Life events are associated with elevated heart rate and reduced heart complexity to acute psychological stress

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ABSTRACT

The current study examined whether the exposure to life events and reported impact of life events are associated with altered cardiac reactivity to an acute psychological stressor. Participants (N = 69) completed the Life Experience Survey (LES) and Positive and Negative Affect Schedule (PANAS) and undertook a standardized social-evaluative stress task. Cardiac activity was measured via heart rate and non-linear heart rate variability (HRV) indices Sample Entropy, SD1, SD2 and SD1/SD2 ratio. Heart rate and non-linear HRV were measured before, during and after stress exposure. Findings suggest higher heart rate reactivity in individuals reporting higher number and impact of negative and total life events. Decreases in Sample Entropy were evident for number as well as impact of life events. No associations were found for SD1, SD2 and SD1/SD2 ratio. Findings suggest that life-events are associated with elevated heart rate and diminished heart rate complexity in response to acute stress.

1. Introduction

Experiencing a greater number of stressful life events might enhance the risk of developing cardiovascular diseases (Bernston, Patel, & Stewart, 2017). A mechanism underlying life events’ associated changes in cardiovascular health might be differences in cardiovascular reactivity (CVR) to acute stress. Of note, differences in physiological stress reactivity have been associated with physical and mental health outcomes (Huang, Webb, Zourdos, & Acevedo, 2013; Salomon, Clift, Karlsson, & Rottenberg, 2009). Acute stress leads to an activation of the sympathetic nervous system (SNS) and a withdrawal of the parasympathetic nervous system (PNS), resulting in stronger heart muscle contractions and increased heart rate (Kemeny, 2003; Turner, 1994).

Importantly, dysregulations in response to acute stress have been associated with either exaggerated or blunted CVR, both of which may impact health (Kibler & Ma, 2004; Souza et al., 2015).

Recent research indicates that life events and chronic challenges might be accompanied by changes in CVR to stress, thus contributing to allostatic load in the long run (e.g., McEwen, 1998), ultimately challenging cardiovascular health (Treiber et al., 2003). Specifically, exposure to critical life events has been associated with both exaggerated (Roy, Steptoe, & Kirschbaum, 1998) and blunted CVR to acute stress (Musante et al., 2000; Tyra, Soto, Young, & Ginty, 2020; Voellmin et al., 2015). Musante et al. (2000) revealed associations between stressful life events and CVR. However, other research failed to verify relations between negative life events and CVR (Vingerhoets, Ratliff-Crain, Jabaaij, Menges, & Baum, 1996), thus suggesting rather heterogeneous evidence.

Of note, previous research on CVR to acute stress typically focused on short-term changes in variables sensitive to sympathetic nervous system activity, as for example, heart rate and blood pressure (e.g., Ginty & Conklin, 2011). However, cardiovascular risk seems to be mediated by a mutual interplay of sympathetic and parasympathetic nervous system activity (Hillebrand et al., 2013), thus arguing for more complex measures of autonomic functioning. In particular, analysis of heart rate variability (HRV) allows to gain insight into the dynamic interplay between the autonomic and the central nervous system (Schwerdtfeger et al., 2020). HRV is based on the analysis of beat-to-beat variations in heart rate (Shaffer & Ginsberg, 2017) and can be quantified using time-domain, frequency-domain and non-linear metrics (e.g., Laborde, Mosley, & Thayer, 2017).

Most research on HRV is based on linear analysis of time- or frequency domains of HRV (Laborde et al., 2017). While time-domain measures quantify the amount of variability in normal-to-normal beat intervals, frequency domain measures index the power in frequency bands via spectral analysis. Of note, HRV time- and frequency domain

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measures change in response to acute stress, with decreases in high frequency (HF-HRV) and increases in low frequency (LF-HRV) being reported most frequently (Kim, Cheon, Bai, Lee, & Koo, 2018). These findings may indicate a shift from parasympathetic to sympathetic dominance. Of note, Xin et al. (2020) examined the role of stressful life events on heart rate and HRV reactivity to acute stress and found that a higher frequency of negative life stress exposure within the last twelve months was associated with a smaller decrease in HRV and a smaller heart rate increase to acute stress, thus suggesting blunted cardiac stress reactivity.

Importantly, heart rate dynamics are chaotic and influenced by non-linear interactions between different physiological systems (Dimitriev, Saperova, & Dimitriev, 2016). While time- and frequency domain measures define quantitative properties of heart rate dynamics, non-linear measures indicate qualitative properties of heart rate dynamics, thus accounting for chaotic influences (Shaffer & Ginsberg, 2017). Specifically, non-linear HRV measures quantify the unpredictability of a time series (Dimitriev et al., 2016) and hence, contain information beyond traditional time and frequency domain HRV parameters (Francesco et al., 2012; Young & Benton, 2015). A less complex series of cardiac beat-to-beat intervals indicates a higher regularity and hence, predictability of the signal. Importantly, lower heart rate complexity has been found to predict paroxysmal atrial fibrillation (Chesnokov, 2008), to be associated with disease (Goldberger, Peng & Lipsitz 2002; Tsai et al., 2020) and contribute to the development of cardiovascular disease (de Godoy, 2016; Lipsitz, 2004). Hence, non-linear HRV measures are of clinical relevance.

Among the most studied non-linear HRV variables are Poincaré plot analysis and entropy measures, both of which seem to be especially suited for examining short-term heart rate complexity (Sassi et al., 2015). The Poincaré plot is a time series analysis tool, which is based on the visual representation of consecutive R-R intervals. Each R-R interval is plotted as a function of the previous R-R interval (Karmakar, Khan, doker, Gubbi, & Palaniswami, 2009). SD1 and SD2 are measures derived from Poincaré plots to quantify short and long-term R-R interval variability (Hoshi, Pastre, Vanderlei, & Godoy, 2013). Of note, SD1 and the time-domain HRV metric root mean square of successive differences (RMSSD) are mathematically equivalent (Cicone et al., 2017). Due to the sensitivity of RMSSD to parasympathetic (i.e., vagal) function (Shaffer, McCraty, & Zerr, 2014), changes in SD1 are also indicative of vagal efference. On the other hand, SD2 and the SD1/SD2 ratio might be sensitive to sympathetic activity and correlate with the LF/HF ratio, possibly indicating autonomic balance (Shaffer & Ginsberg, 2017), although this assumption has been challenged recently (Rahman, Habel, & Contrada, 2018). Of note, both SD1 and SD2 correlate with baroreflex sensitivity and reflect overall predictability of the signal (Shaffer & Ginsberg, 2017). Entropy refers to randomness and predictability of a time series and quantities the repetition of patterns in a signal (Pincus & Goldberger, 1994). A higher value in entropy measures indicates less similarities (and hence, higher unpredictability) in a times series (Lake, Richman, Griffin, & Moorman, 2002).

Importantly, although non-linear HRV measures have received comparably little attention in stress research (relative to time-domain and frequency-domain metrics), there is empirical evidence that such measures are sensitive to changes in stress exposure as well. For example, Dimitriev et al. (2016) examined SD1 and SD2 in a sample of students at rest and before a university exam. They could show that SD1 and SD2 were significantly lower before the exam than at rest. Chan-wimahzang et al. (2017) could observe that entropy measures in a sample of musicians decreased before and during a stage performance, thus suggesting a reduction of complexity. Of note, lower heart rate complexity has also been associated with mental health impairments, such as depression (e.g., Leistredt et al., 2011) or phobia (e.g., Bornas et al., 2006). Together these findings suggest that non-linear heart rate complexity measures could be sensitive to adverse conditions, suggesting higher regularity and predictability of cardiac beat to beat intervals during environmental challenges and compromised psychological functioning.

To our knowledge, studies focusing on associations between life events and non-linear HRV metrics are missing to date. Moreover, most of the previous research examined the mere number of perceived life events. While the frequency of exposure to life events might certainly challenge the organism’s adaptability, it could be assumed that the perceived impact could be even more important in shaping stress reactivity (Tyra et al., 2020). However, to the authors’ knowledge, studies focusing on the perceived impact of life events on CVR are largely missing. Notably, in a study by Phillips, Carroll, Ring, Sweating, and West (2005) the total number of desirable and undesirable life events in young adults was associated with blunted cardiac reactivity to acute stress. However, it was the frequency of exposure to desirable, hence positive events rather than to undesirable events that was consistently connected to blunted reactivity. Thus, there is preliminary evidence that the person’s evaluation of life events could be crucial for research on CVR to acute stress.

Therefore, the current study aimed to investigate the effect of both the number and perceived impact of experienced positive and negative life events on heart rate and heart rate complexity reactivity. Based on the reported findings we expected that individuals with higher numbers of negative and positive life events and higher perceived impact of those life events, would show blunted heart rate and heart rate complexity reactivity to acute stress.

2. Method

2.1. Participants

Participants (N = 69) were young adults (mean age = 22.46, SD = 2.81, female: 68 %, smokers: 11.6 %). The mean waist-to-hip ratio (WHR) was 0.78 (SD = 0.08), the mean Body mass index (BMI) was 23.18 (SD = 4.6). According to BMI criteria 66.66 % of individuals in the sample were classified as having normal weight, 24.64 % as being overweight and 8.7 % as being underweight. A minority of the sample (1.4 %) were without a high school degree, 87.0 % had a high-school degree and 11.6 % had a bachelor or master’s degree. Regular physical exercise was assessed through the question ‘Do you perform regular exercises that cause sweating or shortness of breath (e.g. jogging, walking, cycling, fitness training, team sports)?’ (yes/no). The majority of the sample (66.7 %) agreed to this question. Exclusion criteria were cardiovascular heart disease, receiving psychotropic medication affecting cardiovascular activity, pregnancy, diabetes, and presence of mental disorders. Furthermore, it should be noted that acute life events could severely alter the organism’s physiological adaptability (e.g., Buckley et al., 2012), thus making it difficult to disentangle the cumulative effect of severe life events in the past and more recently experienced aversive circumstances. To prevent influences of recently experienced critical life events on cardiac reactivity, individuals who experienced a critical life event one month before the scheduled experiment were excluded prior to the study. Participants were instructed not to consume alcohol, caffeine, and nicotine two hours prior to study participation. The study was approved by the Ethics Review Board of the University of Graz (GZ. 39/79/63 ex 2017/18).

2.2. Stress task and study procedure

The experiment was carried out on an individual basis in a quiet laboratory. Upon arrival, participant signed informed consent. WHR and BMI were measured. Participants were asked to sit down in front of a computer screen. Electrodes were attached to the chest to record the electrocardiogram (ECG). Participants filled out questionnaires on demographic variables and life events. Affect ratings were obtained before and after baseline measurement, after preparation period and after the stress task. During baseline measurement participants watched
landscape pictures on the computer screen (3 min). Then, general instructions on the stress task were provided. Specifically, participants were instructed to prepare and deliver a speech on at least three personal strengths and three weaknesses. They were offered a pen and paper to take notes if necessary. Stress task appraisal was measured before the stress task. During the preparation period, participants were presented with a timer signaling a countdown of the time left. The speech should last for three minutes, with time used to the full. If the participant finished ahead of time, the experimenter asked for further elaboration. To assure a high level of stress induction, participants were informed that their speech would be audio and video recorded and compared with the performance of other participants. After the preparation period (3 min), the stress task was initialized (3 min). Subsequently, participants were asked to remain in the chair and fixate on a green dot, displayed on the computer screen (5 min). Finally, the electrodes were detached, and participants debriefed. The study procedure is visualized in Fig. 1.

2.3. Variables and instruments

2.3.1. Life events

Life events were assessed using the Life Experiences Survey (LES, Sarason, Johnson, & Siegel, 1978). The LES consists of a list of 47 pre-defined life events, plus three blank spaces in which participants can report unlisted events they experienced. Life events refer to life changes that are considered common to an individual (e.g., ‘marriage’, ‘death of a close family member’). Participants were asked to indicate events that they had experienced over their whole life and to evaluate the positive or negative impact of each experienced life event (7 point-Likert scale, range ‘very negative’ to ‘very positive’). Summing the positive impact ratings provides a positive change score, summing the negative impact ratings provides a negative change score. A total change score can be obtained by adding both values. Change scores represent the rated amount of impact of life events experienced by the participants. Participants’ scores for number of life events (positive, negative, and total number life events) and their respective impact (positive, negative and total impact) were separately added to obtain total scores. Cronbach’s alpha for the overall Life Experiences Survey coefficient was acceptable (.68). Mean value for positive life events was 8.04 (SD = 2.73), for negative life events 7.72 (SD = 4.15) and for total number of life events 15.77 (SD = 4.86). Mean value for positive change score was 16.42 (SD = 6.61), for negative change score 12.45 (SD = 7.69) and for total change score 28.87 (SD = 10.20).

2.3.2. Affect and task appraisal

The German version of the Positive and Negative Affect Schedule (PANAS, Kohn, Egloff, Kohlmann, & Tausch, 1996) was used to assess positive affect (PA) and negative affect (NA). The PANAS consists of 20 items, describing different affective states. Participants rate each item on a 5-point Likert scale ranging from 1 (‘not at all’) to 5 (‘extremely’). PA (e.g., active, strong, excited) and NA (e.g., angry, guilty, scared) is assessed by 10 items each. Mean scores for both dimensions were calculated for each participant. PA and NA were assessed before and after baseline, after the preparation period and after the stress task. Cronbach’s alpha varied between .83 and .92 for PA and .79 and .91 for NA, thus suggesting good reliability. Task appraisal (“How challenging do you consider the upcoming speech?”) was assessed using a unipolar item ranging from 1 (‘not at all’) to 5 (‘extremely’). Similar approaches of examining challenge through self-reported data can be found by Tomaka, Blascovich, Kibler and Ernst (1997).

2.3.3. Physiological variables

2.3.3.1. Heart rate and heart rate complexity. Heart rate and non-linear HRV indices were analyzed based on ECG recordings. The ECG was recorded with a BIOPAC MP150 amplifier system and sampled at 1000 Hz for analyzing HRV. R-R interval detection was carried out via Kubios premium software (vers. 3.2; Tarvainen, Lipponen, & Kuoppa, 2017). Artifacts were corrected through Kubios automatic artifact correction algorithm, with a criterion of 95% of artifact-free HRV data for each task period. Mean heart rate and mean non-linear HRV indices were extracted for each task period (baseline, preparation, stress, recovery). Cardiac variables were analyzed for 3 min in each period. For recovery, the first 3 min were used for analysis. Short-term heart rate complexity was quantified by SD1 (standard deviation of instantaneous beat-to-beat variability) and SD2 (long-term standard deviation of continuous R-R intervals). Sample entropy (SampEn) was calculated to investigate the complexity or irregularity of the signal (Shaffer & Ginsberg, 2017).

2.3.3.2. Respiratory activity. A piezoelectric belt (Braebon Medical Corporation, Canada) was used to record respiratory frequency. The belt was placed around participant’s chest. Data was filtered using the AcqKnowledge 4.3 software (BIOPAC systems, Inc.). The signal was downsamples to 50 samples per second and a bandpass filter was set to 0.05–1 Hz. The mean respiration rate for each task period was extracted for each individual in order to control for respiratory influences on cardiac stress reactivity.

2.4. Statistical analysis

Mixed-effect models were used to analyze changes in heart rate and non-linear HRV from baseline to each task period. Reactivity refers to the change from baseline to stress task (i.e., delta change scores from baseline to stress task). A natural-log transformation was applied for SD1 and SD2 prior to analysis in order to control for skewness. Several confounders need to be considered when analyzing HRV. Specifically, sex and age have been differently associated with HRV (Adjei, Xue, & Mandic, 2018; Koenig & Thayer, 2016; Hernández-Vicente et al., 2020), as well as physical activity and WHR (Koechli, Schtte & Kruger, 2020; Yi, Lee, Shin, Kim & Ki, 2013). Of note, smoking can dampen parasympathetic activity (Talukdar, Nazak, Biswal, Dey & Pal, 2020). Therefore, sex, age, physical activity, WHR and smoking status (yes/no) were included as confounders in the model. Furthermore, fluctuations in respiration rate might influence HRV indices (e.g., Hill, Siebenbrock, Sollers, & Thayer, 2009). Thus, mean respiration rate for each task

Fig. 1. Study procedure with main study phases and main variables, HR, heart rate; SampEn, sample entropy; PA, positive affect; NA, negative affect.
period was included as a confounder in the model. Analyses were conducted using R (version 3.5.2), package lme4 (ver. 1.1–21, Bates et al., 2018). Number and impact of life events scores (negative, positive, total), age and WHR were each treated as a continuous variable and grand mean centered to facilitate the meaning of the intercept. In order to check for the combined effect of number and impact of life events, three additional parameters were calculated by multiplying number and impact of total, negative and positive life events, respectively. Participant was treated as a random effect variable throughout. Shapiro Wilk-tests were used to check for normal distribution. Satterthwaite-approximation was applied to derive p-vaies. The level of significance was fixed at $p < .05$ (two-tailed).

3. Results

3.1. Manipulation check

In order to investigate if the stress task triggered a cardiac stress response, repeated measures ANOVAs (baseline, preparation period, task period, recovery) were conducted. Significant changes in the mean scores between time points (baseline, preparation, stress task, recovery) of all examined variables were evident ($p < .001$). Post hoc Bonferroni-adjusted comparisons between baseline and stress task showed significant results for all examined parameters, except SD1 (see Table 1). To investigate if the stress task impacted affective wellbeing, two repeated measures ANOVAs were conducted. Significant changes in the mean scores between time points (before baseline, after baseline, after preparation and after stress task) were found for negative and positive affect ($p < .001$). For PA, a significant increase from baseline to preparation was observed ($p = .045$), which could also be observed for NA ($p < .001$) (see Table 2). Participants rated the upcoming stress task as moderately challenging ($M = 3.14, SD = 0.98$), which was significantly different from 1 ($t (68) = 18.01, p < .001$).

In order to investigate if the reported strengths and weaknesses during the speech were associated with ratings of affect, correlations between number and duration of reported strengths and weaknesses (seconds) and changes in PA and NA were conducted. Due to technical problems, sound recordings were not available for 11 participants. The videotaped speech recordings were rated by two independent raters (ICC number of strengths = 0.88; ICC number of weaknesses = 0.93, ICC length of reported strengths = 0.81, ICC length of reported weaknesses = 0.90) and aggregated prior to analysis. Correlations were not significant (all $p > .063$). Participants reported a higher number of strengths as compared to number of weaknesses (strengths: $M = 3.35, SD = 1.29$; weaknesses: $M = 2.73, SD = 0.94, t (57) = 3.38, p = .021$) and talked significantly longer about their strengths than about their weaknesses (strengths: $M = 92.93, SD = 29.96$; weaknesses: $M = 72.58, SD = 30.20, t(57) = 2.63, p = .011$).

3.2. Heart rate and heart rate complexity

Results for the mixed effects models to predict cardiac variables are shown in Tables 3–5. Results of the reported models are adjusted for respiratory rate, sex, age, WHR, smoking status, and physical activity. Significant interactions between cardiac reactivity to stress and exposure and impact of life events were found for both heart rate and SampEn (all $p's < .05$). These findings document that heart rate increased while SampEn decreased to the stress task, as number and impact of total life events, respectively increased (see Tables 3 & 4). For heart rate, a significant interaction was observed for number of negative life events and stress task ($p = .039$). More precisely, with each 1-point increase in the number of negative life events, heart rate increased by 0.42 BPM during the stress task, relative to baseline (see Table 3). Similarly, higher negative impact of life events was associated with increases in heart rate during stress and decreases in SampEn, as compared to baseline (all $p's < .05$) (see Table 4). Participants rating a higher number and a higher impact of positive life events, respectively display lower SampEn from baseline to stress task (all $p's < .05$) (see Tables 3 & 4). No significant interactions were found for both heart rate and SampEn with respect to the preparation period.

Furthermore, a stronger decrease in SampEn during stress was found for individuals reporting a higher product of total number and impact of life events, and positive number and impact of life events (all $p's < .05$). Also, higher heart rate reactivity was evident in individuals reporting a higher product of total and negative number and impact of life events (all $p's < .05$) (see Table 5). No significant interactions related to number or impact of positive, negative and total life events and task period were observed for SD1, SD2 and SD1/SD2 ratio (all $p > .343$). Of note, there were no significant effects of examined confounders on results. However, effects of respiratory rate on Sample Entropy from baseline to stress task were observed for models examining positive impact ($p = .047$) and number of positive life events ($p = .045$), thus documenting that a higher breathing rate was associated with decreases in SampEn.

4. Discussion

The aim of the present study was to investigate associations between positive, negative, and total amount of life events and changes in cardiac reactivity to mental stress. Heart rate and non-linear HRV indices (with the exception of SD1) changed significantly from baseline to stress task (for HR around 15 BPM), thus suggesting a successful stress induction. For SD1 it should be noted that the lack of change in response to stress might indicate a lack of parasympathetic withdrawal in response to the stress task (Dimitriev et al., 2016). This finding might partly be explained by the nature of the chosen task. The speech task resulted in an increase of the nature of the chosen task. The speech task resulted in an increase of
negative life events (e.g., Tyra et al., 2020). Moreover, stronger decreases in SampEn were found in association with number of total life events and negative life events, as well as for total, negative and positive impact of life events, respectively. The observed decreases in heart rate complexity indicate a stronger regularity and a more periodic heart rate (Goldberger et al., 2002). Disruptions in the systems’ ability to flexibly adapt to environmental changes. Thus, the present results indicate that exposure to actual stress throughout the lifespan is considered. Our results deviate from previous research, which suggest blunt hyperventilation in individuals facing a higher number of negative life events (e.g., Xin et al., 2020), but are generally in line with other studies hypothesizing that exposure to a higher number of negative life events may promote maladaptive stress responses to acute stress (Matthews, Gump, Block, & Allen, 1997; Roy et al., 1998).

Importantly, our study indicates that both negative and positive life events seem to modulate cardiac stress reactivity. It should be noted though that results might partly be connected to a reporting bias in life events (Pachana, Brillemann, & Dobson, 2011). Participants reported life events that they experienced throughout their whole lifetime. It is possible that potentially stressful past life events (e.g., divorce, moving to a new city) were perceived as stressful at the time of occurrence, but had overall a positive impact on the life of a person. Therefore, these events might be judged as positive in the aftermath of the event, although at the time of occurrence they might have been connected to higher levels of stress. Therefore, both types of life events might provoke adaptation processes that could impact cardiovascular functioning. This seems to be of particular importance, since dysregulation in stress response can increase an individuals’ risk for developing health problems, particularly cardiovascular diseases (Treiber et al., 2003).

It should be noted that higher cardiac regularity has been associated with psychological symptoms (e.g., depression: Leistedt et al., 2011; anxiety: Bornas et al., 2006), which are also linked with increased risk for cardiovascular diseases (Joyn, Whellan, & O’connor, 2003; Vogelzangs et al., 2010). According to the reactivity hypothesis (Treiber et al., 2003) higher CVR to stress might facilitate the development of

Table 3

| Interaction                                                    | SD1 b  | SE  | SD2 b  | SE  | SD1/SD2 b | SE  | SampEn b | SE  | HR b  | SE  |
|---------------------------------------------------------------|--------|-----|--------|-----|-----------|-----|----------|-----|-------|-----|
| Total number of LE                                           | −0.00  | 0.01| 0.01   | 0.01| −0.00     | 0.00| −0.02*   | 0.01| 0.46* | 0.17|
| Number of negative LE                                        | 0.00   | 0.01| 0.01   | 0.01| −0.00     | 0.00| −0.02    | 0.01| 0.42* | 0.20|
| Number of positive LE                                        | −0.01  | 0.02| 0.00   | 0.02| −0.01     | 0.00| −0.03*   | 0.01| 0.45  | 0.31|

LE = Life Events, SD1, SD2, SD1/SD2 = poincaré plot indexes, SampEn = sample entropy, HR = heart rate (BPM).

Table 4

| Interaction                                                    | SD1 b  | SE  | SD2 b  | SE  | SD1/SD2 b | SE  | SampEn b | SE  | HR b  | SE  |
|---------------------------------------------------------------|--------|-----|--------|-----|-----------|-----|----------|-----|-------|-----|
| Total impact of LE                                           | −0.00  | 0.01| 0.00   | 0.01| −0.00     | 0.00| −0.01    | 0.00| 0.21* | 0.08|
| Negative impact of LE                                        | −0.00  | 0.01| 0.00   | 0.01| 0.00      | 0.00| −0.01    | 0.00| 0.24* | 0.11|
| Positive impact of LE                                        | −0.01  | 0.01| 0.00   | 0.01| −0.00     | 0.00| −0.01    | 0.01| 0.17  | 0.01|

LE = Life Events, SD1, SD2, SD1/SD2 = poincaré plot indexes, SampEn = sample entropy, HR = heart rate (BPM).

Table 5

| Interaction                                                    | SD1 b  | SE  | SD2 b  | SE  | SD1/SD2 b | SE  | SampEn b | SE  | HR b  | SE  |
|---------------------------------------------------------------|--------|-----|--------|-----|-----------|-----|----------|-----|-------|-----|
| Total impact of LE x Total number of LE                       | 0.00   | 0.00| 0.00   | 0.00| 0.00      | 0.00| −0.0003* | 0.0001| 0.01* | 0.00|
| Negative impact of LE x Number of negative LE                 | 0.00   | 0.00| 0.00   | 0.00| 0.00      | 0.00| 0.00     | 0.001| 0.01* | 0.01|
| Positive impact of LE x Number of positive LE                 | 0.00   | 0.00| 0.00   | 0.00| 0.00      | 0.00| −0.00007*| 0.00003| 0.01  | 0.00|

LE = Life Events, SD1, SD2, SD1/SD2 = poincaré plot indexes, SampEn = sample entropy, HR = heart rate (BPM).

* p < .05.
cardiovascular diseases. Furthermore, research on cardiac patients showed that CVR might have a prognostic value for future clinical events. For example, Manuck, Olson, Hjernmdahl, and Rehnqvist (1992) could show that myocardial infarction patients, who evidenced larger CVR were at higher risk for cardiovascular complications (i.e., reinfarction or stroke) in the following years. Hence, the findings of the present study might indicate that in a healthy sample the experience of life events, whether positive or negative, could put the individual at risk for cardiovascular diseases via maladaptive cardiac stress responses. Certainly, future research is needed to verify or falsify this hypothesis and to examine whether findings could be generalized to clinical populations.

Importantly, it should be noted that the heterogeneity of findings within this field warrants special attention. Although this study indicates elevated cardiac stress reactivity in individuals having experienced a higher number and impact of negative and positive life events, other studies (also using comparably young samples; e.g., Tyra et al., 2020) found blunted reactivity. Future research should aim to elucidate the exact conditions under which life events are associated with either blunted or exaggerated CVR. We will specify some of these conditions below.

4.1. Limitations

The results of the present study should be interpreted in the light of important limitations. First, it should be noted that findings were contrary to our expectations, thus challenging the robustness of the effects. Hence, replication studies with larger sample sizes are strongly encouraged. Second, the effect of life events on cardiac reactivity may have been influenced by the comparably young age of the study participants. Age-related changes in CVR (Carroll et al., 2000) and variations in the number of experienced life events in different age cohorts (Hatch & Dohrenwend, 2007) limit the generalizability of our findings to older populations. Third, cardiac stress reactivity might be influenced by the nature of the task. Specifically, Musante et al. (2000) revealed that higher exposure to life events was associated with blunted hemodynamic reactivity to a car-driving simulation task, but with elevated reactivity to a social competence interview. Hence, the results of the present study might partly be influenced by the study context, impacting the transferability to studies using different stress tasks. Relatedly, it should be noted that findings were limited to heart rate and SampEn and no significant results were found for SD1 and SD2, thus questioning their sensitivity to life events. Importantly, whereas entropy measures indicate the regularity and complexity of a signal, SD1/SD2 refers to the overall predictability of the signal. Decreases in SD1/SD2 might indicate suppressed parasympathetic and increased sympathetic activation (Hsu et al., 2012). It could thus be assumed that life events are more closely associated with compromised irregularity of the cardiac stress response stemming from various sources rather than to the predictability of autonomic influences on the myocard. Certainly, further research is necessary in order to elucidate the sensitivity of non-linear HRV variables for cardiac stress research. Fourth, it is possible that other hidden variables, such as genes or personality, are driving the association between life events and cardiac reactivity, thus questioning causality. Personality might influence experience and response to life events in various ways. For example, individuals high in neuroticism are more frequently exposed to major life events (Bolger & Schilling, 1991). Furthermore, high levels of neuroticism seem to be connected to altered CVR (Bibbey, Carroll, Roseboom, Phillips, & de Rooij, 2013). Fifth, time elapsed since the experienced life events could affect reporting of the number and impact of experienced life events (Pachana et al., 2011), thus modulating the magnitude of the estimated effect sizes and possibly explaining the heterogeneity of findings in this field.

4.2. Conclusions

Notwithstanding the above-mentioned limitations, the present study suggests that the number as well as the impact of experienced life events in young adults may alter the magnitude of HR and heart rate complexity in response to acute stress. It seems that higher exposure and impact of life events could intensify cardiac stress reactivity and limiting heart complexity, thus eventually imposing a health risk. The findings of this study argue for a more detailed analysis of heart rate complexity metrics in cardiac stress research and non-linear dynamics of HRV in general, since these measures are associated with reduced flexibility in adaption processes and might also be relevant for mental health (Borras et al., 2006; Leistedt et al., 2011). The reported results require replication in future studies.

Declaration of Competing Interest

The authors wish to declare no conflict of interest.

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