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

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ARTICLE INFO

Article history:
Received 6 August 2012
Received in revised form 24 September 2012
Accepted 5 October 2012
Available online 13 October 2012

Keywords:
Forced expiratory volume
Heart rate
Blood pressure
Stress reactivity

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, β = .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.

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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 Roodj 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., 2005), 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
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 (FEV1) 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 FEV1 (p < .001). The present analyses focussed on the middle cohort, as FEV1 (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, FEV1 (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. FEV1 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 FEV1 (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 administration of the PASAT by the same trainer and followed a written protocol. The test comprised a series of single digit numbers presented by audiotaape. The participants were required to add sequential number sequences at one per second measured (FEV1) 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 FEV1 (p < .001). The present analyses focussed on the middle cohort, as FEV1 (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, FEV1 (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.

2.3. Statistical analyses

FEV1 was standardised by height² 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. ƞ² 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 FEV1 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 FEV1 in this and other samples (Carroll et al., 2011, 2012). The covariates were always entered at step 1 and reactivity at step 2. ΔR² was used to indicate effect size.

3. Results

3.1. Sample characteristics

The characteristics of the sample are presented in Table 1. Mean (SD) FEV1 was 3.05 (0.72) L.

3.2. Cardiovascular stress reactivity

The stress successfully perturbed cardiovascular activity. SBP, F (1599) = 718.12, p < .001, ƞ² = .549, DBP, F (1599) = 467.92, p < .001, ƞ² = .439, and HR, F (1599) = 384.49, p < .001, ƞ² = .391, all increased from baseline to stress task exposure. Table 2 presents the summary data.

3.3. FEV1 and cardiovascular stress reactivity

FEV1/height² 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 FEV1/height², the smaller the HR reactions to stress. This association is illustrated by plotting tertiles of HR reactivity against FEV1/height² (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 FEV1/height² was slightly attenuated but remained statistically significant, β = .11, p = .005, ΔR² = .010. In this model, FEV1/height² 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 FEV1/height².

4. Discussion

FEV1 standardised by height², was positively associated with acute stress reactivity. As hypothesised, low FEV1 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 FEV1 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>
<thead>
<tr>
<th>Variable</th>
<th>Mean/N</th>
<th>SD/%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>44.6</td>
<td>0.84</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>280</td>
<td>44.9</td>
</tr>
<tr>
<td>Non-manual occupation group</td>
<td>346</td>
<td>55.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168.4</td>
<td>9.48</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72.8</td>
<td>33</td>
</tr>
<tr>
<td>Current smoker</td>
<td>218</td>
<td>13.92</td>
</tr>
<tr>
<td>PASAT performance score</td>
<td>44.3</td>
<td>9.50</td>
</tr>
</tbody>
</table>
The systems implicated would also appear to be those involved in the neural systems that support motivation and effortful behaviour. More likely that it constitutes a peripheral marker of a dysfunction in blunted stress reactivity: rather than being an adaptive response, it is range but also suggest something about the underlying meaning of dysregulation and deficient \( \beta \)-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. The present study is not without limitations. First, it remains possible that our findings are a product of a confounding by a third variable (Christensen 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.

Acknowledgements

The West of Scotland Twenty-07 Study is funded by the UK Medical Research Council (MC_US_A540_53462) and the data were originally collected by the MRC Social and Public Health Sciences Unit. We are grateful to all of the participants in the Study, and to the survey staff and research nurses who carried it out. The data are employed here with the permission of the Twenty-07 Steering Group (Project No. EC201003). MB (MC_US_A540_5TK10), GD (MC_US_A540_STK30) and KH (MC_US_A540_STK50) are funded by the UK Medical Research Council.

References


| 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 |

Fig. 1. Unadjusted mean (SE) FEV\(_1\)/height\(^2\) by tertiles of heart rate reactivity.