Forced expiratory volume is associated with cardiovascular and cortisol reactions to acute psychological stress

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Abstract
It has been argued that blunted cardiovascular and cortisol reactions to acute psychological stress reflect a dysregulation of the neural system that supports motivation. We examined the association between forced expiratory volume in one second, an effort, hence motivation, dependent measure of lung function measured by spirometry, and cardiovascular and cortisol reactions to a battery of standard psychological stress tasks (Stroop, mirror-tracing, speech), assessed seven years later among 435 men and women from the Dutch Famine Birth Cohort Study. Irrespective of how it was expressed, low forced expiratory volume was associated with blunted heart rate and cortisol stress reactivity. The association survived adjustment for smoking, a range of anthropometric and sociodemographic covariates, and commitment to the stress tasks, as well as cognitive ability.

Descriptors: Forced expiratory volume, Heart rate, Blood pressure, Stress reactivity
Recent 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, Hatsukami, & Davis, 2005; Girdler, Jamner, Jarvik, Soles, & Shapiro, 1997; Lovallo, Dickensheets, Myers, Thomas, & Nixon, 2000; Panknin, Dickensheets, Nixon, & Lovallo, 2002; Phillips, Der, Hunt, & Carroll, 2009; Roy, Steptoe, & Kirschbaum, 1994), illicit substance use among adolescents (Brenner & Beauchaine, 2011), exercise addiction (Heaney, Ginty, Carroll, & Phillips, 2011), depression and risk of depression (Carroll, Phillips, Hunt, & Der, 2007; de Rooij, Schene, Phillips, & Roseboom, 2010; Phillips, Hunt, Der, & Carroll, 2010; Rottenberg, Clift, Bolden, & Salomon, 2007; Salomon, Clift, Karlsdottir, & Rottenberg, 2009; York, et al., 2007), bulimia (Ginty, Phillips, Higgs, Heaney, & Carroll, in press; Koo-Loeb, Pedersen, & Girdler, 1998), obesity and the risk of obesity (Carroll, Phillips, & Der, 2008), and risk of re-offending in delinquent adolescents (De Vries-Bouw, et al., 2011).

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, Phillips, & Lovallo, 2009; Carroll, Phillips, & Lovallo, 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, Phillips, & Lovallo, 2009; Carroll, Phillips, & Lovallo, 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, Spoor, Bohon, & Small, 2008), as well as reduced activation to a fear stimulus in those at high risk of alcoholism (Glahn, Lovallo, & Fox, 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 amydala system to the same stress task (Gianaros, May, Siegle, & Jennings, 2005; Gianaros, et al., 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. We have recently shown associations between blunted stress reactivity and poor cognitive performance on a range of tasks, reasoning, reaction time and memory, that were independent of the tasks used to elicit stress reactions (Ginty, Phillips, Der, Deary, & Carroll, 2011a; Ginty, Phillips, Der, Deary, & Carroll, 2011b; Ginty, Phillips, Roseboom, Carroll, & de Rooij, in press). 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). We have recently shown that low cognitive ability in early adulthood is associated with reduced
forced expiratory volume in middle age (Carroll, Batty, Mortensen, Deary, & Phillips, 2011). A cross-sectional positive relationship between forced expiratory volume and cognitive ability in boys suffering from muscular dystrophy has also been reported (Gauld, Boynton, Betts, & Johnston, 2005). In addition, this study showed that increased visual incentives improved forced expiratory volume.

Accordingly, if forced expiratory volume assessed by spirometry is, to an extent, a measure of motivation, albeit an inadvertant one, we might expect those who perform more poorly to be characterised by blunted stress reactions to acute psychological stress. Data collected as part of the Dutch Famine Birth Cohort Study allowed us to examine this question. We hypothesised that those who showed relatively low forced expiratory volumes would also show blunted cardiovascular and cortisol reactions to acute psychological stress.

Method

Participants

Participants were selected from the Dutch Famine Birth Cohort, which comprises 2414 men and women who were born in Amsterdam, the Netherlands, between November 1943 and February 1947. The selection procedures and subsequent loss to follow up have been described in detail elsewhere (Painter, et al., 2005; Ravelli, et al., 1998). The Dutch Famine Birth Cohort Study was designed to investigate the potential consequences of prenatal exposure to famine on health in later life. It might therefore be suggested that population characteristics may hamper generalization of the present study results. However, this is very unlikely as health differences
have mainly been found in the group of people exposed to famine in early gestation which only comprises 8% of the total study sample. Seven hundred and forty-one of the cohort attended a clinical assessment between 1995 and 1997 during which lung function was measured and 740 attended between 2002 and 2004 during which time cardiovascular and cortisol reactions to acute psychological stress were measured. The effective sample size for the present analyses was 435 (211 men, 224 women), i.e., the participants who provided data on both lung function and stress reactivity. Their mean (SD) age at the time of stress testing was 58.3 (0.90) years. The study was approved by the local Medical Ethics Committee and carried out in accordance with the Declaration of Helsinki and the informed written consent of the participants.

**General Study Parameters**

Between 2002 and 2004 trained research nurses undertook anthropometric measurements and conducted a standardized interview in which, among other things, information was obtained about socio-economic status (SES) and lifestyle. Participants were characterised as current smokers, ex-smokers, or never smokers. Height was measured twice using a fixed or portable stadiometer and weight twice using Seca and portable Tefal scales. SES was defined according to the International Socio-Economic Index (ISEI)-92, which is based on the participant’s or their partner’s occupation, whichever has the higher status (Bakker & Sieben, 1997). Values in the ISEI-92 scale ranged from 16 (low status) to 87.

**Lung function**
Lung function was measured between 1995 and 1997 as forced expiratory volume in one second (FEV₁) using spirometry. Participants underwent spirometric tests carried out by specially trained research nurses using the Vitalograph Compact (Vitalograph Ltd, Buckingham, UK). Participants were seated and used a nose clip. After an acceptable technique had been achieved during a series of practice blows, forced expiratory volume in one second (FEV₁) and forced vital capacity (FVC) were measured at least three times. If the difference between the highest and the second highest values of FEV₁ or FVC was more than 5%, further measurements up to a maximum of eight were performed to improve reproducibility (American Thoracic Society, 1987). The highest values FEV₁ were used in the analysis.

**Stress reactivity**

The stress testing protocol, which started in the afternoon, between the hours of 12.00 – 14.00, began with a 20-minute baseline period after which three psychological stress tasks were performed: Stroop, mirror tracing, and a speech task. Each stress task lasted 5 minutes, with 6 minutes in between, and 30 minutes of recovery following the final stress task. The Stroop task consisted of a single-trial computerized version of the classic Stroop colour-word conflict challenge. After a short introduction, participants were allowed to practise until they fully understood the requirements of the task. Errors and exceeding the response time limit of 5 seconds triggered a short auditory beep. For the mirror-tracing task, a star had to be traced that could only be seen in mirror image (Lafayette Instruments Corp, Lafayette, IN, USA). Every divergence from the line triggered an auditory stimulus. They were allowed to practice one circuit of tracing. Participants were instructed to prioritize accuracy over speed and were told
that most people could perform five circuits of the star without divergence from the line within the given 5 minutes. Prior to the speech task, participants listened to an audio taped instruction in which they were told to imagine a situation in which they were falsely accused of pick pocketing. They were then given 2 minutes to prepare a 3-minute speech in which they had to respond to the accusation. The speech was videotaped and participants were told that the number of repetitions, eloquence, and persuasiveness of their performance would be assessed by a team of communication experts and psychologists. After completion of each of the stress tasks, participants completed 7-point rating scale of stress task impact, including participants’ commitment to the tasks.

Continuous blood pressure (BP) and heart rate (HR) recordings were made using a Finometer or a Portapres Model-2 (Finapres Medical Systems, Amsterdam, the Netherlands). There were no differences in reactivity as a function of the two different measuring devices. Six periods of 5 minutes were designated as the key measurement periods: resting baseline (15 minutes into the baseline period), Stroop, mirror-tracing, speech task (including preparation time), recovery 1 (5 minutes after completing the speech task) and recovery 2 (25 minutes after completing the speech task). Mean systolic blood pressure (SBP), diastolic blood pressure (DBP) and HR were calculated for each measuring period. A total of seven saliva samples were collected using Salivettes (Sarstedt, Rommelsdorf, Germany): at 5 and 20 minutes of the baseline period, at 6 minutes following completion of the Stroop task and the mirror tracing task, and at 10, 20 and 30 minutes after completion of the speech task. Salivary cortisol concentrations were measured using a time-resolved immunofluorescent assay (DELFIA) (Wood, Kilpatrick, & Barnard, 1997).
The assay had a lower detection limit of 0.4 nmol/l and an inter-assay variance of 9-11% and an intra-assay variance of less than 10%.

**Statistical Analyses**

Baseline cortisol was computed as the mean of the first and the second cortisol concentration measures during the baseline period. Cortisol reactivity was derived as a difference between the cortisol concentration measured 20 minutes after completion of the final stress task (speech) and baseline cortisol. Baseline cardiovascular activity was the average of values recorded in the 5-minute period 15 minutes into the baseline. BP and HR measures were averaged across each of the three stress tasks, and the mean of these three averaged then determined. Cardiovascular stress reactivity was defined as the difference between the overall stress mean value and baseline for each of the three cardiovascular variables. FEV₁ was characterised as FEV₁/height² and as a percentage of predicted FEV₁ (Miller, Pedersen, & Dirksen, 2007), which was calculated using a standard algorithm involving age and height (Quanjer, et al., 1993). A number of participants provided insufficient saliva for cortisol determination. Accordingly, the effective sample for the cortisol analyses was 347. Other slight variations in degrees of freedom reflect occasional missing data for some variables.

Repeated measures ANOVAs were used to test for differences between baseline and stress task physiological activity. The associations between FEV₁ and cardiovascular and cortisol stress reactivity were analysed using multiple linear regression with reactivity as the dependent variable, first in models that adjusted only for age, and then in models that additionally adjusted
for baseline physiological activity appropriate to the dependent variable, sex, SES, weight, smoking, and stress task commitment. These covariates were selected as they have been shown to be related to stress reactivity and/or FEV$_1$ in this and other population studies (Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Carroll, Batty, Mortensen, Deary, & Phillips, 2011). Further, as low cognitive ability in this sample is associated with blunted HR and cortisol reactivity in this cohort (Ginty, Phillips, Roseboom, Carroll, & de Rooij, in press), and low cognitive ability is also associated with low FEV$_1$ (Carroll, Batty, Mortensen, Deary, & Phillips, 2011), the analyses were repeated adjusting for all the above covariates plus cognitive ability. Cognitive ability was measured by percentage correct scores on the Alice Heim-4 a test of general mental ability (Heim, 1970). We have described the test, which comprises 33 items measuring numerical reasoning ability and 32 items measuring verbal reasoning ability, and its administration in detail elsewhere (Ginty, Phillips, Roseboom, Carroll, & de Rooij, in press). Regression analyses were first undertaken using FEV$_1$/height$^2$ as the independent variable and then with FEV$_1$ as a percentage of predicted FEV$_1$.

Results

Descriptives

The mean (SD) SES score for participants was 49.1 (0.67) at the time of stress testing. Mean (SD) height and weight were 1.70 (0.43) m and 82.88 (0.74) kg. The mean (SD) sum of the task
commitment ratings across the three stress tasks was 14.76 (0.20). Ninety-eight of the participants were current smokers, 168 were ex-smokers, and 168 indicated that they had never smoked.

**Cardiovascular and cortisol stress reactivity**

Repeated measures ANOVA (baseline, task) indicated that the stress test battery provoked significant increases in SBP, $F(1,431) = 1751.98, p < .001, \eta^2 = .803$, DBP, $F(1,431) = 1931.09, p < .001, \eta^2 = .818$, and HR, $F(1,431) = 482.29, p < .001, \eta^2 = .528$. Stress exposure also elicited a significant increase in cortisol, $F(1, 346) = 54.97, p < .001, \eta^2 = .137$. The summary statistics are presented in Table 1.

Forced expiratory volume and stress reactivity

The mean (SD) FEV$_1$ for the cohort as a whole was 3.13 (0.03) L and FEV$_1$ as a percentage of predicted FEV$_1$ was 100.25 (0.77). FEV$_1$/height$^2$ was not significantly associated with either SBP ($p = .09$) or DBP ($p = .43$) reactivity in the age adjusted regression model. The same was true when FEV$_1$ was expressed as a percentage of predicted FEV$_1$ ($p = .15$ and .81, respectively). However, FEV$_1$/height$^2$ was significantly associated with both HR, $\beta = .19, p < .001, \Delta R^2 = .030$, and cortisol, $\beta = .17, p = .002, \Delta R^2 = .024$, reactivity in this model; the lower the FEV$_1$, the lower
the stress reactivity. These associations are illustrated in Figures 1 and 2, which depict mean HR and cortisol reactivity for tertiles of FEV$_1$/height$^2$. The same outcomes emerged when FEV$_1$ was expressed as a percentage of predicted FEV$_1$, $\beta = .19$, $p < .002$, $\Delta R^2 = .032$ and $\beta = .13$, $p = .02$, $\Delta R^2 = .017$, for HR and cortisol reactivity respectively.

In the model that additionally adjusted for baseline physiological activity appropriate to the dependent variable, sex, SES, weight, smoking, and stress task commitment, FEV$_1$/height$^2$ continued to predict both HR, $\beta = .14$, $p = .009$, $\Delta R^2 = .015$, and cortisol, $\beta = .13$, $p = .04$, $\Delta R^2 = .012$, reactivity. In this model, smoking ($p = .005$) and weighing more ($p < .001$) were also associated with blunted HR reactivity, and there was a tendency for men to exhibit lower HR reactions than women ($p = .09$). Similarly, being male ($p = .002$), smoking ($p = .02$), and being heavier ($p = .04$) were also associated with blunted cortisol stress reactions. In this fully adjusted model, FEV$_1$/height$^2$ was still not associated with SBP ($p = .56$) and DBP ($p = .43$) reactivity.

Almost exactly the same outcomes emerged from this fully adjusted regression model with FEV$_1$ represented as a percentage of predicted FEV$_1$: $\beta = .13$, $p = .006$, $\Delta R^2 = .017$, for HR reactivity and, $\beta = .13$, $p = .02$, $\Delta R^2 = .015$, for cortisol reactivity. Finally, we additionally adjusted for cognitive ability. The mean (SD) percentage correct score for the Alice Heim-4 was 70.1 (15.11). The associations between FEV$_1$/height$^2$ and HR, $\beta = .13$, $p = .02$, $\Delta R^2 = .013$, and cortisol, $\beta = .13$, $p = .04$, $\Delta R^2 = .011$, stress reactivity remained statistically significant. The same was true with FEV$_1$ expressed as a percentage of predicted FEV$_1$: $\beta = .13$, $p = .02$, $\Delta R^2 = .014$
and $\beta = .13$, $p = .02$, $\Delta R^2 = .014$ for HR and cortisol reactivity, respectively. The outcomes were virtually identical, i.e., FEV$_1$ continued to be positively related to HR and cortisol reactivity when we additionally adjusted for whether or not participants were exposed to the Dutch famine in utero.

**Discussion**

FEV$_1$, whether represented as FEV$_1$/height$^2$ or as a percentage of predicted FEV$_1$ was positively associated with acute stress reactivity. Low FEV$_1$ was related to blunted cardiac and cortisol reactions seven years later. This emerged in regression models that adjusted only for age and in models that additionally adjusted for baseline physiological activity appropriate to the dependent variable, sex, SES, weight, smoking, stress task commitment, as well as cognitive ability. As such, our findings are in line with our hypothesis and support our conjecture that blunted physiological stress reactivity is not necessarily an adaptive response and may reflect central motivational dysregulation (Carroll, Lovallo, & Phillips, 2009; Carroll, Phillips, & Lovallo, 2011), and be associated with outcomes that reflect, whether explicitly or inadvertently, individual variations in psychological effort and motivation. With the exception of a small positive correlation with HR reactivity ($r = .10$, $p = .04$), reported stress task commitment was not significantly associated with stress reactivity. As might be expected, stress task commitment was not related to FEV$_1$ measured at the earlier time point ($p > .25$). This and the fact the associations between FEV$_1$ and reactivity withstood adjustment for task commitment 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 reactivity. As smoking affects FEV\(_1\) and is also associated with blunted cardiovascular and cortisol stress reactivity (al'Absi, 2006; al'Absi, Hatsukami, & Davis, 2005; Girdler, Jamner, Jarvik, Soles, & Shapiro, 1997; Phillips, Der, Hunt, & Carroll, 2009; Roy, Steptoe, & Kirschbaum, 1994), it would seem reasonable to propose that smoking could explain the relationship between FEV\(_1\) and reactivity. However, the present associations survive adjustment for smoking status which very much argues against such an account.

Of our cardiovascular measures, only HR reactivity was significantly associated with FEV\(_1\) measured seven years earlier. There were no consistent associations between blood pressure reactivity and FEV\(_1\). Heart rate reflects both β-adrenergic and parasympathetic influences. Thus, low heart rate reactivity could reflect reduced β-adrenergic drive or less of a reduction in vagal tone during the stress tasks (Balanos, et al., 2010; Sloan, Korten, & Myers, 1991). However, previous research would seem to suggest that variations 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). Some have proposed that β-adrenergic activation and HPA activation are dissociated in some circumstances

(Dickerson & Kemeny, 2004; Frankenhaeuser, 1982). However, there is substantial evidence that they frequently covary, such that variations in the magnitude of $\beta$-adrenergic activation, indexed by cardiac stress reactivity, predict subsequent variations in HPA reactions, indexed by cortisol stress reactivity (Al'Absi, et al., 1997; Bosch, et al., 2009; Cacioppo, 1994). It is worth noting that cortisol reactivity in the present study was more strongly correlated with HR reactivity ($r = .38$) than it was with either SBP ($r = .24$) and DBP reactivity ($r = .12$). Tests of difference between correlation coefficients indicates that the correlation for HR reactivity is significantly larger than the other two ($p < .05$).

There is compelling evidence that large magnitude cardiovascular reactions to acute psychological stress are a risk marker for high blood pressure and hypertension (Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Carroll, Smith, Sheffield, Shipley, & Marmot, 1995; Carroll, et al., 2001; Everson, Kaplan, Goldberg, & Salonen, 1996; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998; Matthews, Woodall, & Allen, 1993; Newman, McGarvey, & Steele, 1999; Treiber, Turner, Davis, & Strong, 1997), systemic atherosclerosis (Barnett, Spence, Manuck, & Jennings, 1997; Everson, et al., 1997; Lynch, Everson, Kaplan, Salonen, & Salonen, 1998; Matthews, et al., 1998), and left ventricular mass and/or hypertrophy of the heart (Georgiades, Lemne, de Faire, Lindvall, & Fredrikson, 1997; Kapuku, et al., 1999; Murdison, et al., 1998). However, there is also accumulating evidence that low magnitude stress reactivity, including cortisol reactivity, has a range of adverse behavioural and health corollaries (Carroll, Phillips, & Lovallo, 2009; Carroll, Phillips, & Lovallo, 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. Further neural imaging studies are clearly required to confirm this hypothesis.

The present study is not without limitations. First, FEV\(_1\) and cardiovascular and cortisol stress reactivity were measured at different time points, seven years apart. However, reactivity and FEV\(_1\) appear to be fairly stable over time (Hassellund, Flaa, Sandvik, Kjeldsen, & Rostrup, 2010; Hnizdo, et al., 2007), and it is likely that temporal separation of measurement would, if anything, have underestimated the association between FEV\(_1\) and stress reactivity. Second, it remains possible that our findings are a product of a confounding by some unmeasured variable (Christenfeld, Sloan, Carroll, & Greenland, 2004). However, we were able to discount age, height, weight, baseline physiological activity, sex, SES, smoking, and stress task commitment. Third, it should be acknowledged that the observed effect sizes are small. However, our effects for cardiovascular reactivity are of the same order as or larger than the positive associations between cardiovascular reactivity and future resting blood pressure in other studies (Carroll, et al., 1995; Carroll, et al., 1995; (Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Carroll, et al., 2001; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998; Matthews, Woodall, & Allen, 1993; Newman, McGarvey, & Steele, 1999). Fourth, this is a unique population and it has been suggested that early life adversity may predispose individuals to life-long vulnerability to stress. However, a previous study using this population showed that individuals who experienced prenatal exposure to the Dutch Famine did not differ in cortisol stress reactivity from those who did not (de Rooij, et al., 2006). Fifth, it was not possible to derive performance scores for the all
stress tasks used in this study. Nevertheless, we do have a measure of commitment to the task and have included this as a covariate in the fully adjusted analyses. Finally, we cannot distinguish between the motivational aspects of individual variations in FEV₁ and individual variations in lung function per se. However, there are no compelling reasons we know of to suspect a positive association between stress reactivity and lung function, independently of smoking. On the other hand, as we have indicated there are at least grounds for hypothesising an association between blunted stress reactivity and dysfunctional motivation and hence poor performance in tasks that are effort, i.e., motivation, dependent.

In conclusion, the present analyses show a robust positive association between FEV₁, a motivation dependent measure of lung function, and HR and cortisol reactions to acute stress. As such, the study adds another correlate to the growing list of the corollaries of blunted stress reactivity. More importantly, our results support the notion that blunted reactivity may reflect a general dysfunction of the neural system that supports motivated behaviour.

References


Table 1. Mean (SD) cardiovascular and cortisol activity at baseline and during, and in the case of cortisol after, stress task exposure

<table>
<thead>
<tr>
<th></th>
<th>SBP mmHg</th>
<th>DBP mmHg</th>
<th>HR bpm</th>
<th>Cortisol nmol/L</th>
</tr>
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<tbody>
<tr>
<td>Baseline</td>
<td>128.6 (20.64)</td>
<td>66.0 (11.98)</td>
<td>74.0 (10.62)</td>
<td>4.65 (3.34)</td>
</tr>
<tr>
<td>Stress*</td>
<td>161.2 (25.22)</td>
<td>81.5 (13.38)</td>
<td>80.9 (12.41)</td>
<td>6.44 (4.87)</td>
</tr>
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*Stress differed from baseline for all four variables, \( p < .001 \) in each case.
Figure 1. Mean (SE) heart rate reactivity by tertiles of \( \text{FEV}_1/\text{height}^2 \).

Figure 2. Mean (SE) cortisol reactivity by tertiles of \( \text{FEV}_1/\text{height}^2 \).