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ORIGINAL RESEARCH REPORT

School burnout and cardiovascular functioning in young adult males: a hemodynamic perspective

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Abstract

This study investigated aortic and brachial hemodynamic functioning that may link school burnout to cardiovascular risk factors. Methodological improvements from previous research were implemented including (1) statistical control of depressive and anxiety symptoms (2) resting, stress-induced and cardiac recovery condition comparisons and (3) use of pulse wave analysis. Forty undergraduate young adult males completed self-report measures of school burnout, trait anxiety and depressive symptoms. Participants then completed a protocol consisting of a 10-min seated rest, 5-min baseline (BASE), 3-min cold pressor test (CPT) and a 3-min recovery period (REC). Indices of brachial and aortic hemodynamics were obtained by means of pulse wave analysis via applanation tonometry. Controlling for anxiety and depressive symptoms, planned contrasts identified no differences in cardiovascular parameters at BASE between participants in burnout and non-burnout groups. However, negative changes in hemodynamic indices occurred in burnout participants at CPT and REC as evidenced by increased aortic and brachial systolic and diastolic blood pressures, increased left ventricular work and increased myocardial oxygen consumption. Findings suggest that school burnout symptoms are associated with cardiac hyperactivity during conditions of cardiac stress and recovery and therefore may be associated with the early manifestations of cardiovascular disease. Future studies are suggested to reveal underlying autonomic mechanisms explaining hemodynamics functioning in individuals with school burnout symptomatology.

Keywords

Cardiovascular, cold pressor test, hemodynamics, pulse wave analysis, school burnout, stress

History

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Introduction

Burnout is a multidimensional affective response to stress that has been identified as a risk factor for a host of psychological, psychosocial and physiological ailments including cardiovascular diseases (CVDs) (Kahn & Byosiere, 1992; Melamed et al., 2006; Schaufeli & Buunk, 2003; Shirom, 2003). Although burnout has traditionally been regarded as a work-related disorder (Halbesleben & Buckley, 2004; Maslach et al., 2001) it has recently been applied to educational populations (Kiuru et al., 2008; Salmela-Aro et al., 2009). Within an educational context, school burnout is characterized by chronic exhaustion from school-related work, cynicism toward the meaning of school and feelings of inadequacy toward school related accomplishments (Salmela-Aro et al., 2009). However, school burnout research is limited as the potential physiological impact of school burnout on cardiovascular functioning and risk has yet to be explored. The current study therefore investigated cardiovascular functioning associated with school burnout via pulse wave analysis (PWA).

Research relating (work) burnout to CVD has primarily focused on two mediating physiological stress systems – the sympatheticadrenergic–medullary (SAM) axis and the hypothalamicpituitary–adrenal (HPA) axis – underemphasizing the imbalance of hemodynamic (i.e. blood circulation) functioning due largely to inconsistent and equivocal findings (Danhof-Pont et al., 2011; De Vente et al., 2003; Melamed et al., 2006; van Doornen et al., 2009). It has been argued that previous research investigating burnout that does not adequately account for the influence of related affective symptomatology (especially depressive and anxiety symptoms as suggested by Melamed et al., 2006; Schaufeli & Buunk, 2003; Shirom, 2009), lacks analysis of cardiovascular reactivity (CVR) and recovery (as suggested by Manuck, 1994; Rottenberg et al., 2007; Treiber et al., 2003) and is deficient in the measurement of both peripheral (brachial) and central (aortic) hemodynamics (as suggested by McEniery et al., 2008; Roman et al., 2009) may have contributed to equivocal findings precluding a clear picture of the relationship between burnout, hemodynamics and CVD risk.

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Accordingly, the present study examined hemodynamic functioning as a potential physiological link between school burnout and increased CVD risk by (1) controlling for depressive and anxiety symptoms, (2) evaluating CVR and recovery comparisons and (3) using PWA via applanation tonometry to examine aortic hemodynamics. We hypothesized that individuals with high school burnout would display increased cardiac reactivity (i.e. increased brachial and aortic blood pressure (BP), wave reflection, left ventricular work and myocardial oxygen consumption) and impaired cardiac recovery in response to sympathetic stimulation via a cold pressor test (CPT) compared to individuals with lower school burnout scores. In regard to heart rate (HR), we proposed a specific hypothesis during the recovery phase due to an expected vagal rebound. Vagal rebound is defined as a marked increase in parasympathetic activity above resting levels following an acute stressor (Arai et al., 1989) and is suggested to provide cardioprotection (Mezzacappa et al., 2001). We expected vagal rebound to be elicited in participants with lower school burnout scores but absence in those with high burnout scores.

Methods

Participants

Forty apparently healthy male adult undergraduates (18–30 years of age; $M = 21.32$, $SD = 2.63$) were qualified for study inclusion. Females were excluded from the study due to concerns about hormonal variations influencing pressure wave morphology (Adkisson et al. 2010). Twelve male participants were excluded from study participation. To avoid potential cardiovascular functioning confounds, participants were excluded from study participation through an online health screening assessment if they smoked, exercised regularly as defined as >120 min per week in the previous 6 months, were hypertensive as defined as BP $\geq$140/90 mmHg, had chronic diseases, or were taking beta blockers, antidepressants or stimulants. Participants were asked to abstain from caffeine, alcohol and strenuous physical activity for at least 24 h prior to testing and were asked not to eat any food 4 h prior to testing. Participants were recruited from a university population sample. All participants gave their written consent prior to study participation as approved by The Florida State University Institutional Review Board. The ethnic composition of the sample was 61% Caucasian, 14% African American, 7% Asian and 18% endorsed either biracial or non-disclosed ethnicity.

Instruments and measures

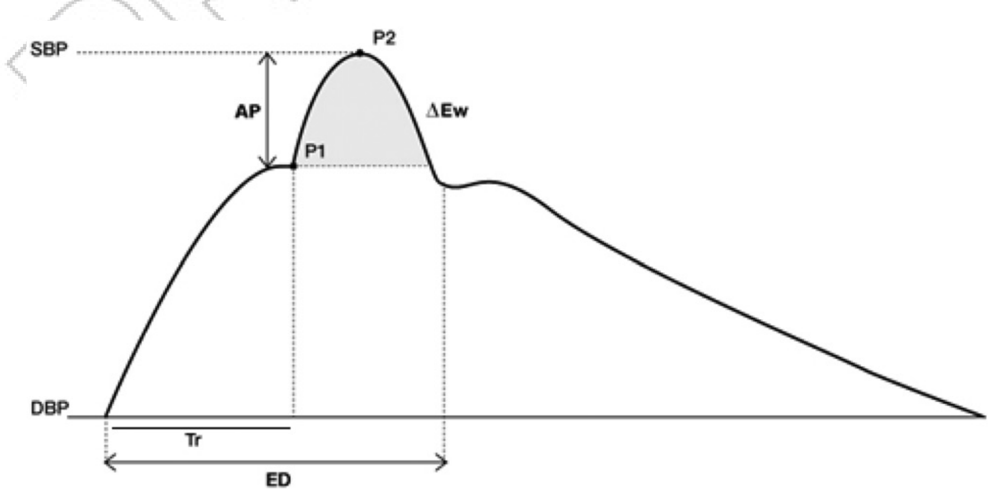
Anthropometrics

Height was measured using a stadiometer and body weight was measured using a Seca scale (Sunbeam Products Inc., Boca Raton, FL). Body mass index (BMI) was calculated as kg/m^2.

Pulse wave analysis

PWA, defined as examination of the characteristics and functioning of the arterial (central) pulse wave, allows for accurate assessment of central hemodynamic functioning (Hashimoto et al., 2007; Nichols & Singh, 2002; Safar et al., 2008). PWA conducted via applanation tonometry allows for a non-invasive examination of the intra-arterial aortic pressure wave form (Figure 1).

Applanation tonometry assesses BP and flow by gently resting a pencil shape device (tonometer) against the skin above an artery. The aortic BP (central) wave comprises a forward wave (P1), caused by stroke volume ejection, and a reflected wave (P2) that returns to the aorta from peripheral sites. Additional indices measured include augmentation index (AIx), transit time of the reflected wave (T*r*), systolic pressure time interval (STI), diastolic pressure time interval (DTI) and subendocardial viability index (SVI). AIx is defined as the augmented pressure ($AP = P2 - P1$) expressed as a percentage of the aortic pulse pressure ($APP = ASBP - ADBP$). AIx is a marker of pressure wave reflection pressure and has been associated with high rates of cardiovascular morbidity and mortality (Mitchell, 2009; Vlachopoulos et al., 2010) and is able to predict clinical



Q2 Figure 1. Schematic representation of a typical aortic pulse pressure waveform. SBP: systolic blood pressure; DBP: diastolic blood pressure; AP: augmentation pressure; P1: pressure of the first systolic peak; P2: pressure of the second systolic peak; T*r*: transit time of the reflected wave; ED: systolic ejection duration.

events independently of peripheral pressures (Vlachopoulos et al., 2010). Since AP and AIx are influenced by HR they are typically adjusted at 75 bpm (AP@75, AIx@75; Wilkinson et al., 2002). T*r* indicates the round-trip travel of the forward wave to the peripheral reflecting sites and back to the aorta. STI has been shown to be an indicator of left ventricular work and myocardial oxygen consumption while DTI is an indicator of coronary perfusion (Bunckberg et al., 1972). SVI is obtained from the ratio of DTI to STI expressed as a percentage of subendocardial perfusion to myocardial demand (Bunckberg et al., 1972).

In this study, PWA assessed vascular function and aortic hemodynamics using brachial BP and applanation tonometry. Brachial BP and applanation tonometry were obtained in duplicates at each time point. Brachial BP was recorded using an automated oscillometric device (HEM-705CP; Omron Healthcare, Vernon Hill, IL). Brachial systolic BP (BSBP) and diastolic BP (BDBP) were used to calibrate radial waveforms obtained from a 10 s epoch using a high-fidelity tonometer (SPT-301B; Millar Instruments, Houston, TX). Brachial mean arterial pressure (MAP) was calculated as (1/3)SBP + (2/3)DBP. Aortic BP waveforms and resulting central pressure indices were derived using a validated generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). Only high-quality measurements (>80% operator index) were considered for analysis.

Depression

Depression was measured using the 10-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977; Santor & Coyne, 1997). The CES-D has been widely used as a stable measure of depressive symptoms in nonclinical samples. It asks participants to respond to a list of ways they may have felt or behaved during the previous week. Sample items include, ''I was bothered by things that usually don't bother me,'' and ''I felt hopeful about the future,'' (reverse coded). Responses ranged from 0 = *rarely or none of the time (less than 1 d)* to 3 = *most or all of the time (5–7 d)*. Responses were summed into one overall score, with a possible range of 0–30. Reliability for the sample was $\alpha = 0.67$.

Anxiety

Anxiety was measured using the 20-item State-Trait Anxiety Inventory (STAI; Spielberger et al., 1970). Participants were asked to respond to anxiety items such as ''upset,'' ''calm,'' ''secure,'' ''at ease'' and ''nervous.'' Responses were scored on a 4-point Likert scale (1 = *not at all* to 4 = *very much so*). Half of the items were reverse coded so that higher scores indicated greater anxiety. Items were then summed to create a composite Anxiety score with a possible range of 20–80. Reliability for the sample was $\alpha = 0.91$.

School burnout

School burnout was measured using the School Burnout Inventory (SBI: Salmela-Aro et al., 2009). The SBI consists of nine items measuring three first-order factors of school burnout: (a) exhaustion at school (four items), (b) cynicism toward the meaning of school (three items) and (c) sense of inadequacy at school (two items). Summed scores from the first-order factors comprise a second-order overall school burnout score. All the items were rated on a 6-point Likert-type scale ranging from 1 (*completely disagree*) to 6 (*strongly agree*). Higher composite scores indicate higher burnout. As validated diagnostic scores have not been established for SBI scores, consistent with the strategy of the Danhof-Pont et al. (2011) meta-analysis of comparing cohorts of burnout based on burnout severity scores, we differentiate individuals with higher burnout (B) from non-burnout (NB) in our sample on the overall SBI score through the use of a median split (*Mdn* = 18). Reliability for the sample was $\alpha = 0.94$.

Procedure

Participants were first introduced to the laboratory setting and familiarized with the study procedures. Body measurements (i.e. height, weight, arm and waist circumference) were taken followed by participants completing a health questionnaire that included a health history form and a questionnaire containing the school burnout, depression and anxiety scales. All data collection were conducted in the afternoon in a quiet, dimly lit, temperature-controlled room ($23 \pm 1\,°C$) at the same time of the day (± 2 h) in order to minimize potential diurnal variations in CVR (Muller, 1999). Before the CPT, participants were seated and given a 10-min rest before any baseline (BASE) measurements were performed. Within 5 min after the rest period, BASE measurements for peripheral brachial BP and applanation tonometry of the radial artery for central aortic hemodynamics were taken. Immediately following the BASE measurements, participants completed the CPT by submerging their hand in cold water (4 °C) for 3 min in order to evoke SNS stimulation. During the CPT a research assistant observed participant completion of the 3 min CPT. All participants were able to keep their hand in the water throughout the entire task. BP and applanation tonometry were obtained between 2 and 3 min of the CPT. After the 3 min CPT, participants were told to remove their hand from the cold water which started a 3-min recovery period (REC). During REC, BP measurements followed directly by hemodynamics measurements were taken within 2–3 min from the start of the recovery period. All REC measurements ended after 3 min.

Statistical analysis

Differences in health characteristics between burnout groups were analyzed with independent samples *t* tests. Multinomial logistic regression evaluated ethnicity and year in school associations with school burnout categorization. Pearson correlations evaluated measurement scale (SBI, CES-D, STAI) associations. Planned univariate contrasts were conducted to compare the hypothesized *a priori* hemodynamic differences between the B and NB groups. Planned univariate contrasts were conducted to compare the hypothesized *a priori* hemodynamic differences between the B and NB groups. The planned contrasts used the adjusted marginal means of hemodynamic indices that were created after controlling for depression and anxiety scores.[1] All statistical analyses were performed using IBM SPSS version 20.

Results

Table 1 shows the health demographic characteristics of the B and NB groups. Independent samples *t* tests indicated no statistically significant differences in health characteristics (height, weight, BMI, age) between classified burnout participants. Multinomial logistic regression analyses indicated that neither ethnicity, $\chi^2(4) = 6.24$, $p = 0.18$, nor year in school, $\chi^2(3) = 7.11$, $p = 0.07$, were associated with school burnout categorization. Pearson correlations were calculated between SBI ($M = 20.19$, $SD = 9.80$), STAI ($M = 31.05$, $SD = 8.19$) and the CES-D ($M = 9.17$, $SD = 2.95$). Significant correlations ($p < 0.01$, one-tailed) between the SBI and the STAI ($r = 0.38$) and the CES-D ($r = 0.45$) support the need to statistically control for anxiety and depressive symptom influences on SBI scores.

Table 2 displays the means and standard deviations of the hemodynamic responses between the B and NB groups at BASE, CPT and REC. Table 3 presents the contrast analyses. At BASE, contrasts indicated no significant differences on any of the cardiovascular indices between the burnout groups (see contrast 1).

As a manipulation check of the CPT procedure increasing SNS stimulation, contrasts were conducted within the B and NB groups comparing their BASE to CPT cardiovascular values. Within both burnout groups all cardiovascular values were significantly different from BASE with increases in HR, BSBP, BDBP, BMAP, ASBP, ADBP, AMAP, AP@75, AIx@75, P1, P2, STI, DTI and decreases in T*r* and SVI (see contrasts 2 and 3).

Contrasts testing predicted differences between burnout groups at CPT indicated that BSBP, BDBP, BMAP, ASBP, ADBP, AMAP, AP@75, Alx@75, P1, P2, STI and DTI were significantly higher for B than NB while T*r* was significantly lower for B than NB (see contrast 4). HR and SVI did not significantly differ between burnout groups at CPT. To examine cardiovascular recovery predictions, contrasts were conducted within B and NB groups comparing BASE to REC cardiovascular values (see contrast 5 and 6). For B, all cardiovascular values at REC except for SVI and HR were still significantly higher (lower for T*r*) than at BASE. For NB no cardiovascular values at REC were significantly different from BASE, except for HR which, as predicted, was significantly lower. Figure 2 displays the mean changes in aortic (panel A and B) and brachial (panel C and D) BP from BASE to CPT and REC between the B and NB groups. Figure 3 displays the mean HR changes from BASE to CPT and REC between the burnout groups. Figure 4 displays mean hemodynamic changes of AIx@75, STI, DTI and SVI from BASE to CPT and from BASE to REC between the B and NB groups.

Table 1. Health characteristics.

Variable	B ($n = 20$)	NB ($n = 20$)
Height (cm)	176.30 ± 8.52	177.61 ± 6.14
Weight (kg)	81.50 ± 15.21	80.34 ± 12.51
BMI (kg/m^2)	25.58 ± 4.00	25.31 ± 4.07
Age (years)	21.20 ± 2.46	21.43 ± 2.79

Data are *M* ± *SD*. B: burnout group; NB: non-burnout group; BMI: body mass index. Independent samples *t* tests examined health characteristic differences.

Discussion

This study examined cardiovascular functioning that may underlie school burnout. Results supported our predictions, demonstrating the novel finding that during exposure to a stressor and in the immediate recovery period, higher levels of school burnout were associated with greater CVR in aortic hemodynamic functioning. These findings identify novel cardiac biomarkers related to school burnout and support the conclusion that burnout may be predictive of an increased risk of future CVD. This study provides the initial investigation into physiological functioning underlying school burnout and attempts to provide a methodological framework for burnout research applicable to additional environments (i.e. workplace burnout).

Methodological suggestions from related literatures (e.g. control of related affective symptomatology, utilization of CVR and recovery phases, and measurement of aortic hemodynamics) were implemented in this study in an attempt to improve the clarity of the potential relationship between burnout, hemodynamics and CVD. First, as affective disorders may have overlapping symptomatology, investigators suggest the need to control for depressive and anxiety symptoms in designs focusing on burnout measurement (Melamed et al., 2006; Schaufeli & Buunk, 2003; Shirom, 2009). Second, only through the exposure to and then recovery from a stressful stimulus may some individuals be identified as at risk of deteriorated cardiovascular functioning. In fact CVR, defined as the magnitude or pattern of hemodynamic responses to stressors, has been identified as serving as both a marker and a mechanism in the pathogenesis of CVD (Manuck, 1994; Treiber et al., 2003). In a review of studies investigating CVR and the development of subclinical and clinical CVD states, BP responses to the cold pressor task (CPT) were noted as predictive of future hypertension in large longitudinal epidemiological studies in initially normotensive samples (Treiber et al., 2003). Also, the degree of cardiovascular and autonomic recovery from a stressful state to homeostasis is also diagnostic of cardiac functioning (Cole et al., 1999). The faster an individual can recover from a stressor and return to a state of homeostasis is predictive of

[1]Instead of traditional omnibus tests, planned comparisons were used to more precisely test the specific, *a priori* hypotheses we proposed. As argued by O'Keefe (2003a,b) and Tutzauer (2003), one of the benefits of testing specific *a priori* hypotheses that are grounded in theory is the latitude of selecting alpha criteria and statistical analyses that appropriately limit the threats to type II error. Therefore, statistical significance for the proposed contrasts where set at $p < 0.05$ with no additional alpha corrections. When traditional omnibus 2×3 analyses of covariance (ANCOVA) with repeated measures were conducted across trials (BASE versus CPT versus REC) and condition (B versus NB) on cardiovascular variables while controlling for depression and anxiety symptomatology, significant ANCOVA interactions were identified ($p < 0.05$) between burnout groups for all cardiovascular indices and simple effect follow-up tests produced near identical conclusions as the planned contrast analyses.

Table 2. Hemodynamic responses to cold pressor test (4 °C) between B and NB groups.

	B			NB		
Variable	BASE	CPT	REC	BASE	CPT	REC
HR (bpm)	62.70 ± 7.66	67.90 ± 8.49	62.00 ± 10.66	61.40 ± 11.66	64.75 ± 9.23	58.25 ± 9.09
BSBP (mmHg)	112.60 ± 8.78	143.70 ± 17.41	121.10 ± 14.51	114.63 ± 5.82	133.25 ± 11.82	116.63 ± 6.17
BDBP (mmHg)	70.50 ± 9.17	94.60 ± 13.67	76.30 ± 9.73	67.75 ± 6.40	81.88 ± 8.62	66.25 ± 6.57
BMAP (mmHg)	84.53 ± 8.38	110.97 ± 14.26	91.23 ± 10.29	83.38 ± 4.94	99.00 ± 8.18	83.04 ± 4.42
ASBP (mmHg)	96.50 ± 8.42	130.60 ± 20.02	105.80 ± 12.23	97.31 ± 4.16	116.50 ± 10.55	97.75 ± 4.25
ADBP (mmHg)	71.60 ± 9.28	95.90 ± 13.65	77.20 ± 9.83	68.48 ± 6.46	83.00 ± 8.72	67.13 ± 6.74
AMAP (mmHg)	79.90 ± 8.70	107.47 ± 15.13	86.73 ± 9.32	78.09 ± 5.33	94.17 ± 8.40	77.33 ± 55.19
AP@75 (%)	1.40 ± 1.83	5.60 ± 5.28	0.70 ± 1.53	0.90 ± 3.02	2.50 ± 1.46	1.38 ± 2.36
AIx@75 (%)	−5.10 ± 6.50	14.60 ± 11.33	1.70 ± 5.43	−2.79 ± 10.26	7.38 ± 6.13	−4.50 ± 8.12
P1 (mmHg)	95.70 ± 8.73	123.50 ± 14.98	102.70 ± 11.64	95.50 ± 4.50	112.13 ± 8.46	96.25 ± 3.17
P2 (mmHg)	95.75 ± 8.12	130.60 ± 20.02	105.60 ± 12.30	96.50 ± 3.96	116.50 ± 10.55	97.13 ± 4.66
Tr (ms)	152.20 ± 14.97	139.40 ± 9.45	144.30 ± 6.81	147.88 ± 7.92	145.13 ± 5.83	151.25 ± 10.92
STI (mmHg/s.min^{-1})	1557.80 ± 227.59	2301.90 ± 478.79	1709.50 ± 290.10	1529.63 ± 222.25	1998.25 ± 224.44	1500.00 ± 151.01
DTI (mmHg/s.min^{-1})	3418.90 ± 328.31	4364.80 ± 550.34	3703.90 ± 432.90	3365.84 ± 296.40	3898.13 ± 416.40	3364.38 ± 253.97
SVI (%)	222.20 ± 24.98	195.00 ± 33.03	221.00 ± 35.97	225.63 ± 40.03	196.88 ± 26.33	226.88 ± 28.67

Data are mean ± SD. B: burnout group; NB: non-burnout group; BASE: baseline; CPT: cold pressor test; REC: recovery; HR: heart rate; BSBP: brachial systolic blood pressure; BDBP: brachial diastolic blood pressure; BMAP: brachial mean arterial pressure; ASBP: aortic systolic blood pressure; ADBP: aortic diastolic blood pressure; AMAP: aortic mean arterial pressure; AP@75: augmentation pressure adjusted at 75 bpm; AIx @75: augmentation index adjusted at 75 bpm; P1: first systolic peak pressure; P2: second systolic peak pressure; *Tr*: reflection time; STI: systolic time interval; DTI: diastolic time interval; SVI: subendocardial viability index.

Table 3. Contrasts analyses of hemodynamic values.

	Contrast 1		Contrast 2		Contrast 3		Contrast 4		Contrast 5		Contrast 6	
	BASE		BASE vs. CPT		BASE vs. CPT		CPT		BASE vs. REC		BASE vs. REC	
	B vs. NB		B		NB		B vs. NB		B		NB	
Variable	Contrast *F*	Partial η^2	Contrast *F*	Partial η^2	Contrast *F*	Partial η^2	Contrast *F*	Partial η^2	Contrast *F*	Partial η^2	Contrast *F*	Partial η^2
HR (bpm)	0.16	0.005	39.95***	0.678	4.96*	0.226	1.13	0.032	0.45	0.023	6.39*	0.299
BSBP (mmHg)	0.63	0.018	106.17***	0.848	66.41***	0.796	4.20*	0.110	18.44***	0.493	1.10	0.068
BDBP (mmHg)	1.03	0.029	599.00***	0.969	339.67***	0.952	10.50**	0.236	21.05***	0.526	2.65	0.150
BMAP (mmHg)	0.24	0.007	161.50***	0.895	71.97***	0.809	8.90**	0.207	22.37***	0.541	0.14	0.009
ASBP (mmHg)	0.12	0.004	94.28***	0.832	52.21***	0.754	6.47*	0.160	26.83***	0.585	0.11	0.007
ADBP (mmHg)	1.31	0.037	196.97***	0.912	57.61***	0.772	10.74**	0.240	24.70***	0.565	0.74	0.047
AMAP (mmHg)	0.53	0.015	149.60***	0.877	64.69***	0.792	9.89**	0.225	25.67***	0.575	0.26	0.017
AP@75 (%)	0.38	0.011	28.00***	0.596	25.67***	0.602	5.18*	0.117	35.81***	0.653	3.37	0.183
AIx@75 (%)	0.68	0.020	50.61***	0.727	30.52***	0.642	5.25*	0.134	40.56***	0.681	2.50	0.143
P1 (mmHg)	0.01	0.000	151.76***	0.889	74.25***	0.814	7.33*	0.177	22.38***	0.541	0.37	0.024
P2 (mmHg)	0.11	0.003	91.81***	0.829	58.11***	0.774	6.47*	0.160	26.58***	0.583	0.21	0.014
Tr (ms)	1.09	0.031	10.21**	0.359	4.38*	0.205	4.49*	0.117	5.53**	0.226	1.48	0.243
STI (mmHg/s.min^{-1})	0.14	0.004	117.03***	0.860	70.77***	0.806	5.45*	0.138	49.19***	0.721	0.92	0.058
DTI (mmHg/s.min^{-1})	0.25	0.007	96.85***	0.836	50.15***	0.747	7.88**	0.188	13.21***	0.410	0.00	0.000
SVI (%)	0.09	0.003	29.44***	0.608	20.33***	0.545	0.03	0.001	0.10	0.005	0.07	0.005

df = (1, 38). B: burnout group; NB: non-burnout group; BASE: baseline; CPT: cold pressor test; REC: recovery. Univariate contrasts examined adjusted marginal means.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

positive physical and mental health outcomes (Rottenberg et al., 2007). Therefore, measuring cardiac function during and after an acute stressor, slight cardiac anomalies undetectable at baseline may then be identified.

Third, although the use of brachial cuff BP measurement is a well-accepted method intended to identify individuals at increased cardiovascular risk, this method may underestimate hemodynamic anomalies. Research has demonstrated that central pressure measurements can predict cardiovascular outcomes such as carotid hypertrophy, extent of atherosclerosis and incident cardiovascular events more accurately than brachial pressure measurements (Roman et al., 2009). Importantly, central pressure cannot be reliably inferred from peripheral pressure measurements (McEniery et al., 2008). Plus specific indices predictive of deteriorated cardiac function (i.e. increases in wave reflection, left ventricular work and myocardial oxygen consumption) can only be derived from central pressure assessment (Hashimoto et al., 2007; O'Rourke & Adji, 2005; Manisty et al., 2010; Safar et al., 2008; Vlachopoulos et al., 2010).

By comparing cardiovascular functioning between baseline, stress and recovery conditions, we were able to demonstrate that even though individuals varying in burnout scores during a restful condition appear equally healthy, under stress and in the direct aftermath of a stressor they are not. Use of PWA identified detrimental changes in aortic

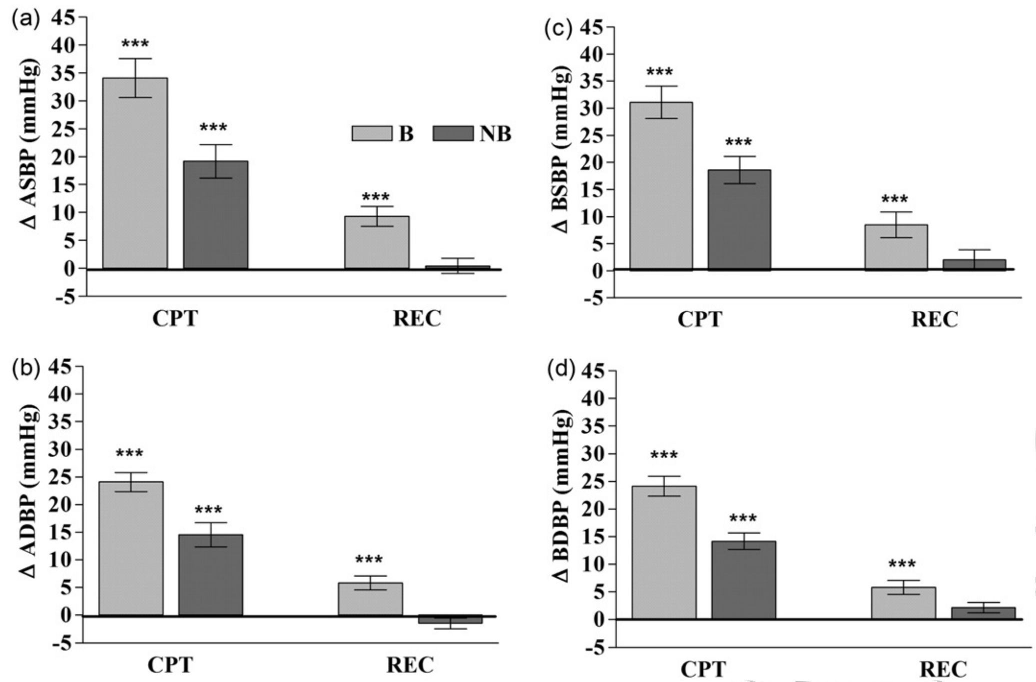


Figure 2. Mean peripheral and central blood pressure changes from baseline to cold pressor test (CPT) and to recovery period (REC) between burnout (B) and non-burnout (NB) groups. Data are mean difference changes and 95% CI. (a) ASBP: aortic systolic blood pressure; (b) ADBP: aortic diastolic blood pressure; (c) BSBP: brachial systolic blood pressure; (d) BDBP: brachial diastolic blood pressure. ***$p < 0.001$ change from baseline. Univariate contrasts examined adjusted marginal means.

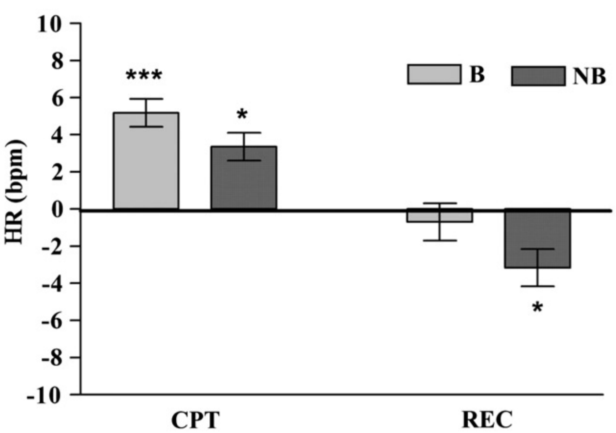


Figure 3. Mean heart rate (HR) changes from baseline to cold pressor test (CPT) and to recovery period (REC) between burnout (B) and non-burnout (NB) groups. Data are mean difference changes with 95% CI. ***$p < 0.001$, *$p < 0.05$ change from baseline. Univariate contrasts examined adjusted marginal means.

hemodynamic indices in individuals with higher burnout scores while performing the CPT and while in cardiac recovery. This finding needs to be viewed in light of the fact that ASBP is more influenced by P2 whereas BSBP is more dependent upon P1 (Nichols, 2005). Thus, changes in P2 after administration of laboratory stressors may reveal cardiovascular anomalies that are not detected using brachial BP cuffs. Our findings are in accordance to those of Casey et al. (2008) that reported CPT evoked increases in peripheral BP and central BP in healthy young adults however the increase in aortic BP during the CPT was higher (∼9%) than the increase in brachial BP suggesting that aortic BP is a more sensitive marker of cardiovascular function than brachial BP. Furthermore, we found greater increases in brachial and aortic BP (Figure 2), wave reflection (AIx in Figure 4a), left ventricular work and myocardial oxygen consumption (STI in Figure 4b) although preserved coronary perfusion (DTI in Figure 4c). As the aforementioned factors are more accurate predictors of cardiovascular health these results suggest that school burnout is associated with increased cardiovascular risk, which may eventually lead to cardiovascular complications such as hypertension, myocardial infarction and stroke (Hashimoto et al., 2007). A novel finding and one worth emphasizing is that we observed these hemodynamic changes during a period of sympathetic nervous system (SNS) stimulation but not while subjects were at rest adding to the notion that cardiovascular anomalies may be undetected at rest (Manuck, 1994; Rottenberg et al., 2007; Treiber et al., 2003). Additionally, statistical analyses that controlled for anxiety and depressive symptoms revealed that burnout symptomatology uniquely accounts for differences in hemodynamic functioning.

A few specific study findings however do need further explanation. First, our analyses indicated that during the CPT, both STI and DTI were significantly higher in the burnout group. Since STI (ventricular work) increased with a concurrent increase in DTI (coronary perfusion) this suggests that burnout may not necessarily attenuate coronary blood flow supply (SVI, see Figure 4d) during sympathetic stimulation. Second, analyses indicate HR did not significantly differ between the burnout groups. However, while not statistically significant, the differences in means were in the predicted directions with HR being higher in the burnout group. Interestingly, the HR response during the post stress recovery period revealed altered cardiovagal modulation as shown by the lack of vagal rebound in the burnout group. Importantly,

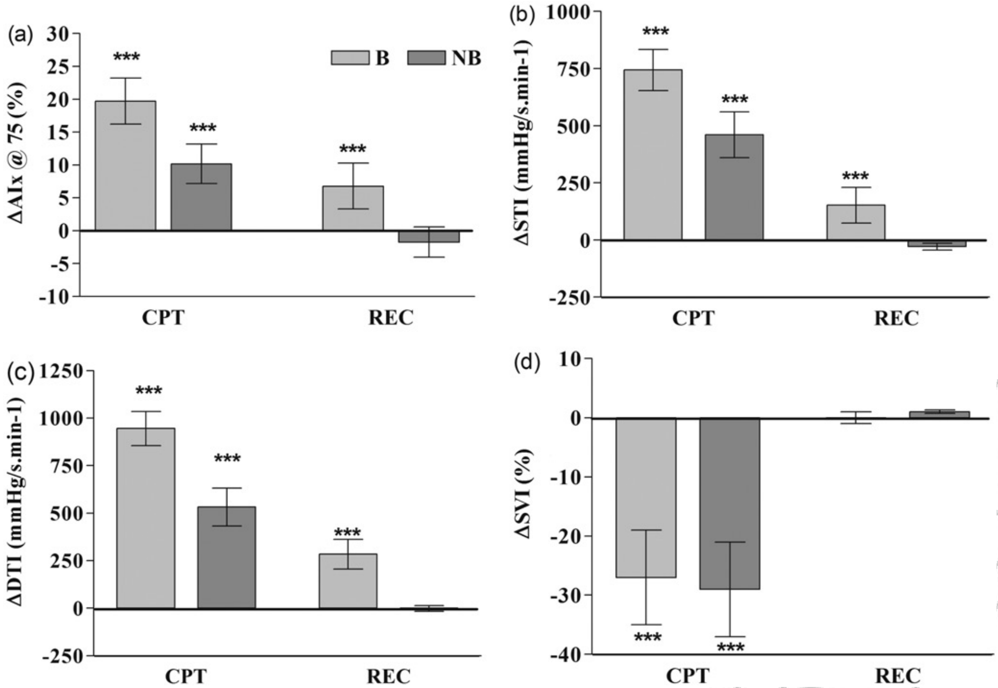


Figure 4. Mean hemodynamic changes from baseline to cold pressor test (CPT) and to recovery period (REC) between burnout (B) and non-burnout (NB) groups. Data are mean difference changes and 95% CI. (a) AIx @75: augmentation index adjusted at 75 bpm; STI: systolic time interval; DTI: diastolic time interval; SVI: subendocardial viability index. ***$p < 0.001$ change from baseline. Univariate contrasts examined adjusted marginal means.

Mezzacappa et al. (2001) demonstrated that impaired cardiac autonomic modulation, specifically lack of vagal rebound, occurs in populations at increased cardiovascular risk such as individuals with hypertension. Accordingly, lack of vagal rebound may be an additional factor linking school burnout to increased CVD risk. However, analysis of cardiac autonomic modulation using HR variability measures is needed to confirm this finding.

The potential mechanisms that may explain increased cardiac reactivity during sympathetic stimulation (CPT) may also be associated with impaired cardiovascular autonomic modulation. As previous research has shown associations between work-related burnout, increased SNS activity and plasma cortisol concentrations (De Vente et al., 2003), the hyperactive CPT cardiovascular responses in the burnout group could be driven by increased adrenergic stimulation owing to altered plasma catecholamines concentration which may ultimately increase smooth muscle vascular tone. Since P2 is influenced by peripheral vascular tone (Munir et al., 2008) and was more affected than the other factors contributing to AIx (e.g. HR), our results suggest that the muscular arteries are hyper responsive in individuals with relatively higher burnout symptomatology. It is worth noting that sympathetic hyperactivity and/or attenuated vagal response during the recovery period may have contributed to the higher levels of ventricular work and AIx in burnout individuals.

Important study limitations are also necessary to note as factors that need to be addressed in future research. First, as this study only included men, additional studies are necessary to determine if these findings are generalizable to females. Second, only global SBI scores were examined; leaving SBI subscale associations with hemodynamic functioning unexamined and of potential future consideration. As this was the first study to examine cardiac biomarkers using the SBI, we were primarily interested in understanding if the overarching concept of school burnout was related to cardiac function; thus leading us to focus our analyses on the composite SBI score. Furthermore, no established clinical diagnostic cutoff points have been established for the SBI. This opens the possibility that what may actually have been examined were subclinical levels of school burnout. However, even assuming subclinical burnout levels, individuals with lower burnout scores still had better cardiac functioning than individuals with higher subclinical burnout scores while under cardiac stress and recovery. Finally, although the HR response pattern of NB individuals was consistent with the elicitation of vagal rebound, more comprehensive HR variability and cardiac autonomic modulation measurements need to be taken in future studies to confirm this finding.

An interesting future direction to this research may be the measurement of vasoactive substances that could help explain the mechanisms responsible for the hemodynamic changes during the CPT. It could be that burnout negatively impacts endothelial cell functioning and vasodilator capacity, but this assumption warrants further investigation. The instrumentation used in this study was not able to fully identify the mechanisms that are determining the cardiac functioning differences found between the burnout groups. Instead, what this study did accomplish was the demonstration of observable cardiovascular differences during sympathetic stimulation and recovery between burnout groups. It is possible that burnout individuals have higher serum catecholamine concentrations which lead to increased vasoconstriction, which is a potential factor that future research should examine (Light et al., 1998).

In conclusion, the important takeaway message from this study is that our results demonstrate that school burnout is associated with increased cardiovascular responses during sympathetic stimulation. This is important as early manifestations of CVDs, such as hypertension, are characterized by increased SNS activity (Goldstein, 1983; Treiber et al., 2003). Increased cardiac reactivity is related to increased SNS activity as well as the future development of cardiovascular complications (Matthews et al., 2004; Treiber et al., 2003). In other words, an increase in school burnout is related to hyperactive responses to cardiac stress that may be related to an increase in SNS activity. Additionally, this study has identified new markers of cardiovascular functioning (such as AIx) that may help identify individuals at increased risk of developing CVD. These findings have important social and clinical implications for the evaluation of school burnout symptoms as they may be associated with the early manifestations of CVD, even in seemingly young healthy men. However, in order to more fully determine the extent of the deleterious relationship between school burnout and cardiovascular functioning, comprehensive prospective studies are needed.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

Adkisson EJ, Casey DP, Beck DT, Gurovich AN, Martin JS, Braith RW. (2010). Central, peripheral and resistance arterial reactivity: fluctuates during the phases of the menstrual cycle. Exp Biol Med 235:111–8.

Arai Y, Saul JP, Albrecht P, Hartley LH, Lilly LS, Cohen RJ, Colucci WS. (1989). Modulation of cardiac autonomic activity during and immediately after exercise. Am J Physiol 256(1Pt2): H132–41.

Bunckberg GD, Fixler DE, Archie JP, Hoffman JI. (1972). Experimental subendocardial ischemia in dogs with normal coronary arteries. Circ Res 30:67–81.

Casey DP, Nichols WW, Braith RW. (2008). Impact of aging on central pressure wave reflection characteristics during exercise. Am J Hypertens 21(4):419–24.

Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. (1999). Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med 341(18):1351–7.

Danhof-Pont MB, Van Veen T, Zitman FG. (2011). Biomarkers in burnout: a systematic review. J Psychosom Res 70:505–24.

De Vente W, Olff M, Van Amsterdam JGC, Kamphuis JH, Emmelkamp PMG. (2003). Physiological differences between burnout patients and healthy controls: blood pressure, heart rate, and cortisol responses. Occup Environ Med 60:54–61.

Goldstein DS. (1983). Plasma catecholamines and essential hypertension. An analytical review. Hypertension 5(1):86–99.

Halbesleben JRB, Buckley RM. (2004). Burnout in organizational life. J Manage 30:859–79.

Hashimoto J, Imai Y, O'Rourke MF. (2007). Indices of pulse wave analysis are better predictors of left ventricular mass reduction than cuff pressure. Am J Hypertens 20(4):378–84.

Kahn RL, Byosiere P. (1992). Stress in organizations. In: Dunnette MD, Hough LM, editors. Handbook of industrial and organizational psychology. 2nd ed. Vol. 3. Palo Alto, CA: Consultin Psychologists Press. p 571–650.

Kiuru N, Aunola K, Nurmi JE, Leskinen E, Salmela-Aro K. (2008). Peer group influence and selection in adolescents' school burnout: a longitudinal. Merrill Palmer Q 54:23–55.

Light KC, Kothandapani RV, Allen MT. (1998). Enhanced cardiovascular and catecholamine responses in women with depressive symptoms. Int J Psychophysiol 28(2):157–66.

Manisty C, Mayet J, Tapp RJ, Parker KH, Sever P, Poulter NH. (2010). Wave reflection predicts cardiovascular events in hypertensive individuals independent of blood pressure and other cardiovascular risk factors: an ASCOT (Anglo-Scandanavian Cardiac Outcome Trial) substudy. J Am Coll Cardiol 56(1):24–30.

Manuck SB. (1994). Cardiovascular reactivity in cardiovascular disease: ''once more unto the breach''. Int J Behav Med 1(1): 4–31.

Maslach C, Schaufeli WB, Leiter MP. (2001). Job burnout. Annu Rev Psychol 52:397–422.

Matthews KA, Katholi CR, McCreath H, Whooley MA, Williams DR, Zhu S, Markovitz JH. (2004). Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. Circulation 110(1):74–8.

McEniery CM, Yasmin BM, Munnery M, Wallace SM, Rowe CV, Cockcroft JR, Wilkinson IB. (2008). Anglo-Cardiff Collaborative Trial Investigators. Central pressure: variability and impact of cardiovascular risk factors: the Anglo-Cardiff Collaborative Trial II. Hypertension 51(6):1476–82.

Melamed S, Shirom A, Toker S, Berliner S, Shapira I. (2006). Burnout and risk of cardiovascular disease: evidence, possible causal paths, and promising research directions. Psychol Bull 132: 327–53.

Mezzacappa ES, Kelsey RM, Katkin ES, Sloan RP. (2001). Vagal rebound and recovery from psychological stress. Psychosom Med 63(4):650–7.

Mitchell GF. (2009). Arterial stiffness and wave reflection: biomarkers of cardiovascular risk. Artery Res 3(2):56–64.

Muller JE. (1999). Circadian variation in cardiovascular events. Am J Hypertens 12(2Pt2):35S–42S.

Munir S, Jiang B, Guilcher A, Brett S, Redwood S, Marber M, Chowienczyk P. (2008). Exercise reduces arterial pressure augmentation through vasodilation of muscular arteries in humans. Am J Physiol Heart Circ Physiol 294(4):H1645–50.

Nichols WW. (2005). Clinical measurement of arterial stiffness obtained from noninvasive pressure waveforms. Am J Hypertens 18(1Pt2): 3S–10S.

Nichols WW, Singh BM. (2002). Augmentation index as a measure of peripheral vascular disease state. Curr Opin Cardiol 17(5):543–51.

O'Keefe DJ. (2003a). Colloquy: should familywise alpha be adjusted? Against familywise alpha adjustment. Hum Commun Res 23:431–47.

O'Keefe DJ. (2003b). Searching for a defensible application of alpha-adjustment tools. Hum Commun Res 29:464–8.

O'Rourke MF, Adji A. (2005). An updated clinical primer on large artery mechanics: implications of pulse waveform analysis and arterial tonometry. Curr Opin Cardiol 20(4):275–81.

Radloff LS. (1977). The CES-D Scale: a self-report depression scale for research in the general population. Appl Psych Measurement 1: 385–401.

Roman MJ, Devereux RB, Kizer JR, Okin PM, Lee ET, Wang W, Howard BV. (2009). High central pulse pressure is independently associated with adverse cardiovascular outcome: the strong heart study. J Am Coll Cardiol 54(18):1730–4.

Rottenberg J, Clift A, Bolden S, Salomon K. (2007). RSA fluctuation in major depressive disorder. Psychophysiology 44: 450–8.

Safar ME, Blacher J, Protogerou A, Achimastos A. (2008). Arterial stiffness and central hemodynamics in treated hypertensive subjects according to brachial blood pressure classification. J Hypertens 26(1): 130–7.

Salmela-Aro K, Kiuru N, Leskinen E, Nurmi JE. (2009). School burnout inventory (SBI): Reliability and validity. Eur J Psychol Assess 25: 48–57.

Santor DA, Coyne JC. (1997). Shortening the CES-D to improve its ability to detect cases of depression. Psychol Assessment 9(3): 233–43.

Schaufeli WB, Buunk BP. (2003). Burnout: An overview of 25 years of research and theorizing. In: Schabracq MJ, Winnubst JAM, Cooper CC, editors. The handbook of work and health psychology. 2nd ed. West Sussex, England: Wiley. p 383–429.

Shirom A. (2003). Job-related burnout. In: Quick JC, Tetrick LE, editors. Handbook of occupational health psychology. Washington, DC: American Psychological Association. p 245–65.

Shirom A. (2009). Epilogue: mapping future research on burnout and health. Stress Health 25:375–80.

Spielberger CD, Gorsuch RL, Lushene RE. (1970). Manual for the state-trait anxiety inventory. Palo Alto, CA: Consulting Psychologists Press.

Treiber FA, Kamarck T, Schneiderman N, Sheffield D, Kapuku G, Taylor T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. Psychosom Med 65(1):46–62.

Tutzauer F. (2003). On the sensible application of familywise alpha adjustment. Hum Commun Res 29:455–63.

van Doornen LJP, Houtveen JH, Langelaan S, Bakker AB, van Rhenen W, Schaufeli WB. (2009). Burnout versus work engagement in their effects on 24-hour ambulatory monitored cardiac autonomic function. Stress Health 25(4):323–31.

Vlachopoulos C, Aznaouridis K, O'Rourke MF, Safar ME, Baou K, Stefanadis C. (2010). Prediction of cardiovascular events and all-cause mortality with central haemodynamics: a systematic review and meta-analysis. Eur Heart J 31(15): 1865–71.

Wilkinson IB, Mohammad NH, Tyrrell S, Hall IR, Webb DJ, Paul VE. (2002). Heart rate dependency of pulse pressure amplification and arterial stiffness. Am J Hypertens 15(1Pt1): 24–30.