Preliminary evidence that exercise dependence is associated with blunted cardiac and cortisol reactions to acute psychological stress

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Abstract

Low or blunted cardiovascular and cortisol reactions to acute psychological stress have been shown to characterise those with a tobacco or alcohol dependency. The present study tested the hypothesis that exercise dependency would be similarly associated with blunted reactivity. Young female exercisers (N = 219) were screened by questionnaire for exercise dependence. Ten women with probable exercise dependence and 10 non dependent controls were selected for laboratory stress testing. Cardiovascular activity and salivary cortisol were measured at rest and in response to a 10-min mental arithmetic stress task. The exercise dependent women showed blunted cardiac reactions to the stress task and blunted cortisol at 10, 20, and 30 minute post stress exposure. These effects could not be accounted for in terms of group differences in stress task performance, nor could the cardiac effects be attributed to group differences in cardio-respiratory fitness. It would seem that low stress reactivity is characteristic of a wide range of dependencies, and is not confined to substance dependence. Our results offer further support for the hypothesis that blunted stress reactivity may be a peripheral marker of a central motivational dysregulation.

1. Introduction

The reactivity hypothesis contends that large magnitude cardiovascular reactions to acute psychological stress play a role in the development of cardiovascular pathology. Evidence in support comes from a number of large scale cross-sectional and prospective observational studies that attest to positive associations between the magnitude of cardiovascular reactions to acute psychological stress tasks and future blood pressure status (Carroll et al., 1995, 2001, 2003; Everson et al., 1996; Newman et al., 1999; Matthews et al., 1993; Markovitz et al., 1998; Treiber et al., 1997), markers of systemic atherosclerosis (Barnett et al., 1997; Matthews et al., 1998; Everson et al., 1997; Lynch et al., 1998), and left ventricular mass and/or hypertrophy of the heart (Georgiades et al., 1997; Kapuku et al., 1999; Murdison et al., 1998). All of these outcomes are predictors of mortality and suggest that large cardiovascular reactions to acute stress are implicated in the development of cardiovascular inflammatory disease.

However, in the understandable enthusiasm to characterise the circumstances and antecedents of excessive reactivity and its consequences for health, the other end of the continuum has been largely neglected. The rarely articulated but implicit assumption is that low physiological
reactivity in the face of acute psychological challenge is the more adaptive response, with no negative consequences for health or behaviour, i.e., low reactivity is benign or even protective. This assumption has recently been subject to challenge (Carroll et al., 2009, in press). Low or blunted cardiovascular reactivity has been reported in epidemiological analyses to be associated with obesity and symptoms of depression cross-sectionally, as well as with an increased risk of becoming obese and developing symptoms of depression five years later (Carroll et al., 2007, 2008; Phillips et al., in press). Analogously, blunted cortisol reactions to acute laboratory stress would seem to characterise patients with atopic dermatitis (Buske-Kirschbaum and Hellhammer, 2003).

Perhaps the most compelling evidence, to date, comes from studies of reactivity and tobacco and alcohol dependence. There is emerging evidence that low or blunted cardiovascular or cortisol reactivity is characteristic of those with substance dependencies and may indeed be a general marker for risk of addiction (Lovallo, 2006). Habitual smokers, for example, have been found to show diminished salivary and plasma cortisol (al'Absi et al., 2003; Kirschbaum et al., 1993, 1994; Rohleder and Kirschbaum, 2006) and cardiovascular (Girdler et al., 1997; Phillips et al., 2009; Roy et al., 1994; Sheffield et al., 1997; Straneva et al., 2000) reactions to acute psychological stress. It is unlikely that these effects reflect temporary abstinence during stress testing and its effects on stress task engagement (Roy et al., 1994). Blunted cardiovascular reactivity has been observed in female smokers regardless of whether they were wearing a nicotine replacement patch or not (Girdler et al., 1997). In addition, cardiovascular and cortisol reactivity has been compared among non-smokers, smokers who abstained from smoking, and smokers who continued to smoke at their usual rate; smokers, irrespective of their assigned condition, showed blunted cardiovascular and cortisol reactions to acute stress (al'Absi et al., 2003).

Those addicted to alcohol have also been found to exhibit blunted cardiovascular and cortisol stress reactivity (Lovallo et al., 2000; Panknin et al., 2002). In addition, relatively low reactivity would appear to be a characteristic of non-alcoholics with a family history of alcoholism. In the Oklahoma Family Health Patterns project, young adults with a positive family history, particularly those with low sociability scores, showed lower cortisol and cardiac reactions to psychological stress than those with a negative family history of alcoholism and high sociability scores (Sorocco et al., 2006). Other studies of the offspring of parents addicted to alcohol or drugs provide further evidence; in a prospective study, boys with a positive family history who showed a blunted cortisol response to stress were more likely to experiment with cigarettes and marijuana (Moss et al., 1999). These data suggest that blunted reactivity may not only be a characteristic of those with a dependency, it may actually pre-date the dependency and signal risk of future addiction.

If low cardiovascular and cortisol reactivity is a marker of motivational dysregulation and linked to inherited risk of addictions (Lovallo, 2006), this should be evident with other dependencies. Although somewhat controversial, it has been argued that exercising has the potential to become an all-consuming and damaging obsession (Morgan, 1979). The reported characteristics of what has come to be called exercise dependence resonate with those that typify substance addiction: experience of withdrawal symptoms on cessation of exercise; increasing tolerance; disturbed psychological functioning; exercising despite medical contraindications; interference with relationships or work (Bamber et al., 2000, 2003; Robbins and Joseph, 1985;
Yates et al., 1983). Indeed, diagnostic criteria, not too dissimilar to those that define substance addiction, have been proposed (Bamber et al., 2003). Accordingly, exercise dependence should provide an interesting model to test the contention that blunted stress reactivity is a marker of motivational dysregulation and is a general feature of addiction and risk of addiction, including non substance dependencies. In the present study we compared the cardiovascular and cortisol reactions to acute psychological stress of individuals who were seemingly exercise dependent and those who were clearly not. It was hypothesised that the former would show blunted stress reactivity.

2. Methods

2.1. Participants

Questionnaires measuring exercise dependence were administered to 219 young adult women who were recruited from sports clubs and exercise classes at the University of Birmingham. Only women were included in the study as exercise dependence is reasonable well characterised in women and is evidently rarer in men (Bamber et al., 2000, 2003). The mean (SD) age of the women recruited was 19.8 (2.25) years and their mean body mass index (BMI) was 21.6 (2.39) kg/m2. Ten participants who scored highest on the exercise dependence criteria (exercise dependent group) and 10 who scored lowest (non exercise dependent group) were selected to attend a laboratory session to measure cardiovascular and cortisol reactions to an acute psychological stress task. Age, BMI, and scores on the exercise dependence questionnaires for the two groups are presented in Table 1. The majority of these 20 participants (95%) indicated that they were “white”, with one describing herself as “Indian”. None smoked, and none had a history of cardiovascular disease, a current endocrine or immune disorder, an acute infection or another chronic illness, with the exception of one participant with asthma and one reporting Pfeiffer syndrome. The exercise dependent group included three runners, two rowers, two soccer players, one swimmer, one cricketer, and one tri-athlete. The non dependent group comprised two gymnasts, one runner, one judoist, one rower, one walker, and four women whose predominant physical activity was undertaken at organised exercise classes. Participants were paid £10 for completing the stress testing session; all gave written informed consent and the study was approved by the appropriate ethics committee.

2.2. Questionnaires

2.2.1. Exercise attitudes and beliefs questionnaire (EABQ)
The EABQ is a questionnaire specifically designed for this study. The 12 items were 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 criteria that informed the items were: a) an extreme preoccupation with exercise such that it impairs functioning in the psychological, social, occupational, or physical domains; b) symptoms of withdrawal as evidenced by adverse reactions to the interruption of exercise or unsuccessful attempts at exercise control. An example of the former would be loss of friends as a result of unreliability due to exercise preoccupation. An example of the latter would be depressed mood and/or extreme irritability when unable to exercise. The items were scored on a 7-point Likert-type scale where 0 = definitely not to 6 = definitely yes. Accordingly, total scores could range from 0 to 72. In the present screening sample, the EABQ showed acceptable internal consistency, Cronbach’s α = .88. The EABQ is reproduced in full in Appendix 1.

2.2.2. Exercise dependence questionnaire (EDQ)

The EDQ is designed to measure relationships between exercise dependence and eating disorders and distinguish between primary and secondary independence (Ogden et al., 1997). It is a 29 item measure which is scored on a 1–7 point Likert scale calculating dependence based on eight subscales. High internal reliabilities (Cronbach’s α) for each of the subscales have been reported: interference with family social life, α = .81; positive reward, α = .80; withdrawal symptom s, α = .80; exercise for weight control, α = .78; insight into a problem, α = .76; exercise for social reasons, α = .75; exercise for health reasons, α = .70; stereotyped behaviour, α = .52; total score, α = .84 (Ogden et al., 1997). The internal reliability for the whole scale in the present screening sample was α = .88. A score of ≥ 116 out of a possible 203 on the EDQ is considered the threshold for possible exercise dependence (Bamber et al., 2000). Scores on the EDQ correlated highly with those on the EABQ, r (215) = .72, p b .001.

(Insert Table 1)

2.2.3. Exercise beliefs questionnaire (EBQ)

The EBQ is a 21-item measure of beliefs about the consequences of not exercising. It has been validated against clinical measures of psychological distress. Example items are ‘if I do not exercise my brain will become unhealthy’ and ‘if I do not exercise I will be sexually unattractive’. Each item is responded to by selecting a position on 100 cm line anchored at I do not believe this thought at all = 0 and I am completely convinced this thought is true. The EBQ has four factors with the following reported internal and test–retest reliabilities: social desirability, α = .87, r = .76; physical appearance, α = .83, r = .77; mental and emotional functioning, α = .89, r = .70; vulnerability to disease and ageing, α = .67, r = .67 (Loumindo s and Wells, 1998). For the purposes of the study only the full scale score was used as an additional check on propriety of group allocation. The internal reliability in the present screening sample for the full EBQ scale was high, α = .94.

2.2.4. Physical activity assessment and the estimation of cardio-respiratory fitness

Physical activity was measured using the scale from the Whitehall II study (Marmot et al., 1991). Participants were asked how much time they spent in activities of light, moderate, and vigorous exercise intensity (0, 1–2, 2–5, 6–8, 9–10, and 11+ hours per week). Category scores (0, 1, 2, 3, 4, or 5), derived from the above were multiplied by a weighting of 1, 2, and 3 for light, moderate, and vigorous intensity exercise, respectively. To facilitate the estimation of cardio-respiratory fitness (Jurca et al., 2005), the final values generated from the questionnaire were categorised as physical activity levels 1–5, where 1 signifies inactivity and 5 indicates participation in 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, 1.76, and 3.03, respectively (Jurca et al., 2005). Cardio-respiratory fitness in METS was estimated using the following formula, ((age *.10) – ((BMI) *.17) – (resting heart rate) * .03) + (physical activity score) + 18.07 (Jurca et al., 2005).

2.2.5. SCOFF eating disorders questionnaire

Since exercise dependence and disordered eating behaviour are often co-morbid (Bamber et al., 2000), the SCOFF questionnaire (Morgan et al., 1999) was administered; it contains 5 items which screen for the existence of an eating disorder. The questions focus on 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).

2.2.6. Psychological stress task questionnaire

This is a 7-item questionnaire, administered immediately following the stress task exposure. It requires participants to rate to what extent they found the task to be difficult, stressful, exciting, confusing, engaging, and embarrassing, as well as how they thought they had performed. Responses were made on a 0 (not at all) to 6 (extremely) Likert-type scale.

2.3. Cardiovascular and salivary cortisol measurements

The laboratory session consisted of four periods: 10 min adaptation, 10 min baseline, 10 min stress task and 30 min recovery. During the laboratory session systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were measured discontinuously from the non-dominant arm using a standard brachial artery cuff and a semi-automatic sphygmomanometer (Critikon Inc, Tampa, FL). During the formal 10-min baseline, cardiovascular measurements were made at 2, 4, and 6 min and a saliva sample initiated at the 7th min.
measurements were made at 2, 4, 6, and 8 min during the acute stress task and at the same time points during the recovery period (latter data not reported). Four stimulated saliva samples were obtained using salivettes. Samples were obtained at 10, 20 and 30 min following the stress task. Participants placed the salivette dental swab into 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 4000 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). This assay is based on the competition principle and microplate separation. An unknown amount of cortisol present in the sample and a fixed amount of cortisol conjugated with horseradish peroxidase compete for the binding sites of antibodies coated on to wells. After an hour the microplate is washed to stop the competition reaction. After addition of a substrate solution the concentration of cortisol is inversely proportional to the optical density measured at 450 nm. The mean intra-assay coefficient of variation was 9% and the inter-assay coefficient was b 10%.

2.4. Acute psychological stress task

Participants undertook an acute psychological stress task: the 10 min paced auditory serial addition test (PASAT). The PASAT has been shown previously to reliably perturb both the cardiovascular system and salivary cortisol (Phillips et al., 2006, 2009; Ring et al., 2002). Participants were presented with a series of single digit numbers by compact disc and required in each case to add the present number to the number previously presented and call out the answer. The intervals between the numbers were 4.5 s for the first 2 min and shortened by .5 s every subsequent 2 min. An experimenter sitting directly adjacent to the participants obtrusively scored participants’ answers. The task also involved elements of competition and social evaluation. A leader board was displayed in the laboratory of the top five scores and the participants were instructed to attempt to beat the displayed scores. Participants started with 1000 points and were deducted five points for every wrong answer. If they made an error or hesitated they received a brief burst of loud aversive noise. If they did not make any errors within each block of 10 numbers, they received the noise at a point during the last five numbers of every block. All participants received the same number, i.e. 21, noise bursts. They were also videotaped throughout the task and instructed to watch themselves live on a television screen directly in front of them throughout the task; they were informed that the tape would be assessed by “body language experts”. In reality, no such assessment was made. If attention drifted, participants were prompted by an experimenter to keep looking at the television screen.

2.5. Procedure
The exercise dependent group was formed from the 10 highest scorers on the EABQ who also had EDQ scores of ≥ 116 and the non dependent group from the 10 lowest EABQ scores who also had scores of ≤ 116 on the EDQ. They were all non smokers. These 20 participants then attended a laboratory session starting at either 15:00 or 16:30. They were required to abstain: from alcohol 12 h, vigorous exercise, caffeine 2 h and food 1 h before the session. During the adaptation phase, participants were seated and the blood pressure cuff attached; they sat quietly and completed the EPQ-R. At the end of the baseline period the participants were read the instruction of the PASAT and completed a brief practice to ensure they understood the task. Participants completed the PASAT task ratings immediately after the task. The schedule of cardiovascular and salivary cortisol measures are indicated above.

2.6. Data reduction and analysis

For the cardiovascular data, averages of each period (baseline and task) were computed. To check that the task perturbed cardiovascular activity, repeated measures ANOVAs were performed. The analogous analysis for the cortisol data was by multivariate ANOVA. Initial analyses of group differences were by means of repeated measures ANOVAs, using the Greenhouse–Geisser correction. Reactivity was then calculated as average task minus average baseline for HR, SBP and DBP. Salivary cortisol reactivity was calculated separately for the 10, 20, and 30 min post task samples by subtracting the baseline concentration in each case. Reactivity data were analysed using univariate ANOVA. Throughout, partial η² is reported as an index of effect size. Univariate ANOVA was also used to test for group differences in age, BMI, estimated cardio-respiratory fitness, data from the exercise dependence and attitudes questionnaires, the personality inventory, the stress task ratings, and stress task performance.

3. Results

3.1. Validating allocation to exercise dependent and non dependent groups

As would be expected, the exercise dependent group had significantly higher total scores than the non dependent group on the EABQ. They also scored significantly higher on the EDQ and all its subscales, as well as on the EBQ. Groups did not differ significantly in either age or BMI. The relevant statistics are displayed in Table 1. Few participants met the SCOFF criterion for an eating disorder: two in the exercise dependent group and none in the non dependent group.

3.2. Physical activity and estimated cardio-respiratory fitness

There was a tendency for the exercise dependent group to register higher physical activity scores than the non dependent group; the respective mean (SD) activity scores were 2.8 (.54) and 2.3 (.66). However, this difference was not quite statistically significant $F(1,18) = 3.60, p = .074, \eta^2 = .167$. Nevertheless, the exercise dependent group, 13.3 (.87) METS, had higher estimated cardio-respiratory fitness than the non dependent group, 12.2 (1.03) METS, $F(1,18) = 6.97, p = .017, \eta^2 = .279$.

3.3. Menstrual cycle phase and oral contraceptives

No one was taking any prescribed medication with the exception of the contraceptive pill which 55% of participants reported taking (40% in the exercise dependent group and 70% of the non dependent group), although this was not significantly different, $\chi^2 (1) = 1.81, p = .178$. Seven women reported being in the follicular phase of the menstrual cycle in exercise dependent group, compared to three in the non dependent group. However, the distribution of those in the follicular and luteal phases between groups was not significantly different, $\chi^2 (1) = 3.20, p = .074$.

The exercise dependent group tended to perform better on the PASAT (675.0, SD = 29.06) than the non dependent group (601.5, SD = 29.06) but the difference was not statistically significant ($p = .09$). Groups did not differ on their ratings of how stressful or difficult the task was nor in the extent that it engaged them. Indeed, the only group difference to emerge from these analyses was in rated performance; the exercise dependent group (3.1, SD = 1.20) judged that they had performed significantly better than the non dependent group, (1.9, SD = 1.10), $F(1,18) = 5.02, p = .04, \eta^2 = .218$.

3.5. Cardiovascular reactions to acute psychological stress

Baseline and reactivity values for SBP, DBP and HR are displayed in Table 2. There were no significant differences between groups in cardiovascular activity at baseline, with one exception. The exercise dependent group had significantly lower baseline SBP (107.8, SD = 10.94) than the non dependent group (118.7, SD = 5.16), $F(1,18) = 8.12, p = .01, \eta^2 = .331$. As revealed by analyses of the difference between average baseline and average stress task levels, the PASAT reliably perturbed SBP, $F(1,19) = 37.16, p = .001, \eta^2 = .662$, DBP, $F(1,19) = 20.67, p = .001, \eta^2 = .521$, and HR, $F(1,19) = 39.92, p = .001, \eta^2 = .678$. Repeated measures ANOVA revealed a significant time effect, $F$

\[(2, 36) = 38.76, p < .001, \eta^2 = .680\] and a significant group \( \times \) time interaction for HR, \( F(2, 36) = 3.80, p = .049, \eta^2 = .174 \); this is illustrated in Fig. 1. There was no group main effect. There were no group main effects or group \( \times \) time interaction effects for SBP or DBP. As would be expected from the above, there was a significant difference in HR reactivity between groups, \( F(1, 18) = 5.12, p = .04, \eta^2 = .221 \); the exercise dependent group exhibited significantly smaller HR reactions to the acute stress. There were no significant group differences in SBP or DBP reactivity.

### 3.6. Salivary cortisol reactions to acute psychological stress

Repeated measures ANOVA revealed a significant effect of time, \( F(3, 54) = 3.53, p = .03, \eta^2 = .164 \), and a group \( \times \) time interaction effect, \( F(3, 54) = 4.36, p = .01, \eta^2 = .195 \). These effects are illustrated in Fig. 2. In terms of cortisol reactivity, there were significant differences between groups at all three sampling points: for 10 min post task, \( F(1, 18) = 7.59, p = .01, \eta^2 = .297 \); for 20 min post task, \( F(1, 18) = 7.06, p = .02, \eta^2 = .282 \); for 30 min post task, \( F(1, 18) = 7.16, p = .02, \eta^2 = .285 \). At each time point, the exercise dependent group showed blunted cortisol reactivity. Indeed, positive cortisol responses were apparent only for the non dependent group (Table 3).

### 3.7. Sensitivity analyses

Given that cardio-respiratory fitness is negatively associated with cardiovascular reactivity (for a meta-analysis sees Forcier et al., 2006) and that the groups differed on estimated fitness, the HR reactivity analysis was re-visited adjusting for estimated fitness in ANCOVA. The group difference in HR reactivity survived such adjustment, \( F(1, 17) = 6.76, p = .02, \eta^2 = .284 \). Since there is no similarly compelling evidence that cardio-respiratory fitness affects cortisol stress reactivity (Sinyor et al., 1983) we did not adjust for fitness in our cortisol analysis. However, women in the luteal phase of the menstrual cycle have been shown to exhibit greater cortisol responses to acute stress than women in the follicular phase or women taking oral contraceptives (Kirschbaum et al., 1999), thus we revisited the cortisol analysis adjusting for menstrual cycle phase and/or oral contraception. For this, we created a binary variable which contrasted those in the luteal phase not taking oral contraceptives with the rest of the sample. The observed group differences in cortisol reactivity at 10, 20 and 30 min post task remained significant after adjusting for menstrual cycle phase and oral contraception; \( F(1, 17) = 4.48, p = .02, \eta^2 = .276 \), \( F(1, 17) = 6.11, p = .02, \eta^2 = .264 \), and \( F(1, 17) = 5.61, p = .03, \eta^2 = .248 \), respectively.

There was also a tendency for the groups to differ on PASAT performance, which may signal variations in mental effort and commitment. Thus the main group analyses for HR and cortisol reactivity were repeated adjusting for performance. The effects reported above survived such adjustment: for HR reactivity, \( F(1, 17) = 6.36, p = .02, \eta^2 = .275 \); for cortisol reactivity at 10
Outcomes

The administered (2000; 10 physiological reactivity. More assumptions found = research, cortisol lower cardiovascular and respiratory reactions for smokers (Carroll et al., 1997; Carroll, 2009). An alternative explanation for the HR reactivity and cortisol reactivity at 10 min post task, F(1,16) = 8.54, p = .01, η² = .348; for cortisol reactivity at 20 min, F(1,16) = 6.51, p = .02, η² = .289; or for cortisol reactivity at 30 min, F(1,16) = 7.58, p = .01, η² = .322.

(In Insert Tables 2 & 3, Figures 1 & 2)

4. Discussion

The results of the present study indicated that individual's identified as exercise dependent exhibited blunted cardiac and salivary cortisol reactions to acute psychological stress compared to their non dependent counterparts. Given that high cardio-respiratory fitness is associated with lower cardiovascular reactivity (Forcier et al., 2006), it was important to discount this as an alternative explanation for the present group differences in HR reactivity. We estimated cardio-respiratory fitness using an established algorithm (Jurca et al., 2005); the exercise dependent group was fitter than the non dependent group. However, the group difference in HR reactivity remained significant when controlling for estimated fitness. In addition, the present effects could not be accounted for by group differences in objective and subjective stress task performance. Nor were they attributable to a co-morbid eating disorder. Only two exercise dependent participants were found to have a possible eating disorder and their exclusion did not materially change the outcomes.

This is the first evidence we know of that a non substance dependency is associated with blunted stress reactivity. However, our findings are very much in line with those from previous research showing that smokers are characterised by relatively blunted cardiovascular (Girdler et al., 1997; Phillips et al., 2009; Roy et al., 1994; Sheffield et al., 1997; Straneva et al., 2000) and cortisol (al'Absi et al., 2003; Kirschbaum et al., 1993, 1994; Rohleder and Kirschbaum, 2006) reactivity. They also resonate with the findings from studies linking alcoholism (Lovallo et al., 2000; Panknin et al., 2002) and risk of alcohol dependence (Sorocco et al., 2006) with blunted cardiovascular and cortisol reactivity. The present results also offer further challenge to the assumption that low stress reactivity is necessarily benign (Carroll et al., 2009, in press).

More specifically, our findings lend further support to the hypothesis that blunted physiological reactivity may be a peripheral marker of central motivational dysregulation linked to inherited or acquired risk of a wide range of addictions (Lovallo, 2006). What might be the underlying neurophysiological mechanisms? We are some way from certainty but clues are emerging. The neural circuits that converge at the striatum and ventromedial prefrontal cortex would appear to shape the motivation of our behaviour, and these appear to be the same circuits that underlie the process of addiction. Neurochemical communication among these structures and among these areas change as experimental animals are exposed to increasing amounts of self-administered drugs of abuse (Koob, 2003), and recent research shows actual reconfiguration of

neural connectivity in these areas following alcohol exposure (Xie et al., 2009). In short, these structures and their patterns of interaction may not only affect our physiology, but also our behaviour. However, in the present context much of this remains highly speculative. As yet, nothing is known about the neurophysiology of exercise dependence and only human brain imaging studies will allow determination of whether or not substance and non substance dependencies have common circuitry and, accordingly, are just different manifestations of a general motivational dysregulation. However, given the preliminary nature of the present findings, replication in a larger sample is an essential first step.

The present study is not without its limitations. First, the sample is solely female which raises the issue of generalisation. However, as indicated, exercise dependence is much better characterised in women and would appear to be more prevalent (Bamber et al., 2000, 2003). Second, the sample size is small. However, it represented the extremities of what is almost certainly a continuous distribution from problematic to non problematic exercise behaviour and was of the same order of magnitude as samples in some of the studies of reactivity and tobacco and alcohol addiction. Nevertheless, the modesty of the sample limits our capacity to control for multiple potential confounding variables. Accordingly, it remains possible that our findings are a product of confounding by some unmeasured variable (Christenfeld et al., 2004). However, we were able discount stress task performance and perceptions, and our results could not be attributed to a co-morbid eating disorder in the exercise dependent. Further, the group differences in HR reactivity could not be accounted for by variations in cardio-respiratory fitness, although it should be conceded that we relied on an estimation of fitness. The group differences in cortisol reactivity could not be accounted for by variations in menstrual cycle phase and/or oral contraceptive use. Finally, it should be conceded that the cortisol reactions were modest and only evident in the non dependent group. Therefore in retrospect, the stress task may not have been optimal to adequately stimulate the hypothalamus–pituitary–adrenal axis.

In sum, like smoking and alcohol addiction, exercise dependence would appear to be characterised by blunted cardiac and cortisol reactions to acute psychological stress. This adds moment to the notion that low reactivity, far from being benign, may signify a state of central motivational dysregulation. The challenge for the future is to understand the neural processes that may be common to different behavioural dependencies, as well as their influence on physiological reactions to psychological stress.

Acknowledgements

The authors would like to thank Bethan Jones, Manoj Sharma, Andrew Rigpen, and Christopher Evans, for help in recruitment, screening, and testing.

Appendix 1. Exercise attitudes and behaviour
Below are a number of statements about attitudes to exercise and exercise behaviour. Please indicate the extent to which these statements are in line with your attitudes and behaviour, by circling the appropriate number between 0 (definitely not) and 6 (definitely yes) for each statement.

1. I am constantly thinking about exercise.
2. I prioritise exercise above all other social activities.
3. I sometimes continue to exercise against medical advice.
4. I have lost friends because of my commitment to exercise.
5. It is very important to me that I keep to a strict exercise regime.
6. I feel bad when I have to interrupt my exercise schedule.
7. I find the thought that I may not be able to exercise as a result of injury depressing.
8. When I have tried to reduce the amount I exercise I have generally been unsuccessful.
9. I worry that I have to exercise more and more to get the same 'buzz'.
10. Exercise does not make me feel good, but not exercising makes me feel worse.
11. Most of the time I exercise alone.
12. I often underestimate to others the amount of time I spend exercising.

Appendix

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Characteristics of the exercise dependent and non dependent group.</th>
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<tbody>
<tr>
<td></td>
<td>Non dependent</td>
</tr>
<tr>
<td>Age in years</td>
<td>19.0 (.94)</td>
</tr>
<tr>
<td>BMI in kg/m²</td>
<td>23.5 (4.93)</td>
</tr>
<tr>
<td>EDQ total score</td>
<td>69.6 (11.52)</td>
</tr>
<tr>
<td>EDQ subscales</td>
<td>13.8 (3.67)</td>
</tr>
<tr>
<td>Interference – social/family/work</td>
<td>7.4 (2.96)</td>
</tr>
<tr>
<td>Withdrawal symptoms</td>
<td>8.6 (3.03)</td>
</tr>
<tr>
<td>Exercise br weight control</td>
<td>7.3 (3.34)</td>
</tr>
<tr>
<td>High intake problem</td>
<td>10.4 (2.37)</td>
</tr>
<tr>
<td>Exercise br social reasons</td>
<td>10.0 (4.97)</td>
</tr>
<tr>
<td>Exercise br health reasons</td>
<td>6.8 (1.48)</td>
</tr>
<tr>
<td>Stereotyped behaviour</td>
<td>5.2 (2.00)</td>
</tr>
<tr>
<td>EDQ totalscore</td>
<td>443.0 (226.77)</td>
</tr>
</tbody>
</table>
Table 2
Baseline cardiovascular levels and cardiovascular reactivity for the exercise dependent and non dependent groups.

<table>
<thead>
<tr>
<th></th>
<th>Non dependent</th>
<th>Dependent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Reactivity</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>118.7 (5.16)</td>
<td>8.1 (6.52)</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>71.5 (637)</td>
<td>5.7 (5.67)</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>73.7 (9.02)</td>
<td>15.5 (9.58)</td>
</tr>
</tbody>
</table>

Fig. 1. Mean (SE) HR activity at baseline and during and after the acute stress task in exercise dependent and non dependent women.


### Table 3
Baseline cortisol levels and cortisol reactivity for the exercise dependent and non dependent groups.

<table>
<thead>
<tr>
<th>Cortisol (nmol/L)</th>
<th>Non dependent</th>
<th>Exercise dependent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>8.4 (4.54)</td>
<td>7.9 (2.75)</td>
</tr>
<tr>
<td>Reactivity 10 min post task</td>
<td>1.7 (2.33)</td>
<td>-.6 (1.12)</td>
</tr>
<tr>
<td>Reactivity 20 min post task</td>
<td>1.1 (3.33)</td>
<td>-1.9 (1.43)</td>
</tr>
<tr>
<td>Reactivity 30 min post task</td>
<td>.3 (2.23)</td>
<td>-2.0 (1.40)</td>
</tr>
</tbody>
</table>

![Graph showing cortisol levels](image)

**Fig. 2.** Mean (SE) salivary cortisol at baseline and at 10 minute, 20 minute, and 30 minute post stress tasks in exercise dependent and non dependent women.

### References


