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The level of physical activity affects adrenal and cardiovascular reactivity to psychosocial stress

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Summary Physical activity plays a key role in the control of neuroendocrine, autonomic, and behavioral responses to physical and psychosocial stress. However, little is known about how the level of physical activity modulates stress responsiveness. Here, we test whether different levels of physical activity are associated with different adrenal, cardiovascular, and psychological responses to psychosocial stress. In addition, competitiveness is assessed as a personality trait that possibly modulates the relationship between physical activity and stress reactivity. Eighteen elite sportsmen, 50 amateur sportsmen, and 24 untrained men were exposed to a standardized psychosocial laboratory stressor (Trier Social Stress Test). Repeated measures of salivary free cortisol, heart rate, and psychological responses to psychosocial stress were compared among the 3 study groups. Elite sportsmen exhibited significantly lower cortisol, heart rate, and state anxiety responses compared with untrained subjects. Amateur sportsmen showed a dissociation between sympathetic and hypothalamic—pituitary—adrenal responsiveness to stress, with significantly reduced heart rate responses but no difference in cortisol responses compared with untrained men. Different levels of competitiveness among groups did not mediate stress reactivity. Our results are in line with previous studies indicating reduced reactivity of the autonomic nervous system to psychosocial stress in trained individuals. More importantly, these findings imply a differential effect of the level of physical activity on different stress-related neurophysiological systems in response to psychosocial stress.

1. Introduction

Psychosocial stress increases the risk of developing cardiovascular and mental diseases, such as hypertension or

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depression (Gold et al., 1988a,b; McEwen, 2000, 2002; Vanitallie, 2002). Physical activity is commonly regarded as beneficial to both physical and psychological health, and is seen as an effective preventive measure and treatment for stress-related diseases (Perkins et al., 1986; Ross and Hayes, 1988; Steptoe et al., 1993; Blumenthal et al., 1999; Babyak et al., 2000; Talbot et al., 2002; Ketelhut et al., 2004; Barlow et al., 2006; Nabkasorn et al., 2006). Physically active people show reduced reactivity to physical stressors as well as reduced susceptibility to the adverse influences of life stress (Tucker et al., 1986; Luger et al., 1987; Deuster et al., 1989; Steptoe et al., 1989; Dishman, 1997; Throne et al., 2000; Dishman et al., 2006; Rimmele et al., 2007). Moreover, it has been proposed that physical activity influences stress reactivity more generally, with protective effects also on non-physical stress, such as mental stressors (Claytor, 1991; Cox, 1991; Sothmann et al., 1991, 1996). Although findings are not uniform (de Geus and van Doornen, 1993; Moyna et al., 1999; Jackson and Dishman, 2006), there is substantial evidence showing that physical activity is associated with lower reactivity of the sympathetic nervous system and the hypothalamic–pituitary–adrenal (HPA) axis to psychological stress protocols. Specifically, physically active subjects showed lower cortisol increase (Rimmele et al., 2007), lower cardiovascular reactivity (Crews and Landers, 1987; Spalding et al., 2004; Rimmele et al., 2007), and more rapid cardiovascular recovery (Jackson and Dishman, 2006) to psychological laboratory stressors in comparison with less active controls. Interestingly, stress reactivity differs not only between extreme groups of physically active and inactive controls, but also between groups with less distinct differences in activity levels. For example, heart rate reactivity to a mental stressor is lower and heart rate recovery is faster in elite sportsmen compared with amateur sportsmen (Moya-Albiol et al., 2001). Thus, the level of physical activity might differentially affect stress reactivity, which could also explain some inconsistent results of previous studies.

Personality traits may be important modulators of the relationship between physical activity and stress reactivity. Compared with untrained controls, sportsmen typically show higher levels of self-efficacy (Netz et al., 2005; Rimmele et al., 2007) and competitiveness (Frederick, 2000), with elite sportsmen scoring significantly higher on competitiveness in comparison to amateur sportsmen (Houston et al., 1997). Notably, self-efficacy has been associated with lower anxiety and physiological stress reactivity (Schwarzer, 1992; Bandura, 1997; Butki et al., 2001), and competitiveness has been related to higher cardiovascular reactivity to competition (Harrison et al., 2001). However, studies relating to the modulating influence of self-efficacy and competitiveness on stress reactivity in sportsmen are scarce. In a recent study from our laboratory, higher levels of self-efficacy in elite sportsmen were found to be unrelated to lower physiological and subjective stress responses compared with untrained men (Rimmele et al., 2007). Sportsmen with higher levels of competitiveness exhibit lower anxiety before a competition (Jones and Swain, 1992). However, whether or not competitiveness likewise accounts for differences in reactivity to a psychosocial stressor is unclear.

The aim of the present study was twofold. First, we examined the effect of different levels of physical activity on psychosocial stress reactivity by including three study groups that clearly differ in their level of physical activity (elite sportsmen, amateur sportsmen, and untrained men). Second, we investigated possible modulating effects of competitiveness on stress responsiveness. Physical fitness was characterized by reported training regimens and by a lactate test. Psychological and physiological responses to a psychosocial laboratory stressor were assessed by repeated measurement of state anxiety, mood and calmness, heart rate, and salivary free cortisol levels.

2. Methods

2.1. Participants

The final study sample comprised 18 elite sportsmen, 50 amateur sportsmen, and 24 untrained men. All participants were recruited by the Swiss Federal Office of Sports, local sports clubs, and through advertisements in newspapers and at the local universities in Zürich. Based on the physical activity levels and endurance capacity of trained subjects assessed by a physical fitness test and a self-report questionnaire (see Section 2.2), subjects were classified into groups of elite sportsmen, amateur sportsmen, and untrained

Table 1 Description of the study groups

	Elite sportsmen (n = 18)	Amateur sportsmen (n = 50)	Untrained men (n = 24)
Age (years)	24.17 ± 0.89	24.82 ± 0.43	23.65 ± 0.61
Body mass index (kg/m^2)	22.27 ± 0.54	21.8 ± 0.25	21.46 ± 0.42
Perceived stress (PSS)	19.79 ± 1.36	21.64 ± 0.94	22.91 ± 1.30
Training (h/week)**	11.61 ± 1.07	5.44 ± 0.34	0.37 ± 0.12
Running (h/week)**	4.67 ± 1.01	2.43 ± 0.21	–
Running distance (km/week)**	42.95 ± 8.34	21.43 ± 1.72	–
V 4 mmol/l (km/h)**	15.38 ± 0.51	13.16 ± 0.25	–
Maximum velocity (km/h)*	18.84 ± 0.38	17.35 ± 0.32	–
Perceived exertion (BORG rating scale)	18.33 ± 0.27	18.67 ± 0.19	–

Data are expressed as mean ± S.E.M.
V 4 mmol/l—velocity at 4 mmol/l blood lactate concentration; PSS—perceived stress scale.
* $p < 0.05$.
** $p < 0.01$.

men (Table 1). Sportsmen were medium to long distance runners. A MANOVA was used to verify group differences in physical activity levels. Exclusion criteria were medication intake, reported medical illness, symptoms of psychopathology (Derogatis, 1983), substance abuse, smoking more than 5 cigarettes per day, increased levels of chronic stress (Perceived Stress Scale (PSS) (Cohen et al., 1983)), and overtraining measured with the Recovery-Stress Questionnaire for Athletes (RESTQ-Sport) (Kellmann and Kallus, 2001). Eight of the original 100 subjects did not meet the eligibility criteria and were excluded: four with acute seasonal allergic rhinitis, two who met criteria for a mental health disorder based on the Symptom Checklist (SCL-90-R) (Derogatis, 1983), and two due to overtraining (RESTQ-Sport). The study was approved by the institutional review board of the University of Zürich. Before participation, all subjects provided written informed consent and were informed of their right to discontinue participation at any time. After completion of the experiment, subjects were paid 50 Swiss francs for their participation.

2.2. Physical fitness test

The physical fitness test determined the maximum velocity and the velocity at 4 mmol/l blood lactate concentration (anaerobic threshold). The physical fitness test was carried out at the end of the sports season in the afternoon. It consisted of a submaximal, non-aversive 4 × 1000 m running test (Held et al., 2000) at self-selected but increasing running velocities (prescribed as slow, medium, and fast training speed and maximal velocity) interspersed with short recovery periods of 2 min. During recovery, blood samples were taken from the earlobe for assessment of lactate concentration, and perceived exertion was measured with the Borg scale (Borg et al., 1987). The self-report questionnaire assessed the frequency and duration of exercise activities on a weekly basis, the number of sports competitions per year, and the achievements in sports competitions.

2.3. Procedure

All experimental sessions started between 13:00 and 15:00 h and lasted for 2.5 h. The physical fitness test took place at least one week before the psychosocial stress session. Subjects were instructed to refrain from eating and drinking anything but water for 2 h prior to the experiment and from intense physical activity, caffeine, nicotine, and alcohol during the 24 h before the experiment. Psychosocial stress was induced by the Trier Social Stress Test (TSST) (Kirschbaum et al., 1993), which reliably induces two- to threefold increase in HPA axis and cardiovascular responses (Singh et al., 1999; Heinrichs et al., 2001, 2003; Dickerson and Kemeny, 2004). All subjects were naïve to the applied stress procedure. In this stress protocol, subjects are exposed to a 5-min public speaking task (mock job interview) and a subsequent 5-min mental arithmetic task (serial subtraction) performed out loud in front of an unknown panel of two evaluators and a conspicuous video camera. For the public speaking task, participants were instructed to apply for a job of their choice. To further emphasize the socio-evaluative character of the TSST, the panel of evaluators was presented as experts in the evaluation of nonverbal behavior. After the participants had entered the TSST room, they remained in a standing position throughout the 10-min stress protocol. Following completion of the stress session, subjects were instructed to rest quietly for 90 min until saliva sampling was completed.

2.4. Endocrine and autonomic measures

Salivary free cortisol is a valid indicator of the biologically active fraction of cortisol (Vining et al., 1983; Kirschbaum and Hellhammer, 1989, 1994). Saliva was collected using Salivette collection devices (Sarstedt, Rommelsdorf, Germany). In order to minimize anticipatory stress reactions (Kirschbaum et al., 1992), which could affect baseline cortisol measurement, participants were told about the task they had to perform after the baseline saliva sample had already been collected. Seven saliva samples were collected immediately before (−1 min relative to the onset of the TSST) and after stress exposure (+10, +20, +30, +45, + 60, +90 min). Samples were stored at −20 °C until assaying. For biochemical analyses of free cortisol concentration, saliva samples were thawed and spun at 3000 rpm for 10 min to obtain 0.5–1.0 ml clear saliva with low viscosity. Cortisol concentrations were determined using a commercially available chemiluminescence immunoassay (CLIA; IBL Hamburg, Germany). Intra- and interassay coefficients of variation were below 10%.

Heart rate was monitored continuously for subsequent 60-s intervals from 1 min before stress exposure until 5 min after cessation of the stressor using a wireless chest heart rate transmitter and a wrist monitor recorder (Polar S810i™, Polar Electro, Finland).

2.5. Psychological measures

General competitiveness was assessed with the Competitiveness Index (CI) (Houston et al., 1992), and sports-specific competitiveness was assessed with the Sports Orientation Questionnaire (SOQ) (Gill and Deeter, 1988). At baseline (−40 min relative to the onset of stress exposure), and during anticipation of the stressor (−1 min) as well as after stress exposure (+10 min), mood, calmness, and state anxiety were repeatedly measured using the state scale of the State-Trait Anxiety Inventory (STAI) (Spielberger et al., 1970) and the Multidimensional Mood Questionnaire (Steyer et al., 1997). All questionnaires have been broadly used and have shown satisfactory internal consistency and validity. The calculated Cronbach's index of internal consistency show good internal consistency for the STAI (between α = 0.85 and 0.91) and for the subscales mood (between α = 0.75 and 0.87) and calmness (between α = 0.77 and 0.83) of the Multidimensional Mood Questionnaire.

2.6. Statistical analysis

Cortisol and psychological data were analyzed using two-way analysis of variance (ANOVA) with repeated measurement (group [3 groups: elite sportsmen vs. amateur sportsmen vs. untrained men] by time [repeated factor: 7 for cortisol and 3 for psychological measures]). Due to significant baseline differences between groups in heart rates using a one-way

analysis of variance, an analysis of covariance (ANCOVA) with baseline measures as covariate was calculated with 15 repeated measurements. We verified repeated measures results with Greenhouse-Geisser corrections where the Mauchly test of sphericity determined heterogeneity of covariance. To determine group differences, we calculated separate ANOVAs and *post hoc* comparisons using Tukey's HSD test. The areas under the individual response curves with respect to increase ($AUC_{Increase}$) were calculated with the trapezoid formula (Pruessner et al., 2003), which allows a sensitive measurement of physiological changes over time. Recovery was calculated as the percentage change in cortisol levels from 10 min (peak) to 60 min (back to baseline) post-TSST and from +1 min (peak) to +12 min (back to baseline) for heart rate, respectively. To assess group differences in the two competitiveness measures, a MANOVA was calculated. As competitiveness differed between groups, mediating effects of competitiveness were additionally assessed using a mediator analysis based on linear regression modelling with competitiveness entered as mediator (Baron and Kenny, 1986). As independent variables dichotomized group variables were entered. As dependent variables, we used AUCi's of cortisol and heart rate for assessment of physiological stress reactivity. For the assessment of psychological stress reactivity, the differences between pre- and post-stressor values were calculated. Data were analyzed using SPSS 14 (SPSS Inc., Chicago, IL). Data are presented as mean ± S.E.M. All analyses were two-tailed, with the level of significance set at $p < 0.05$.

3. Results

The three study groups did not significantly differ in terms of age, BMI, psychological symptoms, and perceived stress (all $p > 0.1$). A significant difference in the duration of training (h/week) was observed among the three groups ($F(2, 88) = 107.15$, $p < 0.01$). The MANOVA for the selected variables indicative of physical activity level (training (h/week), running (h/week), running distance (km/week), velocity at 4 mmol/l blood lactate concentration, maximum velocity (km/h)) revealed a significant difference between elite sportsmen and amateur sportsmen ($F(1, 65) = 13.12$; $p < 0.001$). Elite sportsmen exhibited significantly higher scores in velocity at 4 mmol/l blood lactate concentration ($t(64) = 4.21$, $p < 0.01$) and in maximum velocity (km/h) ($t(64) = 2.58$, $p < 0.05$) compared with amateur sportsmen. There was no difference in perceived exertion between elite sportsmen and amateur sportsmen. Characteristics of the sample are summarized in Table 1.

3.1. Cortisol responses to stress

The psychosocial stress protocol induced the expected significant increase in salivary free cortisol levels in all three groups (main effect of time, $F(1.90, 163.42) = 120.6$, $p < 0.01$). Cortisol levels did not differ between groups at baseline ($F(2, 88) = 1.67$, $p = 1.95$). However, study groups differed significantly in their cortisol responses to the stressor (main effect of group, $F(2, 86) = 4.78$, $p < 0.05$; group by time interaction, $F(3.8, 163.42) = 2.54$, $p < 0.05$), with the group of elite sportsmen exhibiting the lowest cortisol

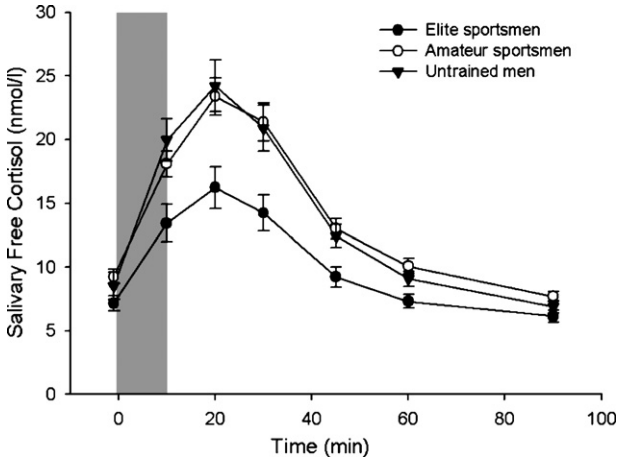

Figure 1 Mean salivary free cortisol levels before, during (shaded area), and after a standardized psychosocial stressor (Trier Social Stress Test) in elite sportsmen, amateur sportsmen, and untrained men. Error bars are standard errors of the mean (S.E.M.).

responses as compared to the groups of amateur sportsmen (*post hoc* analysis, $p = 0.01$) and untrained men (*post hoc* analysis, $p = 0.03$) (Fig. 1). The mean absolute increase in salivary cortisol in response to stress was 15.69 nmol/l in untrained men, 14.15 nmol/l in amateur sportsmen, and 9.08 nmol/l in elite sportsmen.

For cortisol increase (−1 to +90 min), a one-way ANOVA with $AUC_{Increase}$ showed no significant differences between groups ($F(2, 88) = 0.89$, $p = 0.42$), indicating that sportsmen and untrained men show the same overall cortisol reactivity. Recovery did not significantly differ between groups ($F(2, 88) = 1.75$, $p > 0.17$).

3.2. Heart rate responses to stress

Heart rate at baseline (−1 min before the onset of stress) differed significantly among groups ($F(2, 83) = 4.1$, $p < 0.05$). The mean absolute baseline heart rates were 79.9 ± 3.9 beats/min in elite sportsmen, 79.9 ± 2.3 beats/min in amateur sportsmen, and 92.5 ± 5.0 beats/min in untrained men. Results obtained by two-way ANCOVA with repeated measures indicated that the stress protocol induced significant increases in heart rate in all groups (main effect of time, $F(6.8, 545.8) = 5.31$, $p < 0.01$). In response to the stressor, there was a significant attenuating effect of physical activity on heart rate (main effect of group, $F(2, 80) = 4.02$, $p < 0.05$; group by time interaction, $F(13.6, 545.8) = 1.85$, $p < 0.05$), with the lowest heart rate levels during the stress protocol in elite sportsmen and amateur sportsmen compared to untrained men (Fig. 2). Groups did not differ in heart rate recovery ($p > 0.44$).

3.3. Psychological responses to stress

State anxiety (STAI) significantly increased during the anticipation period of the stress protocol in all groups (main effect of time, $F(2, 176) = 71.95$, $p < 0.01$) (Fig. 3). A main effect of group on state anxiety was observed ($F(2, 88) = 3.49$, $p < 0.05$), demonstrating the highest anxiety levels in

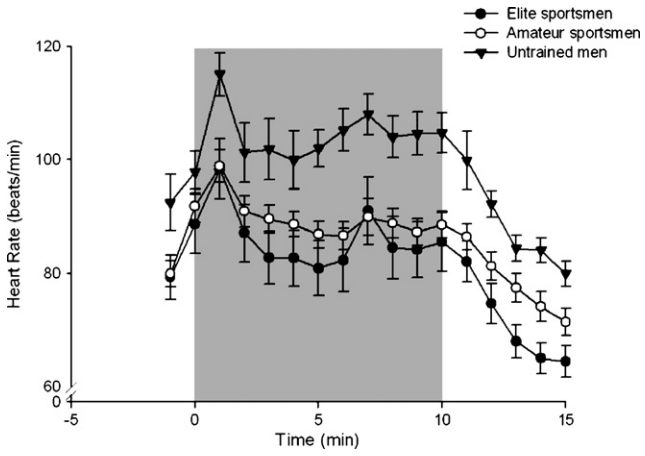

Figure 2 Mean heart rates before, during (shaded area), and after a standardized psychosocial stressor in elite sportsmen, amateur sportsmen, and untrained men. Error bars are S.E.M.

untrained men and the lowest anxiety levels in elite sportsmen throughout the experimental session (*post hoc* analysis, $p < 0.05$). State anxiety levels in amateur sportsmen did not significantly differ from untrained men and elite sportsmen ($p > 0.31$).

The stress protocol significantly worsened mood (main effect of time, $F(1.84, 163.69) = 25.39$, $p < 0.01$) (before introduction to stress, 16.6 ± 2.30; after stress, 15.0 ± 3.20) and calmness (main effect of time, $F(2, 178) = 66.63$, $p < 0.01$) (before introduction to stress, 14.95 ± 2.75; after stress, 13.2 ± 3.23) in all groups, with the highest negative mood (14.3 ± 2.69) and lowest calmness scores (10.4 ± 3.00) occurring when subjects were anticipating the stressor. No significant differences were observed among groups (all $p > 0.1$).

3.4. Effects of competitiveness on stress responsiveness

A MANOVA for the Competitiveness Index and the Sports Orientation Questionnaire showed a significant global effect

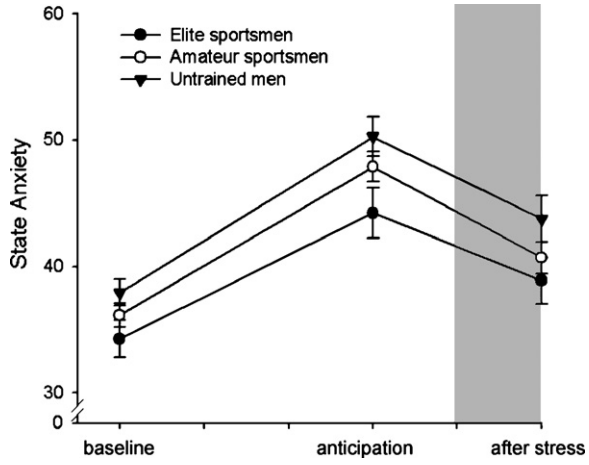

Figure 3 Mean levels (with S.E.M. bars) of anxiety before and during anticipation, and after psychosocial stress exposure (shaded area) in elite sportsmen, amateur sportsmen, and untrained men.

($F(1, 88) = 25.74$, $p < 0.001$). Groups differed significantly in competitiveness scores as assessed by the Competitiveness Index (CI) ($F(2, 88) = 9.2$, $p < 0.01$) and the Sports Orientation Questionnaire (SOQ) ($F(2, 88) = 24.35$, $p < 0.01$) (Fig. 4). For the CI, elite sportsmen (15.61 ± 0.51) differed significantly from amateur sportsmen (12.43 ± 0.56) and untrained men (10.57 ± 0.87) in competitiveness levels (*post hoc* analyses, all $p < 0.01$). For the SOQ, elite sportsmen displayed the highest competitiveness (55.94 ± 1.55), with amateur sportsmen showing intermediate (47.74 ± 0.94), and untrained men the lowest (40.08 ± 1.81) competitiveness levels (*post hoc* analyses, all $p < 0.01$). Competitiveness was negatively correlated with baseline heart rate (for CI $r = -0.51$; $p < 0.01$; for SOQ $r = -0.33$; $p < 0.01$).

In order to determine whether differences in competitiveness among groups may modulate stress responses, a mediator analysis was conducted (Baron and Kenny, 1986). However, competitiveness did not mediate group differences in cortisol, heart rate, and psychological responses to stress (all $p > 0.1$).

4. Discussion

In the present study, we examined whether different levels of physical activity are associated with different psychological, adrenal, and autonomic responses to a standardized psychosocial stressor. In addition, we tested the potential influence of competitiveness on stress reactivity. We measured cortisol and heart rate repeatedly at time points that covered their full stress reactivity time courses. The stress protocol significantly increased cortisol and heart rate responses, worsened mood and calmness, and increased state anxiety in all three study groups. However, the pattern of stress responses was related to the level of physical activity, with the lowest

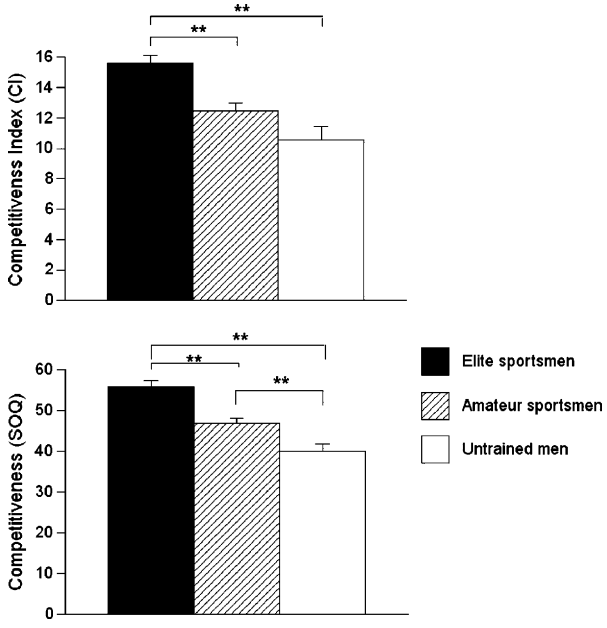

Figure 4 Mean levels (with S.E.M. bars) of general competitiveness (Competitiveness Index, CI) and sports-related competitiveness (Sports Orientation Questionnaire, SOQ) in elite sportsmen, amateur sportsmen, and untrained men.

cortisol, heart rate, and psychological responses in those individuals with the highest physical activity level (elite sportsmen). Most importantly, we found a dissociation between the sympathetic nervous system (heart rate) and HPA axis (cortisol) responses depending on the level of physical activity. Whereas elite sportsmen exhibited generally attenuated physiological and psychological stress responses compared with untrained men, amateur sportsmen showed lower heart rate responses but similar cortisol responses compared with untrained men. Competitiveness was not associated with differences in stress reactivity.

The present data build on previous research by demonstrating lower cortisol responses to psychosocial stress in elite sportsmen compared with untrained men even though the two groups did not differ at baseline levels. This finding confirms previous findings of lower cortisol responses to psychosocial stress of elite sportsmen compared with untrained men in another sample (Rimmele et al., 2007). The similar cortisol responses in amateur sportsmen and untrained men observed here are also consistent with previous studies that likewise did not reveal differences in cortisol reactivity to psychological stressors among amateur sportsmen of differing levels of fitness or between amateur sportsmen and sedentary subjects (Sinyor et al., 1983; Moyna et al., 1999). Chronic physical exercise is associated with reduced HPA axis activation if the experimental stressor is of the same absolute physical intensity (Luger et al., 1987), and high HPA axis responders to exercise stress are also highly responsive to psychological stress (Singh et al., 1999). In this context, it is possible 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. Alternatively, some other aspects of elite sportsmen (e.g., regular exposure to the stress of competition) may have led to such adaptations.

Both elite sportsmen and amateur sportsmen showed significantly lower heart rate responses to stress compared with untrained men, replicating previous reports from several cross-sectional studies (Heidbreder et al., 1983; Holmes and Roth, 1985; Brooke and Long, 1987; Claytor, 1991; Rimmele et al., 2007). In addition, a longitudinal study showed that aerobic training reduces the cardiovascular response to psychological stress (Spalding et al., 2004). In contrast, a recent meta-regression analysis reported cardiorespiratory fitness to be linked to slightly stronger cardiovascular responses to psychological stress (Jackson and Dishman, 2006). Possible reasons that might explain this divergent outcome include different types of psychological stressors, the exclusion of elite sportsmen, differences in physical activity and fitness levels, variations in age and gender of the subjects, the method of measurement, and the time of day of stress induction of the studies included in the meta-regression analysis.

It is important to note that the comparison of physiological changes across the three groups revealed a dissociation of the weekly amount of physical activity between sympathetic nervous system (heart rate) and HPA axis (cortisol) responses. Only elite sportsmen showed consistently lower stress responses compared with untrained men in both heart rate and cortisol responses to the psychosocial stressor, whereas amateur sportsmen showed lower heart rate but similar cortisol responses as compared to untrained men. Although the mechanisms underlying this dissociation are unknown, it might be suggested that the sympathetic nervous system (as the main regulator of heart rate) is more sensitive to the adaptive consequences of physical activity and that potential adaptations of the HPA axis occur later on, at a higher level of physical activity.

With regard to the psychological stress response, elite sportsmen showed the lowest, amateur sportsmen intermediate, and untrained men the highest state anxiety responses, which is in accordance with previous findings (Sinyor et al., 1983; Rimmele et al., 2007). Acute periods of exercise as well as prolonged exposure (over 10 weeks) to aerobic exercise are related to a reduction in anxiety (Petruzzello et al., 1991, 1997; Salmon, 2001). Our findings suggest that the level of physical activity is associated with state anxiety responses to a psychosocial stressor.

We also examined whether competitiveness might modulate psychosocial stress reactivity. Both general and sports-specific competitiveness levels were highest in elite sportsmen, differing markedly from levels in amateur sportsmen or untrained men, which concurs with many previous reports (Houston et al., 1997). However, although negatively correlated with baseline heart rate, competitiveness per se did not mediate adrenal and autonomic stress responsiveness. Thus, higher competitiveness may be linked to a higher level of physical activity, but seems to be unrelated to stress reactivity in our study.

Regarding possible clinical implications, it seems important to note that both hyper-reactivity of the sympathetic nervous system and the HPA axis are associated with psychiatric and cardiovascular disease (McEwen, 1998). Exaggerated cardiovascular responsiveness to psychological stress contributes to the pathogenesis of cardiovascular disease (Linden et al., 2003). Our findings indicate that higher levels of physical activity are associated with lower heart rate reactivity to stress, but there are no differences in heart rate recovery, suggesting that higher levels of physical activity might be protective against the development of stress-related diseases. Accordingly, extreme levels of exercise have been associated with increased susceptibility to immunosuppression and infection (Gleeson, 2006). Thus, it may be speculated that an intermediate level of physical activity is most effective for stress protection, whereas too high levels might cause long-term dysfunctional effects on health.

As we used a cross-sectional study design, our data do not allow a causal interpretation of the effects of physical activity on stress responsiveness. Prospective longitudinal studies are needed in order to confirm that physical activity decreases stress responses and, as a long-term effect, may protect against stress-related disorders. Similarly, the relationship of the type and the amount of physical activity and the possible influence of psychological variables in terms of stress protection should also be investigated in longitudinal study designs. While our participants were selected to be medium to long distance runners, our study is limited in that we did not assess complete data on the other kinds of physical activity that they were carrying out. As our total sample included healthy young men, the results cannot be generalized to the population as a whole, and particularly not to older persons. However, not only is there evidence that aging is associated with stronger HPA responses to stress but there are also findings indicating a robust attenuating effect of

moderate physical activity on these responses in elderly people (Traustadottir et al., 2005). Thus, changes in HPA axis reactivity induced by physical activity may even be more pronounced in older compared with younger subjects.

In summary, our data suggest that the level of physical activity differentially influences the physiological and psychological reactivity to psychosocial stress. Interestingly, the reactivity of the sympathetic nervous system to stress was revealed to be sensitive to higher levels of physical activity (elite and amateur sportsmen), whereas cortisol responses were attenuated only in elite sportsmen. Future prospective longitudinal studies should evaluate whether an intermediate level of physical activity might induce an optimal stress-protective effect against stress-related diseases.

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Conflict of interest

The authors declare that they have no conflicts of interest.

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