortisol (Pirke et al., 1992) and blunted cardiovascular (Koo-Loeb et al., 1998) reactions to acute mental stress tasks in women with bulimia nervosa. In the Koo-Loeb et al. (1998) study, group differences emerged for blood pressure, HR and the preejection period, but not for SV, CO, or TPR. However, they used thoracic impedance cardiography and we used a relatively novel technology in this field, Doppler echocardiography, to measure cardiac activity. A recent study comparing measures of cardiac output and stroke volume using impedance cardiography and Doppler echocardiography concluded that the latter provided a more reliable and clinically acceptable and accurate method of measuring cardiac activity during haemodynamic challenge (Fellahi et al., 2009). Such differences in sensitivity could go some way explaining why blunted cardiovascular reactivity was somewhat differently manifest in the two studies. In contrast, our results are seemingly at odds with those of another study that reported bulimics had relatively high heart rate reactivity (Messerli-Burgy et al., 2010). However, Messerli-Burgy et al. compared
bulimics with a binge eating disorder group and a control group of obese individuals. There is evidence that obesity is also associated with blunted cardiovascular reactivity (Carroll et al., 2008), and so bulimics may only appear to show elevated cardiac reactivity when compared to controls who show blunted reactivity. It is worth noting that similar magnitude HR reactivity characterised their bulimic patients and our disordered eating individuals. In sum, it is difficult to draw firm conclusions about the relative reactivity status of bulimics in the absence of a healthy control group. Finally, the present results varied from those found in two previous studies examining individuals with high scores on eating disorder questionnaires (Cattanach et al., 1988; Koo-Loeb et al., 2000). This may have to do with the stringency of the selection criteria for extreme groups. In the present study, all 12 of the disordered eating disorder group met EDE-Q clinical criteria and all reported self-induced vomiting (Fairburn and Beglin, 1994).

The present results add further weight to the contention that individuals with disordered eating may have a dysregulated stress response (Cattanach and Rodin, 1988; Lo Sauro et al., 2008). The precise pattern of the present group differences, with the disordered eating group showing blunted cardiac reactivity and attenuated systemic vasodilatory reactions to stress, would seem to implicate b-adrenergic processes (Balanos et al., 2010). It is possible that the disordered eating behaviour is associated with diminished b-adrenergic activation. Alternatively, individuals vary in the sensitivity of b-adrenergic receptors (Mills et al., 1994), and it is also possible that disordered eating is associated with reduced receptor sensitivity. There is at least some evidence in favour of the former as bulimics have been found to show blunted norepinephrine reactions to stress (Pirke et al., 1992). The present results also implicate differences in HPA axis activation. The only previous study to examine both sympathetic and HPA axis activation found blunted cortisol stress reactivity in bulimic patients, but blunted a-amylase reactivity in patients with anorexia nervosa, suggesting some specificity in the two branches of the stress effector system (Monteleone et al., 2011). Others have proposed that sympathetic and HPA activation can be dissociated in some circumstances (Frankenhaeuser, 1982; Dickerson and Kemeny, 2004). However, there is also substantial evidence that they frequently covary, such that variations in the magnitude of the acute stress reactions of the sympathetic nervous system, as indexed by cardiac reactivity, predict subsequent variation in HPA reactions, as indexed by cortisol reactivity (Cacioppo, 1994; Al'Absi et al., 1997; Bosch et al., 2009). The present results suggest that disordered eating behaviour may be characterised by dysregulation in both branches of the stress response system.

The results of the present study offer further support for the hypothesis that blunted cortisol and cardiovascular reactivity may be a maladaptive response. Blunted cortisol and cardiovascular reactivity is characteristic of those with substance dependencies and may indeed be a general marker for risk of addiction (Lovallo, 2006). For example, habitual smokers have been found to show diminished salivary and plasma cortisol reactivity (Kirschbaum et al., 1993, 1994; Al’Absi et al., 2003; Rohleder and Kirschbaum, 2006) and cardiovascular reactions (Girdler et al., 1997; Roy et al., 1994; Phillips et al., 2009b), to a range of acute stress tasks, not attributable to temporary abstinence during a stress testing session (Girdler et al., 1997). Those addicted to alcohol have also been found to exhibit blunted cortisol and cardiovascular stress reactivity (Lovallo et al., 2000; Panknin et al., 2002), as have the offspring of alcoholic parents (Moss et al., 1999; Sorocco et al., 2006), suggesting that blunted reactivity may actually predict addiction and signal future risk of addiction. Blunted reactivity has also been associated with obesity (Carroll et al., 2008), lower self-reported health (Phillips et al., 2009a; De Rooij and Roseboom, 2010), and depression (Carroll et al., 2007; York et al., 2007; Salomon et al., 2009; De Rooij et al., 2010). Most recently, blunted reactivity has been found in those reporting high perceived stress relative to their actual life events exposure (Ginty and Conklin, 2011) and in those with exercise dependence (Heaney et al., 2011). Although it may be premature to try to integrate these varied correlates of blunted reactivity, it is possible that
they are all reflect problems in motivation and emotion. Accordingly, it has been proposed that blunted physiological stress reactivity may be a peripheral marker of central motivational dysregulation (Carroll et al., 2009, 2011). By central motivational dysregulation we mean the suboptimal functioning of those systems in the brain, converging at the striatum and ventromedial prefrontal cortex that appear to shape the motivation of our behaviour.

This study is not without limitations. First, the sample size is relatively small. However, it represented the extremities of what is almost certainly a continuous distribution from problematic to non problematic eating behaviour and was of the same order of magnitude of the studies of reactivity and eating disorders. Second, it remains possible that our findings are a product of a confounding by some unmeasured variable (Christenfeld et al., 2004). However, we were able to discount stress task performance and subjective task impact and engagement, BMI, cardio-respiratory fitness and age, nor could the group difference be attributed to co-morbid exercise dependence. Third, our sample was exclusively female which raises the issue of generalisation. However, as indicated, disordered eating is much better characterised in women and is decidedly more prevalent (Anderson, 1995; Woodside et al., 2001). Finally, participants did not have a formal diagnosis of an eating disorder, neither did we assess past history of eating disorders nor past or current treatment. Nevertheless, the EDE-Q has been proven to be as effective as clinical diagnosis interviews in identifying potential eating disorders (Fairburn and Beglin, 1994). Further, all our disordered eating participants reported, on two questionnaires, self-induced vomiting, and all met the criteria for an eating disorder on the SCOFF.

In conclusion, disordered eating was associated with a dysregulation of the autonomic stress-response, as evidenced by blunted cortisol and cardiovascular reactions to a standard mental stress task. The latter would appear to reflect reduced b-adrenergic activation. Our results, together with some, but not all, previous findings suggest that bulimia may indeed be characterised by suboptimal stress responses. Finally, the present data add further weight to the hypothesis that low reactivity may signify a state of central motivational dysregulation and may be characteristics of a number of maladaptive behaviours. Role of funding source There were no study sponsors in the study design; in the collection, analysis and interpretation of data; in the writing of the report; and in the decision to submit the paper for publication.

Conflict of interest

The authors have no conflict of interest.

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Balanos, Sports Cattanach, reactions Disord. physiological illusion Crowther, Chatterton Disorders http://dx.doi.org/10.1016/j.psyneuen.2011.09.004


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Tables and Figures

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<tr>
<th>Table 1</th>
<th>Physical characteristics of the control and DE groups.</th>
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<td></td>
<td>Control</td>
</tr>
<tr>
<td>Age (years)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.7 (.07)</td>
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<tr>
<td>Weight (kg)</td>
<td>64.7 (7.6)</td>
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<td>BMI (kg/m²)</td>
<td>22.6 (2.10)</td>
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<td>Estimated cardio-respiratory fitness (METS)</td>
<td>18.6 (.97)</td>
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There were no significant differences between groups.

<table>
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<tr>
<th>Table 2</th>
<th>Eating behaviour characteristics of the control and DE groups.</th>
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<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>SCOFF total</td>
<td>0.0 (.00)</td>
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<tr>
<td>EDE-Q 1–15 total</td>
<td>4.2 (3.7)</td>
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<td>EDE-Q 29–36 total</td>
<td>0.1 (.29)</td>
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<th>Table 3</th>
<th>Task performance and reported task impact for the control and DE groups.</th>
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<tr>
<td></td>
<td>Control Mean (SD)</td>
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<tr>
<td>PASAT total score</td>
<td>700.4 (89.99)</td>
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<tr>
<td>Difficulty</td>
<td>3.7 (.65)</td>
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<tr>
<td>Stressful</td>
<td>4.3 (.90)</td>
</tr>
<tr>
<td>Engaging</td>
<td>3.5 (.93)</td>
</tr>
</tbody>
</table>

There were no significant differences between groups.

Figure 1  
(a) Mean (SE) cardiac output at baseline and during and after the acute stress task in the control and disordered eating groups. (b) Mean (SE) heart rate at baseline and during and after the acute stress task in the control and disordered eating groups. (c) Mean (SE) stroke volume at baseline and during and after the acute stress task in the control and disordered eating groups.
Figure 2  (a) Mean (SE) systolic blood pressure at baseline and during and after the acute stress task in the control and disordered eating groups. (b) Mean (SE) diastolic blood pressure at baseline and during and after the acute stress task in the control and disordered eating groups. (c) Mean (SE) total peripheral resistance at baseline and during and after the acute stress task in the control and disordered eating groups.

Figure 3  Mean (SE) salivary cortisol at baseline and at 20 min post stress task in the control and disordered eating groups.