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Low forced expiratory volume is associated with blunted cardiac reactions to acute psychological stress in a community sample of middle-aged men and women

Douglas Carroll [a,*], Anna C. Phillips [a], Geoff Der [b], Kate Hunt [b], Adam Bibbey [a], Michaela Benzeval [b], Annie T. Ginty [a]

[a] School of Sport and Exercise Sciences, University of Birmingham, Birmingham, United Kingdom
[b] MRC/CSO Social and Public Health Sciences Unit, Glasgow, Scotland, United Kingdom

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ABSTRACT

It has been argued recently that blunted cardiovascular reactions to acute psychological stress have adverse behavioural and health corollaries that reflect dysregulation of the neural systems that support motivation. We examined the association between cardiovascular reactions to a standard stress task, the paced auditory serial arithmetic rest, and forced expiratory volume in one second, an effort, hence motivation, dependent assessment of lung function measured by spirometry. Low forced expiratory volume, expressed as a ratio to height squared was associated with blunted heart rate, but not blood pressure, stress reactivity, $r = .17$, $p < .001$. The association survived adjustment for smoking, a range of anthropometric and sociodemographic covariates, resting heart rate and stress task performance, $\beta = .11$, $p = .005$. As such, our results provide support for the hypothesis that blunted stress reactivity may be a peripheral marker of a dysfunction in the brain systems that support motivated behaviour.

1. Introduction

Accumulating evidence implicates low or blunted cardiovascular and/or cortisol reactions to acute psychological stress in a range of adverse behavioural and health outcomes such as tobacco and alcohol dependence, as well as risk of dependence (al'Absi, 2006; al'Absi et al., 2005; Girdler et al., 1997; Lovallo, 2005; Panknin et al., 2002; Phillips et al., 2009; Roy et al., 1994), illicit substance use among adolescents (Brenner and Beauchaine, 2011), risk of re-offending in delinquent adolescents (De Vries-Bouw et al., 2011), exercise addiction (Heaney et al., 2011), depression and risk of depression (Carroll et al., 2007; de Rooij et al., 2010; Phillips et al., 2011; Rottenberg et al., 2007; Salomon et al., 2009; York et al., 2007), bulimia (Ginty et al., 2012; Koo-Loeb et al., 1998), and obesity and the risk of obesity (Carroll et al., 2008).

Although it is difficult to see the commonality among some of these apparently diverse outcomes, we have recently argued that they all are different manifestations of the same underlying central corollary of a deficient peripheral stress response. Specifically, all of these outcomes, to different degrees, reflect motivational dysregulation, i.e., a dysfunction of the neural systems that support motivated behaviour (Carroll et al., 2009, 2011). Areas within the greater amygdala system that converge at the striatum and ventromedial prefrontal cortex are not only implicated in the regulation of the stress response but also shape our feelings and the motivation of our behaviour (Carroll et al., 2009, 2011; Lovallo, 2005). There is at least preliminary evidence from imaging studies that areas within this system exhibit blunted reactions to pleasant stimuli in depressed patients relative to controls (Epstein et al., 2006), blunted reactions to food intake in those with a high body mass index (Stice et al., 2008), as well as reduced activation to a fear stimulus in those at high risk of alcoholism (Glahn et al., 2007). There is also some evidence that individuals who show blunted cardiovascular reactions to an acute psychological stress task show blunted neural reactions in the greater amygdala system to the same stress task (Gianaros et al., 2005, 2008).

If this speculation has any foundation, we would expect low or blunted stress reactivity to be associated with relatively poor performance on tasks that require psychological effort, i.e., are dependent, at least in part, on the integrity of central motivational processes. One outcome measure generally acknowledged to be effort, i.e., motivation, dependent is forced expiratory volume in one second, a widely used assessment of lung function measured by spirometry (Miller et al., 2005). Contributors to variability in forced expiratory volume measurement are reported to be failure of effort (Becklake, 1990) and differences in intrinsic motivation (Crim et al., 2011), as well, obviously, as lung function itself. We have recently found preliminary evidence that low forced expiratory volume is associated with blunted heart rate and cortisol stress reactions; the associations survived adjustment for smoking, a range of anthropometric

* Corresponding author at: School of Sport and Exercise Sciences, University of Birmingham, Birmingham B15 2TT, United Kingdom. Tel.: +44 121 414 7240; fax: +44 121 414 4121.
E-mail address: carrolld@bham.ac.uk (D. Carroll).

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and sociodemographic covariates, as well as commitment to the stress tasks (Carroll et al., 2012). Clearly, replication is essential. Accordingly, the present study examined the association between forced expiratory volume and cardiovascular stress reactivity in a different sample. We hypothesised that blunted cardiovascular reactivity would be associated low expiratory volume.

2. Materials and methods

2.1. Participants

Data were collected as part of the West of Scotland Twenty-07 Study. Participants were all from Glasgow and surrounding areas in Scotland and have been followed up at intervals since the initial baseline survey in 1987/8 (Benzeval et al., 2009). Full details of the sampling methodology and the structure of the sample are provided elsewhere (Carroll et al., 2008). The analyses here are of data from the third follow-up, at which the participants had forced expiratory volume in one second measured (FEV_1) measured and underwent standard cardiovascular stress testing. The West of Scotland study comprises three distinct age cohorts who were, aged on average, respectively 24, 44, and 64 years old at the time of the third follow-up. The three cohorts differed substantially in FEV_1 ($p<.001$). The present analyses focussed on the middle cohort, as FEV_1 ($M=3.62$ L) in the youngest cohort would be broadly optimal and, accordingly, considered to be less susceptible to variation as a result of motivational commitment. Similarly, FEV_1 ($M=2.35$ L) in the oldest cohort was suboptimal and again less likely to be affected by variations in psychological effort and motivation. The effective sample size for the present analyses was 600. Their mean (SD) age was 44.56 (0.84), so there was a modest age spread within the cohort. Ethical approval was gained from the local research ethics committee, with all the participants providing informed consent.

2.2. Apparatus and procedure

Testing sessions were conducted by trained nurses in a quiet room in the participants' homes. Demographic details at the third follow-up were obtained by questionnaire. Household socioeconomic status was characterised as manual or non-manual from the occupational status of the head of household, using the Registrar General classification system (Register General, 1980). Height and weight were measured. FEV_1 was measured using spirometry (Micro Medical Micro Plus Ms03 spirometer). Three measurements were taken from each participant, and the maximum expired volume achieved over the three recorded as FEV_1 (American Thoracic Society, 1987). The acute stress task was the paced auditory serial arithmetic test (PASAT), which has been shown in numerous studies to reliably perturb the cardiovascular system (Ring et al., 2002; Winzer et al., 1999), and to demonstrate good test–retest reliability (Willemsen et al., 1998). The nurses were all trained in administering the PASAT by the same trainer and followed a written protocol. The test comprised a series of single digit numbers presented by audiotape. The participants were required to add sequential number pairs, while at the same time retaining the second of the pair in memory to add to the next number presented. Answers were given orally and the correctness of the answers recorded as a measure of performance. The first sequence of 30 numbers was presented at a rate of one every 4 s, and the second at one every 2 s. The task lasted 3 min. Only those who registered a score on the PASAT were included in the analyses. Of a possible score of 60, the mean (SD) score was 44.3 (9.50).

2.3. Statistical analyses

FEV_1 was standardised by height^2 to take account of the different lung capacities of the participants of varying stature (Miller et al., 2007). Repeated measures (baseline, task) ANOVA was used to confirm that the PASAT significantly increased cardiovascular activity. η^2 was adopted as a measure of effect size. Reactivity was calculated as mean task value minus the baseline value. The associations between cardiovascular reactivity and FEV_1 were examined by correlation and then by multiple linear regression using the following covariates: age, sex, socioeconomic status, smoking, weight, PASAT performance, and the appropriate resting cardiovascular activity. These covariates were chosen because previous research has shown that they are associated with cardiovascular stress reactivity and/or FEV_1 in this and other samples (Carroll et al., 2011, 2012). The covariates were always entered at step 1 and reactivity at step 2. ΔR^2 was used to indicate effect size.

3. Results

3.1. Sample characteristics

The characteristics of the sample are presented in Table 1. Mean (*SD*) FEV_1 was 3.05 (0.72) L.

3.2. Cardiovascular stress reactivity

The stress successfully perturbed cardiovascular activity. SBP, $F(1599)=718.12$, $p<.001$, $\eta^2=.545$, DBP, $F(1599)=467.92$, $p<.001$, $\eta^2=.439$, and HR, $F(1,599)=384.49$, $p<.001$, $\eta^2=.391$, all increased from baseline to stress task exposure. Table 2 presents the summary data.

3.3. FEV_1 and cardiovascular stress reactivity

FEV_1/height^2 was positively correlated with HR reactivity, $r(598)=.17$, $p<.001$, but not with SBP, $r(598)=.07$, $p=.11$, or DBP, $r(598)=.07$, $p=.10$, reactivity: thus, the lower the FEV_1/height^2, the smaller the HR reactions to stress. This association is illustrated by plotting tertiles of HR reactivity against FEV_1/height^2 (see Fig. 1). In a multiple regression model, that adjusted for age, sex, socioeconomic status, smoking, weight, PASAT performance, and resting HR, the positive association between HR reactivity and FEV_1/height^2 was slightly attenuated but remained statistically significant, $\beta=.11$, $p=.005$, $\Delta R^2=.010$. In this model, FEV_1/height^2 was also lower in smokers, women, those who were older, and those from the manual socioeconomic status group. In similar analyses, there was no association between either SBP reactivity ($p=.96$) or DBP reactivity ($p=.90$) and FEV_1/height^2.

4. Discussion

FEV_1 standardised by height^2, was positively associated with acute stress reactivity. As hypothesised, low FEV_1 was associated with blunted HR reactions to a standard mental stress task. This association was evident not only from correlational analyses but also in regression models that adjusted for age, sex, socioeconomic status, smoking, weight, PASAT performance, and resting HR. The present findings are very much in line with those that emerged from analyses of a different data set and reactions to a different stress task; low FEV_1 was again associated with blunted HR reactivity (Carroll et al., 2012). Accordingly, they add further support to the contention that blunted physiological stress reactivity may not necessarily be an adaptive response. Rather, they may reflect central motivational

Table 1
Characteristics of the sample.

Variable	Mean/N	SD/%
Age	44.6	0.84
Sex (male)	280	44.9
Non-manual occupation group	346	55.4
Height (cm)	166.4	9.48
Weight (kg)	72.8	33
Current smoker	218	13.92
PASAT performance score	44.3	9.50

Table 2
Mean (*SD*) SBP, DBP, and HR baseline, during PASAT, and reactivity.

	Baseline	During PASAT	Reactivity
Systolic blood pressure (mmHg)	127.3 ± 18.14	139.7 ± 18.72	12.4 ± 11.35
Diastolic blood pressure (mmHg)	80.7 ± 11.13	87.7 ± 11.31	7.0 ± 7.92
Heart rate (bpm)	66.6 ± 11.09	74.5 ± 12.12	7.9 ± 9.88

dysregulation (Carroll et al., 2009, 2011), and be associated with outcomes that reflect, whether explicitly or inadvertently, individual variations in psychological effort and motivation. That the association between FEV_1 and HR reactivity withstood adjustment for PASAT performance argues against the parsimonious explanation that individuals who do not engage fully in an assessment of FEV_1 will similarly fail to engage with psychological stress tasks, and hence register lower reactivity. Rather, we would argue that a more nuanced and covert process provides a better account of the associations we observe; it is physiological disengagement, reflecting central motivational dysregulation, rather than psychological disengagement that underlies the association between FEV_1 and stress reactivity. The association also withstood adjustment for key confounders which might be expected to relate to FEV and/or reactivity including age, sex, occupational group, smoking status, weight, and baseline cardiovascular measures.

Only HR reactivity was significantly associated with FEV_1; there were no consistent associations between blood pressure reactivity and FEV_1. This was also the case in our earlier study of a different sample (Carroll et al., 2012). As HR reflects both β-adrenergic and parasympathetic influences, low HR reactivity could reflect reduced β-adrenergic drive or less of a reduction in vagal tone during the stress tasks (Balanos et al., 2010; Sloan et al., 1991). However, previous research would seem to suggest that a variation in β-adrenergic activation is the primary source of individual differences in HR reactivity during psychological stress. Although blood pressure is also affected by β-adrenergic influences, SBP and DBP reactivity would seem less determined by β-adrenergic activation than is the case for HR activation (Balanos et al., 2010). For example, β-adrenergic blockade has been observed to attenuate cardiac reactivity, but not SBP or DBP reactivity (Winzer et al., 1999).

There is accumulating evidence that low magnitude stress reactivity has a range of adverse behavioural and health corollaries (Carroll et al., 2009, 2011). The findings of the present study not only add to that range but also suggest something about the underlying meaning of blunted stress reactivity: rather than being an adaptive response, it is more likely that it constitutes a peripheral marker of a dysfunction in those neural systems that support motivation and effortful behaviour. The systems implicated would also appear to be those involved in the regulation of peripheral stress reactivity; accordingly, motivational dysregulation and deficient β-adrenergic activation during acute stress exposure may have common neural origins (Carroll et al., 2009, 2011). Neural imaging studies are clearly required to confirm this hypothesis.

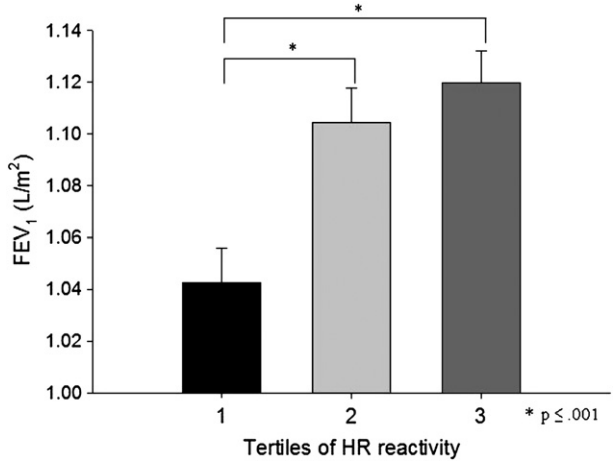

Fig. 1. Unadjusted mean (SE) $FEV_1/height^2$ by tertiles of heart rate reactivity.

The present study is not without limitations. First, it remains possible that our findings are a product of a confounding by a third variable (Christenfeld et al., 2004). However, it is difficult to envisage what that variable might be given that we were able to discount age, height, weight, baseline HR, sex, socioeconomic status, smoking, and stress task performance. Second, it should be acknowledged that the observed effect sizes are small. However, our effects are of the same order as or larger than the positive associations between cardiovascular stress reactivity and adverse cardiovascular outcomes reported previously (Carroll et al., 1995, 2001, 2003; Markovitz et al., 1998; Matthews et al., 1993; Newman et al., 1999). Finally, we had to rely on stress task performance score as our measure of engagement. Although this seems reasonable, in retrospect it might have proved useful to have included self-report measures of stress task impact.

5. Conclusion

The present analyses show a robust positive association between FEV_1, an effort, and hence motivation, dependent measure of lung function, and HR reactions to acute stress; blunted HR reactivity was related to low FEV_1. As such, the study confirms another correlate to the growing list of the corollaries of blunted stress reactivity. More importantly, our results support the notion that blunted stress reactivity may reflect a general dysfunction of the neural systems that supports motivated and goal-directed behaviour.

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