Sedentary Behaviour, Physical Activity and Psychobiological Stress Reactivity: A Systematic Review

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ABSTRACT

Background: Sedentary behaviour, physical activity, and psychobiological reactivity to acute psychological stress are independent risk factors for cardiovascular disease. Sedentary behaviour and physical activity influence autonomic, haemodynamic, and inflammatory pathways under resting conditions, and these pathways become activated under acute psychological stress. However, it is unclear whether sedentary behaviour and physical activity relate to psychobiological responses to stress. Thus, the aim of this study is to systematically review sedentary behaviour and physical activity in the context of psychobiological reactivity to acute psychological stress.

Methods: Sedentary behaviour, physical activity and psychobiological stress reactivity search terms were combined, and several databases were searched in duplicate. Eligibility criteria included: (1) a validated measure of sedentary behaviour/physical activity; (2) cardiovascular, inflammatory, neuroendocrine, or respiratory markers measured at rest and in response to laboratory-induced acute psychological stress.

Results: 6084 articles were screened, with 11 included in a narrative synthesis. No studies measured postural components of sedentary behaviour, but 2/4 studies found that markers of sedentary behaviour (e.g., physical inactivity) were associated with elevated heart rate, dysregulated heart rate variability, or lowered cortisol responses to stress. Higher volumes of physical activity were linked to lower HR, cortisol, or immune responses to stress in 4/7 studies.

Conclusions: Extensive methodological variability precludes conclusions from being drawn. This review should be used to guide a more homogeneous and gold-standard literature, which accounts for postural components of sedentary behaviour using inclinometry, and the whole physical activity intensity spectrum using universal and reproducible approaches.

Key words: Sedentary behaviour, physical activity, stress reactivity, acute psychological stress, cardiovascular disease, systematic review
INTRODUCTION

Cardiovascular disease, sedentary behaviour, and physical activity

Cardiovascular disease (CVD) is the leading cause of global mortality and morbidity, which is reflected by a disability-adjusted life-year count of 393 million in 2019 (Vos et al., 2020). Sedentary behaviour is an emerging risk factor for CVD, and is defined as “any waking behaviour characterized by an energy expenditure ≤1.5 metabolic equivalents (METs), while in a sitting, reclining or lying posture” (Tremblay et al., 2017, p. 9). Conversely, physical activity is defined uniquely as “any bodily movement produced by skeletal muscles that results in energy expenditure” (Caspersen, Powell and Christenson, 1985, p. 126). Importantly, the physical activity intensity spectrum includes light intensity physical activity (< 3 METs), moderate intensity physical activity (3-6 METs) and vigorous intensity physical activity (> 6 METs) (Pate et al., 1995). Given their respective definitions, and that individuals can be both highly physically active (i.e., by performing daily physical activity at a level that exceeds daily physical activity guidelines) and highly sedentary (e.g., by spending the rest of the day sitting; Hamer et al., 2020), physical activity and sedentary behaviour should be considered separate, independent behaviours. However, in recent years many studies have relied on lower physical activity volumes, physical inactivity (i.e., not meeting physical activity guidelines), or a lack of movement detected via accelerometry, to index sedentary behaviour (Prince et al., 2020).

Although these markers can sometimes provide a robust estimation of sedentary behaviour (Tremblay et al., 2017; Prince et al., 2020), they fail to account for postural sedentary behaviour components, which might be important in the context of CVD risk (Dempsey et al., 2018; Edwardson et al., 2020).

Cardiovascular disease risk factors associated with sedentary behaviour and physical activity

British adults spend approximately 9.3 hours/day engaging in sedentary behaviour (Hamer et al., 2020), and physical inactivity is rife at 36% (Guthold et al., 2018). This is concerning, as sedentary behaviour (Patterson et al., 2018; Ekelund et al., 2019) and physical (in)activity (Ramakrishnan et al., 2021) are independently implicated in CVD aetiology. Sedentary behaviour is also related to elevations in CVD risk markers, including blood pressure (BP) (Lee and Wong, 2015), inflammation (Parsons et al., 2017), autonomic dysfunction (dos Santos et al., 2019), cortisol (Gubelmann et al.,...
2018) and metabolic dysregulation (Hadgraft et al., 2021). Conversely, physical activity is inversely (i.e., beneficially) associated with the abovementioned risk factors (Mora et al., 2007; Batty et al., 2020).

**Psychobiological reactivity to acute psychological stress and links to sedentary behaviour and physical activity**

Acute psychological stress perturbs the cardiovascular (Chida and Steptoe, 2010), inflammatory/immune (Marsland et al., 2017), cortisol (Dickerson and Kemeny, 2004) and respiratory (Plourde et al., 2017) systems. Interestingly, the risk factors above that are characteristic of sedentary behaviour and physical (in)activity play a role in determining psychobiological changes (i.e., reactivity and recovery) to acute psychological stress (e.g., Veldhuijzen Van Zanten et al., 2005; Balanos et al., 2010; Kidd, Carvalho and Steptoe, 2014; Steptoe et al., 2014). For example, sedentary behaviour increases resting levels of BP, with one pathway being heightened sympathetic tone (Dempsey et al., 2018, 2020), and higher resting BP is associated with exaggerated cardiovascular (e.g., blood pressure, heart rate, cardiac output) (Sheffield et al., 1997; Balanos et al., 2010), inflammatory (Steptoe et al., 2016) and respiratory (Sims et al., 1988) responses to acute psychological stress. Importantly, exaggerated psychobiological responses to stress, and impaired recovery post-stress, are prospectively associated with CVD risk factors (Chida and Steptoe, 2010; Turner et al., 2020), and can trigger major adverse cardiovascular events acutely (Paine, Bosch and Veldhuijzen van Zanten, 2012). Therefore, if sedentary behaviour and low physical activity are associated with exaggerated stress reactivity and poor stress recovery, then this could be an important mechanism linking these behaviours with CVD outcomes.

*The current literature summarising sedentary behaviour and physical activity in the context of psychobiological stress reactivity*

The cross-stressor adaptation hypothesis postulates that exercise and physical activity triggers stress responses that over time contribute to the attenuation of responses to psychological stress (Sothmann et al., 1996). However, findings from randomised controlled trials that have examined the effect of exercise training on stress reactivity measures are inconsistent, with both lower psychobiological
responses to stress (e.g., Klaperski et al., 2014) and null findings (e.g., Sloan et al., 2021) reported in the literature. Systematic reviews have shown that higher cardiorespiratory fitness (which can be used as a marker of habitual physical activity) is related to smaller systolic BP (SBP) stress reactivity and faster heart rate (HR) recovery post-stress (Forcier et al., 2006), as well as larger HR stress reactivity and improved SBP/HR recovery (Jackson and Dishman, 2006). However, although fitness and physical activity are linked there are also major differences, including that physical activity is a behaviour, and there is a large genetic component to fitness that is not found for physical activity (Schutte et al., 2016). A more recent systematic review reported that higher volumes of physical activity were related to attenuated HR (3/5 studies) and cortisol (5/8 studies) responses to stress (Mücke et al., 2018). However, many of the studies included in this review used exercise behaviours as a proxy for total physical activity volume and the whole physical activity intensity spectrum. Importantly, exercise has a unique definition, usually reflects only a small proportion of total (e.g., incidental/lifestyle) physical activity volume, and often represents the higher end of the physical activity intensity spectrum (Caspersen, Powell and Christenson, 1985). This previous review also exclusively examined HR and cortisol changes to the Trier Social Stress Test in healthy populations (Mücke et al., 2018), thereby excluding other psychobiological markers, populations and stress tasks. Our review will take a broader approach and investigate a wider range of psychobiological responses (including other key cardiovascular parameters such as blood pressure, inflammatory markers, and respiratory indices) to all commonly used psychological stress tasks (e.g., mirror tracing, cold pressor, the Stroop Colour and Word Test) in a variety of populations. This is because these responses are important for stress-related CVD risk (Zhao et al., 2012; Sullivan et al., 2020; Turner et al., 2020) and might also be impacted by physical activity (Hamer and Steptoe, 2007). No prior work has reviewed the sedentary behaviour and psychobiological stress reactivity literature.

**Aim and hypotheses of the current systematic review**

The aim of this systematic review is to summarise research exploring sedentary behaviour and physical activity in the context of cardiovascular, inflammatory, neuroendocrine, and respiratory responses to acute psychological stress. We hypothesised that higher volumes of sedentary behaviour
would be associated with exaggerated psychobiological responses to stress, whereas higher volumes
of physical activity would be associated with smaller stress responses.

METHODS

Protocol
This systematic review followed Preferred Reporting Items for Systematic reviews and Meta-analyses
(Moher et al., 2009) (Supplementary File 1) and Meta-Analyses and Systematic Reviews of
Observational Studies (Stroup et al., 2000) guidelines.

Information sources and search strategy
Key words (formulated by the research team and academic librarian) representing sedentary
behaviour and physical activity were combined with terms relating to psychobiological stress
reactivity (Supplementary File 2). Several electronic bibliographic databases (PubMed, Web of
Science, PsycINFO and MEDLINE) were searched in duplicate on the 31st of October 2019, with a
final updated search completed on the 10th of November 2021. This retrieved articles published from
the date inception to the 10th of November 2021 (inclusive). No limits were used and the “all
fields/text” option was applied. Manual searches were undertaken through the reference lists of our
included studies, as well as previous reviews on complementary topics (Forcier et al., 2006; Jackson
and Dishman, 2006; Mücke et al., 2018). All articles were imported into reference management
software (Mendeley desktop version 1.19.4, Elsevier, London, UK) and duplicates were removed.

Eligibility criteria
Inclusion criteria were: (1) studies with an aim or hypothesis relating to sedentary behaviour or
physical activity in the context of psychobiological stress reactivity; (2) peer-reviewed journal articles
written in English language; (3) primary or secondary analysis of quantitative data. We only included
human adult studies (mean sample age between 18 years and 60 years) using a previously cited time-
limited active (i.e., where individuals can alter their performance/outcomes of a task) or passive (i.e.,
where participants endure an aversive stimulus) psychological (i.e., metabolically undemanding)
laboratory stress task. Psychobiological outcomes needed to be collected before and during or after psychological stress, and could include any cardiovascular, inflammatory/immune, neuroendocrine, or respiratory markers. Studies were required to measure sedentary behaviour or physical activity using a validated wearable device or questionnaire, covering at least a three-day period. Given that the current definition of sedentary behaviour has only recently been formulated (Tremblay et al., 2017), any studies that claimed to measure sedentary behaviour were included, regardless of how sedentary behaviour was defined or assessed. In the physical activity domain, studies had to measure total volume of physical activity (e.g., lifestyle and/or incidental physical activity), rather than rely on exercise/sport behaviours which often represent only a small proportion of total physical activity volume. No study design restrictions were imposed, and therefore all observational and intervention studies were included.

**Study selection and data collection process**

Two reviewers independently screened all articles based on title and abstract. Reasons for exclusion were detailed and any discrepancies were resolved by the senior author. The full text screening process was then completed using the same approach. If any full texts could not be found, then corresponding authors were contacted.

**Study quality and risk of bias**

Risk of bias at the study level was assessed independently by two reviewers. First, an adapted Downs and Black checklist (Downs and Black, 1998) was used, but only items relevant to this systematic review were retained, as per others (e.g., Plourde et al., 2017). Total scores could range from 0-15, and tertiles were formed to indicate high (0-5), moderate (6-10) and low (>10) risk of bias (e.g., Silva, Jayawardana and Meyer, 2018). Second, the Risk Of Bias In Non-randomized Studies of Interventions (ROBINS-I; Sterne et al., 2016) tool was employed, which comprised items devised specifically for this review (e.g., Slavish and Szabo, 2019). Again, tertiles were used to categorise high (0-7), moderate (8-14) and low (>14) risk of bias. The two risk of bias tools that were used in this study can be found in Supplementary File 3.
RESULTS

Study selection

A total of 6271 articles with cardiovascular endpoints, 3835 with neuroendocrine endpoints, 2609 with inflammatory/immune endpoints, and 4936 with respiratory endpoints, were retrieved by four separate searches, one for each physiological system (see Figure 1). After combining, and with duplicates removed, this yielded 6084 articles for screening. A total of 11 unique studies were included in this narrative synthesis, four assessing markers of sedentary behaviour in the context of psychobiological stress reactivity, and seven assessing physical activity in the context of psychobiological stress reactivity.

Methodological characteristics

As shown in Table 1 and Table 2, 8/11 studies were observational and cross-sectional. In the sedentary behaviour domain there was one randomised cross-over trial (Endrighi, Steptoe and Hamer, 2016). In the physical activity domain there was one randomised control trial (Hanson et al., 2013) and one experimental study (Taylor and Katomeri, 2006), but the physical activity and stress reactivity data that were derived from these two studies were observational, focusing on person-level physical activity.

Participant characteristics

Sample size ranged from $N=31$ (Buckworth, Dishman and Cureton, 1994) to $N=96$ (Zaffalon Júnior et al., 2018) with mean sample age extending from 20.8 years (Buckworth, Dishman and Cureton, 1994) to 50.0 years (Ferreira-Silva et al., 2018). Seven studies exclusively tested healthy participants (See Tables 1 and 2), whereas others assessed healthy individuals with parental history of hypertension (Buckworth, Dishman and Cureton, 1994), temporarily abstinent smokers (Taylor and Katomeri, 2006), individuals with moderately elevated psychological distress (Poole et al., 2011) and patients with obstructive sleep apnoea (Ferreira-Silva et al., 2018). Body mass index (BMI) ranged from 22.1 kg/m$^2$ (Gerber et al., 2017) to 29.0 kg/m$^2$ (Ferreira-Silva et al., 2018) and two studies tested participants with an overweight BMI (Hong et al., 2004; Ferreira-Silva et al., 2018). The remaining participant characteristics are summarised in Tables 1 and 2.
Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram.

Total Number of Articles (n=17,112)

Duplicates Removed (n=11,028)

Excluded at Title & Abstract Screening Stage (n=5895)

Excluded at Full Text Stage (n=163). Reasons Included:
- Did not measure sedentary behaviour or physical activity (n=122);
- Did not examine sedentary behaviour or physical activity in the context of psychobiological stress reactivity (n=33);
- Not a peer reviewed journal article (n=9);
- Non-validated/cited sedentary behaviour or physical activity tool (n=4);
- Full text not in English language (n=3);
- Inappropriate stress task (e.g., metabolically demanding) (n=2);
- Not primary research (e.g., previous review) (n=2);
- Physiology not measured pre and during or post stress (n=1).

Cardiovascular endpoints (n=6272)

Neuroendocrine endpoints (n=3835)

Inflammatory/immune endpoints (n=2609)

Respiratory endpoints (n=4396)

Title & Abstract Screening (n=6084)

Articles Included (n=11)
Measurement of sedentary behaviour

No research in this review quantified postural and metabolic components of sedentary behaviour in line with the widely accepted definition (Tremblay et al., 2017). Instead, studies used physical inactivity or a lack of movement as indirect markers of sedentary behaviour. These markers of sedentary behaviour are frequently used within the sedentary behaviour literature (Tremblay et al., 2017; Prince et al., 2020) and associations with certain (e.g., body fat), but not all (e.g., diastolic blood pressure), CVD risk factors are comparable when comparing posturally-determined sedentary behaviour and markers of sedentary behaviour (Edwardson et al., 2020). In line with other systematic reviews in the field of sedentary behaviour (e.g., Prince et al., 2020), this present review will hereafter refer to sedentary behaviour estimated from physical inactivity or a lack of movement as a marker of “sedentary behaviour”. However, this a literature-wide limitation that is considered in the discussion.

Specifically, one study indexed sedentary behaviour as a lack of movement (calculated as the difference between daily total wear time and daily total active time) using a hip-mounted ActiGraph GT1M (ActiGraph, Pensacola, Florida, USA) across seven days (Endrighi, Steptoe and Hamer, 2016). The remaining studies used the International Physical Activity Questionnaire (IPAQ). Ferreira-Silva et al. (2018) defined their “sedentary” group as doing < 30 min of physical activity per week, and their active group as doing >150 min of physical activity per week. Zaffalon Júnior et al. (2018) classified their “sedentary” group as sedentary or irregularly active, and their active group as active or very active (but provided no quantification). Dziembowska et al. (2019) operationalised their “sedentary” group as individuals who are sedentary and do not regularly train, and their active group as elite volleyball players who are active and train >four times per week.

Measurement of physical activity

Two studies measured physical activity via wearable devices (hip worn ActiGraph GT1M (ActiGraph, Pensacola, Florida, USA) during waking hours for 7 days; Poole et al., 2011; Gerber et al., 2017), but Poole et al. (2011) also administered the short-form version of the IPAQ. The remaining five studies exclusively used self-report: the full IPAQ (Hanson et al., 2013), Seven-Day Physical Activity Recall Interview (Buckworth, Dishman and Cureton, 1994; Taylor and Katomeri, 2006), Godin-Shephard
Leisure-Time Physical Activity Questionnaire (Hong et al., 2004), and the short version of the Freiburg Questionnaire of Physical Activity (Hermann et al., 2019).

*Inducement of acute psychological stress*

The studies in this review adopted psychological stress tasks that have been well used in the literature and reported in previous systematic reviews (e.g., Turner et al., 2020). Two studies employed the Stroop Colour and Word Test, using a 3-min version (Ferreira-Silva et al., 2018), or one of unspecified duration (Zaffalon Júnior et al., 2018). In addition, the Trier Social Stress Test (TSST) of 10 minutes (Gerber et al., 2017; Hermann et al., 2019), and 5 minutes (the mental arithmetic component only; Hanson et al., 2013) was used. A 10-min dual task paradigm (5-min mirror tracing followed immediately by 5-min public speaking; Poole et al., 2011; Endrighi, Steptoe and Hamer, 2016), 5-min mental arithmetic task (Dziembowska et al., 2019) and 6-min speech task (Hong et al., 2004) were also used. One study used a paradigm involving one active (5-min mental arithmetic) and one passive (2-min forehead cold pressor; Buckworth, Dishman and Cureton, 1994) task, and another study used a combination of two active tasks (Stroop [3-min] and speech [2-min preparation, 2-min speech]) and one passive task (temporarily handling a lit cigarette without smoking [duration not reported]) (Taylor and Katomeri, 2006). All studies excluding three (Ferreira-Silva et al., 2018; Zaffalon Júnior et al., 2018; Dziembowska et al., 2019) induced elements of social evaluation.

*Psychobiological measures taken under stress*

As detailed in Tables 1 and 2, ten studies measured cardiovascular responses to stress: nine examined BP and/or HR responses, two measured heart rate variability (HRV) responses and one measured forearm blood flow and forearm vascular conductance responses. Six studies assessed neuroendocrine markers: five measured salivary cortisol and one measured plasma adrenaline and noradrenaline. One study measured inflammatory cytokines (interleukin-6), and one study explored immune cell responses (lymphocyte subset and L-selectin [CD62L+] cells). No studies (sedentary behaviour or physical activity) have assessed respiratory changes to acute psychological stress.
Markers of sedentary behaviour and psychobiological responses to acute psychological stress

Cross-over intervention study

As shown in Table 1, one study found that a free-living intervention significantly increased an index of sedentary behaviour (mean [SE] increase of 31.49 [12.13] min/day for sedentary behaviour, defined as non-movement), but this did not impact SBP, DBP, IL-6 or cortisol responses to stress, relative to a “normal lifestyle” condition (Endrighi, Steptoe and Hamer, 2016). There was a significant interaction for HR, but post-hoc analyses revealed no significant differences in stress reactivity or stress recovery across conditions.

Observational studies

Zaffalon Júnior et al. (2018) demonstrated that relative to their active group, their sedentary group (indexed by lower PA) had higher HR and low frequency (LF)/high frequency (HF) ratio, lower time domain HRV, and lower absolute HF HRV, during recovery from stress (indicative of an “unhealthy” sympathetic-dominant response). However, two other studies found no differences across sedentary and active groups (indexed by high and low PA, respectively) in BP and HR (Ferreira-Silva et al., 2018) or HR (Dziembowska et al., 2019) during stress. Nevertheless, the latter study demonstrated a time-by-group interaction effect for cortisol, which was lower 45-min post-stress in their sedentary group versus their active group (Dziembowska et al., 2019). Ferreira-Silva et al. (2018) found that forearm blood flow and forearm vascular conductance under stress were lower in their sedentary (low PA) group, compared to their active group (Table 1).

Physical activity and psychobiological responses to acute psychological stress

Although two studies were acute interventions, the physical activity and stress reactivity data were derived from cross-sectional, observational analyses. As shown in Table 2, one of these studies demonstrated lower HR under stress in those with high vs low vigorous physical activity, but no effect on HRV (HF-HRV and RMSSD) was found. This suggests no association with stress-related autonomic pathways (Hanson et al., 2013). The other study found no correlation between moderate-to-vigorous physical activity and HR and/or BP responses to stress (Taylor and Katomeri, 2006).
In a highly active group compared to a moderately active group, Buckworth et al. (1994) demonstrated lower HR during their mental arithmetic task and recovery period, as well as lower HR during the final minute of recovery from the cold-pressor task. Another study found similar HRV (RMSSD, HF HRV, LF HRV, LF/HF ratio) and cortisol concentrations during stress across their PA groups (Hermann et al., 2019), whereas Poole et al. (2011) reported no correlation between light or moderate-to-vigorous physical activity (either when ActiGraph or IPAQ measured) and cortisol, BP, or HR responses to stress. One study did not demonstrate any significant findings for HR, but a time-by-group interaction emerged for cortisol, with larger cortisol concentrations during stress in those who did not meet the American College of Sports Medicine guidelines (vigorous physical activity >20 min, ≥3 times per week) versus those who did meet the guidelines (Gerber et al., 2017). Finally, Hong et al. (2004) did not find any differences in catecholamine concentrations during stress across their PA groups, but during stress the high PA group (compared to low PA group) demonstrated smaller perturbations in the number of naïve T<sub>SC</sub> cells, memory T<sub>SC</sub> cells, CD62L<sup>+</sup> T<sub>SC</sub> cells, memory T<sub>H</sub> cells, CD62L<sup>-</sup> natural killer cells and lymphocyte CD8<sup>+</sup>CD62L<sup>+</sup> cells. Importantly, this suggests a more resilient response to non-pathogenic stimuli whilst preserving function for antigenic challenge.

**Risk of bias and quality assessment**

As shown in Table 3, Downs and Black scores indicated that one study had a low risk of bias (Endrighi, Steptoe and Hamer, 2016), with the remaining articles showing moderate risk of bias. In general, reporting, and internal validity were rated highly (i.e., lower bias), whereas scores for external validity and statistical power were low. Based on our ROBINS-I assessment tool, five studies had low risk of bias and six had moderate risk of bias (see Table 3)
Table 1. Markers of sedentary behaviour and psychobiological responses to acute psychological stress.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study design</th>
<th>Sample characteristics</th>
<th>Index of sedentary behaviour</th>
<th>Stress paradigm</th>
<th>Psychobiological measures</th>
<th>Main findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endrighi et al. (2016)</td>
<td>Randomised crossover trial with two conditions:</td>
<td></td>
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<tr>
<td></td>
<td>1) 2-week sedentary condition (physical activity replaced with sedentariness).</td>
<td>N=43 healthy participants (44% female).</td>
<td>Hip-mounted ActiGraph GT1M worn for 7 days, with the following cut-points: sedentary = &lt;190, LPA = 191–573, MPA = 574–2099, VPA = &gt;2099 (cpm). Average daily wear time across the week = 12h.</td>
<td>Baseline: duration not reported. Stress: mirror tracing (5 min) and public speaking (5 min) with social evaluation. Recovery: 45 min.</td>
<td>Cardiovascular: Continuous HR (Actiheart) and BP (Finometer). Measurements taken for last 5 min of baseline and recovery, and during whole of stress paradigm (mean of speech and mirror tracing)</td>
<td>Cardiovascular: Effect of time: Increases in SBP, $F(2,82)=64.04$, $p&lt;.001$, DBP, $F(2,82)=77.96$, $p&lt;.001$ and HR, $F(1.58, 64.80)=48.72$, $p&lt;.001$. Interaction effect: mixed-model ANOVA revealed no time by condition interaction for SBP, $F(2,82)=2.45$, $p=.09$ or DBP, $F(2,82)=1.53$, $p=.22$. The time-by-condition interaction for HR was significant, $F(2,82)=4.53$, $p=.01$, but post-hoc t-tests revealed no significant differences across conditions in reactivity (stress minus baseline) or recovery (recovery minus baseline). Inflammatory: Plasma IL-6 assayed in duplicate. Measurements taken for last 5 min of baseline and recovery, and during whole of stress paradigm (mean of speech and mirror tracing)</td>
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</tbody>
</table>
Time of testing: either 10:00 or 14:00, but same for each participant across both conditions.

Interaction effect: mixed-model ANOVA revealed no time-by-condition interaction for cortisol, $F(3,123)=1.60, p=.19$.

Area under the curve (AUC) for cortisol was not significantly different across conditions, $t(42)=1.50, p=.14$.

### Observational studies

<table>
<thead>
<tr>
<th>Dziembowska et al., (2019).</th>
<th>Cross-sectional observational study comparing HR and cortisol during stress in sedentary vs active participants.</th>
<th>$N=55$ healthy participants (100% female).</th>
<th>IPAQ Created 2 groups: Sedentary group ($n=30$): Individuals who were sedentary and did not participate in sport or regularly train. Mean PA = 420 MET/min/week. Mean age ($SD$)=22.5 (1.1) years.</th>
<th>Baseline: 5 min. Stress: mental arithmetic without social evaluation (5 min). Recovery: 35 min.</th>
<th>Cardiovascular: Continuous HR (emWavePro®). Measurements taken throughout whole of baseline and stress, and during first 4 min of recovery.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sessions began between 9-11am.</td>
<td>Mean BMI ($SD$)=23.8 (1.3) kg/m².</td>
<td>Active group ($n=25$): Elite volleyball players who are active, with &gt;five years of training experience, and who train &gt;four times/week. Mean PA = 2700 MET/min/week.</td>
<td></td>
<td></td>
<td>Neuroendocrine: Salivary cortisol assayed in duplicate. Samples collected: start of baseline and every 15 min thereafter.</td>
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</tbody>
</table>

**Cardiovascular:**
- Effect of time: HR increased $F(2,120)=21.34$, $p<.001$, $\eta^2=0.26$.
- Effect of group: repeated measure ANCOVA revealed no group differences in HR during the stress protocol, $F(1,60)=2.69, p=.106$, $\eta^2=0.04$.

**Neuroendocrine:**
- Effect of time: cortisol increased, $F(3,180)=11.73, p<0.001$, $\eta^2=0.16$.
- Effect of group: repeated measure ANCOVA revealed lower cortisol in sedentary group, vs active group, during stress protocol, $F(1,60)=4.69, p=0.034$, $\eta^2=0.07$.
- Interaction effect: Cortisol only significantly increased in the active group. At 45-min post stress, the sedentary group had lower cortisol concentration, relative to the active group, $F(3,180)=3.07, p=.029$, $\eta^2=0.05$. 

Dziembowska et al., (2019). Cross-sectional observational study comparing HR and cortisol during stress in sedentary vs active participants. $N=55$ healthy participants (100% female). Mean age ($SD$)=22.5 (1.1) years. Mean BMI ($SD$)=23.8 (1.3) kg/m². Active group ($n=25$): Elite volleyball players who are active, with >five years of training experience, and who train >four times/week. Mean PA = 2700 MET/min/week. Baseline: 5 min. Stress: mental arithmetic without social evaluation (5 min). Recovery: 35 min. Cardiovascular: Continuous HR (emWavePro®). Measurements taken throughout whole of baseline and stress, and during first 4 min of recovery. Neuroendocrine: Salivary cortisol assayed in duplicate. Samples collected: start of baseline and every 15 min thereafter.
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Participants</th>
<th>Methods</th>
<th>Findings</th>
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<tbody>
<tr>
<td>Ferreira-Silva et al., (2018).</td>
<td>Cross sectional observational study comparing HR and HRV during stress in sedentary vs physically active individuals.</td>
<td>N=40 obstructive sleep apnoea patients who were otherwise healthy (40% female). Mean (SE) age=50.0 (1.0) years. Mean (SE) BMI=29.0 (0.8) kg/m².</td>
<td>IPAQ Created 2 groups: Sedentary group (n=21), PA &lt;30 min/week. PA group (n=19), PA ≥150 min/week, including MVPA ≥ 3 days/week and ≥ 30 min/bout. Cardiovascular: Intermittent BP (cuff on the ankle) and continuous HR (ECG). BP measurements taken minutely. HR measurements taken continuously throughout baseline and stress.</td>
<td>Baseline: 4 min. Stress: Stroop (3 min) with no social evaluation. No recovery period reported. Cardiovascular: Effect of time: significant increase in BP and HR (all p &lt;.05) Effect of group: unpaired Student t-tests revealed no group differences in BP or HR during stress (all p &gt;.05)</td>
</tr>
<tr>
<td>Zaffalon Júnior et al., (2018).</td>
<td>Cross sectional, observational study comparing HR and HRV during stress recovery in sedentary vs physically active women.</td>
<td>N=96 healthy participants (100% female). Mean (SD) age=23.2 (3.8) years. Mean (SD) BMI=22.2 (3.3) kg/m².</td>
<td>IPAQ Created 2 groups: Sedentary group (n=48): sedentary or irregularly active. Active group (n=48): active or very active. The quantification strategy that was used to split the groups was not reported. Cardiovascular: Continuous HR and HRV (Polar watch). Time domain HRV measures: RR, RR SD, RR variance and RMSSD. Frequency domain HRV measures: LF HRV (0.03–0.15 Hz), HF HRV (0.15–0.4 Hz), LF/HF ratio. HR and HRV measured during baseline and</td>
<td>Baseline: 15 min. Stress: Stroop (duration not reported) with no social evaluation. Recovery: 15 min. Cardiovascular: HR Effect of time: HR increased (p&lt;.01). Effect of group: unpaired student t-test revealed that the sedentary group had higher HR during baseline and recovery, relative to the active group (p=.035) Time domain HRV Effect of time: not reported. Effect of group: unpaired student t-test revealed that the sedentary group had lower RR, RR SD, RR variance and RMSSD, relative to the active group (all p&lt;.05). No further data provided. Frequency domain HRV Effect of time: not reported.</td>
</tr>
</tbody>
</table>
recovery and analysed in three segments during baseline (times not specified) and min 2–5 and 6–9 of recovery. Effect of group: unpaired student t-test revealed no group differences in LF HRV ($p>.05$). The sedentary group had lower absolute HF HRV, and a higher HF/LF ratio, compared to the active group (all $p <.05$). This was shown during both time periods (2-5min and 6-9min post stress).

*Note. SD=standard deviation, SE=standard error, BMI=body mass index, IPAQ=International Physical Activity Questionnaire, PA=physical activity, LPA=light intensity physical activity, MPA=moderate intensity physical activity, VPA=vigorous intensity physical activity, MVPA=moderate to vigorous physical activity, J/kg/week=joules per kilogram per week, BP=blood pressure, SBP=systolic blood pressure, DBP=diastolic blood pressure, ECG=electrocardiogram, HR=heart rate, HRV=heart rate variability, HF=high frequency, LF=low frequency, RMSSD=Root Mean Square of the Successive Differences, RR=time between two heartbeats, RR (SD)=standard deviation of R-R interval, i.e., time between two heart beats), IL-6=interleukin 6, AUC=area under the curve, ANOVA = analysis of variance, CPM=counts per minute, p/w = per week.*
Table 2. Physical activity and psychobiological responses to acute psychological stress.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study design</th>
<th>Sample characteristics</th>
<th>Assessment of physical activity</th>
<th>Stress paradigm</th>
<th>Psychobiological measures</th>
<th>Main findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hanson et al., (2013).</td>
<td>Randomized trial investigating the effect of 20g of serotonin reuptake inhibitor (or placebo) and VPA, on HR/HRV during stress (only placebo data presented) AM sessions.</td>
<td>N=44 healthy participants (100% female). Mean (SD) age=23.7 (5.9) years. Mean (SD) BMI=22.6 (3.0) kg/m².</td>
<td>IPAQ Created 2 groups based on national VPA guidelines: High active = &gt;30 min of VPA ≥ 3 days/week (n=22). Low active = &lt; 30 min of VPA &lt; 3 days/week (n=18).</td>
<td>Baseline: 5 min. Stress: Mental arithmetic component of the TSST (5 min) with social evaluation. Recovery: not reported</td>
<td>Cardiovascular: Continuous HR and HRV (Polar watch RS800CX). HRV: HF HRV (0.15–0.40 Hz) and RMSSD. Data collected and averaged during baseline and stress.</td>
<td>Cardiovascular (only placebo data is presented). HR effect of time: HR increased F(1, 38)=210.43, p&lt;.001, η²p=0.85. HR effect of group: HR during stress was lower in highly active (84.62bpm, SD=17.93) vs low active (94.80bpm, SD=10.45) group; t(38) = 2.13, p=.040, d=0.48. HRV effect of time: HF HRV, F(1, 38)=84.60, p&lt;.001, d=1.47, and RMSSD decreased, F(1, 38)=35.99, p&lt;.001, d=0.96. HRV effect of group: non-significant.</td>
</tr>
<tr>
<td>Taylor et al., (2006).</td>
<td>Experimental study comparing HR and BP reactivity after 15 min walk with 15 min resting control. Session time not reported.</td>
<td>N=60 healthy temporary abstinent smokers (57% female). Mean (SD) age = 28.6 (7.6) years. Mean (SD) BMI = 23.0 (3.2) kg/m².</td>
<td>7-day PA recall interview Did not split sample into groups – treated PA data continuously.</td>
<td>Baseline: 10 min. Stress: Stroop (3min), speech (2 min) &amp; holding cigarette (duration not reported). Recovery: not reported.</td>
<td>Cardiovascular: Intermittent BP and HR (arm cuff). Δ in BP &amp; HR was calculated separately for each task by subtracting BP and HR measured immediately post-stress from baseline.</td>
<td>Cardiovascular: Effect of time: SBP (by 4.3 mmHg), DBP (by 2.9 mmHg) and HR (by 2.3 bpm) increased (all p &lt;.05). Correlational analysis: No association between hours of MVPA in the previous 7 days and the Δ in SBP, DBP or HR in response to any of the three stress tasks (all p&gt;0.05).</td>
</tr>
</tbody>
</table>
## Observational studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Type</th>
<th>Description</th>
<th>Participants</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buckworth et al., (1994).</td>
<td>Cross-sectional observational study</td>
<td>Comparing HR and BP during stress in individuals with high vs moderate levels of PA.</td>
<td>N=31 healthy participants with parental history of hypertension (100% female). Mean (SD) age=20.8 (2.0) years. Mean (SD) BMI=22.6 (2.9) kg/m².</td>
<td>7-day PA recall interview. Created 2 groups based on age and sex norms: Highly active (n=16, 1217.7 ± 98.4 J/kg/wk). Moderately active (n=15, 1015.5 ± 49.4 J/kg/wk).</td>
<td>Baseline: 5 min. Stress: mental arithmetic with social evaluation (5 min) - and forehead cold pressor (3°C to 4°C; 2 min. Recovery: 6 min. Cardiovascular: Continuous BP and HR (Finometer). Measurements taken throughout baseline, min 2 to 3 and 4 to 5 of mental arithmetic and min 2 to 3 and 5 to 6 of recovery. No measurements during cold pressor task but taken during min 2 to 3 and 5 to 6 cold pressor recovery. Cardiovascular: Effect of time: not reported. Effect of group: non-significant. Interaction effect: mixed-model ANOVA revealed no time-by group interaction for HR, SBP or DBP, when comparing high vs moderately active groups (all p &gt;.05). HR in the highly active group was lower during mental arithmetic task and recovery (d=0.30), and during last minute of cold pressor recovery (d=0.48) vs moderately active group (all p &lt;.05).</td>
</tr>
<tr>
<td>Gerber et al., (2017).</td>
<td>Cross sectional, observational study</td>
<td>Comparing cardiovascular and cortisol stress responses across four groups: 1) high VPA + high perceived stress (measured by the PSS). 2) low VPA + high perceived stress.</td>
<td>N=42 healthy participants (52% female). Mean (SD) age=21.2 (2.2) years. Mean (SD) BMI = 22.1 (1.8) kg/m².</td>
<td>Hip-mounted ActiGraph GT1M worn for 7 days, with the following Freedson cut-points: MPA (1952–5724 cpm), VPA (≥5724 cpm). Mean (SD) wear=5.95 (0.84) days, wear time=861.42 (72.54) min. 1) PSS &gt;22, VPA&gt;20 min ≥ 3x p/w (n=9).</td>
<td>Baseline: 20 min. Stress: TSST (10 min; 5 min public speaking and 5 min mental arithmetic) with social evaluation Recovery: 90 min. Cardiovascular: Continuous HR (chest HR monitor with wrist device) HR was measured from 1 min pre- to 2 min post-stress. Neuroendocrine: Salivary cortisol assayed in duplicate. Samples collected: 20- and 1-min pre-stress, and 10, 20, 30, 45, 60, 90 min post-stress. Cardiovascular: Effect of time: HR increased, F(1,38)=62.01, p&lt;.001, η²=.620. Effect of group: ANCOVA revealed no between-group differences in HR, in terms of: AUCG, F(3,38)=2.21, p=.102, η²=.149, AUCI, F(3,38)=0.91, p=.445, η²=.067, and peak minus baseline F(3,38)=1.31, p=.286, η²=.094. Interaction effects: ANCOVA revealed no significant time-by-group interaction for HR F(3,38)=2.48, p=.076, η²=.164.</td>
</tr>
</tbody>
</table>

Cross sectional, observational study comparing HRV and cortisol during stress across PA groups.

<table>
<thead>
<tr>
<th>Group Description</th>
<th>PSS and VPA Criteria</th>
<th>Neuroendocrine:</th>
<th>Cardiovascular:</th>
</tr>
</thead>
<tbody>
<tr>
<td>2) high VPA + low perceived stress.</td>
<td>PSS &gt;22, VPA&lt;20 min &lt; 3x p/w (n=12)</td>
<td>Effect of time: significant increase in cortisol, F(3,36)=18.69, p&lt;.001, η²=.601.</td>
<td>Effect of time: RMSDD, F(1.75, 54.29)=15.59, p&lt;.001, η²=.34, and LF HRV, F(2.16, 67.06)=4.05, p=.019, η²=.12, decreased, and LF/HF ratio increased, F(2.04, 63.18)=3.17, p=.048, η²=.09.</td>
</tr>
<tr>
<td>3) high VPA + low perceived stress.</td>
<td>PSS &lt;22, VPA&gt;20 min ≥ 3x p/w (n=10)</td>
<td>Effect of group: ANCOVA revealed smaller concentrations of cortisol in the low VPA groups, versus the high VPA groups (all p&lt;.05)</td>
<td>Effect of group: ANOVA revealed no difference in HR during the stress protocol across PA groups, F(3.38)=5.31, p=.01, η²=.295. Similar findings for AUCG, F(3,38)=5.40, p&lt;.01, η²=.310, AUCi, F(3,38)=5.28, p&lt;.01, η²=.294.</td>
</tr>
<tr>
<td>Sessions started at either 11:00 or 15:00.</td>
<td>PSS &lt;22, VPA&lt;20 min &lt; 3x p/w (n=11).</td>
<td>Interaction effects: ANCOVA was significant for cortisol, F(9,88)=2.57, p&lt;.05, η²=.174.</td>
<td>Cortisol responses to stress were always greatest in groups with low VPA, and smallest in groups with high VPA.</td>
</tr>
</tbody>
</table>

**Neuroendocrine:**


**Cardiovascular:**

- Freiburg Questionnaire of Physical Activity—short version.
- Created 3 groups.
- Mean (SD) age=24.31 (3.35).
- Mean BMI=23.56 (2.30).
- Baseline: 60 min.
- Stress: TSST (10 min; 5 min public speaking, and 5 min mental arithmetic) with social evaluation.
- HRV (HRV watch system): RMSSD, HF HRV (0.15-0.40 Hz), LF HRV (0.04-0.15 Hz), and the LF/HF ratio.

**Cardiovascular:**

- Effect of time: significant increase in cortisol, F(3,36)=18.69, p<.001, η²=.601.
- Effect of group: ANCOVA revealed smaller concentrations of cortisol in the low VPA groups, versus the high VPA groups (all p<.05).
- Interaction effects: ANCOVA was significant for cortisol, F(9,88)=2.57, p<.05, η²=.174.
- The baseline to peak (20 min pre- to 20 min post-stress) response was highest in the low VPA groups, relative to the high VPA groups, F(3,38)=5.31, p=.01, η²=.295. Similar findings for AUCG, F(3,38)=5.40, p<.01, η²=.310, AUCi, F(3,38)=5.28, p<.01, η²=.294.
- Cortisol responses to stress were always greatest in groups with low VPA, and smallest in groups with high VPA.

N=32 healthy participants (100% male).

Cross sectional, observational study comparing HRV and cortisol during stress across PA groups.

Sessions ran between 1-5pm.
### Vigorously active: PA >6 hours per week \((n=14)\).

### Recovery: 75 min.

and 1-, 5-, 10-, 20-, 30-, 45-, 60- and 75-min post-stress.

### Neuroendocrine:
- **Effect of time:** cortisol increased \((p<.001)\).
- **Effect of group:** ANOVA revealed no difference in cortisol during the stress protocol across PA groups, \(F(3.25, 47.09)=0.98, p=.417\).

### Hong et al., (2004).

Cross sectional, observational study comparing immune and catecholamine responses to stress across high v low PA groups.

Sessions ran between 8:15 am and 12:00 noon.

<table>
<thead>
<tr>
<th>Session</th>
<th>Participants</th>
<th>Mean (SD) age</th>
<th>Mean (SD) BMI</th>
<th>Baseline</th>
<th>Stress</th>
<th>Recovery</th>
<th>Neuroendocrine</th>
<th>Immune</th>
<th>Neuroendocrine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>24</td>
<td>37.3 (8.3)</td>
<td>26.4 (4.4)</td>
<td>30 min</td>
<td>speech (2 x 3 min speeches) with social evaluation</td>
<td>15 min</td>
<td>Plasma adrenaline and noradrenaline assayed in duplicate.</td>
<td>Immune cells measured (baseline, immediately-post, and 15 min-post stress) using flow cytometry/cell counter</td>
<td>Effects of time: adrenaline, (F(1,40)=8.02, p&lt;.01), and noradrenaline, (F(1,43) = 18.97, p&lt;.001), increased.</td>
</tr>
<tr>
<td>High</td>
<td>24</td>
<td>37.3 (8.3)</td>
<td>26.4 (4.4)</td>
<td>30 min</td>
<td>speech (2 x 3 min speeches) with social evaluation</td>
<td>15 min</td>
<td>Plasma adrenaline and noradrenaline assayed in duplicate.</td>
<td>Immune cells measured (baseline, immediately-post, and 15 min-post stress) using flow cytometry/cell counter</td>
<td>Effects of time: adrenaline, (F(1,40)=8.02, p&lt;.01), and noradrenaline, (F(1,43) = 18.97, p&lt;.001), increased.</td>
</tr>
</tbody>
</table>

Raw questionnaire score was generated by multiplying frequencies of weekly PA by 3 (light intensity PA), 5 (moderate intensity PA) and 9 (vigorous PA).

### Neuroendocrine:
- **Effect of time:** adrenaline, \(F(1,40)=8.02, p<.01\), and noradrenaline, \(F(1,43) = 18.97, p<.001\), increased.

### Immune:
- There were time (pre, immediately post stress, and 15-min post-stress) x group (low and high PA) interactions for: naive T cells, \(F(1,45)=5.97, p<.05\); memory T cells, \(F(1,45)=7.86, p<.01\); CD62L+ T cells, \(F(1,46)=4.02, p<.05\); and memory CD8+ T cells, \(F(1,43)=4.72, p<.05\); CD62L−NK cells, \(F(1,44)=4.82, p<.05\); lymphocyte CD8+CD62L−, \(F(1,45)=9.56, p<.05\); and NK CD62L+ cells, \(F(1,44)=20.27, p<.05\).

The low PA group had a higher number of the immune cells above across the stress protocol, relative to the high PA group.
Poole et al., (2011). Cross sectional, observational study correlating PA with BP, HR, and cortisol responses to stress. Sessions ran between 12.00 noon and 17.00.

N=40 healthy participants with elevated psychological distress (100% female).
Mean (SD) age = 28.7 (6.1) years.
Mean (SD) BMI = 23.0 (4.4) kg/m².
PA (min/day): LPA = 99.9 (22.6), MPA = 81.2 (28.3), VPA = 57.1 (24.3).

ActiGraph GT1M for 7 days on the hip and IPAQ.
The following cut-points were used (cpm): LPA = 191–573, MPA = 574–2099, VPA >2099 (Matthews, 2005).
Mean (SD) daily wear time across the week = 857 (58) min/day.
IPAQ scores computed by multiplying PA (minutes) by frequency (days) of the subscales.
Baseline: 50 min.
Stress: mirror tracing (5 min) and public speaking (5 min) with social evaluation.
Recovery: 25 min.

Cardiovascular: Continuous BP and HR (Finometer) measured during final 5 min of baseline, throughout stress, and final 5 min of recovery. Reactivity (task minus baseline) and recovery (recovery minus baseline) were computed.

Neuroendocrine: Salivary cortisol assayed in duplicate.
Samples taken at the end of baseline, immediately post-stress, and 20-min post-stress.

Note. SD=standard deviation, SE=standard error, BMI=body mass index, PSS=Perceived Stress Scale, IPAQ=International PA Questionnaire, PA=physical activity, LPA=light intensity physical activity, MPA=moderate intensity physical activity, VPA= vigorous intensity physical activity, J/kg/week=joules per kilogram per week, BP=blood pressure, SBP=systolic blood pressure, DBP=diastolic blood pressure, HR=heart rate, HRV=heart rate variability, HF=high frequency, LF=low frequency, RMSSD= Root Mean Square of the Successive Differences, AUC=area under the curve, ANOVA = analysis of variance, CPM=counts per minute, p/w = per week, Tₜ cells=T helper cells, Tₑ = T cytotoxic cell, NK cell=natural killer cell.
Table 3. Risk of bias assessment

<table>
<thead>
<tr>
<th>Study</th>
<th>Downs and Black (reporting)</th>
<th>Downs and Black (external validity)</th>
<th>Downs and Black (internal validity)</th>
<th>Downs and Black (power)</th>
<th>Downs and Black risk of bias</th>
<th>ROBINS-I (total score)</th>
<th>ROBINS-I risk of bias</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td><strong>Sedentary Behaviour</strong></td>
<td></td>
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<tr>
<td>Dziembowska et al. (2019)</td>
<td>6</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>9</td>
<td>Moderate</td>
<td>15</td>
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<tr>
<td>Endrighi et al. (2016)</td>
<td>7</td>
<td>0</td>
<td>3</td>
<td>1</td>
<td>11</td>
<td>Low</td>
<td>15</td>
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<tr>
<td>Ferreira-Silva et al. (2018)</td>
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<td>3</td>
<td>0</td>
<td>10</td>
<td>Moderate</td>
<td>10</td>
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<tr>
<td>Zaffalon Júnior et al. (2018)</td>
<td>6</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>9</td>
<td>Moderate</td>
<td>11</td>
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<tr>
<td><strong>Physical Activity</strong></td>
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</tr>
<tr>
<td>Buckworth et al. (1994)</td>
<td>6</td>
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<td>2</td>
<td>1</td>
<td>9</td>
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<td>14</td>
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<tr>
<td>Gerber et al. (2017)</td>
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<td>13</td>
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<td>3</td>
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<td>Hermann et al. (2019)</td>
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<td>3</td>
<td>1</td>
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<td>Moderate</td>
<td>11</td>
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<tr>
<td>Hong et al. (2004)</td>
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<td>3</td>
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<tr>
<td>Poole et al. (2011)</td>
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<td>3</td>
<td>0</td>
<td>10</td>
<td>Moderate</td>
<td>15</td>
</tr>
<tr>
<td>Taylor &amp; Katomeri (2006)</td>
<td>6</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>9</td>
<td>Moderate</td>
<td>13</td>
</tr>
</tbody>
</table>

*Note: Downs and Blacks score of 0-5 = high risk of bias, 6-10 = moderate risk of bias and >10 = low risk of bias. ROBINS-I score of 0-7 = high risk of bias, 8-14 = moderate risk of bias and >14 = low risk of bias*
DISCUSSION

This is the first systematic review of the sedentary behaviour and psychobiological stress reactivity literature. This is also the first review to summarise the physical activity-stress reactivity literature whilst solely accounting for total (i.e., lifestyle/incidental) physical activity, as previous reviews have quantified physical activity using both physical activity and/or exercise behaviours. Our review furthers the existing literature as exercise behaviours can be unrepresentative of total physical activity volume and the whole physical activity intensity spectrum. Across this review findings were inconsistent, likely due to large methodological heterogeneity, and therefore drawing conclusions remains difficult at this time. This review should be used to guide future studies in the area.

Measurement of sedentary behaviour

Although four studies claimed to assess sedentary behaviour, none assessed postural and metabolic components of sedentary behaviour in line with the widely accepted definition (Tremblay et al., 2017). Instead, these studies used physical inactivity or a lack of movement as indirect indices of sedentary behaviour. Although associations with certain CVD risk factors (e.g., body fat) are similar when comparing posturally-determined sedentary behaviour and markers of sedentary behaviour (Edwardson et al., 2020), and that markers of sedentary behaviour are frequently used in the literature (Prince et al., 2020), they fail to account for postural components of sedentary behaviour. This is important because a regularly adopted sedentary posture might be an important determining factor for psychobiological stress reactivity, possibly via heightened pathways blood pressure (Dempsey et al., 2018), inflammation (Dogra et al., 2019) and sympathetic activity (Dempsey et al., 2018, 2020) under conditions of rest. For example, a seated posture induces the bending of arteries in the leg, which promotes turbulent blood flow and negatively impacts resting blood pressure control (Thosar et al., 2014). This might also be further exacerbated through a pathway of heightened resting sympathetic nervous system activity that is observed in a seated posture (Dempsey et al., 2016, 2018, 2020). Importantly, higher resting blood pressure is predictive of exaggerated cardiovascular (Sheffield et al., 1997; Balanos et al., 2010), inflammatory (Steptoe et al., 2016) and respiratory (Sims et al., 1988) responses to acute psychological stress. In summary, this review has shown that no pre-existing
research has measured sedentary behaviour (per the current definition; Tremblay et al., 2017) in the
context of psychobiological stress reactivity, which highlights an urgent methodological consideration
for future research, which would benefit greatly by using gold-standard inclinometry.

Markers of sedentary behaviour and psychobiological responses to stress
One study used a cross-over design to increase sedentary time over 14 days, but found no differences
across condition (“sedentary” vs normal lifestyle) in BP, HR, IL-6, or cortisol responses to stress
(Endrighi, Steptoe and Hamer, 2016). This might be explained by their highly active sample, with
emerging research suggesting that sedentary behaviour is most deleterious for resting blood pressure
(Spehar et al., 2020), markers of inflammation (Henson et al., 2013) and CVD mortality (Ekelund et
al., 2019) in physically inactive populations. Consequently, future research in this area should aim to
recruit inactive populations. The lack of differences across condition might also be partially explained
by the limited intervention duration, small increases in sedentary time, and because it cannot be
ascertained whether individuals were more “sedentary” or just performed less movement. Acute (i.e.,
< 1 day) and tightly controlled laboratory-based interventions to manipulate sitting time may be
beneficial before further real-world interventions are implemented.

In the context of habitual markers of sedentary behaviour, Zaffalon Júnior et al. (2018) found that
“sedentary” females had higher HR, lower time domain HRV, and lower HF-HRV, during recovery
from stress, compared to active females. This suggests that sympathetic hyperactivity during stress
might be observed in sedentary individuals, which is supported by research under resting conditions
(dos Santos et al., 2019). Larger HR responses to stress are prospectively associated with CVD risk
factors (Chida and Steptoe, 2010; Turner et al., 2020) and therefore this might represent a novel
mechanism linking markers of sedentary behaviour with CVD. However, other studies in this current
review using comparable designs and grouping approaches found no association between sedentary
behaviour indices and HR and/or BP responses to stress (Ferreira-Silva et al., 2018; Dziembowska et
al., 2019). One explanation for these discrepancies might relate to sample variability, including that
Zaffalon Júnior et al.’s (2018) “sedentary” group had poorer self-reported physical health, higher
resting HR, and maladaptive HRV metrics at rest. Consequently, future research might benefit from
recruiting populations at risk for CVD, rather than purely healthy samples. Finally, Ferreira-Silva et al. (2018) found that blood flow and vascular conductance of the forearm was attenuated under stress in their sedentary (low PA) group, compared to their active group. This suggests that larger vasoconstrictory responses to stress were observed in those with an elevated index of sedentary behaviour (Ferreira-Silva et al., 2018). This is a potential mechanism linking sedentary behaviour and CVD that warrants further investigation, particularly given that exaggerated peripheral vasoconstriction during stress (as indexed by a lower stress/baseline peripheral arterial tonometry ratio) is associated with increased risk of major adverse cardiovascular events (Kim et al., 2019).

Dziembowska et al. (2019) found statistically lower concentrations of cortisol 45-min post-stress in their sedentary group, relative to an active group. Research has shown that attenuated cortisol responses to stress can be maladaptive, with prospective links to CVD risk factors, including obesity and depression (Carroll et al., 2017). This may be explained by the potent anti-inflammatory effects of cortisol under stress (Kunz-Ebrecht et al., 2003), especially given that obesity and depression are characterised by elevated basal levels of inflammation (Ouakinin, Barreira and Gois, 2018). Consequently, the findings of Dziembowska et al. (2019) highlight the potential for a link between higher volumes of sedentary behaviour and larger pro-inflammatory responses to stress, but this must remain speculative until future research has tested this.

**Physical activity and psychobiological responses to stress**

Two studies found lower HR during stress in groups with higher volumes of habitual physical activity (Buckworth, Dishman and Cureton, 1994; Hanson et al., 2013), although the latter found this exclusively for vigorous intensity physical activity. Given that large HR responses to stress are associated with hypertension and sudden coronary death (Turner et al., 2020), this lowered HR response to stress might be a cardioprotective mechanism induced by regular physical activity. HR under stress is also a marker of autonomic functioning (Brindle et al., 2014), and therefore these findings might also suggest that physical activity improves stress-induced autonomic tone, which is aligned with the resting literature (Tebar et al., 2020). However, others in this review found no link between physical activity and HR (Taylor and Katomeri, 2006; Poole et al., 2011; Gerber et al.,
HRV (Hanson et al., 2013; Hermann et al., 2019) or catecholamine (Hong et al., 2004) responses to stress. Further research using accelerometry and a range of autonomic measures is needed to confirm whether physical activity impacts autonomic tone under stress. Gerber et al. (2017) found that groups with higher volumes of device-assessed vigorous physical activity (relative to groups with low volumes of vigorous physical activity) showed attenuated cortisol output during stress. This possibly highlights the importance of higher intensity physical activity for reducing cortisol stress reactivity, which is important because large cortisol responses to stress are associated with higher risk of hypertension, and the progression of coronary artery calcification (Turner et al., 2020). However, others found no link between physical activity (including moderate-vigorous physical activity) and cortisol responses to stress (Poole et al., 2011; Hermann et al., 2019).

The non-significant findings of Poole et al. (2011) might be partially explained by the weak socially evaluative nature of their stress paradigm (Dickerson and Kemeny, 2004), which should be noted for future research. The non-significant findings of Hermann et al. (2019) could be explained by limited group differences in physical activity; although self-reported physical activity differed across groups, objectively measured VO2 max was homogenous. This may reflect well-known biases regarding self-report physical activity methodologies, and highlight the importance for forthcoming studies to select device-based approaches (Van Poppel et al., 2010).

One study revealed that active (relative to inactive) individuals had smaller concentrations of lymphocyte populations during stress, including CD62L expressing lymphocytes (Hong et al., 2004). This might possibly be explained by healthier autonomic tone under stress as a result of regular physical activity (Hong et al., 2004; Tebar et al., 2020). As L-selectin is a key adhesion molecule implicated in lymphocyte migration from circulation to tissue (Ivetic, Green and Hart, 2019), this attenuated L-selectin immune response to stress could potentially reflect a reduced likelihood of experiencing inflammatory events in those who are physically active (Ivetic, Green and Hart, 2019). However, this would need to be directly examined.

Cross-stressor adaptation hypothesis and links to previous reviews
The physical activity-reactivity literature is governed by the cross-stressor adaptation hypothesis (Sothmann et al., 1996), and this hypothesis was generally supported by a recent systematic review (Mücke et al., 2018). However, our work provides only minimal support to this hypothesis. One explanation relates to the fact that this previous review included many studies which used exercise behaviours to index total physical activity volume (as well as including studies that actually measured habitual physical activity), rather than solely focusing on physical activity (Mücke et al., 2018).

Although often used interchangeably, exercise and physical activity are separate behaviours with unique definitions (Caspersen, Powell and Christenson, 1985), such that exercise often represents a singular session of movement (which can often be higher intensity), and is usually unreflective of total physical activity volume (Caspersen, Powell and Christenson, 1985). Consequently, our review is arguably more reflective of daily/lifestyle physical activity, the wider physical activity intensity spectrum, and current physical activity guidelines.

This work also extends this earlier review (Mücke et al., 2018) by examining a wider range of psychobiological responses to any stress task. However, no sedentary behaviour studies and only two physical activity papers utilised passive stressors, even though dysregulated reactivity to passive stress also relates to CVD risk (Zhao et al., 2012). Examining responses to both types of stress is important due to the different response patterns they induce. Passive stressors induce primarily α-adrenergic reactions with more vascular perturbation, whereas active stressors evoke primarily β-adrenergic pathways with increased myocardial responses (Sherwood, Dolan and Light, 1990). As sedentary behaviour and physical activity influence autonomic pathways under rest, future work should compare reactivity to both types of stress, as this might help untangle any underlying mechanisms driving associations between sedentary behaviour/physical activity and stress reactivity. Future research may also choose to explore possible psychological mechanisms. For example, it is plausible that higher levels of physical activity and lower levels of sedentary behaviour might relate to increased self-efficacy, which leads to stressful events being perceived as a controllable challenge rather than an uncontrollable threat, with the inducement of a healthier psychobiological response to stress (Meijen et al., 2020). Finally, our review supports a previous systematic review which found inconsistent
evidence linking sedentary behaviour to objective markers of stress (e.g., cortisol, blood pressure, heart rate) under resting conditions (Teychenne et al., 2019).

**Recommendations for future research**

Our review has highlighted key areas for future research to investigate. For the sedentary behaviour literature, it is critical that future studies accurately quantify postural and metabolic components of sedentary behaviour using inclinometry, as this was lacking in all reviewed studies. It would also be interesting for future studies to explore how prolonged bouts of sedentary behaviour influence psychobiological stress reactivity. Finally, the interaction between sedentary behaviour and physical activity (Ekelund et al., 2019) could be explored in the context of stress reactivity. In the physical activity domain, 24hr accelerometry methodologies, in combination with universal cross-brand data analysis techniques (e.g., using raw gravitational acceleration) should be adopted. Research should also explore whether physical activity influences inflammatory responses to stress and examine whether a higher intensity of physical activity (such as with exercise intensity; Mücke et al., 2018) is most important for attenuating measures of stress reactivity. There is also a critical need for longitudinal studies and further randomised controlled/crossover trials in this area, so that potential causal links between sedentary behaviour/physical activity and stress reactivity can be investigated.

This review has revealed that no studies have investigated respiratory responses to acute psychological stress in the context of sedentary behaviour or physical activity. Like with other stress reactivity markers, respiratory stress responses are driven by autonomic pathways (Dampney, 2015), and can be used to non-invasively index key metabolic changes under stress. For example, stress-induced changes in oxygen consumption reflects changes in metabolic output, and a reduction in end-tidal partial pressure of carbon dioxide in response to stress is indicative of hyperventilation (Meuret and Ritz, 2010). Interestingly, the “metabolically appropriateness” of cardiovascular responses to stress is theorized to be a potential mechanism linking exaggerated cardiovascular reactivity to CVD (Balanos et al., 2010). In addition, dysregulated respiratory responses to stress are associated with CVD risk markers, including vasoconstriction and restricted cardiac blood flow (Grossman, 1983; Rutherford, Clutton-Brock and Parkes, 2005). Therefore, measuring respiratory responses to stress in
the context of sedentary behaviour and physical activity might provide a unique insight into novel metabolic pathways that might link sedentary behaviour and physical activity to cardiometabolic disease. Moreover, given that there are known interactions and similar underpinning mechanisms (e.g., sympathetic pathways) between the psychobiological systems that this review examined (e.g., cardiovascular, inflammatory/immune, cortisol and respiratory systems) future work may benefit from adopting a “multisystem” approach and examining these systems concurrently. It might also be valuable for research to look at multiple markers of reactivity (e.g., peak latency, peak response, recovery, curvature) because this approach is likely to provide a more comprehensive insight into the stress response patterns associated with sedentary behaviour/physical activity.

Methodological considerations of this present review

Strengths include the broad nature of this systematic review (e.g., accounting for a wide range of psychobiological responses to any active or passive stress task), duplication of every stage of the review, and using multiple risk of bias tools. Limitations were the large methodological variability that precluded meta-analyses and drawing conclusions from the literature. We included a wide range of psychological stress tasks in our review, including the Stroop Colour and Word Test. However, while this paradigm is widely used in the stress reactivity literature, there remains some controversy surrounding its capability to sufficiently stimulate the psychobiological stress axes. Next, only peer-reviewed articles were included, but this choice was made to ensure high rigour. There were occasional missing study data, and unfortunately some authors did not respond to requests for further information. Finally, the focus of this review was person-level sedentary behaviour/physical activity, and it should be noted that acute sedentary behaviour/physical activity interventions (e.g., lasting < 1 day) might yield different results.

Conclusion

This systematic review is the first to summarise the sedentary behaviour and psychobiological stress reactivity literature, although no studies measured postural and metabolic components of sedentary behaviour as per the widely accepted definition (Tremblay et al., 2017). In the physical activity–reactivity domain, we only included studies that measured total (i.e., incidental/lifestyle physical
activity) volume, and excluded those studies focusing on exercise behaviours, which are often unrepresentative of total physical activity volume. The methodological variability of this literature is substantial and therefore conclusions cannot be drawn at this present time. We hope this review can encourage future research to adopt more homogenous and gold-standard methodologies, including the assessment of postural components of sedentary behaviour with inclinometry, and the measurement of different physical activity intensities using 24hr accelerometry with universal analytical techniques.
REFERENCES


Parsons, T. J. et al. (2017) ‘Physical activity, sedentary behavior, and inflammatory and hemostatic


