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Behavioral and self-reported sensitivity to reward are linked to stress-related differences in positive affect

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ABSTRACT

Despite the high prevalence of stress exposure healthy adaptation or resilience is a common response. Theoretical work and recent empirical evidence suggest that a robust reward system, in part, supports healthy adaptation by preserving positive emotions even under exceptionally stressful circumstances. We tested this prediction by examining empirical relations among behavioral and self-reported measures of sensitivity to reward, trait resilience, and measures of affect in the context of experimentally induced stress. Using a quasi-experimental design we obtained measures of sensitivity to reward (self-report and behavioral), as well as affective and physiological responses to experimental psychosocial stress in a sample of 140 healthy college-age participants. We used regression-based moderation and mediational models to assess associations among sensitivity to reward, affect in the context of stress, and trait resilience and found that an interaction between exposure to experimental stress and self-reported sensitivity to reward predicted positive affect following experimental procedure. Participants with high sensitivity to reward reported higher positive affect following stress. Moreover, positive affect during or after stress mediated the relation between sensitivity to reward and trait resilience. Consistent with the prediction that a robust reward system serves as a protective factor against stress-related negative outcomes, our results found predictive associations among sensitivity to reward, positive affect, and resilience.

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1. Introduction

The role of stress in the etiology of mood and anxiety disorders is well documented (Hammen, 2005; Pizzagalli et al., 2007; van Praag, 2004). Although individual responses to a stressor (traumatic or otherwise) vary, with many individuals reacting positively, relatively little is known of the factors contributing to this positive adaptation (Bonanno, 2004). Extensive cross-species research documents the role of reward and reward-related neural circuitry in the development of psychiatric disorders (Bogdan et al., 2013; Bogdan and Pizzagalli, 2006; Corral-Frías et al., 2015, 2013; Epstein et al., 2006; Franklin et al., 2012; Keedwell et al., 2005; Krishnan et al., 2007; Pizzagalli et al., 2009, 2007; Steele et al., 2007). Reduced ability to experience reward or pleasure (i.e. anhedonia) is a central feature of many stress-related disorders (Elman et al., 2009; Knutson et al., 2008; Pizzagalli et al., 2007) and evidence suggests that stress-induced dysregulation of the reward system increases vulnerability to some of these disorders (e.g., depression, posttraumatic stress disorder, substance use) (Bogdan et al., 2013; Corral-Frías et al., 2015; Elman et al., 2009; Knutson et al., 2008). Recent studies reveal marked reductions in reward approach behavior and reduced reward-related neural reactivity in the context of early-life or acute experimental stress (Bogdan and Pizzagalli, 2006; Dillon et al., 2009; Lighthall et al., 2012; Mehta et al., 2010; Treadway et al., 2013), suggesting a prominent role of stress in the appearance of anhedonic symptoms and related psychiatric disorders (Corral-Frías et al., 2015; Nikolova et al., 2012). However, cross-species evidence has demonstrated that stress may lead to an increase in reward salience (Chaijale et al., 2015), burst firing of rodent ventral tegmental area (VTA) dopamine neurons (Anstrom and Woodward, 2005), and increased dopamine release, reward-related behaviors and neural activation in humans (Mather and Lighthall, 2012; Scott et al., 2006), altogether highlighting the

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importance of understanding the complex relationships between the reward system, stress and related psychopathology.

A robust reward system appears to protect against the deleterious effects of stress and the presence of positive trait-like emotions decreases the risk of psychopathology following stress (Charney, 2004; Southwick et al., 2005). Optimism, humor, and an ability to experience reward or pleasure predict responses to stress (Bonanno, 2004; Charney, 2004; Folkman and Moskowitz, 2000; Fredrickson, 2001; Haglund et al., 2007; Southwick et al., 2005; Tugade and Fredrickson, 2004). Congruently, increased reward-related neural activity (e.g., ventral striatal reactivity in response to reward) appears to protect against the damaging effects of recent-life (Nikolova et al., 2012) and early-life (Corral-Frías et al., 2015) stress. However, these studies have explored the moderating role of reward processing on the relationship between stress and positive affect using retrospective measures of stress. Laboratory studies would be the most informative way to analyze the interactions between reward- and stress-related behaviors, but to date, no studies have examined the relationships between sensitivity to reward and the responses to an experimentally induced laboratory-based stressor.

Given this previous evidence (Corral-Frías et al., 2015; Nikolova et al., 2012), the present study used a quasi-experimental design to test the hypothesis that sensitivity to reward moderates the relationship between stress exposure and positive affect after stress. Moreover, based on previous literature suggesting positive affect is used to cope with stressful life experiences and thus mediate the relationship between stress and resilience (Gloria et al., 2013; Tugade and Fredrickson, 2004) we examined the prediction that positive affect following and during exposure to stress mediates relations between sensitivity to reward and self-reported trait resilience. We hypothesized, in congruence with existent literature, higher reward sensitivity will be associated with higher positive affect in the context of stress and in turn with greater reports of trait resilience.

2. Materials and methods

2.1. Participants

One hundred fifteen undergraduate and twenty five graduated students participated. Undergraduate students were recruited through an online University of Arizona subject pool sign-up system, available only to undergraduate students enrolled in INDV101 courses. Graduate students were recruited through a graduate student list serve and completed the study as volunteers. Graduate students were evenly distributed in both groups. Nine (14.1%) and 16 (23.9%) of the participants in the Control group and Experimental group respectively were graduate students. A Pearson's chi square test showed that graduate students were not unevenly represented in one group or the other ($\chi^2(1) = 2.043$; $p = .15$). Participants were at least 18 years of age (mean = 21.35 ± 4.32, ranging from 18 to 32). Both male ($N = 59$) and female ($N = 81$) participants were recruited; 64.4% of whom self-identified as White, 18.4% as Hispanic, 8.1% as Asian, 3.7% as Black, 2.2% as Hawaiian or Pacific Islander, and 0.7% as Native American (two did not provide this information).

Participants were pseudo-randomly assigned to a Control or Experimental group before arriving in the laboratory. Demographic characteristics did not differ significantly between the Control and Experimental groups (Table 1), nor did initial anxiety indices (Beck et al., 1988), defense style (Muris and Merckelbach, 1996), or self-report sensitivity to reward (Carver and White, 1994). Study exclusion criteria included: (1) not completing the majority of the study (2) self-reported psychiatric diagnosis, and (3) medical diagnosis of neurological, metabolic, or hormonal disorders.

Table 1
Demographic characteristics of the sample.

	Control	Experimental	*t*	*P*
Age	20.93 ± 4.21	21.68 ± 4.44	.986	.362
Sex	F: 36 M: 24	F: 37 M: 30	−.768	.444
BAI	13.31 ± 12.01	12.77 ± 12.23	−.253	.801
BIS	19.33 ± 2.39	19.67 ± 2.35	−.788	.433
BAS (Drive)	11.05 ± 2.48	11.61 ± 2.80	−1.17	.243
BAS (Fun Seeking)	12.03 ± 2.27	12.39 ± 2.23	−.899	.371
BAS (RR)	17.30 ± 2.19	17.50 ± 2.28	−.750	.455
DSQ (Mature)	5.64 ± 0.95	5.76 ± 0.96	.685	.495
DSQ (Immature)	3.87 ± 0.88	3.93 ± 1.04	.248	.379
DSQ (Neurotic)	4.69 ± 1.02	4.87 ± 1.05	.883	.680

Means ± standard deviations; BAI, Beck Anxiety Inventory; BIS, Behavioral Inhibition Scale; BAS, Behavioral Activation Scale; RR, Reward Responsiveness; DSQ, Defense Style Questionnaire.

Thus data from four participants who did not complete the study were excluded from the analysis, three participants in the Control and three in the Experimental group were additionally excluded due to self-reported psychiatric diagnosis. No participant reported neurological, metabolic, or hormonal disorders. Additionally, 42 participants (27 Control and 15 Experimental) were excluded in cortisol and 12 participants from heart rate statistical analysis (3 Control and 9 Experimental) due to a malfunction of the freezer where samples were stored and malfunction for heart rate collection device respectively.

2.2. Consenting and online procedures

Participants completed an online consent form before completing a set of online questionnaires, which included a general demographics questionnaire, the Beck Anxiety (Beck et al., 1988), a resilience questionnaire (Wagnild and Young, 1993), the Behavioral Inhibition Scale/Behavioral Activation Scale (BIS/BAS;(Carver and White, 1994)), and the Defense Style Questionnaire (Muris and Merckelbach, 1996). Participants additionally were asked to report any history of neuroendocrine, neurological or psychiatric disorder as well as current or past use of medications.

2.3. Experimental procedures

Participants were asked to come to the laboratory at least five hours after waking to reduce time-related circadian changes in levels of free salivary cortisol (peak levels occur shortly after awakening (Hansen et al., 2008)). Upon arrival, participants read and signed a written informed consent form and then completed a monetarily rewarded task (Monetary Incentive Delay; MID) (Knutson et al., 2000). Those in the Experimental group experienced the Trier Social Stress Test (TSST) (Kirschbaum et al., 1993); those in the Control group experienced a placebo version of the TSST (Het et al., 2009). Finally, participants in both groups completed the MID a second time.

Physiological response (heart rate and salivary cortisol) and self-reported affective state were measured before, during and after the stress/control manipulation (see Fig. 1A for a timeline). The entire experimental protocol lasted about 90 min. Upon completion of the study, all participants were debriefed, and dismissed. Undergraduate students were granted research credits for their participation.

2.3.1. Resilience measure

A 25-item 7-point Likert-style self-report questionnaire assessed trait resilience (Wagnild and Young, 1993). This scale includes two subscales: *personal competence* (17 items), which reflects determination and resourcefulness, and *acceptance of self and life* (8 items), which reflects acceptance of life and sense of peace in the face of adversity.

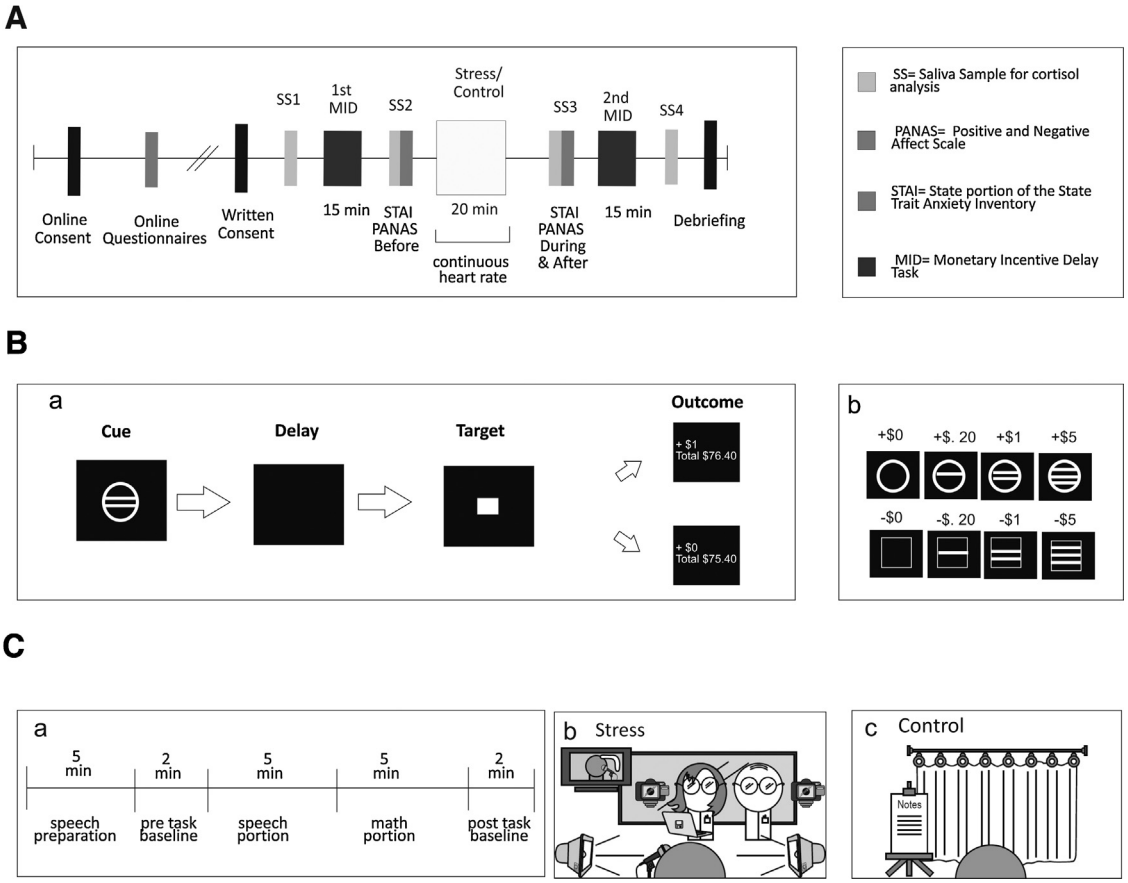

Fig. 1. Experimental Protocol (A) Timeline. Participants completed a series of online questionnaires before participating in the study. After written consent, participants performed a reward sensitivity task (Monetary incentive delay task; MID) and, after exposure to a psychosocial stressor or control, performed the same reward sensitivity tasks a second time. Physiological responses (salivary cortisol and heart rate) and self-reported affect (PANAS and state portion of STAI) were collected throughout. MID. a). Participants hit the spacebar as quickly as possible when a white target square appeared on the screen. A cue denoting the possible reward or loss and a delay preceded the target. (b) Cues signaled a potential reward represented by a circle or a potential penalty represented by a square. (C) Experimental Stress/Control Manipulations. a. Participants prepared and presented a speech, after which they perform an arithmetic task. Participants were randomly assigned to either (b) Experimental group (stress) or (c) Control group (no stress).

2.3.2. Sensitivity to reward measures

2.3.2.1. Behavioral activation scale. Three BAS subscales: *Reward-Responsiveness*, which measures positive affect and excitability (5 items), *Drive*, which measures the pursuit of appetitive goals (4 items) and *Fun-Seeking*, which measures the inclination to seek out new rewarding situations (4 items) assessed trait sensitivity to reward (Carver and White, 1994).

2.3.2.2. Monetary incentive delay task. Each participant completed a 90-trial computerized reward task (Knutson et al., 2000) twice, once before and once after experimental manipulation (stress/control), to measure their behavioral sensitivity to reward. Each trial consisted of the presentation of a cue (a circle or a square), a brief delay, and the presentation of a target; participants were previously instructed to respond to the target as quickly as possible (see Fig. 1Ba). A sufficiently quick response to the target produced a reward or avoided a penalty. A circle signaled a potential reward ($0, $0.20, $1.00, $5.00) and a square signaled a potential penalty (−$0, −$0.20, −$1.00, −$5.00) (Fig. 1Bb). There were 72 potential reward and punishment trials of which half of the cues presented represented potential rewards (36 trials) and half represented potential punishments (36 trials). The number of trials and percentage of each type of cue was identical for all participants. Starting cutoff reaction time was set to 850 ms to maximize reward outcomes and the program adapted to the participant's reaction times over trials (i.e., cutoff was shortened for faster and lengthened for slower participants). Thus, the presentation time of the target varied between individuals and was determined using response times of previous trials. Although participants did not receive actual monetary rewards, most received research credits for participation. There were no statistical differences in reaction times between those that received course credit (undergraduate) and those who did not (graduate; see Supplemental Table 1). Based on previous evidence demonstrating that cues representing higher monetary rewards elicit greater BOLD activation in reward-related neural regions (Knutson et al., 2000, 2008; Knutson and Cooper, 2005; Spreckelmeyer et al., 2009) and previous research using reaction time to reward cues as proxies for motivation (Pizzagalli et al., 2009) pre-manipulation average reaction times to the highest reward cue were used as measures of behavioral reward sensitivity.

2.3.3. Stress and control manipulations

2.3.3.1. Trier Social Stress Test (TSST). The TSST consisted of a five-minute speech preparation, followed by a two-minute pre-task phase, a five-minute extemporaneous presentation of the speech, a five-minute arithmetic task, and a two-minute post-task phase (Fig. 1Ca; (Kirschbaum et al., 1993)). A Research Assistant (RA) instructed participants to write an outline of a speech designed to indicate their qualifications for a job of their choice. After the 5-minute speech preparation, the RA attached the equipment to measure heart rate (Vernier Software and Technology) then instructed the participant to stand and remain still for two minutes.

This provided a baseline heart rate measure. The RA then removed the participant's previously prepared notes and instructed each participant to deliver the prepared speech in the presence of two "judges" (usually one male and one female; age ranged from 19 to 25) wearing white lab coats. Immediately following the speech, the RA instructed the participant to count backwards from 1876 by 17s as quickly and as accurately as possible for five minutes. If the participant made a mistake, the judges instructed them to start again from the beginning. On the other hand, if the participant answered correctly, the judges instructed them to go faster. Finally, the judges instructed the participant to stand and remain still for two minutes. This permitted us to obtain post-manipulations heart rate measurements (Kirschbaum et al., 1993). The judges did not answer questions and were instructed to show little or no emotion. Participants were left with the impression their performance was audio and video recorded and were told that a prominent professor was watching and analyzing their performance from behind a one-way mirror. A pair of industrial lights was also turned on, adding heat and brightness to the stressful conditions outlined above (Fig. 1Cb).

2.3.3.2. Placebo Trier Social Stress Test. The participants in the Control group underwent a placebo version of the TSST (Het et al., 2009). The control manipulation consisted of a two-minute pre-task phase, a five-min speech preparation during which the participants wrote an outline of a recent event, followed by a five-min speech during which the participants retained their notes and talked about the chosen topic while alone, followed by a five-min arithmetic task during which they read the answers to a 2nd grade subtraction task, again while alone, followed by a two-minute post-task phase (Fig. 1Ca).

2.3.4. Stress outcomes measures

2.3.4.1. Self-report measures of affect. The State-Trait Anxiety Inventory the (STAI; (Watson et al., 1988)) and the Positive and Negative Affect Schedule (PANAS; (Watson et al., 1988)) assessed subjective affect before, during, and after stress. The RA handed the self-administered questionnaires to participants at two different times (Fig. 1A), immediately before the psychosocial stressor to measure baseline affect with instructions to fill out the questionnaires keeping in mind how they felt at that moment (i.e., before experimental manipulation) and then two questionnaires immediately after the experimental manipulation. The RA instructed the participant to complete the first of these two questionnaires while keeping in mind how s/he felt at that moment (i.e., after experimental manipulation) and the second with instructions to keep in mind how s/he felt during the experimental manipulation.

2.3.4.2. Heart rate. Heart rate was recorded continuously for two minutes before (pre-task), during, and two minutes after (post-task) the speech and math portion of the experimental manipulation (Fig. 1Ca). A handgrip heart-rate monitor (one sample per 5-s) using Logger pro data collection software collected heart-rate data. Electrodes embedded in the hand-grip heart-rate Monitor measured the signal on the surface of the skin.

2.3.4.3. Salivary cortisol. Immediately after written consent, participants rinsed their mouths thoroughly. Participants were instructed to abstain from eating or drinking for the remainder of the experiment. Saliva samples were collected, using Salivette collection devices (Salimetrics, LLC), four times during the experiment: at the beginning of the experiment, immediately before and immediately after the experimental manipulation, and after the second MID (Fig. 1A). Using in-house facilities, saliva samples were assayed for cortisol in duplicate with a commercially available enzyme immunoassay kit (ELISA, Salimetrics, LLC).

2.4. Statistical analyses

All statistical analyses were conducted using SPSS v21. Two sets of analyses were performed. First, the overall effects of Experimental psychosocial stress on stress outcomes were probed. Exposure to stress served as the independent variable; stress outcomes (self-report affect and physiological responses) served as dependent variables. Repeated measures analyses of variance were obtained with condition (Experimental versus Control) as between factors and time (before, during, and after the experimental manipulation) as within factors.

Second, given previous evidence that self-reported stress together with reward-related neural reactivity predicts positive affect (Corral-Frías et al., 2015; Nikolova et al., 2012) we tested if behavioral and self-reported reward sensitivity moderated the association between Experimental stress and positive affect using a series of linear regressions and regression-based moderation models via the PROCESS macro of SPSS (Hayes, 2013). The association between the BAS scales and behavioral responses to MID and positive affect were tested independently. Significant interactions were assessed post-hoc using the Johnson–Neyman method (Johnson and Fay, 1950) which allows for post-hoc analyses of continuous variables and determines the value of the moderator at which curves significantly differ (Lazar and Zerbe, 2011). We used this method to calculate the range of sensitivity to reward for which exposure to Experimental stress was associated with self-reported affect. Additionally, based on previous evidence demonstrating that positive affect may be used to cope with stressful life experiences and thus mediate the relationship between stress and resilience (Gloria et al., 2013; Tugade and Fredrickson, 2004) we further tested whether the interaction between stress exposure and reward sensitivity was indirectly associated with resilience through positive affect using a series of mediational and moderated mediational models. Biological sex was added as a covariate in all models because of the previously documented sex differences in reactivity to reward stimuli (Spreckelmeyer et al., 2009).

3. Results

3.1. Responses to psychosocial stress

Exposure to experimental stress successfully altered both positive and negative affect. A repeated measures ANOVA comparing blocks of time detected a significant decrease in positive affect ($F_{(2,248)} = 25.32$, $p < .01$, $\eta^2_p = .170$; Fig. 2A) and increase in self-reported negative affect in the Experimental but not the Control group ($F_{(2,238)} = 28.86$, $p < .01$, $\eta^2_p = 195$; Fig. 2B). Post-hoc *t*-tests revealed that participants in the Experimental group self-reported decreased positive affect during ($t_{(126)} = 4.66$; $p < .01$) and after stress ($t_{(126)} = 6.06$ $p < .01$). Likewise, there was an increase in negative affect in the Experimental group relative to the Control group during ($t_{(121)} = -5.09$; $p < .01$) and after stress ($t_{(126)} = -6.343$; $p < .01$).

The stress manipulation also significantly increased physiological responses. A repeated measures ANOVA detected a significant time × condition interaction in heart rate ($F_{(2,342)} = 13.33$, $p < .01$, $\eta^2_p = .105$) and salivary cortisol ($F_{(2,246)} = 7.62$, $p < .01$, $\eta^2_p = .105$). Post-hoc *t*-test detected no baseline differences in heart rate between Experimental and Control groups ($t_{(1,117)} = -1.67$; $p = .09$), but detected significant differences between these groups during the speech portion ($t_{(1,117)} = -3.97$; $p < .01$), math portion ($t_{(1,118)} = -3.944$; $p < .01$), and after the manipulation subsided ($t_{(1,116)} = -2.22$; $p < .05$). Additionally, post-hoc *t* test detected no differences in salivary cortisol between Experimental and Control groups during baseline (before MID: $t_{(1,87)} = .27$; $p = .78$ and before

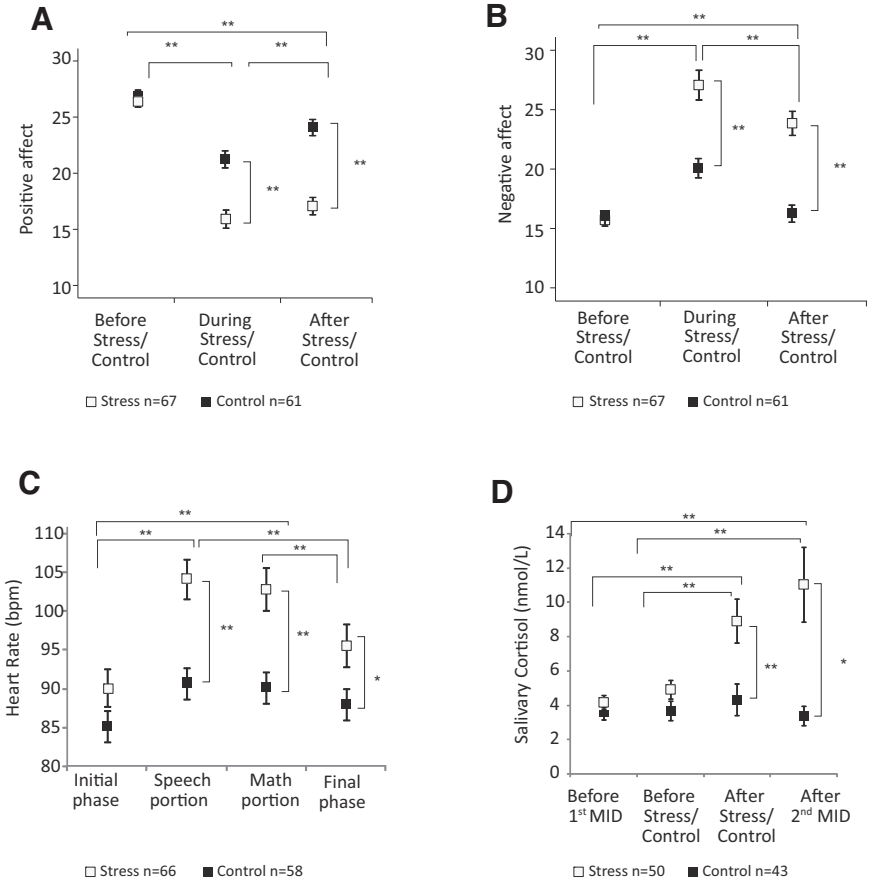

Fig. 2. Changes in affect and physiological responses after stress and control manipulations. (A) Positive affect. (B) Negative affect. (C) Heart rate. (D) Salivary cortisol. All figures illustrate means plus standard errors. (Split plot ANOVA * = $p < .05$, ** = $p < .01$).

stress/control: $t_{(1,88)} = -.65$; $p = .512$), but detected significant differences in salivary cortisol immediately after stress/control ($t_{(1,87)} = -2.38$; $p < .05$) and 15 min after the manipulation had ended ($t_{(1,84)} = -3.17$; $p < .01$).

3.2. Sensitivity to reward, positive affect, and resilience

Our analyses detected independent main effect associations between self-report (BAS Drive: $t_{(1,121)} = 2.203$; $p < .05$), and behavioral sensitivity to reward (MID reaction time to highest rewarding cues: ($t_{(1,121)} = -2.228$; $p < .05$)), and self-reported positive affect during manipulation, regardless of condition. Fig. 3 illustrates the association between self-report sensitivity to reward and positive affect throughout the stress procedure (For figures illustrating association with behavioral measures see Supplemental Fig. 3). Higher sensitivity to reward (as exemplified by higher self-reported reward BAS Drive and lower reaction time in response to MID cues representing high rewards) was associated with higher positive affect during experimental procedures in both the stress and control conditions. This effect was not present before (*BAS Drive*: $t_{(1,121)} = 719$, $p = .474$; *MID reaction time*: $t_{(1,121)} = -.43$; $p = .66$; Fig. 3A) or after Experimental or Control procedures (*BAS Drive:* $t_{(1,121)} = 91$; $p = .36$; *MID high reaction time:* $t_{(1,118)} = -.98$; $p = .32$). Our analyses additionally detected a significant two-way interaction between self-report sensitivity to reward (but not behavioral; see Supplemental material and Fig. 3) and stress exposure that predicted positive affect after stress ($\Delta R^2 = .0286$, $b = .9209$; $p < .05$). Post-hoc analyses (Johnson–Neyman method) revealed that those who were exposed to experimental stress and reported relatively higher sensitivity to reward also self-reported higher positive affect after the experimental procedure compared to those with relatively lower sensitivity to reward (Experimental: $t = -2.6190$, $p = .01$; Control: $t = .5617$, $p = .57$; Fig. 3C). To ensure that our results were not confounded by differences in behavioral inhibition, neurotic defense style, or trait anxiety, these variables were added as covariates in the moderation models ($\Delta R^2 = 0.0308$, $b = 0.4336$; $p = 0.02$). Additionally, to confirm that the results were not further confounded by differences in trait resilience we ran moderation analyses with trait resilience as a covariate and our results remained significant ($\Delta R^2 = 0.032$, $b = 1.0069$; $p = 0.02$). The data suggest that during an unknown experimental procedure (either stress or control), higher sensitivity to reward aided participants in the maintenance of positive emotions. Moreover, once the manipulation ceased, sensitivity to reward continued to be protective—but only in the context of stress.

To further explore the association between sensitivity to reward and resilience, we investigated the prediction that positive affect during or after stress exposure mediated the relationship between sensitivity to reward and trait resilience. Consistent with this hypothesis, higher self-report and behavioral sensitivity to reward predicted greater positive affect during experimental procedures which, in turn, predicted higher self-reported trait resilience (Fig. 4A and B). Interestingly, positive affect after the manipulation mediated the relationship between self-reported sensitivity to reward and resilience, but only for those in the stress group (Fig. 4C). Those in the stress group reporting higher sensitivity to reward also self-reported higher positive affect after stress exposure and in turn higher trait resilience. To further test the moderating effect of stress in the association between reward sensitivity, positive affect and resilience we ran moderated mediation models using the Mature

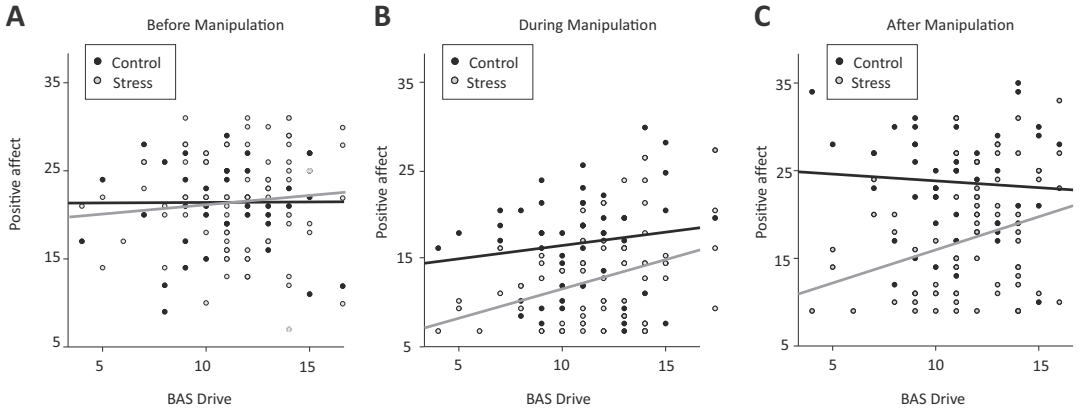

Fig. 3. Sensitivity to reward moderates the relationship between stress exposure and positive affect after stress but not during or before. (A) Positive affect before Stress/Control (ΔR^2 = .0023 b = .1966 p = .61). (B) Positive affect during Stress/Control (ΔR^2 = .0069 b = .4263 p = .32). (C) Positive affect after Stress/Control (ΔR^2 = .0286 b = .9209 p = .03).

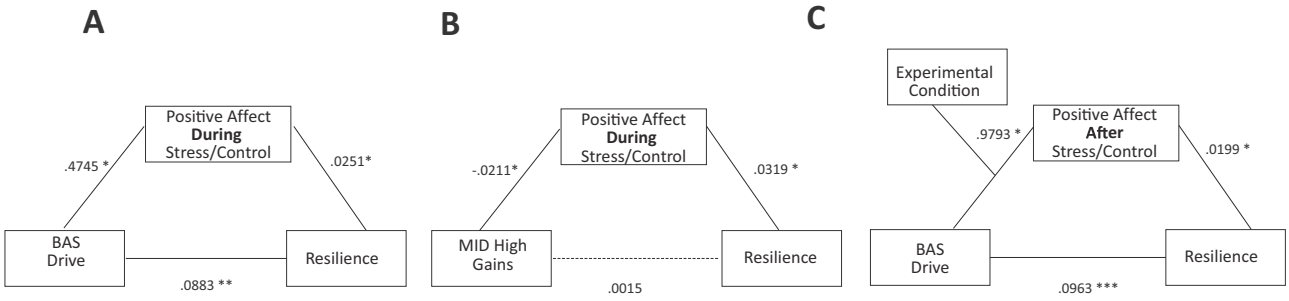

Fig. 4. Mediational Models. Standard regression coefficients for the (A) relationship between self-report and (B) behavioral sensitivity to reward and self-report resilience as mediated by positive affect during experimental stress. (C) Standard regression coefficients for the association between self-report sensitivity to reward and self-report resilience as mediated by positive affect after experimental manipulation. (* = p < .05, *** = p < .001).

subscale of the Defense Style Questionnaire (where significant differences between the Experimental and Control group were not present; see Table 1) which has been previously utilized as a measure of resilience in stress research (Simeon et al., 2007). We found a trending effect in a consistent direction (Supplemental Fig. 6). The conditional indirect effect was only significant for the stress group. These moderated mediational models suggest that sensitivity to reward, through the maintenance of positive affect in the context of stress exposure, protects against the deleterious effects of stress.

4. Discussion

The present results join a growing body of evidence highlighting the importance of reward and positive emotions as protective against the effects of stress (Bijttebier et al., 2012; Corral-Frías et al., 2015; Geschwind et al., 2010; Nikolova et al., 2012; Ryba and Hopko, 2012; Vythilingam et al., 2009) by demonstrating a significant role of sensitivity to reward in moderating the relationship between positive affect and exposure to experimental stress. In contrast to most studies which examine this interaction only using self-reported measures of stress, the present study documents a theoretically important relationship between self-reported and behavioral sensitivity to reward, and *positive* affect during and following experimental stress (Fig. 3). Moreover, the results demonstrate that positive affect in the context of experimental stress mediates the relationship between sensitivity to reward and trait resilience (Fig. 4).

Although extensive research links reward system dysfunctions to various psychiatric disorders, such as depression, PTSD and substance use disorder (Diekhof et al., 2008; Pizzagalli et al., 2009; Sailer et al., 2008), little is known about how individual differences in reward function lead to variation in outcomes after exposure to stress or trauma. Animal research has demonstrated that the mesolimbic system, critical for reward processing, is essential for determining individual differences in susceptibility or resistance to social defeat stress (Krishnan et al., 2007). Current human literature regarding this issue is, however, limited. Heller and colleagues (2013) demonstrated that individuals with sustained reward-related neural circuit reactivity to positive stimuli self-reported greater well-being and exhibited less diurnal cortisol output than controls (Heller et al., 2013). Although Heller et al. (2013) probed the effect of reward-related neural function on well-being and its relationship with stress-related neuroendocrine output, they did not provide an empirical link to stress exposure. In a previous study, Geschwind et al. (2010) found that high reward experience was associated with reduced future affective symptoms in the context of either early-life or recent-life stress. The data presented in the present study extend this evidence by demonstrating a link between sensitivity to reward and positive affect following stress using experimental stress procedures. Further, the association between positive affect and sensitivity to reward in the context of stress is consistent with previous evidence showing increased reward system response in the context of acute stress (Anstrom and Woodward, 2005; Chaijale et al., 2015; Mather and Lighthall, 2012; Scott et al., 2006; Treadway et al., 2013).

Most previous studies have explored the moderating role of reward processing on the relationship between stress and positive affect using retrospective measures of stress (Corral-Frías et al., 2015; Geschwind et al., 2010; Nikolova et al., 2012). By using stress manipulations such as the Trier Social Stress Test we were able to explore how sensitivity to reward influenced emotional outcomes before, during, and after exposure to a stressful

situation. The present data support the hypothesis that sensitivity to reward may have different roles during and after exposure to stress. For example, during the experimental procedure, higher sensitivity to reward was associated with higher positive affect regardless of manipulation type—suggesting that higher sensitivity to reward helped maintain positive emotions during unpredictable situations. Interestingly, once the manipulation had ceased, the association was present only in those who were exposed to stress—suggesting that reward function was particularly important during recovery from stress.

The mechanisms through which higher sensitivity to reward lead to better outcomes after stress are under active investigation and many of the insights have come from findings in the animal model literature. Recent reports based on animal models have suggested that resilience or resistance to stress is in part due to increased plasticity or robustness of neural reward centers such as the ventral tegmental area or ventral striatum (Krishnan et al., 2007). Similarly, recent neuroimaging studies reported that reward-related ventral striatal activity moderates the association between recent-life stress and positive affect, suggesting that higher sensitivity to reward provides some protection against the deleterious effects of stress (Corral-Frías et al., 2015; Nikolova et al., 2012).

In interpreting the results presented in this manuscript, it is important to consider them in the context of some study limitations. The present study used a healthy university sample, thus the generalizability of these data to the general population needs to be demonstrated. A systematic replication of this research, using a representative community sample, would further help elucidate the links between sensitivity to reward and adaptation after stress exposure. As these studies used a cross-sectional design it is difficult to establish if individual differences in reward function precedes stress exposure or if this exposure has long-lasting effects on the development and function of reward brain circuits. Thus, longitudinal research is needed to more clearly define specific roles of the biological mechanisms underlying reward function and its links to resilience or resistance to stress. Additionally, we did not assess early-life or recent-life stress in this sample. Early-life stress has a marked effect on reward system function (Dillon et al., 2009; Mehta et al., 2010) as well as the anticipation of future exposure to life stressors (Heim et al., 2002) and thus may have predisposed some members of the current sample to have lower responses to reward as well as worse outcomes after stress. Moreover, the present results were only significant using the Drive subscale of the BAS as a predictor. Carver and White (1994), however, mention that the Drive subscale is the best predictor of happiness response following reward-related cues and suggest that this subscale is the best predictor of sensitivity to reward. Consistent with this suggestion, previous studies have shown that BAS subscales are associated with increased neural sensitivity to reward (Simon et al., 2010). Specifically, the Drive subscale is the best predictor of certain types of rewarding cues (Beaver, 2006); thus conveying more confidence to our results. Another clear difference between our study and previous studies probing reward using the MID task is the lack of payment to our participants. It is clear that payment increases effort and performance (Bonner and Sprinkle, 2002). Although, previous research suggests that participants respond similarly to real and hypothetical rewards (Madden et al., 2003), the use of hypothetical money in our task may have produced reaction times that are not directly comparable to those observed in tasks where actual money was awarded. The evidence here is mixed. Consistent with this assertion, reaction times in the present study were, in average, about 100 ms slower than in previous studies (Spreckelmeyer et al., 2009). On the other hand, the reaction time profile was directly comparable: reaction times were significantly faster for higher reward cues than smaller reward cues (see Supplemental Fig. 1). Finally, although the data demonstrate some behavioral effects (see Supplemental materials), neural measures evaluating the contrast between high rewards and no rewards would be a more appropriate measure of sensitivity to reward. Future studies evaluating the effect of reward sensitivity on positive affect in the context of stress should include reward-related neural reactivity.

These limitations notwithstanding, the present study provides empirical evidence consistent with the theoretically predicted protective characteristics of sensitivity to reward in participants experiencing experimentally induced psychosocial stress. Further understanding of the biological, cognitive, and behavioral underpinnings and mechanisms of reward processing and its relationship to vulnerability as well as resilience to psychopathology is needed (Bogdan et al., 2013; Bogdan and Pizzagalli, 2006; Elman et al., 2009; Knutson et al., 2008; Pizzagalli et al., 2009). Better understanding of these mechanisms and how they relate to individual differences in stress outcomes may lead to preventative efforts as well as more efficacious, and individually tailored treatments (Bogdan et al., 2013; Kupfer and Regier, 2011).

Conflict of interest

None.

Contribution of authors

NSCF helped with the design of the study, collected data, performed data analysis and took the lead with writing the manuscript. WJJ helped with the design of the study, oversaw the collection and analysis of the data, and contributed to the writing the manuscript. LN helped with the design of the study and provided feedback on the manuscript. JMF helped with the design of the study, oversaw the collection of the data and provided feedback on the manuscript.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.psyneuen.2016.01.012.

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