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Recent life stress predicts blunted acute stress response and the role of executive control

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ABSTRACT
The present study examined the associations between recent life stress and responses to acute psychological stress, and how these associations varied with executive control. Heart rate (HR), heart rate variability (HRV), salivary cortisol, and affective states were measured before, during and after the Trier Social Stress Test (TSST), an effective laboratory stressor, in 54 healthy participants, and executive control function was tested with a Go/No-Go task in a neutral context on a different day. The hierarchical multiple regression analysis showed that high frequency of life stress during the last twelve months predicted blunted cardiovascular acute stress response, i.e., smaller HR and HRV reactivity. Moreover, the low executive control group showed a significant association between higher recent life stress and blunted acute stress response, which was not apparent in the high executive control group. The results suggested that greater executive control may benefit us with adaptive acute stress response under recent life stress.

HIGHLIGHTS
- The Trier Social Stress Test induces cardiovascular and cortisol responses.
- Higher life event frequency (LEF) predicts smaller cardiovascular stress response.
- Executive control plays a role in the link of LEF to stress response.

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KEYWORDS
Recent life stress; executive control; acute stress; heart rate variability; Go/No-Go; cortisol

1. Introduction
Stress is pervasive in real life. Exposure to an unpredictable, uncontrollable stressor can evoke allostasis physiologically and emotionally (Ulrich-Lai & Herman, 2009). A considerable amount of studies from animals and humans have revealed that acute stress could temporally elicit different responses from autonomic neural system (ANS). Firstly, sympathetic-adrenal-medullary (SAM) axis shows the most immediate responses, e.g., heart rate (HR) increases (see a review, Allen, Kennedy, Cryan, Dinan, & Clarke, 2014), while heart rate variability (HRV) decreases (Castaldo et al., 2015; Li et al., 2009; Tharion, Parthasarathy, & Neelakantan, 2009). Then, the hypothalamic-pituitary-adrenocortical (HPA) axis, which functions as a relatively slower stress response, would modulate cortisol secretion (Dickerson & Kemeny, 2004). Along with these physiological responses, acute stress also induce some emotional response, e.g., increased fear, anxiety and tension (Kelly, Tyrka, Anderson, Price, & Carpenter, 2008; Sparrenberger et al., 2009).

Proper acute stress response is considered as an adaptive function (McEwen, 1998), but excessive and prolonged stress exposure might cause deleterious consequences including prefrontal cognition dysfunction (e.g., attenuated performance in working memory, cognitive flexibility and cognitive inhibition, for a review, see Shields, Szema, & Yonelinas, 2016), physical disease (Dhabhar, 2014; Lovallo, 2011), and mental disorders like anxiety, depression, eating disorder (Allen et al., 2014; De Kloet, Joëls, & Holsboer, 2005) and post-traumatic stress disorder (PTSD) (Davidson, Stein, Shalev, & Yehuda, 2004).

Individuals vary substantially in their response to acute stress. Recently, studies have showed increasing interest in the factors predicting individual differences in stress response. A meta-analysis study reviewed that age, gender, genetics, personality traits, and social factors (i.e., social support, social status and social culture) modulate individual stress responses to acute stressors (Allen et al., 2014). In addition, cognitive factors including general intelligence (Ginty, Phillips, Der, Deary, & Carroll, 2011; Ginty, Phillips, Roseboom, Carroll, & DeRooij, 2012), executive function (Hendrawan, Yamakawa, Kimura, Murakami, & Ohira, 2012), cognitive control (Compton et al., 2011, 2008; Pleger et al., 2017), error-awareness (Wu et al., 2017), and attention bias (Fox, Cahill, & Zougkou, 2010; Pilgrim, Marin, & Lupien, 2010), have also been found associated with stress response magnitude.
Recent life stress has been found to be one factor contributing to individual differences in stress responses. However, previous studies have yielded mixed results. For example, while most studies indicated a negative association between recent life stress and heart rate responses to acute stressors in adults and adolescents (Matthews et al., 2001; Murali & Chen, 2005; Phillips, Carroll, Ring, Sweeting, & West, 2005), some studies did not show a statistically significant relationship between life events and heart rate stress responses (Cohen, Simons, Rose, McGowan, & Zelson, 1986; Jorgensen & Houston, 1989; Lepore et al., 1997).

The mixed results have been found to be due to some modulating variables, such as personality, coping style, and social network (Peng et al., 2012; Phillips et al., 2005; Roy, Steptoe, & Kirschbaum, 1998). Among many potential moderators of the relationship between life stress and acute stress responses, executive control function could be an important one. Executive control has strong link with stress regulation. On behavioral level, executive control performance is predictive of dampened responses to acute stress (Hendrawan et al., 2012; Williams, Suchy, & Rau, 2009). On neural level, prefrontal cortex is crucial both in executive control (Ridderinkhof, Van Den Wildenberg, Segalowitz, & Carter, 2004) and stress regulation (Cerqueira, Mailliet, Almeida, Jay, & Sousa, 2007). In addition, previous studies found that executive control can modulate the relationship between life stress and stress response. For instance, greater neural activity during error-monitoring process has been found to predict less stress reactivity to daily stress (Compton et al., 2011). Thus, executive control may be a crucial modulator in the relationship between acute stress response and life stress.

Most studies on the relationship between recent life stress and acute stress response focused on HR or blood pressure stress reactivity. However, studies on other aspects of stress response, i.e., HRV, cortisol level, and subjective affect, are limited. HRV is a measure of the continuous interplay between the influences of SNS and PNS on heart rate, indicating adaptive ability of the heart under variable circumstances (Appelhans & Luecken, 2006; Castaldo et al., 2015). Up to now, one study has reported that accumulation of violence experiences was associated with smaller HRV decline towards acute stressor in adolescent (Murali & Chen, 2005). For cortisol response, one study showed that more stressful life events were associated with a reduced cortisol stress response in children (8–12 years) (Armbuster et al., 2012); a recent study revealed that participants’ exposure to entire life stressors predicts a blunted cortisol response to acute stress in adults (Lam, Shields, Trainor, Slavich, & Yonelinas, 2019). However, how recent life stress would predict these different aspects of acute stress responses in young adults remains unclear.

This study aimed to examine whether recent life stress would predict acute stress responses and how executive control function would modulate their associations. We used a standard laboratory stressor to elicit acute stress responses in healthy adults. The stress responses were collected and calculated comprehensively before, during and after the acute stress induction, including HR, HRV, cortisol, positive and negative affect, and control feeling of the cute stressor. Furthermore, executive control function was measured as false alarm rate (FAR) in the Go/No-Go task (Cheung, Mitsis, & Halperin, 2004). Based on previous findings, we firstly test the hypothesis that high frequency of recent life stress would be associated with lower stress responses. We also explored the role of executive control in the relationship between recent life stress and acute stress responses.

2. Methods

2.1. Participants

This study was based on a secondary analysis of data from a project on individual psychophysiological response to acute stress (Xin et al., 2017). Fifty-four participants (35 males), aged from 18 to 25 years (mean 22.57 ± 1.67) with 13–18 education years (mean 15.89 ± 1.34) were recruited. Participants were excluded in case of (a) psychiatric illnesses, neurological diseases, endocrine disorders or major physical illness; (b) severe head trauma or brain damage (e.g., brain surgery, cerebral haemorrhage); and (c) major operation in the last 6 months. Also, participants were not to be ill, taking medicines, suffering from certain chronic diseases, be pregnant, live an irregular lifestyle (i.e., prolonged reversed day and night schedule) or staying up during the 3 days prior to the study. Female subjects were not in their ovulation phase of menstrual cycle. All participants were right-handed, not heavy smokers (no more than five cigarettes a day) and not alcoholics (no more than two alcoholic drinks a day), with normal or corrected-to-normal vision. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Human Experimentation in the Institute of Psychology, Chinese Academy of Sciences. The written informed consent was obtained from every subject before participating in the experiment.

2.2. General procedure

To avoid the influence of circadian rhythm of cortisol levels (Dickerson & Kemeny, 2004) the study was conducted during afternoon, starting at approximately 13:30. Participants were asked not drinking or eating anything except water and avoid vigorous exercise within one hour before coming to the laboratory. All participants reported that they did comply with these requirements. Participants came twice in 2 weeks. On the first afternoon, participants were asked to complete a Go/No-Go task. On the second afternoon, participants were instructed to remain seated for 30 min in a quiet room before the acute stress induction. During this resting period, they filled out questionnaires including demographic information (i.e., age, sex, education), personality with the mini-International Personality Item Pool (see details in Xin et al., 2017) and the Adolescent Self-Rating Life Events Check List (Liu et al., 1997) (see details below). Baseline measurements of salivary sample (SS), HR, positive affect (PA) and negative affect (NA) were collected immediately after rest. Then, the stress induction was conducted with the Trier Social Stress Test (TSST, see details below). HR was constantly recorded.
during the preparation, speech, and arithmetic parts of the TSST. After the TSST task, SS, HR (continuous recording for 5 min), PA, NA, and feeling of Control (FoC) on TSST were collected again. Then SS and HR were collected at 20 min, 45 min, and 60 min after the end of TSST. The general procedure is illustrated in Figure 1.

2.2.1. Go/No-Go task
Participants were asked to complete a Go/No-Go task similar to that of previous studies in our project (Wu et al., 2017). The task included one practice block of 10 trials and two experimental blocks of 240 trials in each one. Stimuli were the digits of "1" or "9". The probability was 20% for No-Go trials and 80% for Go trials. In each trial, one digit was presented in the center of the screen for 150 ms followed by an inter-stimulus interval varied randomly between 1200 and 1500 ms. Participants were asked to respond as quickly as possible to Go trials (either "1" or "9") by pressing a button on the keyboard and inhibit the response in No-Go trials ("9" or "1", respectively). The target stimulus for Go trials ("1" or "9") was counterbalanced across participants.

2.2.2. The laboratory stress task-Trier Social Stress Test
A modified TSST from Kirschbaum and his colleagues (Buchanan, Tranel, & Kirschbaum, 2009) was used to induce acute stress. During the preparation period, participants were asked to prepare a 5-min speech to defend themselves (in an imaginary situation) against a store manager who charged them of shoplifting. They could take notes at preparation but were not allowed to take the notes during speaking. After the preparation, participants completed the speech and mental arithmetic tasks with a microphone and were videotaped (which they were aware of). Throughout the speech and mental arithmetic tasks, three experimenters (two females and one male) with white coats and neutral facial expressions were present. For the mental arithmetic task, participants were asked to subtract 13 consecutively, beginning with 1022. They were asked to respond as quickly and accurately as possible and restart at 1022 if a mistake occurred.

2.2.3. Recent life stress
Recent life stress was assessed using the Adolescent Self-Rating Life Events Check List (ASLEC) (Liu et al., 1997). This scale has 27 items, including six dimensions of interpersonal relationship, study pressure, being punished, bereavement, change for adaption, and others, which are all typical negative life event in college student. Subjects were asked whether they had experienced events pertaining to these items in the last 12 months and their perceived severity of the event they experienced. The life events frequency (LEF) was reported as the recent life stress score. The Cronbach's alpha was 0.85 for perceived severity, but not applied for the frequency (Liu et al., 1997).

2.2.4. The positive and negative affect
The subjective affective was rated using the Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988) with a 5-point Likert scale (1-not at all, to 5-very much). The scale consists of 20 items, with 10 for positive affect (interested, excited, inspired and alert) and 10 for negative affect (distressed, nervous, scared and upset). The PANAS has strong internal consistency for moment-measurement with Cronbach's alphas of 0.89 for PA and 0.85 for NA (Watson et al., 1988).

2.2.5. Feeling of control
Participants rated their subjective control levels on the speech and arithmetic task of the TSST with a visual analog scale from 0- "out of control" to 10- "a strong sense of control".
2.2.6. Physiological stress response measurement

HR was recorded with a Biopac Amplifier System (MP150; Biopac, Goleta, CA, USA) with a sample rate of 1000 Hz. During the recording, three electrocardiograph electrodes were placed on the right side of the neck and the left and right inner ankles. HR was defined as the number of beats per minute (bpm), calculated by the mean of 5-min continuous recording at each collection point using AcqKnowledge software. HRV was analyzed with Kubios Analysis software (Biomedical Signal Analysis Group, University of Kuopio, Finland). Root mean square of successive differences (RMSSD) of inter beat intervals and high frequency (0.15–0.4 Hz) component of HRV (HF-HRV) were used as the indices of HRV, which are mainly mediated by vagal activity (Malik, Bigger, Camm, & Kleiger, 1996). Saliva samples were collected using Salivettes (Sarstedt, Rommelsdorf, Germany). Samples were frozen at −22°C until analysis. Before analysis, samples were dissolved and centrifuged at 3000 rpm for 10 min. Then cortisol in saliva was measured using electrochemiluminescence immunoassay (Cobas e 601, Roche Diagnostics, Numbrecht, Germany). The lower sensitivity for cortisol was 0.5 nmol/L. Intra- and inter-assay variations were less than 10%.

2.3. Data analysis

To examine whether the stress elicitation was effective, the repeated measure ANOVA was conducted on HR, RMSSD, HF-HRV and Cortisol across time points, and the paired repeated measure ANOVA was conducted on HR, RMSSD, HF-HRV, and Cortisol across time points. To examine whether the stress elicitation was effective, the paired repeated measure ANOVA was conducted on HR, RMSSD, HF-HRV, and Cortisol across time points. The affect control score was the mean of control feeling on speech and mental arithmetic task. As studies have showed sex difference in acute stress responses (see a meta-analysis, Liu et al., 2017), and neuroticism was found significantly linked with acute stress responses in our previous study (Xin et al., 2017), we were entered at step 1 as covariates in the hierarchical regression analysis. The recent life stress was entered at step 2.

Next, we performed a moderation analysis using PROCESS macro for SPSS (Hayes, 2017). Firstly, subjects were divided into high and low executive control groups by FAR median (Cerqueira et al., 2007). The FAR was calculated by the proportion of error in all No-Go trials, with higher FAR meaning lower executive control. Then the moderation analysis was conducted with 5000 bootstrap samples with executive control as moderator, stress responses as dependent variables, recent life stress as independent variable, and sex and neuroticism as covariates.

Extreme values (values larger than three times of the interquartile range) in independent variables were deleted before the hierarchical multiple regression analyses. Two participants were excluded from the HR and HRV analyses. Recent life stress score was transformed to normal distribution with Box-Cox transformation (Box and Cox, 1964) with MedCalc Statistical Software version 15.6.1 (MedCalc Software bvba, Ostend, Belgium; https://www.medcalc.org; 2015). Other statistical analyses were implemented with SPSS 20.0 (IBM Corp. Armonk, NY). All reported p-values were two-tailed with the significance level of .05.

3. Results

3.1. Description of recent life stress and false alarm rate

Subjects had an average of 7.593 (SD = 6.045) stressful life events in the last 12 months, and an average FAR of 13.5% (SD = 9.8%).

3.2. Physiological and psychological stress response

For HR, the repeated measure ANOVA revealed a significant main effect of time, \(F(3,237,171.542) = 85.483, p < 0.001, \) partial Eta-square = 0.617. Post hoc analysis indicated that HR during speech and mental arithmetic periods was higher than HR at all other time points, with all \(p \) values < 0.05. HR during preparation was higher than HR at baseline and time points after the end of the TSST, \(p \) values < 0.01. No other significant differences were found between time points, with all \(p \) values > 0.10. The mean (± SD) of ΔHR was 11.728 (±8.209) bpm (see Figure 2(a)).

For RMSSD, the repeated measure ANOVA revealed a significant main effect of time, \(F(3,456, 183.165) = 23.601, p < 0.001, \) partial Eta-square = 0.308. Post hoc analysis indicated that RMSSD during the speech and mental arithmetic periods was significantly lower than measures at other time points, with all \(p \) values < 0.01. No other significant differences were found between time points, with all \(p \) values > 0.05. The mean (± SD) of ΔRMSSD was −9.601 (± 16.585) (see Figure 2(b)).

For HF-HRV, the repeated measure ANOVA revealed a significant main effect of time, \(F(3,936, 208.590) = 17.140, p < 0.001, \) partial Eta-square = 0.562. Post hoc analysis indicated that HF-HRV during the speech and mental arithmetic periods was significantly lower than measures at other time points, with all \(p \) values < 0.05. No other significant differences were found between time points, with all \(p \) values > 0.05. The mean (± SD) of ΔHF-HRV was −208.55 (± 880.10) (see Figure 2(c)).

For cortisol, results of the repeated measure ANOVA using the factor of time revealed a significant main effect, \(F(2.405, 127.479) = 40.465, p < 0.001, \) partial Eta-square = 0.433. Cortisol reached its peak at 20 min after the completion of TSST (M = 14.693, SD = 5.550), which was significantly higher.
than cortisol concentration measured at other time, with all \( p \) values < 0.001. Cortisol at 0 min and 45 min after the completion of TSST was higher than that of baseline and 60 min after the completion of the TSST, with all \( p \) values < 0.01. However, there was no difference in cortisol between 0 min and 45 min after the completion of TSST, and between baseline and 60 min after the completion of the TSST. The mean (± SD) of ΔCortisol was 5.924 (± 5.855) nmol/L (see Figure 2(d)).

For PA, the paired sample \( t \)-test revealed no significant difference between PA at baseline and end of TSST, \( t(53) = 1.204, p = 0.234 \). The mean (± SD) of ΔPA was 0.852 (± 5.199) (see Figure 2(e)).

For NA, participants experienced more negative affect (NA) at the end of TSST than baseline, \( t(53) = 4.836, p < 0.001 \). The mean (± SD) of ΔNA was 3.278 (± 4.981) (see Figure 2(f)).

The mean (± SD) of FoC on TSST was 5.300 (± 1.678).

### 3.3. Results of the hierarchical regression analyses

Table 1 shows the hierarchical multiple regression results of recent life stress on stress responses towards TSST.

#### 3.3.1. Regression of recent life stress on HR stress response

The result showed that recent life stress significantly added 7.2% of the explained variance after controlling for covariates (\( \Delta R^2 = 0.072, R^2 = 0.156, F (3, 48) = 2.955, p = 0.042 \). And recent life stress could independently predict HR response (\( \beta = -0.274, t = -2.025, p = 0.048 \)). The more stressful life events subjects experienced, the smaller HR acute stress response was, for scatterplot of simple correlation, see Figure 3(a).

![Figure 2](image-url)

**Figure 2.** Trend of (a) HR, (b) RMSSD, (c) HF-HRV, (d) Cortisol, (e) PA, and (f) NA across measure time. Error bar represents standard deviation. 0, 20, 45, 60 are 0 min, 20 min, 45 min and 60 min after the end of the Trier Social Stress Test (TSST) separately. HR: heart rate; RMSSD: root mean square of successive differences; HF-HRV: high frequency component of heart rate variability; PA: positive affect; NA: negative affect.

### Table 1. Results of hierarchical multiple regression on stress responses.

<table>
<thead>
<tr>
<th>Responses</th>
<th>Predictors</th>
<th>( \beta )</th>
<th>( t )</th>
<th>( p )</th>
<th>( R^2 )</th>
<th>( \Delta R^2 )</th>
<th>( F )</th>
<th>sig-( F )</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔHR</td>
<td>Sex</td>
<td>0.098</td>
<td>0.736</td>
<td>0.465</td>
<td>0.084</td>
<td>0.001</td>
<td>0.084</td>
<td>0.117</td>
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<tr>
<td></td>
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<td>-1.714</td>
<td>0.093</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ΔRMSSD</td>
<td>Sex</td>
<td>-0.247</td>
<td>-2.025</td>
<td>0.048</td>
<td>0.156</td>
<td>0.072</td>
<td>2.955</td>
<td>0.042</td>
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<tr>
<td></td>
<td>Neuroticism</td>
<td>-0.171</td>
<td>-1.208</td>
<td>0.204</td>
<td>0.038</td>
<td>0.038</td>
<td>0.977</td>
<td>0.384</td>
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<tr>
<td>ΔHF-HRV</td>
<td>Sex</td>
<td>0.066</td>
<td>0.486</td>
<td>0.629</td>
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<tr>
<td></td>
<td>Neuroticism</td>
<td>0.362</td>
<td>2.693</td>
<td>0.010</td>
<td>0.165</td>
<td>0.126</td>
<td>3.151</td>
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<tr>
<td>ΔCortisol</td>
<td>Sex</td>
<td>-0.104</td>
<td>-0.747</td>
<td>0.220</td>
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<tr>
<td></td>
<td>Neuroticism</td>
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<td>-0.203</td>
<td>0.840</td>
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</tr>
<tr>
<td>ΔPA</td>
<td>Sex</td>
<td>0.336</td>
<td>2.678</td>
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<td>0.211</td>
<td>0.211</td>
<td>6.823</td>
<td>0.002</td>
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<tr>
<td></td>
<td>Neuroticism</td>
<td>-0.361</td>
<td>-2.849</td>
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<td>4.864</td>
<td>0.005</td>
</tr>
<tr>
<td>ΔNA</td>
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<td>0.022</td>
<td>0.570</td>
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<tr>
<td></td>
<td>Neuroticism</td>
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<tr>
<td>ΔFoC</td>
<td>Sex</td>
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<td>-0.698</td>
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<td>0.023</td>
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<td>-2.979</td>
<td>0.026</td>
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</table>

Note. ΔHR/ΔRMSSD/ΔHF-HRV/ΔCortisol/ΔPA/ΔNA: HR/RMSSD/HF-HRV/Cortisol/PA/NA changes to TSST. FoC: average feeling of control on speech and mental arithmetic. \( \beta \): standard coefficient. LEF: recent life event frequency. Enter method: enter. * \( p < 0.05 \); ** \( p < 0.01 \); *** \( p < 0.001 \).
The result showed that recent life stress significantly added 12.6% of the explained variance after controlling for covariates (ΔR² = 0.126, R² = 0.165, F(3, 48) = 3.151, p = 0.033). And recent life stress could independently predict RMSSD response (β = 0.362, t = 2.693, p = 0.010), the more stressful life event subjects experienced, the smaller RMSSD decrease was, for scatterplot of simple correlation, see Figure 3(b).

The result showed that recent life stress added 9.2% of the explained variance (ΔR² = 0.092, R² = 0.108, F(3, 48) = 1.942, p = 0.135). And recent life stress could independently predict the HF-HRV response after controlling for covariates (β = 0.310, t = 2.227, p = 0.031), the more stressful life event subjects experienced, the smaller HF-HRV decrease was, for scatterplot of simple correlation, see Figure 3(c).

The predictions of recent life stress or covariates on Cortisol change were not significant, see Table 1.

The result showed that recent life stress was not a significant predictor to PA change after controlling for covariates (β = 0.124, t = 0.978, p = 0.333). In addition, the covariates significantly contributed 21.1% explained variance to the regression model (ΔR² = 0.211, R² = 0.226, F (3, 50) = 4.864, p = 0.005).

The predictions of recent life stress or covariates on NA change were not significant, see Table 1.

Figure 3. Scatter plots showing associations between LEF and (a) ΔHR, (b) ΔRMSSD, (c) ΔHF-HRV. LEF: life event frequency. ΔHR/ΔRMSSD/ΔHF-HRV: HR/RMSSD/HF-HRV change during TSST to baseline.

3.3.6. Regression of recent life stress on NA response to TSST

4. Discussion

The current study was aimed to examine the relationship between recent life stress and acute stress responses, and how executive control moderated this relationship. The result has showed that the TSST caused an increase in HR, a decrease in HRV, and an increase in cortisol, indicating a successful stress induction. Importantly, recent life stress negatively predicted cardiovascular responses to acute stress (blunted HR increment and HRV decrement), but did not predict cortisol or subjective stress responses. Notably, people with different levels of executive control function manifest different patterns of relationship between recent life stress and HRV stress response, i.e., the group with low executive control showed a negative correlation in line with the overall
trend, however, such correlation was not found in the high executive control group.

This study related high frequency of life stress exposure during the last twelve months to a low level of cardiovascular responses to acute stress. The finding is consistent with prior studies on cardiovascular stress responses (see a cohort study, Phillips et al., 2005) and extended earlier reports by finding the relationship between recent life stress and HRV, i.e., a smaller HRV decline to acute stress (representing lower stress response). Our finding is also consistent with the result of a previous study in adolescence which focused on violence-related experiences in life events (Murali & Chen, 2005) and extended this study by measuring a broad range of stressful life events in adults, including interpersonal relationship problems, study pressure, and being punished, thus providing new evidence that general stressful life experience was related to heart rate dynamic change under acute stress in adults.

The findings may be interpreted in two different perspectives. Firstly, people experiencing a lot of adverse events over a period may become hypo-sensitive to emergencies (Boyce & Chesterman, 1990), thus the laboratory-stressor was evaluated as less stressful, plausibly arouse relatively smaller autonomic acute stress response. Secondly, being exposed to stressful life events frequently over a defined period, the body might be chronically stressed and stay in a high arousal state (Vente, De Olff, Amsterdam, Van Kamphuis, & Emmelkamp, 2003), causing psychophysiological resources being occupied and consumed. This allostatic load (Duan et al., 2015; Logan & Barksdale, 2008) then makes it hard to mobilize and reallocate resource to deal with emergency, thus showing a dampened acute response.

It is worth noticing that executive control moderated the relationship between recent life stress and HRV stress response in the present study. Subjects were divided into lower group and higher group by the median of executive control score. The group in low executive control showed the negative association found in the whole group, i.e., greater recent life stress exposure, smaller RMSSD/HF-HRV decline to the TSST. According to our knowledge, no similar study has explored how executive control moderated the relationship between recent life stress and acute stress response. The lack of association between recent life stress and acute stress response in high executive control group might be explained by that higher executive control is linked to more effective prefrontal cortex function. Competitive PFC function in the higher executive control group may provide them with greater capacity to cope with life stressors, i.e., they may appraise some stressful life events in more flexible and adaptive ways (Williams et al., 2009). Thus, accumulated life events may not be experienced much stressful to cause maladaptive reactivity to acute stress. Our finding of the role of executive control on acute stress response under recent life stress may also help to explain the mixed results in literature regarding the relationship between recent life stress and acute stress response.

There are some limitations. Firstly, although the TSST is a standard task for acute stress induction and stress responses can be conveniently measured in the laboratory, the intensity of this laboratory-based acute stressor is in the moderate level, which may not necessarily represent real-world life stressors. Secondly, the present study indicated that one aspect of executive control, inhibitory control, played a role in modulating the relationship between recent life stress and HRV reactivity to stress, roles of other aspects of executive control (e.g., cognitive flexibility, working memory, emotion regulation, decision making and so on), need further consideration. Third, being limited to the relatively small sample size which had a test power (1-β) range of 0.74–0.92 by post-hoc analyses with G*Power 3.1 (http://www.gpower.hhu.de/en.html), more work is needed to validate this finding in a generalized population.

5. Conclusion

In conclusion, this study suggested that individual’s cardiovascular responses to acute stress was predicted by recent life stress and this relationship was moderated by executive control, i.e., this association was less apparent in individuals with higher executive control. Notwithstanding the relatively limited sample, these findings suggest that higher frequency of recent life stress could probably influence our adaptive response to acute stress, which may be caused by the allostatic load or by the under-evaluation of stressful event, but greater executive control can possibly prevent such adverse effects.
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