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 inclinometery, 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 accelerometery, 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.

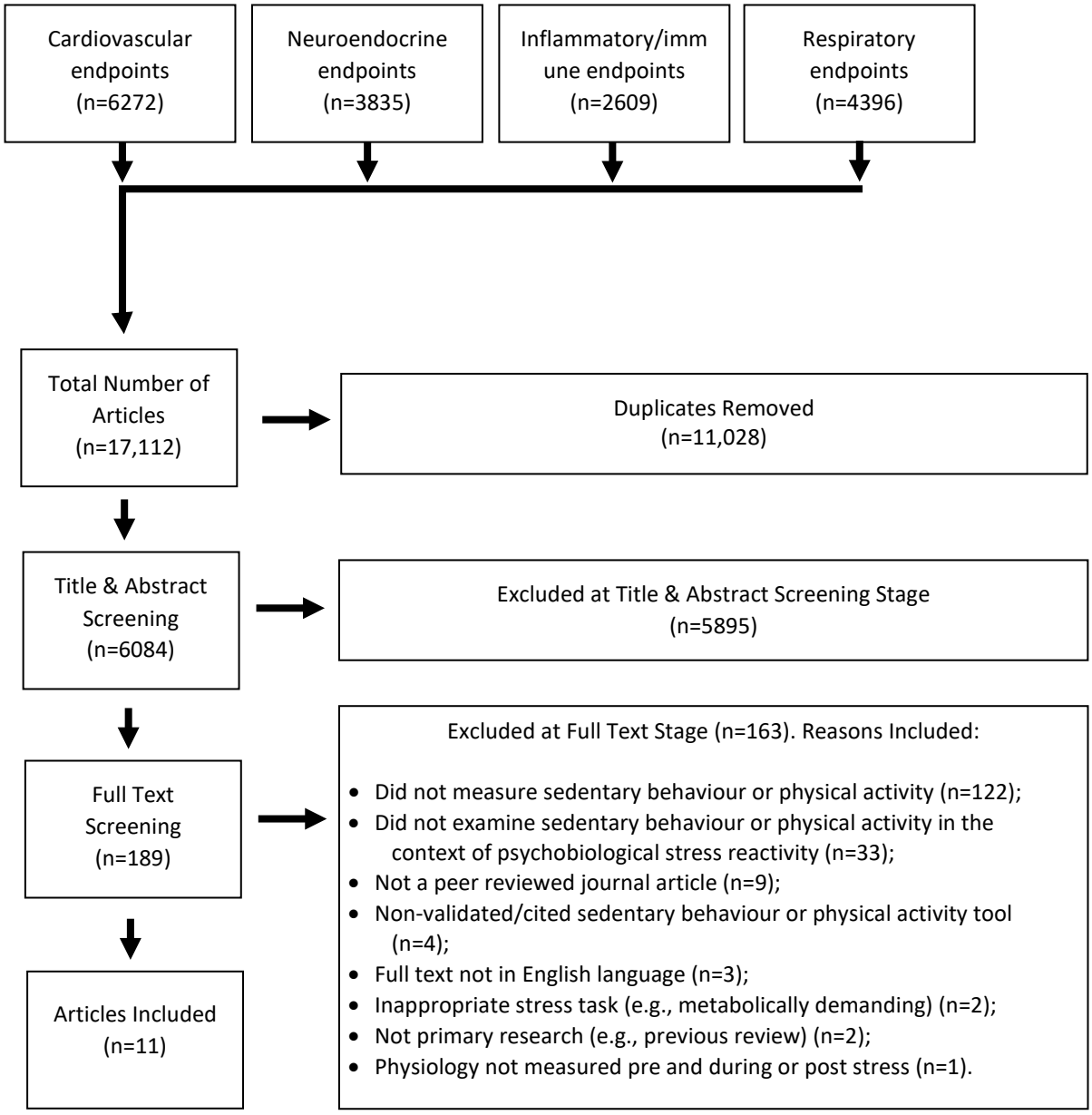

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram.

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 $T_{S/C}$ cells, memory $T_{S/C}$ cells, $CD62L^+$ $T_{S/C}$ cells, memory T_H cells, $CD62L^-$ natural killer cells and lymphocyte $CD8^+CD62L^+$ 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.

Reference	Study design	Sample characteristics	Index of sedentary behaviour	Stress paradigm	Psychobiological measures	Main findings
			Intervention study			
Endrighi *et al*. (2016).	Randomised cross-over trial with two conditions: 1) 2-week sedentary condition (physical activity replaced with sedentariness). 2) 2-week "normal lifestyle" control condition HR, BP, IL-6, and cortisol were measured at rest, during stress and during post-stress recovery after both conditions.	*N*=43 healthy participants (44% female). Mean *(SD)* age=24.8 (4.5) years. Mean *(SD)* BMI=23.3 (2.4) kg/m^2. During control condition, mean *(SD)* "sedentary" time=575 (7) min/day, LPA=82 (39) min/day, MVPA=140 min/day. During sedentary condition, mean *(SD)* "sedentary time" =607 (86) min/day, LPA=71 (24) min/day, MVPA=103 (35) min/day.	Hip-mounted ActiGraph GT1M worn for 7 days, with the following cut-points: sedentary = <190, LPA = 191–573, MPA = 574–2099, VPA = >2099 (cpm). Average daily wear time across the week = 12h. Sedentary time was calculated as the daily total wear time minus total daily active time. Mean (*SE*) increase of 31.49 (12.13) min/day of "sedentary behaviour" in the sedentary condition versus the normal lifestyle condition.	Baseline: duration not reported. Stress: mirror tracing (5 min) and public speaking (5 min) with social evaluation. Recovery: 45 min.	***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) ***Inflammatory:*** Plasma IL-6 assayed in duplicate. Samples collected: end of baseline and 45-min post-stress. ***Neuroendocrine:*** Salivary cortisol assayed in duplicate. Samples collected: end of baseline, immediately post-	***Cardiovascular:*** Effect of time: Increases in SBP, $F(2,82)=64.04$, $p<.001$, DBP, $F(2,82)=77.96$, $p<.001$ and HR, $F(1.58, 64.80)=48.72$, $p<.001$. 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:*** Effect of time: non-significant for IL-6, $F(1,41)=0.29$, $p=0.59$. Interaction effect: mixed-model ANOVA revealed no time-by-condition interaction for IL-6, $F(1,41)=1.44$, $p=.23$. ***Neuroendocrine:*** Effect of time: Increase in cortisol concentration, $F(3,123) = 8.80$, $p<.001$.

	Time of testing: either 10:00 or 14:00, but same for each participant across both conditions.				stress, 20- and 45-min post-stress.	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			
Dziembowska *et al*., (2019).	Cross-sectional observational study comparing HR and cortisol during stress in sedentary vs active participants. Sessions began between 9-11am.	*N*=55 healthy participants (100% female). Mean age *(SD)*=22.5 (1.1) years. Mean BMI *(SD)*=23.8 (1.3) kg/m^2.	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. 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.	***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. $F(3,180)=3.07$, $p=.029$, $\eta^2=0.05$.

Ferreira-Silva *et al*., (2018).	Cross sectional observational study comparing HR and HRV during stress in sedentary vs physically active individuals. Session ran in the morning (no specific time reported).	*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/m^2.	IPAQ Created 2 groups: Sedentary group (*n*=21), PA <30 min/week. PA group (*n*=19), PA ≥150 min/week, including MVPA ≥ 3 days/week and ≥ 30 min/bout.	Baseline: 4 min. Stress: Stroop (3 min) with no social evaluation. No recovery period reported.	***Cardiovascular:*** Intermittent BP (cuff on the ankle) and continuous HR (ECG). BP measurements taken minutely. HR measurements taken continuously throughout baseline and stress.	***Cardiovascular:*** Effect of time: significant increase in BP and HR (all p <.05) Effect of group: unpaired Student t-tests revealed no group differences in BP or HR during stress (all p >.05)
Zaffalon Júnior et al., (2018).	Cross sectional, observational study comparing HR and HRV during stress recovery in sedentary vs physically active women. Session time not reported.	*N*=96 healthy participants (100% female). Mean (*SD*) age=23.2 (3.8) years. Mean (*SD*) BMI=22.2 (3.3) kg/m^2.	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.	Baseline: 15 min. Stress: Stroop (duration not reported) with no social evaluation. Recovery: 15 min.	***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	***Cardiovascular:*** *HR* Effect of time: HR increased (p<.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<.05). No further data provided. *Frequency domain HRV* Effect of time: not reported.

		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.

Reference	Study design	Sample characteristics	Assessment of physical activity	Stress paradigm	Psychobiological measures	Main findings
			Intervention studies			
Hanson *et al.*, (2013).	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.	*N*=44 healthy participants (100% female). Mean *(SD)* age=23.7 (5.9) years. Mean *(SD)* BMI=22.6 (3.0) kg/m^2.	IPAQ Created 2 groups based on national VPA guidelines: High active = >30 min of VPA ≥ 3 days/week (*n*=22). Low active = < 30 min of VPA < 3 days/week (*n*=18).	Baseline: 5 min. Stress: Mental arithmetic component of the TSST (5 min) with social evaluation. Recovery: not reported	***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.	***Cardiovascular*** (only placebo data is presented). HR effect of time: HR increased $F(1, 38)=210.43$, $p<.001$, η^2 p=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, $F(1, 38)=84.60$, $p<.001$, $d=1.47$, and RMSSD decreased, $F(1, 38)=35.99$, $p<.001$, $d=0.96$. HRV effect of group: non-significant.
Taylor *et al*., (2006).	Experimental study comparing HR and BP reactivity after 15 min walk with 15 min resting control. Session time not reported.	*N*=60 healthy temporary abstinent smokers (57% female). Mean *(SD)* age = 28.6 (7.6) years. Mean *(SD)* BMI = 23.0 (3.2) kg/m^2.	7-day PA recall interview Did not split sample into groups – treated PA data continuously.	Baseline: 10 min. Stress: Stroop (3min), speech (2 min) & holding cigarette (duration not reported). Recovery: not reported.	***Cardiovascular:*** Intermittent BP and HR (arm cuff). Δ in BP & HR was calculated separately for each task by subtracting BP and HR measured immediately post-stress from baseline.	***Cardiovascular:*** Effect of time: SBP (by 4.3 mmHg), DBP (by 2.9 mmHg) and HR (by 2.3 bpm) increased (all p <.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>0.05).

			Observational studies			
Buckworth *et al*., (1994).	Cross-sectional observational study comparing HR and BP during stress in individuals with high vs moderate levels of PA. Time of testing not reported.	*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/m^2.	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.	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 >.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 <.05)
Gerber *et al*., (2017).	Cross sectional, observational study 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.	*N*=42 healthy participants (52% female). Mean *(SD)* age=21.2 (2.2) years. Mean *(SD)* BMI = 22.1 (1.8) kg/m^2.	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 >22, VPA>20 min ≥ 3x p/w (*n*=9).	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<.001, η^2=.620. Effect of group: ANCOVA revealed no between-group differences in HR, in terms of: AUC_G, F(3,38)=2.21, p=.102, η^2=.149, AUC_I, F(3,38)=0.91, p=.445, η^2=.067, and peak minus baseline F(3,38)=1.31, p=.286, η^2=.094. Interaction effects: ANCOVA revealed no significant time-by-group interaction for HR F(3,38)=2.48, p=.076, η^2=.164.

	3) high VPA + low perceived stress. 4) low VPA + low perceived stress. Sessions started at either 11:00 or 15:00.	2) PSS >22, VPA<20 min < 3x p/w (*n*=12) 3) PSS <22, VPA>20 min ≥ 3x p/w (*n*=10) 4) PSS <22, VPA<20 min < 3x p/w (*n*=11).			***Neuroendocrine:*** Effect of time: significant increase in cortisol, $F(3,36)=18.69$, $p<.001$, $\eta^2=.601$. 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, $F(9,88)=2.57$, $p<.05$, $\eta^2=.174$. 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, $F(3,38)=5.31$, $p<.01$, $\eta^2=.295$. Similar findings for AUC_G, $F(3,38)=5.40$, $p<.01$, $\eta^2=.310$, AUC_I, $F(3,38)=5.28$, $p<.01$, $\eta^2=.294$. Cortisol responses to stress were always greatest in groups with low VPA, and smallest in groups with high VPA.
Hermann *et al*., (2018).	Cross sectional, observational study comparing HRV and cortisol during stress across PA groups. Sessions ran between 1-5pm.	N=32 healthy participants (100% male). Mean *(SD)* age=24.31 (3.35). Mean BMI=23.56 (2.30).	Freiburg Questionnaire of Physical Activity–short version. Created 3 groups. Inactive: PA <2 hours per week (*n*=8). Moderately active: PA between 2 and 6 hours per week (*n*=10).	Baseline: 60 min. Stress: TSST (10 min; 5 min public speaking, and 5 min mental arithmetic) with social evaluation.	***Cardiovascular:*** HRV (HRV watch system): RMSSD, HF HRV (0.15-0.40 Hz), LF HRV (0.04-0.15 Hz), and the LF/HF ratio. ***Neuroendocrine:*** Serum cortisol assayed in duplicate. Blood samples collected: 15- and 1-min pre-stress	***Cardiovascular:*** Effect of time: RMSDD, $F(1.75, 54.29)=15.59$, $p<.001$, $\eta^2=0.34$, and LF HRV, $F(2.16, 67.06)=4.05$, $p=.019$, $\eta^2=0.12$, decreased, and LF/HF ratio increased, $F(2.04, 63.18)=3.17$, $p=.048$, $\eta^2=0.09$. Effect of group: ANOVA revealed no difference in HR during the stress protocol across PA groups, $F(3.38, 49,06)=0.398$, $p=.778$. HRV data was non-significant, but no further details presented.

			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.	*N*=48 healthy participants (52% female). Mean *(SD)* age=37.3 (8.3) years. Mean *(SD)* BMI =26.4 (4.4) kg/m^2.	Godin-Shephard Leisure-Time PA Questionnaire. Created 2 groups based on median split: Low active, mean *(SD)* raw questionnaire score of 18.56 (1.77) (*n*=24). High active, mean *(SD)* raw questionnaire score of 55.90 (3.50) (*n*=24). Raw questionnaire score was generated by multiplying frequencies of weekly PA by 3 (light intensity PA), 5 (moderate intensity PA) and 9 (vigorous PA).	Baseline: 30 min Stress: speech (2 x 3 min speeches) with social evaluation Recovery: 15 min	***Neuroendocrine*** Plasma adrenaline and noradrenaline assayed in duplicate. ***Immune:*** Immune cells measured (baseline, immediately-post, and 15 min-post stress) using flow cytometry/cell counter lymphocytes ($CD3^+$), T_H cells ($CD3^+CD4^+$), Naïve T_H cells ($CD45RA^+CD62L^+$), Memory T_H cells ($CD45RO^+$), T_C cells ($CD3^+CD8^+$), Naïve T_C cells ($CD45RA^+CD62L^+$), Memory T_C cells ($CD45RO^+$), NK cells ($CD3^-CD16^+56^+$), $CD4^+CD62L^+$, $CD4^+CD62^-$ and $CD8^+CD62L^+$.	***Neuroendocrine*** Effects of time: adrenaline, F(1,40)=8.02, p<.01, and noradrenaline, F(1,43) = 18.97, p<.001, increased. Interaction effects: time (pre, immediately post stress, and 15-min post-stress) x group (low and high PA) repeated measure ANOVAs revealed no significant interaction for adrenaline or noradrenaline (all p>.05). ***Immune*** Effects of time: lymphocyte subsets and CD62L cells increased (all p<.05). Interaction effects: There were time (pre, immediately post stress, and 15-min post-stress) by group (low and high PA) interactions for: naïve $T_{S/C}$ cells, F(1,45)=5.97, p<.05; memory $T_{S/C}$ cells, F(1,45)=7.86, p<.01; $CD62L^+$ $T_{S/C}$ cells, F(1,46)=4.02, p<.05; memory T_H 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; 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/m^2. 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.	***Cardiovascular:*** Effect of time: BP and HR increased (all p <.05). Correlational analyses: no association between self-reported or ActiGraph-determined total PA, LPA, MPA and VPA, and the Δ in BP or HR in response to, or recovery from, stress (all p>.05). ***Neuroendocrine:*** Effect of time: cortisol concentration increased Correlational analyses: no association between self-reported or ActiGraph-determined total PA, LPA, MPA and VPA, and the Δ in cortisol during recovery from stress (all p>.05).

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, 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, 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, T_H cells=T helper cells, T_c = T cytotoxic cell, NK cell=natural killer cell

Table 3. Risk of bias assessment

Study	Downs and Black (reporting)	Downs and Black (external validity)	Downs and Black (internal validity)	Downs and Black (power)	Downs and Black (total score)	Downs and Black risk of bias	ROBINS-I (total score)	ROBINS-I risk of bias
Sedentary Behaviour								
Dziembowska *et al*. (2019)	6	0	3	0	9	Moderate	15	Low
Endrighi *et al*. (2016)	7	0	3	1	11	Low	15	Low
Ferreira-Silva *et al*. (2018)	7	0	3	0	10	Moderate	10	Moderate
Zaffalon Júnior *et al*. (2018)	6	0	3	0	9	Moderate	11	Moderate
Physical Activity								
Buckworth *et al*. (1994)	6	0	2	1	9	Moderate	14	Moderate
Gerber *et al*. (2017)	7	0	3	0	10	Moderate	13	Moderate
Hanson *et al*. (2013)	7	0	3	0	10	Moderate	15	Low
Hermann *et al*. (2019)	5	0	3	1	9	Moderate	11	Moderate
Hong *et al*. (2004)	6	0	3	0	9	Moderate	15	Low
Poole *et al*. (2011)	7	0	3	0	10	Moderate	15	Low
Taylor & Katomeri (2006)	6	0	3	0	9	Moderate	13	Moderate

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.*,

2017), HRV (Hanson *et al.*, 2013; Hermann *et al.*, 2019) or catecholamine (Hong *et al.*, 2004) responses to stress. Further research using accelerometery 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 VO_2 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 inclinometery, 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 accelerometery 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

488 the context of sedentary behaviour and physical activity might provide a unique insight into novel
489 metabolic pathways that might link sedentary behaviour and physical activity to cardiometabolic
490 disease. Moreover, given that there are known interactions and similar underpinning mechanisms
491 (e.g., sympathetic pathways) between the psychobiological systems that this review examined (e.g.,
492 cardiovascular, inflammatory/immune, cortisol and respiratory systems) future work may benefit from
493 adopting a "multisystem" approach and examining these systems concurrently. It might also be
494 valuable for research to look at multiple markers of reactivity (e.g., peak latency, peak response,
495 recovery, curvature) because this approach is likely to provide a more comprehensive insight into the
496 stress response patterns associated with sedentary behaviour/physical activity.

497 **Methodological considerations of this present review**

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

510 **Conclusion**

511 This systematic review is the first to summarise the sedentary behaviour and psychobiological stress
512 reactivity literature, although no studies measured postural and metabolic components of sedentary
513 behaviour as per the widely accepted definition (Tremblay *et al.*, 2017). In the physical activity–
514 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 accelerometery with universal analytical techniques.

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