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

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

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

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

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

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

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

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

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

Method

Participants

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

Procedure

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

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

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

Measures

Lifetime Non-Sport Stressor Exposure

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

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

Lifetime Sport-Specific Stressor Exposure

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

Heart Rate

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

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

Salivary Cortisol

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

Laboratory-Based Social Stressor

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

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

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

Data Analyses

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

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

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

[1] 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.

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

Results

Descriptive Statistics

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

Cardiovascular and Endocrine Reactivity to the Acute Stressor

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

[2] 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.

Lifetime Stressor Exposure and Psychophysiological Reactivity to an Acute Social Stressor

Lifetime Non-Sport Stressor Count

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

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

Lifetime Non-Sport Stressor Severity

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

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

Lifetime Sport-Specific Stressor Count

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

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

Lifetime Sport-Specific Stressor Severity

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

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

Lifetime Stressor Exposure and Psychophysiological Habituation to a Repeated Acute Social Stressor

Lifetime Non-Sport Stressor Count

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

Salivary cortisol. Despite non-significant quadratic (ΔR^2 = .000, p = .911) and cubic (ΔR^2 = .010, p = .339) components, a significant linear relationship (ΔR^2 = .070, β = -.27, p = .014) was found between total count of lifetime non-sport stressors and cortisol habituation.

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

Lifetime Non-Sport Stressor Severity

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

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

Lifetime Sport-Specific Stressor Count

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

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

Lifetime Sport-Specific Stressor Severity

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

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

Discussion

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

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

Psychophysiological Reactivity to an Acute Social Stressor

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

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

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

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

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

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

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

Psychophysiological Habituation to a Repeated Acute Stressor

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

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

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

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

Strengths, Limitations, and Implications

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

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

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

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

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

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

Conclusion

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

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

References

al'Absi, M., Ginty, A., & Lovallo, W. (2021). Neurobiological mechanisms of early life adversity, blunted stress reactivity and risk for addiction. *Neuropharmacology, 188,* 108519. doi: 10.1016/j.neuropharm.2021.108519

Arnold, R., & Fletcher, D. (2021). Stressors, hassles, and adversities. In R. Arnold & D. Fletcher (Eds.), *Stress, well-being, and performance in sport (*pp. 31-62). Taylor & Francis.

Blascovich, J. (2008). Challenge and threat. In A. J. Elliot (Ed.), *Handbook of approach and avoidance motivation* (pp. 431-445). Psychology Press.

Brindle, R., Ginty, A., & Conklin, S. (2013). Is the association between depression and blunted cardiovascular stress reactions mediated by perceptions of stress? *International Journal of Psychophysiology, 90*(1), 66-72. doi: 10.1016/j.ijpsycho.2013.06.003

Brindle, R., Pearson, A., & Ginty, A. (2022). Adverse childhood experiences (ACEs) relate to blunted cardiovascular and cortisol reactivity to acute laboratory stress: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews, 134*, 104530. doi: 10.1016/j.neubiorev.2022.104530

Carroll, D., Ginty, A., Painter, R., Roseboom, T., Phillips, A., & de Rooij, S. (2012a). Systolic blood pressure reactions to acute stress are associated with future hypertension status in the Dutch Famine Birth Cohort Study. *International Journal of Psychophysiology, 85*(2), 270-273. doi: 10.1016/j.ijpsycho.2012.04.001

Carroll, D., Ginty, A., Der, G., Hunt, K., Benzeval, M., & Phillips, A. (2012b). Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality. *Psychophysiology, 49*(10), 1444-1448. doi: 10.1111/j.1469-8986.2012.01463.x

Carroll, D., Ginty, A., Whittaker, A., Lovallo, W., & de Rooij, S. (2017). The behavioural,

cognitive, and neural corollaries of blunted cardiovascular and cortisol reactions to acute psychological stress. *Neuroscience & Biobehavioral Reviews*, *77*, 74-86. doi: 10.1016/j.neubiorev.2017.02.025

Cazassa, M. J., Oliveira, M. D. S, Spahr, C. M., Shields, G. S, & Slavich, G. M. (2020). The Stress and Adversity Inventory for Adults (Adult STRAIN) in Brazilian Portuguese: Initial validation and links with executive function, sleep, and mental and physical health. *Frontiers in Psychology*, *10*, 30383. doi:10/gmr8

Charloux, A., Lonsdorfer-Wolf, E., Richard, R., Lampert, E., Oswald-Mammosser, M., & Mettauer, B. et al. (2000). A new impedance cardiograph device for the non-invasive evaluation of cardiac output at rest and during exercise: comparison with the "direct" Fick method. *European Journal of Applied Physiology*, *82*(4), 313-320. doi: 10.1007/s004210000226

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status. *Hypertension*, *55*(4), 1026-1032. doi: 10.1161/hypertensionaha.109.146621

Chrousos, G. P. (2009). Stress and disorders of stress system. *Nature Reviews Endocrinology*, *5*(7), 374-381. doi: 10.1038/nrendo.2009.106

Dienstbier, R. A. (1992). Mutual impacts of toughening on crises and losses. In L. Montada, S.-H. Filipp, & M. J. Lerner (Eds.), *Life crises and experiences of loss in adulthood* (pp. 367–384). Lawrence Erlbaum Associates, Inc.

Dooley, L. N., Slavich, G. M., Moreno, P. I., & Bower, J. E. (2017). Strength through adversity: Moderate lifetime stress exposure is associated with psychological resilience in breast cancer survivors. *Stress And Health*, *33*(5), 549-557. doi: 10.1002/smi.2739

Elzinga, B., Roelofs, K., Tollenaar, M., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse

events. *Psychoneuroendocrinology*, *33*(2), 227-237. doi: 10.1016/j.psyneuen.2007.11.004

Epel, E. S., Crosswell, A. D., Mayer, S. E., Prather, A. A., Slavich, G. M., Puterman, E., & Mendes, W. B. (2018). More than a feeling: A unified view of stress measurement for population science. *Frontiers in Neuroendocrinology, 49*, 146-169. doi:10/gdp5hv

Field, A. (2018). *Discovering statistics using IBM SPSS statistics* (5th ed.). SAGE Publications.

Fletcher, D., Hanton, S., & Mellalieu, S. D. (2006). An organizational stress review: Conceptual and theoretical issues in competitive sport. In S. Hanton & S. D. Mellalieu (Eds.), *Literature reviews in sport psychology* (pp. 321–374). Hauppauge, NY: Nova Science Publishers.

Freeman, P. (2021). Social support. In R. Arnold & D. Fletcher (Eds.), *Stress, well-being, and performance in sport* (pp. 240-258). Oxford, UK: Taylor & Francis.

Furman, D., Campisi, J., Verdin, E., Carrera-Bastos, P., Targ, S., Franceschi, C., … & Slavich, G. M. (2019). Chronic inflammation in the etiology of disease across the life span. *Nature Medicine, 25*, 1822-1832. doi: 10.1038/s41591-019-0675-0

Ginty, A., & Conklin, S. (2011). High perceived stress in relation to life events is associated with blunted cardiac reactivity. *Biological Psychology*, *86*(3), 383-385. doi: 10.1016/j.biopsycho.2011.01.002

Ginty, A., Phillips, A., Roseboom, T., Carroll, D., & deRooij, S. (2012). Cardiovascular and cortisol reactions to acute psychological stress and cognitive ability in the Dutch Famine Birth Cohort Study. *Psychophysiology, 49*(3), 391-400. doi: 10.1111/j.1469-8986.2011.01316.x

Howard, S., & Hughes, B. (2012). Benefit of social support for resilience-building is contingent on social context: examining cardiovascular adaptation to recurrent stress in women. *Anxiety, Stress & Coping*, *25*(4), 411-423. doi: 10.1080/10615806.2011.640933

Howells, K., Sarkar, M., & Fletcher, D. (2017). Can athletes benefit from difficulty? A systematic review of growth following adversity in competitive sport. In M. R. Wilson, V. Walsh & B. Parkin (Eds.), *Sport and the Brain: The Science of Preparing, Enduring and Winning, Part B, Progress in Brain Research* (pp. 117-159). UK: Academic Press.

Hughes, B., Lü, W., & Howard, S. (2018). Cardiovascular stress-response adaptation: Conceptual basis, empirical findings, and implications for disease processes. *International Journal of Psychophysiology*, *131*(8), 4-12. doi: 10.1016/j.ijpsycho.2018.02.003

Kirschbaum, C., Pirke, K., & Hellhammer, D. (1993). The 'Trier Social Stress Test' – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*(1-2), 76-81. doi: 10.1159/000119004

Kudielka, B., von Känel, R., Preckel, D., Zgraggen, L., Mischler, K., & Fischer, J. (2006). Exhaustion is associated with reduced habituation of free cortisol responses to repeated acute psychosocial stress. *Biological Psychology*, *72*(2), 147-153. doi: 10.1016/j.biopsycho.2005.09.001

Labuschagne, I., Grace, C., Rendell, P., Terrett, G., & Heinrichs, M. (2019). An introductory guide to conducting the Trier Social Stress Test. *Neuroscience & Biobehavioral Reviews, 107*, 686-695. doi: 10.1016/j.neubiorev.2019.09.032

Lam, J. C. W., Shields, G. S., Trainor, B. C., Slavich, G. M., & Yonelinas, A. P. (2019). Greater lifetime stress exposure predicts blunted cortisol but heightened DHEA responses to acute stress. *Stress and Health, 35*, 15-26. doi:10/gd3p4k

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. Springer.

Lepore, S., Miles, H., & Levy, J. (1997). Relation of chronic and episodic stressors to psychological distress, reactivity, and health problems. *International Journal of Behavioral Medicine*, *4*(1), 39-59. doi: 10.1207/s15327558ijbm0401_3

Lovallo, W. (2013). Early life adversity reduces stress reactivity and enhances impulsive behavior: Implications for health behaviors. *International Journal of Psychophysiology*, *90*(1), 8-16. doi: 10.1016/j.ijpsycho.2012.10.006

Matthews, K., Gump, B., & Owens, J. (2001). Chronic stress influences cardiovascular and neuroendocrine responses during acute stress and recovery, especially in men. *Health Psychology*, *20*(6), 403-410. doi: 10.1037/0278-6133.20.6.403

McEwen, B. S. (1998). Stress, adaptation, and disease. Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *840*(1), 33–44. doi: 10.1111/j.1749-6632.1998.tb09546.x

McEwen, B., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *896*(1), 30-47. doi: 10.1111/j.1749-6632.1999.tb08103.x

McLoughlin, E., Arnold, R., Fletcher, F., Spahr, C. M., Slavich, G. M., & Moore, L. J. (2022). Assessing lifetime stressor exposure in sport performers: Associations with trait stress appraisals, health, well-being, and performance. *Psychology of Sport and Exercise, 58*, 102078. doi: 10.1016/j.psychsport.2021.102078

McLoughlin, E., Fletcher, D., Slavich, G. M., Arnold, R., & Moore, L. J. (2021). Cumulative lifetime stress exposure, depression, anxiety, and well-being in elite athletes: A mixed method study. *Psychology of Sport and Exercise*, *52*, 101823. doi:10/gfj5

McMullin, S. D., Shields, G. S., Slavich, G. M., & Buchanan, T. W. (2021). Cumulative lifetime stress exposure predicts greater impulsivity and addictive behaviors. *Journal of Health Psychology*, *26*(14), 2921-2936. doi: 10.1177/1359105320937055

Meijen, C., Jones, M. V., Mccarthy, P. J., Sheffield, D. & Allen, M. S. (2013). Cognitive and affective components of challenge and threat states. *Journal of Sports Sciences, 31*(8), 847-855. doi: 10.1080/02640414.2012.753157

Moore, L. J., Young, T., Freeman, P., & Sarkar, M. (2018). Adverse life events, cardiovascular responses, and sports performance under pressure. *Scandinavian Journal of Medicine & Science in Sports*, *28*(1), 340-347. doi:10/gjgb7k

Navazesh, M. (1993). Methods for collecting saliva. *Annals of the New York Academy of Sciences, 694*, 72–77. doi: 10.1111/j.1749-6632.1993.tb18343.x

Olvera Alvarez, H. A., Provencio-Vasquez, E., Slavich, G. M., Laurent, J. G. C., Browning, M., McKee-Lopez, G., Robbins, L., & Spengler, J. D. (2019). Stress and health in nursing students: The Nurse Engagement and Wellness Study. *Nursing Research, 68*(6), 453-463. doi: 10.1097/NNR.0000000000000383

Phillips, A. (2011). Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health. *Biological Psychology*, *86*(2), 106-113. doi: 10.1016/j.biopsycho.2010.03.016

Phillips, A., Carroll, D., Ring, C., Sweeting, H., & West, P. (2005). Life events and acute cardiovascular reactions to mental stress: A cohort study. *Psychosomatic Medicine*, *67*(3), 384-392. doi: 10.1097/01.psy.0000160464.63264.5d

Phillips, A., Roseboom, T., Carroll, D., & de Rooij, S. (2012). Cardiovascular and cortisol reactions to acute psychological stress and adiposity. *Psychosomatic Medicine*, *74*(7), 699-710. doi: 10.1097/psy.0b013e31825e3b91

Richard, R., Lonsdorfer-Wolf, E., Charloux, A., Doutreleau, S., Buchheit, M., & Oswald-Mammosser, M. et al. (2001). Non-invasive cardiac output evaluation during a maximal progressive exercise test, using a new impedance cardiograph device. *European Journal of Applied Physiology*, *85*(3-4), 202-207. doi: 10.1007/s004210100458

Roos, L., Janson, J., Sturmbauer, S., Bennett, J., & Rohleder, N. (2019). Higher trait reappraisal predicts stronger HPA axis habituation to repeated stress. *Psychoneuroendocrinology, 101*, 12-18. doi: 10.1016/j.psyneuen.2018.10.018

Roy, M., Steptoe, A., & Kirschbaum, C. (1998). Life events and social support as moderators of individual differences in cardiovascular and cortisol reactivity. *Journal of Personality and Social Psychology, 75*(5), 1273-1281. doi: 10.1037/0022-3514.75.5.1273

Seery, M. D., Holman, E. A., & Silver, R. C. (2010). Whatever does not kill us: Cumulative lifetime adversity, vulnerability, and resilience. *Journal of Personality and Social Psychology, 99*(6), 1025–1041. doi:10.1037/a0021344

Seery, M., Leo, R., Lupien, S., Kondrak, C., & Almonte, J. (2013). An upside to adversity? Moderate cumulative lifetime adversity is associated with resilient responses in the face of controlled stressors. *Psychological Science, 24*(7), 1181-1189. doi: 10.1177/0956797612469210

Shapero, B. G., Hamilton, J. L., Stange, J. P., Liu, R. T., Abramson, L. Y., &Alloy, L. B. (2015). Moderate childhood stress buffers against depressive response to proximal stressors: A multi-wave prospective study of early adolescents. *Journal of Abnormal Child Psychology, 43*(8),1403–1413. doi:10.1007/s10802-015-0021-z

Shields, G. S., Fassett-Carman, A., Gray, Z. J., Gonzales, J. E., Snyder, H. R., & Slavich, G. M. (2022). Why is subjective stress severity a stronger predictor of health than stressor exposure? A preregistered two-study test of two hypotheses. *Stress and Health*. doi: 10.1002/smi.3165

Slavich, G. M. (2020). Social safety theory: A biologically based evolutionary perspective on life stress, health, and behavior. *Annual Review of Clinical Psychology, 16*, 265-295. doi: 10.1146/annurev-clinpsy-032816-045159

Slavich, G. M. (2022). Social Safety Theory: Understanding social stress, disease risk, resilience, and behavior during the COVID-19 pandemic and beyond. *Current Opinion in Psychology, 45*:101299. doi: 10.1016/j.copsyc.2022.101299

Slavich, G. M., & Shields, G. S. (2018). Assessing lifetime stress exposure using the Stress and Adversity Inventory for Adults (Adult STRAIN). *Psychosomatic Medicine*, *80*(1), 17-27. doi:10/ggvmjk

Slavich, G. M., Stewart, J. G., Esposito, E. C., Shields, G. S., & Auerbach, R. P. (2019). The Stress and Adversity Inventory for Adolescents (Adolescent STRAIN): Associations with mental and physical health, risky behaviors, and psychiatric diagnoses in youth seeking treatment. *Journal of Child Psychology and Psychiatry*, *60*(9), 998-1009. doi:10/gg8ggf

Straneva, P., Hinderliter, A., Wells, E., Lenahan, H., & Girdler, S. (2000). Smoking, oral contraceptives, and cardiovascular reactivity to stress. *Obstetrics and Gynecology*, *95*(1), 78-83. doi: 10.1097/00006250-200001000-00016

Turner, A., Smyth, N., Hall, S., Torres, S., Hussein, M., & Jayasinghe, S., … & Clow, A. J. (2021). Psychological stress reactivity and future health and disease outcomes: A systematic review of prospective evidence. *Psychoneuroendocrinology*, *114*, 104599. doi: 10.1016/j.psyneuen.2020.104599

Tyra, A., Soto, S., Young, D., & Ginty, A. (2020). Frequency and perceptions of life stress are associated with reduced cardiovascular stress-response adaptation. *International Journal of Psychophysiology*, *157*, 51-60. doi: 10.1016/j.ijpsycho.2020.08.006

Whittaker, A., Ginty, A., Hughes, B., Steptoe, A., & Lovallo, W. (2021). Cardiovascular stress reactivity and health: Recent questions and future directions. P*sychosomatic Medicine, 83*(7), 756-766. doi: 10.1097/psy.0000000000000973

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.

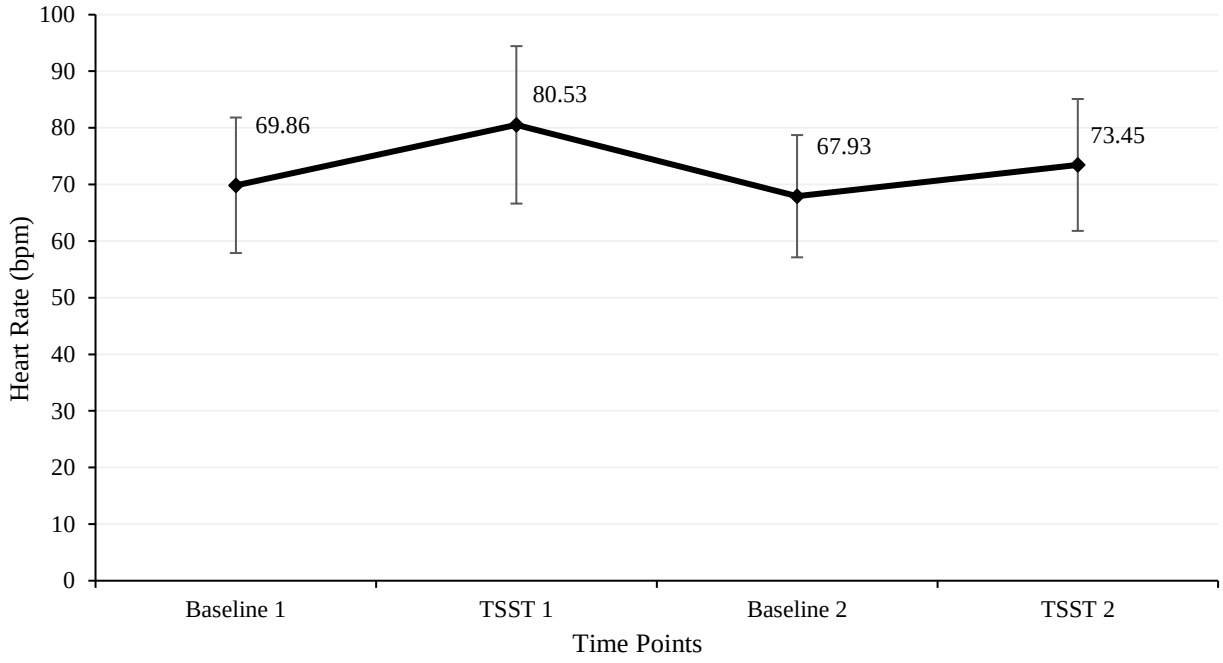


Figure 1b. Endocrine (e.g., salivary cortisol) measures assessed across all time points from pre- to post-TSST. Values show mean cortisol concentration, and error bars represent standard deviations.

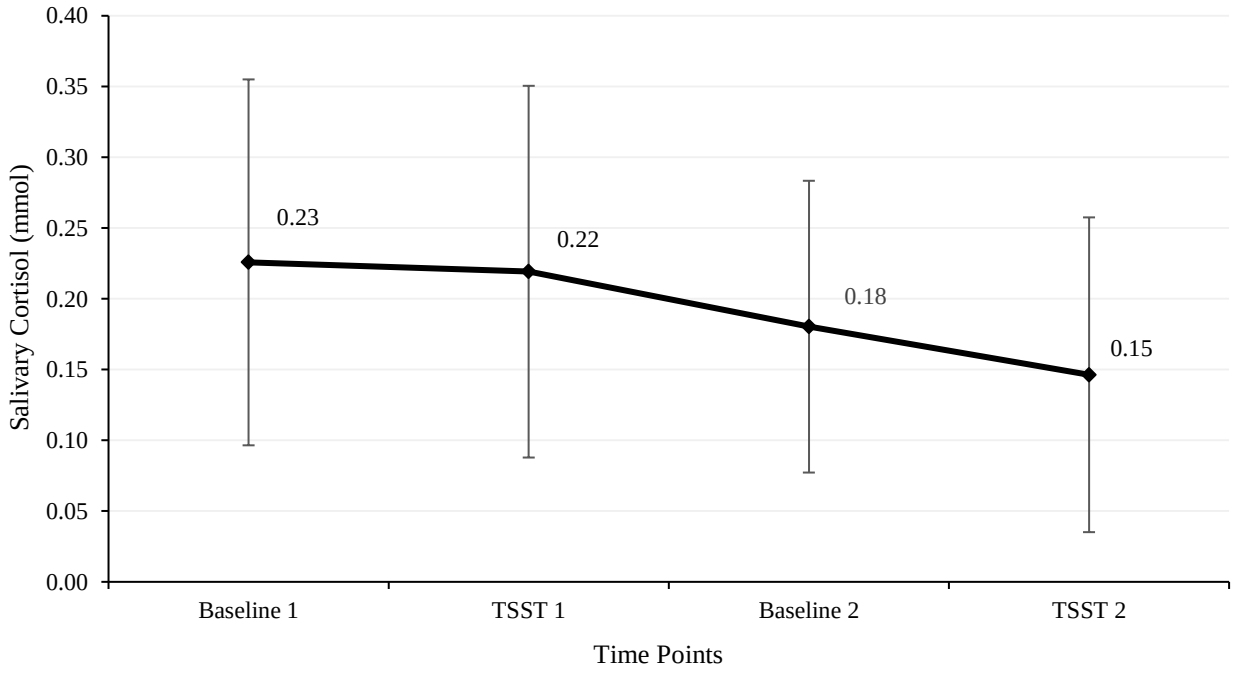
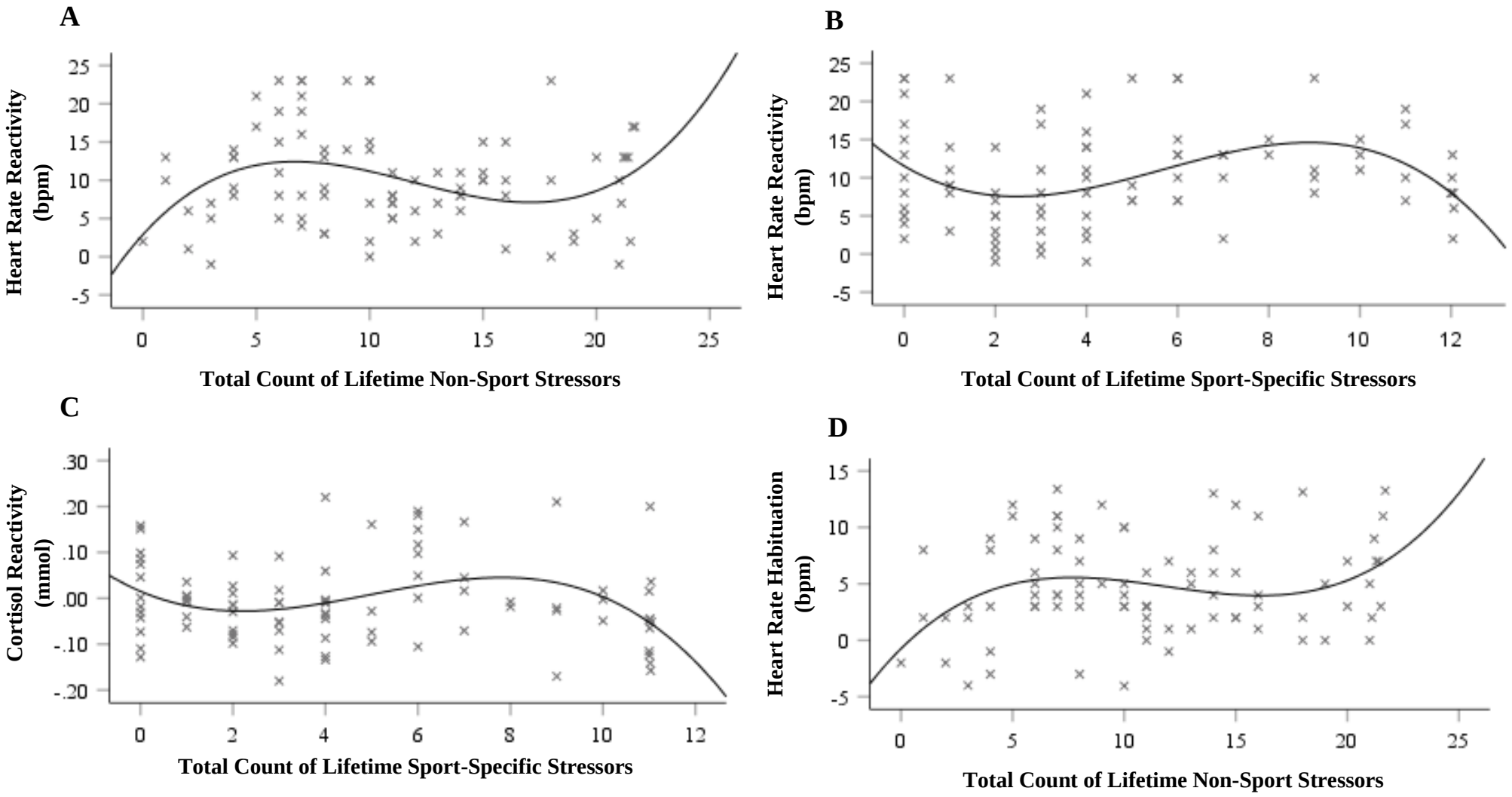


Figure 2. The significant cubic relationships between lifetime non-sport and sport-specific stressor count and psychophysiological (i.e., heart rate and salivary cortisol) reactivity and habituation.



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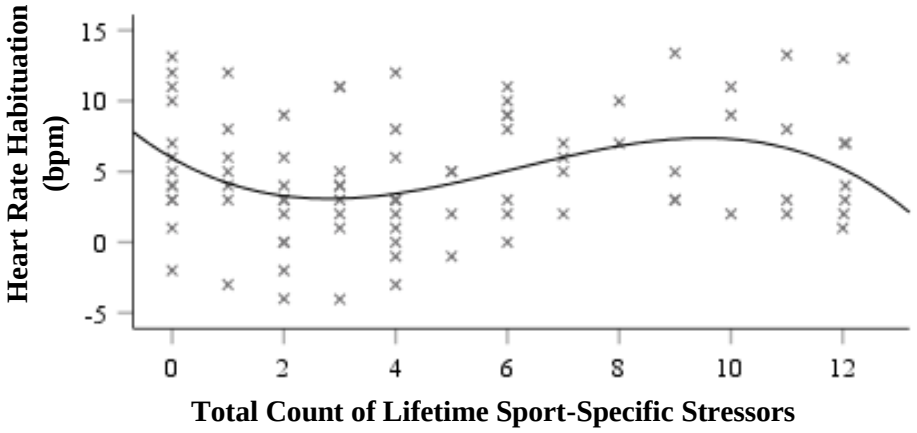
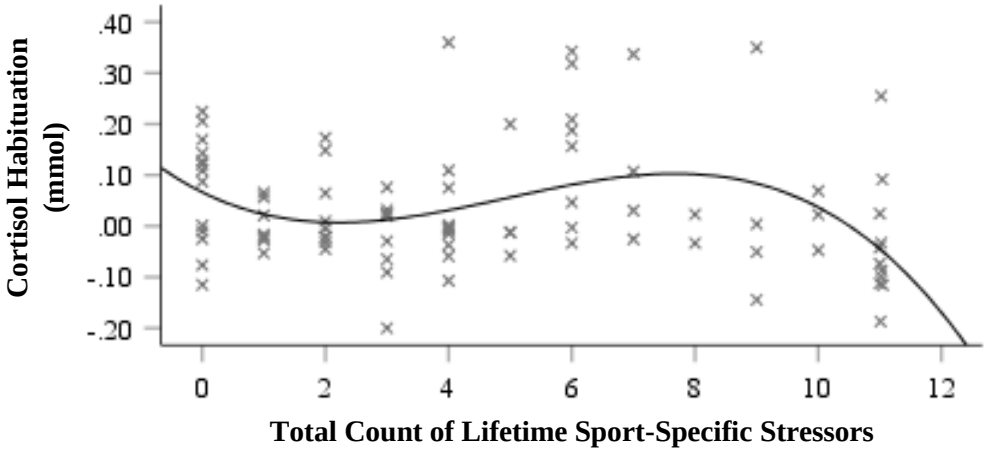

Heart Rate Habituation (bpm)
15
10
5
0
-5
0
2
4
6
8
10
12
Total Count of Lifetime Sport-Specific Stressors

F


Cortisol Habituation (mmol)
.40
.30
.20
.10
.00
-.10
-.20
0
2
4
6
8
10
12
Total Count of Lifetime Sport-Specific Stressors