

# Does the level of physical exercise affect physiological and psychological responses to psychosocial stress in women?

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## ABSTRACT

**Objectives:** To test the Cross-Stressor Adaptation hypothesis for females by examining whether physically exercising young women show reduced physiological and psychological stress responses to a psychosocial stressor.

**Design:** Forty-seven healthy young women with different levels of physical exercise (17 not or rarely exercising, 15 moderately exercising, 15 vigorously exercising) underwent the Trier Social Stress Test for Groups (TSST-G); physiological and psychological stress responses during and after stress induction were compared.

**Method:** ANOVAs with repeated measures were used to compare stress reactivity and recovery between the three exercise groups. Heart rate and salivary free cortisol were used as indicators of physiological stress response, state anxiety, mood, and calmness as indicators of psychological stress response. For physiological stress reactivity, the areas under the curve with respect to the ground ( $AUC_G$ ) were compared.

**Results:** In all three exercise groups, experimentally induced stress led to a significant rise in heart rate, cortisol, and state anxiety; mood and calmness significantly decreased. As hypothesized, the pattern of the physiological stress response differed for the three exercise groups, with lowered reactivity in the more active groups. However, the psychological stress response partly went in the opposite direction: Exercising participants reported a higher mood decrease, suggesting a dissociation of the physiological and psychological stress responses.

**Conclusions:** The findings suggest that the Cross-Stressor Adaptation hypothesis is also valid for young women; however, only with regard to physiological stress response. The unexpected findings for psychological stress response need to be further explored in experimental studies.

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## Introduction

There is profound evidence suggesting that regular physical exercise is beneficial not only for physical health but also for mental health (Raglin & Wilson, 2012). In particular, physical exercise has been found to exert antidepressant and anxiolytic effects that are clinically relevant (Rethorst, Wipfli, & Landers, 2009). However, although stress is one of the major threats for physical and mental health (Chrousos, 2009), surprisingly little is known about the stress-regulatory role of physical exercise (Fuchs & Klaperski, 2012). In the literature, one of the most discussed assumptions on how

physical exercise might influence the stress-coping system is the so-called “stress-buffer hypothesis of physical exercise.” Physical exercise is thought to act as a moderator of the stress-health relationship by reducing the detrimental effects of chronic stress (e.g., at the workplace) on physical and mental health, or in other words by “buffering” the negative health effects of stress (Gerber, Kellmann, Hartmann, & Pühse, 2010; Tsatsoulis & Fountoulakis, 2006). However, only a small majority of studies conducted on this topic found full or at least partial support for the stress-buffering hypothesis (Gerber & Pühse, 2009; Klaperski, Seelig, & Fuchs, 2012).

Several authors cite the “Cross-Stressor Adaptation hypothesis” (CSA hypothesis) as a possible mechanism for the stress-buffering effect of physical exercise (Hamer, Taylor, & Steptoe, 2006; Sothmann, 2006). The CSA hypothesis states that regular exercise leads to biological adaptations which contribute to a reduced

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physiological reaction of the sympathetic nervous system (SNS) and the hypothalamic–pituitary–adrenal (HPA) axis to stressors in general (Hamer et al., 2006; Tsatsoulis & Fountoulakis, 2006). A reduction in physiological stress reaction is reflected by (a) a lower stress *reactivity*, i.e. the deviation from the physiological baseline is smaller, and (b) a faster stress *recovery*, i.e. less time is needed to return to the physiological baseline (Forcier et al., 2006). Chronic hyper-elevated activations and slow recovery of the stress-systems have a negative impact on health (Carroll, Lovallo, & Phillips, 2009; Chrousos, 2009), and a high reaction to laboratory stressors is associated with a higher risk of stress-related diseases (Chida & Steptoe, 2010; Ellenbogen, Hodgins, Walker, Couture, & Adam, 2006). A confirmation of the CSA hypothesis would emphasize the importance of physical exercise for health, as it would result in a health-protective lower physiological reactivity and faster physiological recovery from stressful events (Kemeny, 2003; Tsatsoulis & Fountoulakis, 2006). So far it is unclear whether an exercise-induced attenuated physiological stress reaction is paralleled by a lowered psychological stress reaction (e.g., reduced anxiety).

There are conflicting empirical findings regarding the effects of physical exercise (and fitness) on reactivity and recovery in psychosocial stress situations (Dishman & Jackson, 2000). First of all, several cross-sectional studies with non-clinical samples demonstrated that regularly exercising or fit persons show reduced physiological stress reactions and partly reduced psychological stress reactions when confronted with experimentally induced psychosocial stressors. Rimmele et al. (2007), for example, found a lower heart rate reactivity and a reduced salivary free cortisol response along with a smaller mood decline and a tendency for a greater increase in anxiety in elite sportsmen compared to untrained men in response to the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), a standardized stress-induction protocol. More recently, Rimmele et al. (2009) replicated and extended the results of this study and showed that amateur sportsmen also had a significantly lower heart rate *reactivity* than untrained men, but that both groups had a significantly greater cortisol response than elite sportsmen; however, no *recovery* differences were found between the three groups. Furthermore, throughout the study the highest anxiety levels were observed in untrained men and the lowest in elite sportsmen. However, no significant time by group interactions emerged (Rimmele et al., 2009). Conversely, other cross-sectional studies did not find differences in stress response between trained and untrained men (e.g., Moyna et al., 1999).

More profound support of the CSA hypothesis stems from randomized controlled training studies (RCT) with non-clinical samples. Spalding, Lyon, Steel, and Hatfield (2004), for instance, found a reduced cardiovascular stress reactivity and better recovery from a mental arithmetic stressor with auditory distraction in males and females who had completed a six-week aerobic exercise program, compared to a weight training and a non-intervention group. However, other RCT studies showed no such or rather ambiguous effects (e.g., de Geus, van Doornen, & Orlebeke, 1993). Meta-analyses focusing on cardiovascular stress responses to psychosocial stress (Forcier et al., 2006; Hocking Schuler & O'Brien, 1997; Jackson & Dishman, 2006) found physically active persons to possess a better cardiovascular *recovery* after stress exposure. However, while Forcier et al. (2006) state that physical exercise is also linked to a lower cardiovascular *reactivity*, the meta-analysis from Jackson and Dishman (2006) did not support this assumption. In the only meta-analysis to also analyze endocrine stress response, van Doornen and de Geus (1993) concluded that there is no link between physical fitness and hormonal components of the stress response. Sothmann (2006) summarized the state of research in this area by reasoning that “based on the neuroendocrine data

one cannot substantively argue for an impact of exercise training in augmenting or reducing stress reactivity in psychosocial settings” (p. 156). The author calls for more sophisticated experimental designs and more research with currently underrepresented female samples.

Two of the rare studies with women are from Summers, Lustyk, Heitkemper, and Jarrett (1999) and Traustadóttir, Bosch, and Matt (2005). Summers et al. (1999) did not find any differences between fit and unfit women in the catecholamine response (pre and post urine sample) to a mild psychological stressor (Stroop Color–Word Test) in the follicular phase. In contrast, Traustadóttir et al. (2005) showed that higher physical fitness in post-menopausal women was linked to a lower endocrine stress response (plasma cortisol). However, findings focusing on different stress responses from different age groups and genders must be compared carefully, as age and gender strongly affect physiological and psychological stress response (see Jackson & Dishman, 2006; Kudielka, Hellhammer, & Wüst, 2009). Kelly, Tyrka, Anderson, Price, and Carpenter (2008), for example, found women to report greater psychological stress than men on the TSST, although both groups showed similar physiological stress responses. These findings also illustrate that caution must be exercised when transferring conclusions for physiological stress response to psychological response or vice versa. According to Campbell and Ehler's (2012) review of the empirical evidence, physiological and psychological stress responses are often not, as usually assumed, interrelated; rather, they found “dissociations” of the stress responses to be a common phenomenon in studies applying the TSST to examine stress responses.

In sum, there is some evidence in support of the CSA hypothesis that regular exercise results in a more beneficial cardiovascular and endocrine stress response. However, less is known about exercise-induced changes in psychological stress responses and about how these psychological reactions relate to physiological reactions. We identify two main limitations of the current research on the CSA hypothesis: (1) Most studies were conducted with men, and thus more research is needed with female samples. (2) The majority of previous studies used non-standardized and non-validated methods of stress induction, hampering comparability and raising the question whether stressors were appropriate (ecologically valid) for detecting differences in stress responses. Hence, we set out to test the CSA hypothesis in a sample of young women with different levels of physical exercise by comparing their physiological (heart rate, salivary free cortisol) and psychological (anxiety, calmness, mood) stress responses to a standardized psychosocial laboratory stressor in a group format (TSST for Groups; von Dawans, Kirschbaum, & Heinrichs, 2011).

## Methods

### Participants

Fifty women were recruited by advertisements and personal address at the University of Freiburg, in local sporting teams, and at local sports clubs in Freiburg, Germany. The participants were between 18 and 28 years of age, reported that they engaged regularly in at least 3 h of exercise<sup>1</sup> a week or did not exercise regularly at all, did not take any hormonal contraceptives (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999), and did not smoke more than five cigarettes per day. Further exclusion criteria in the study were symptoms of psychopathology, reported medical

<sup>1</sup> The term “exercise” covers all forms of sporting exercise but does not cover daily physical activity, e.g., housework.

**Table 1**  
Description (mean, SEM) of the study groups.

Variables	Inactive ( <i>n</i> = 17)	Moderately active ( <i>n</i> = 15)	Vigorously active ( <i>n</i> = 15)	<i>p</i>
Age (years)	21.41 ± 0.37	23.33 ± 0.77	21.47 ± 0.71	<i>p</i> = .06
Body Mass Index (kg/m <sup>2</sup> )	22.09 ± 0.83	21.23 ± 0.37	21.34 ± 0.39	<i>p</i> = .54
Chronic stress (TICS <sup>a</sup> )	1.22 ± 0.11	1.10 ± 0.17	1.19 ± 0.15	<i>p</i> = .83
Trait anxiety (STAI <sup>b</sup> )	36.94 ± 1.65	39.47 ± 2.07	37.73 ± 2.44	<i>p</i> = .67
Symptoms of psycho-pathology (BSI–GSI <sup>c</sup> )	0.38 ± 0.06	0.34 ± 0.05	0.31 ± 0.07	<i>p</i> = .72
Physical exercise (min/week)	22.02 ± 8.35	246.51 ± 13.96	684.65 ± 46.52	<i>p</i> < .001

Note. Data are expressed as mean ± SEM.

<sup>a</sup> TICS = The Trier Inventory for Chronic Stress.

<sup>b</sup> STAI = State Trait Anxiety Inventory.

<sup>c</sup> BSI–GSI = Brief Symptom Inventory–Global Severity Index.

illness, an irregular menstrual cycle, medication intake or use of cortisone compounds and substance abuse, the study of psychology, prior participation in a similar stress experiment, lack of fluency in German. Three of the original 50 subjects did not meet the eligibility criteria and were excluded from the analysis: One subject proved in the study session to not being able to speak German fluently and two other subjects met criteria for a mental health disorder on the basis of the Brief Symptom Inventory (Franke, 2000). Results are therefore reported for the remaining *N* = 47 women. Due to technical problems, we did not record the heart rate of one subject. Moreover, although all participants were scheduled for assessment in the luteal phase,<sup>2</sup> the verification of the actual length of the cycle (see Procedure) revealed that 16 participants were not tested in the luteal phase or had irregular cycles that paused or lasted longer than 48 days, leaving *n* = 31 women eligible for the cortisol analysis. In the course of the study session, detailed exercise behavior was assessed: The subjects named a maximum of four exercise types they regularly engaged in and indicated the frequency (per month) and duration (per episode) for each activity (Fuchs, 2012). Based on the self-reported amount of regular physical exercise and on the exercise classification Rimmele et al. (2007, 2009) used, subjects were classified as either inactive (engaging in less than 2 h of exercise per week),<sup>3</sup> moderately active (engaging in 2–6 h of exercise per week), or vigorously active (engaging in more than 6 h of exercise per week). In the overall study sample, 17 women were inactive (mean ± SD age: 21.4 ± 1.54), 15 women were moderately active (mean ± SD age: 23.3 ± 2.97), and 15 women were vigorously active (mean ± SD age: 21.5 ± 2.75). The type of exercise the participants engaged in varied within but not between the groups, with the majority of the subjects engaging in ball sports (e.g., basketball), endurance (e.g., jogging) or fitness-orientated (e.g., aerobics) exercise. Characteristics of the sample are presented in Table 1.

### Procedure

In a telephone interview prior to the laboratory session, we screened all participants for eligibility and asked them when the first day of their last menstruation was in order to schedule them during the luteal phase, where the free salivary cortisol reactivity reaches its maximum and is comparable to the male response (Kirschbaum et al., 1999). Participants were also instructed to refrain

from exercising, taking any medication, and drinking alcohol or coffee 24 h prior to the study session, to have regular breakfast and lunch but to refrain from eating 2 h prior to the study session, and to not come in haste. Participants completed the study in random groups of three. All study sessions lasted for 2 h and took place between 2:15 p.m. and 7:00 p.m.,<sup>4</sup> allowing us to control for diurnal variations of cortisol release (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). Before participation, all subjects provided written informed consent and were informed of their right to discontinue the study at any time and withdraw their consent afterward. Psychosocial stress was induced by the Trier Social Stress Test for Groups (TSST-G; von Dawans et al., 2011), which was adapted for study purposes to groups of three instead of six participants. The TSST-G is a standardized motivated performance task protocol which reliably and validly induces psychosocial stress (von Dawans et al., 2011): After a preparation period, subjects deliver a free speech and complete a mental arithmetic task in front of a camera and two judges who withhold any feedback, combining high levels of uncontrollability and socio-evaluative threat (Dickerson & Kemeny, 2004).

As depicted in Fig. 1, a study session comprised a preparation period (45 min), a presentation period (14 min), and a resting period (60 min). During *preparation*, participants were randomly assigned to one of three tables as they arrived and requested not to communicate with each other. They were further asked to either drink a standardized drink of grape juice (250 ml) or eat 25 g of grape sugar (to obtain similarly high levels of blood glucose), after which we carried out initial psychological and physiological measures. Then the participants were introduced to the upcoming TSST-G task (for details, see von Dawans et al., 2011). In the *presentation period* subjects were led into another room, where they stood separated by dividing walls in front of a camera and two women representing the selection committee. Each participant was given 3 min for her speech. Afterward, the subjects did an unexpected serial subtraction task (three 30 s turns). More details on the TSST protocol for groups of six are described elsewhere (von Dawans et al., 2011). During the *resting period*, participants were thanked by the committee and instructed to sit down and fill out questionnaires. After 5 min the investigator reentered the room and guided the subjects back to the first room, where they were instructed to sit quietly for 60 more minutes until saliva sampling was completed and to fill in some additional questionnaires. At the end of the study session the selection committee entered the room, the women were debriefed, thanked, and paid 10 Euros for their participation. They were further asked to report the beginning of their next menstruation. The procedure was approved by the ethical review board of the University of Freiburg.

<sup>2</sup> Women were classified as being in the luteal phase when the examination took place in the 2nd half of the cycle as well as a maximum of 15 days and no less than one day before the next menstruation (Raith-Paula, Frank-Herrmann, Freundl, & Strowitzki, 2008).

<sup>3</sup> Due to our recruitment strategy, the women in this group reported predominantly that they did not exercise at all (*n* = 11). However, six women reported small amounts of exercise; in order not to exclude any eligible subjects, we placed all inactive and hardly active women in one group, which we refer to as “inactive”.

<sup>4</sup> Time and day of measurement varied to the same extend within all three exercise groups.

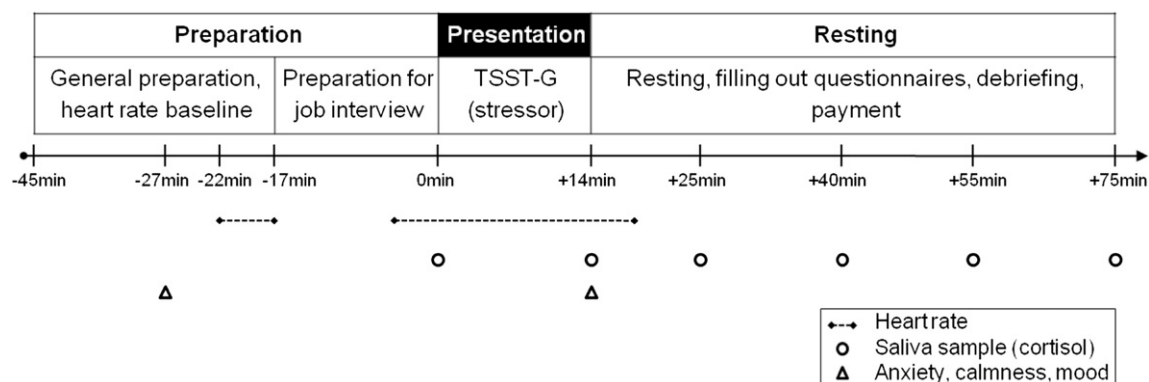


Fig. 1. Study design: Sequence of events and measurements on timeline.

### Physiological measures

We used heart rate as an indicator of the autonomic stress response and salivary free cortisol as an indicator of the endocrine stress response. Heart rate (beats/min, bpm) was monitored continuously at 5-s intervals from 5 min before the stress onset until 5 min after cessation of the stressor. For analyses, aggregated 1-min intervals were computed. Additionally, the participants' heart rate baseline was assessed in an upright standing position about 23 min–18 min before the stress task; the mean of this 5 min interval was used as the individual heart rate baseline value. For all heart rate assessments, a wireless chest heart rate transmitter with a wrist monitor recorder (Polar RS400X, Polar Electro, Finland) was used.

Cortisol release is a valid indicator for HPA activity as a response to an acute psychosocial stressor, especially when psychosocial stress is induced by a performance task containing social-evaluative threat and uncontrollability (Dickerson & Kemeny, 2004). The free, biologically active cortisol fraction in the blood can be reliably and validly assessed through the measurement of salivary free cortisol – a non-invasive method for assessing cortisol levels repeatedly. Salivary free cortisol gradually increases within about 10 min, with its peak around 10–30 min after stressor cessation (Foley & Kirschbaum, 2010). We collected six saliva samples from each participant before (–1 min relative to stressor onset) and after stress exposure (+14, +25, +40, +55, +60 min; Fig. 1) using a commercially available sampling device (Salivette®; Sarstedt, Germany). Saliva samples were stored at –20 °C and sent to Dresden LabService GmbH (Germany) for biochemical analysis of free cortisol concentration. Saliva samples were thawed and spun at 21 °C at 3000 rpm for 3 min to obtain clear saliva; cortisol concentrations (nmol/l) were determined by a luminescence immunoassay for the in-vitro-diagnostic quantitative determination of cortisol in human saliva (IBL International, Germany). Interassay coefficients of variation were below 3.6%.

### Psychological measures

Before (–27 min relative to the stress onset) and directly after cessation of the stressor, state anxiety, calmness, and mood were assessed as indicators for psychological stress reactivity. We measured *state anxiety* using the German subscale “state anxiety” of the State Trait Anxiety Inventory (STAI; Laux, Glanzmann, Schaffner, & Spielberger, 1981), revealing a good internal consistency (Cronbach's  $\alpha$  before/after stress = .85/.95). *Calmness* and *mood* were assessed with the two subscales “calm-nervous” and “good-bad mood” from the German version of the Multidimensional Mood State Questionnaire (MDBF; Steyer, Schwenkmezger, Notz, & Eid, 1997). The subscales “calm-nervous” (Cronbach's  $\alpha$  before/after

stress = .87/.93) and “good-bad mood” (Cronbach's  $\alpha$  before/after stress = .87/.93) also showed good internal consistencies. In order to control for systematic differences in trait anxiety and chronic stress levels between the groups, we also asked the participants to complete the STAI “trait anxiety” scale and the short form of the German Trier Inventory for Chronic Stress (TICS; Schulz, Schlotz, & Becker, 2004). The internal consistency was good for both scales, with a Cronbach's  $\alpha$ -score of .88 for “trait anxiety” and .84 for “chronic stress.”

### Statistical analysis

We analyzed baseline differences of heart rate and cortisol baseline using analyses of variance (ANOVA). To control for baseline values we did further analyses with baseline-adjusted data (data minus baseline), unless otherwise indicated. For adjustment of cortisol levels, we used the last salivary sample as baseline, because, due to our results (the cortisol levels of the first salivary sample differed significantly between the groups but converged over time; see Results), we assume that the first saliva sample was taken too late to assess the true baseline (see Discussion). We analyzed whether the stressor led to a significant increase in heart rate and cortisol by using ANOVAs with repeated measures (group [inactive vs. moderately active vs. vigorously active] by time [repeated factor; 10 measurements for heart rate: –5 min before stress to 5 min after stress onset; 3 measurements for cortisol: stress onset to 25 min after stress onset]). *Stress reactivity*: To assess stress reactivity, we calculated the “area under the individual response curve with respect to the ground” ( $AUC_G$ ; Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) for each participant.<sup>5</sup> The  $AUC_G$  is more suitable than repeated measures analyses for analyzing physiological data with numerous points of measurement and sometimes different time distances between measurements (Pruessner et al., 2003). For heart rate, we calculated the  $AUC_G$  during the stress exposure (14 min time interval); for cortisol, due to its delayed response, we calculated the  $AUC_G$  for the time period between the 1st and 3rd sample (25 min time interval). We used separate ANOVAs and a priori comparison to determine differences in  $AUC_G$  between groups. To analyze differences in psychological stress reactivity, we used three separate ANOVAs with repeated measures (before and after stress), followed by contrast analyses with the difference value from the two time points. *Stress recovery*: For heart rate, we calculated *recovery time* (time [in min] needed to reach

<sup>5</sup> We preferred the  $AUC_G$  to the  $AUC$  with respect to increase ( $AUC_I$ ), as the  $AUC_I$  is based on the reference to the first value, allowing for negative results in the case of a decrease (Pruessner et al., 2003).

baseline level after cessation of stressor [end of stressor at +14 min; see Fig. 1]) and *recovery speed* (baseline adjusted heart rate at time point +14 min divided by recovery time [distance away from baseline/time needed to reach baseline]). For cortisol, we assessed the percentage of change in cortisol level from sample 3–6.<sup>6</sup> We used ANOVAs to determine group differences.

For all analyses the level of significance was set at  $p < .05$ . Where the Mauchly test of sphericity indicated heterogeneity of covariance, Greenhouse-Geisser corrections were used. All data were analyzed with SPSS Statistics 17.0 and are presented as mean  $\pm$  SEM.

## Results

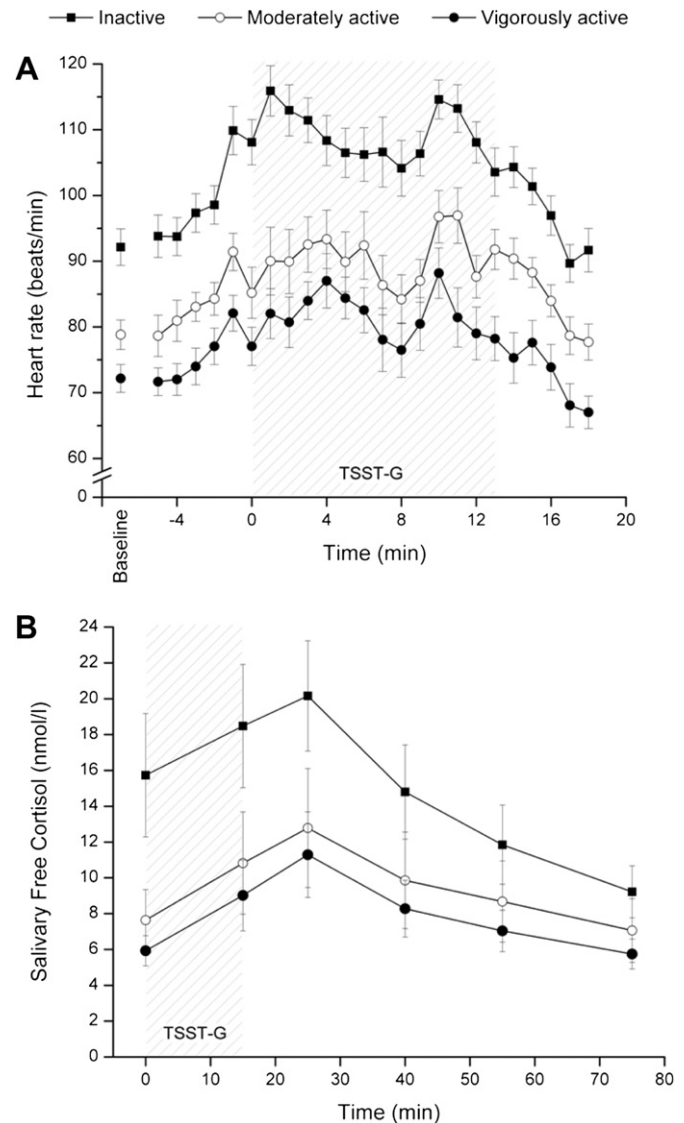
The three exercise groups did not differ significantly in terms of age, BMI, chronic stress, trait anxiety, or symptoms of psychopathology (all  $p > .05$ , see Table 1 for exact  $p$  values). In accordance with the exercise group classification, the three exercise groups differed significantly in levels of self-reported physical exercise,  $F(2,44) = 152.89$ ,  $p < .001$ ,  $\eta_p^2 = .874$ : On average, inactive women exercised less than 0.5 h/week, while moderately and vigorously active women exercised more than 4 h/week and 11 h/week, respectively.

### Heart rate responses to stress

Heart rate at baseline differed significantly among exercise groups,  $F(2,43) = 18.22$ ,  $p < .001$ ,  $\eta_p^2 = .459$ , with the lowest heart rate in the vigorous group ( $72.18 \pm 2.11$  bpm), a medium heart rate in the moderate group ( $78.82 \pm 2.24$  bpm) and the highest heart rate in the inactive group ( $92.15 \pm 2.77$  bpm) (Fig. 2A). Contrast analyses showed that the inactive group's baseline differed significantly from those in the moderate and the vigorous group (both  $p < .001$ ), whereas the difference between the latter two just missed significance ( $p = .07$ ). A significant large main effect of time in the repeated measures ANOVA,  $F(3,6,155.1) = 22.63$ ,  $p < .001$ ,  $\eta_p^2 = .345$ , indicated that the stressor induced a significant increase in heart rate in all groups; as the significant main effect of group [ $F(2,43) = 4.63$ ,  $p = .02$ ,  $\eta_p^2 = .177$ ] displays, the average height of heart rate differed between the three exercise groups, with the highest values for the inactive group. *Stress reactivity*: During stress exposure, the groups differed significantly in their deviation from baseline as indicated by the  $AUC_G$  parameter [ $F(2,43) = 3.76$ ,  $p = .03$ ,  $\eta_p^2 = .149$ ]: The inactive group showed a significantly higher stress reactivity than the vigorous group ( $p = .01$ ) and a trend toward a higher stress reactivity than the moderate group ( $p = .07$ ). The two active groups did not differ ( $p = .49$ ). *Stress recovery*: The time needed to recover also differed significantly between groups,  $F(2,43) = 5.64$ ,  $p = .01$ ,  $\eta_p^2 = .208$ : The vigorously active women's heart rates decreased significantly faster than those of the moderate ( $p = .02$ ) or inactive women ( $p = .002$ ), yet there was no difference between the latter two groups. In contrast to recovery time, there were no significant differences in recovery speed,  $F(2,43) = 1.17$ ,  $p = .32$ .

### Cortisol responses to stress

$N = 31$  women were tested during their luteal phase (10 inactive, 11 moderately active, 10 vigorously active). However, 6 of the 31 women did not have an elevation in their cortisol level as a response to the stressor and were therefore classified as *non-responders* (2 inactive, 3 moderately active, 1 vigorously active).



**Fig. 2.** (A) Mean baseline heart rates and mean heart rates before, during (shaded area), and after the psychosocial stressor (TSST-G) in inactive ( $n = 17$ ), moderately active ( $n = 14$ ), and vigorously active ( $n = 15$ ) women. (B) Mean salivary free cortisol levels before, during, and after the psychosocial stressor (TSST-G, shaded area) in inactive ( $n = 8$ ), moderately active ( $n = 8$ ), and vigorously active ( $n = 9$ ) women. Error bars are SEM.

Results from the analyses with and without *non-responders* were identical in terms of significance. For ease of reference, only the results for *responders* ( $n = 25$ ) are displayed. Mean salivary cortisol levels are presented in Fig. 2B.

There was a significant difference in cortisol levels at the time of the first salivary sample,  $F(2,22) = 5.68$ ,  $p = .01$ ,  $\eta_p^2 = .340$ : The highest cortisol levels were found in the inactive group ( $15.73 \pm 3.44$  nmol/l) and differed significantly from the moderately active ( $7.64 \pm 1.71$  nmol/l) and the vigorously active ( $5.93 \pm 0.84$  nmol/l) groups' cortisol levels ( $p = .02$  resp.  $p = .004$ ), which did not differ from each other ( $p = .58$ ). The groups' cortisol levels did not differ significantly at the time of the subsequent samples ( $p = .06$ ;  $p = .10$ ;  $p = .14$ ;  $p = .21$ ;  $p = .22$ ; the last sample was used as the baseline level; analyses were done with baseline adjusted data [see Methods]). The significant main effect of time,  $F(1,4,29.9) = 16.46$ ,  $p < .001$ ,  $\eta_p^2 = .428$ , illustrates that the TSST-G induced a significant increase in cortisol levels. *Stress reactivity*: Comparison of the  $AUC_G$  (salivary samples 1–3) indicates that the three groups differed in their stress reactivity [ $F(2,22) = 4.47$ ,  $p = .02$ ,

<sup>6</sup> As sample 6 itself is part of the recovery calculation, the unadjusted baseline values were used.

$\eta_p^2 = .289$ ], with the inactive group displaying a significantly higher endocrine stress reactivity than the moderately ( $p = .02$ ) and the vigorously active groups ( $p = .01$ ), whereas the two latter groups did not differ ( $p = .86$ ). *Stress recovery*: For stress recovery, no differences emerged between the three groups,  $F(2,22) = 1.03$ ,  $p = .37$ .

### Psychological responses to stress

Before stress exposure, the three exercise groups showed no significant differences in *state anxiety* [ $F(2,44) = 0.90$ ,  $p = .42$ ], *calmness* [ $F(2,44) = 0.22$ ,  $p = .80$ ], and *mood* [ $F(2,44) = 0.61$ ,  $p = .55$ ]. The same baseline levels can thus be assumed. *State anxiety* (Fig. 3A): The exposure to the psychosocial stressor caused a significant increase in state anxiety in the total group (main effect of time:  $F(1,44) = 53.14$ ,  $p < .001$ ,  $\eta_p^2 = .547$ ), but the three exercise groups neither responded differently to the stressor (no time by group interaction effect:  $F(2,44) = 1.24$ ,  $p = .30$ ), nor did they differ in their mean state anxiety levels (no main effect of group:  $F(2,44) = 0.73$ ,  $p = .49$ ). *Calmness* (Fig. 3B): After the stress exposure, calmness was significantly reduced in the total group of subjects (main effect of time:  $F(1,44) = 25.38$ ,  $p < .001$ ,  $\eta_p^2 = .366$ ); however, there was no significant time by group interaction effect,  $F(2,44) = 0.53$ ,  $p = .59$ , or main effect of group,  $F(2,44) = 0.76$ ,  $p = .47$ . *Mood* (Fig. 3C): The stressor significantly worsened the subjects' mood (main effect of time:  $F(1,44) = 57.10$ ,  $p < .001$ ,  $\eta_p^2 = .565$ ). Furthermore, we found a significant time by group interaction effect,  $F(2,44) = 3.60$ ,  $p = .04$ ,  $\eta_p^2 = .140$ , with further analyses showing that—contrary to our expectations—the vigorously active group's mood worsened significantly more than the inactive group's mood ( $p = .01$ ); for the moderately active group we found a trend toward a greater mood decrease compared with the inactive group ( $p = .06$ ). Averaged over both time points, the mood levels of the three groups did not differ ( $p = .26$ ).

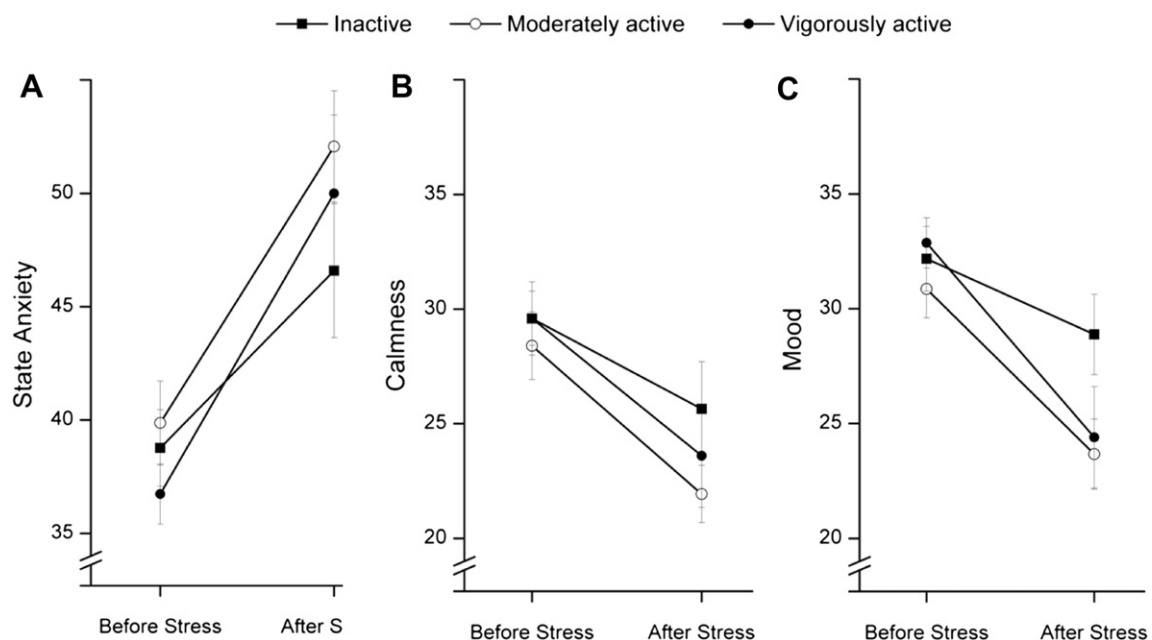
### Discussion

This is the first study to examine the effects of different levels of physical exercise on physiological and psychological stress responses

to a naturalistic standardized psychosocial stressor (TSST-G) in young women. Four major results have been identified: First, in all three exercise groups (inactive, moderately active, vigorously active) the TSST-G induced a significant rise in heart rate, cortisol, and state anxiety, while mood and calmness significantly worsened. Second, as hypothesized, the response pattern of the physiological stress reaction differed significantly for the three exercise groups, with lowered physiological stress reactions in the more active women. Third, contrary to expectations, the psychological stress responses did not parallel the physiological stress responses, with the more physically active groups even showing an *elevated* psychological stress reaction in terms of change in mood. Fourth, our results do not support the idea of a difference in sensitivity between the SNS and the HPA axis (Rimmele et al., 2009), as the moderately active women resembled the vigorously active women in their cardiovascular and endocrine stress response. In the following, we discuss the findings for heart rate, cortisol, and psychological stress reactions separately.

### Physiological stress indicators

For heart rate, our findings confirm for women what has been reported previously by Rimmele et al. (2009) for men. During stress exposure *heart rate reactivity* significantly differed for the three exercise groups, with the lowest heart rate levels in the vigorously active group, the second lowest in the moderately active group, and the highest in the inactive group (Fig. 2A). This result supports the assumption of the CSA hypothesis for women: Persons with higher levels of regular exercise showed alleviated cardiovascular stress reactions in psychosocial stress situations. For vigorously exercising women, the CSA hypothesis was also confirmed in terms of heart rate *recovery time*: The women showed a significantly faster recovery time than their moderately active and inactive counterparts. However, for heart rate *recovery speed*—taking into account the “distance to recover”—no significant differences between the exercise groups were found. Thus, for heart rate recovery time and speed our results are ambiguous, as is the literature on this topic. According to meta-analyses by Jackson and Dishman (2006) as well as Forcier et al. (2006), regular exercise is linked to a better stress



**Fig. 3.** Mean levels of state anxiety (A), calmness (B), and mood (C) before and after the psychosocial stressor (TSST-G) in inactive ( $n = 17$ ), moderately active ( $n = 15$ ), and vigorously active ( $n = 15$ ) women. Error bars are SEM.

recovery ability, yet Rimmele et al. (2009) did not find significant differences between exercise groups for recovery speed (percentage of change from peak to baseline). This illustrates again the assessment problem we pointed out above: The use of different indicators of stress response (i.e. recovery speed vs. time) can lead to incongruous results.

Our data also confirm a link between physical exercise and endocrine stress responses: Physically inactive women showed significantly higher cortisol reactivity than their active counterparts (Fig. 2B). This is in line with findings from Traustadóttir et al. (2005) for older women and Rimmele et al. (2007, 2009) for men. Contradictory results were reported by Long (1991) and Summers et al. (1999), yet in these studies stress was not induced by a standardized and validated psychosocial stressor. In our data, both the vigorously active and the moderately active group showed a significantly lower cortisol response than the inactive group. At least for women, this result contradicts findings by Rimmele et al. (2009), who concluded from their study “that only a markedly enhanced level of physical activity, such as in elite sportsmen, rather than graded increases causes a significant adaptation of the HPA response that may generalize to other stressors” (p. 195). Our data do not support this view; instead, we found evidence for a graded endocrine response to an increasing level of physical exercise: Participants exercising 2–6 h per week already showed alleviated endocrine stress reactions as assumed by the CSA hypothesis. Regarding cortisol recovery, our data did not confirm the CSA hypothesis. Similar to results by Rimmele et al. (2009), the cortisol recovery in our study did not differ between the three exercise groups. However, these non-significant differences in cortisol recovery might be the reflection of a measurement problem: Due to fixed assessment points for the salivary cortisol samples on the one hand and delayed cortisol response on the other hand, the examination of cortisol recovery was more imprecise than the examination of heart rate recovery.

Two limitations must be taken into account when interpreting our findings on cortisol reactivity: First, only 31 women were eligible for the cortisol analyses, six of whom did not show a cortisol increase when confronted with the stressor (non-responders). This drop in sample size causes a reduction of the statistical power. As we found a very large effect in terms of cortisol reactivity, a post-hoc power analysis indicated an acceptable power of .77 for this parameter; however in terms of cortisol recovery a smaller effect might not have been detected due to insufficient statistical power. Second, the exercise groups' cortisol levels already differed significantly at the time of the first sample of salivary cortisol. This difference was neither expected nor can it easily be explained. Contrary to heart rate, regular physical exercise does not lead to a generally decreased cortisol level, although acute physical exercise stimulates cortisol release (Duclos & Tabarin, 2011). Because the exercise groups' cortisol levels converged in the course of time and did not significantly differ anymore at the time of the last salivary sample, a general baseline difference between the groups is not likely. We rather suppose that the first saliva sample was taken too late to allow assessment of the baseline: At the time of the first saliva sample, the women had already known about the upcoming stress-task for 10 min and had been participating in the study for about 45 min; the anticipatory stress might have led to an increase of cortisol even before the first saliva sample. If the inactive women, as hypothesized, show a larger endocrine stress response in general, this might also be true for anticipatory stress. Results from Gaab, Rohleder, Nater, and Ehlert (2005) support the assumption that the TSST excites anticipatory stress and as a result increases the level of cortisol before the TSST even starts. However, as the inactive male participants in the studies by Rimmele et al. (2007, 2009) did not show elevated cortisol levels directly before the TSST, it remains

unclear whether our findings are distinctive to inactive women or are rather an experimental artifact.

#### Psychological stress indicators

In all three exercise groups, anxiety, calmness, and mood worsened significantly due to the stress induction (Fig. 3). However, significant differences between the groups were found only for mood: The vigorously active women's mood, and to a lesser extent also that of the moderately active women, deteriorated more under stress than the inactive women's mood. This result was not expected as earlier findings indicated larger mood worsening in untrained men compared to trained men (Rimmele et al., 2007). What could be a possible explanation for the dissociation between physiological and psychological stress reactions we found in our data? Campbell and Ehlert (2012) showed that physiological and psychological stress responses are not automatically linked: Significant correlations between perceived emotional stress variables and physiological responses measured by heart rate and saliva cortisol emerged in less than 30% of 49 analyzed TSST studies. Strahler, Kirschbaum, and Rohleder (2011) furthermore found a negative association between physiological and psychological stress response: Before and during a ballroom dancing competition, older dancers showed a larger endocrine stress response but reported lower psychological stress and relevance of results compared to younger dancers. With regard to our findings, one might speculate that a good performance on the stress task might have been more relevant to the physically active women, as sport and exercise engagement is associated with a higher competitiveness and achievement motivation (Wartenberg & McCutcheon, 1998). Being more competitive and motivated to achieve, the physically active women might have found it more important to perform well or better than the other participants in the group stress task. Thus, physically active women may have experienced more psychological stress, while their physiological stress responses—due to CSA effects—remained less affected. However, this speculation is not supported by Rimmele et al. (2009): Although elite and amateur sportsmen had a higher competitiveness than untrained men, there was no dissociation of the physiological and psychological stress responses.

We need further research to find out whether the dissociation between physiological and psychological stress responses found in the present study emerged due to the gender of the sample (women), the nature of the stress task (the TSST-G might cause larger reactions in more competitive subjects than the single TSST), or due to other moderating variables.

#### Strengths and limitations

Our study sheds light on the relationship between regular physical exercise and stress responses in young women. By applying a standardized psychosocial stressor (TSST-G), we assessed physiological (heart rate, salivary free cortisol) and psychological (state anxiety, calmness, mood) stress responses in three exercise groups (inactive, moderately active, and vigorously active), allowing for comparisons with similar studies conducted with males. Our results suggest that the CSA hypothesis is valid for physiological but not for psychological stress responses in women—a challenging finding that certainly needs further examination. However, as our data were only cross-sectional, no causal conclusions can be drawn. Longitudinal experimental intervention studies manipulating regular exercise engagement are needed to clarify causality in this regard. It is also unclear whether physical exercise or physical fitness accounts more for changes in the stress responses. Apart from that, the generalizability of our findings is limited to young and healthy

women having a rather high educational level—a broadening to other groups and different clinical samples would provide more insight into the influence of gender, age, and other factors on exercise-related Cross-Stressor Adaptation processes. All in all, our study underlines the importance of valid and standardized stress protocols and measures for interpreting and comparing results in a meaningful way.

## Outlook

Low reactions to laboratory stressors are linked to a lower risk for stress-related diseases (e.g., Chida & Steptoe, 2010), and health is affected negatively by long-lasting hyper-elevated activations and slow recovery of the stress systems (e.g., Chrousos, 2009). If it holds true that regular physical exercise leads to reduced stress reactivity and improved stress recovery even in psychosocial stress situations (e.g., at the workplace), this would strengthen the idea of physical exercise as an effective “buffer” against the negative health effects of chronic stress. However, it should not be disregarded that an increasing number of studies have shown chronic physiological hypo-reactivity to be linked to an increased risk of other chronic diseases such as obesity (Carroll, Phillips, & Der, 2008). Thus, reduced stress reactivity might be a double-edged sword: It is associated with a lowered risk for some diseases, like cardiovascular illnesses, but at the same time heightens the risk for other diseases (Carroll et al., 2009). Future studies need to explore the long-term relationship between physical exercise, physiological and psychological stress responses (in terms of hyper- as well as hypo-elevated reactions), and stress-related chronic disorders.

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