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**Lifetime Stressor Exposure and Psychophysiological Reactivity and Habituation to  
Repeated Acute Social Stressors**

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### Abstract

This study addressed whether lifetime stressor exposure was associated with psychophysiological reactivity and habituation to a novel laboratory-based stressor. Eighty-six participants ( $M_{age} = 23.31$  years,  $SD = 4.94$ ) reported their exposure to lifetime non-sport and sport-specific stressors before completing two consecutive trials of the Trier Social Stress Test while cardiovascular (i.e., heart rate) and endocrine (i.e., salivary cortisol) data were recorded. Exposure to a moderate number of lifetime non-sport and sport-specific stressors was associated with adaptive cardiovascular reactivity, whereas very low or very high stressor exposure was related to maladaptive reactivity. Moreover, experiencing a very low number of lifetime non-sport (but not sport-specific) stressors was associated with poorer habituation. In contrast, lifetime stressor severity was unrelated to cardiovascular reactivity. Finally, greater lifetime non-sport and sport-specific stressor count were associated with blunted cortisol reactivity and poorer habituation. These results suggest that lifetime stressor exposure may influence sport performers' acute stress responses.

*Keywords:* adaptation; adversity; cardiovascular reactivity; cortisol reactivity; stress

## **Lifetime Stressor Exposure and Psychophysiological Reactivity and Habituation to Repeated Acute Social Stressors**

Greater lifetime stressor exposure has been related to more mental (e.g., depression; Slavich et al., 2019) and physical (e.g., respiratory infections; Cazassa et al., 2020) health complaints. One population of particular interest is sport performers, given that the sporting environment imposes numerous stressors on them (Arnold & Fletcher, 2021). Indeed, stressors are a particularly salient feature of sport performers' lives, which can potentially have damaging effects on health, well-being, and performance (Fletcher et al., 2006). Although some research has demonstrated that adverse events can act as a catalyst for positive change (e.g., Howells et al., 2017), recent research has found that exposure to a greater number of lifetime non-sport and sport-specific stressors was associated with more mental and physical health problems including anxiety and respiratory infections (McLoughlin et al., 2022). Collectively, this research also suggests that stressors may degrade health, particularly when they are chronic (vs. acute) or have occurred more recently (vs. in childhood; Lam et al., 2019; McLoughlin et al., 2021). Moreover, experiencing more severe stressors over the life course has been identified as a relatively stronger predictor of ill-health as compared to the total count of such stressors (Slavich et al., 2019; Shields et al., 2022). Despite these findings, the psychophysiological (e.g., cardiovascular, endocrine) mechanisms through which lifetime stressor exposure affects health remain unclear.

One theoretical framework that explains how stressor exposure affects health is the integrative model of lifespan stress and health (Epel et al., 2018). This model comprises three main elements: (a) contextual factors, including individual and environmental factors (e.g., genetics), cumulative life stressor exposure (e.g., past and current stressors), and protective factors (e.g., social, psychological, and behavioural processes); (b) psychophysiological stress responses (e.g., cognitive appraisals, cardiovascular reactivity); and (c) biological aging and

1 disease (e.g., cardiovascular disease). This was chosen as the guiding theory for this study  
2 because it seeks to address the limitations associated with historical theories of stress by  
3 understanding how individual components of the multi-level stress process interact over the  
4 lifespan to impact health (Epel et al., 2018). This is particularly noteworthy given that a missing  
5 factor from many stress and health models (e.g., transactional model of stress and coping;  
6 Lazarus & Folkman, 1984) is cumulative lifetime stressor exposure (i.e., the total count or  
7 severity of all stressors an individual has experienced in their life; Lam et al., 2019).

8         According to the integrative model of lifespan stress and health (Epel et al., 2018),  
9 contextual factors (e.g., socio-economic status) and cumulative stress, together with protective  
10 factors (e.g., social support), shape how people habitually view events and respond to stressors  
11 psychologically and physiologically (e.g., exaggerated reactivity to acute stressors).  
12 Specifically, this model posits that psychophysiological responses to stress may, at least in part,  
13 explain how lifetime stressor exposure portends poor health outcomes. The integrative model  
14 is also useful as it helps to refine key conceptual dimensions of lifetime stressor exposure,  
15 whereby stressors are distinguished by their timing (e.g., early life vs adulthood), type (e.g.,  
16 acute life events vs chronic difficulties), primary life domain (e.g., housing, education, work),  
17 and social-psychological characteristics (e.g., interpersonal loss, physical danger). Consistent  
18 with the definition of lifetime stressor exposure and theory, it is important to assess domain  
19 specific stressors (e.g., sport vs. non-sport) to assess which are particularly salient for sport  
20 performers' health, well-being, and performance (McLoughlin et al., 2021).

21         When exposed to acute stressors (e.g., delivering a speech), the nervous system  
22 responds immediately by activating the sympathetic-adrenal-medullary (SAM) system, which  
23 stimulates increases in heart rate and blood pressure (Turner et al., 2021). The endocrine system  
24 also responds to acute stressors by activating the hypothalamic-pituitary-adrenal (HPA) axis,  
25 which releases cortisol from the adrenal cortex (Chrousos, 2009). An optimal response occurs

1 when these physiological systems are activated and then de-activated quickly, returning the  
2 body to a relaxed state (Turner et al., 2021). However, maladaptive physiological responses  
3 can also occur, characterized by exaggerated or blunted reactivity to an acute stressor or a lack  
4 of habituation when this stressor is repeated (Hughes et al., 2018). Along these lines, extreme  
5 cardiovascular and cortisol stress responses (i.e., exaggerated or blunted) have been associated  
6 with adverse health outcomes, suggesting that a normative response is more optimal (Turner et  
7 al., 2021). For example, exaggerated cardiovascular and cortisol reactivity has been related to  
8 hypertension (e.g., Carroll et al., 2012a), cardiovascular disease (Chida & Steptoe, 2010), and  
9 all-cause mortality (Carroll et al., 2012b). Similarly, blunted cardiovascular and cortisol  
10 reactivity has been related to depression (Brindle et al., 2013), obesity (Phillips et al., 2012),  
11 poorer cognitive function (Ginty et al., 2012), and substance abuse (al'Absi et al., 2021).

12 In examining the antecedents of disproportionate stress reactivity, research has  
13 suggested that experiencing lifetime stressors may alter regulation of the SAM system and  
14 HPA axis (e.g., Carroll et al., 2017; Tyra et al., 2020). For instance, Lovallo (2013) found that  
15 exposure to many psychosocial stressors before 16 years old was associated with blunted  
16 cortisol responses to acute stress. When assessing stressor exposure over the entire lifespan,  
17 Elzinga et al. (2008) found that stressful life events were associated with reduced cortisol  
18 reactivity. However, studies investigating the influence of stressor exposure on cardiovascular  
19 reactivity have yielded mixed results (Ginty & Conklin, 2011). For example, exposure to more  
20 lifetime stressors has been linked with higher (e.g., Lepore et al., 1997; Roy et al., 1998) and  
21 lower (e.g., Matthews et al., 2001; Phillips et al., 2005) cardiovascular reactivity. One potential  
22 explanation for these mixed findings could involve stress appraisals (Blascovich, 2008).  
23 Indeed, a moderate number of stressful life events was associated with a challenge state (i.e.,  
24 relatively higher cardiac output and lower total peripheral resistance reactivity), as compared  
25 to a lower or higher number of events, which was related to a threat state (Moore et al., 2018).

1 To date, however, few studies have examined how stressor exposure over the entire life course  
2 is related to cardiovascular responses to an acute social stressor, and we know of no studies  
3 that have examined how lifetime stressor exposure is related physiological habituation to  
4 repeatedly experiencing the same acute stressor over time.

5 To our knowledge, only one study has examined how cumulative lifetime stressor  
6 exposure is associated with differences in HPA axis reactivity to a single acute stressor (Lam  
7 et al., 2019). This study used the Stress and Adversity Inventory (Adult STRAIN; Slavich &  
8 Shields, 2018) to assess the frequency, exposure timing, duration, and severity of stressors over  
9 the entire life course. The results suggested that greater lifetime stressor exposure was  
10 associated with blunted salivary cortisol reactivity (Lam et al., 2019). However, this study did  
11 not examine how lifetime stressor exposure was related to SAM activation using markers of  
12 cardiovascular reactivity, such as heart rate. Furthermore, Lam et al. (2019) did not investigate  
13 how lifetime stressor exposure is related to differences in physiological reactivity to the same  
14 stressor over time. This is particularly important given that failure to habituate to repeated  
15 stressors has been hypothesized to be a key process leading to allostatic load and increased  
16 disease risk (Hughes et al., 2018; McEwen, 1998).

17 To extend prior literature, this study addressed how lifetime non-sport and sport-  
18 specific stressor exposure was associated with psychophysiological reactivity to an acute  
19 laboratory-based social stressor, as indexed by heart rate and salivary cortisol levels, as well as  
20 habituation when this stressor was repeated. Given that prior research has yielded mixed results  
21 (see Turner et al., 2021), we tested several possible models examining linear, quadratic, and  
22 cubic relationships between lifetime stressor exposure and the psychophysiological outcomes  
23 assessed—namely heart rate and salivary cortisol.

## 24 **Method**

### 25 **Participants**

1           An *a priori* power calculation using G\*Power software revealed that a minimum  
2 sample of 77 participants was required to perform multiple regression analyses. The effect size  
3 entered into this calculation was based on the medium effect ( $\beta = 0.30$ ) reported between  
4 lifetime stressor exposure and cortisol reactivity in Lam et al. (2019), with an alpha of 0.05 and  
5 power of 0.80. In total, 86 sport performers (45 female, 41 male;  $M_{age} = 23.31$  years,  $SD =$   
6  $4.94$ ) were recruited. Participants competed in a variety of individual (e.g., tennis) and team  
7 (e.g., rugby) sports, and had an average of 8.35 years ( $SD = 6.07$ ) experience in their sport.  
8 Participants competed in their sports at various levels, with 2% performing at senior  
9 international, 11% at international, 16% at national, 15% at regional, 26% at university, 7% at  
10 county, and 23% at club level. Participants were not able to take part if they had any known  
11 respiratory or cardiovascular conditions, a history of diabetes, were currently ill, injured or  
12 pregnant, at high-risk of infection, or taking a medication that increases infection risk (e.g.,  
13 steroids), smoked (i.e., at least one cigarette per day), or were obese (i.e., body mass index  
14  $>30\text{kg/m}^2$ ).

## 15   **Procedure**

16           Following institutional ethical approval, participants were recruited using convenience  
17 sampling methods using the researchers' existing contacts and university sports clubs. Prior to  
18 taking part, participants were advised of their ethical rights (e.g., anonymity, right to withdraw)  
19 using an information sheet and provided written informed consent. Once recruited, participants  
20 completed a ~20-minute online questionnaire assessing lifetime stressor exposure at least 48  
21 hours prior to their laboratory visit to ensure that answers did not influence their acute stress  
22 responses.

23           To minimise the impact of diurnal variability in salivary cortisol, participants were  
24 invited to the laboratory between 13:00-18:00 for a 3-hour laboratory session. To rule out any  
25 exogenous effects on cardiovascular and endocrine measures, participants were asked to

1 abstain from caffeine, alcohol, and moderate-to-vigorous exercise for 24 hours. Additionally,  
2 participants were asked to refrain from eating or drinking anything other than water for 4 hours  
3 prior to their visit. After providing informed consent, an impedance cardiograph and blood  
4 pressure monitor were fitted. Participants were then asked to sit and relax for 20 minutes to  
5 gather baseline cardiovascular data. At the end of this rest period, participants provided a saliva  
6 sample for the assessment of cortisol activity. Next, the researcher gave instructions about the  
7 Trier Social Stress Test (TSST; Kirschbaum et al., 1993). Following five minutes of  
8 preparation, participants performed the TSST. Participants were then instructed to relax for 20  
9 minutes, before providing a saliva sample ~20 minutes after completing the TSST. This TSST  
10 protocol was then repeated to enable the assessment of habituation, with a 10-minute rest period  
11 enforced between trials (see Supplementary Materials).

## 12 **Measures**

### 13 *Lifetime Non-Sport Stressor Exposure*

14 Lifetime non-sport stressor exposure was assessed using the Adult STRAIN (Slavich  
15 & Shields, 2018), which assesses 55 major life stressors including 26 acute life events (e.g.,  
16 death of a loved one) and 29 chronic difficulties (e.g., ongoing health problems). Once a  
17 stressor is endorsed, follow-up questions are asked to assess the stressor's severity (1 = *not at*  
18 *all to 5 = extremely*), frequency (1 to 5 or more times), exposure timing (1 = *ongoing to 7 =*  
19 *over 5 years ago*), and duration (*years and/or months*). Analyses were conducted using the  
20 STRAIN's two main outcome variables: (a) total count of lifetime stressors (range = 0-166)  
21 and (b) total severity of lifetime stressors (range = 0-265), with higher scores indicating greater  
22 lifetime stressor burden. The Adult STRAIN has demonstrated excellent test-retest reliability  
23 over one month ( $r_s = 0.90$  to  $0.95$ ), and very good concurrent ( $r_s = 0.15$  to  $0.62$ ) and predictive  
24 validity in relation to a variety of health-related outcomes, including general mental and  
25 physical health complaints, doctor-diagnosed health problems and autoimmune disorders, and



1 anxiety and depression symptom severity, as well as positive and negative health behaviours,  
2 impulsivity and addictive behaviours, birth intendedness, sleep difficulties, executive  
3 dysfunction, neural reactivity and connectivity, and cardiovascular, metabolic, and immune  
4 function (e.g., Cazassa et al., 2020; McMullin et al., 2021; Olvera Alvarez et al., 2019).

### 5 ***Lifetime Sport-Specific Stressor Exposure***

6 Lifetime sport-specific stressor exposure was assessed using the STRAIN's Sport  
7 Stress Assessment Module (Sport SAM; McLoughlin et al., 2022), which assesses eight  
8 stressors that are commonly encountered by sport performers (e.g., underperformance). Once  
9 a stressor is endorsed, follow-up questions are asked to determine its severity (1 = *not at all* to  
10 5 = *extremely*), frequency (1 to 5 or more times), exposure timing (1 = *ongoing* to 7 = *over 5*  
11 *years ago*), and duration (*years and/or months*). Analyses were based on the Sport SAM's two  
12 main outcome variables: (a) total count of sport-specific stressors (range = 0-24), and (b) total  
13 severity of sport-specific stressors (range = 0-40). The Sport SAM has demonstrated good  
14 usability and acceptability, concurrent ( $r_s = 0.23$  to  $0.29$ ) and predictive ( $r_s = 0.13$  to  $0.32$ )  
15 validity, and very good test-retest reliability ( $r_{icc} = 0.87$  to  $0.89$ ; McLoughlin et al., 2022).

### 16 ***Heart Rate***

17 A non-invasive impedance cardiograph device (PhysioFlow Enduro, PF07, Manatec  
18 Biomedical, France) estimated heart rate. The PhysioFlow provides hemodynamic parameters  
19 using analysis of trans-thoracic bioimpedance signals. Specifically, it measures the change in  
20 impedance through a high frequency alternating electrical current (66 kHz) of low magnitude  
21 (4.5 mA peak to peak) using electrodes. Following skin preparation, six spot electrodes  
22 (BlueSensor R, Ambu, Ballerup, Denmark) were placed on the left side of the neck, the middle  
23 of the sternum, the rib closest to V6, on the xiphoid process, and laterally on the rib. After  
24 details were entered (e.g., body mass), the Physioflow was calibrated over 30 heart cycles while  
25 participants sat quietly resting in an upright position. To complete calibration and ensure

1 accurate estimates of heart rate, an ambulatory blood pressure monitor (Welch Allyn, 7100,  
2 Germany) was used to obtain systolic and diastolic blood pressure estimates every two minutes,  
3 which were regularly inputted into the PhysioFlow. Due to the non-continuous nature of the  
4 blood pressure measurement, we did not include blood pressure data in any analysis. The  
5 Physioflow has been validated and has been used in >100 peer-reviewed publications (see  
6 Charloux et al. 2000; Richard et al. 2001), and the literature suggests that its accuracy is  
7 comparable to invasive techniques, and its clinical reproducibility and sensitivity are excellent.

### 8 *Salivary Cortisol*

9 Participants provided four saliva samples, collected over a four-minute period, using a  
10 passive drool method (Navazesh, 1993). Specifically, participants allowed saliva to accumulate  
11 on the floor of their mouth without stimulation by orofacial movement or swallowing, before  
12 drooling saliva into pre-weighed 15 mL centrifuge tubes at ~60-s intervals. Samples were  
13 stored at 4°C for up to 24 hours prior to being centrifuged at 2000 ×g for 10 minutes to remove  
14 particulate matter. The supernatant was aliquoted into micro-centrifuge tubes (Eppendorf,  
15 Hamburg, Germany), and were then placed into a freezer and stored at -20°C until analysis.  
16 Salivary cortisol was analysed in duplicate using enzyme-linked immuno-sorbent assay  
17 (ELISA) kits according to the manufacturer's instructions (Salimetrics, Philadelphia, PA,  
18 United States). Absorbance values were measured using a microplate reader  
19 (SPECTROstarNano; BMG Labtech, Ortenberg, Germany). The inter- and intra-assay  
20 coefficients of variation for salivary cortisol were 5.63% and 7.07%, respectively.

### 21 *Laboratory-Based Social Stressor*

22 A modified version of the TSST was used to induce acute stress (Labuschagne et al.,  
23 2019). The TSST includes a 5-minute speech task, followed by a 5-minute mental arithmetic  
24 task. For the speech portion of the TSST, participants were given 5 minutes to prepare and then  
25 5 minutes to deliver a speech to “senior management” describing why they would be “the

1 perfect applicant for the vacant position”. They were told that their speech would be videotaped  
2 “so that a video analysis of behaviours, voice frequency, and performance may be conducted.”  
3 Following the speech, participants performed a 5-minute mental arithmetic task in which they  
4 were told to start at 1022 and “serially subtract the number 13 as fast and as accurately as  
5 possible.” They were told to stop and start again at 1022 if they paused, counted too slowly, or  
6 made an error.

7 To assess habituation, participants performed the TSST again following a 50-minute  
8 rest period. This rest period was longer than that used in prior stress habituation research to  
9 ensure that cortisol levels returned to normal (e.g., Hughes et al., 2018). On the second TSST  
10 trial, participants gave a speech about a sporting situation that they found particularly stressful  
11 (as Meijen et al., 2013). If a participant stopped talking before the 5 minutes had elapsed,  
12 identical prompts were used to those in the first speech. Next, participants were given identical  
13 instructions to those used in the first mental arithmetic task but were asked to start at 3891 and  
14 count backwards in increments of 13 (see Supplementary Materials).

### 15 **Data Analyses**

16 Mean values were computed for heart rate and salivary cortisol at each time point (i.e.,  
17 Baseline 1, TSST 1, Baseline 2, TSST 2). Reactivity scores were calculated by subtracting  
18 baseline values from stressor exposure values. This generated two reactivity scores for heart  
19 rate and cortisol, one for each TSST trial. Habituation was calculated by subtracting reactivity  
20 2 values from reactivity 1 values. Zero or positive values indicated more successful habituation,  
21 whereas negative values indicated poorer habituation (as Lü et al., 2016). Theoretically,  
22 successful habituation can be defined as the reduction of exaggerated cardiovascular responses  
23 to mid-range ones that are then maintained for subsequent exposures to the stressor concerned  
24 (Hughes et al., 2018).

1 Data were analyzed using IBM SPSS software. To ensure data were normally  
2 distributed, outlier analyses were performed. This analysis revealed 26 univariate outliers (i.e.,  
3 z-scores greater or lesser than 1.96), which were Winsorized to a value 1% larger or smaller  
4 than the next most extreme score (Field, 2018). Supplementary analyses were conducted on  
5 the Winsorized and non-Winsorized data. In these supplementary analyses, the hierarchical  
6 regression results did not substantially differ when non-Winsorized data was inputted into the  
7 statistical analysis. Given the benefits associated with performing statistical analyses on  
8 normally distributed data (e.g., to reduce bias in standard errors; Field, 2018), we have chosen  
9 to report the Winsorized data in the manuscript. As a result, data were normally distributed  
10 (i.e., skewness and kurtosis  $z$  scores did not exceed 1.96). After calculating descriptive  
11 statistics, a series of repeated measures ANOVAs with post-hoc paired samples  $t$ -tests assessed  
12 changes in heart rate and cortisol over time. These analyses checked that both trials of the TSST  
13 initiated a stress response. Throughout these analyses, sphericity violations were corrected  
14 using Greenhouse–Geisser corrections where appropriate. Alpha was set at 0.05, and partial  
15 eta-squared ( $\eta_p^2$ ) effect sizes were calculated with values of .01, .06, and .14 reflecting small,  
16 medium, and large effects, respectively.

17 To examine the relationships between lifetime non-sport and sport-specific stressor  
18 exposure and psychophysiological outcomes, hierarchical regression analyses were conducted.  
19 Mean centered lifetime non-sport or sport-specific stressor count or severity was entered at step  
20 1, the quadratic term (stressor count or severity<sup>2</sup>) was entered at step 2, and the cubic term  
21 (stressor count or severity<sup>3</sup>) was entered at step 3<sup>1</sup>. The significance of the additional variance  
22 explained in the outcomes at each step was assessed. This approach allowed us to assess  
23 additional bends in the modelled curve, accounting for the influence of a small number of

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<sup>1</sup> Regression analyses were repeated while controlling for potential confounding variables (i.e., age, sex, and competitive level). Notably, controlling for these variables did not alter the results.

1 extreme values (Seery et al., 2013). To explore significant quadratic or cubic components, the  
2 linear simple slopes at the following levels of stressor exposure were assessed: 2 SDs and 1SD  
3 below the mean, at the mean, and 1 SD and 2 SDs above the mean, representing very low, low,  
4 moderate, high, and very high lifetime stressor exposure, respectively<sup>2</sup>. This post-hoc probing  
5 used values from the variance-covariance matrix of the regression coefficients to calculate the  
6 standard errors of the regression slopes and their 95% confidence intervals (CIs). The slopes  
7 were significant if their 95% CIs did not cross zero.

## 8 **Results**

### 9 **Descriptive Statistics**

10 The means and *SDs* of cardiovascular (heart rate) and endocrine (salivary cortisol)  
11 measures assessed across all time points from pre- to post-TSST are presented in Figures 1a  
12 and 1b, respectively. Values show mean heart rate and salivary cortisol concentration, and error  
13 bars represent standard deviations. Participants experienced an average of 17 stressors over  
14 their lifetime (range = 0-54), including an average of 12 non-sport stressors (range = 0-35) and  
15 five sport-specific stressors (range = 0-19). The mean cumulative lifetime (non-sport) stressor  
16 severity was 27.07 (*SD* = 17.70; range = 0-265), and mean cumulative lifetime (sport-specific)  
17 stressor severity was 8.28 (*SD* = 7.19; range = 0-40).

### 18 **Cardiovascular and Endocrine Reactivity to the Acute Stressor**

19 The first TSST significantly increased heart rate,  $F(1.78, 151.42) = 126.34, p < .001,$   
20  $\eta_p^2 = .60,$  and cortisol,  $F(1.97, 167.35) = 20.68, p < .001, \eta_p^2 = .19.$  Post-hoc analyses showed  
21 that heart rate was significantly higher during TSST 1 and TSST 2 than the preceding baselines  
22 ( $ps < .001$ ). Although salivary cortisol concentration levels did not change from Baseline 1 to  
23 TSST 1 ( $p = .661$ ), it significantly decreased from Baseline 2 to TSST 2 ( $p < .001$ ).

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<sup>2</sup> To further support these findings, we cross-validated the results by randomly splitting the sample in two and ran the multiple regression analyses twice (i.e., one on each sample) and the results largely followed the same pattern as to those reported in the present study.

## 1 **Lifetime Stressor Exposure and Psychophysiological Reactivity to an Acute Social** 2 **Stressor**

### 3 *Lifetime Non-Sport Stressor Count*

4 **Heart rate.** Beyond non-significant linear ( $R^2 = .007, p = .442$ ) and quadratic ( $\Delta R^2 =$   
5  $.007, p = .457$ ) components, a significant cubic ( $\Delta R^2 = .086, p = .006$ ) relationship was observed  
6 between total count of lifetime non-sport stressors and heart rate reactivity (see Figure 2a).  
7 Within this model, there was a significant cubic relationship at mean total count of lifetime  
8 non-sport stressors ( $b = 0.10, p = .006, sr^2 = .09$ ). The slope of this curve was significant and  
9 positive 2 *SDs* (slope = 4.07, 95% CI [1.59, 6.55]) and 1 *SD* (slope = 0.62, 95% CI [0.29, 0.94])  
10 below the mean, significant and negative at the mean (slope = -0.75, 95% CI [-1.28, -0.21]),  
11 not significant 1 *SD* above the mean (slope = -0.02, 95% CI [-0.44, 0.40]), and significant and  
12 positive 2 *SDs* above the mean (slope = 2.79, 95% CI [0.82, 4.76]). Thus, very low lifetime  
13 non-sport stressor count was associated with more blunted heart rate reactivity (~2 bpm), while  
14 low and very high stressor count was linked with more exaggerated reactivity (~13 bpm).

15 **Salivary cortisol.** There were no significant linear ( $R^2 = .038, p = .071$ ), quadratic ( $\Delta R^2$   
16  $= .006, p = .478$ ), or cubic ( $\Delta R^2 = .006, p = .459$ ) relationships between total count of lifetime  
17 non-sport stressors and cortisol reactivity.

### 18 *Lifetime Non-Sport Stressor Severity*

19 **Heart rate.** There were no significant linear ( $R^2 = .018, p = .224$ ), quadratic ( $\Delta R^2 =$   
20  $.001, p = .818$ ), or cubic ( $\Delta R^2 = .038, p = .074$ ) relationships between total severity of  
21 lifetime non-sport stressors and heart rate reactivity.

22 **Salivary cortisol.** Despite non-significant quadratic ( $\Delta R^2 = .000, p = .952$ ) and cubic  
23 ( $\Delta R^2 = .174, p = .678$ ) components, a significant linear relationship ( $\Delta R^2 = .052, \beta = -.23, p =$   
24  $.034$ ) was found between total severity of lifetime non-sport stressors and cortisol reactivity.  
25 Thus, greater lifetime non-sport stressor severity was related to more blunted cortisol reactivity.

### 1 *Lifetime Sport-Specific Stressor Count*

2       **Heart rate.** Beyond non-significant linear ( $R^2 = .009, p = .386$ ) and quadratic ( $\Delta R^2 =$   
3  $.000, p = .879$ ) components, a significant cubic ( $\Delta R^2 = .117, p = .001$ ) relationship was observed  
4 between total count of lifetime sport-specific stressors and heart rate reactivity (see Figure 2b).  
5 Within this model, there was a significant cubic relationship at mean total count of lifetime  
6 sport-specific stressors ( $b = -0.054, p = .001, sr^2 = .12$ ). The slope of this curve was significant  
7 and negative 2 *SDs* (slope = -10.35, 95% CI [-14.39, -6.30]) and 1 *SD* (slope = -2.07, 95% CI  
8 [-3.33, -0.80]) below the mean, significant and positive at the mean (slope = 1.49, 95% CI  
9 [0.60, 2.39]), not significant 1 *SD* above the mean (slope = 0.34, 95% CI [-1.02, 1.71]), and  
10 significant and negative 2 *SDs* above the mean (slope = -5.52, 95% CI [-10.23, -0.81]). Thus,  
11 both very low and high lifetime sport-specific stressor count were associated with more  
12 exaggerated heart rate reactivity, while very high stressor count was linked with more blunted  
13 reactivity.

14       **Salivary cortisol.** Beyond non-significant linear ( $R^2 = .003, p = .634$ ) and quadratic  
15 ( $\Delta R^2 = .012, p = .324$ ) components, a significant cubic ( $\Delta R^2 = .082, p = .008$ ) relationship was  
16 observed between total count of lifetime sport-specific stressors and cortisol reactivity (see  
17 Figure 2c). Within this model, there was a significant cubic relationship at mean total count of  
18 lifetime sport-specific stressors ( $b = -0.01, p = .008, sr^2 = .08$ ). The slope of this curve was  
19 significant and negative 2 *SDs* (slope = -0.16, 95% CI [-0.26, -0.05]) and 1 *SD* (slope = -0.03,  
20 95% CI [-0.05, -0.003]) below the mean, significant and positive at the mean (slope = 0.02,  
21 95% CI [0.004, 0.03]), and significant and negative 1 *SD* (slope = -0.01, 95% CI [-0.02, -0.003])  
22 and 2 *SDs* (slope = -0.13, 95% CI [-0.20, -0.06]) above the mean. Thus, very low lifetime sport-  
23 specific stressor count was associated with increases in cortisol concentration, while very high  
24 stressor count was related to decreases in cortisol concentration.

### 25 *Lifetime Sport-Specific Stressor Severity*

1           **Heart rate.** There were no significant linear ( $R^2 = .004, p = .541$ ), quadratic ( $\Delta R^2 =$   
2            $.000, p = .961$ ), or cubic ( $\Delta R^2 = .011, p = .336$ ) relationships between total severity of lifetime  
3           sport-specific stressors and heart rate reactivity.

4           **Salivary cortisol.** There were no significant linear ( $R^2 = .001, p = .761$ ), quadratic ( $\Delta R^2 =$   
5            $.000, p = .895$ ), or cubic ( $\Delta R^2 = .026, p = .144$ ) relationships between total severity of lifetime  
6           sport-specific stressors and cortisol reactivity.

## 7           **Lifetime Stressor Exposure and Psychophysiological Habituation to a Repeated Acute** 8           **Social Stressor**

### 9           *Lifetime Non-Sport Stressor Count*

10           **Heart rate.** Beyond non-significant linear ( $R^2 = .017, p = .236$ ) and quadratic ( $\Delta R^2 =$   
11            $.004, p = .553$ ) components, a significant cubic ( $\Delta R^2 = .059, p = .025$ ) relationship was observed  
12           between total count of lifetime non-sport stressors and heart rate habituation (see Figure 2d).  
13           Within this model, there was a significant cubic relationship at mean total count of lifetime  
14           non-sport stressors ( $b = .005, p = .025, sr^2 = .06$ ). The slope of this curve was significant and  
15           positive 2 SDs below the mean (slope = 2.20, 95% CI [0.11, 4.29]), but not significant 1 SD  
16           below the mean (slope = 0.47, 95% CI [-0.18, 1.12]), at the mean (slope = -0.22, 95% CI [-  
17           0.58, 0.14]), or 1 SD (slope = 0.14, 95% CI [-0.40, 0.68]) and 2 SDs (slope = 1.54, 95% CI [-  
18           0.28, 3.36]) above the mean. Thus, very low lifetime non-sport stressor count was associated  
19           with poorer habituation, while low, moderate, high, and very high lifetime stressor count were  
20           linked with more successful habituation to repeated exposure to the acute laboratory-based  
21           social stressor.

22           **Salivary cortisol.** Despite non-significant quadratic ( $\Delta R^2 = .000, p = .911$ ) and cubic  
23           ( $\Delta R^2 = .010, p = .339$ ) components, a significant linear relationship ( $\Delta R^2 = .070, \beta = -.27, p =$   
24            $.014$ ) was found between total count of lifetime non-sport stressors and cortisol habituation.



1 Thus, greater lifetime stressor exposure was associated with more blunted cortisol habituation  
2 to repeated exposure to the acute laboratory-based social stressor.

### 3 *Lifetime Non-Sport Stressor Severity*

4 **Heart rate.** There were no significant linear ( $R^2 = .004, p = .580$ ), quadratic ( $\Delta R^2 =$   
5  $.002, p = .675$ ), or cubic ( $\Delta R^2 = .014, p = .276$ ) relationships between total severity of lifetime  
6 non-sport stressors and heart rate habituation to repeated exposure to the acute laboratory-based  
7 social stressor.

8 **Salivary cortisol.** Despite non-significant quadratic ( $\Delta R^2 = .002, p = .661$ ) and cubic  
9 ( $\Delta R^2 = .004, p = .555$ ) components, a significant linear relationship ( $\Delta R^2 = .089, \beta = -.30, p =$   
10  $.005$ ) was found between total severity of lifetime non-sport stressors and cortisol habituation.  
11 Thus, greater lifetime stressor severity was related to more blunted cortisol habituation to  
12 repeated exposure to the acute laboratory-based social stressor.

### 13 *Lifetime Sport-Specific Stressor Count*

14 **Heart rate.** Beyond non-significant linear ( $R^2 = .020, p = .196$ ) and quadratic ( $\Delta R^2 =$   
15  $.013, p = .291$ ) components, a significant cubic ( $\Delta R^2 = .072, p = .012$ ) relationship was observed  
16 between total count of lifetime sport-specific stressors and heart rate habituation (see Figure  
17 2e). Within this model, there was a significant cubic relationship at mean total count of lifetime  
18 sport-specific stressors ( $b = -0.028, p = .012, sr^2 = .07$ ). The slope of this curve was significant  
19 and negative 2 *SDs* (slope = -6.02, 95% CI [-7.69, -4.36]) and 1 *SD* (slope = -1.40, 95% CI [-  
20 2.19, -0.61]) below the mean, significant and positive at the mean (slope = 0.78, 95% CI [0.17,  
21 1.38]), and significant and negative 1 *SD* (slope = 0.51, 95% CI [0.04, 0.97]) and 2 *SDs* (slope  
22 = -2.20, 95% CI [-3.25, -1.16]) above the mean. Despite differences in the direction and  
23 magnitude of the slope of this curve, however, all values were above zero; therefore, all levels  
24 of lifetime sport-specific stressor count were associated with successful habituation to repeated  
25 exposure to the acute laboratory-based social stressor.

1           **Salivary cortisol.** Beyond non-significant linear ( $R^2 = .010, p = .351$ ) and quadratic  
2 ( $\Delta R^2 = .019, p = .201$ ) components, a significant cubic ( $\Delta R^2 = .096, p = .004$ ) relationship was  
3 observed between total count of lifetime sport-specific stressors and cortisol habituation (see  
4 Figure 2f). Within this model, there was a significant cubic relationship at mean total count of  
5 lifetime sport-specific stressors ( $b = -0.001, p = .004, sr^2 = .10$ ). The slope of this curve was  
6 significant and negative 2 *SDs* below the mean (slope = -0.15, 95% CI [-0.28, -0.02]), not  
7 significant 1 *SD* below the mean (slope = -0.02, 95% CI [-0.05, 0.005]), significant and positive  
8 at the mean (slope = 0.03, 95% CI [0.007, 0.04]), not significant 1 *SD* above the mean (slope =  
9 -0.007, 95% CI [-0.02, 0.007]), and significant and negative 2 *SDs* above the mean (slope = -  
10 0.12, 95% CI [-0.20, -0.04]). Thus, very high lifetime sport-specific stressor count was  
11 associated with poorer habituation, while very low, low, moderate, and high stressor count  
12 were linked with more successful habituation to repeated exposure to the acute laboratory-  
13 based social stressor.

#### 14 *Lifetime Sport-Specific Stressor Severity*

15           **Heart rate.** There were no significant linear ( $R^2 = .000, p = .839$ ), quadratic ( $\Delta R^2 =$   
16  $.024, p = .155$ ), or cubic ( $\Delta R^2 = .001, p = .777$ ) relationships between total severity of lifetime  
17 sport-specific stressors and heart rate habituation to repeated exposure to the acute laboratory-  
18 based social stressor.

19           **Salivary cortisol.** Finally, there were no significant linear ( $R^2 = .007, p = .446$ ),  
20 quadratic ( $\Delta R^2 = .002, p = .677$ ), or cubic ( $\Delta R^2 = .016, p = .243$ ) relationships between total  
21 severity of lifetime sport-specific stressors and cortisol habituation to repeated exposure to the  
22 acute laboratory-based social stressor.

#### 23 **Discussion**

24           Despite growing interest in how lifetime stressor exposure affects mental and physical  
25 health-related outcomes such as depression and hypertension, the mechanisms underlying this

1 relationship remain unclear (Whittaker et al., 2021). Moreover, to date, no studies have  
2 examined how lifetime stressor exposure is associated with psychophysiological reactivity to  
3 a novel acute stressor, or habituation when this stressor is repeated, which may improve our  
4 understanding of how lifetime stressors affect disease risk (McEwen, 1998). Additionally, we  
5 are not aware of any studies that have examined if the context in which stressors are  
6 experienced is important in shaping psychophysiological stress responses. This is particularly  
7 important given that recent theory and empirical research hypothesizes that individual and  
8 environmental contextual factors shape how individuals habitually view events and respond to  
9 stressors both psychologically and physiologically (Epel et al., 2018). Thus, this study  
10 addressed how lifetime non-sport and sport-specific stressor exposure was related to  
11 psychophysiological reactivity and habituation to an acute laboratory-based social stressor and  
12 habituation when this stressor was repeated. Overall, the results suggest that lifetime stressor  
13 exposure may influence sport performers' acute stress responses. It should be noted, that  
14 despite the significant findings discussed, the effect sizes for linear and cubic effects were  
15 typically small (e.g., significant cubic effects ranged from  $\Delta R^2 = 0.05$  to 0.12) but even these  
16 may hold practical relevance in applied or clinical settings (Slavich, 2020,2022).

### 17 **Psychophysiological Reactivity to an Acute Social Stressor**

18 Consistent with Social Safety Theory (Slavich 2020, 2022), a significant cubic  
19 relationship was found between lifetime non-sport stressor count and heart rate reactivity.  
20 Specifically, participants who had experienced very low lifetime non-sport stressor count  
21 exhibited more blunted heart rate reactivity, whereas those who experienced both low and very  
22 high lifetime non-sport stressor counts exhibited more exaggerated heart rate reactivity. These  
23 findings suggest that experiencing a moderate or high number of lifetime non-sport stressors  
24 may promote more adaptive cardiovascular reactivity, whilst exposure to a very low, low, or  
25 very high number of stressors may lead to more maladaptive reactivity. These findings are thus

1 consistent with Social Safety Theory and with prior research showing that moderate stressor  
2 exposure is associated with greater resilience to stressors (Seery et al., 2010; Shapero et al.,  
3 2015). This is particularly important given that both exaggerated and blunted cardiovascular  
4 stress responses have been linked with negative health outcomes, including cardiovascular  
5 disease (Carroll et al., 2017; Turner et al., 2021). In contrast, the severity of lifetime non-sport  
6 stressors was unrelated to heart rate reactivity, possibly due to participant characteristics (e.g.,  
7 oral contraceptive use) that could have masked associations between lifetime stressor exposure  
8 and the outcomes assessed here (Straneva et al., 2000).

9         A significant cubic relationship was also found between lifetime sport-specific stressor  
10 count and heart rate reactivity. Specifically, participants who had experienced very low and  
11 high lifetime sport-specific stressor count both exhibited more exaggerated heart rate reactivity.  
12 In contrast, those who experienced very high lifetime sport-specific stressor count exhibited  
13 more blunted heart rate reactivity. These results suggest that experiencing a moderate number  
14 of lifetime sport-specific stressors may promote more adaptive cardiovascular reactivity.  
15 Theory and empirical research have suggested that moderate exposure to general stressors  
16 occurring over the life course may provide individuals with opportunities to develop personal  
17 resources (e.g., adaptive coping strategies) that can enhance the ability to cope with future  
18 stressors (e.g., Dienstbier, 1989; Dooley et al., 2017; Slavich, 2020). These data yielded  
19 polarised results to those observed for lifetime non-sport stressor count, suggesting, for the first  
20 time, that the context in which a stressor is experienced could be an important dimension for  
21 future research to consider when examining the stress-health relationship (Arnold & Fletcher,  
22 2021). One potential explanation for these contrasting results could be because the stressors  
23 commonly experienced in sport are generally more predictable and controllable (e.g., poor  
24 performance, deselection), as opposed to the stressors experienced in their personal lives (e.g.,  
25 undergoing a personal experience of physical or verbal abuse; Lazarus & Folkman, 1986).

1 There was, however, a lack of association between lifetime sport-specific stressor severity and  
2 heart rate reactivity, adding to the consistent null relationship observed between lifetime  
3 stressor severity and cardiovascular reactivity to the acute stressor.

4 Lifetime non-sport stressor count was not related to salivary cortisol reactivity.  
5 However, a significant negative relationship existed between lifetime non-sport stressor  
6 severity and cortisol reactivity, suggesting that experiencing more severe lifetime stressors was  
7 linked with more blunted reactivity. This finding is consistent with prior research suggesting  
8 that adversity predicts blunted cortisol reactivity to psychosocial stress (see Brindle et al.,  
9 2022). Despite this, in contrast to the present results, one prior study found that cortisol  
10 reactivity is more strongly associated with lifetime stressor count than severity (Lam et al.,  
11 2019). Although blunted psychophysiological stress responses have been found to be  
12 potentially beneficial in the short-term, they are considered maladaptive in the long-term as  
13 they have been prospectively related to obesity (e.g., Phillips, 2011) and depression (e.g.,  
14 Carroll et al., 2017). Therefore, these findings extend prior research by suggesting that the  
15 severity of lifetime non-sport stressors may alter regulation of the HPA axis (McEwen, 1998).

16 A significant cubic relationship existed between total count of lifetime sport-specific  
17 stressors and salivary cortisol reactivity. Specifically, only those who had experienced very  
18 low lifetime sport-specific stressor count exhibited increases in cortisol concentration in  
19 reaction to the laboratory-based social stressor. In contrast, participants who reported a very  
20 high lifetime sport-specific stressor count exhibited decreases in cortisol concentration. This  
21 finding is consistent with prior research reporting that greater lifetime stressor exposure is  
22 associated with more blunted responses to acute stressors (Shapero et al., 2015). Therefore,  
23 these results provide evidence that moderate lifetime stressor exposure may help to buffer an  
24 individual against the negative effects of future stressors (Dooley et al., 2017). Although this  
25 finding is consistent with prior research examining lifetime non-sport stressor exposure (e.g.,

1 Lam et al., 2019), our research advances extant literature by examining the role of sport-  
2 specific stressors. Finally, the results also suggest that greater lifetime non-sport stressor  
3 severity was associated with more blunted cortisol reactivity to an acute social stressor.

#### 4 **Psychophysiological Habituation to a Repeated Acute Stressor**

5       When investigating whether participants habituated to the acute stressor when it was  
6 repeated (i.e., TSST), a significant cubic relationship was found between lifetime non-sport  
7 stressor count and heart rate habituation. Specifically, experiencing a very low number of  
8 lifetime non-sport stressors was associated with poorer habituation to acute social stress. This  
9 may be problematic given that an insufficient ability to habituate to repeated stressor exposure  
10 has been hypothesized to be a key physiological profile that increases risk for allostatic load  
11 (McEwen, 1998). Although potentially adaptive in the short term, allostatic load is  
12 hypothesized to accelerate the onset of disease and increase mortality risk (McEwen & Seeman,  
13 1999). In combination with prior research, these findings help advance our understanding of  
14 the determinants of habituation, suggesting that lifetime stressor exposure may be an important  
15 factor that influences how well an individual adapts to repeated social stressors over time.

16       In addition, a significant cubic relationship existed between lifetime sport-specific  
17 stressor count and heart rate habituation. Despite differences in the direction and magnitude of  
18 the slope of this curve, all values were above zero, indicating successful habituation. One  
19 potential explanation for this finding could be that sport performers are better able to adapt to  
20 acute social stressors, given that lifetime stressors classified as ‘sport-specific’ are not *typically*  
21 as long-lasting or impactful as compared with lifetime non-sport stressors (e.g., death of a  
22 family member; McLoughlin et al., 2022). However, the results also suggest no significant  
23 relationship between lifetime sport-specific stressor severity and heart rate habituation, a  
24 finding that contributes to the predominately null effects of lifetime stressor severity on  
25 psychophysiological outcomes observed in the present study.

1           With regards to HPA axis habituation, significant negative relationships existed  
2 between lifetime non-sport stressor exposure and salivary cortisol habituation for both stressor  
3 count and severity. These results suggest that exposure to more and more severe non-sport  
4 stressors across the life course is linked with poorer cortisol habituation. These results can be  
5 likened to the ‘persistent blunter’ typology within the habituation hypothesis by Hughes et al.  
6 (2018), which is characterised by a blunted (or low) response to a first stressor exposure that  
7 remains low during a second exposure. Crucially, failure to habituate has been linked with poor  
8 health outcomes (e.g., depression; Kudielka et al., 2006). To our knowledge, this is the first  
9 study to examine how lifetime non-sport stressor exposure shapes HPA axis habituation to  
10 repeated acutely stressful tasks, thus adding unique information to the literature.

11           A significant cubic relationship was observed between lifetime sport-specific stressor  
12 count and salivary cortisol habituation. Specifically, participants exposed to a very high  
13 number of lifetime sport-specific stressors tended to habituate poorly to the repeated acutely  
14 stressful tasks. Therefore, the present study extends extant literature by suggesting that lifetime  
15 sport-specific stressor exposure may predict HPA axis habituation (Roos et al., 2019). In  
16 contrast to stressor count, lifetime sport-specific stressor severity was unrelated to cortisol  
17 habituation. This pattern of results (i.e., count vs. severity) is consistent with prior research  
18 showing that lifetime stressor count, not severity, predicts cortisol reactivity to acute stress  
19 (Lam et al., 2019). Our results therefore extend this finding to habituation to repeated acute  
20 stressors.

### 21 **Strengths, Limitations, and Implications**

22           Several strengths of this study are notable. First, we used a state-of-the-art instrument  
23 for assessing lifetime stressor exposure (i.e., STRAIN; Slavich & Shields, 2018), a well-  
24 validated laboratory task for inducing acute social stress responses (i.e., TSST; Labuschagne  
25 et al., 2019), and a novel study design that enabled us to assess both reactivity and habituation

1 to a laboratory-based social stressor. In doing so, this is the first study to address how lifetime  
2 non-sport and sport-specific stressors are related to psychophysiological responses to a  
3 repeated laboratory-based social stressor.

4         Despite these strengths, several limitations should also be noted. First, participants were  
5 relatively young ( $M_{age} = 23.31$  years,  $SD = 4.94$ ), which could have limited variability in the  
6 number and severity of stressors experienced. Therefore, future studies should examine the  
7 generalizability of these findings in a more diverse sample. Second, this study collected data  
8 from participants who represented a range of competitive levels, which could have influenced  
9 the findings. Future research should thus examine whether these results differ when data is  
10 collected from a sample of participants with more homogenous characteristics (e.g., athletes  
11 competing at different levels). Third, the trials of the acutely stressful task in this study were  
12 not counterbalanced, and this could have contributed to an order-effect (e.g., learning). Fourth,  
13 this research assessed a relatively limited number of outcomes (e.g., heart rate, cortisol), and  
14 future studies are needed to investigate how lifetime stressors affect other health-related, such  
15 as heart rate variability and inflammatory cytokines (Furman et al., 2021). Finally, our  
16 approach to assessing lifetime stressors was based on retrospective reports that can be  
17 susceptible to memory recall bias. Although self-report checklist measures are relatively  
18 inexpensive and easy to administer, researchers have raised concerns about their reliability and  
19 validity (vs. in-depth interview methods, for example; Dohrenwend, 2006). Despite this,  
20 research has shown that the STRAIN has excellent test-retest reliability (Cazassa et al., 2020)  
21 and that individuals can reliably recall major life stressors over long periods of time (Brown &  
22 Harris, 1978; Slavich & Shields, 2018).

23         These findings can contribute to applied practice as they provide critical information  
24 regarding how best to identify sport performers at risk for poor health and support these  
25 individuals by helping them respond more adaptively to acute stressors. First, the results



1 suggest that practitioners (e.g., coaches, sport psychologists) should assess sport performers'  
2 historical exposure to lifetime non-sport and sport-specific stressors to help identify those who  
3 may be most susceptible to developing dysfunctional responses to stress that may be health-  
4 damaging. Once identified, interventions could then be implemented to modify reactivity to  
5 acute stressors or habituation. For example, social support has been associated with more  
6 adaptive psychophysiological stress responses and better cardiovascular health (e.g., Howard  
7 & Hughes, 2012). Thus, practitioners could utilise evidence-based social support interventions  
8 to help sport performers build stronger social support networks (see Freeman, 2021).

9       Finally, this study has some important theoretical contributions. In accordance with the  
10 integrative model of lifespan stress and health (Epel et al., 2018), this study investigated how  
11 the total count and severity of both non-sport and sport-specific lifetime stressors was  
12 associated with psychophysiological (i.e., heart rate and salivary cortisol) reactivity to a novel  
13 and then repeated acute laboratory-based psychosocial stressor. Although the integrative model  
14 suggests that lifetime stressor exposure impacts long term health via repeated maladaptive  
15 stress responses (Epel et al., 2018), there is currently little data on this specific question.  
16 Therefore, this study represented a novel test of this proposition and advanced our  
17 understanding of how lifetime stressor exposure is associated with sport performers' acute  
18 psychophysiological stress reactivity, and thus health. This furthers our theoretical  
19 understanding of the lifetime stressor-health relationship and suggests that more exploration is  
20 required with additional biomarkers of stress (e.g., dehydroepiandrosterone).

## 21 **Conclusion**

22       In conclusion, the present study is the first to address how lifetime non-sport and sport-  
23 specific stressor exposure, both in terms of count and severity, are associated with  
24 psychophysiological reactivity and habituation to an acute laboratory-based social stressor. The  
25 results suggest that experiencing a moderate number of lifetime stressors was related to more

1 adaptive psychophysiological responses. In contrast, exposure to a very high or low number of  
2 lifetime stressors led to more maladaptive responses to acute stress (e.g., exaggerated or  
3 blunted reactivity) and to poorer habituation. Furthermore, whereas exposure to a very low  
4 number of lifetime non-sport stressors was linked with poorer habituation, lifetime sport-  
5 specific stressor exposure was unrelated to habituation. These associations differed depending  
6 on the context of the stressor experienced (e.g., non-sport vs. sport-specific) and its underlying  
7 dimensions (e.g., lifetime count vs. severity). Overall, this study advances the literature on  
8 stress and health in sport and has important implications for identifying sport performers most  
9 at-risk of stress-related ill-health.

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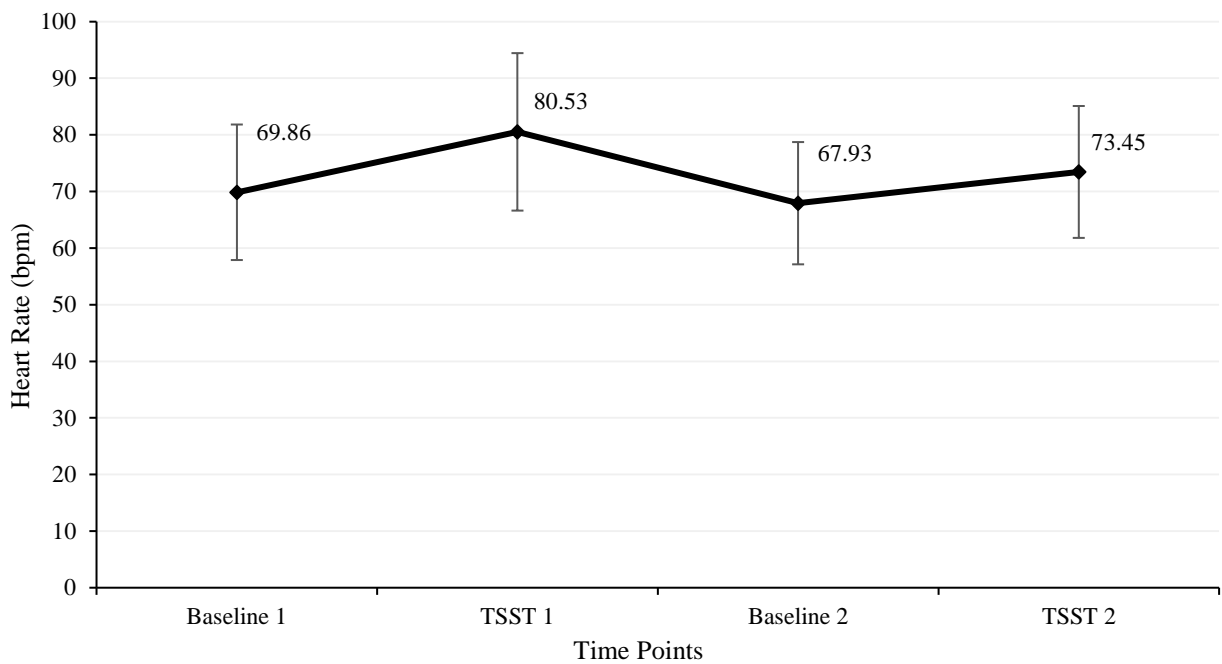
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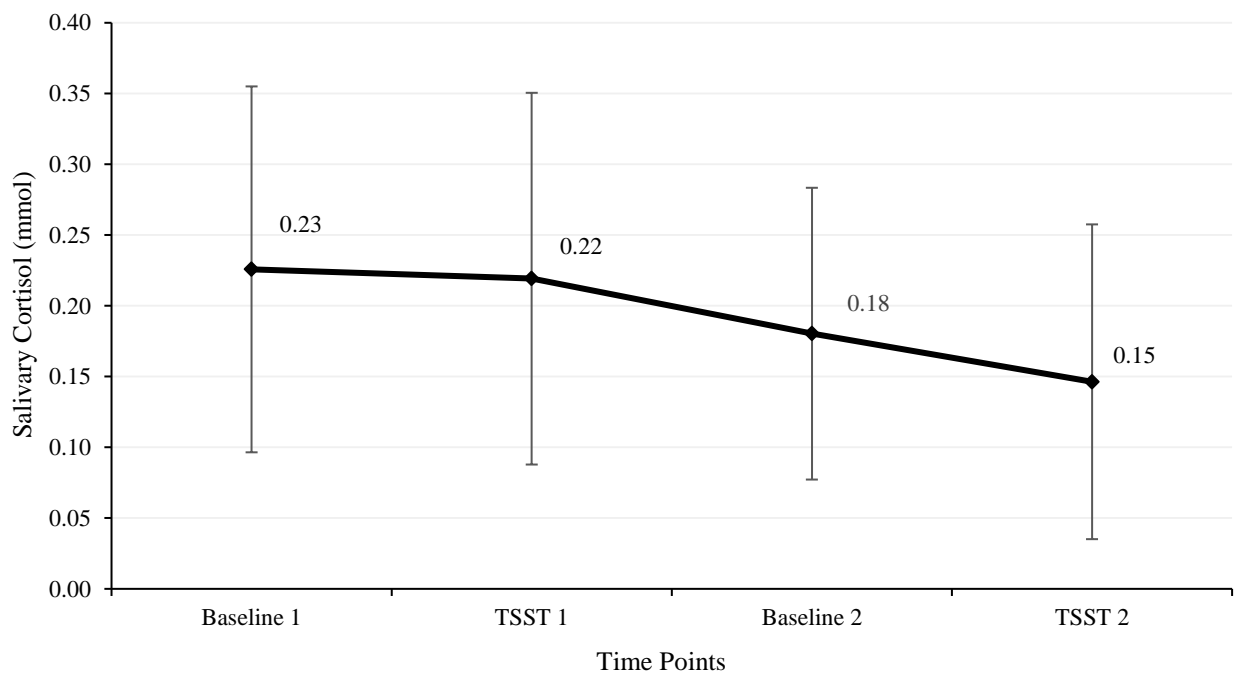
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**Figure 1a.** Cardiovascular (e.g., heart rate) measures assessed across all time points from pre- to post-TSST. Values show mean heart rate, and error bars represent standard deviations.



**Figure 1b.** Endocrine (e.g., salivary cortisol) measures assessed across all time points from pre- to post-TSST. Values show mean cortisol concentration, and error bars represent standard deviations.



**Figure 2.** The significant cubic relationships between lifetime non-sport and sport-specific stressor count and psychophysiological (i.e., heart rate and salivary cortisol) reactivity and habituation.

