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Sedentary behaviour, but not moderate-to-vigorous physical activity, is associated with respiratory responses to acute psychological stress

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ABSTRACT

Background: Acute psychological stress induces respiratory responses, and stress-induced respiratory changes can be used to non-invasively reflect metabolic regulation. Respiratory and cardiovascular responses to stress are both driven by sympathetic mechanisms. Higher volumes of sedentary behaviour and lower volumes of physical activity are associated with elevated sympathetic tone and larger cardiovascular responses to stress. The aim of this study was to test whether these associations translate to measures of respiratory stress reactivity.

Methods: Daily hours of sedentary behaviour (thigh-mounted activPAL) and moderate-to-vigorous physical activity (MVPA; wrist-mounted ActiGraph) were assessed across seven days. Breath-by-breath respiratory (e.g., breathing frequency [BF], end-tidal carbon dioxide partial pressure [PetCO₂], carbon dioxide output [$\dot{V}CO_2$] and respiratory exchange ratio [RER]) responses to an 8-min Paced Auditory Serial Addition Test were then measured using a Cortex MetaLyzer3B.

Results: Healthy participants (N = 61, mean age \pm SD = 25.7 \pm 8.9 years) recorded high volumes of sedentary behaviour (9.96 \pm 1.48 hours/day) and MVPA (1.70 \pm 0.71 hours/day). In adjusted models (with the inclusion of sedentary behaviour, MVPA, and other *a priori* selected covariates) hours of daily sedentary behaviour were associated with baseline to stress changes in BF (B = 0.695, 95% CI = 0.281 — 1.109, *p* = .014), V_T (B = -0.042, 95% CI = -0.058 — -0.026, *p* = .014), PetCO₂ (B = -0.537, 95% CI = -0.829 — -0.245, *p* = .014), $\dot{V}CO_2$ (B = -0.008, 95% CI = -0.014 — -0.003, *p* = .030), and RER (B = -0.013, 95% CI = -0.021 — -0.005, *p* = .022). Daily hours of MVPA were not linked with respiratory responses to stress.

Discussion: Sedentary behaviour, but not MVPA, is associated with respiratory stress reactivity. Future work should untangle the underlying mechanisms of these findings and explore the consequences for cardiometabolic disease.

Keywords: Sedentary behaviour, physical activity, acute psychological stress, stress reactivity, respiratory, gas exchange, ventilation, metabolic.

INTRODUCTION

Cardiometabolic disease is one of the leading causes of global mortality and morbidity (Vos et al., 2020). Acute psychological stress induces a range of psychobiological responses across the cardiovascular, immune, and neuroendocrine systems (Plourde et al., 2017; Turner et al., 2020). There are large individual differences in these responses, which can reflect cardiometabolic disease susceptibility (Hackett & Steptoe, 2017; Turner et al., 2020). For example, large cardiovascular (e.g., systolic and diastolic blood pressure, heart rate) responses to stress are prospectively associated with hypertension (Chida & Steptoe, 2010; Turner et al., 2020), and individuals with type 2 diabetes show attenuated systolic blood pressure responses to stress (Steptoe et al., 2014).

Metabolic dysregulation is a mechanism that is theorised to underlie links between exaggerated cardiovascular responses to stress and cardiometabolic disease (Carroll et al., 2009; Obrist, 1976; Turner et al., 2020). During exercise there is amplified cardiovascular output which is tightly coupled to the augmented metabolic demand caused by increased muscular activity (Balanos et al., 2010; Carroll et al., 2009). However, laboratory (Balanos et al., 2010; Carroll et al., 2009) and field (Blix et al., 1974) studies have demonstrated that psychological stress induces little metabolic demand, yet cardiovascular (e.g., heart rate, blood pressure) responses of large magnitude are still ignited.

Consequently, large cardiovascular responses to psychological stress are metabolically inappropriate, which can acutely threaten homeostasis and activate pathophysiological mechanisms if mounted repeatedly over time (Carroll et al., 2009; Obrist, 1976; Turner et al., 2020). In support, metabolically excessive systolic blood pressure responses to psychological stress are associated with increased carotid artery intima-media thickness (Lambiase et al., 2012).

One non-invasive method that can be used to reflect metabolic processes is respiratory assessment (Ritz, 2012). For example, oxygen consumption ($\dot{V}O_2$) reflects metabolic rate (Tyagi et al., 2014), end-tidal carbon dioxide partial pressure ($P_{et}CO_2$) can be used to indicate hyperventilation (i.e., breathing in excess of current metabolic demand; Meuret and Ritz, 2010), and respiratory exchange ratio (RER) quantifies fuel substrate utilisation (e.g., fat vs carbohydrate oxidation; Pendergast, Leddy and Venkatraman, 2000). Importantly, acute psychological stress induces respiratory responses,

including the outcomes above (Balanos et al., 2010; Plourde et al., 2017; Ritz & Kullowatz, 2005).

Evidence also links acute episodes of psychological stress with the triggering of asthma attacks (Ritz et al., 2011; Sandberg et al., 2000) and dysregulated respiratory reactivity has been outlined as a potential mechanism (Plourde et al., 2017; Ritz et al., 2011).

Previous literature in healthy populations has shown that acute psychological stress tasks evoke responses across a range of ventilatory (e.g., breathing frequency, tidal volume) and gas exchange (e.g., $\dot{V}O_2$ and $P_{et}CO_2$) parameters (Boiten, 1998; Boiten et al., 1994; Ley & Yelich, 1998; Mador & Tobin, 1991; Suess et al., 1980; Wientjes et al., 1998; Wilhelm et al., 2017). However, the literature is vastly limited when considering modifiable health behaviours that might relate to these responses. Sedentary behaviour is defined as “any waking behaviour characterized by an energy expenditure \leq 1.5 metabolic equivalents (METs), while in a sitting, reclining or lying posture” (Tremblay et al., 2017, p. 9). Physical activity is operationalised as “any bodily movement produced by skeletal muscles that results in energy expenditure” (Caspersen et al., 1985, p.126). Sedentary behaviour (Brocklebank et al., 2015; Campbell Jenkins et al., 2014; Edwardson et al., 2020) and physical activity (Campbell Jenkins et al., 2014; Cheng et al., 2003; Myers et al., 2019) are linked with metabolic parameters under resting conditions. These activity behaviours are also associated with elevated sympathetic tone (Tebar et al., 2020; Zaffalon Júnior et al., 2018), which is a key mechanism that drives large respiratory and cardiovascular stress response patterns (Brindle et al., 2014; Dampney, 2015). Previous work has demonstrated that high volumes of sedentary behaviour and low volumes of physical activity are associated with heightened cardiovascular stress reactivity (Chantry et al., 2022; Mücke et al., 2018), but it is unknown whether these activity behaviours relate to dysregulated respiratory responses to stress. If so, this could be a novel pathway linking sedentary behaviour and physical activity with cardiometabolic disease.

One study has shown that individuals who undertake regular physical activity (yoga vs non-yoga practitioners) show attenuated $\dot{V}O_2$ during a mental arithmetic stress task (Tyagi et al., 2014). This could represent a healthier metabolic response in those who are more physically active, as lower metabolic activity during stress (indicated by lower $\dot{V}O_2$) is likely compatible with the small

metabolic demand imposed by acute psychological stress (Balanos et al., 2010; Carroll et al., 2009). However, it cannot be ascertained whether this finding was driven by the psychological stress reducing effects of yoga, or the direct effect of physical activity (Tyagi et al., 2014). No pre-existing research has assessed the link between sedentary behaviour and respiratory responses to acute psychological stress.

Thus, the aim of this study was to investigate relationships between sedentary behaviour, physical activity, and respiratory stress reactivity. We hypothesised that higher volumes of sedentary behaviour would be associated with heightened ventilatory and gas exchange responses to stress (e.g., hyperventilation, higher $\dot{V}O_2$), whereas larger volumes of moderate-to-vigorous physical activity (MVPA) would be associated with smaller ventilatory and gas exchange (e.g., lower $\dot{V}O_2$) stress responses.

METHODS

Participants

Healthy adults (N = 61) were recruited as part of a wider study (Chaunty et al., 2022). Exclusion criteria included: acute illness, pre-existing medical conditions (e.g., asthma, hypertension, type 2 diabetes), taking regular medication (excluding oral contraceptives), body fat percentage > 32% (male); > 45% (female), and any smoking/vaping history. Written informed consent was gained and the study was approved by Loughborough University's Ethics Sub-Committee (R19-P011).

Measures

Sedentary behaviour

A gold-standard thigh-mounted inclinometer (20hz activPAL3 micro, PAL Technologies Ltd, Glasgow, UK) measured postural and metabolic components of sedentary behaviour. The activPAL defines sedentary behaviour as sitting or lying (thigh angle < 20 degrees above or below the horizontal plane) with MET of 1.25, which is in line with the widely accepted sedentary behaviour definition (Tremblay et al., 2017). Research has demonstrated near perfect agreement between

sedentary behaviour derived from the activPAL and direct observation (Edwardson et al., 2016; Kim et al., 2015). Data were recorded for seven continuous days and nights, starting from the first midnight after deployment. If reattachment was necessary, then participants were given detailed instructions of how to do this and were supported by a member of the research team. Validated software (Processing PAL, version 1.3, Leicester, UK) processed the data, including the removal of any periods of non-wear and/or sleep (Winkler et al., 2016). Any obvious algorithm-produced errors were manually corrected using heatmaps and participant log diaries, such as when a nap was reported in the diary but coded by the activPAL as a sedentary bout of the same duration (Edwardson et al., 2017). In line with other research, each participant required \geq one weekend day and \geq three weekdays of valid daily data for data inclusion (a valid day was defined as > 499 steps, $< 95\%$ of time in any one posture, > 10 hours of wear time) (Edwardson et al., 2017). Sedentary behaviour data is reported as mean hours per day.

Physical activity

An ActiGraph GT3X-BT+ triaxial accelerometer (ActiGraph, Florida, USA) was initialized to record data at 100 Hz using ActiLife version 6 (ActiGraph, Florida, USA), and worn on the non-dominant wrist continuously (24 hours/day) for seven days (e.g., Hildebrand *et al.*, 2014; Mikkelsen *et al.*, 2020). However, due to missing sleep time stamps on the final day of recording, there was a maximum of six analysable days of data per participant. Nevertheless, our wear time criteria still required \geq four valid days of data (\geq one weekend day and \geq three weekdays) with a valid day specified as ≥ 16 hours of wear time (Migueles et al., 2019). Data were analysed using the open-source R-package “GGIR”, version 2.0 (<http://cran.r-project.org>) which is described in full elsewhere (Migueles et al., 2019). MVPA was defined as > 100 milli-g (Hildebrand et al., 2017). All incidental physical activity was recorded and presented as mean hours per day.

Continuous respiratory activity

Breath-by-breath respiratory parameters were measured using the Cortex MetaLyzer 3B online gas-analysis system (Cortex Biophysic GmbH, Leipzig, Germany) during the final 8-min of baseline (baseline), during the 8-min PASAT (stress), and during the first 8-min of the post-stress recovery period (recovery). This yielded continuous quantification of breathing frequency (BF), tidal volume

(V_T), minute ventilation (\dot{V}_E), end-tidal carbon dioxide partial pressure ($P_{et}CO_2$), oxygen consumption ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$) and respiratory exchange ratio (RER). The cortex methodology has been described elsewhere and shows good reliability and validity (Meyer et al., 2001). Briefly, the system was calibrated in line with the manufacturer's instructions, with reference to atmospheric pressure, gas concentration, gas volume and participant anthropometry (MSS software, Cortex Biophysic GmbH, Leipzig, Germany). Next, a face mask (Hans Rudolph incorporated, Kansas City, USA) was tightly fitted, which included a Triple V low resistance volume transducer (Erich Jaeger GmbH, Hoechberg, Germany) and Nafion sample line (Perma Pure LLC, Lakewood, USA), connected into the MetaLyzer 3B base system. After each testing session continuous data were screened and cleaned for erroneous values (e.g., during any coughing/talking) and then collapsed into 8-min averages for baseline, stress, and recovery. Due to equipment failure, there were missing data for two participants, returning an analysable sample of $n = 59$ participants.

Psychological stress task

Participants completed an 8-min version of the Paced Auditory Serial Addition Test (PASAT) (Gronwall, 1977), a task which has been previously shown to induce changes in respiratory system activity (Plourde et al., 2017). Participants were presented with single digit numbers (1-9) via a standardised audio file and were required to add consecutive numbers, whilst retaining the most recent number in memory, to add to the next presented number. To prevent interference with respiratory assessment, participants reported their answers non-verbally by pointing to a number on a sheet of paper. The speed of number delivery progressively increased, such that during the first two minutes the time interval between the numbers was 2.4 seconds and was shortened by 0.4 seconds every two minutes thereafter.

To increase the provocativeness of the task, our PASAT protocol included additional components of social evaluation, competition and punishment (Chantry et al., 2022; Paine et al., 2014; Veldhuijzen Van Zanten et al., 2004). A researcher positioned one metre away obtrusively scored each participants performance (scores could range from 0 to 218; one point for each correct answer) and a single aversive noise burst was administered using a buzzer to signify error or hesitation (to ensure

standardisation, this buzzer was pressed once only during the last five numbers of every block of ten, and if there was no error or hesitation during this five number block, then the noise burst was administered after the 10th number had been presented – consequently, all participants received the same number of noise bursts). A large leader board was presented, and participants were informed that the highest scoring participant would receive an Amazon voucher. Finally, participants were video recorded and watched their PASAT performance live on a television screen directly in front of them. It was announced that “independent body language experts” would analyse their tape to scrutinise components of anxiety, but this was false, and the tape was deleted when the participant left the laboratory. There was a full debrief upon conclusion of the laboratory session.

The PASAT was specifically selected as our stress protocol because it is well used in the literature (Chantry et al., 2022; Turner et al., 2020), can be conducted with high levels of consistency, induces strong psychobiological responses to stress, and does not require vocalisation. Self-reported appraisal scores (stress, engagement, arousal, difficulty, perceived performance) were collected shortly after the stress task (as well as after the baseline and recovery periods) using 0 – 7 simple unipolar rating scales (e.g., Paine et al., 2013). Perceived performance during baseline and recovery measured how well participants perceived they could sit quietly and rest. These self-reported appraisal scores were used to support the physiological manipulation check data for the stress task but were not included in any main analyses.

Screening tools

Brachial blood pressure (Omron M6 comfort, Omron Healthcare, Milton Keynes, UK) was measured and socio-demographic data was collected via standard questionnaires (e.g., age, sex, occupational category). Participants were also asked to complete the 16-item Anxiety Sensitivity Index (Reiss et al., 1986), which measures physical, psychological and social concerns relating to anxiety. This questionnaire yields robust reliability and validity statistics (Vujanovic et al., 2007) and was selected because anxiety sensitivity is a potential covariate that may impact respiratory responses to sympathetic stimuli (Pané-Farré et al., 2015). Finally, anthropometric measures of height (274

stadiometer, Seca, Hamburg, Germany), weight and body fat percentage (mBCA 515 bioimpedance scales, Seca, Hamburg, Germany) were taken.

Procedure

A two-session observational design was employed. Session one checked participant eligibility, which included measuring resting brachial blood pressure and gathering socio-demographic data. The acitivPAL and ActiGraph devices were also fitted.

After a seven-day period of activity behaviour monitoring, participants reported to a temperature-controlled (20-22°C) laboratory (between 1pm and 2pm) to complete a stress reactivity protocol. Participants refrained from over-the-counter medication for 7 days, vigorous exercise for 24 hours, alcohol for 12 hours, drinking anything other than water for 4 hours, and eating for 2 hours. All relevant equipment was attached, and then whilst seated participants completed a 20-min resting baseline period (baseline), followed by an 8-min active psychological stress task (stress) and 45-min recovery period (recovery). Participants quietly watched a nature documentary (Blue Planet or Planet Earth 2; BBC) when data were not being collected, with any episodes/scenes likely to induce higher arousal avoided.

Data analysis

Generalized estimating equations with an autoregressive (AR(1)) correlation structure and suitable distribution and link selection (e.g., gamma distribution with log link for variables with residuals showing positive skew) were used to investigate the effect of time for each respiratory and self-reported appraisal score parameter (i.e., manipulation check regarding the stress task).

Fully adjusted models: For our primary analyses, generalized linear regression models were tested in which the outcomes of interest (each resting baseline respiratory parameter and each respiratory parameter computed as a change variable from baseline to stress) were associated with (1) the predictors of interest (daily hours of sedentary behaviour and MVPA), and (2) a priori defined covariates. There were separate models for each respiratory parameter. Log links were applied to models with respiratory variables showing positively skewed residuals, and unstandardized B-

coefficients are reported to reflect effect size, along with 95% Wald confidence intervals. These models were also adjusted for covariates selected *a priori* based on pre-existing literature (i.e., these covariates were selected because they are known to relate to respiratory/sympathetic responses to sympathetic stimuli [such as psychological stress] and/or sedentary behaviour/physical activity): age, sex, body fat percentage, anxiety sensitivity index score, activPAL waking wear time, ActiGraph waking wear time, and for the models assessing rest to stress respiratory changes, the appropriate respiratory parameter under resting baseline conditions (Hamer et al., 2020; Plourde et al., 2017; Steptoe et al., 2016; Turner et al., 2020). The associations between these covariates (including sedentary behaviour and MVPA) are shown in Supplementary Material 1 (Supplementary Table 1). In sum, these fully adjusted analyses reflect our strongest models for assessing independent associations between activity behaviours and respiratory stress reactivity, by adjusting for the effects of covariates that might otherwise explain variance in the data.

Partially adjusted models: Anxiety sensitivity was selected as a covariate because it has been shown to relate to respiratory responses to sympathetic stimuli (Pané-Farré et al., 2015). However, as anxiety sensitivity also predicts psychological responses to respiratory perturbations (Blechert et al., 2013), it could be considered a controversial covariate to include. Therefore, we also re-ran our fully adjusted models, which included sedentary behaviour, MVPA, and our *a priori*-selected covariates, with the exception of anxiety sensitivity – these models can also be found in Supplementary Material 1 (Supplementary Tables 4 and 5).

Unadjusted models: Due to the large number of covariates that were included in the primary models above, there is a possibility of statistical model overfitting. Therefore, we also ran unadjusted analyses, allowing the crude relationships between activity behaviours and respiratory stress reactivity to be explored. Generalized linear models examined associations between daily hours of sedentary behaviour (without MVPA and other covariates) and (1) each resting baseline respiratory parameter, and (2) each respiratory parameter computed as a change variable from baseline to stress. The same procedure was then conducted for daily hours of MVPA (i.e., without sedentary behaviour and other

covariates). These unadjusted models can be found in Supplementary Material 1 (Supplementary Tables 6 and 7).

All data were analysed using IBM SPSS version 27, with significance set at 0.05. The Holm-Bonferroni correction (Holm, 1979) was used to adjust for multiple testing of our generalized linear models, which yielded adjusted p values for the predictors in each model (i.e., sedentary behaviour and MVPA). As we did not draw any conclusions regarding the association between our covariates and respiratory stress reactivity measures, these covariates have not been included in this Holm-Bonferroni adjustment process.

RESULTS

Participant characteristics

The characteristics of our sample ($n = 61$) are summarised in Table 1. Participants were mainly young adults (mean age \pm SD = 25.7 ± 8.9 years), 29 (48%) were male, and body fat percentage levels were variable (ranging from 8.4% to 43.7%). Device adherence was strong, including that 80% of participants registered seven valid days of activPAL data, with 83% recording the maximum of six days for the ActiGraph. All participants satisfied activPAL and ActiGraph wear time criteria to be included in analyses. Daily hours of sedentary behaviour (mean \pm SD = 9.96 ± 1.48 hours) and MVPA (mean \pm SD = 1.70 ± 0.71 hours) are comparable to national averages derived from similar methodological approaches (Hamer et al., 2020; Ramakrishnan et al., 2021). Although MVPA volume appears high, this was expected given that all incidental physical activity was measured using a wearable device with no bout criteria (Thompson et al., 2016).

Table 1. Characteristics of the sample (N=61).

	Mean (SD) / N (%)
<i>Sociodemographic, anthropometric, and psychological data</i>	
Age (years)	25.7 (8.9)
Sex (male)	29 (48)
Body mass index (kg/m ²)	24.4 (4.2)
Body fat percentage (%)	25.5 (9.6)
White ethnicity	50 (82)
A non-manual occupation category for the head of the household	39 (64)
PASAT performance score (from 0 to 281)	101 (36)
Anxiety Sensitivity Index score (from 0 to 64)	16.9 (9.3)
<i>Sedentary behaviour data (activPAL)</i>	
Average daily hours of sedentary behaviour (hours)	10.0 (1.5)
Average number of days that the activPAL was worn (days)	6.7 (0.2)
Average daily waking hours that the activPAL was worn (hours)	15.2 (1.1)
Participants with 100% activPAL compliance (i.e., 7 days wear)	49 (80)
<i>Physical activity data (ActiGraph)</i>	
Average daily hours of moderate-to-vigorous physical activity (MVPA; hours)	1.7 (0.7)
Average number of days that the ActiGraph was worn (days)	5.8 (0.5)
Average daily waking hours that the ActiGraph was worn (hours)	15.9 (0.9)
Participants with 100% ActiGraph compliance (i.e., 6 days wear)	51 (84)

Note. PASAT=Paced Auditory Serial Addition Test. Higher anxiety sensitivity index scores indicate greater levels of anxiety sensitivity.

Psychorespiratory responses to the stress task (manipulation check)

As demonstrated in Table 2, our PASAT paradigm induced respiratory responses. There were significant changes in reaction to stress for ventilatory parameters: BF [Wald $\chi^2(2) = 148.57, p < .001, V = .65$], \dot{V}_E [Wald $\chi^2(2) = 145.07, p < .001, V = .85$] and PetCO₂ [Wald $\chi^2(2) = 33.23, p < .001, V = .32$], as well as gas exchange parameters: $\dot{V}O_2$ [Wald $\chi^2(2) = 139.43, p < .001, V = .63$], $\dot{V}CO_2$ [Wald $\chi^2(2) = 184.16, p < .001, V = .72$] and RER [Wald $\chi^2(2) = 57.21, p < .001, V = .40$]. The stress task did not alter V_T [Wald $\chi^2(2) = 0.40, p = .82, V = .03$]. Associations between these respiratory stress reactivity markers can be found in Supplementary Material 1.

For the self-reported appraisal scores (see Table 2), participants reported the PASAT to be stressful [Wald $\chi^2(2) = 549.33, p < .001, V = .89$], engaging [Wald $\chi^2(2) = 82.40, p < .001, V = .47$], arousing [Wald $\chi^2(2) = 63.04, p < .001, V = .42$], and difficult [Wald $\chi^2(2) = 864.07, p < .001, V = .91$], and believed they performed poorly [Wald $\chi^2(2) = 176.71, p < .001, V = .69$].

Table 2. Psychorespiratory responses to the Paced Auditory Serial Addition Test (PASAT).

	Baseline	Stress (PASAT)	Recovery	Wald χ^2	DF	<i>p</i>	Cra mér' s <i>V</i>
Ventilatory parameters							
Breathing frequency (BF; breaths/min)	15.09 ± 0.41	20.21 ± 0.58*	16.71 ± 0.46†*	148.5 7	2	<.001	0.65
Tidal volume (V_T ; l/breath)	0.62 ± 0.02	0.63 ± 0.02	0.62 ± 0.02	0.40	2	.82	0.03
Minute ventilation (\dot{V}_E ; l/min)	8.66 ± 0.25	11.62 ± 0.31*	9.40 ± 0.25†*	145.0 7	2	<.001	0.85
End-tidal carbon dioxide partial pressure (PetCO ₂ ; mmHg)	33.87 ± 0.56	33.16 ± 0.59*	32.54 ± 0.53†*	33.23	2	<.001	0.32
Gas exchange parameters							
Oxygen consumption ($\dot{V}O_2$; l/min)	0.26 ± 0.01	0.32 ± 0.01*	0.27 ± 0.01†*	139.4 3	2	<.001	0.63
Carbon dioxide output ($\dot{V}CO_2$; l/min)	0.23 ± 0.01	0.30 ± 0.01*	0.24 ± 0.01†*	184.1 6	2	<.001	0.72
Respiratory exchange ratio ($\dot{V}CO_2/\dot{V}O_2$)	0.89 ± 0.01	0.95 ± 0.01*	0.88 ± 0.01†	57.21	2	<.001	0.40
Self-reported psychological parameters							
Stress (from 0 to 7)	0.61 ± 0.11	4.49 ± 0.16*	0.38 ± 0.09†	549.3 3	2	<.001	0.89
Engagement (from 0 to 7)	2.61 ± 0.22	4.30 ± 0.16*	2.43 ± 0.20†	82.40	2	<.001	0.47
Difficulty (from 0 to 7)	0.98 ± 0.16	4.90 ± 0.12*	0.48 ± 0.11†*	864.0 7	2	<.001	0.91
Arousal (from 0 to 7)	1.34 ± 0.19	2.97 ± 0.19*	1.30 ± 0.17†	63.04	2	<.001	0.42
Perceived performance (from 0 to 7)	3.92 ± 0.19	1.49 ± 0.14*	4.38 ±0.17†*	176.7 1	2	<.001	0.69

Note. Data shows generalized estimating equation (GEE) repeated measure models and represents main effects of time in terms of psychorespiratory changes in response to stress. PASAT=Paced Auditory Serial Addition Test, DF=degrees of freedom. Data are mean ± standard error, *p* values are unadjusted. Bold text indicates statistical significance at the *p* < .05 level. Wald χ^2 =Wald chi-square statistic. * = significantly different from baseline, † = significantly different from stress (*p* < .05). For self-reported psychological responses, higher scores imply greater levels.

Fully adjusted associations between sedentary behaviour, moderate-to-vigorous physical activity (MVPA), and respiratory parameters under baseline conditions

In fully adjusted models including daily hours of sedentary behaviour, daily hours of MVPA, and all covariates, the only significant relationship that emerged was between hours per day of sedentary behaviour and baseline BF (*B* = 0.702, 95% CI = 0.339 — 1.066, *p* = .014). As detailed in Supplementary Material 1 (Supplementary Table 3), sedentary behaviour and MVPA were not associated with baseline V_T , V_E , PetCO₂, $\dot{V}O_2$, $\dot{V}CO_2$ or RER (all *p* > .05).

Fully adjusted associations between sedentary behaviour, moderate-to-vigorous physical activity (MVPA), and magnitude of respiratory changes from baseline to stress

As shown in Table 3, in fully adjusted models including daily hours of sedentary behaviour, daily hours of MVPA, and all covariates, daily hours of sedentary behaviour were positively associated with baseline to stress changes in BF ($B = 0.695$, 95% CI = 0.281 — 1.109, $p = .012$), and negatively related with baseline to stress changes in V_T ($B = -0.042$, 95% CI = -0.058 — -0.026, $p < .001$), $P_{et}CO_2$ ($B = -0.537$, 95% CI = -0.829 — -0.245, $p < .001$), $\dot{V}CO_2$ ($B = -0.008$, 95% CI = -0.014 — -0.003, $p = .030$) and RER ($B = -0.013$, 95% CI = -0.021 — -0.005, $p = .022$). There was evidence of a negative relationship between hours of daily sedentary behaviour and the baseline to stress $\dot{V}O_2$ response, but this was reduced to a trend after the Holm-Bonferroni correction was applied ($B = -0.007$, 95% CI = -0.012 — -0.002, $p = .088$). Again, hours of daily MVPA were not linked with baseline to stress changes for any respiratory parameter (Table 3; all $p > .05$).

Table 3. Generalized linear regression models that examine associations between activity behaviours (sedentary behaviour and MVPA; predictors) and baseline-to-stress respiratory responses (outcomes), with full adjustment for covariates.

	B	SE	95% Wald Confidence Interval		p
			Lower	Upper	
<i>Δ Breathing frequency from baseline to stress (BF; breaths/min)</i>					
Daily hours of sedentary behaviour	0.695	0.21	0.281	1.109	.012^b
Daily hours of MVPA	0.403	0.41	-0.399	1.204	1.00 ^b
Age (years)	-0.065	0.03	-0.123	-0.008	.027 ^c
Sex (males) ^a	-0.779	0.67	-2.087	0.530	.24 ^c
Body fat percentage	0.023	0.04	-0.051	0.098	.54 ^c
activPAL waking wear time (hours)	-0.835	0.31	-1.437	-0.233	.007 ^c
ActiGraph waking wear time (hours)	0.012	0.01	0.002	0.022	.020 ^c
Anxiety sensitivity index score	-0.057	0.03	-0.114	0.000	.049 ^c
Baseline breathing frequency (breaths/min)	-0.190	0.08	-0.352	-0.029	.021 ^c
<i>Δ Tidal volume from baseline to stress (V_T; l/breath)</i>					
Daily hours of sedentary behaviour	-0.042	0.01	-0.058	-0.026	<.001^b
Daily hours of MVPA	-0.036	0.02	-0.069	-0.004	.18 ^b
Age (years)	0.004	0.00	0.002	0.006	<.001 ^c
Sex (males) ^a	0.149	0.03	0.094	0.205	<.001 ^c
Body fat percentage	0.004	0.00	0.001	0.007	.006 ^c
activPAL waking wear time (hours)	0.032	0.01	0.008	0.057	.010 ^c
ActiGraph waking wear time (hours)	-0.001	0.00	-0.001	0.000	.002 ^c
Anxiety sensitivity index score	0.002	0.00	0.000	0.005	.040 ^c
Baseline tidal volume (l/breath)	-0.587	0.06	-0.708	-0.467	<.001 ^c
<i>Δ Minute ventilation from baseline to stress (V̇_E; l/min)</i>					
Daily hours of sedentary behaviour	-0.125	0.13	-0.373	0.123	1.00 ^b
Daily hours of MVPA	-0.083	0.25	-0.580	0.414	1.00 ^b
Age (years)	-0.014	0.02	-0.049	0.021	.44 ^c
Sex (males) ^a	1.665	0.43	0.813	2.498	<.001 ^c
Body fat percentage	0.091	0.02	0.045	0.137	<.001 ^c
activPAL waking wear time (hours)	0.170	0.19	-0.204	0.544	.34 ^c
ActiGraph waking wear time (hours)	-0.002	0.00	-0.008	0.004	.53 ^c
Anxiety sensitivity index score	-0.005	0.02	-0.039	0.030	.79 ^c
Baseline minute ventilation (l/min)	-0.335	0.08	-0.500	-0.171	<.001 ^c
<i>Δ End-tidal carbon dioxide partial pressure from baseline to stress (PetCO₂; mmHg)</i>					
Daily hours of sedentary behaviour	-0.537	0.15	-0.829	-0.245	<.001^b
Daily hours of MVPA	-0.770	0.28	-1.319	-0.220	.054 ^b
Age (years)	0.022	0.02	-0.015	0.060	.24 ^c
Sex (males) ^a	2.032	0.48	1.093	2.971	<.001 ^c

Body fat percentage	-0.003	0.03	-0.056	0.050	.90 ^c
activPAL waking wear time (hours)	0.680	0.23	0.225	1.14	.003 ^c
ActiGraph waking wear time (hours)	-0.007	0.00	-0.014	0.000	.050 ^c
Anxiety sensitivity index score	0.003	0.02	-0.037	0.044	.87 ^c
Baseline PetCO ₂ (mmHg)	-0.133	0.04	-0.212	-0.054	.001 ^c
<i>Δ Oxygen consumption from baseline to stress ($\dot{V}O_2$; l/min)</i>					
Daily hours of sedentary behaviour	-0.007	0.01	-0.012	-0.002	.088 ^b
Daily hours of MVPA	-0.009	0.01	-0.019	0.002	.50 ^b
Age (years)	0.000	0.00	-0.001	0.001	.81 ^c
Sex (males) ^a	0.027	0.01	0.007	0.047	.007 ^c
Body fat percentage	0.001	0.00	0.000	0.002	.010 ^c
activPAL waking wear time (hours)	0.008	0.00	0.000	0.016	.041 ^c
ActiGraph waking wear time (hours)	0.000	0.00	0.000	0.001	.89 ^c
Anxiety sensitivity index score	0.001	0.00	0.000	0.002	.022 ^c
Baseline oxygen consumption (l/min)	-0.078	0.06	-0.201	0.045	.21 ^c
<i>Δ Carbon dioxide output from baseline to stress ($\dot{V}CO_2$; l/min)</i>					
Daily hours of sedentary behaviour	-0.008	0.01	-0.014	-0.003	.030^b
Daily hours of MVPA	-0.011	0.02	-0.022	0.001	.37 ^b
Age (years)	0.000	0.00	-0.001	0.000	.26 ^c
Sex (males) ^a	0.046	0.01	0.026	0.066	<.001 ^c
Body fat percentage	0.002	0.00	0.001	0.003	<.001 ^c
activPAL waking wear time (hours)	0.011	0.00	0.003	0.019	.010 ^c
ActiGraph waking wear time (hours)	0.000	0.00	0.000	0.001	.48 ^c
Anxiety sensitivity index score	0.001	0.00	0.000	0.001	.10 ^c
Baseline carbon dioxide output (l/min)	-0.096	0.07	-0.237	0.046	.18 ^c
<i>Δ Respiratory exchange ratio from baseline to stress (RER; $\dot{V}CO_2/\dot{V}O_2$)</i>					
Daily hours of sedentary behaviour	-0.013	0.01	-0.021	-0.005	.022^b
Daily hours of MVPA	-0.005	0.01	-0.021	0.012	1.00 ^b
Age (years)	0.000	0.00	-0.001	0.001	.66 ^c
Sex (males) ^a	0.031	0.01	0.005	0.058	.022 ^c
Body fat percentage	0.002	0.00	0.001	0.004	.006 ^c
activPAL waking wear time (hours)	0.006	0.00	-0.007	0.019	.36 ^c
ActiGraph waking wear time (hours)	0.000	0.00	0.000	0.001	.46 ^c
Anxiety sensitivity index score	-0.001	0.00	-0.002	0.000	.20 ^c
Baseline respiratory exchange ratio ($\dot{V}CO_2/\dot{V}O_2$)	-0.389	0.06	-0.509	-0.270	<.001 ^c

Note. ^a = compared to females as the reference group, ^b = adjusted *p* value (Holm-Bonferroni corrected for multiple testing), ^c = unadjusted *p* value. SE = standard error, MVPA = moderate-to-vigorous physical activity. Each model (separate for each respiratory parameter) included sedentary behaviour, MVPA, and *a priori* selected covariates (age, sex, body fat %, anxiety sensitivity index score, activPAL waking wear time, ActiGraph waking wear time and the appropriate baseline respiratory parameter). Bold font indicates statistical significance for either sedentary behaviour or MVPA (Holm-Bonferroni adjusted *p* < .05).

DISCUSSION

This is the first study to explore associations between device-assessed activity behaviours (sedentary behaviour and MVPA) and respiratory responses to acute psychological stress. In fully adjusted models with both sedentary behaviour and MVPA (and covariates) included, daily hours of sedentary behaviour were positively associated with BF responses to stress (and BF under resting conditions), and negatively associated with V_T , $PetCO_2$, $\dot{V}CO_2$, and RER stress responses. There was no link between sedentary behaviour and \dot{V}_E or $\dot{V}O_2$ stress reactivity. MVPA was not related to responses to stress for any respiratory outcome.

Sedentary behaviour and respiratory responses to stress

Ventilatory responses to stress

Sedentary behaviour was positively associated with baseline and stress-induced BF, as well as negatively associated with V_T responses to stress, but there was no relationship with \dot{V}_E reactivity. Thus, while it seems that sedentary behaviour does not relate to stress-induced changes in the volume of inhaled and exhaled air per minute (i.e., \dot{V}_E), those with higher volumes of habitual sedentary produced a more rapid and shallow breathing pattern under stress. However, it must be noted that rapid and shallow breathing is a common response to laboratory-based stress tasks (Boiten et al., 1994; Suess et al., 1980; Wilhelm et al., 2017) and therefore may not be uniquely associated with sedentary behaviour. Nevertheless, the breathing pattern above is a possible explanation for the negative relationship that was found between sedentary behaviour and the baseline to stress change in $PetCO_2$, given that a decrease in $PetCO_2$ can reflect hyperventilation (Meuret & Ritz, 2010).

However, it is of interest to point out that as there was not a positive association between sedentary behaviour and \dot{V}_E , it is plausible that the ventilatory impact of the baseline-to-stress increase in BF was counteracted by decreased in \dot{V}_T , and that the observed negative association with $PetCO_2$ was a consequence of faster, shallower breathing on alveolar gas elimination, rather than a reflection of hypocapnia and hyperventilation. In essence, alveolar CO_2 may not have been captured in full due to fast and shallow breathing under stress, therefore making $PetCO_2$ appear lower.

It is also important to note that a valid estimation of arterial PCO_2 via the measurement of PetCO_2 requires thorough washout of dead-space air during expiration, because incomplete washout leads to the mixing of alveolar and dead-space air, and, consequently, an underestimation of arterial PCO_2 levels (McSwain et al., 2010). Importantly, incomplete washout is often observed during fast, shallow breathing (which occurs during acute psychological stress tasks), and unfortunately, due the methods and equipment used in this study it was not possible to inspect whether the expiratory PCO_2 curve reached a flat plateau at the end of expiration, which would verify whether PetCO_2 provides a valid estimation of arterial PCO_2 levels. In sum, further research is needed to confirm whether sedentary behaviour is linked to hyperventilation under stress.

If future research were to confirm that hyperventilation under stress is observed in those with high volumes of sedentary behaviour, then this may be important because stress-induced hyperventilation can predict cardiometabolic disease risk factors, such as elevated levels of vasoconstriction (Grossman, 1983; Rutherford et al., 2005), and might reflect physiological-metabolic uncoupling during stress, which is considered a mechanism from exaggerated psychobiological stress reactivity to cardiometabolic disease (Balanos et al., 2010; Carroll et al., 2009).

Interestingly, our other work within the same sample of participants found that activPAL-measured sedentary behaviour was positively associated with responses to stress for cardiovascular (diastolic blood pressure and total peripheral resistance), inflammatory (interleukin-6), and cortisol outcomes (Chantry et al., 2022). Collectively, these stress responses suggest that sedentary behaviour is related to heightened sympathetic tone under stress (Brindle et al., 2014; Dampney, 2015), which provides a mechanistic rationale for future research that may choose to explore sedentary behaviour and hyperventilation during stress. This later work would also benefit from adopting a more precise threshold of hyperventilation and using equipment that can assess PetCO_2 plateau (e.g., Gardner, 1996).

Sedentary behaviour and gas exchange responses to stress

Sedentary behaviour was negatively associated with stress-induced changes in $\dot{V}\text{CO}_2$, with a negative trend for $\dot{V}\text{O}_2$ changes. However, given that the relationship between sedentary behaviour and $\dot{V}\text{O}_2$

stress reactivity was reduced to a trend after the Holm-Bonferroni correction was applied, caution must be taken when interpreting this finding, and further replication work is clearly needed. Nevertheless, as $\dot{V}O_2$ and $\dot{V}CO_2$ changes can be indicative of metabolic activity (Tyagi et al., 2014), our results suggest that larger decreases in baseline to stress metabolic activity were found in those with higher volumes of habitual sedentary behaviour. Studies examining other populations with poor cardiometabolic health (e.g., those with type 2 diabetes) have found attenuated cardiovascular, cortisol, inflammatory, and cholesterol responses to acute psychological stress (Steptoe et al., 2014), but to our knowledge no studies have examined respiratory stress responses in this context. Nevertheless, lower metabolic (e.g., $\dot{V}O_2$) responses to exercise challenge in type 2 diabetic individuals have been observed (Regensteiner et al., 1995), which may be relevant because psychological and sub-maximal exercise stimuli are often comparable in terms of the cardiovascular activation they induce (Balanos et al., 2010; Carroll et al., 2009).

Our findings for $\dot{V}O_2$ and $\dot{V}CO_2$ could reflect the inability to physiologically or sympathetically respond to stress in the expected manner, although it should be noted that this is highly speculative. This could potentially be important because there is an emerging literature linking attenuated cardiovascular and sympathetic stress reactivity to CVD risk factors (Turner et al., 2020). From a cardio-metabolic perspective, because sedentary behaviour is associated with positive cardiovascular (Chantry et al., 2022) and negative metabolic (indicated by $\dot{V}CO_2$), responses to stress, this could possibly highlight cardio-metabolic uncoupling under stress (Balanos et al., 2010; Carroll et al., 2009; Lambiase et al., 2012) in individuals with higher volumes of sedentary behaviour.

However, from a purely metabolic perspective, a negative relationship between sedentary behaviour and metabolic responses to stress could reflect a healthy level of synchronicity between the metabolic activity of the body and the small metabolic burden induced by psychological stress. Further research is needed to untangle the cardiometabolic consequences of these findings, which would benefit from integrating cardiovascular (including heart rate variability) and respiratory responses to psychological stressors within the same study.

Sedentary behaviour was negatively associated with RER responses to acute psychological stress, which might reflect higher levels of whole body fat oxidation, and smaller rates of carbohydrate metabolism, under stress (Pendergast et al., 2000; Ramos-Jiménez et al., 2008). Others have reported similar findings under conditions of rest when comparing endurance-trained and sedentary (categorised by low physical activity) individuals (Smorawiński et al., 2001). Our study furthers the literature by examining associations between posturally-measured sedentary behaviour and RER responses to psychological stimuli, but further research is needed to explore the longer-term implications for health outcomes, including obesity and type 2 diabetes mellitus.

Moderate-to-vigorous physical activity and respiratory responses to stress

Links to previous research

Daily hours of MVPA were not associated with baseline to stress respiratory (gas exchange or ventilatory) responses (see Table 3). This contrasts a previous study which showed that individuals who undertake regular physical activity (yoga) show attenuated $\dot{V}O_2$ during psychological stress, when compared to individuals who do not regularly partake in yoga (Tyagi et al., 2014). One reason could be the stress-reducing effects of yoga, which were not accounted for in our study. A secondary explanation is that yoga is primarily lower intensity physical activity, whereas we explored higher intensity physical activity; it is possible that lower intensity physical activity is more important for attenuating metabolic responses to stress, but this needs to be directly tested.

Potential reasons for the non-significant findings between moderate-to-vigorous physical activity and respiratory stress reactivity

The lack of significant findings for MVPA in this current study contrasts the cardiovascular reactivity literature, where MVPA appears to be an important factor linked to the attenuation of cardiovascular responses to stress (Mücke et al., 2018). As MVPA is strongly associated with sympathetic pathways (Soares-Miranda et al., 2011), this could indicate that sympathetic mechanisms are not the only driving force underpinning our observed respiratory findings. For example, the direct influence of neural signals from the dorsomedial hypothalamus is also likely to be important (Dampney, 2015). One speculative explanation for the absence of significant MVPA findings in this study relates to the

low metabolic nature of our stress protocol. MVPA is highly reflective of cardiorespiratory fitness (Naylor et al., 2021), and therefore might be more important for respiratory changes in response to paradigms with a metabolic component.

The potential use of psychological stress testing to indicate psychobiological dysregulation

There were relatively consistent findings between sedentary behaviour and respiratory stress reactivity measures (see Table 3), but associations between sedentary behaviour and the same respiratory parameters under resting conditions did not always manifest (see Supplementary Material 1; Supplementary Table 3). Interestingly, this phenomena has been observed for other cardiometabolic disease risk factors (e.g., depression, tobacco smoking) and stress reactivity measures across the cardiovascular and neuroendocrine systems (Kiecolt-Glaser et al., 2020; Kirschbaum et al., 1993). Our preliminary findings appear to provide further support for psychological stress testing as a paradigm to explore physiological dysregulation that has not yet surfaced under resting conditions (Kiecolt-Glaser et al., 2020; Turner et al., 2020; Zaffalon Júnior et al., 2018). We have taken a novel approach by attempting to extend this “stress-induced physiological dysregulation” model to the respiratory system, but further research is clearly warranted.

Methodological considerations

This study has key strengths. It is the first piece of research to explore the link between respiratory responses to acute psychological stress and activity behaviours. We also used gold-standard device-based approaches to measure sedentary behaviour (accounting for postural and metabolic components) and physical activity (analysed using highly reproducible methods based on raw gravitational acceleration). Finally, we adjusted for a diverse range of covariates within generalized statistical models that are less susceptible to overfitting, although a limitation of using these models is the lack of standardised effect size metrics. It is also possible that other potentially important covariates (e.g., cardiorespiratory fitness, other metabolic traits) were missed.

There are also other considerations to take into account. First, correcting for multiple testing may have increased type II error rate, but this was deemed necessary due to the large number of analyses that were run. Second, our findings are only applicable to young healthy adults. Future research could test

clinical respiratory populations (e.g., individuals with asthma and chronic obstructive pulmonary disease), given that these populations show poor metabolic health, and increasing physical activity volume is a key component of pulmonary rehabilitation (Spruit et al., 2013). Third, intrusive measurement techniques (e.g., using a face mask) within a laboratory environment may have altered natural breathing patterns (Askanazi et al., 1980) (including the possibility that these factors may have contributed to the lower-than-expected PetCO₂ values at rest and in response to stress) and negatively influenced ecological validity. Although unobtrusive techniques could have been adopted, these were not selected because it would not have been feasible to quantify gas exchange parameters. For future studies it would also be advisable to combine metabolic assessment and spirometry with capnographic measurement that allows for a careful visual or other inspection of the pCO₂ curves in order to avoid that breaths that fail to reach a flat end-expiratory plateau are included in the analyses. Fourth, given the novelty of this exploratory study and lack of prior research, we were not able to conduct an *a priori* power calculation. Our data was collected as part of another ongoing study (Chantry et al., 2022) which was powered to detect associations between sedentary behaviour and cardiovascular stress reactivity outcomes. Given the opportunistic nature of our respiratory stress reactivity analyses, being underpowered is highly possible, which is important in relation to the risk of statistical model overfitting, caused by the high number of covariates relative to our modest sample size. Although as mentioned above, generalized statistical models are less susceptible to overfitting and we also presented unadjusted models without covariates. It is important to highlight the implications if our study is underpowered, including that associations which failed to attain statistical significance should be viewed guardedly (as they may have been significant with a larger sample size) and it is possible that the magnitude of our effects was overestimated. While the pattern of our findings was largely consistent with the unadjusted models, future replication studies that are confirmed as adequately powered are essential. Finally, because of compositional and collinearity issues we were not able to introduce light intensity physical activity into our models (van der Ploeg & Hillsdon, 2017).

Conclusion

This is the first study to examine associations between activity behaviours and respiratory responses to acute psychological stress. Hours of daily sedentary behaviour, but not MVPA, were associated with respiratory responses to acute psychological stress, including positive associations with BF responses, and negative links with PetCO₂, V_T, \dot{V} CO₂, and RER responses. Further research is needed to unravel the underlying mechanisms of our preliminary findings and explore the possible consequences for cardiometabolic disease.

REFERENCES

- Askanazi, J., Silverberg, P. A., Foster, R. J., Hyman, A. I., Milic-Emili, J., & Kinney, J. M. (1980). Effects of respiratory apparatus on breathing pattern. *Journal of Applied Physiology Respiratory Environmental and Exercise Physiology*, *48*(4), 577–580. <https://doi.org/10.1152/jappl.1980.48.4.577>
- Balanos, G. M., Phillips, A. C., Frenneaux, M. P., McIntyre, D., Lykidis, C., Griffin, H. S., & Carroll, D. (2010). Metabolically exaggerated cardiac reactions to acute psychological stress: The effects of resting blood pressure status and possible underlying mechanisms. *Biological Psychology*, *85*(1), 104–111. <https://doi.org/10.1016/j.biopsycho.2010.06.001>
- Blechert, J., Wilhelm, F. H., Meuret, A. E., Wilhelm, E. M., & Roth, W. T. (2013). Experiential, autonomic, and respiratory correlates of CO₂ reactivity in individuals with high and low anxiety sensitivity. *Psychiatry Research*, *209*(3), 566–573. <https://doi.org/10.1016/j.psychres.2013.02.010>
- Blix, A. S., Stromme, S. B., & Ursin, H. (1974). Additional heart rate - an indicator of psychological activation. *Aerospace Medicine*, *45*(11), 1219–1222.
- Boiten, F. A. (1998). The effects of emotional behaviour on components of the respiratory cycle. *Biological Psychology*, *49*(2), 29–51. [https://doi.org/10.1016/S0301-0511\(98\)00025-8](https://doi.org/10.1016/S0301-0511(98)00025-8)
- Boiten, F. A., Frijda, N. H., & Wientjes, C. J. E. (1994). Emotions and respiratory patterns: review and critical analysis. *International Journal of Psychophysiology*, *17*(2), 103–128. [https://doi.org/10.1016/0167-8760\(94\)90027-2](https://doi.org/10.1016/0167-8760(94)90027-2)
- Brindle, R. C., Ginty, A. T., Phillips, A. C., & Carroll, D. (2014). A tale of two mechanisms: A meta-analytic approach toward understanding the autonomic basis of cardiovascular reactivity to acute psychological stress. *Psychophysiology*, *51*(10), 964–976. <https://doi.org/10.1111/psyp.12248>
- Brocklebank, L. A., Falconer, C. L., Page, A. S., Perry, R., & Cooper, A. R. (2015). Accelerometer-measured sedentary time and cardiometabolic biomarkers: A systematic review. *Preventive Medicine*, *76*, 92–102. <https://doi.org/10.1016/j.ypmed.2015.04.013>
- Campbell Jenkins, B. W., Sarpong, D. F., Addison, C., White, M. S., Hickson, D. M. A., White, W., & Burchfiel, C. (2014). Joint effects of smoking and sedentary lifestyle on lung function in African Americans: The Jackson heart study cohort. *International Journal of Environmental Research and Public Health*, *11*(2), 1500–1519. <https://doi.org/10.3390/ijerph110201500>
- Carroll, D., Phillips, A. C., & Balanos, G. M. (2009). Metabolically exaggerated cardiac reactions to acute psychological stress revisited. *Psychophysiology*, *46*(2), 270–275. <https://doi.org/10.1111/j.1469-8986.2008.00762.x>
- Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise and physical fitness definitions for health-related research. *Public Health Reports*, *100*(2), 126–131.

- Chantry, A. J., Bishop, N. C., Hamer, M., Kingsnorth, A. P., Chen, Y. L., & Paine, N. J. (2022). Sedentary behaviour is associated with heightened cardiovascular, inflammatory and cortisol reactivity to acute psychological stress. *Psychoneuroendocrinology*, *141*(3), 105756. <https://doi.org/10.1016/j.psyneuen.2022.105756>
- Cheng, Y. J., Macera, C. A., Addy, C. L., Sy, F. S., Wieland, D., & Blair, S. N. (2003). Effects of physical activity on exercise tests and respiratory function. *British Journal of Sports Medicine*, *37*(6), 521–528. <https://doi.org/10.1136/bjism.37.6.521>
- Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension*, *55*(4), 1026–1032. <https://doi.org/10.1161/HYPERTENSIONAHA.109.146621>
- Dampney, R. A. L. (2015). Central mechanisms regulating coordinated cardiovascular and respiratory function during stress and arousal. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology*, *309*(5), 429–443. <https://doi.org/10.1152/ajpregu.00051.2015>
- Edwardson, C. L., Henson, J., Biddle, S. J. H., Davies, M. J., Khunti, K., Maylor, B., & Yates, T. (2020). activPAL and ActiGraph assessed sedentary behavior and cardiometabolic health markers. *Medicine and Science in Sports and Exercise*, *52*(2), 391–397. <https://doi.org/10.1249/MSS.0000000000002138>
- Edwardson, C. L., Rowlands, A. V., Bunnewell, S., Sanders, J., Esliger, D. W., Gorely, T., O'Connell, S., Davies, M. J., Khunti, K., & Yates, T. (2016). Accuracy of posture allocation algorithms for thigh- and waist-worn accelerometers. *Medicine and Science in Sports and Exercise*, *48*(6), 1085–1090. <https://doi.org/10.1249/MSS.0000000000000865>
- Edwardson, C. L., Winkler, E. A. H. H., Bodicoat, D. H., Yates, T., Davies, M. J., Dunstan, D. W., & Healy, G. N. (2017). Considerations when using the activPAL monitor in field-based research with adult populations. *Journal of Sport and Health Science*, *6*(2), 162–178. <https://doi.org/10.1016/j.jshs.2016.02.002>
- Gardner, W. N. (1996). The pathophysiology of hyperventilation disorders. *Chest*, *109*(2), 516–534. <https://doi.org/10.1378/chest.109.2.516>
- Gronwall, D. M. A. (1977). Paced Auditory Serial-Addition Task: A measure of recovery from concussion. *Perceptual and Motor Skills*, *44*(2), 367–373. <https://doi.org/10.2466/pms.1977.44.2.367>
- Grossman, P. (1983). Respiration, stress, and cardiovascular function. *Psychophysiology*, *20*(3), 284–300. <https://doi.org/10.1111/j.1469-8986.1983.tb02156.x>
- Hackett, R. A., & Steptoe, A. (2017). Type 2 diabetes mellitus and psychological stress—a modifiable risk factor. *Nature Reviews Endocrinology*, *13*(9), 547–560. <https://doi.org/10.1038/nrendo.2017.64>
- Hamer, M., Stamatakis, E., Chastin, S., Pearson, N., Brown, M., Gilbert, E., & Sullivan, A. (2020). Feasibility of measuring sedentary time using data from a thigh-worn accelerometer. *American Journal of Epidemiology*, *189*(9), 963–971. <https://doi.org/10.1093/aje/kwaa047>
- Hildebrand, M., Hansen, B. H., van Hees, V. T., & Ekelund, U. (2017). Evaluation of raw acceleration sedentary thresholds in children and adults. *Scandinavian Journal of Medicine and Science in Sports*, *27*(12), 1814–1823. <https://doi.org/10.1111/sms.12795>
- Hildebrand, M., Van Hees, V. T., Hansen, B. H., & Ekelund, U. (2014). Age group comparability of raw accelerometer output from wrist- and hip-worn monitors. *Medicine and Science in Sports and Exercise*, *46*(9), 1816–1824. <https://doi.org/10.1249/MSS.0000000000000289>
- Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of*

- Statistics*, 6(2), 65–70. <https://doi.org/10.2307/4615733>
- Kiecolt-Glaser, J. K., Renna, M. E., Shrout, M. R., & Madison, A. A. (2020). Stress reactivity: what pushes us higher, faster, and longer—and why it matters. *Current Directions in Psychological Science*, 29(5), 492–498. <https://doi.org/10.1177/0963721420949521>
- Kim, Y., Barry, V. W., & Kang, M. (2015). Validation of the ActiGraph GT3X and activPAL accelerometers for the assessment of sedentary behavior. *Measurement in Physical Education and Exercise Science*, 19(3), 125–137. <https://doi.org/10.1080/1091367X.2015.1054390>
- Kirschbaum, C., Strasburger, C. J., & Langkrär, J. (1993). Attenuated cortisol response to psychological stress but not to CRH or ergometry in young habitual smokers. *Pharmacology, Biochemistry and Behavior*, 44(3), 527–531. [https://doi.org/10.1016/0091-3057\(93\)90162-M](https://doi.org/10.1016/0091-3057(93)90162-M)
- Lambiase, M. J., Dorn, J., & Roemmich, J. N. (2012). Metabolic and cardiovascular adjustments during psychological stress and carotid artery intima-media thickness in youth. *Physiology and Behavior*, 105(5), 1140–1147. <https://doi.org/10.1016/j.physbeh.2011.12.012>
- Ley, R., & Yelich, G. (1998). Fractional end-tidal CO₂ as an index of the effects of stress on math performance and verbal memory of test-anxious adolescents. *Biological Psychology*, 49(2), 84–94. [https://doi.org/10.1016/S0301-0511\(98\)00028-3](https://doi.org/10.1016/S0301-0511(98)00028-3)
- Mador, M. J., & Tobin, M. J. (1991). Effect of alterations in mental activity on the breathing pattern in healthy subjects. *American Review of Respiratory Disease*, 144(3), 481–487. https://doi.org/10.1164/ajrccm/144.3_pt_1.481
- McSwain, S. D., Hamel, D. S., Smith, B. P., Gentile, M. A., Srinivasan, S., Meliones, J. N., & Cheifetz, I. M. (2010). End-tidal and arterial carbon dioxide measurements correlate across all levels of physiologic dead space. *Respiratory Care*, 55(3), 288–293.
- Meuret, A. E., & Ritz, T. (2010). Hyperventilation in panic disorder and asthma: Empirical evidence and clinical strategies. *International Journal of Psychophysiology*, 78(1), 68–79. <https://doi.org/10.1016/j.ijpsycho.2010.05.006>
- Meyer, T., Georg, T., Becker, C., & Kindermann, W. (2001). Reliability of gas exchange measurements from two different spiroergometry systems. *International Journal of Sports Medicine*, 22(8), 593–597. <https://doi.org/10.1055/s-2001-18523>
- Migueles, J. H., Cadenas-Sanchez, C., Rowlands, A. V., Henriksson, P., Shiroma, E. J., Acosta, F. M., Rodriguez-Ayllon, M., Esteban-Cornejo, I., Plaza-Florido, A., Gil-Cosano, J. J., Ekelund, U., van Hees, V. T., & Ortega, F. B. (2019). Comparability of accelerometer signal aggregation metrics across placements and dominant wrist cut points for the assessment of physical activity in adults. *Scientific Reports*, 9(1), 1–12. <https://doi.org/10.1038/s41598-019-54267-y>
- Mikkelsen, M. L. K., Berg-Beckhoff, G., Frederiksen, P., Horgan, G., O’Driscoll, R., Palmeira, A. L., Scott, S. E., Stubbs, J., Heitmann, B. L., & Larsen, S. C. (2020). Estimating physical activity and sedentary behaviour in a free-living environment: A comparative study between fitbit charge 2 and Actigraph GT3X. *PLoS ONE*, 15(6), e0234426. <https://doi.org/10.1371/journal.pone.0234426>
- Mücke, M., Ludyga, S., Colledge, F., & Gerber, M. (2018). Influence of regular physical activity and fitness on stress reactivity as measured with the trier social stress test protocol: A systematic review. *Sports Medicine*, 48(11), 2607–2622. <https://doi.org/10.1007/s40279-018-0979-0>
- Myers, J., Kokkinos, P., & Nyelin, E. (2019). Physical activity, cardiorespiratory fitness, and the metabolic syndrome. *Nutrients*, 11(7), 1–18. <https://doi.org/10.3390/nu11071652>
- Nayor, M., Chernofsky, A., Spartano, N. L., Tanguay, M., Blodgett, J. B., Murthy, V. L., Malhotra, R., Houstis, N. E., Velagaleti, R. S., Murabito, J. M., Larson, M. G., Vasani, R. S., Shah, R. V., & Lewis, G. D. (2021). Physical activity and fitness in the community: the Framingham Heart

- Study. *European Heart Journal*, Online ahead of print.
<https://doi.org/10.1093/eurheartj/ehab580>
- Obrist, P. A. (1976). Cardiovascular-behavioral interaction – as it appears today. *Psychophysiology*, *13*(2), 95–107. <https://doi.org/10.1111/j.1469-8986.1976.tb00081.x>
- Paine, N. J., Ring, C., Aldred, S., Bosch, J. A., Wadley, A. J., & van Zanten, J. J. C. S. V. (2013). Eccentric-exercise induced inflammation attenuates the vascular responses to mental stress. *Brain Behavior and Immunity*, *30*, 133–142. <https://doi.org/10.1016/j.bbi.2013.01.082>
- Paine, N. J., Ring, C., Bosch, J. A., Drayson, M. T., Aldred, S., & Veldhuijzen van Zanten, J. J. C. S. (2014). Vaccine-induced inflammation attenuates the vascular responses to mental stress. *International Journal of Psychophysiology*, *90*(3), 340–348. <https://doi.org/10.1016/j.ijpsycho.2014.06.016>
- Pané-Farré, C. A., Alius, M. G., Modeß, C., Methling, K., Blumenthal, T., & Hamm, A. O. (2015). Anxiety sensitivity and expectation of arousal differentially affect the respiratory response to caffeine. *Psychopharmacology*, *232*(11), 1931–1939. <https://doi.org/10.1007/s00213-014-3828-3>
- Pendergast, D. R., Leddy, J. J., & Venkatraman, J. T. (2000). A perspective on fat intake in athletes. *Journal of the American College of Nutrition*, *19*(3), 345–350. <https://doi.org/10.1080/07315724.2000.10718930>
- Plourde, A., Lavoie, K. L., Raddatz, C., & Bacon, S. L. (2017). Effects of acute psychological stress induced in laboratory on physiological responses in asthma populations: A systematic review. *Respiratory Medicine*, *127*, 21–32. <https://doi.org/10.1016/j.rmed.2017.03.024>
- Ramakrishnan, R., Doherty, A., Smith-byrne, K., Rahimi, K., Bennett, D., Woodward, M., Walmsley, R., & Dwyer, T. (2021). Accelerometer measured physical activity and the incidence of cardiovascular disease: Evidence from the UK Biobank cohort study. *PLoS Medicine*, *18*(9), e1003809. <https://doi.org/10.1371/journal.pmed.1003487>
- Ramos-Jiménez, A., Hernández-Torres, R. P., Torres-Durán, P. V., Romero-Gonzalez, J., Mascher, D., Posadas-Romero, C., & Juárez-Oropeza, M. A. (2008). The respiratory exchange ratio is associated with fitness indicators both in trained and untrained men: A possible application for people with reduced exercise tolerance. *Clinical Medicine. Circulatory, Respiratory and Pulmonary Medicine*, *2*(1), 1–9. <https://doi.org/10.4137/ccrpm.s449>
- Regensteiner, J. G., Sippel, J., McFarling, E. T., Wolfel, E. E., & Hiatt, W. R. (1995). Effects of non-insulin-dependent diabetes on oxygen consumption during treadmill exercise. *Medicine & Science in Sports & Exercise*, *27*(6), 875–881.
- Reiss, S., Peterson, R. A., Gursky, D. M., & McNally, R. J. (1986). Anxiety sensitivity, anxiety frequency and the prediction of fearfulness. *Behaviour Research and Therapy*, *24*(1), 1–8. [https://doi.org/10.1016/0005-7967\(86\)90143-9](https://doi.org/10.1016/0005-7967(86)90143-9)
- Ritz, T. (2012). Airway responsiveness to psychological processes in asthma and health. *Frontiers in Physiology*, *3*(3), 1–17. <https://doi.org/10.3389/fphys.2012.00343>
- Ritz, T., & Kullowatz, A. (2005). Effects of emotion and stress on lung function in health and asthma. *Current Respiratory Medicine Reviews*, *1*(2), 209–218. <https://doi.org/10.2174/1573398054022993>
- Ritz, T., Simon, E., & Trueba, A. F. (2011). Stress-induced respiratory pattern changes in asthma. *Psychosomatic Medicine*, *73*(6), 514–521. <https://doi.org/10.1097/PSY.0b013e318222050d>
- Rutherford, J. J., Clutton-Brock, T. H., & Parkes, M. J. (2005). Hypocapnia reduces the T wave of the electrocardiogram in normal human subjects. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology*, *289*(58), 148–155.

<https://doi.org/10.1152/ajpregu.00085.2005>

- Sandberg, S., Paton, J. Y., Ahola, S., McCann, D. C., McGuinness, D., Hillary, C. R., & Oja, H. (2000). The role of acute and chronic stress in asthma attacks in children. *Lancet*, *356*(9234), 982–987. [https://doi.org/10.1016/S0140-6736\(00\)02715-X](https://doi.org/10.1016/S0140-6736(00)02715-X)
- Smorawiński, J., Nazar, K., Kaciuba-Uscilko, H., Kamińska, E., Cybulski, G., Kodrzycka, A., Bicz, B., & Greenleaf, J. E. (2001). Effects of 3-day bed rest on physiological responses to graded exercise in athletes and sedentary men. *Journal of Applied Physiology*, *91*(1), 249–257. <https://doi.org/10.1152/jappl.2001.91.1.249>
- Soares-Miranda, L., Sandercock, G., Vale, S., Silva, P., Moreira, C., Santos, R., & Mota, J. (2011). Benefits of achieving vigorous as well as moderate physical activity recommendations: Evidence from heart rate complexity and cardiac vagal modulation. *Journal of Sports Sciences*, *29*(10), 1011–1018. <https://doi.org/10.1080/02640414.2011.568513>
- Spruit, M. A., Singh, S. J., Garvey, C., Zu Wallack, R., Nici, L., Rochester, C., Hill, K., Holland, A. E., Lareau, S. C., Man, W. D. C., Pitta, F., Sewell, L., Raskin, J., Bourbeau, J., Crouch, R., Franssen, F. M. E., Casaburi, R., Vercoulen, J. H., Vogiatzis, I., ... Wouters, E. F. M. (2013). An official American Thoracic Society/European Respiratory Society statement: key concepts and advances in pulmonary rehabilitation. *American Journal of Respiratory and Critical Care Medicine*, *188*(8), 13–64. <https://doi.org/10.1164/rccm.201309-1634ST>
- Steptoe, A., Hackett, R. A., Lazzarino, A. I., Bostock, S., La Marca, R., Carvalho, L. A., & Hamer, M. (2014). Disruption of multisystem responses to stress in type 2 diabetes: Investigating the dynamics of allostatic load. *Proceedings of the National Academy of Sciences*, *111*(44), 15693–15698. <https://doi.org/10.1073/pnas.1410401111>
- Steptoe, A., Kivimäki, M., Lowe, G., Rumley, A., & Hamer, M. (2016). Blood pressure and fibrinogen responses to mental stress as predictors of incident hypertension over an 8-year period. *Annals of Behavioral Medicine*, *50*(6), 898–906. <https://doi.org/10.1007/s12160-016-9817-5>
- Suess, W. M., Alexander, A. B., Smith, D. D., Sweeney, H. W., & Marion, R. J. (1980). The effects of psychological stress on respiration: A preliminary study of anxiety and hyperventilation. *Psychophysiology*, *17*(6), 535–540. <https://doi.org/10.1111/j.1469-8986.1980.tb02293.x>
- Tebar, W. R., Ritti-Dias, R. M., Mota, J., Farah, B. Q., Saraiva, B. T. C., Damato, T. M. M., Delfino, L. D., Aguilar, B. A. S., dos Santos, A. B., Silva, S. C. B., Vanderlei, L. C. M., & Christofaro, D. G. D. (2020). Relationship between domains of physical activity and cardiac autonomic modulation in adults: a cross-sectional study. *Scientific Reports*, *10*(1), 1–7. <https://doi.org/10.1038/s41598-020-72663-7>
- Thompson, D., Batterham, A. M., Peacock, O. J., Western, M. J., & Booso, R. (2016). Feedback from physical activity monitors is not compatible with current recommendations: A recalibration study. *Preventive Medicine*, *91*, 389–394. <https://doi.org/10.1016/j.ypmed.2016.06.017>
- Tremblay, M. S., Aubert, S., Barnes, J. D., Saunders, T. J., Carson, V., Latimer-Cheung, A. E., Chastin, S. F. M., Altenburg, T. M., Chinapaw, M. J. M., Aminian, S., Arundell, L., Hinkley, T., Hnatiuk, J., Atkin, A. J., Belanger, K., Chaput, J. P., Gunnell, K., Larouche, R., Manyanga, T., ... Wondergem, R. (2017). Sedentary Behavior Research Network (SBRN) - terminology consensus project process and outcome. *International Journal of Behavioral Nutrition and Physical Activity*, *14*(1), 1–17. <https://doi.org/10.1186/s12966-017-0525-8>
- Turner, A. I., Smyth, N., Hall, S. J., Torres, S. J., Hussein, M., Jayasinghe, S. U., Ball, K., & Clow, A. J. (2020). Psychological stress reactivity and future health and disease outcomes: A systematic review of prospective evidence. *Psychoneuroendocrinology*, *114*, 104599. <https://doi.org/10.1016/j.psyneuen.2020.104599>

- Tyagi, A., Cohen, M., Reece, J., & Telles, S. (2014). An explorative study of metabolic responses to mental stress and yoga practices in yoga practitioners, non-yoga practitioners and individuals with metabolic syndrome. *BMC Complementary And Alternative Medicine*, *14*, 1–10. <https://doi.org/10.1186/1472-6882-14-445>
- van der Ploeg, H. P., & Hillsdon, M. (2017). Is sedentary behaviour just physical inactivity by another name? *International Journal of Behavioral Nutrition and Physical Activity*, *14*(1), 142–146. <https://doi.org/10.1186/s12966-017-0601-0>
- Veldhuijzen Van Zanten, J. J. C. S., Ring, C., Burns, V. E., Edwards, K. M., Drayson, M., & Carroll, D. (2004). Mental stress-induced hemoconcentration: Sex differences and mechanisms. *Psychophysiology*, *41*(4), 541–551. <https://doi.org/10.1111/j.1469-8986.2004.00190.x>
- Vos, T., Lim, S. S., Abbafati, C., Abbas, K. M., Abbasi, M., Abbasifard, M., Abbasi-Kangevari, M., Abbastabar, H., Abd-Allah, F., Abdelalim, A., Abdollahi, M., Abdollahpour, I., Abolhassani, H., Aboyans, V., Abrams, E. M., Abreu, L. G., Abrego, M. R. M., Abu-Raddad, L. J., Abushouk, A. I., ... Murray, C. J. L. (2020). Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *The Lancet*, *396*(10258), 1204–1222. [https://doi.org/10.1016/S0140-6736\(20\)30925-9](https://doi.org/10.1016/S0140-6736(20)30925-9)
- Vujanovic, A. A., Arrindell, W. A., Bernstein, A., Norton, P. J., & Zvolensky, M. J. (2007). Sixteen-item anxiety sensitivity index: Confirmatory factor analytic evidence, internal consistency, and construct validity in a young adult sample from the Netherlands. *Assessment*, *14*(2), 129–143. <https://doi.org/10.1177/1073191106295053>
- Wientjes, C. J. E., Grossman, P., & Gaillard, A. W. K. (1998). Influence of drive and timing mechanisms on breathing pattern and ventilation during mental task performance. *Biological Psychology*, *49*(2), 53–70. [https://doi.org/10.1016/S0301-0511\(98\)00026-X](https://doi.org/10.1016/S0301-0511(98)00026-X)
- Wilhelm, F. H., Rattel, J. A., Wegerer, M., Liedlgruber, M., Schweighofer, S., Kreibitz, S. D., Kolodyazhniy, V., & Blechert, J. (2017). Attend or defend? Sex differences in behavioral, autonomic, and respiratory response patterns to emotion-eliciting films. *Biological Psychology*, *130*, 30–40. <https://doi.org/10.1016/j.biopsycho.2017.10.006>
- Winkler, E. A. H., Bodicoat, D. H., Healy, G. N., Bakrania, K., Yates, T., Owen, N., Dunstan, D. W., & Edwardson, C. L. (2016). Identifying adults' valid waking wear time by automated estimation in activPAL data collected with a 24 h wear protocol. *Physiological Measurement*, *37*(10), 1653–1668. <https://doi.org/10.1088/0967-3334/37/10/1653>
- Zaffalon Júnior, J. R., Viana, A. O., de Melo, G. E. L., & De Angelis, K. (2018). The impact of sedentarism on heart rate variability (HRV) at rest and in response to mental stress in young women. *Physiological Reports*, *6*(18), 1–8. <https://doi.org/10.14814/phy2.13873>

HIGHLIGHTS

- The link between activity behaviours and respiratory stress reactivity is unknown.
- Sedentary behaviour was related to some measures of respiratory stress reactivity.
- Physical activity was not linked with respiratory stress reactivity.
- The implications of dysregulated respiratory stress reactivity need further study.