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Early life stress moderated the influence of reward anticipation on acute psychosocial stress responses

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Abstract

Recent studies suggest that reward anticipation decreases individuals' acute stress responses. However, individuals who have experienced early life stress (ELS) may have a blunted capacity for reward anticipation, which reduces its buffering effect on psychosocial stress responses. To investigate this phenomenon, 66 young adults completed the Trier Social Stress Test following a reward anticipation task, and their ELS levels were measured using the Childhood Trauma Questionnaire (CTQ). Meanwhile, the current study collected biological and psychological measures of stress by analysing cortisol levels, heart rates, heart rate variability (HRV) as well as subjective stress levels, in response to the Trier Social Stress test. Results showed that reward anticipation successfully decreased acute stress responses in general, and it also improved participants' HRV. However, this effect was more evident in individuals with low ELS than those with high ELS. These findings help us deepen understanding of the role of reward anticipation in fostering resilience under stress and the potentially important implications for individuals who have been exposed to ELS are also discussed.

KEYWORDS

cortisol response, early life stress, heart rate variability, reward anticipation, Trier Social Stress Test

1 | INTRODUCTION

Physical or mental imbalances caused by unexpected or uncontrollable stimuli may induce stress as an adaptive compensatory response to maintain homeostasis (Pacak & Palkovits, 2001). Excessive, prolonged, or inadequate regulation of the stress response systems will invariably cause individuals to suffer harmful health consequences (Guilliams & Edwards, 2010). For example, repeated or chronic stressors can lead to hypothalamic-pituitary-adrenal (HPA) axis dysregulation, which changes appropriate cortisol secretion and affects end-organ function (Lupien et al., 2009). Moreover, stress can leave people feeling anxious and distressed, making them more susceptible to numerous physical and mental diseases (Lazarus & Folkman, 1984).

Since reducing excessive stress responses may promote health, a lot of studies have been undertaken to find ways to reduce excessive stress responses (Creswell & Lindsay, 2014; Haslam et al., 2016). For example, a recent study showed that imagining a positive future through long-term interventions reduced cortisol reactivity to acute stress (Nicolson et al., 2020). Our existing study further indicated that reward anticipation via experimental manipulation relieved participants' subjective feelings of stress, and decreased the overall cortisol secretion and heart rate induced by psychosocial stress (Hu & Yang, 2021). Regarding the mechanism, several studies theorized that anticipation of future rewards may increase participants' positive affect, which further promotes positive cognitive reappraisal of stressors (Folkman & Moskowitz, 2000; Kringelbach & Berridge, 2009; Nicolson

et al., 2020). This creates the inverse pairing of activating the reward system against the HPA axis stress response (Dutcher & Creswell, 2018). Notably, the buffering effect of anticipating future reward may be dependent on emotional states and life experiences. Thus, understanding the contributing factors for interpersonal heterogeneity in the role of reward anticipation is key in preventing and treating stress-related disorders (Gan et al., 2019; Russo et al., 2012; Rutter, 2006; Zhou et al., 2018).

Early life stress (ELS) refers to considerable adversities and stressful social experiences in early life, such as child neglect or abuse (Brown et al., 2009). Converging evidence indicates that ELS causes persisting changes to emotional cognitive processing. This creates considerable pathogenic factors for the development of mental illness, addictive behavior, and personality disorders (Carr et al., 2013; Neigh et al., 2009; Pechtel & Pizzagalli, 2011). Furthermore, a recent review indicated that individuals with high ELS have deficits in generating and perceiving reward anticipation (Novick et al., 2018). For example, adults who experienced childhood abuse rated reward cues less positively than adults with no history of abuse (Dillon et al., 2009). Additionally, neuroimaging studies also support this perspective. Such studies have indicated that individuals with higher level of early stress exposure showed lower neural responses to reward cues in the basal ganglia, including in the nucleus accumbens, caudate nucleus, putamen, and globus pallidus during reward anticipation processing (Boecker et al., 2014; Dillon et al., 2009; Goff et al., 2013; Hanson et al., 2016; Mehta et al., 2010). In addition, many studies have found that ELS can affect the HPA axis response to acute stress (e.g., Carpenter et al., 2009; Grimm et al., 2014; Heim et al., 2008), and the question of how ELS shapes the activity of the HPA axis in individual's growth particularly important (Del Giudice et al., 2011). Reward anticipation could protect the stress system from the negative effects of ELS by alleviating an excessive cortisol response, and the dysfunction of reward anticipation may aggravate the existing damage to the stress system. In this study, we sought to understand the moderating role of ELS on the buffering effect of reward anticipation.

In summary, exposure to adversity during early development can affect individuals' reactivity to reward anticipation. This manifests in the decline of anticipatory pleasure and approach motivation during the processing of reward anticipation-related stimuli or events (Novick et al., 2018). Furthermore, the buffering effect of reward anticipation is based on an individual's perception and anticipation of expected future reward. Therefore, the impaired sensitivity toward reward anticipation and the dysfunction of the reward system in individuals with high ELS may moderate the effect of reward anticipation on the acute stress response. Thus, we hypothesize that reward anticipation would decrease the HPA axis and autonomic nervous system stress responses to acute psychosocial stress in individuals with low ELS, but not those with high ELS. To test this hypothesis, we recruited participants to complete the Trier Social Stress Test (TSST), following a reward anticipation task. Participants' ELS levels were measured using the CTQ. Indicators of stress responses included participants' subjective stress reports, cortisol and heart rates, and heart rate variability (HRV), which stems from the heart responding to physiological oscillation signals.

2 | MATERIAL AND METHOD

2.1 | Participants

We used G*Power to estimate the sample size for the linear regression analyses, with $\alpha = .05$, power $(1\text{-}\beta) = 0.8$, and medium effect size (Faul et al., 2009). We thus arrived at a sample size of $n = 68$. Seventy-two participants were recruited via advertisements from a local university. A total of six participants were excluded due to missing endocrine or cardiovascular data. The final sample comprised 66 participants (42 females and 24 males), with ages ranging between 18 and 26 years (mean age = 20.02, $SD = 1.26$). We ascertained participants' eligibility, current health status, and health behaviors using self-reports from the potential participants. Exclusion criteria were acute or chronic psychiatric or somatic diseases, intake of psychotropic or glucocorticoid medication, alcohol/drug abuse, and enrolment in other TSST studies. Participants were informed that the study was designed to investigate social cognitive function, and would last approximately 1 hr 30 min. Participants were randomly assigned to the reward anticipation (RA) and non-reward anticipation (NA) groups. Demographic data for each group are presented in Table 1. Participants were asked to refrain from smoking, engaging in strenuous exercise, and drinking alcohol or caffeine on the

TABLE 1 Descriptive data of demographic variables

	Reward anticipation	Non-reward anticipation	*p*
Age in years, mean (*SD*)	20.26 (1.24)	19.75 (1.24)	.097
Gender			
Male, *n*	14	10	
Female, *n*	20	22	
ELS, mean (*SD*)	37.50 (11.90)	37.75 (14.69)	.940
Emotional neglect	9.06 (4.36)	9.66 (5.45)	.623
Emotional abuse	8.15 (3.29)	8.06 (4.06)	.926
Physical neglect	7.44 (2.93)	7.84 (2.95)	.580
Physical abuse	5.91 (1.56)	6.16 (3.11)	.685
Sexual abuse	5.50 (1.08)	5.50 (1.65)	1.000

Abbreviations: ELS, early life stress; *SD*, standard deviation.

day of their appointment. They were instructed not to eat or brush their teeth in the hour prior to the session.

This study was approved by the Research Ethics Committee of the University and was performed in line with the Declaration of Helsinki. We obtained informed oral consent from all participants prior to conducting the experiment. All participants received monetary compensation to the value of 40 yuan for their participation. Participants in the reward anticipation group earned extra cash rewards.

2.2 | Measurement of ELS

ELS was measured using the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1998). The CTQ is a 28-item self-report questionnaire designed to retrospectively assess five types of adverse childhood experiences. These include emotional neglect, emotional abuse, physical neglect, physical abuse, and sexual abuse. Scores range from 5 to 25 for each subscale, with higher scores indicating frequent exposure to the corresponding type of maltreatment. This study utilized the Chinese version of the CTQ, which has good validity and reliability in Chinese populations (Fu et al., 2005). The current sample indicated an adequate internal consistency for CTQ-SF (Cronbach' s $\alpha = .91$).

2.3 | Reward anticipation manipulation

Participants were instructed to draw a token from a box, either labeled as "reward" or "no reward". Participants in the reward anticipation condition were told: "You have won an entry into a raffle draw after the experiment. You may win between 10 to 50 yuan in extra cash." Participants in the non-reward anticipation condition were told: "You did not win an entry into the raffle draw." To reinforce the effect of reward anticipation manipulation, the experimenter reminded participants about the raffle every 10 min following the stress task in the reward anticipation group.

Participants' moods were measured using the positive and negative affect schedule (PANAS) before and after the reward anticipation tasks (Watson et al., 1988). After the session, participants were asked to indicate: "How much are you looking forward to the raffle after the reward anticipation task?" Responses were rated on a 7-point Likert-scale to determine the perceived level of reward anticipation. Scores ranged from 1 ("not at all") to 7 ("very much").

2.4 | Stress treatment

The TSST is a standardized psychosocial stress test that can effectively activate HPA axis responses in laboratory research (Kirschbaum et al., 1993). In the TSST group, we set up two interviewers of different genders, one camera, and one microphone. Participants were asked to prepare for a mock job interview, which involved delivering a five-minute long application speech to the two interviewers. The speeches were video recorded. The interviewers remained impassive throughout the interview. Afterwards, participants were asked to complete a five-minute continuous verbal subtraction task. This included problems such as subtracting 13 from 1,022 as quickly and accurately as possible. If participants answered incorrectly, the interviewer would interrupt them and ask them to restart.

2.5 | Stress response measurement

2.5.1 | Subjective stress

Participants were asked to indicate their subjective stress levels during the stress task on a 7-point Likert-scale. Responses ranged from 1 ("not stressful") to 7 ("very stressful").

2.5.2 | Neuroendocrine response

Salivary cortisol was collected as the neuroendocrine indicator of the stress response. A saliva collector (salivate SARSTEDT, Germany) was used to store samples. Participants were asked to place a cotton bud into their mouth, chew on it for one minute, and spit it back into the sampler. Participants were reminded to refrain from touching the cotton buds with their hands or any other objects during the process to avoid contaminating the sample. Samples were stored in a refrigerator at −20℃. The concentrations of cortisol in the saliva samples were analyzed using an enzyme-linked immunosorbent assay (IBL-Hamburg, Germany), according to the manufacturer's instructions. The sensitivity of the cortisol assay was 0.005 μg/dl. The inter- and intra-assay coefficient of variation for the cortisol assays were 3.1% and 6.4%, respectively.

2.5.3 | Cardiovascular response

Cardiac activity data were monitored continuously at a sampling rate of 1 kHz using a Biopac MP150 system. Specifically, participants' cardiovascular activity was recorded using an electrocardiogram amplifier module and three disposable electrodes positioned on the chest, left armpit, and abdomen. Heart rate and HRV were assessed from baseline to the end of the experimental task. The variability in the interval between successive R peaks (R-R interval, RRIs) was identified from electrocardiogram (ECG) recordings to calculate HRV.

The tasks examining heart rate and HRV did not commence until a clear and accurate ECG recording was obtained. All relevant segments of ECG recordings were visually inspected before determining heart rate and HRV. Undetected R waves were manually inserted where appropriate. Ectopic beats and artifacts were excluded from the analysis. The raw ECG signal was filtered using a 0.5 to 35 Hz band-pass filter, sampled at a rate of 2,000 Hz.

Heart rate was reported in beats per minute (BPM). The root mean square of successive differences (RMSSD), measured in milliseconds, was calculated for HRV. It is a stable (Li et al., 2009) and valid (Thayer & Sternberg, 2010) time-domain measure of vagally mediated HRV. Participants' successive heart rates and RMSSD were extracted and analyzed using the AcqKnowledge software package (Biopac Systems, Goleta, CA). RMSSD values were natural log-transformed (ln) to fit the assumptions of linear analyses (Ellis et al., 2008).

2.6 | Procedure

Experiments were conducted from 2:30 p.m. to 5:00 p.m. to control for the diurnal rhythm of cortisol. Figure 1 details the experimental procedure. The experiment was conducted in a quiet room. Participants were asked to rest for at least 30 min upon arrival, while completing the questionnaire. Following the acclimation period, participants provided a baseline saliva sample for the assessment of cortisol levels. Participants then completed the reward anticipation task and evaluated their emotions before and after the task, using the PANAS. Next, participants prepared for the stress task. After 10 min, participants were sent to the testing room to complete the TSST. Upon completion, participants were instructed to return to the waiting room and rest. Participants were allowed to withdraw from the experiment at any time.

Throughout the experiment, participants' heart rates were monitored continuously and computed at eight time points (see Figure 1). The time points were as follows: T0 = −20 min (baseline measurement), T1 = −15 min (reward anticipation task end), T2 = 0 min (TSST start), T3 = 5 min (mental arithmetic start), T4 = 10 min (TSST end), T5 = +20 min (Rest 1), T6 = +30 min (Rest 2), T7 = +40 min (recovery end). Saliva samples were obtained at three time points: T0, T5, and T7. Subjective stress reports were collected at seven time points: T0, T1, T2, T4, T5, T6, and T7.

2.7 | Statistical analyses

All variables were examined for distributional properties and cases were deleted for univariate outliers. Participants' overall cortisol levels, heart rates, and subjective stress ratings were summarized, applying a method to calculate the area under the curve regarding increases (AUC_I) contingent on the baseline level (Pruessner et al., 2003). See Supplementary Information for the means, standard deviations and correlations between gender, ELS, and each stress response indicators in different groups. Linear regression models were used to investigate the interaction effect of reward anticipation and ELS on stress responses (AUC_I of cortisol responses, heart rates, subjective stress levels, and lnRMSSD). Besides, previous studies suggested that gender could be an important variable affecting sensitivity to reward anticipation in individuals with ELS experience and stress response; therefore, we included gender as a covariate in our analyses (Casement et al., 2014; Rincón-Cortés et al., 2019). Analyses were conducted using IBM SPSS Statistics for Windows, version 20.0 (IBM, Armonk, NY, USA).

First, gender was entered as a covariate. Meanwhile, reward anticipation and ELS were entered as main predictors, successively. Then, a two-way interaction reward anticipation (RA) × ELS was entered. All predictors were mean-centred. To examine the moderating effects, we tested whether successive regression steps significantly increased the variance described by the model (ΔR^2). To facilitate the interpretation of significant interaction terms, tests of the simple slope at high (+1 SD) and low (−1 SD) levels of ELS were conducted (Aiken et al., 1991).

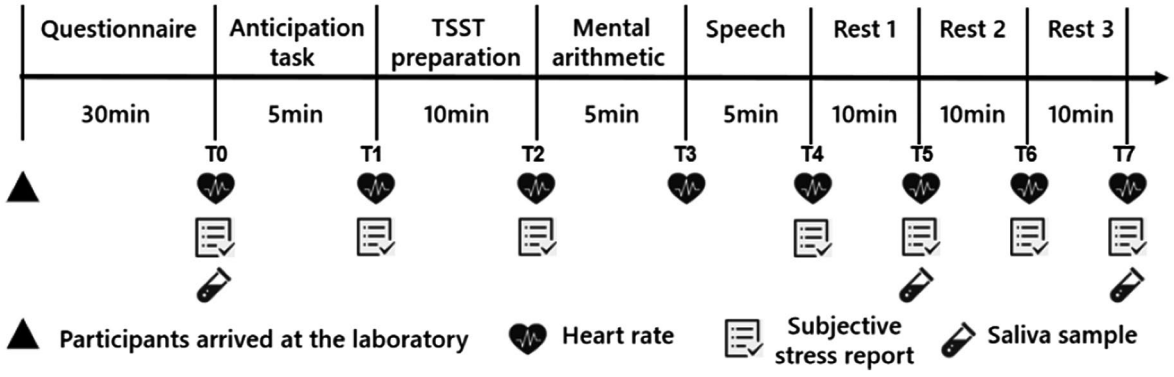

FIGURE 1 Experimental procedure. T, time point; TSST, Trier Social Stress Test

3 | RESULTS

3.1 | Manipulation check

Participants' reward anticipation and mood levels are listed in Table 2. Independent-sample *t*-tests showed that participants in the reward anticipation group reported a higher level of reward anticipation than those in the non-reward anticipation group ($M_{RA} = 4.62$, $SD = .89$; $M_{NA} = 1.28$, $SD = .46$; $t(1, 64) = 19.01$, $p < .001$, 95% CI: 2.97, 3.68). Participants in the reward anticipation group further reported higher level of positive feeling than those in the non-reward anticipation group after the reward anticipation task ($M_{RA} = 26.50$, $SD = 5.62$; $M_{NA} = 23.69$, $SD = 5.31$; $t(1, 64) = 2.088$, $p = .041$, 95% CI: −5.503, −0.122). However, no significant difference was indicated between the two groups regarding positive feelings before the reward anticipation task ($M_{RA} = 24.74$, $SD = 5.52$; $M_{NA} = 25.06$, $SD = 5.32$; $t(1, 64) = 0.245$, $p = .807$, 95% CI: −2.341, 2.996). Furthermore, no significant difference was observed in negative feelings before ($M_{RA} = 16.63$, $SD = 4.70$; $M_{NA} = 15.91$, $SD = 3.49$; $t(1, 64) = 0.703$, $p = .485$, 95% CI: −1.314, 2.740) and after the reward anticipation task ($M_{RA} = 14.41$, $SD = 3.28$; $M_{NA} = 15.03$, $SD = 4.02$, $t(1, 64) = 0.688$, $p = .494$, 95% CI: −1.179, 2.418).

3.2 | The moderation effect of ELS

Participants' cortisol concentrations during the experiment are illustrated in Figure 2a. The results of moderating effect analysis indicated that ELS negatively predicted AUC_I cortisol levels ($\beta = -.240$, $p = .044$, 95% CI: −0.008, 0.000). The interaction of ELS × RA on AUC_I cortisol levels was significant ($\beta = .259$, $p = .032$, $\Delta R^2 = 0.064$, 95% CI: 0.001, 0.017) (see Table 3, $Model_{cortisol}$). Follow-up tests revealed that AUC_I cortisol levels in the reward anticipation group were significantly lower than those in the non-reward anticipation group among participants with low ELS ($p = .002$), but not among those with high ELS ($p = .103$) (Figure 2b).

Participants' heart rate changes during the experiment are illustrated in Figure 3a. Results indicated that AUC_I heart rates in the reward anticipation group were significantly lower than those in the non-reward anticipation group ($\beta = -.455$, $p < .001$, 95% CI: −55.943, −21.611). The interaction of ELS × RA on AUC_I heart rates was significant ($\beta = .408$, $p < .001$, $\Delta R^2 = 0.159$, 95% CI: 1.315, 3.974) (see Table 3, $Mode_{heart\ rate}$). Follow-up tests revealed that AUC_I heart rates in the reward anticipation group were significantly lower than those in the non-reward anticipation group among participants with low ELS ($p = .025$), but not among those with high ELS ($p = .306$) (see Figure 3b).

Further analyses revealed that the lnRMSSD in the reward anticipation group was significantly higher than that in the non-reward anticipation group ($\beta = .274$, $p = .022$, 95% CI: 0.028, 0.343). Moreover, the interaction of ELS × RA on lnRMSSD was significant ($\beta = -.295$, $p = .015$, $\Delta R^2 = 0.083$, 95% CI: −0.027, −0.003) (see Table 3, $Model_{lnRMSSD}$). Follow-up tests revealed that lnRMSSD in the reward anticipation group was significantly higher than that in the non-reward anticipation group among participants with low ELS ($p = .036$), but not among participants with high ELS ($p = .288$), as shown in Figure 3c.

Participants' subjective stress reports during the experiment are illustrated in Figure 4. Results showed that the AUC_I subjective stress levels in the reward anticipation group were significantly lower than those in the non-reward anticipation group ($\beta = -.293$, $p = .020$, 95% CI: −6.525, −0.591). However, the interaction of ELS × RA on AUC_I subjective stress levels was nonsignificant ($\beta = .124$, $p = .323$, $\Delta R^2 = 0.015$, 95% CI: −0.115, 0.344), as shown in Table 3, $Model_{Subjective\ Stress}$.

TABLE 2 Descriptive data of emotions before and after reward anticipation task and the ratings of self-reported reward anticipation

	Reward anticipation	Non-reward anticipation	*p*
Reward anticipation, mean (*SD*)	4.62 (0.89)	1.28 (0.46)	.000***
Positive emotion			
Before, mean (*SD*)	24.74 (5.52)	25.06 (5.32)	.807
After, mean (*SD*)	26.50 (5.62)	23.69 (5.31)	.041*
Negative emotion			
Before, mean (*SD*)	16.63 (4.70)	15.91 (3.49)	.485
After, mean (*SD*)	14.41 (3.28)	15.03 (4.02)	.554

Abbreviation: *SD*, standard deviation.

*$p < .05$; ***$p < .001$.

4 | DISCUSSION

The present study was designed to explore the individual differences of the buffering effect of reward anticipation on acute stress responses. Consistent with the findings of previous research, including our existing experiment (Hu & Yang, 2021; Nicolson et al., 2020), reward anticipation was found to relieve the subjective feeling of stress and the increased heart rate induced by psychosocial stress. Furthermore, this study found that reward anticipation increased HRV throughout the experimental process. Importantly, the buffering effect was

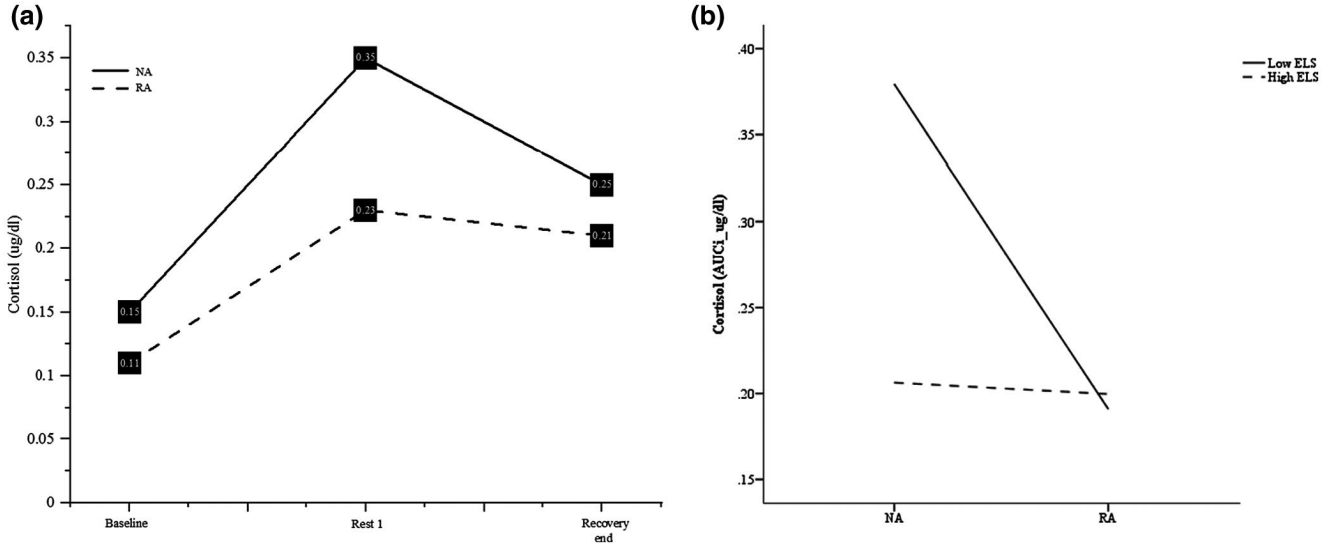

FIGURE 2 (a) Cortisol concentrations at all time points in the reward anticipation and control groups during TSST, and (b) the simple slope analysis of ELS moderating reward anticipation and AUC_I cortisol levels. AUC_I, area under the curve with respect to increase; NA, non-reward anticipation group; RA, reward anticipation group; TSST, Trier Social Stress Test

TABLE 3 Linear regression models predicting stress responses

	B	*SE*	*β*	*t*	*p*	ΔR^2
$Model_{cortisol}$						
Gender	0.027	0.054	0.059	0.502	.617	
RA	−0.081	0.052	−0.181	−1.561	.124	
ELS	−0.004	0.002	−0.240	−2.052	.044*	
RA × ELS	0.009	0.004	0.259	2.201	.032*	0.064
$Model_{heart\ rate}$						
Gender	2.831	8.97	0.032	0.316	.753	
RA	−38.777	8.585	−0.455	−4.517	.000***	
ELS	−0.190	0.33	−0.058	−0.574	.568	
RA × ELS	2.644	0.665	0.408	3.978	.000***	0.159
$Model_{heart\ rate\ variability}$						
Gender	−0.053	0.082	−0.075	−0.64	.525	
RA	0.186	0.079	0.274	2.357	.022*	
ELS	−0.004	0.003	−0.139	−1.181	.242	
RA × ELS	−0.015	0.006	−0.295	−2.491	.015*	0.083
$Model_{subjective\ stress}$						
Gender	−0.447	1.551	−0.035	−0.288	.774	
RA	−3.558	1.484	−0.293	−2.398	.020*	
ELS	0.007	0.057	0.016	0.127	.899	
RA × ELS	0.115	0.115	0.124	0.997	.323	0.015

Abbreviations: ELS, early life stress; RA, reward anticipation.

*$p < .05$;; ***$p < .001$.

shown to be more prevalent in participants with low ELS than those with high ELS. Specifically, reward anticipation mitigated the enhanced cortisol secretion and increased heart rate, and promoted the HRV in participants with low ELS. However, this effect was not observed in participants with high ELS. Taken together, the findings reveal that the anticipation of a reward may be a powerful means of fostering resilience under stress. Thus, this study highlighted the moderating effect of ELS on the buffering effect of reward anticipation in stressful circumstances.

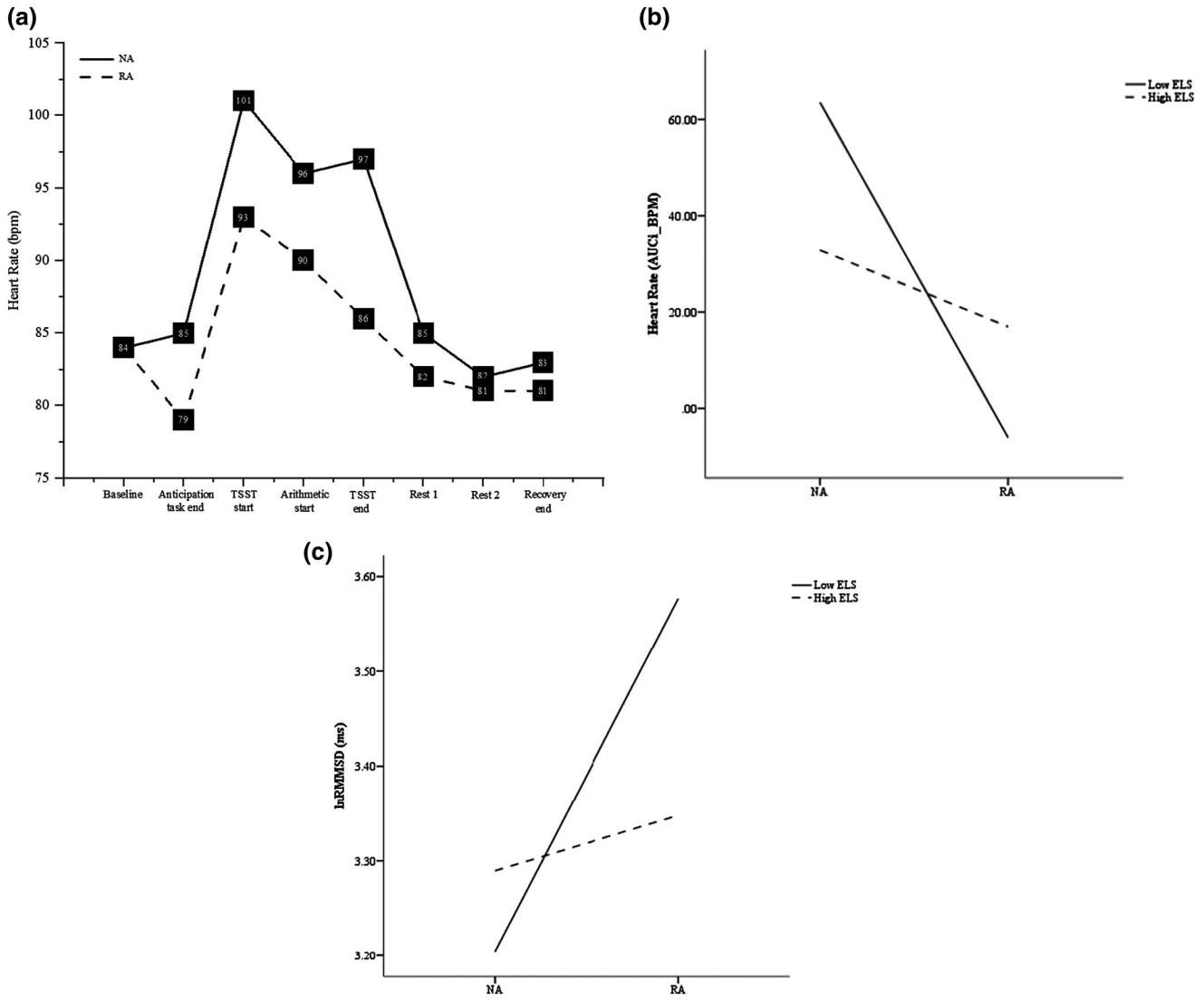

FIGURE 3 (a) Heart rates at all time points in the reward anticipation and control groups during TSST, (b) the simple slope analysis of ELS moderating reward anticipation and AUC_I heart rates, and (c) the simple slope analysis of ELS moderating reward anticipation and HRV. AUC_I, area under the curve with respect to increase; BPM, beats per minute; NA, non-reward anticipation group; RA, reward anticipation group; TSST, Trier Social Stress Test

The finding that reward anticipation alleviated subjective feelings of stress and reduced the heart rate under acute stress may be due to several psychological mechanisms. First, anticipation of a reward may be accompanied by positive moods which have widely been reported to dampen stress responses and restore stress-induced deficits (Dutcher & Creswell, 2018; Heller et al., 2009; Kringelbach & Berridge, 2009). Consistent with previous studies, our results indicated that participants in the reward anticipation group showed significant increases in positive moods. Positive emotions can enhance psychological resources and offset the potentially damaging psychological consequences of social evaluative threat, which are crucial factors behind the psychosocial stress response (Dickerson & Kemeny, 2004). Furthermore, previous studies have found that focusing on a positive future promoted individuals' positive reappraisals of stressors and changed their negative thought patterns, thus reducing the physiological stress response (Folkman & Lazarus, 1984; Fontaine et al., 1993; Gaab et al., 2003; Nicolson et al., 2020). In addition, a recent review highlighted the importance of affective and motivational properties in reward-related processes for fostering an enhanced perception of control (Ly et al., 2019). As an appraised characteristic of a stressful context, controllability can also facilitate active coping behavior, which can relieve excessive psychological and physical stress responses (Folkman et al., 1986; Sinha et al., 2016). Additionally, Tu et al. (2020) reported that reward-related expectations can shape one's perception of pain. Thus, we theorize that the changes in emotion, cognition, and coping behavior induced by reward anticipation may be the underlying psychological mechanisms of the buffering effect.

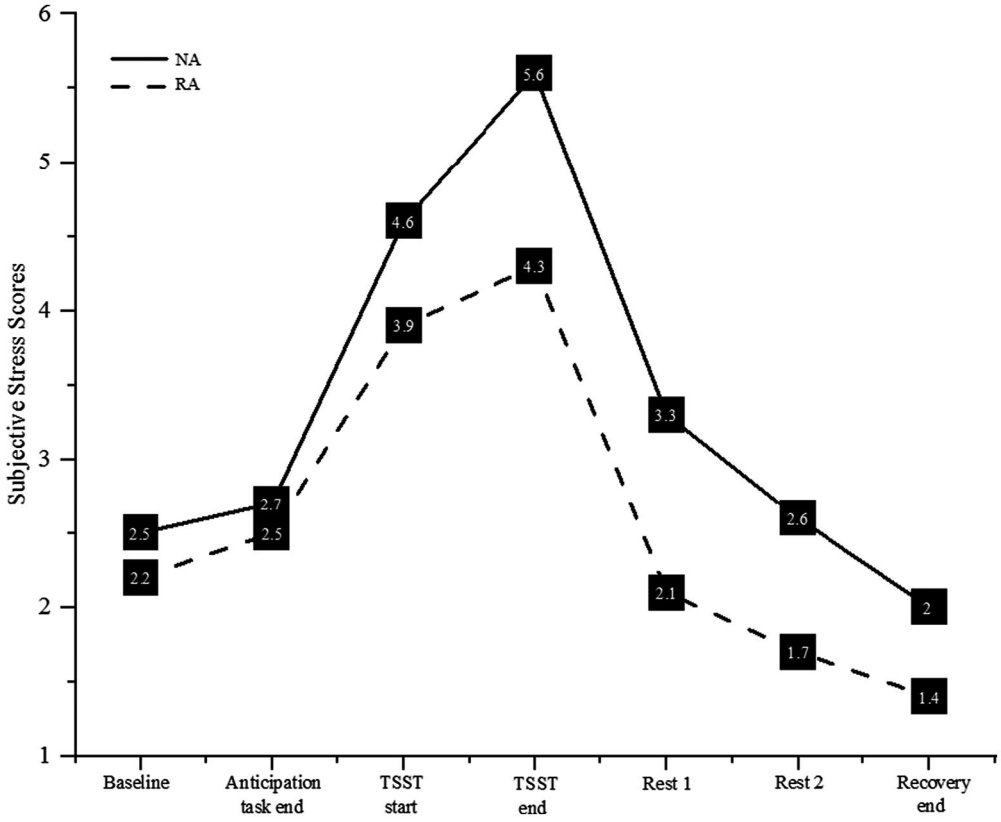

FIGURE 4 Subjective stress ratings at all time points in the reward anticipation and control groups during TSST. RA, reward anticipation group; NA, non-reward anticipation group; TSST, Trier Social Stress Test

Furthermore, the enhancement of positive emotions and sense of control caused by reward anticipation may occur via neuroplasticity mechanisms within the corticostriatal pathways and dopaminergic transmission (Fu & Depue, 2019; Ly et al., 2019). Previous studies demonstrated that during cognitive processing of reward anticipation, the ventral tegmental area dopamine–nucleus accumbens pathway is a primary neural circuit for incentive motivation and its accompanying subjective state of reward and positive affect (Bromberg-Martin et al., 2010; Haber & Knutson, 2010; Sesack & Grace, 2010). Dopamine is a well-established reward system neurotransmitter, and rewarding stimuli lead to dopamine release (Haber & Knutson, 2010). Animal studies have indicated that blocking dopamine significantly exaggerated the stress-induced increases in plasma adrenocorticotrophic hormone and corticosterone (Sullivan & Dufresne, 2006), suggesting a role for dopamine in stress regulation. Moreover, a review of the brain reward pathways in stress resilience inferred that the activation of reward-related brain regions such as the nucleus accumbens may be a pathway for lowered cortisol reactivity to a stressor (Dutcher & Creswell, 2018). Therefore, the dopamine transmission and activities of reward-related brain regions may be the neuroendocrine basis for the stress-relieving effect of reward anticipation.

In assessing cardiac responses to psychosocial stressors, this study investigated both the sympathetic (i.e., heart rate) activities and parasympathetic (i.e., HRV) measures. We further observed the anticipated increase of HRV in the reward anticipation group. HRV is the result of increased parasympathetic activity. During times of perceived stress, the sympathetic nerves produce a "stress response" by increasing adrenaline and reducing vagal tone, while the parasympathetic nerves attempt to "regulate" arousal (Sharpley, 2002). In this study, the lnRMSSD was used as the index of HRV. Higher lnRMSSD is associated with stronger top-down self-regulation and is related to a higher stress adaptability (Holzman & Bridgett, 2017; Lehrer & Gevirtz, 2014). Stress often reduces lnRMSSD, which is the manifestation of homeostasis and emotional disorder (Ottaviani et al., 2016; Taelman et al., 2009). Contrarily, receiving rewards can increase the lnRMSSD in both humans and animals, as shown in previous studies (Landolt et al., 2017; Zebunke et al., 2011). Additionally, aggregated measures of momentary positive affect were accompanied by elevated ambulatory lnRMSSD (Schwerdtfeger & Gerteis, 2014; Steptoe et al., 2007). Consistent with previous studies, our results showed significant differences in positive emotions and stress responses between the reward anticipation and control groups. This finding suggests that reward anticipation may enhance participants' lnRMSSD values by evoking positive emotions and reducing the negative effects of stress. Moreover, the opposite effect of reward anticipation on heart rate and HRV may

indicate that reward anticipation improved the ability of the parasympathetic nervous system to regulate the excessive cardiovascular response caused by persistent anxiety and tension during stressful situation. And the dissimilar effects of reward anticipation on these stress indicators reflect the functional differences and collaborations among stress systems.

Furthermore, our findings showed that individual variation in ELS moderated the buffering effect of reward anticipation on stress responses. Limited research on human participants has shown that individuals with ELS were widely found to have deficits regarding the processing of reward anticipation (Novick et al., 2018). Additionally, the decreased reactivity to reward anticipation has a conceptual basis in learning and attachment, which can be powerfully shaped by environmental experiences in the developmental pathway. Individuals learn about reward anticipation from the parent-child relationship in early life. Thus, problematic parental relationships may impair the ability to process various reward signals, making it difficult for such individuals to anticipate and pursue future rewards (Guyer et al., 2006; Pechtel & Pizzagalli, 2013; Pollak, 2015). For example, abused children raised in environments characterized by danger and negative social feedback may become hypervigilant to threat cues, thus neglecting reward cues. Additionally, neglected children lack social interactions with primary caregivers. This damages their understanding of social rules, such as the association between approach behavior and reward outcomes. On the other hand, people with high ELS are exposed to inconsistent or poorly conveyed emotional and reward signals in caregiver behavior. Thus, they are unlikely to believe that they would receive rewards, thereby decreasing their reactivity to positive cues and their anticipation of rewarding events (Bugental et al., 1990). This occurs especially when the pending rewards are uncertain, both in experimental paradigms and real-world contexts (Nelson et al., 2014). Thus, this study highlights the importance of cultivating the ability to anticipate and pursue future rewards at an early age for stress adaptability. It further suggests that the establishment of positive attachment relationships and the reduction of the effects of childhood trauma are feasible means for achieving stress adaptability. Future research should explore how additional changes in social behavior and the developmental neuroscience of individuals with high levels of ELS influence the moderating effect of reward systems on stress responses.

Some studies also reported increased neurological and behavioral reactivity during the anticipation of reward in individuals with ELS both in human studies and animal models (e.g., Casement et al., 2014; Cuenya et al., 2015). For example, for boys who suffered from depression, low maternal warmth in early childhood was associated with increased neural activity when anticipating a monetary reward (Morgan et al., 2014). A similar association was found for adolescent females with low maternal warmth, and increased activity was associated with increased depressive symptoms (Casement et al., 2014). These studies suggest that depression may be an important factor leading to higher neural activities for individuals with low maternal warmth. Because participants recruited in the current study are college students without depression or other mental disorders, and they mainly experienced emotional abuse and neglect, we infer that the participants in the present study, like those in many previous studies, have decreased sensitivity during the anticipation of reward (Novick et al., 2018). Future studies can investigate how ELS modulates the stress-relieving effect of reward anticipation in more clinical samples.

Notably, we found no significant moderation effect of ELS on subjective reported stress. Indeed, previous studies have evidenced a complete dissociation between ratings of stressfulness and physiological stress reactivity. For example, a Dutch famine birth cohort study showed that participants with depression or anxiety had slower cardiovascular and cortisol responses to a series of stress tasks. However, they subjectively reported experiencing greater levels of pressure during the tasks (Bibbey et al., 2013; Carroll et al., 2017). Furthermore, subjective stress ratings reflected interoceptive and subjective awareness of stress signals, which is a conscious step toward regaining perceived and behavioral control over stress (Gratz & Roemer, 2004; Gross & John, 2003; Sinha et al., 2016). Therefore, the increase in subjective stress may improve stress regulation. This may be especially true for individuals who experienced childhood trauma, since they have trouble detecting possible threats, and have difficulty monitoring their reactions (Dodge et al., 1995; Luke & Banerjee, 2013). Future research should consider using the state-trait anxiety inventory (STAI, Spielberger et al., 1970) to better elucidate participants' subjective stress levels (Hu et al., 2018).

In relation to our study, individuals with ELS appear to show different reactivity patterns during distinct reward processes, that is, reward anticipation versus reward consumption (Dennison et al., 2016). Boecker and colleagues reported that people who experienced socio-economic adversity in early life showed reduced neural responses in the ventral striatum during anticipation of reward. However, they reported heightened reactivity during reward delivery in the putamen, right pallidum, and insula. This pattern was particularly observed with reactivity to the positive verbal feedback (Boecker et al., 2014, Boecker-Schlier et al., 2016). Thus, the processing stage and type of reward may affect the moderating effects of ELS, and individuals with high ELS may experience better stress recovery because of the greater pleasure brought by reward attainment. Therefore, the sensitivity and moderating effects of reward anticipation may be an essential factor for individuals with high ELS to withstand stress and adversity, especially given the deficiency of reward consumption in early development. Consistent with this perspective, DelDonno and colleagues (2019) suggested that the

increased network activation during reward anticipation in individuals who experienced childhood trauma may signify resilience. Additionally, lower neural responses to reward anticipation predicted higher stress reactivity in children with high ELS (Vidal-Ribas et al., 2019). Our findings thus elucidate the role of reward in stress resilience in the context of ELS. Previous studies reported that higher reward reactivity was a crucial moderating factor for fostering resilience to the adverse consequences of stress (Dennison et al., 2016; Telzer et al., 2014). The current study approached this issue from a development perspective. Our findings suggested that the impaired buffering effect of reward anticipation on stress responses in individuals with high ELS may compound the adverse effects of frequent stressors on their physical and mental health. This may create a vicious cycle regarding the reward system dysfunction and stress susceptibility (Hanson et al., 2015; Vidal-Ribas et al., 2019).

Remarkably, the current study also showed that ELS was negatively correlated with cortisol secretion under acute stress. This suggests that ELS may lead to a reduced response of the HPA axis to psychosocial stress. According to the stress inoculation hypothesis, exposure to moderate and brief intermittent stress in early life may lead to decreased cortisol reactivity and the development of subsequent stress resistance. However, severe stress increases HPA-axis reactivity as a further prototypical responsivity pattern, such as that among individuals with a history of severe physical or sexual abuse and major depressive disorder or post-traumatic stress disorder (Del Giudice et al., 2011; Heim et al., 2008; Parker et al., 2006; Rao et al., 2010). Our sample included healthy young adults with no history of psychiatric disorders, who had experienced moderate emotional abuse and neglect. Thus, the hypocortisolism of the HPA system under acute stress may reflect their enhanced ability to adapt to stress, serving as a marker for biological resilience (Grimm et al., 2014). In addition, severe ELS may also develop into a blunted stress response pattern that signifies central motivational and emotional dysregulation or serves as a counter-regulatory adaptation to continuous stress exposure during development (Cărnuţă et al., 2015; Carroll et al., 2017; Fries et al., 2005; Miller et al., 2007; Pryce et al., 2005). In sum, it remains controversial whether passive stress responses in individuals with an ELS history are a manifestation of resilience or vulnerability; it could depend on the psychiatric diagnoses of participants and the characteristics of early stress exposure, including their severity, nature, and timing (Fogelman & Canli, 2018; Mello et al., 2009; Tyrka et al., 2013). It should be mentioned that the blunted cortisol response caused by ELS may partly explain the attenuated cortisol-relieving effects of reward anticipation in the present study, which maybe due to the "floor effect". This reminds us that the function of processing reward anticipation needs to be measured to clarify its role in this moderation effect.

This study has three main limitations. The present study did not balance data across the genders. Previous studies indicated that low maternal warmth may increase the brain's sensitivity to the anticipation of reward, depending on gender (Casement et al., 2014). Furthermore, ELS alters opioid receptor mRNA levels, as well as dopamine D1 receptor expression within the nucleus accumbens, in a gender-dependent manner (Chang et al., 2019; Fuentes et al., 2018). Thus, gender is an important factor in researching the long-term impact of early life experiences on the reward system. Additionally, emerging evidence from preclinical and human research suggests that differences in gender exist in response to both chronic and acute stress exposure (Rincón-Cortés et al., 2019). Thus, future studies should balance the number of male and female participants to control for the potential impact of gender. In this study, when we included gender as a covariate for moderating effect analysis, the present findings remained.

Secondly, the participants recruited in this study were healthy and with moderate ELS, so the current results cannot be extended to people with mental illness or those with severe ELS, who may show enhanced reward anticipation reactivity and different stress response patterns (Grimm et al., 2014; Novick et al., 2018). Therefore, the conclusions of the present study may apply to the healthy population with moderate ELS only. In addition, the childhood trauma that was investigated in the present study is only one component of ELS. Indeed, individuals with other early experiences of chronic stressors, such as peer bullying and medical problems, were also found to be dysfunctional in reward anticipation processing (Casement et al., 2014; Hanson et al., 2016), and the potential moderating effect of additional types of ELS needs to be explored in the future.

Thirdly, psychological variables related to stress in this study have largely been overlooked in previous literature. This prevented us from further elucidating the psychological mechanisms underlying the current findings. For example, social evaluative threat and uncontrollability are the core induced factors of psychosocial stress responses (Dickerson & Kemeny, 2004). In the reward anticipation condition, the social evaluative threat may be reduced by positive cognitive reappraisal (Folkman & Moskowitz, 2000). Additionally, controllability may be increased by active engagement (Zahn et al., 2016). Both effects are related to stress resilience, and may be moderated by ELS. Future research should address these limitations by investigating the psychological pathways of the moderating effect of ELS.

5 | CONCLUSION

In conclusion, this study indicated that reward anticipation mitigates the cortisol and cardiovascular responses to acute psychosocial stress in healthy adults. It also improved

participants' HRV. Moreover, the buffering effect was moderated by ELS experience. Together, the present findings elucidate the effect of ELS on the role of reward anticipation in stress resilience as a potential inter-individual variability.

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CONFLICT OF INTEREST

This manuscript has not been published or presented elsewhere in part or in entirety and is not under consideration by another journal. All the authors approved the manuscript, agree with its submission to your esteemed journal, and contributed significantly to its creation. All study participants provided informed consent, and the study design was approved by the appropriate ethics review board. We have read and understood your journal's policies, and we believe that neither the manuscript nor the study violates any of these. There are no conflicts of interest to declare.

AUTHOR CONTRIBUTIONS

Weiyu Hu: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Resources; Software; Writing-original draft; Writing-review & editing. **Yadong Liu:** Investigation. **Jiwen Li:** Investigation. **Xiaolin Zhao:** Investigation. **Juan Yang:** Conceptualization; Funding acquisition; Methodology; Project administration; Supervision; Validation; Visualization; Writing-review & editing.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

Supplementary Material

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Impact Statement

The contents of this page will be shown on the eTOC on the online version only. It will not be published as part of the article PDF.

Our findings add a new perspective on dysfunction of reward anticipation processing as an underlying mechanism relating early life stress (ELS) to stress-related negative consequences. The present study found that not only reward anticipation successfully decreased acute stress responses in general which was consistent with previous studies but also improved participants' HRV. Furthermore, this effect was moderated by ELS.