Trait Anxiety Predicts Acute Psychological Stress Response: Postprint

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Date: 2018-09-07T00:00:00+00:00

Abstract

Acute stress response enables individuals to rapidly mobilize psychophysiological resources in emergency situations, thereby better coping with current challenges. This study employed the Trier Social Stress Test (TSST), using heart rate and salivary cortisol as stress response indicators, and investigated the predictive effect of trait anxiety on acute psychological stress response while controlling for three demographic variables: gender, age, and years of education. The results revealed: (1) TSST successfully induced stress responses in participants, manifested as increased heart rate and elevated salivary cortisol levels; (2) hierarchical regression results demonstrated that trait anxiety predicted heart rate changes under acute stress, while its predictive effect on salivary cortisol changes did not reach statistical significance. These findings indicate that trait anxiety is closely associated with the rapid-response indicator of acute stress response—the sympathetic nervous system; higher trait anxiety levels correspond to smaller sympathetic nervous system responses under acute stress. Individuals with higher trait anxiety levels may experience restricted acute stress responses due to excessive consumption of cardiomyocytes resulting from chronic anxiety.

Full Text

Trait Anxiety Predicts Acute Psychological Stress Response

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Abstract

Acute stress responses enable individuals to rapidly mobilize psychophysiological resources during emergencies, thereby better confronting immediate challenges. Using the Trier Social Stress Test (TSST) and measuring stress responses through heart rate and salivary cortisol, this study examined the predictive effect of trait anxiety on acute psychological stress responses while controlling for three demographic variables: gender, age, and years of education. The results revealed: (1) TSST successfully induced stress responses, manifested as accelerated heart rate and elevated salivary cortisol; (2) Hierarchical regression analysis showed that trait anxiety predicted heart rate changes during acute stress, while its predictive effect on salivary cortisol changes did not reach significance. These findings indicate that trait anxiety is closely associated with the sympathetic nervous system—a rapid-response indicator of acute stress reactions. Higher trait anxiety levels correspond to smaller sympathetic nervous system responses during acute stress. Individuals with elevated trait anxiety may experience impaired acute stress responses due to chronic anxiety-induced excessive consumption of myocardial cells.

Keywords: trait anxiety; acute psychological stress; heart rate; salivary cortisol; prediction

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Introduction

Stress is an integral part of human life. Daily experiences such as losing a loved one, natural disasters, college entrance examinations, graduate school tests, and job interviews present various threats and challenges. Acute stressors trigger a cascade of physiological stress responses, with the sympathetic-adrenal medulla (SAM) system and the hypothalamus-pituitary-adrenal (HPA) axis serving as the two primary physiological indicators. During acute stress, the SAM system activates rapidly, causing increased heart rate (Allen, Kennedy, Cryan, Dinan, & Clarke, 2014). Research demonstrates that heart rate is the most convenient and effective measure for assessing rapid physiological stress reactions, widely applied in stress research (Porges, 1995). HPA axis activation proceeds relatively slowly, with enhanced activity leading to increased cortisol secretion (Sandi & Haller, 2015). Acute stress responses enable rapid threat detection and mobilization of psychophysiological resources to meet challenges (de Kloet, Joëls, & Holsboer, 2005). However, excessive and persistent stress may lead to adverse consequences, including psychiatric disorders such as post-traumatic stress disorder, depression, anxiety disorders, and schizophrenia, as well as somatic diseases like cardiovascular and cerebrovascular conditions (Sapolsky, 2015; Ventriglio, Gentile, Baldessarini, & Bellomo, 2015).

Individuals exhibit substantial variability in stress response patterns and intensity when facing identical stressors (Kudielka & Wüst, 2010). On one hand, laboratory stressors such as TSST have been employed to examine relationships

between demographic factors (e.g., gender, age, education) and psychological factors (e.g., personality and cognition) with stress responses in healthy individuals (Bale & Epperson, 2015; Fiocco, Joober, & Lupien, 2007; Novais et al., 2016; Wu et al., 2017). On the other hand, studies using naturally occurring traumatic events have investigated how biological factors (e.g., amygdala activity and genetics) and psychological factors collected before trauma relate to post-traumatic stress disorder symptoms or psychological stress responses (Admon et al., 2009; Beevers, Lee, Wells, Ellis, & Telch, 2011; Bomyea, Risbrough, & Lang, 2012; Cobb, Lancaster, Meyer, Lee, & Telch, 2017).

Some research has specifically examined the relationship between trait anxiety and acute stress responses. Trait anxiety represents a relatively stable, individual-difference tendency toward anxiety (Spielberger, Gorsuch, & Lusthene, 1970). Previous studies have primarily focused on acute psychological stress responses in anxiety disorder patients (e.g., generalized anxiety disorder, social phobia, panic disorder), yielding inconsistent findings. Some research indicates that anxiety disorder patients show elevated acute psychological stress responses compared to controls (Gola et al., 2012) or no differences (Klumbies, Braeuer, Hoyer, & Kirschbaum, 2013), while others report opposite conclusions (Wichmann, Kirschbaum, Lorenz, & Petrowski, 2017). Among healthy populations, most studies find that high trait anxiety individuals show lower acute psychological stress responses than their low trait anxiety counterparts. For example, Jezova, Makatsori, Duncko, Moncek, and Jakubek (2004) used the public speaking component of TSST with healthy male participants, finding that high trait anxiety groups exhibited lower SAM system responses (epinephrine and norepinephrine) and lower HPA axis responses (adrenocorticotropic hormone, plasma cortisol, and salivary cortisol) compared to low trait anxiety groups. Villada, Hidalgo, Almela, and Salvador (2014) similarly employed TSST as an acute stressor and found lower salivary cortisol responses in high trait anxiety groups. However, Wirtz et al. (2007) found no correlation between trait anxiety levels and salivary cortisol stress responses in middle-aged men.

Most existing research on healthy populations has analyzed trait anxiety as a categorical variable by grouping participants based on questionnaire scores (Jezova et al., 2004; Villada et al., 2014). Haslam, Holland, and Kuppens (2012) reviewed nearly 20 years of taxometric research, concluding that most normal personality traits and psychiatric disorders represent continuous rather than discrete categorical variables, with no distinct taxa separating normal personality from personality disorders. For instance, social anxiety disorder (Boyers et al., 2017), generalized anxiety disorder (Kertz, Mchugh, Lee, & Björgvinsson, 2014), and anxiety sensitivity (Asmundson, Weeks, Carleton, Thibodeau, & Fetzner, 2011) all exhibit continuous structures. Analyzing trait anxiety across its full continuum provides more information, yielding greater statistical power and less parameter estimation bias (Steudte-Schmiedgen et al., 2015).

Different studies employ various stress response indicators that reflect different aspects of stress reactivity (Kloet et al., 2005). Acute stressors elicit multi-

ple physiological responses, including the rapid-response SAM system and the slower-response HPA axis. Research indicates that both systems play crucial roles in mobilizing resources to address threats and restore homeostasis (Kloet et al., 2005). However, most studies measure only one physiological indicator. For example, Villada et al. (2014) and Wirtz et al. (2007) measured only HPA axis changes in salivary cortisol to reflect acute psychological stress response levels. This study simultaneously employs a rapid-response indicator—sympathetic nervous system heart rate changes—and a slow-response indicator—HPA axis salivary cortisol changes—to reflect acute psychological stress responses.

This study treats trait anxiety scores from all healthy participants as continuous variables and examines trait anxiety' s predictive effect on acute psychological stress responses using both rapid-response (sympathetic nervous system heart rate changes) and slow-response (HPA axis salivary cortisol changes) indicators. Previous research demonstrates significant correlations between gender (Bale & Epperson, 2015; Novais, Monteiro, Roque, Correia-Neves, & Sousa, 2016), age (Novais et al., 2016; Gunnar, Talge, & Herrera, 2009), and education years (Fiocco, Joober, & Lupien, 2007) with acute psychological stress responses. For instance, males show higher salivary cortisol responses than females during acute stress; salivary cortisol responses increase with age; and education years correlate negatively with salivary cortisol responses. However, Jezova et al. (2004) used only male participants and controlled for age but not education years. This study investigates trait anxiety' s predictive effect on acute psychological stress responses while controlling for these three demographic variables. Based on previous findings (Jezova et al., 2004; Villada et al., 2014), we hypothesized that trait anxiety levels would negatively predict acute psychological stress responses —higher trait anxiety would correspond to lower acute stress responses.

Method

Participants

Participants were recruited from universities in Beijing through posted advertisements. Recruitment criteria included: (1) university students aged 18–28 years; (2) good physical health. Telephone interviews screened potential participants based on exclusion criteria: (1) history of psychiatric or neurological disorders; (2) history of endocrine disorders (e.g., Cushing' s syndrome); (3) history of other major chronic physical illnesses such as diabetes, heart disease, meningitis, severe kidney disease, malignant tumors, epilepsy, and migraine; (4) history of brain injury (e.g., brain surgery, cerebral hemorrhage, severe brain trauma); (5) long-term use of antipsychotic, neurological, or adrenocortical hormone medications; (6) current pregnancy; (7) long-term reversed day-night schedules; (8) major surgery within the past 6 months; (9) alcohol abuse (more than 2 drinks daily) or excessive smoking (more than 5 cigarettes daily). The final sample comprised 54 participants (35 males, 19 females) aged 18–25 years (mean age = 22.57 ± 1.67) with 13–18 years of education (mean = 15.89 ± 1.34). All participants provided informed consent and received compensation upon completion.

Procedure

All experiments were conducted between 1:30 PM and 5:00 PM, a period when salivary hormones (e.g., cortisol) are at relatively low and stable diurnal levels (Nelson, 2005). Participants were contacted by telephone the day before the experiment and instructed to avoid strenuous exercise on the experimental day. All participants reported compliance upon arrival. After arriving at the laboratory, participants rested on a sofa for 30 minutes, during which they completed demographic questionnaires (age, gender, education years) and the Trait Anxiety Inventory (T-AI). Following the rest period, saliva samples were collected and heart rate was recorded (baseline time point). Participants then completed the TSST, which consisted of 5-minute preparation, speech, and mental arithmetic phases, with continuous heart rate recording during each phase. Immediately after TSST completion, saliva samples were collected and heart rate was recorded continuously for 5 minutes (time point 1). Saliva collection and heart rate recording were repeated at 35 minutes (time point 2), 60 minutes (time point 3), and 75 minutes (time point 4) after TSST onset. The experimental procedure and data collection timeline are illustrated in Figure 1 [Figure 1: see original paper].

Measures

The State-Trait Anxiety Inventory (STAI), developed by Spielberger and Gorsuch (1983), was used. This study employed the Chinese version of the trait anxiety subscale (T-AI) (Wang, Wang, & Ma, 1999), which includes 20 items rated on a 4-point scale: 1 = almost never, 2 = sometimes, 3 = often, 4 = almost always. The T-AI total score reflects participants' general or typical anxiety levels.

Stress Induction

The TSST, developed by Kirschbaum and colleagues in 1993 (Kirschbaum, Pirke, & Hellhammer, 1993), is a standardized psychosocial stress test. Modified versions have proven equally or more effective than the original in eliciting stress responses (Buchanan, Bagley, Stansfield, & Preston, 2012). Therefore, this study used a modified TSST consisting of three 5-minute components: preparation, speech, and mental arithmetic. Participants received instructions for a speech task requiring them to imagine being accused of shoplifting while shopping and to defend themselves before a store manager—differing from the original TSST's job interview simulation. During the 5-minute preparation phase, participants could take notes in one room, though notes were confiscated before speaking. Participants were informed that their entire speech would be video-recorded for later analysis and that "managers" would take notes and evaluate their performance. After preparation, participants were escorted to another room, positioned appropriately, and required to deliver their speech while standing. Three experimenters (two female, one male) 扮演 ed managers, wearing white coats, maintaining neutral expressions, and providing no feedback. When par-

ticipants fell silent, experimenters prompted them with questions. Immediately following the speech, participants completed a mental arithmetic task requiring them to serially subtract 13 from 1022, vocalizing only the results while performing calculations silently. Incorrect responses required restarting from 1022.

Physiological Measures

Saliva Collection Saliva was collected using salivette tubes (Sarstedt, Rommelsdorf, Germany) and stored at -22°C. Before analysis, samples were thawed and centrifuged at 3000 rpm for 10 minutes. Salivary cortisol concentrations were determined using electrochemiluminescence immunoassay (Cobas e601, Roche Diagnostics, Numbrecht, Germany), with a sensitivity of 0.5 nmol/L (lower limit). Intra- and inter-assay coefficients of variation were both less than 10%.

Heart Rate Recording Heart rate was recorded using the Biopac Amplifier-System (MP150; Biopac, Goleta, CA, USA) with three electrocardiogram electrodes placed on the right side of the neck, left medial ankle, and right medial ankle. The signal sampling frequency was 1000 Hz. AcqKnowledge software averaged heartbeats during each 5-minute recording period at each time point, calculating beats per minute (bpm) as the heart rate index for that time point.

Data Analysis

Heart rate and salivary cortisol responses served as stress response indicators. To confirm TSST effectiveness, one-way repeated measures ANOVA tested differences across time points, followed by Bonferroni post-hoc comparisons. Heart rate peaked during the speech phase; therefore, the heart rate stress response index (Δheart rate) was calculated as the average heart rate during the TSST speech phase minus baseline heart rate. Salivary cortisol peaked at 35 minutes post-TSST onset (time point 2); thus, the salivary cortisol stress response index (Δsalivary cortisol) was calculated as salivary cortisol at 35 minutes post-TSST onset (time point 2) minus baseline salivary cortisol.

Pearson correlation analysis examined relationships between trait anxiety and acute stress responses. Hierarchical regression analysis then assessed trait anxiety's predictive effect on acute stress responses after controlling for demographic variables potentially influencing acute psychological stress responses. The procedure involved: Step 1, entering demographic variables (gender, age, education years) as the first layer; Step 2, entering trait anxiety as the second layer. The change in R^2 (ΔR^2) between layers determined whether trait anxiety predicted stress responses beyond the shared influence of gender, age, and education years. All variables were entered using forced entry method. Regression model dependent variables were stress responses (Δheart rate and Δsalivary cortisol). Data were processed using SPSS 19.0, with all reported p-values from two-tailed tests and significance set at 0.05.

Results

Stress Response

Means and standard deviations of TSST-elicited physiological responses (heart rate and salivary cortisol) are presented in Table 1 .

Heart Rate Stress Response Heart rate peaked during the speech phase. The mean Δheart rate was 16.3 bpm (SD = 12.1). One-way repeated measures ANOVA across time points revealed a significant main effect of time, $F(7, 371) = 25.12$, $p < 0.001$, $p^2 = 0.29$. Post-hoc comparisons showed that heart rate during both TSST speech and arithmetic phases was significantly higher than baseline, preparation, and all other time points (ps < 0.01). Heart rate did not differ significantly between speech and arithmetic phases ($p = 0.835$). Heart rates at 15 minutes (time point 1), 35 minutes (time point 2), 60 minutes (time point 3), and 75 minutes (time point 4) post-TSST did not differ significantly from baseline (ps > 0.05). Heart rate trends are illustrated in Figure 2 [Figure 2: see original paper].

Salivary Cortisol Stress Response Salivary cortisol peaked at 35 minutes post-TSST onset (time point 2). The mean Δsalivary cortisol was 5.92 nmol/L (SD = 5.85). One-way repeated measures ANOVA across time points showed a significant main effect of time, $F(4, 212) = 19.01$, $p < 0.001$, $p^2 = 0.22$. Post-hoc comparisons revealed that salivary cortisol at 35 minutes post-TSST onset (time point 2) was significantly higher than all other time points (ps < 0.01). Salivary cortisol levels at 15 minutes (time point 1), 35 minutes (time point 2), and 60 minutes (time point 3) post-TSST were significantly higher than baseline and 75 minutes (time point 4) post-TSST (ps < 0.05). Salivary cortisol trends are shown in Figure 3 [Figure 3: see original paper].

Correlation Analysis

Trait anxiety correlated negatively with acute heart rate stress response ($r = -0.32$, $p < 0.05$) but not significantly with acute salivary cortisol stress response ($r = -0.06$, $p = 0.68$). A scatterplot of the simple correlation between trait anxiety and heart rate stress response appears in Figure 4 [Figure 4: see original paper].

Regression Analysis

Detailed results for each stress response regression model are presented in Table 2 .

Heart Rate Stress Response Model The first-layer demographic variables model was marginally significant, $F(2, 51) = 2.40$, $p = 0.078$. Education years positively predicted heart rate stress response ($= 0.55$, $p < 0.05$), uniquely explaining 11.50% of heart rate stress response variance. The second-layer predictive model was significant, $F(2, 51) = 3.85$, $p < 0.05$. Adding trait anxiety

increased explained variance by 11%. Trait anxiety negatively predicted heart rate stress response (= -0.35, $p < 0.05$).

Salivary Cortisol Stress Response Model Both the first-layer model ($F(2, 51) = 0.35$, $p = 0.792$) and second-layer model ($F(2, 51) = 0.27$, $p = 0.898$) for salivary cortisol stress response were non-significant, with no variables showing significant predictive effects (ps > 0.05).

Discussion

This study investigated trait anxiety' s predictive effect on acute psychological stress responses in healthy participants using TSST as a stress induction method, with objective heart rate and salivary cortisol as acute stress response indicators. Trait anxiety was treated as a continuous rather than categorical variable while controlling for gender, age, and education years. Results demonstrated that TSST successfully induced significant stress responses, evidenced by accelerated heart rate and elevated salivary cortisol—findings consistent with previous TSST research (Kirschbaum et al., 1993; Villada et al., 2014). Yang, Hou, Yang, and Zhang (2011) demonstrated TSST' s effectiveness in eliciting acute stress responses in Chinese participants, establishing its cross-cultural applicability—conclusions further supported by the present results.

The most important finding was that higher trait anxiety levels predicted lower acute heart rate stress responses, while trait anxiety showed no significant predictive effect on salivary cortisol stress responses. These results indicate that trait anxiety negatively predicts heart rate responses during acute stress; individuals with higher trait anxiety exhibit smaller sympathetic nervous system responses to acute stressors. This conclusion aligns with Jezova et al. (2004), who used epinephrine and norepinephrine as SAM system indicators. Furthermore, analyzing trait anxiety across its full continuum provides greater statistical power and less parameter estimation bias (Steudte-Schmiedgen et al., 2015). However, most previous research has categorized participants based on trait anxiety scores (Jezova et al., 2004; Villada et al., 2014). The current study' s innovation lies in using the more easily collected heart rate metric and analyzing trait anxiety as a continuous variable. Importantly, while numerous studies demonstrate relationships between gender, age, education years, and acute stress responses (Bale & Epperson, 2015; Fiocco, Joober, & Lupien, 2007; Gunnar, Talge, & Herrera, 2009; Novais et al., 2016), previous trait anxiety research has not comprehensively controlled these demographic factors—unlike the present study.

Regarding mechanisms underlying the negative correlation between trait anxiety and heart rate stress responses, one possible explanation involves reduced -adrenergic receptor responsivity in high trait anxiety individuals. Research has documented diminished -adrenergic receptor responsivity in anxiety disorder patients such as those with panic disorder (Brown, Charney, Woods, Heninger, & Tallman, 1988) and in healthy populations experiencing chronic extreme stress

(Dimsdale, Mills, Patterson, Ziegler, & Dillon, 1994) or scoring high on tension-anxiety dimensions (Yu, Dimsdale, & Mills, 1999). The mechanism may involve chronic anxiety elevating plasma norepinephrine and other catecholamines (Axelrod & Reisine, 1984), causing sustained -adrenergic receptor activation that leads to excessive myocardial cell consumption and apoptosis (Bisognano et al., 2000), resulting in compensatory reduced -adrenergic receptor responsivity during acute stress (Young, Nesse, Weder, & Julius, 1998). -adrenergic receptors on myocardial cells produce positive inotropic effects when activated, increasing myocardial excitation, contractility, ejection velocity, and heart rate to manage emergencies and facilitate "fight or flight" responses (Grimm & Brown, 2010). Thus, reduced -adrenergic receptor responsivity in high trait anxiety individuals may impair rapid sympathetic resource mobilization during acute stress, hindering effective emergency responses.

An alternative explanation suggests that the relationship between trait anxiety and heart rate stress responses may share common genetic pathways. The serotonin transporter gene-linked polymorphic region (5HTTLPR) critically determines central/peripheral serotonin levels and effects, relating closely to emotional regulation and physiological responses (Phillips, Hunt, Der, & Carroll, 2011). Research indicates that individuals carrying the long allele of 5HTTLPR exhibit both higher trait anxiety levels and lower blood pressure and heart rate responses to laboratory stress tasks (Williams et al., 2008). Therefore, carrying the 5HTTLPR long allele may represent a mechanism linking higher trait anxiety with lower heart rate stress responses.

Although TSST successfully elicited salivary cortisol stress responses, this study found no predictive effect of trait anxiety on salivary cortisol stress responses. This result aligns with Wirtz et al. (2007), who also used TSST and treated trait anxiety as a continuous variable, but contradicts findings from Jezova et al. (2004) and Villada et al. (2014). The latter studies categorized participants based on trait anxiety scores, treating it as a categorical variable. Additionally, Jezova et al. (2004) used only male participants and controlled for age but not education years. In contrast, the present study treated trait anxiety as a continuous variable, included both healthy males and females, and controlled for gender, age, and education years. These demographic and methodological differences may account for divergent findings across laboratories, suggesting that the relationship between trait anxiety and slow stress responses (HPA axis activity) is not unidimensional and may involve multiple contributing factors. Furthermore, research demonstrates that different personality traits relate to distinct components of the stress response system (HPA axis vs. SAM system) (Puig-Perez et al., 2015). Chida and Hamer' s (2008) meta-analysis of 161 acute stress response studies in healthy populations found that negative psychological traits such as trait anxiety, hostility, and neuroticism correlated with sympathetic acute stress responses (heart rate) but not HPA axis responses (salivary cortisol). Conversely, positive psychological traits like optimism, self-enhancement, and well-being related to HPA axis responses but not sympathetic responses. Thus, as a negative psychological trait, trait anxiety predicts heart rate but not

salivary cortisol stress responses.

The divergent predictive effects of trait anxiety on heart rate versus salivary cortisol stress responses align with evidence that SAM system and HPA axis response mechanisms and functions are independent during stress (de Boer, De Beun, Slangen, & van Gugten, 1990). The SAM system constitutes a rapid stress response system, with adrenal medulla innervation by sympathetic nerves enabling immediate activation during acute stress (Rimmele et al., 2007). The HPA axis represents a slower stress response system; a meta-analysis of 173 TSST studies found cortisol peaks distributed broadly between 20–30 minutes post-TSST (i.e., 35–45 minutes after TSST onset) (Goodman, Janson, & Wolf, 2017). These two response mechanisms cooperate to address emergency challenges. The present findings further demonstrate that acute stress response research should incorporate both SAM system and HPA axis indicators to capture the complete stress response profile.

This study also found that education years positively predicted heart rate stress responses, contrary to Fiocco et al. (2007). This discrepancy may arise because Fiocco et al. controlled for self-esteem levels across different education groups, whereas the present study did not control for self-esteem. Li, Wang, Chen, and Bi (2016) found that longer education duration correlated with higher self-esteem in Chinese populations. Additionally, research using TSST with Chinese participants found positive correlations between self-esteem and acute heart rate stress responses (Chen et al., 2014). Thus, higher self-esteem among more educated individuals may elevate sympathetic acute stress responses, facilitating rapid psychophysiological resource mobilization to meet higher social approval needs.

Study limitations include: (1) The sample comprised university students, limiting generalizability to other populations. (2) Animal research indicates an inverted-U relationship between stress intensity and behavior, with different stress levels showing distinct relationships with psychological functioning (Sapolsky, 2015). Ethical constraints limit laboratory stress induction to low-to-moderate intensity responses (Wu & Yan, 2017). Although some studies have examined relationships between trait anxiety and stress responses to naturally occurring major stressors, these have used only post-traumatic stress disorder symptoms or subjective emotional responses as outcome measures (Cobb et al., 2017; Shell, Gazelle, & Faldowski, 2014), which may be subject to interpretation biases (Compton et al., 2008). Future research should incorporate objective physiological measures to improve validity when assessing severe stress responses to major stressors.

In summary, this study demonstrates that trait anxiety significantly predicts sympathetic nervous system responses to acute psychological stress, with higher trait anxiety associated with smaller sympathetic responses. This finding provides a scientific predictive indicator for individual stress responses during emergencies. Future research should employ longitudinal designs to examine how psychological traits measured during non-stressful periods predict physiological

and psychological responses to naturally occurring life events.

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Note: Figure translations are in progress. See original paper for figures.