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SYSTEMATIC REVIEW

Influence of Regular Physical Activity and Fitness on Stress Reactivity as Measured with the Trier Social Stress Test Protocol: A Systematic Review

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Abstract

Background Psychosocial stress is associated with multiple health complaints. Research to date suggests that regular physical activity (PA) and higher cardiorespiratory fitness may reduce stress reactivity and therefore contribute to a reduction of stress-related risk factors. While previous reviews have not differentiated between stressors, we focus on psychosocial stress elicited with the Trier Social Stress Test (TSST).

Objective Our objective was to examine the effect of regular PA and cardiorespiratory fitness on stress reactivity, with a particular focus on the TSST. The TSST is the laboratory task most widely used to induce socio-evaluative stress and elicits stronger stress reactions than most other cognitive stressor tasks.

Methods A systematic search within various databases was performed in January 2018. The following outcomes were considered: cortisol, heart rate, psychological stress reactivity, and potential moderators (age, sex, exercise intensity, assessment mode, and psychological constructs).

Results In total, 14 eligible studies were identified. Cortisol and heart rate reactivity were attenuated by higher PA or better fitness in seven of twelve studies and four of nine studies, respectively. Two of four studies reported smaller increases in anxiety and smaller decreases in calmness in physically active/fitter participants. Three of four studies found that higher PA/fitness was associated with more favorable mood in response to the TSST.

Conclusion About half of the studies suggested that higher PA/fitness levels were associated with an attenuated response to psychosocial stress. Currently, most evidence is based on cross-sectional analyses. Therefore, a great need for further studies with longitudinal or experimental designs exists.

Key Points

Higher physical activity and fitness levels were associated with an attenuated adrenocortical stress reactivity in response to the Trier Social Stress Test (TSST) in about 60% of the studies, indicated by lower increases in salivary cortisol.

Higher physical activity and fitness levels were associated with a reduced cardiovascular reactivity in response to the TSST in about 40% of the studies, indicated by lower increases in heart rate.

Higher physical activity and fitness levels were associated with more favorable psychological responses following the TSST in about half of the studies.

Some evidence indicated a more favorable stress reactivity among people who typically engage in exercise activities with higher intensity levels.

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1 Introduction

In modern societies, psychosocial stress is a major issue associated with psychological and physiological health complaints [1, 2]. While reasonable levels of stress may have beneficial effects on individuals' development, exposure to excessively high and chronic stress that exceeds an individual's coping capacity increases allostatic load and poses substantial health risks across all age groups [3–5]. High subjective stress is associated with a higher risk of cardiovascular diseases [6], stroke [7], metabolic syndrome [8], immune system dysfunction [9] and higher all-cause mortality [10]. Furthermore, cognitive functions can be impaired [11], and stress can contribute to the development of depression or burnout [12].

Most researchers agree that regular physical activity (PA) and higher fitness levels are beneficial to health and well-being [13–16]. Obvious advantages of PA and exercise in comparison with other health-promoting or illness-preventing interventions are cost effectiveness, easy accessibility, and the absence of unwanted side effects, making them increasingly interesting for research [17]. Some researchers argue that PA/fitness not only promotes health through a direct reduction of risk factors for major diseases but also acts indirectly via stress-buffering effects [18–20]. According to a review by Gerber and Pühse [18], half of the reviewed studies supported the claim that people with high exercise levels exhibited fewer health problems if they were exposed to high stress levels.

Several physiological and psychological explanations for a possible attenuating effect of regular PA and better cardiorespiratory fitness on stress have been suggested. According to the cross-stressor-adaptation (CSA) hypothesis, exposure to physical stress (e.g., vigorous exercise) triggers a stress response comparable to that found in reaction to psychosocial stressors [21, 22]. The basic assumption behind the CSA hypothesis is that the (beneficial) adaptation of hypothalamic–pituitary–adrenocortical (HPA) axis activity and the sympathoadrenal medullary (SAM) system during the physical stress of regular exercise can generalize to other, non-physical (e.g., cognitive or psychosocial) stressors [23]. In stress reactivity studies, salivary free cortisol or blood cortisol are usually measured as the main (adrenocortical) parameter [24], with increases indicating a stimulation of the HPA axis. Heart rate (HR) is reported as a criterion for the reactivity of the cardiovascular system, which in turn is modulated by the autonomic nervous system (ANS) [2]. Higher increases in cortisol levels and HR indicate a higher stress response. Less frequently, researchers also assessed blood pressure [25], catecholamine concentrations [26], saliva alpha-amylase [27], or HR variability [28]. Parameters such as anxiety, mood, and calmness are most commonly assessed as psychological outcomes [29–31].

The importance of stress reactivity-related research was highlighted by meta-analytic findings by Chida and Steptoe [32], which showed that a higher stress reactivity, defined as the magnitude of the reaction to acute mental stress, was associated longitudinally with poorer cardiovascular status and a higher risk of subsequent cardiovascular diseases.

The most recent systematic reviews and meta-analyses on the effects of cardiorespiratory fitness on stress reactivity date back to 2006 [33, 34]. In their meta-analysis, Forcier et al. [33] found that participants with higher cardiorespiratory fitness levels showed lower cardiovascular reactivity in response to a wide range of different stressors. More specifically, they reported point estimates of -1.84 ($p < 0.005$) and -3.69 ($p < 0.001$) for HR and systolic blood pressure, respectively. While these findings suggest that higher fitness levels are associated with a blunted stress reactivity, they cannot be generalized to other physiological parameters or subjective stress reactions. In contrast, Jackson and Dishman [34] included a wider range of physiological outcomes in their meta-analysis (e.g., HR, blood pressure, catecholamines, cardiac function, cortisol). Their study also had a number of strengths, such as the exclusion of studies without maximal or submaximal fitness testing, the exclusion of stressors with a PA component, or the exclusion of studies with a mixed battery of active and passive stressor tasks. A combination of these various reactivity outcomes provided only limited support for the validity of the CSA hypothesis, and high cardiovascular fitness levels were even associated with a small, heterogeneous increase in stress reactivity ($\Delta = 0.08$, $p = 0.001$). Nevertheless, we hold that this global effect size must be interpreted with utmost caution because different physiological reactivity parameters are regulated by distinct physiological and psychological mechanisms. Moreover, combining different stressors is problematic as different stressor tasks such as physiological stressors (e.g., cold pressor task, forehead cold), cognitive stressors (e.g., Stroop task, mental arithmetic), and socio-evaluative stressors (e.g., public speaking) [33, 35] can elicit different physiological stress reactions [36]. For instance, Dickerson and Kemeny [35] showed that effects on cortisol levels vary greatly across tasks, with highest cortisol reactivity found in motivated performance tasks with the additional element of uncontrollability and socio-evaluative threat.

This may explain why the Trier Social Stress Test (TSST) has become one of the most widely used psychosocial stressor tasks during the past two decades. Developed and validated by Kirschbaum et al. [37], this test consists of an anticipation phase followed by a 5-min mock job interview and a 5-min mental arithmetic task, both in front of a non-responsive jury of two or three people. Over the years, different test versions for children (TSST-C) [38] and for simultaneous measurement in groups of six people (TSST-G) [39] have been developed. The TSST shows high

ecological validity and reliability. For instance, the TSST typically induces a two- to threefold increase in cortisol levels from baseline to peak [40, 41]. As shown by Dickerson and Kemeny [35], the socio-evaluative character of the TSST leads to a significantly stronger stress reaction than other cognitive stressor tasks (e.g., simple arithmetic tasks, Stroop task).

Given that the meta-analyses of Forcier et al. [33] and Jackson and Dishman [34] are more than a decade old and were conducted before researchers started using the TSST in CSA studies, we hold that it is time to expand current reviews by examining the effect of regular PA and cardiorespiratory fitness on stress reactivity with a special focus on the TSST, and separately for indicators of adrenocortical, cardiovascular, and psychological stress reactivity.

Considering that the relationship between PA/fitness and stress reactivity might be influenced by a variety of moderating factors, we also aim to provide an overview of moderators that have been examined in previous studies. For instance, as other researchers have pointed out, age might strongly affect physiological and psychological stress responses [34, 42]. Another potential moderator is participants' sex. As highlighted by Kudielka and Kirschbaum [43], glucocorticoid levels were usually higher in females after HPA axis stimulation in animal studies, whereas sex differences in humans seemed to depend on participants' age. Moreover, in a review article on the role of exercise as a stress modifier, Hackney [44] argued that, with increasing exercise intensities, the immediate neuroendocrine stress response (e.g., concentrations of cortisol and adrenocorticotropic hormone in the blood) rises proportionally. He therefore assumed that the intensity with which people typically exercise may have an impact on stress reactivity during psychosocial stressor tasks. Furthermore, scholars have argued that personality affects the perception and regulation of stress, which may also impact on physiological stress reactivity [45, 46]. Personality traits such as competitiveness have been shown to differ between athletes and non-athletes [47] and might lead to variations in cardiovascular stress reactivity [48]. In an attempt to provide a comprehensive model of emotion regulation and dysregulation, Thayer and Lane [49] developed the theory of neurovisceral integration and therein emphasized the importance of inhibitory processes. In a state of dysregulation (e.g., through high perceived or chronic stress), these negative feedback circuits can be disrupted, resulting in perseveration and continued activation of systems. According to Brosschot et al. [50], perseverative cognitions such as rumination, combined with prolonged psychological representations of stressors, can be a factor leading to altered physiological activation in response to stressors.

2 Methods

2.1 Search Strategy

The current systematic review was conducted according to the guidelines provided in the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement [51]. A research protocol with orientation to the PRISMA-P 2015 checklist was used [52, 53]. The systematic literature search was conducted independently by the first and third author of this article in January 2018 using the online databases PubMed, Web of Science, and PsycINFO. The search terms were ("TSST" OR "social stress*") AND ("physical activity" OR fitness OR exercise OR train* OR sport). As filters within the databases, "abstract availability", "publication date > 1993", and "human subjects" (if available) were used.

2.2 Study Selection and Data Extraction

Studies with cross-sectional and longitudinal design, as well as exercise intervention studies, were eligible for this review. Eligible studies had to (1) investigate the effect of regular PA and/or cardiovascular fitness on stress reactivity, (2) assess stress reactivity with the TSST (studies using an adapted version of the TSST were included if the original structure of the test, consisting of a preparation phase, a free speech task, and a mental arithmetic task, was still recognizable), (3) be published in peer-reviewed journals (in English or German), and (4) focus on healthy human subjects. Studies were excluded if (1) neither cortisol nor HR was measured as a stress reactivity indicator (see Sect. 1), (2) they showed no sufficient differentiation of subjects' PA or fitness levels (e.g., Rohleder et al. [54]), (3) subjects were recruited from non-healthy populations (e.g., Sjörs et al. [55]), or (4) if medication was tested on the subjects (e.g., Sommer et al. [56]). Since the focus of this review was on the effects of regular PA and cardiorespiratory fitness, we further excluded studies investigating the effects of acute bouts of PA on stress reactivity. Given that exposure to psychosocial and socio-evaluative stress is an issue that concerns children, adults, and the elderly (although the effects of acute stress might vary across age groups), no age-specific restrictions were imposed in this review.

After titles and abstracts were screened, full texts of the remaining studies were reviewed with regard to inclusion and exclusion criteria. Additionally, reference lists of available articles and contents of relevant journals were reviewed. If abstracts of studies met the inclusion criteria, but full texts were not available, or if data necessary for the review could not be found in the article, corresponding authors were contacted.

Relevant data were extracted from each article included in the review: age range, number and sex of participants, assessment methods for PA or fitness, group description, TSST version. Table 1 shows the main outcomes. With regard to stress reactivity, saliva or blood cortisol (HPA axis), HR (ANS), and anxiety, calmness, and mood (psychological reactivity) were regarded as main outcomes. Studies were only considered supportive of the CSA hypothesis if group differences or associations were statistically significant ($p < 0.05$).

3 Results

3.1 Overview of Studies

3.1.1 Number of Studies

Figure 1 shows the search process, which was in accordance with the PRISMA guidelines [52]. From the 645 studies initially identified, 14 met the inclusion criteria and are discussed in this review, comprising 13 cross-sectional and one experimental study. Note that the publication by Jayasinghe et al. [57] refers to a subsample of Jayasinghe et al. [26], and Strahler et al. [27] published new data from the study by Klaperski et al. [28].

3.1.2 Participants

In total, 1334 participants (60.7% male) were tested. Sample sizes varied between 34 and 258 participants per study (median 84). Participants' age ranged from 8 to 82 years, with two studies focusing on children [58, 59], seven on young adults aged 18–32 years [25, 29–31, 60–62], four on adults aged 18–65 years [26–28, 57], and one on older adults aged 54–82 years [63].

3.1.3 Stressor Task

As Table 1 shows, 12 studies used one of the TSST standard protocols (five TSST, two TSST-C, and five TSST-G [in groups of three to six participants]), and two studies modified the protocol to some extent (modified speech task to fit the target group [31, 63]).

3.1.4 Assessment of Physical Activity

PA was measured using subjective and objective approaches (Table 1). Validated questionnaires such as the Child Health and Illness Profile—Parent Form [58], the Measurement of Daily Activities and Exercise Questionnaire [28], or the International Physical Activity Questionnaire [62] were administered, or items assessing exercise frequency, duration, and intensity (excluding activities of daily living) were used instead [25, 30, 60, 63]. One study defined different exercise groups via recruitment methods [31]. Objective measurement of PA was achieved via accelerometry over the course of 5–7 days [29, 59].

3.1.5 Assessment of Fitness

Table 1 shows that fitness levels were determined using (spiro)ergometry [26–28, 57], a multilateral fitness test designed for the Swiss army [62], a 4×1000 m running test at increasing subjective exertion [60], or a 3.1 km walking task at an average speed of 5.75 km/h [61].

3.1.6 Outcomes

Twelve of the studies measured cortisol [25–31, 58–61, 63], and nine studies [25, 26, 28–31, 57, 60, 62] reported HR values, as these indicators are known to represent central pathways of the human stress system (see Sect. 1). With regard to psychological parameters, mood [25, 29–31, 60] (five studies) and anxiety and calmness [29–31, 60] (four studies each) were assessed before and after exposure to the stressor.

3.1.7 Calculation of Stress Reactivity

For calculation of cortisol reactivity, most studies used one of the two formulas suggested by Pruessner et al. [64], providing a certain degree of standardization. Accordingly, cortisol reactivity is reported as the area under the curve (AUC) (from baseline to peak) either with respect to the increase from baseline (AUC_I) or with respect to the ground (AUC_G). A different but comparable approach was chosen by Puterman et al. [63], who used multilevel growth curve modeling. Some of the studies also used the difference between peak and baseline cortisol to define reactivity [26, 29, 59]. HR variations in response to the stressor were typically reported as AUC for the time interval from about 5 min before to 5 min after TSST or as the difference between peak and baseline [62]. For reference, some study designs implemented the measurement of resting HR before the TSST instruction in an upright standing position to standardize the conditions (e.g., Jayasinghe et al. [26], Klaperski et al. [28], Gerber et al. [29]).

Table 1 Influence of physical activity and fitness level on TSST outcomes

Study, location	*N* (M/F); age, years [range (mean)]	Assessment of PA/fitness	Group description	Task	Main outcomes (endocrine, cardiovascular, psychological)
Cross sectional studies					
Childs and de Wit [25], USA	111 (42/69); 18–32 (22.05)	Subjective PA: exercise frequency per week	Sedentary vs. regular exercisers	TSST and non-stressful control task	Cortisol (saliva) → no group differences HR → no group differences Mood → greater decrease in positive affect (elation, friendliness) in non-exercisers
Dockray et al. [58], UK & USA	111 (56/55); 8–13 (10.97)	Subjective PA: CHIP-P questionnaire	Continuous variable (no groups)	TSST-C	Cortisol (saliva) → no association between PA and AUC_I; for girls significant correlation of PA and AUC_I: $r = -0.41$ ($p < 0.05$)
Gerber et al. [29], CH	42 (20/22); 18–31 (21.40)	Objective PA: Accelerometer worn for $M = 5.95$ days (VPA)	Low stress/higher VPA (G1); low stress/lower VPA (G2); high stress/higher VPA (G3); high stress/lower VPA (G4)	TSST	Cortisol (saliva) → group differences in reactivity, with reactivity being highest in G4 and lowest in G1 HR → no group differences State anxiety → no group differences Calmness/mood → group differences in reactivity, with G1 being most calm/having best mood
Jayasinghe et al. [26], AUS	44 F; 30–50 (39.25)	Fitness: VO_{2max} test	High fit vs. low fit (median split)	TSST	Cortisol (blood) → no group difference; no correlation with VO_{2max} HR → no group difference; no correlation with VO_{2max}
Jayasinghe et al. [57], AUS[a]	34 F; 30–50 (39.50)	Fitness: VO_{2max} test	High fit vs. low fit (median split)	TSST	HR → higher reactivity in highly fit women
Klaperski et al. [30], GER	47 F; 18–28 (22.07)	Subjective PA: exercise type, frequency and duration of exercise	Inactive (G1) vs. moderately active (G2) vs. vigorously active (G3)	TSST-G	Cortisol (saliva) → AUC_G differed between groups, being higher in G1 than G2 and G3 HR → average and AUC_G differed between groups (highest in G1) State anxiety → no group differences Calmness/mood → no group differences for calmness; more worsened mood in G3 vs. G1

Table 1 (continued)

Study, location	*N* (M/F); age, years [range (mean)]	Assessment of PA/fitness	Group description	Task	Main outcomes (endocrine, cardiovascular, psychological)
Martikainen et al. [59], FIN	258 (126/132); 8–9 (8.15)	Objective PA: accelerometer worn for mean 5.93 days (overall PA and % of VPA)	Low (G1) vs. intermediate (G2) vs. high overall PA/VPA (G3)	TSST-C	Cortisol (saliva) → overall PA: significant increase only in G1 ($p < 0.001$); lower reactivity in G2 and G3 vs. G1; vigorous PA: significant increase in all groups; lower reactivity in G3 vs. G1 and G2
Puterman et al. [63], USA	46 F; 54–82 (65.41)	Subjective PA: time in vigorous exercise over 3 days	Sedentary vs. active	TSST (modified for elderly)	Cortisol (saliva) → PA did not predict cortisol response; PA moderated the effect of rumination on cortisol response (only in sedentary, high rumination caused an increase in cortisol response)
Rimmele et al. [31], CH	44 M; NR (21.67)	Recruitment-based group differences in endurance training status	Elite athletes vs. untrained men	TSST (modified for athletes)	Cortisol (saliva) → lower cortisol responses in athletes HR → lower HR responses in athletes State anxiety → less state anxiety in athletes Calmness/mood → higher calmness in athletes, less decrease in mood in athletes
Rimmele et al. [60], CH	92 M; 18–32 (24.39)	Fitness: 4 × 1000 m running test (v_{max} and v at 4 mmol/l lactate); subjective PA: frequency and duration of exercise	Elite runners vs. amateur athletes vs. untrained men	TSST	Cortisol (saliva) → significant group differences (lowest cortisol response in elite sportsmen) HR → significant attenuating effect of exercise State anxiety → significant attenuating effect of exercise on state anxiety Calmness/mood → no differences between groups
Strahler et al. [27], GER[b]	115 M; 19–64 (45.70)	Fitness: ergometer test (P_{IAT}/BW)	2- and 3-group-version using age-specific percentiles	TSST-G	Cortisol (saliva) → 2 groups: no group differences; no association between AUC_G or AUC_I and fitness level; 3 groups: significant effect of fitness status
Wood et al. [61], UK	75 F[c]; 18–40 (19.30)	Fitness: HR during a 3 km moderate walk 30 min before TSST	Fit vs. unfit (split at HR = 141)	TSST-G	Cortisol (saliva) → significant lower overall AUC_G in fit group

Table 1 (continued)

Study, location	*N* (M/F); age, years [range (mean)]	Assessment of PA/fitness	Group description	Task	Main outcomes (endocrine, cardiovascular, psychological)
Wyss et al. [62], CH	219 M; NR (20.20)	Fitness: Swiss Army physical fitness test battery; subjective PA: IPAQ	Continuous variable (no groups)	TSST-G	Subjective PA remained insignificant and was not included in the models. HR → positive (enhancing) effect of aerobic fitness (estimated VO_{2max}) and muscle power on HR AUC_G in multiple linear regression ($R^2 = 0.15$); first quartile (highest fitness, high muscle power) with higher reactivity than fourth quartile
Experimental studies					
Klaperski et al. [28], GER	96 M; 19–64 (46.26)	Fitness: ergometer test (P_{IAT}/BW); subjective PA: type, frequency and duration/episode; BSA	Untrained and physically inactive participants	TSST-G	Intervention: 12-week running training program (EG) vs. relaxation training (RG) vs. wait list control (CG); TSST-G and fitness test before (T1) and after (T2) intervention Cortisol (saliva) → within groups: reactivity dropped from T1 to T2 in EG and RG, but not in CG; between groups: higher reactivity improvement in EG vs. CG HR→ within groups: reduction of reactivity from T1 to T2 only in EG; between groups: greater reactivity reduction in EG vs. RG

Studies are presented in alphabetical order

AUC_G area under the curve with respect to the ground, AUC_I baseline-adjusted area under the curve, *AUS* Australia, *BSA* Bewegungs- und Sportaktivität Fragebogen (Physical Activity, Exercise and Sport Questionnaire) [86], *CG* control group, *CH* Switzerland, *CHIP-P* Child Health and Illness Profile—Parent Form, *EG* exercise group, *F* female, *FIN* Finland, *G* group, *GER* Germany, *HR* heart rate, *IPAQ* International Physical Activity Questionnaire, *M* male, *NR* not reported, *PA* physical activity, P_{IAT}/BW power at individual anaerobic threshold/body weight, *RG* relaxation group, *TSST* Trier Social Stress Test, *TSST-C* Trier Social Stress Test for Children [38], *TSST-G* Trier Social Stress Test in Groups [39], v_{max} maximum velocity, VO_{2max} maximum oxygen consumption, *VPA* vigorous physical activity

[a]Subsample of Jayasinghe et al. [26]

[b]Sample overlapping with Klaperski et al. [28]

[c]From the total sample of 164, only 75 performed the fitness task

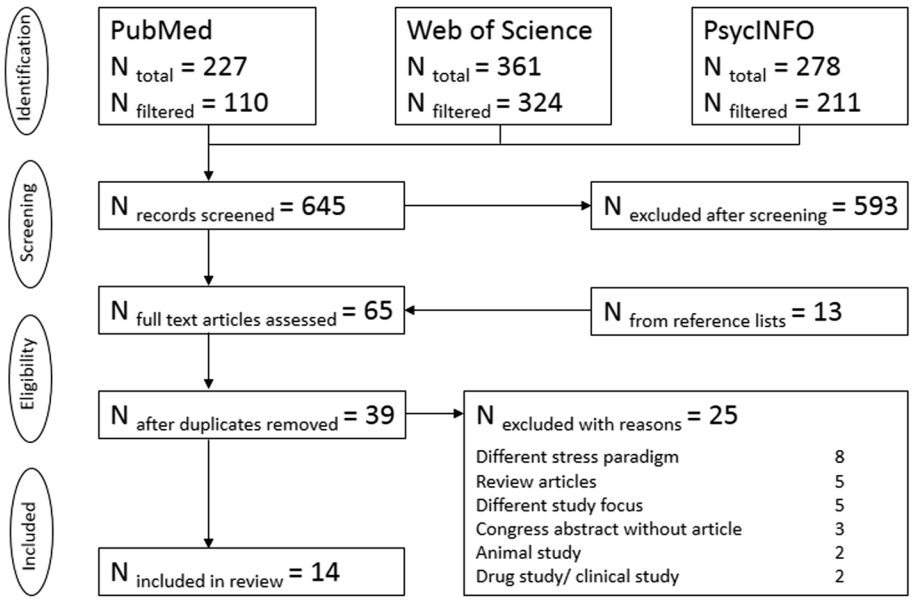

Fig. 1 Flow chart of the different phases of study screening and selection

3.2 General Pattern of Results

3.2.1 Stress Reactivity

In support of the ability of the TSST to induce stress, the measured stress reactivity parameters showed significant changes in response to the TSST in the expected direction across (almost) all studies. Only two exceptions were observed: in Martikainen et al. [59], increases in cortisol levels were detected only in children in the two lower thirds of overall PA, and in Rimmele et al. [31], the stress test did not induce changes with regard to the psychological variable "calmness".

3.2.2 Cross-Stressor Adaptation Hypothesis

With regard to cortisol, seven of twelve studies fully supported the hypothesis of a reduced reactivity in more physically active or fitter participants, pointing towards an association between increased PA and fitness and a reduced reactivity of the HPA axis in response to psychosocial stress [28–31, 59–61]. Three additional studies found at least partial evidence for the CSA hypothesis [27, 58, 63]. In two studies, cortisol reactivity and PA/fitness were unrelated [25, 26].

With regard to HR, four of nine studies detected lower reactivity in more physically active or fitter participants, indicating that participants with higher PA/fitness levels showed a lower reactivity of the ANS to psychosocial stress [28, 30, 31, 60]. However, three studies found no significant effects [25, 26, 29], and Jayasinghe et al. [57] and Wyss et al. [62] even found that fitter participants showed higher HR reactivity than their less fit counterparts.

With regard to psychological variables, two studies supported the hypothesis that higher PA or fitness levels are associated with lower responses in state anxiety [31, 60] and higher calmness [29, 31] in response to the stressor, whereas two other studies found that this was not the case for state anxiety [29, 30] and for calmness [30, 60]. Moreover, three studies found more positive mood responses to the stressor in participants with higher PA or fitness levels [25, 29, 31]; one study found no significant relationship [60], whereas one study [30] found that vigorously exercising participants displayed stronger decreases in positive mood than their sedentary counterparts.

3.3 Potential Moderators

3.3.1 Age

Five of the reviewed studies included participants' age in their statistical analysis. In their sample of 111 children aged 8–13 years, Dockray et al. [58] calculated regression analyses on cortisol reactivity, with age and pubertal stage as additional independent variables, finding that age but not pubertal stage was significantly positively associated with stress reactivity in girls. However, there was no association with age or pubertal stage for boys. Gerber et al. [29] used analyses of covariance adjusted for age only if they showed significant associations with physiological and psychological outcomes; however, in their sample of 42 undergraduate students, no significant associations between age and one of the outcomes were present. Klaperski et al. [28] reported participants' age to be a significant covariate for HR baseline values, but no influence of age was observed on the magnitude of stress reactivity. Puterman et al. [63] replicated all analyses with age as covariate, with unchanged results.

Wood et al. [61] included age as a covariate in their hierarchical multiple regression model but did not report the amount of variance explained by the variable. Nine of the studies did not assess or report the influence of participants' age on their results [25–27, 30, 31, 57, 59, 60, 62].

3.3.2 Sex

With four studies providing information on sex as a potential moderator, our review revealed the following results: For children aged 8–13 years, Dockray et al. [58] found no sex differences in PA and the magnitude of cortisol reactivity, whereas Martikainen et al. [59] found higher levels of overall ($p = 0.01$) and vigorous PA (VPA) ($p = 0.001$) and lower cortisol reactivity ($p = 0.004$) in boys compared with girls. In their sample of young adults, Childs et al. [25] reported significantly higher cortisol ($p < 0.001$, $\eta^2\rho = 0.16$) responses in men than women but no sex differences in HR and mood responses to the TSST. However, in a similar age group, Gerber et al. [29] found no sex differences regarding cortisol, HR, and psychological reactivity. All other studies investigated either only men or women and therefore did not provide insights into sex as a covariate.

3.3.3 Exercise Intensity

In this review, seven studies provided information regarding an influence of different exercise intensities and fitness levels. In four studies, a three-group design with different fitness or VPA levels (e.g., sedentary vs. active vs. vigorously active) was created. In three of them, participants classified into the highest PA/fitness group showed a reduced cortisol reactivity compared with participants with moderate PA/fitness levels, and to an even greater degree compared with physically inactive/untrained participants [30, 59, 60]. However, in one study, this was not the case [27]. Furthermore, negative Pearson correlations were reported between cortisol reactivity and PA (only in girls) [58], indicating a linear inverse relationship. In contrast to the aforementioned findings, Jayasinghe et al. [57] reported a positive linear correlation between HR reactivity and fitness level as represented by the maximum oxygen consumption (VO_{2max}), indicating possible different mechanisms in the ANS compared with the HPA axis. However, Jayasinghe et al. [26] reported no significant association between VO_{2max} and cortisol or HR reactivity, respectively.

3.3.4 Objective vs. Subjective Physical Activity

Studies using objective measures of PA [29, 59] were more likely to find changes in cortisol reactivity, which fit the CSA hypothesis, compared with studies using self-report questionnaires [25, 58, 63]. This was not the case for variables concerning psychological reactivity, with only one study showing mixed results [29].

3.3.5 Physical Activity vs. Fitness

When differentiating between PA ($N = 8$) and fitness ($N = 6$), the following pattern emerged: With regard to cortisol reactivity, no systematic differences were found. That is, five of eight studies investigating effects of PA found evidence in favor of the CSA hypothesis [29–31, 59, 60]. Three studies found no [25] or only partial [58, 63] evidence. Two of four studies investigating effects of fitness found evidence in favor of the CSA hypothesis [28, 61]. Two studies found either partial [27] or no [26] evidence. With regard to HR reactivity, three of five studies found lower HR reactivity in participants with higher PA [30, 31, 60], whereas two found no significant between-group differences [25, 29]. However, for fitness, two of five studies showed elevated HR reactivity in fitter participants [57, 62], whereas two studies found no significant results [26, 27]. In contrast, one randomized controlled trial reported lower HR reactivity in participants who engaged in 12-week fitness training compared with a control group [28]. None of the fitness-related studies investigated psychological stress reactivity.

3.3.6 Psychological Covariates

Four studies within this review investigated the influence of psychological factors on the relationship between PA/fitness and stress reactivity. Puterman et al. [63] found that, in women aged 54–82 years, rumination seemed to alter this relationship in the sense that, in less active women, only those with high scores in rumination showed elevated cortisol reactivity, whereas the more active participants exhibited values similar to those of the sedentary low ruminators. Rimmele et al. [60] investigated the stress reactivity of 18 elite and 50 amateur athletes compared with 24 untrained men and reported that the personality trait of competitiveness did not modulate stress reactivity in their sample. Wyss et al. [62] investigated potential moderating effects of the so-called Big Five personality traits (neuroticism, extraversion, openness, agreeableness, and conscientiousness) and found no influence on the cardiac response to the TSST. Finally, Gerber et al. [29] used a four-group design, with groups defined by combining high versus low objectively measured VPA with high versus low perceived stress during the last month. They showed, in a sample of 42 undergraduate students, that those with high chronic stress and low VPA displayed the highest cortisol reactivity and the

greatest decrease in calmness and mood, whereas students with low chronic stress and high VPA showed the lowest cortisol reactivity and the least decrease in calmness and mood, underlining the potential of subjective stress perception over a longer period of time to moderate the impact of PA on the stress response.

4 Discussion

The purpose of this systematic review was to evaluate the association between regular PA and cardiorespiratory fitness and stress reactivity and to provide an overview of moderators of this relationship that have been examined in prior investigations. In contrast to previous reviews, only studies that used the TSST to experimentally induce stress were included. The TSST is a highly effective standardized socio-evaluative stressor task that can provoke stronger stress reactions than other (e.g., cognitive) tasks.

Our review shows that, in seven of twelve of the studies, higher PA or fitness levels were associated with an attenuated stress reactivity of the HPA axis. The pattern of reactivity of the ANS was less clear: Four of nine studies supported the CSA hypothesis, whereas three studies found no connection and two other studies showed an increased reactivity in participants with higher PA/fitness. With regard to psychological stress reactivity, two of four studies found less severe anxiety and loss of calmness in more physically active/fit participants, and three of four studies showed more positive mood in more physically active/fit than in less active/fit participants.

Furthermore, preliminary evidence suggests a dose–response relationship, indicating that differences compared with inactive participants are stronger among participants with high PA levels than in those with moderate PA levels. Nevertheless, most evidence is derived from cross-sectional analyses, which precludes conclusions about cause and effect. However, one experimental study (randomized controlled trial by Klaperski et al. [28]) suggested that regular exercise training may indeed lead to a reduced reactivity in response to psychosocial stress. The results of the outcomes addressed in this review are discussed separately in the following sections.

4.1 Cortisol

The reactivity of the HPA axis to acute stress is measured by the glucocorticoid hormone cortisol. The present review confirmed that this parameter reacts sensitively to differences in PA/fitness. Thus, the CSA hypothesis was supported in six cross-sectional studies and one randomized controlled trial. Nevertheless, five studies found no or only limited associations. More specifically, one study showed that, in older women, the variables were only associated in the presence of "rumination" as a moderator, meaning that, in less active women, only those with high scores in rumination showed elevated cortisol reactivity [63]. In Jayasinghe et al. [26], the absent association could be explained by the relatively high fitness scores in the low-fitness group, which precluded observation of the differences that would be expected in a truly low-fit sample. This is the only study in this review measuring total blood cortisol instead of salivary free cortisol. According to the free hormone hypothesis, only the unbound (free) cortisol is considered biologically active [65]. In blood cortisol, the rate of free cortisol is only 6–30%. A comprehensive overview of the issue is given by Levine et al. [66]. Moreover, Dockray et al. [58] reported lower PA-related cortisol reactivity only in girls. However, Martikainen et al. [59] conducted a larger study with participants within the same age range and observed no sex-related differences.

At least two other reasons exist to explain why the CSA was not supported in all studies. As demonstrated by Wolfram et al. [67], cortisol reactivity in response to the TSST and to a real-life stressor is only weakly associated. Following Campbell and Ehlert [68] and Roy [69], emotional involvement might be limited during experimentally induced stress, because failure has no real negative consequences for the participants. In line with this notion, Zanstra and Johnston [70] argued that participants might show less strong stress reactions during laboratory stress than during real-life stress. Another factor might be insufficient statistical power. Based on estimations with G*Power software, we found that at least 128 participants are needed to detect moderate between-group differences (Cohen's $f = 0.25$) via analyses of covariance (assuming an alpha error probability of 0.05 and a power of 0.80). As shown in Table 1, only two studies had samples with more than 128 participants [59, 62]. Thus, the majority of the studies did not have sufficient statistical power to detect effects of moderate magnitude. In light of these limitations (which also apply to the other outcomes), it is all the more noteworthy that the CSA hypothesis was supported in almost 60% of all studies.

4.2 Heart Rate

Unlike the HPA axis, the ANS appears to show a more diverse reaction to acute psychosocial stress. Five of nine of the studies did not find the expected reduced HR reactivity in physically more active and fit participants. Beyond that, two studies reported an association in the opposite direction [57, 62]. By contrast, four studies reported lower HR reactivity in highly trained and more active participants. This indicates that a cross-stressor adaptation not only might result in adaptation in the sense of habituation but under certain circumstances may also have a sensitization effect on HR

reactivity [71]. Interestingly, Wyss et al. [62] reported the aforementioned changes in reactivity for cardiorespiratory fitness and muscle power but not for balancing, indicating a possible moderating effect of the type of fitness on HR reactivity.

Given that higher cardiovascular fitness levels are generally related to lower resting HR, one could argue that this might have confounded the results. In fact, HR baseline differences between fit and unfit (or physically active and inactive, respectively) participants were found in six of nine studies [25, 28, 30, 57, 60, 62]. However, all studies controlled for this potential bias either by using the AUC_I or baseline minus peak to calculate reactivity or by including baseline HR as a covariate. Nevertheless, as highlighted by Jayasinghe et al. [57], it is still conceivable that a "ceiling" effect prevented participants with a high baseline HR from showing higher reactivity levels.

Moreover, little is known so far about the mechanisms underlying individual differences in physiological stress reactivity. Lovallo [72] created a model with a special focus on stress reactivity regulating brain structures. They argued that dysregulation on the central nervous level (prefrontal cortex, limbic system, hypothalamus, and brain stem), in particular, contributes to poor behavioral homeostasis. Lovallo suggests that "stress reactivity ranging from very low to very high has a normative midrange of intensity and present evidence that negative health outcomes may be associated with both exaggerated and diminished stress reactivity since both tendencies imply a loss of homeostatic regulation" [72, p. 121]. Phillips et al. [73] reported that not only a strongly elevated but in some cases also a (neurally based) blunted stress reactivity might correspond to negative health outcomes. This could explain why Wyss et al. [62] and Jayasinghe et al. [57] obtained results at odds with the CSA hypothesis. As Wyss et al. [62] argued, their results would indicate a shift from an unhealthily low to a normal, healthy stress reactivity. But it remains unclear which magnitude of the individual stress reactivity can be considered healthy and how inter-individual differences can be identified, and their assumption does not explain why about 40% of the studies (including one randomized controlled trial) found evidence supporting the traditional CSA hypothesis. One possible conclusion would be that the CSA hypothesis should be adjusted to state that repeated exercise does not necessarily reduce stress reactivity but instead contributes to a normalization to healthy levels that allow the person to improve their homeostatic regulation. However, as the results of our review suggest, this "range of optimal reactivity" explanation seems to be more evident for adaptations of the SAM system but does not reflect current results of the HPA axis reactivity to psychosocial stress.

4.3 Psychological Stress Reactivity

Earlier studies showed that a physiological adaptation might not necessarily be consistent with the pattern and intensity of psychological stress perception [74]. Accordingly, some of the reviewed studies also collected psychological stress parameters. Findings pointed towards positive effects of PA/fitness on reactions to stressful situations (reduced anxiety, improved mood and calmness), with some inconsistencies. In Klaperski et al. [30], changes in mood in athletes were worse than in non-athletes, which the authors attributed to greater competitiveness and achievement motivation in athletes. However, Rimmele et al. [60] found that competitiveness did not moderate stress reactivity. Both studies used similar assessment approaches; reasons for the contrasting findings remain unclear. According to a review by Campbell and Ehlert [68], dissociation between physiological and psychological stress reactivity is an often observed phenomenon and potentially influenced by assessment features, psychological traits and states, and physiological dispositions. Therefore, the assessment of potential moderating factors is crucial.

4.4 Potential Moderators

According to the CSA hypothesis, exercise acts as a stressor itself and leads to beneficial generalized adaptations of the stress systems. Sufficient exercise intensity seems to be a precondition: According to Hackney [44], a minimum exercise intensity of 50–60% of VO_{2max} must be reached to elicit cortisol responses to exercise and thus generate adaptations that would match those required by the CSA hypothesis. The direct dependence of cortisol levels on exercise intensity (during exercise) [44] leads to the assumption that there might be a dose–response relationship regarding the influence of exercise on stress reactivity. The results of this review mostly corroborate this assumption. Rimmele et al. [60] showed that ambitious athletes benefit more in terms of reduced stress reactivity than do recreational athletes. Klaperski et al. [30] found a similar pattern in women. Martikainen et al. [59] demonstrated in a sample of children that differences in VPA, but not moderate PA, account for a reduced reactivity to stress, supporting the hypothesis that a higher exercise intensity is related to more pronounced positive effects on stress reactivity. Nevertheless, prospective studies comparing different exercise intensities are needed to draw more reliable conclusions.

Higher age, e.g., in relation to the amount and intensity of past stressful life events, might contribute to blunted stress reactivity [73, 75]. Strong evidence points towards changes in HPA axis activity with progressing age [34]. However, within this review, only one study reported an influence of participants' age on stress reactivity, and only in girls [58].

More than half of the studies did not include age in their statistical analysis. This might be partly because most of the studies included only participants within a specific age range (e.g., 18–30 years), which might not be sufficient to detect age-specific differences. The study by Dockray et al. [58] was conducted with children aged 8–13 years, an age associated with many behavioral and hormonal changes. This might explain why only these researchers detected significant influences of age on stress reactivity. We therefore suggest that future studies should consider a wider age range in their inclusion criteria to more systematically examine the influence of age on stress reactivity.

Our review revealed that, in the majority of studies, the authors tested either men or women. This likely indicates that sex differences were anticipated and, where possible, avoided as a potential confounding factor. However, within this review, studies that included both male and female participants showed inconsistent results for children [58, 59] and young adults [25, 29]. Interestingly, whereas Martikainen et al. [59] found lower cortisol reactivity in boys than in girls, Childs et al. [25] reported the opposite for young men and women, indicating a possible age-dependent alteration of the influence of sex on cortisol reactivity to acute stress. In their review from 2006, Kajantie et al. [76] focused on sex differences in HPA axis responses to acute psychosocial stress, in the aggregate showing that, between puberty and menopause, women normally show lower reactivity than men of the same age. However, menstrual cycle, intake of oral contraceptives, and pregnancy can alter women's cortisol reactivity [76, 77]. Possible underlying mechanisms that have been investigated in earlier research include sex-specific differences (in premenopausal women and men of similar age) regarding the following hormonal properties: adrenal responsiveness to adrenocorticotropic hormone (ACTH), resulting in different secretion rates of cortisol in the adrenal cortex; production rate of arginine vasopressin (AVP) and HPA axis responsiveness to AVP, which is known to potentiate corticotropin-releasing hormone (CRH)-evoked ACTH release in the pituitary and also directly stimulate cortisol secretion; corticosteroid-binding globulin (CBG), which influences the proportion of circulating unbound, metabolically active cortisol and whose production is stimulated by estrogen; and general sex differences in testosterone and estrogen concentration, whose multiple interactions on a central nervous level are still not fully understood [76]. With regard to children's HPA axis reactivity, a recent review focusing on this topic reported higher reactivity in girls than in boys in a majority of studies [78]. Differences were also explained by possible interactions between the HPA axis and the hypothalamic–pituitary–gonadal (HPG) axis.

As already noted, psychological covariates may play a role in regulation of stress reactivity. Within this review, rumination, agreeableness, extraversion, and stress perception within the last month were shown to moderate the relationship between PA and stress reactivity, whereas competitiveness, neuroticism, openness, and conscientiousness were not found to be involved. According to Bibbey et al. [45], cortisol and cardiovascular stress reactivity are consistently associated with a number of personality traits. In a large middle-aged cohort ($N = 352$), they showed that participants with higher neuroticism scores showed lower cortisol and HR reactivity, and greater agreeableness and openness were associated with higher cortisol and HR reactivity. However, in the studies reviewed by Kudielka et al. [41], personality only influenced stress reactivity after repeated exposure to the TSST. In conclusion, results on psychological covariates are inconsistent and mechanisms remain unclear.

4.5 Strengths and Limitations

A strength of this systematic review is the specific focus on the TSST. While other reviews included a great variety of stressor tasks with different grades of effectiveness and possibly different effects on the human stress system, we concentrated on a stressor task that, compared with other known laboratory stressors, typically triggers a more than twofold increased cortisol reaction and has therefore become the most widespread psychosocial stressor task. Thus, we excluded one factor of potential heterogeneity and decreased the likelihood of a beta error. A second strength is the differentiation between markers of HPA axis, the ANS, and psychological parameters. This allows a more precise analysis of the effects of PA/fitness on the different pathways of stress reactivity. Lastly, within this review, an analysis and discussion of potential moderating factors is offered.

However, some potential limitations need to be taken into consideration. First, some characteristics in terms of study design—for instance, measurement of PA/fitness—still varied across studies. Moreover, in some cases, PA and exercise were not clearly differentiated or were used synonymously [26, 60, 63]. PA was measured variously via validated questionnaires, via self-reported frequency, duration and/or time of exercise (excluding activities of daily living), or, more reliably, objectively via accelerometry [29, 59]. Fitness levels were determined using (spiro)ergometry [26–28, 57], a multilateral fitness test designed for the Swiss army [62], a 4×1000 m running test at increasing subjective exertion [60], and a 3 km walking task at 5.75 km/h [61]. While ergometer tests allow a more standardized measurement of fitness status, the latter two correspond more to participants' real-life situations.

Second, cut-offs for differentiation between groups with high and low PA or fitness levels varied across studies, depending on sample characteristics, chosen outcome variables, and study designs. Three different approaches

were identified: institutional recommendations for minimum weekly PA were applied [29, 61, 63], a median or tertiary split was performed [26, 27, 57, 59], or arbitrary cut-offs were used [25, 30, 31, 60]. Therefore, the different approaches for measurement and categorization of PA/fitness levels mean that inter-study comparisons of PA/fitness are limited to some extent.

Third, because of changes in cortisol secretion through oral contraceptives and throughout menstrual cycles, obtaining valid cortisol samples in women is challenging [77, 79]. Several strategies were used to control this factor, including exclusion of intake of oral contraceptives [25, 30], controlling for menstrual phase in statistical analyses [61], or scheduling all women during the same menstrual phase (mid follicular: Jayasinghe et al. [26]; luteal: Gerber et al. [29], Klaperski et al. [30]). These different approaches might lead to inconsistent results concerning cortisol reactivity in women.

Fourth, we acknowledge that different versions of the TSST were used in the studies. However, all versions are structured identically (preparation phase, public speech, mental arithmetic) and elicit similar stress reactions [38, 39], reducing the likelihood of a potential bias attributable to different TSST versions.

Fifth, as mentioned in Sect. 2.2, we focused only on significance of study results, meaning that findings were only considered as supporting the CSA hypothesis if they were statistically significant. This might have caused a bias, since the likelihood of identifying significant results is greater in larger samples. As mentioned, most of the studies were underpowered, leading to a relatively conservative interpretation of the current state of the art.

Sixth, while stress reactivity was used as an outcome in the present review, we acknowledge that some scholars have argued that recovery from stress is just as important as stress reactivity [80]. Stress recovery is generally defined as the time elapsed between peak reactivity and return to baseline [81]. However, we decided not to consider stress recovery for the following reasons. (1) Recovery is highly dependent on the peak stress reaction. If a person shows a stronger stress reaction, his/her organism may also need more time to return to baseline. Thus, recovery is to some degree confounded by stress reactivity. (2) In most studies, cortisol concentrations were only assessed in 15-min intervals during the recovery phase, which makes it difficult to establish the exact time at which cortisol levels have returned to baseline. (3) The recovery period was relatively short in some studies, so cortisol concentrations were unlikely to have returned to baseline.

Lastly, no meta-analytical techniques were applied to calculate a summary effect over the individual studies as some of the limitations mentioned above have contributed to a large heterogeneity between studies (e.g., different designs, outcomes, and sample populations). In such a case, examination of the source of heterogeneity rather than calculating a summary effect is recommended [82]. However, the small number of studies do not allow subgroup analysis/meta-regression to further investigate potential moderators.

5 Conclusions and Future Perspectives

Despite methodological differences, 58% of the studies included in this review suggested that higher PA/fitness was associated with an attenuated adrenocortical stress response. Although less marked, a similar pattern was observed for the ANS and for psychological stress reactivity. Some evidence points towards a more reduced stress reactivity with increasing exercise intensity. Study results partly support the notion of an optimal stress reactivity in cardiovascular parameters in particular, and higher PA and better fitness contribute to gaining or maintaining this status.

Elevated or impaired stress reactivity is associated with a variety of health issues, including higher risk of cardiovascular disease [83], musculoskeletal problems [84], or depression [1], with potential negative consequences for health systems and the economy of a country. As our review suggests, people with higher PA/fitness levels may react less strongly than their less active/fit counterparts if exposed to acute stress. However, currently, as most evidence is based on cross-sectional analyses, evidence remains insufficient to draw definite conclusions regarding the question of whether stress reactivity can be deliberately improved by exercise training or by encouraging people to integrate more PA into their daily lives. Therefore, there is a great need for further studies with longitudinal or experimental designs. Future studies should also include health outcomes and examine whether additional factors moderate the relationship between PA/fitness and stress reactivity (e.g., socio-economic status, chronic stress, specific personality traits, influence of genetic factors, or the gene–environment interaction). However, the inclusion of moderating factors should be guided by specific hypotheses and based on theoretical considerations. This is important to mention because, so far, the selection of moderators seems quite arbitrary. Moreover, it is noteworthy that all existing studies were conducted either in the USA or in European countries and mostly focused on Caucasian populations. We therefore suggest that more research is needed in other regions and with more diverse ethnic populations to examine the generalizability of the findings. Furthermore, more basic research is required on underlying mechanisms, especially regarding processes in the brain (e.g., interactions between stress, exercise, and brain structures, such as prefrontal cortex, hippocampus, and amygdala [85]) and genetic factors, which are still not fully understood.

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