



# Acute Stress Selectively Reduces Reward Sensitivity

## Citation

Berghorst, Lisa Hinckley, Ryan Bogdan, Michael J. Frank, and Diego A. Pizzagalli. 2013. Acute stress selectively reduces reward sensitivity. *Frontiers in Human Neuroscience* 7:133.

## Published version

<https://doi.org/10.3389/fnhum.2013.00133>

## Link

<http://nrs.harvard.edu/urn-3:HUL.InstRepos:10582747>

## Terms of use

This article was downloaded from Harvard University's DASH repository, and is made available under the terms and conditions applicable to Open Access Policy Articles (OAP), as set forth at

<https://harvardwiki.atlassian.net/wiki/external/NGY5NDE4ZjgzNTc5NDQzMGIzZWZhMGFIOWI2M2EwYTg>

## Accessibility

<https://accessibility.huit.harvard.edu/digital-accessibility-policy>

## Share Your Story

The Harvard community has made this article openly available. Please share how this access benefits you. [Submit a story](#)

1 Abstract Word Count: 175 (1,373 characters with spaces)  
2 Manuscript Word Count: 7,327  
3 Total Number of Figures and Tables: 6  
4  
5  
6

7 Running Title: Stress selectively reduces reward sensitivity  
8  
9  
10

## 11 **Acute stress selectively reduces reward sensitivity**

12  
13 Lisa H. Berghorst<sup>1,2</sup>, Ryan Bogdan<sup>3</sup>, Michael J. Frank<sup>4</sup>, and Diego A. Pizzagalli<sup>2\*</sup>  
14  
15  
16  
17

18 <sup>1</sup>Department of Psychology, Harvard University, Cambridge, MA, USA

19 <sup>2</sup>Center for Depression, Anxiety and Stress Research, McLean Hospital, Harvard Medical  
20 School, Belmont, MA, USA

21 <sup>3</sup>BRAIN Laboratory, Department of Psychology, Washington University in St. Louis, St.  
22 Louis, MO, USA

23 <sup>4</sup>Brown Institute for Brain Science, Departments of Psychiatry and Cognitive, Linguistic,  
24 & Psychological Sciences, Brown University, Providence, RI, USA  
25  
26  
27  
28  
29  
30  
31

32 \*Correspondence:

33 Diego A. Pizzagalli, Ph.D.

34 Harvard Medical School

35 Center for Depression, Anxiety and Stress Research, McLean Hospital

36 115 Mill Street,

37 Belmont, MA 02478

38 E-mail: [dap@mclean.harvard.edu](mailto:dap@mclean.harvard.edu)  
39  
40  
41  
42  
43

1 **Abstract**

2 Stress may promote the onset of psychopathology by disrupting reward processing.  
3 However, the extent to which stress impairs reward processing, rather than incentive  
4 processing more generally, is unclear. To evaluate the specificity of stress-induced  
5 reward processing disruption, 100 psychiatrically healthy females were administered a  
6 probabilistic stimulus selection task enabling comparison of sensitivity to reward-driven  
7 (Go) and punishment-driven (NoGo) learning under either ‘no stress’ or ‘stress’ (threat-  
8 of-shock) conditions. Cortisol samples and self-report measures were collected. Contrary  
9 to hypotheses, the groups did not differ significantly in task performance or cortisol  
10 reactivity. However, further analyses focusing only on individuals under ‘stress’ who  
11 were high responders with regard to both cortisol reactivity and self-reported negative  
12 affect revealed reduced reward sensitivity relative to individuals tested in the ‘no stress’  
13 condition; importantly, these deficits were reward-specific. Overall, findings provide  
14 preliminary evidence that stress-reactive individuals show diminished sensitivity to  
15 reward but not punishment under stress. While such results highlight the possibility that  
16 stress-induced anhedonia might be an important mechanism linking stress to affective  
17 disorders, future studies are necessary to confirm this conjecture.

18  
19

20 **Keywords:** affect-cognition interactions, stress, anhedonia, reward, punishment, cortisol,  
21 depression, emotion

# 1 INTRODUCTION

2  
3 Unraveling the connection between life stress and the onset of affective disorders  
4 continues to be a critical but complex endeavor. The reward system is often dysfunctional  
5 in affective disorders (American Psychiatric Association, 2000) and may play a central  
6 role in bridging these phenomena. Specifically, mounting evidence suggests that stress  
7 attenuates reward responsiveness through its influence on underlying neurobiological  
8 processes (Anisman and Matheson, 2005). However, a central point of ambiguity in this  
9 domain concerns the specificity of the impact of stress on reward processing. In order to  
10 gain a more comprehensive understanding of the mechanisms at play, it is necessary to  
11 clarify whether such effects might be generalizable to other valence-laden stimuli (e.g.,  
12 punishment) and thus reflective of incentive processing more broadly.

13 A large body of preclinical work suggests that uncontrollable negative stressors  
14 blunt sensitivity to reward via disruption of mesocorticolimbic pathways. The majority  
15 of research investigating relationships between stressors and reward processing has been  
16 performed in non-human animal studies. In rodents, uncontrollable stress leads to  
17 “anhedonic” behavior and dysfunction within mesocorticolimbic dopaminergic pathways  
18 critically implicated in incentive motivation and hedonic coding (Anisman and Matheson,  
19 2005; Henn and Vollmayr, 2005). Surprisingly, relatively few researchers have  
20 empirically examined putative relationships between stress and the reward system in  
21 humans. In an early human study, Berenbaum and Connelly (1993) found that real-life  
22 acute stressors, including military training and final examinations, reduced self-reported  
23 pleasure and positive affect in two separate samples. Moreover, this stress-induced  
24 reduction in hedonic capacity was strongest in participants with family histories of  
25 depression. In a controlled laboratory setting, Bogdan and Pizzagalli (2006) reported that  
26 an acute stressor (threat-of-shock) blunted reward responsiveness—specifically,  
27 participants’ ability to modulate behavior as a function of rewards (see Bogdan *et al.*,  
28 2011 and Liu *et al.*, 2011 for independent replications). Using the same probabilistic  
29 reward task, participants with high levels of perceived life stress were characterized by  
30 decreased reward responsiveness (Pizzagalli *et al.*, 2007). Recently, Cavanagh and  
31 colleagues (2010) employed a social evaluative threat stress manipulation while  
32 participants completed a probabilistic stimulus selection task. They found that stress led  
33 to relatively decreased reward learning in individuals with high trait-level punishment  
34 sensitivity (as assessed using the Behavioral Inhibition System (BIS) scale) as compared  
35 to an enhanced reward learning bias in individuals with lower trait-level punishment  
36 sensitivity. Complementing these behavioral findings, two recent neuroimaging studies  
37 reported that stress inductions (e.g., cold pressor task, aversive movie clips)  
38 superimposed on reward processing paradigms reduced activity in brain areas involved in  
39 reward processing, such as the medial prefrontal cortex, orbitofrontal cortex, and dorsal  
40 striatum (Ossewaarde *et al.*, 2011; Porcelli *et al.*, 2012).

41 In spite of these findings, it remains unclear whether such stress-induced effects  
42 are specific to rewards or extend to negatively-valenced stimuli, such as punishment. In  
43 Cavanagh’s aforementioned study (2010), social evaluative stress led to heightened  
44 sensitivity to punishment in individuals with high trait-level punishment sensitivity, but  
45 lower sensitivity to punishment in individuals with low trait-level punishment sensitivity.  
46 In related research, various prior studies have examined aversive processing changes

1 using threat of shock manipulations and reported stress-induced increases in aversive  
2 processing during affective Stroop tasks (e.g., Edwards, Burt, & Lipp, 2006; Edwards,  
3 Burt, & Lipp, 2010; Robinson, Letkiewicz, Overstreet, Ernst, & Grillon, 2011). In a  
4 recent fMRI study investigating the neural circuitry underlying such findings, Robinson  
5 and colleagues (2012) reported that enhanced dorsomedial prefrontal cortex amygdala  
6 connectivity during the processing of aversive stimuli under stress (threat of  
7 unpredictable foot shock in the scanner) might underlie stress-induced threat biases.  
8 Collectively, these studies raise the possibility that, unlike reward sensitivity,  
9 punishment sensitivity might be potentiated under stress.

10 The current study was designed to assess the specificity of the deleterious effect  
11 of stress on reward processing by comparing the impact of stress on reward-related (e.g.,  
12 positive feedback) versus punishment-related (e.g., negative feedback) learning. To  
13 achieve this aim, a probabilistic stimulus selection task (PSSST; modified from Frank *et*  
14 *al.*, 2004) was implemented in conjunction with an acute stressor (threat-of-shock) using  
15 a between-subjects design (e.g., ‘stress’ vs. ‘no-stress’). The current study design  
16 differed from previous studies in this area (e.g., Bogdan and Pizzagalli, 2006; Bogdan *et*  
17 *al.*, 2011) because it allowed evaluation of responsiveness to both positive and negative  
18 feedback. This enabled us to ascertain whether purported stress-induced reward  
19 processing deficits reflected specific reductions in sensitivity to reward feedback vs.  
20 broad reductions in sensitivity to feedback in general (regardless of valence). In addition,  
21 our experiment was initially designed to test whether the impact of stress on reward  
22 processing was conditional upon the stress being perceived as uncontrollable. This was  
23 attempted by implementing both a ‘controllable’ and ‘uncontrollable’ stress condition,  
24 along with a ‘no stress’ condition. However, this aspect of our stress manipulation was  
25 unsuccessful (see *Supplement* for detailed analyses) and thus the present report focuses  
26 on the comparison between ‘stress’ (collapsed across the two controllability subgroups)  
27 and ‘no-stress’ conditions. Based on prior findings, we hypothesized that individuals  
28 under acute stress would exhibit reduced reward sensitivity (e.g., lower reward-related  
29 accuracy and a reduced reward-related RT bias, as detailed in the *Methods* section)  
30 relative to individuals in the no-stress condition. Moreover, we hypothesized that reward  
31 sensitivity would be selectively more reduced relative to punishment sensitivity in those  
32 individuals completing the task under stress.

## 33 34 **MATERIALS AND METHODS**

### 35 **Participants**

36 All study procedures were approved by Harvard University’s Committee on the Use of  
37 Human Subjects in Research. One hundred (n = 100) female participants, 18 to 25 years  
38 old, were recruited through community advertisements and the Harvard University  
39 Department of Psychology Study Pool. Only females were recruited due to sex  
40 differences in psychological and hormonal responses to stress, and because women tend  
41 to demonstrate a more pronounced stress response than men (Nolen-Hoeksema and Hilt,  
42 2009). All subjects were right-handed, non-smokers, with normal or corrected-to-normal  
43 vision, no color-blindness, and no known current or past neurological, psychiatric or  
44 medical illnesses. Prior to participation, all individuals were screened over the phone to  
45 determine study eligibility. The evaluation included diagnostic screening questions from  
46 the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First, Spitzer,

1 Gibbon and Williams, 1995), more detailed questions from the depression and substance  
2 abuse modules, and a handedness questionnaire (Chapman and Chapman, 1987).  
3 Subjects were excluded if they could speak or read Japanese because one of the tasks  
4 (PSST) included Hiragana symbols. Individuals who met eligibility requirements were  
5 invited for an experimental session. Prior to the session, participants were randomized to  
6 one of three experimental conditions: ‘no stress’ (n = 29), ‘controllable stress’ (n = 35),  
7 or ‘uncontrollable stress’ (n = 36). Data from five participants (two from the ‘no stress’  
8 group, one from the ‘controllable stress’ group and two from the ‘uncontrollable stress’  
9 group) were excluded because they never met performance criteria (see *Modified*  
10 *Probabilistic Stimulus Selection Task (PSST)* section) in the training phase of the PSST.  
11 Thus, 95 participants were included in the analyses: ‘no stress’ group (n = 27),  
12 ‘controllable stress’ group (n = 34), and ‘uncontrollable stress’ group (n = 34). However,  
13 given the lack of success of the controllability aspect of our stress manipulation (see  
14 *Supplement* for detailed analyses), data from the two stress groups were combined into a  
15 single ‘stress’ group in subsequent analyses.

## 16 17 18 **Procedures**

19 Figure 1 presents a summary of the session timeline. After arriving to the  
20 laboratory, the first written informed consent was obtained using a general consent form  
21 with no mention of the stress manipulation. This procedure allowed us to obtain  
22 unbiased baseline self-report ratings and physiological indices. Participants were then  
23 asked to complete a battery of self-report questionnaires, including a demographics form,  
24 the Beck Depression Inventory-II (BDI-II; Beck, Steer, and Brown, 1996), the Mood and  
25 Anxiety Symptom Questionnaire (MASQ-short; Watson *et al.*, 1995), the Perceived  
26 Stress Scale (PSS; Cohen *et al.*, 1983), the Temporal Experience of Pleasure Scale  
27 (TEPS; Gard *et al.*, 2006), and the Behavioral Inhibition and Behavioral Activation  
28 Scales (BIS/BAS; Carver and White, 1994).

29 Twenty minutes after arrival, the first of three saliva samples was collected to  
30 measure baseline cortisol levels. Next, participants completed the first set of “in-the-  
31 moment” state self-report questionnaires to obtain baseline ratings of their current mood  
32 (= “baseline” timepoint for analyses). These included the state versions of the State Trait  
33 Anxiety Inventory (STAI-S; Spielberger *et al.*, 1983) and the Positive and Negative  
34 Affect Schedule (PANAS-S; Watson *et al.*, 1988).

35 Next, the second written informed consent was obtained using either a ‘no stress’  
36 condition or a ‘stress’ condition consent form. The ‘stress’ consent form stated that  
37 participants might receive electrical shocks (via two electrodes attached to their right  
38 hand) during two ensuing computer games: “up to two” shocks during the first task (a  
39 “filler” task) and “up to three” shocks during the second task (the PSST). Participants  
40 then completed a computerized basic attention task that acted as a “filler” task, during  
41 which all participants in the ‘stress’ condition received one electrical shock (performance  
42 in this task was extraneous to study hypotheses). This task served the purpose of making  
43 the potential for shock a credible threat given that we did not actually administer any  
44 shock during the main task of interest (PSST). Following the “filler” task, participants  
45 completed a second identical set of “in-the-moment” state self-report questionnaires (=“  
46 “post-filler-task/pre-PSST” timepoint); additionally, participants were asked to provide a

1 second saliva sample for cortisol level analyses (approximately 13 minutes after the  
2 shock).

3       Thereafter, participants who completed the “filler” task in the ‘stress’ condition  
4 were further subdivided into ‘controllable stress’ and ‘uncontrollable stress’ conditions,  
5 and participants received the appropriate set of instructions for the PSST. Between the  
6 training and test phases of the PSST, participants completed a third set of “in-the-  
7 moment” state self-report questionnaires (= “PSST” timepoint) probing affect  
8 experienced during the training phase of the task (i.e., the phase of the task involving the  
9 stress manipulation). Following the test phase of the PSST, participants were asked to  
10 provide a third saliva sample for cortisol analyses (time-locked to 10 minutes from the  
11 end of the training phase of the PSST in order to capture cortisol levels when participants  
12 in the stress conditions were under perceived ‘threat of shock’). Then, they completed a  
13 final set of “in-the-moment” state self-report questionnaires (= “post-task” timepoint).  
14 Participants also completed a post-task questionnaire to probe their experiences during  
15 the session. At the end of the experiment, all participants were debriefed and either paid  
16 (\$10/hour) or awarded study credit for their time. The overall session took approximately  
17 1.5 to 2 hours, and subjects received \$15-\$20 or 1.5-2 study credits. Please see  
18 *Supplement* for detailed descriptions of trait and state measures.

### 19 20 ***Stress manipulation***

21       Two electrodes were placed on the right hand of each participant assigned to  
22 either of the stress conditions, and the electrode wires were attached to a shock box  
23 placed on the table in front of the participant. The shock level was adjusted to what each  
24 participant felt was “aversive, but not painful.” This was done by beginning at the lowest  
25 level of shock intensity and having the participant experience a brief shock at each level  
26 to have the participant identify a level that she felt was “aversive, but not painful.” The  
27 maximum current intensity (4 mA; Coulbourn E13-22) was approved by the local IRB.  
28 Prior to the “filler” task, these participants were told that they could receive up to two  
29 electrical shocks, but the task was actually programmed to administer only one shock. In  
30 the PSST, all participants were told that they would see a multicolored bar on either side  
31 of the computer screen with a tick mark that would periodically move up and down. In  
32 the ‘no stress’ condition, they were told that the bars had no meaning. They were also  
33 told that occasionally the border of the computer screen would flash red and they should  
34 press down on a foot pedal when they saw this visual cue in order to indicate that they  
35 were attending to the task. The task was programmed for the cue to appear 1 - 2 times  
36 during each practice block, but participants were not given information about the  
37 frequency of this occurrence. For participants in both the ‘controllable stress’ and  
38 ‘uncontrollable stress’ conditions, the border flashing red indicated that a shock might  
39 occur in the next 15-30 seconds and they were told that the location of the tick mark  
40 within the multicolored bars would indicate the likelihood they would receive a shock.  
41 For these participants, the multicolored bars were labeled with “danger” at the top and  
42 “safe” at the bottom, and the closer the tick mark was to the top of the bar, the higher the  
43 likelihood of receiving a shock. Moreover, participants in the stress conditions were told  
44 that the movement of the tick mark was determined by the computer and was thus  
45 unrelated to their performance on the task. However, participants in the ‘controllable  
46 stress’ condition were told that if they pressed the foot pedal when they saw the red

1 border visual cue, they could override the computer and lower the location of the tick  
2 mark in the bars, thus reducing (albeit not fully eliminating) the likelihood they would  
3 receive a shock. When these participants pressed down on the foot pedal, the tick mark  
4 did shift down closer to the “safe” zone at the bottom of the bar, providing some visual  
5 feedback. In contrast, participants in the ‘uncontrollable stress’ condition were instructed  
6 to press down on the foot pedal to indicate they were attending to the task (i.e., they  
7 received the same instructions about the foot pedal as those in the ‘no stress’ condition)  
8 and this had no effect on the location of the tick mark. Participants in both stress  
9 conditions were told they could receive up to three electrical shocks during the PSST; in  
10 reality, no shock was administered during this task. Of note, the threat-of-shock stress  
11 manipulation was only in effect during the training phase of the PSST. This was the  
12 target of our stress manipulation because reward and punishment feedback were only  
13 provided during that phase of the task.

#### 14 15 ***“Filler” task***

16 Participants completed a brief version (~8 min) of a Continuous Performance  
17 Task (CPT; Conners, 1995) as a “filler” task. They were presented with a series of letters  
18 (“O,” “T,” “H,” “Z,” or “X”) on a computer screen, one at a time, and were instructed to  
19 press the space bar immediately following any letter except for “X.” Participants  
20 completed two blocks of 125 trials, with each letter appearing in 25 trials; on each trial,  
21 the letter stimulus was presented for 500 ms, followed by an interstimulus interval that  
22 varied between 1250-1550 ms.

#### 23 24 ***Modified Probabilistic Stimulus Selection Task (PSST)***

25 The PSST included a training phase and a test phase (Figure 2). During the  
26 training phase, participants were presented with three different stimuli pairs (AB, CD,  
27 EF) in random order, and were instructed to choose one of the two stimuli by pressing  
28 one of two response buttons. Following a subject’s response, feedback was given to  
29 indicate whether the choice was “correct” or “incorrect.” Importantly, this feedback was  
30 probabilistic, such that for AB trials, a choice of stimulus A led to correct (positive)  
31 feedback in 80% of the trials, while a choice of stimulus B led to incorrect (negative)  
32 feedback in these trials (with the relations reversed for the other 20% of AB trials). The  
33 stimulus pair CD was less reliable, with stimulus C correct in 70% of CD trials, and the  
34 stimulus pair EF was the least reliable, with stimulus E correct in 60% of the EF trials.  
35 During this training phase, subjects learned to choose stimuli A, C, and E more  
36 frequently than B, D, or F. Of note, selection of A over B could be achieved either by  
37 learning that choosing A usually leads to positive feedback or learning that choosing B  
38 usually leads to negative feedback, or both. Participants completed the training phase  
39 either under a ‘no stress,’ ‘controllable stress,’ or ‘uncontrollable stress’ condition. The  
40 training phase was terminated after participants reached performance criteria (65% A in  
41 AB, 60% C in CD, and 50% E in EF) or after the completion of 6 blocks. The  
42 performance criteria were set so that all participants would be at approximately the same  
43 performance level before proceeding to the test phase (i.e., there was no ‘overtraining’ for  
44 subjects who had already learned the contingencies because they would advance to the  
45 test phase earlier).

1 In the test phase, subjects were presented with the same three stimuli pairs, as  
2 well as all novel combinations of stimuli pairs, and feedback was not provided (Figure 2).  
3 In order to examine whether subjects learned more about the positive or negative  
4 outcomes of their decisions in the training phase, the stimuli pairs of primary interest in  
5 the test phase were those involving an A or B stimulus paired with a novel stimulus (e.g.,  
6 AC, AD, AE, and AF; BC, BD, BE, and BF), referred to as “transfer pairs.” These  
7 transfer pairs enabled assessment of the degree to which participants learned from prior  
8 positive feedback to choose the most reinforced stimulus (“Choose A”) and/or learned  
9 from prior negative feedback to avoid the most punished stimulus (“Avoid B”). Prior  
10 studies have shown that these conditions are differentially sensitive to dopaminergic  
11 manipulation and that performance in the “Choose A” condition is correlated with neural  
12 responses to positive outcomes, whereas performance in the “Avoid B” condition is  
13 correlated with neural responses to negative outcomes.

14 The stimuli presented in the PSST were black-and-white Hiragana characters. In  
15 the training phase, each trial began with a fixation cross in the middle of the screen for  
16 1000 ms, followed by a stimuli pair for 2000 ms or until the participant made a response.  
17 Thereafter, visual feedback was provided for 1500 ms as either “Correct” in blue letters,  
18 “Incorrect” in red letters, or “No response detected” in red letters (if the subject did not  
19 respond within 2000 ms). Each block of the training phase had 60 trials with 20 trials per  
20 stimuli pair. In the test phase, each trial began with a fixation cross for 1000 ms,  
21 followed by a stimuli pair for 3000 ms or until the participant made a response. The test  
22 phase consisted of one block of 90 trials, with six trials of each of the 15 possible  
23 stimulus pairs.

### 24 ***Saliva samples***

26 For saliva collection, participants were instructed to put a small cotton roll  
27 (Salivette) in their mouth for approximately 90 seconds, and then place the saliva-soaked  
28 cotton into a small plastic tube. Saliva samples were subsequently stored in a freezer  
29 ( $\leq -20$  degrees Celsius) until assayed. The timing of the collection of cortisol samples  
30 (specified in the procedures section above) was based on prior research indicating that  
31 cortisol typically peaks about 10-20 minutes after stressor onset (e.g., Kudielka, Buske-  
32 Kirschbaum, Hellhammer, & Kirschbaum, 2004). To control for diurnal rhythms in  
33 cortisol levels, all participants were run between the hours of 1pm and 6pm (Dickerson  
34 and Kemeny, 2004). To further control for fluctuations in hormone levels, participants  
35 were asked to adhere to the following instructions: no eating or brushing their teeth for at  
36 least an hour before the session; no consumption of yogurt for at least two hours before  
37 the session; no consumption of any caffeine-containing products or alcohol the day of the  
38 session; no strenuous exercise the day of the session. Information was also collected  
39 regarding the time of day participants woke up and the time of the session.

### 40 **Data Analyses**

#### 41 ***Trait and dispositional self-report measures***

44 Total and subscale scores were computed for the BDI, MASQ, PSS, TEPS, and  
45 BIS/BAS, and t-tests were run to compare participants who completed the task under  
46 ‘stress’ versus ‘no-stress’ conditions.

1  
2 ***“In-the-moment” state self-report measures***

3 To assess the effectiveness of the stress manipulation, separate mixed ANOVAs  
4 were conducted on STAI-S, PANAS-PA (positive affect), and PANAS-NA (negative  
5 affect) scores, with *Time* (Baseline, PSST) as a repeated measure and *Group* (Stress, No-  
6 Stress) as a between-subjects factor. Significant findings were followed up with t-tests.  
7

8 ***PSST training phase***

9 To evaluate potential group differences in training, t-tests were conducted to  
10 compare groups on the number of blocks required to reach performance criteria; separate  
11 mixed ANOVAs were run for accuracy and RT on the final training block with *Trial*  
12 *Type* (AB, CD, EF) and *Group* as factors. Significant differences were followed up with  
13 t-tests.  
14

15 ***PSST test phase***

16 Prior to the main analyses of interest, a t-test was run to compare accuracy on AB  
17 trials (the “easiest” trial type) in the test phase to confirm that there were no significant  
18 differences between ‘stress’ and ‘no stress’ groups with regard to participants learning the  
19 basic task. Although the performance criteria in the training phase was intended to  
20 address this issue, it is possible that participants could have become confused by the lack  
21 of feedback and the addition of novel stimuli pairs in the test phase, so this served to  
22 verify that learning carried over to the test phase.

23 Thereafter, to assess whether participants learned more from the positive or  
24 negative feedback they received during training, data from the test phase were analyzed  
25 with respect to performance on the test trials involving novel combinations of stimuli  
26 pairs that included either an A or a B stimulus, respectively. For trials involving an A  
27 stimulus paired with a novel stimulus (“Choose A” trials), accuracy was calculated as the  
28 proportion of trials on which the participant chose A (the most frequently reinforced  
29 stimulus) over the novel stimulus. For trials involving a B stimulus paired with a novel  
30 stimulus (“Avoid B” trials), accuracy was calculated as the proportion of trials on which  
31 the participant avoided B (the most frequently punished stimulus) and chose the novel  
32 stimulus instead. Next, ANOVAs were performed with *Trial Type* (‘Choose A,’ ‘Avoid  
33 B’) and *Group* as factors to examine accuracy and RT separately. Significant differences  
34 were followed up with the appropriate t-tests.  
35

36 ***Saliva samples (cortisol)***

37 In order to obtain cortisol levels, saliva samples were sent to the Laboratory for  
38 Biological Health Psychology (Brandeis University, MA, USA) and analyzed in a single  
39 batch to avoid assay variability (intra-assay CV = 6.48%; inter-assay CV = 6.06%).  
40 These values were then entered into an ANOVA using *Time* (T1 = baseline, T2 = post-  
41 “filler” task/pre-PSST, T3 = post-PSST) and *Group* as factors. Given the diurnal drop in  
42 cortisol levels throughout the day (Schmidt-Reinwald *et al.*, 1999), and the inevitable  
43 variability in wake-up time across participants, we also calculated the difference between  
44 waking time and time of the first saliva collection; this value was used as a covariate in  
45 the aforementioned ANOVA. Next, in line with previous studies (e.g., Townsend *et al.*,  
46 2011), we calculated cortisol reactivity scores (i.e., difference scores from T1 to T2, or

1 T1 to T3) for all participants. Finally, an ANOVA was run to compare cortisol reactivity  
2 scores with *Group*.

3  
4 ***Follow-up analyses: Using changes in cortisol levels and self-reported state anxiety to***  
5 ***identify stress-reactive subgroup***

6 Given that ‘threat of shock’ might only have been stressful for a sub-group of  
7 participants, we identified individuals who were relatively high stress responders based  
8 on changes in cortisol levels and self-reported state anxiety from T1 (baseline) to T2 (~13  
9 minutes after subjects received the shock administered in the “filler” task). Initially, we  
10 examined descriptive statistics on the distribution of cortisol reactivity scores from T2 –  
11 T1 within ‘no-stress’ and ‘stress’ groups to examine if there was indeed considerable  
12 variability in reactivity scores within each group. In order to obtain a new ‘stress  
13 reactive’ group with only stress-reactive participants, we first standardized the T2 – T1  
14 cortisol reactivity scores across all participants. Next, using these standardized values,  
15 participants were divided into 3 tiers: high responders ( $> 0.24$ ), medium responders  
16 ( $-0.27 \geq$  and  $\leq 0.24$ ), and low responders ( $< -0.27$ ). These cut-off scores were selected so  
17 that approximately 1/3 of participants were in each tier. Similarly, we standardized the  
18 T2 - T1 change scores in self-reported state anxiety levels (using STAI scores), and again  
19 divided participants into 3 tiers: high responders ( $> 0.44$ ), medium responders ( $-0.66 \geq$   
20 and  $\leq 0.44$ ), and low responders ( $< -0.66$ ). Thereafter, a new ‘stress reactive’ group was  
21 created that included only participants who completed the task under stress *and* were  
22 relatively high stress responders, defined as being in the ‘high responder’ tier with regard  
23 to both changes in cortisol levels and self-reported state anxiety. Using this new ‘stress  
24 reactive’ group, all of the aforementioned analyses were re-run to compare the ‘stress  
25 reactive’ and ‘no-stress’ groups on demographics, trait and state self-report measures, and  
26 performance on the PSSST task.

27  
28  
29 **RESULTS**

30  
31 **Trait and dispositional self-report measures (No-Stress vs. Stress Groups)**

32 As evident in Table 1, there were no significant differences between the ‘no-  
33 stress’ and ‘stress’ groups on the trait or dispositional self-report measures collected at  
34 baseline [all  $t_s \leq 1.67$ ,  $p_s \geq 0.10$ ]. Accordingly, putative differences in behavioral  
35 performance or stress reactivity were not confounded by group differences in trait or  
36 dispositional affect, or ongoing stress levels.

37  
38

1 **Table 1:**

	No Stress (NS) Group	Stress (S) Group	Stress Reactive (SR) Group	NS v. S Statistic	p	NS v. SR Statistic	p
<b>Gender (% female)</b>	100%	100%	100%	N/A	N/A	N/A	N/A
<b>Age (years)</b>	21.43 (± 1.79)	21.32 (± 2.20)	22.05 (± 1.92)	t(93) = 0.22	0.83	t(43) = 1.11	0.28
<b>Education (years)</b>	14.81 (± 1.39)	14.35 (± 1.61)	14.94 (± 1.35)	t(93) = 1.31	0.19	t(43) = 0.31	0.76
<b>Marital Status (% single)</b>	100%	93%	89%	$\chi^2(2) = 2.10$	0.35	$\chi^2(1) = 3.14$	0.08
<b>Income* (% &lt; \$50,000)</b>	90%	74%	69%	$\chi^2(1) = 2.29$	0.13	$\chi^2(1) = 2.29$	0.13
<b>Compensation Form (% monetary)</b>	85%	90%	78%	$\chi^2(1) = 0.39$	0.54	$\chi^2(1) = 0.41$	0.52
<b>Ethnicity (% Caucasian)</b>	85%	59%	61%	$\chi^2(2) = 10.07$	<b>0.01</b>	$\chi^2(1) = 3.39$	0.07
<b>BDI-II</b>	1.85 (± 2.38)	2.21 (± 2.34)	1.67 (± 2.03)	t(93) = -0.66	0.51	t(43) = 0.27	0.79
<b>MASQ: GDA</b>	15.52 (± 4.74)	15.66 (± 3.90)	16.22 (± 3.21)	t(93) = -0.15	0.88	t(43) = -0.55	0.59
<b>MASQ: GDD</b>	16.85 (± 5.25)	18.10 (± 5.12)	17.72 (± 5.79)	t(93) = -1.07	0.29	t(43) = -0.52	0.60
<b>MASQ: AA</b>	20.52 (± 4.82)	19.59 (± 3.62)	19.28 (± 3.05)	t(93) = 1.03	0.31	t(43) = 0.97	0.34
<b>MASQ: AD</b>	49.56 (± 10.90)	49.71 (± 10.68)	45.83 (± 8.99)	t(93) = -0.06	0.95	t(43) = 1.20	0.24
<b>Perceived Stress Scale</b>	19.67 (± 6.33)	20.68 (± 5.86)	20.83 (± 4.62)	t(93) = -0.74	0.46	t(43) = -0.67	0.51
<b>TEPS: Anticipatory</b>	64.67 (± 6.68)	64.65 (± 9.78)	66.11 (± 7.80)	t(93) = 0.01	0.99	t(43) = -0.67	0.51
<b>TEPS: Consummatory</b>	48.41 (± 5.56)	50.66 (± 6.06)	52.22 (± 5.70)	t(93) = -1.67	0.10	t(43) = -2.23	<b>0.03</b>
<b>BIS/BAS: Reward Responsiveness</b>	7.48 (± 1.67)	7.51 (± 2.18)	7.56 (± 2.09)	t(93) = -0.07	0.94	t(43) = -0.13	0.90
<b>BIS/BAS: Drive</b>	9.19 (± 1.96)	9.06 (± 2.13)	9.06 (± 1.73)	t(93) = 0.27	0.79	t(43) = 0.23	0.82
<b>BIS/BAS: Fun Seeking</b>	8.04 (± 2.16)	7.78 (± 2.23)	8.00 (± 2.47)	t(93) = 0.51	0.61	t(43) = 0.05	0.96
<b>BIS/BAS: Inhibition</b>	16.00 (± 2.82)	15.40 (± 2.83)	15.33 (± 2.74)	t(93) = 0.94	0.35	t(43) = 0.79	0.44

BDI-II = Beck Depression Inventory-II; MASQ = Mood and Anxiety Symptom Questionnaire; GDA = General Distress Anxious; GDD = General Distress Depressive; AA = Anxious Arousal; AD = Anhedonic Depression; TEPS = Temporal Experience of Pleasure Scale; BIS/BAS = Behavioral Inhibition and Behavioral Activation Scales  
 \* = Participants who chose not to report income are not included in the Income statistics; this applies to 7 out of 27 (26%) 'no stress' participants and 15 out of 68 (22%) 'stress' participants.

2  
3  
4  
5  
6  
7

1  
2 **“In-the-moment” state self-report measures (No-Stress v. Stress Groups)**

3 Analyses of both state anxiety (STAI-S scores) and negative affect (PANAS-NA  
4 scores) revealed similar effects: significant *Time x Group* interactions [ $F(1,93) > 5.06$ ,  
5  $ps < 0.03$ ], along with significant main effects of *Time* [ $F(1,93) > 8.80$ ,  $ps < 0.01$ ] and  
6 *Group* [ $F(1,93) > 4.87$ ,  $ps \leq 0.03$ ]. Importantly, at baseline, groups did not differ in their  
7 levels of state anxiety or negative affect [ $ts(93) < 0.46$ ,  $ps > 0.64$ ]. During the PSST,  
8 participants in the ‘stress’ group reported significantly higher levels of state anxiety and  
9 negative affect than participants in the ‘no-stress’ group [ $ts(93) > 3.00$ ,  $p < 0.01$ ].  
10 Within-group paired t-tests indicated that anxiety increased from baseline to PSST in the  
11 ‘no stress’ group [ $t(26) = 2.17$ ,  $p = 0.04$ ] and, to a much greater degree, in the ‘stress’  
12 group [ $t(67) = 8.54$ ,  $p < 0.01$ ]. Meanwhile, negative affect increased significantly from  
13 baseline to PSST in the ‘stress’ group [ $t(67) = 4.45$ ,  $p < 0.01$ ] but not in the ‘no stress’  
14 group [ $t(26) = 0.62$ ,  $p = 0.54$ ]. The mixed ANOVA on PANAS-PA scores revealed only  
15 a significant main effect of *Time* [ $F(1,93) = 11.33$ ,  $p < 0.01$ ; all other  $Fs < 2.58$ ,  $ps >$   
16  $0.11$ ], with levels of positive affect decreasing from baseline to PSST in both groups.  
17

18 **PSST training phase (No-Stress v. Stress Groups)**

19 Groups did not differ in the number of completed training blocks [ $t(93) = 0.27$ ,  $p$   
20  $= 0.79$ ]; all groups took approximately 3 blocks to advance to the test phase [No-Stress:  
21  $3.15 \pm 1.75$ ; Stress:  $3.25 \pm 1.62$ ]. A *Trial Type* (AB, CD, EF) x *Group* (‘no stress,’  
22 ‘stress’) mixed ANOVA on accuracy scores in the final training block indicated only a  
23 significant main effect of *Trial Type* [ $F(1,93)=24.71$ ,  $p < 0.01$ ; all other  $Fs < 2.41$ ,  $ps >$   
24  $0.12$ ]; as expected, participants were most accurate on the AB trial type and least accurate  
25 on the EF trial type. No significant differences emerged from the mixed ANOVA for RT  
26 in the final training block [all  $Fs < 1.06$ ,  $ps > 0.30$ ]. Altogether, these findings indicate  
27 that (1) the probabilistic contingencies elicited the intended behavioral effects, and (2)  
28 groups did not differ in performance during the training phase.  
29

30 **PSST test phase (No-Stress v. Stress Groups)**

31 The groups did not differ significantly in their accuracy on AB trials in the test  
32 phase [No-Stress Group = 90% ( $\pm 12\%$ ); Stress Group = 86% ( $\pm 23\%$ ); [ $t(93)= 0.94$ ,  $p =$   
33  $0.35$ ], confirming that learning carried over to the test phase similarly for the two groups.  
34 Contrary to hypotheses, the *Trial Type* (“Choose A,” “Avoid B”) x *Group* ANOVA on  
35 accuracy scores revealed no significant effects [all  $Fs < 1.82$ ,  $ps > 0.17$ ].

36 For RT scores, the analogous *Trial Type x Group* ANOVA yielded a significant  
37 main effect of *Trial Type* [ $F(1,93) = 29.52$ ,  $p < 0.01$ ] and a trend for a *Trial Type x Group*  
38 interaction [ $F(1,93) = 3.29$ ,  $p = 0.07$ ]. These results reflected both groups being faster on  
39 “Choose A” trials than “Avoid B” trials, with the ‘no-stress’ group demonstrating this  
40 pattern to a greater extent.  
41

42 **Stress-reactive subgroup (defined by changes in cortisol levels and self-reported**  
43 **state anxiety)**

44 An examination of descriptive statistics on the distribution of cortisol reactivity  
45 scores at T2-T1 within ‘no-stress’ and ‘stress’ groups revealed considerable variability in  
46 reactivity scores within each group: scores in the ‘no stress’ group ranged from -5.51 to

1 1.71 [mean:  $-1.56 \pm 1.57$ ]; scores in the ‘stress’ group ranged from -7.82 to 11.78 [mean:  
2  $-0.95 \pm 2.40$ ]. Per design, cortisol reactivity scores at T2-T1 were significantly higher in  
3 the new ‘stress reactive’ group than the ‘no-stress’ group [ $t(42) = 4.01$ ,  $p < 0.01$ ; degrees  
4 of freedom reduced by 1 because cortisol data missing for one subject at T2].  
5 Importantly, cortisol reactivity scores at T3-T1 continued to be significantly higher in the  
6 ‘stress reactive’ group than the ‘no-stress’ group [ $t(41) = 3.75$ ,  $p < 0.01$ ; degrees of  
7 freedom reduced by 2 because cortisol data missing for two subjects at T3], suggesting  
8 that subjects in the ‘stress reactive’ group continued to be more physiologically stressed  
9 during the PSST than subjects in the ‘no stress’ group. The new groups did not differ  
10 significantly from each other on any of the following demographic variables: gender, age,  
11 years of education, marital status, income level, form of compensation, or ethnicity (see  
12 Table 1).

### 14 **Trait and dispositional self-report measures (No-Stress v. Stress-Reactive Groups)**

15 As compared to the ‘no-stress’ group, the ‘stress reactive’ group reported  
16 significantly higher scores on the consummatory subscale of the Temporal Experiences  
17 of Pleasure Scale (TEPS), which assesses individual trait dispositions in consummatory  
18 experiences of pleasure [ $t(43) = 2.23$ ,  $p = 0.03$ ; all other  $t(43) \leq 1.36$ ,  $ps \geq 0.18$ ]. Due to  
19 this finding, the TEPS consummatory subscore was used as a covariate.

### 21 **“In-the-moment” state self-report measures (No-Stress v. Stress-Reactive Groups)**

22 **State anxiety.** As shown in Figure 3, and in line with the new group design, the  
23 ANCOVA on STAI-S scores revealed only a significant *Time x Group* interaction  
24 [ $F(1,42) = 13.33$ ,  $p < 0.01$ ], whereas the *Time* [ $F(1,42) = 0.29$ ,  $p = 0.59$ ] and *Group*  
25 [ $F(1,42) = 3.52$ ,  $p = 0.07$ ] effects were not significant. At baseline, groups did not differ  
26 in their state anxiety levels [ $t(43) = -0.48$ ,  $p = 0.63$ ]. During the PSST, participants in the  
27 ‘stress reactive’ group reported significantly higher levels of state anxiety than  
28 participants in the ‘no-stress’ group [ $t(43) = 3.57$ ,  $p < 0.01$ ]. Within-group paired t-tests  
29 indicated that anxiety increased from baseline to PSST in both the ‘stress reactive’ group  
30 [ $t(17) = 6.31$ ,  $p < 0.01$ ] and ‘no stress’ group [ $t(26) = 2.17$ ,  $p = 0.04$ ].

31 **State negative affect.** The ANCOVA on PANAS-NA scores indicated only a  
32 significant *Time x Group* interaction [ $F(1,42) = 6.00$ ,  $p = 0.02$ ]; *Time* [ $F(1,42) = 0.95$ ,  $p =$   
33  $0.33$ ] and *Group* [ $F(1,42) = 3.57$ ,  $p = 0.07$ ]; see Figure 3. At baseline, groups did not  
34 differ in their levels of negative affect [ $t(43) = -0.12$ ,  $p = 0.90$ ]; during the PSST, the  
35 ‘stress reactive’ group reported significantly more negative affect than the ‘no stress’  
36 group [ $t(43) = 2.90$ ,  $p < 0.01$ ]. Paired t-tests indicated that negative affect increased  
37 significantly from baseline to PSST in the ‘stress reactive’ group [ $t(17) = 3.03$ ,  $p < 0.01$ ],  
38 but not in the ‘no stress’ group [ $t(26) = 0.62$ ,  $p = 0.54$ ].

39 **State positive affect.** The ANCOVA revealed no significant effects [all  $F_s < 1.95$ ,  
40  $ps > 0.17$ ].

### 42 **PSST training phase (No-Stress v. Stress-Reactive Groups)**

43 Groups did not differ in the number of completed training blocks [ $t(43) = 0.57$ ,  $p$   
44  $= 0.58$ ]; all groups took approximately 3 blocks to advance to the test phase [No-Stress:  
45  $3.15 \pm 1.75$ ; Stress-Reactive:  $3.44 \pm 1.69$ ]. Separate *Trial Type* (AB, CD, EF) x *Group*

1 ('no stress,' 'stress reactive') ANCOVA on accuracy scores and RT scores revealed no  
2 significant effects [all  $F_s < 3.13$ , all  $p_s > 0.08$ ].

#### 3 4 **PSST test phase (No-Stress v. Stress-Reactive Groups)**

5 The ANCOVA comparing accuracy on AB trials in the test phase with *Group*  
6 ('no stress,' 'stress reactive') revealed no significant group differences [No-Stress Group  
7 = 90% ( $\pm 12\%$ ); Stress-Reactive Group = 92% ( $\pm 16\%$ );  $F(1,42) = 0.63$ ,  $p = 0.43$ ],  
8 confirming that learning carried over to the test phase similarly for the two groups.  
9 Critically, the *Trial Type* ("Choose A," "Avoid B")  $\times$  *Group* ('no stress,' 'stress reactive')  
10 ANCOVA on accuracy scores revealed a main effect of *Trial Type* [ $F(1,42) = 5.72$ ,  $p =$   
11  $0.02$ ], which was qualified by a significant *Group*  $\times$  *Trial Type* interaction [ $F(1,42) =$   
12  $6.45$ ,  $p = 0.015$ ], whereas the *Group* main effect was not significant [ $F(1,42) = 0.14$ ,  $p =$   
13  $0.71$ ]. As shown in Figure 4, these findings indicate that the 'stress reactive' group  
14 displayed relatively lower accuracy on reward-related trials than punishment-related trials  
15 compared to the 'no stress' group, which exhibited the opposite pattern.

16 For RT, an analogous *Group*  $\times$  *Trial Type* ANCOVA yielded only a significant  
17 main effect of *Group* [ $F(1,42) = 7.59$ ,  $p < 0.01$ ; all other  $p_s > 0.18$ ], due to faster RTs in  
18 the 'no-stress' group than the 'stress reactive' group (Figure 4). Follow-up analyses  
19 indicated that, compared to the 'no stress' group, participants in the 'stress reactive'  
20 group demonstrated significantly slower RTs on the "Choose A" trials [ $F(1,42) = 13.67$ ,  
21  $p < 0.01$ ], but not the "Avoid B" trials [ $F(1,42) = 3.13$ ,  $p = 0.08$ ]. Moreover, participants  
22 within the 'no stress' group were faster on their "Choose A" trials than their "Avoid B"  
23 trials [ $t(26) = -4.47$ ,  $p < 0.01$ ], suggestive of a reward-related RT bias, whereas those in  
24 the 'stress reactive' group had similar RTs on both trial types [ $t(17) = -1.41$ ,  $p = 0.18$ ]  
25 and did not show this effect.

#### 26 27 **DISCUSSION**

28  
29 This study was designed to extend our understanding of stress-related anhedonic  
30 behavior by examining whether stress specifically reduces reward processing (i.e.,  
31 learning from positive feedback) or more generally influences incentive processing (i.e.,  
32 learning from both positive and negative feedback). The stress manipulation induced  
33 significantly higher levels of negative affect and anxiety in those individuals who  
34 completed the Probabilistic Stimulus Selection Task under stress versus no-stress  
35 conditions. Yet, contrary to our hypotheses, the stress manipulation did not have a  
36 significant differential impact on cortisol reactivity or task performance at the group  
37 level, likely due to large individual differences. Importantly, however, individuals with  
38 heightened cortisol reactivity and increased negative affect following acute stress did  
39 demonstrate deficits specific to reward processing. These latter findings suggest that, in  
40 highly stress-reactive individuals, stress may selectively result in reward processing  
41 deficits with no reduction in punishment processing.

42 Given that the 'threat-of-shock' stressor did evoke significantly higher levels of  
43 self-reported negative affect and anxiety in the 'stress' group than the 'no-stress' group,  
44 which was in line with prior independent studies (Bogdan and Pizzagalli, 2006; Bogdan  
45 *et al.*, 2011), we were surprised to find that the 'stress' group did not demonstrate  
46 significantly higher levels of cortisol reactivity. In light of these patterns, it is possible

1 that our stress manipulation may not have elicited as strong of a physiological stress  
2 response as intended because only a single shock was administered during the “filler”  
3 task and none were administered during the PSST. In addition, the stress manipulation  
4 did not include any social evaluative component, which has been shown to reliably  
5 produce physiological stress responses (Kirschbaum *et al.*, 1993). Moreover, for  
6 participants in the ‘stress’ group, the border of the computer screen flashing red during  
7 the PSST indicated that a shock could occur in the next 15-30 seconds; it is possible that  
8 this cue may have reduced the stressfulness of the ‘threat-of-shock’ by increasing the  
9 perceived predictability of the stressor. In fact, predictable stressors typically elicit  
10 smaller physiological stress responses and are experienced as less aversive than  
11 unpredictable stressors (Anisman and Matheson, 2005). In light of these null cortisol  
12 findings, it was not entirely surprising that initial analyses of task performance across  
13 groups yielded no significant between-group differences during the training or test phases  
14 of the PSST.

15 One potential explanation for the lack of significant findings in this initial set of  
16 analyses may be that there was a broad range of individual differences within the group  
17 of individuals who completed the task under stress in terms of how physiologically  
18 “stressed out” participants became in response to the ‘threat-of-shock.’ An examination  
19 of cortisol reactivity scores within each group indeed confirmed that there was substantial  
20 intra-group variability. Accordingly, we conducted follow-up analyses by identifying a  
21 stress-reactive subgroup based on cortisol reactivity as well as self-reported anxiety  
22 levels; the new ‘stress reactive’ group included only those participants who completed the  
23 task under stress and were ‘high responders’ from both a physiological (cortisol levels)  
24 and self-reported experiential (STAI scores) perspective. In line with these demarcations,  
25 the new ‘stress reactive’ group also demonstrated a significant increase in negative affect  
26 (PANAS-NA scores) that was not apparent in the ‘no stress’ group, reinforcing  
27 coalescence between biological measures and self-report measures of stress response.

### 28 29 **Stress-Sensitive Individuals Demonstrate Reward-Specific Impairments**

30 Consistent with previous studies (Bogdan and Pizzagalli, 2006; Bogdan *et al.*,  
31 2010; Pizzagalli *et al.*, 2007), and our main hypotheses, participants in the new ‘stress  
32 reactive’ group demonstrated reduced reward sensitivity relative to participants in the  
33 ‘no-stress’ group. This was supported in the following ways: First, there was a  
34 significant *Group* (‘no stress,’ ‘stress reactive’) x *Trial Type* (“Choose A,” “Avoid B”)  
35 interaction for accuracy during the test phase of the PSST, which was due to relatively  
36 lower accuracy on reward-related (“Choose A”) trials than punishment-related (“Avoid  
37 B”) trials in the ‘stress reactive’ group, compared with the opposite pattern exhibited by  
38 the ‘no-stress’ group (i.e., relatively higher accuracy on reward-related than punishment-  
39 related trials). This finding suggests that stress-sensitive participants did not experience a  
40 global decrease in accuracy on the task under stress, but rather a more specific reduction  
41 in accuracy on reward-related trials only. This reward-processing deficit may reflect  
42 reduced sensitivity to positive feedback (during the training phase of the PSST), evident  
43 in an impaired ability to use this reward information to guide decision making in novel  
44 contexts (during the test phase of the PSST). Secondly, participants in the ‘no-stress’  
45 group demonstrated a reward-related RT bias that was absent in the ‘stress reactive’  
46 group. Specifically, the ‘no stress’ group demonstrated faster RTs on reward-related

1 trials than punishment-related trials, while the RTs of the ‘stress reactive’ group were not  
2 significantly different between trial types. Moreover, participants in the ‘no-stress’ group  
3 were significantly faster than participants in the ‘stress reactive’ group on the reward-  
4 related trials but not the punishment-related trials. Importantly, these findings suggest  
5 that speed-accuracy tradeoffs did not play a significant role in the present results. For  
6 example, the fact that the ‘stress reactive’ group, as compared to the ‘no stress’ group,  
7 had poorer accuracy *and* slower RTs on reward-related trials runs counter to the notion  
8 that poorer accuracy could have been due to a speed-accuracy tradeoff of faster RTs.  
9 Overall, our results expand prior lines of research on stress-induced reductions in reward  
10 responsiveness by suggesting that stress may selectively reduce sensitivity to reward  
11 feedback and does not more broadly reduce sensitivity to feedback in general.

12 During the test phase, there were no group differences in accuracy on the most  
13 salient trials from the training phase (e.g., AB trials), which (1) suggests that all  
14 participants learned the basic task and this learning carried over to the test phase, and (2)  
15 provides further evidence that stress did not induce a global performance deficit across  
16 the task (e.g., differences only emerged for novel trial types in the test phase). These  
17 findings, in combination with the fact that participants across groups needed a  
18 comparable number of training blocks to reach performance criteria during the training  
19 phase, also suggest that results were not likely the byproduct of psychometric artifacts.  
20 More specifically, as highlighted in experiments assessing the effects of threat on  
21 working memory performance (Shackman et al., 2006), it is important to address whether  
22 results could be merely the artifact of an additional load on attentional resources in the  
23 stress condition, rather than stress per se. If this were the case, however, we would  
24 expect to see global deficits in task performance for individuals who completed the task  
25 under stress. In addition, a predominant lack of group differences on trait and  
26 dispositional self-report measures (the one exception being the consummatory subscale of  
27 the TEPS, which was controlled for in the analyses), and no group differences at baseline  
28 on any affective state self-report measures, suggests that putative differences in  
29 behavioral performance or stress reactivity were not confounded by group differences in  
30 affect, mood, or ongoing life stress.

31 In related research that warrants acknowledgement, Lighthall and colleagues  
32 (2012) recently reported that participants who completed the same probabilistic stimulus  
33 selection task *after* exposure to a cold pressor stress manipulation had relatively reduced  
34 punishment learning and increased reward learning. However, the stressor was  
35 terminated well before the beginning of the PSST (and an unrelated memory task was  
36 administered between the stressor and the PSST); this sequence of events raises the  
37 possibility that their observed results may have stemmed from ‘relief’ experienced by  
38 participants after the stressor. In line with the conceptualization of ‘stress relief’ as  
39 rewarding, ‘relief’ from stressors has been associated with activation of reward-related  
40 neural regions (Leknes, *et al.*, 2011) and increased dopamine levels (Navratilova *et al.*,  
41 2012). Clearly, more research is needed to examine the putative relationship between  
42 negative stressors and decreased reward sensitivity, with particular focus on the temporal  
43 unfolding of such processes.

#### 44 **Limitations**

1           There are several limitations to the current study that should be acknowledged.  
2 First, the study included only female participants due to sex differences in psychological  
3 and hormonal responses to stress (e.g., women demonstrate a more pronounced stress  
4 response than men; Nolen-Hoeksema and Hilt, 2009). Thus, future studies will be  
5 required to determine if the current stress-induced reward-specific deficits generalize to  
6 males. Second, the strength of findings is limited by the fact that significant between-  
7 group results only emerged after re-running the main analyses of interest using a ‘stress  
8 reactive’ subgroup defined based on physiological and self-reported experiential indices  
9 of stress responsiveness. This new ‘stress reactive’ group had a relatively small sample  
10 size and contained participants who had received two different sets of instructions  
11 regarding controllability of the stressor. However, the lack of significant differences  
12 between these participants (with regard to both self-report and physiological measures;  
13 see *Supplemental Analyses*) mitigates the potential effect of this latter limitation. Third,  
14 it is important to acknowledge the inherently limited ecological validity of an acute  
15 ‘threat-of-shock’ laboratory stressor and the potentially diminished strength of laboratory  
16 stressors that do not include a social evaluative component. Fourth, given that findings  
17 from this study pertain to learning from positive vs. negative feedback, it remains to be  
18 seen whether the patterns found will generalize to other types of rewards and  
19 punishments. Finally, in order to further evaluate whether stress-induced hedonic deficits  
20 are a potential mechanism underlying the link between stress and depression, it will be  
21 imperative to run parallel experiments in MDD individuals. In spite of these limitations,  
22 the current study has significant strengths, including the use of a well-controlled  
23 experimental procedure (threat-of-shock) that allowed us to superimpose an acute stress  
24 manipulation to a primary task (the PSST) and has substantial translational value.  
25  
26

## 27 **Conclusions**

28           In sum, results from these biologically informed analyses support *a priori*  
29 hypotheses and previous research findings (Bogdan and Pizzagalli, 2006; Bogdan *et al.*,  
30 2010; Pizzagalli *et al.*, 2007) by demonstrating that stress-reactive individuals under  
31 stress exhibit reduced reward processing (i.e., reduced sensitivity to positive feedback,  
32 evident in an impaired ability to use this reward information to guide decision making in  
33 novel contexts) relative to individuals not under stress. These results are also in line with  
34 recent neuroimaging studies that have shown reduced activation in reward-related neural  
35 areas in response to stress inductions implemented immediately prior to reward  
36 processing tasks (Ossewaarde *et al.*, 2011; Porcelli *et al.*, 2012). Critically, findings from  
37 the current study extend this area of research by providing initial evidence that these  
38 stress-induced deficits appear to be reward-specific and not generalizable to punishment  
39 processing. Given that negative life stress often precedes depression onset (Kendler *et al.*,  
40 1999) and predicts clinical severity (Tennant, 2002), the current results also provide  
41 support for the possibility that stress-induced hedonic deficits may be a potential  
42 mechanism underlying the connection between negative stress and depressive episodes.  
43 In this way, such results are in line with conceptualizations of stress-induced anhedonia  
44 as a potential vulnerability factor for depression (Berghorst and Pizzagalli, 2010, for  
45 review). Although promising, it is important to emphasize that (1) these findings  
46 emerged in the context of an only partially successful stress manipulation (see

1 *Supplement*); (2) findings emerged only after a subgroup of stress-reactive participants  
2 was identified; and (3) the ecological validity of the stress manipulation was limited.  
3 Accordingly, these findings await replications and conclusions should be tempered.  
4 Future studies also need to examine whether the stress-induced rapid activation of the  
5 mesocortical DA system and inhibition of the mesolimbic DA system in animal models  
6 (Cabib *et al.*, 2002; Cabib and Puglisi-Allegra, 1996) represent biological mechanisms  
7 fundamental to the current study findings.

8  
9 **Acknowledgements:**

10 This project was supported in part by a Sackler Fellowship in Psychobiology awarded to  
11 Lisa Berghorst and NIMH grants (R01 MH068376, R01 MH095809) awarded to DAP.  
12 The authors would like to thank Drs. Wendy Berry Mendes and Jeremy Jamieson for  
13 their guidance in the methods of cortisol data collection and analysis; and Dr. Jill Hooley  
14 for her valuable feedback and support throughout the project.

15  
16 **Disclosures:**

17 Dr. Pizzagalli has received consulting fees from ANT North America Inc. (Advanced  
18 Neuro Technology), AstraZeneca, Shire, Servier, and Ono Pharma USA, as well as  
19 honoraria from AstraZeneca for projects unrelated to the current research. All other  
20 authors report no competing interests.

21

1 **References:**

- 2 American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental*  
3 *disorders* (4<sup>th</sup> ed., text revision). Washington, DC: American Psychiatric Press.
- 4 Anisman, H., and Matheson, K. (2005). Stress, depression, and anhedonia: caveats  
5 concerning animal models. *Neurosci Biobehav Rev.* 29, 525-546.
- 6 Beck, A. T., Steer, R. A., and Brown, G. K. 1996. *Beck Depression Inventory Manual*  
7 (2<sup>nd</sup> ed.). San Antonio: The Psychological Corporation.
- 8 Berenbaum, H., and Connelly, J. (1993). The effect of stress on hedonic capacity. *J*  
9 *Abnorm. Psychol.* 102, 474-481.
- 10 Berghorst, L., and Pizzagalli, D. A. (2010). Defining depression endophenotypes. In C.E.  
11 Beyer and S.A. Stahl (Eds.), *Next Generation Antidepressants. Moving Beyond*  
12 *Monoamines To Discover Novel And Differentiated Treatment Strategies For Mood*  
13 *Disorders* (pp. 70-89). New York, NY: Cambridge University Press.
- 14 Bogdan, R., and Pizzagalli, D. A. (2006). Acute stress reduces reward responsiveness:  
15 implications for depression. *Biol. Psychiatry* 60, 1147-1154.
- 16 Bogdan, R., Perlis, R. H., Fagerness, J., and Pizzagalli, D. A. (2010). The impact of  
17 mineralocorticoid receptor ISO/VAL genotype (rs5522) and stress on reward  
18 learning. *Genes Brain Behav.* 9, 658-667.
- 19 Bogdan, R., Santesso, D. L., Fagerness, J., Perlis, R. H., and Pizzagalli, D. A. (2011).  
20 Corticotropin-releasing hormone receptor type 1 (CRHR1) genetic variation and  
21 stress interact to influence reward learning. *J. Neurosci.* 31, 13246-13254.
- 22 Cabib, S., and Puglisi-Allegra, S. (1996). Stress, depression and the mesolimbic  
23 dopamine system. *Psychopharmacology (Berl.)* 128, 331-342.
- 24 Cabib, S., and Puglisi-Allegra, S. (1994). Opposite responses of mesolimbic dopamine  
25 system to controllable and uncontrollable aversive experiences. *J. Neurosci.* 14,  
26 3333-3340.
- 27 Cabib, S., Ventura, R., and Puglisi-Allegra, S. (2002). Opposite imbalances between  
28 mesocortical and mesoaccumbens dopamine responses to stress by the same  
29 genotype depending on living conditions. *Behav. Brain Res.* 129, 179-185.
- 30 Campbell-Sills, L., Liverant, G. I., and Brown, T. A. (2004). Psychometric evaluation of  
31 the behavioral inhibition/behavioral activation scales in a large sample of outpatients  
32 with anxiety and mood disorders. *Psychol. Assessment* 16, 244-254.
- 33 Carver, C. S., and White, T. L. (1994). Behavioral inhibition, behavioral activation, and  
34 affective responses to impending reward and punishment: the BIS/BAS scales. *J.*  
35 *Pers. Soc. Psychol.* 67, 319-333.
- 36 Cavanagh, J. F., Frank, M. J., and Allen, J. J. (2010). Social stress reactivity alters reward  
37 and punishment learning. *Soc. Cogn. Affect. Neurosci.* 6, 1-10.
- 38 Chapman, L. J., and Chapman, J. P. (1987). The measurement of handedness. *Brain*  
39 *Cogn.* 6(2), 175-183.
- 40 Cohen, S., Kamarck, T., and Mermelstein, R. (1983). A global measure of perceived  
41 stress. *J. Health Soc. Behav.* 24, 385-396.
- 42 Conners, C. K. (1995). *Conners' Continuous Performance Test*. Toronto: Multi-Health  
43 Systems.
- 44 Dickerson, S. S., and Kemeny, M. E. (2004). Acute stressors and cortisol responses: a  
45 theoretical integration and synthesis of laboratory research. *Psychol. Bull.* 130, 355-  
46 391.

- 1 Edwards, M.S., Burt, J.S., & Lipp, O.V. (2010). Selective attention for masked and  
2 unmasked emotionally toned stimuli: Effects of trait anxiety, state anxiety, and test  
3 order. *Br. J. Psychol.* 101, 325-343.
- 4 Edwards, M.S., Burt, J.S., & Lipp, O.V. (2006). Selective processing of masked and  
5 unmasked verbal threat material in anxiety: Influence of an immediate acute stressor.  
6 *Cognition Emotion* 20, 812-835.
- 7 First M. B., Spitzer R. L., and Gibbon, M. (1995). *Structured clinical interview for DSM-*  
8 *IV*. New York: Biometrics Research Department.
- 9 Frank, M. J., Seeberger, L. C., and O'Reilly, R. C. (2004). By carrot or by stick:  
10 Cognitive reinforcement learning in Parkinsonism. *Science* 306, 1940-1943.
- 11 Gard, D. E., Gard, M. G., Kring, A. M., and John, O. P. (2006). Anticipatory and  
12 consummatory components of the experience of pleasure: a scale development  
13 study. *J. Res. Pers.* 40, 1086-1102.
- 14 Henn, F. A., and Vollmayr, B. (2005). Stress models of depression: forming genetically  
15 vulnerable strains. *Neurosci. Biobehav. Rev.* 29, 799-804.
- 16 Kendler, K.S., Karkowski, L.M., and Prescott, C.A. (1999). Causal relationship between  
17 stressful life events and the onset of major depression. *Am. J. Psychiatry* 156, 837-  
18 841.
- 19 Kirschbaum, K. M., Pirke, and Hellhammer, D. H. (1993). The Trier Social Stress Test—  
20 a tool for investigating psychobiological stress responses in a laboratory setting.  
21 *Neuropsychobiology* 28, 76-81.
- 22 Leknes, S., Lee, M., Berna, C., Andersson, J., and Tracey, I. (2011). Relief as a reward:  
23 hedonic and neural responses to safety from pain. *PLoS ONE* 6 (4), e17870.
- 24 Lighthall, N. R., Gorlick, M. A., Schoeke, A., Frank, M. J., and Mather, M. (2012).  
25 Stress modulates reinforcement learning in younger and older adults. *Psychol.*  
26 *Aging* 2012 Sep 3, [Epub ahead of print].
- 27 Liu, W.H., Chan, R.A., Wang, L.Z., Huang, J., Cheung, E.F., Gong, Q.Y., and Gollan,  
28 J.K. (2011). Deficits in sustaining reward responses in subsyndromal and syndromal  
29 major depression. *Prog. Neuropsychopharmacol. Biol. Psychiatry* 35, 1045-1052.
- 30 Mendes, W. B., Blascovich, J., Major, B., and Seery, M. D. (2001). Challenge and threat  
31 responses during downward and upward social comparisons. *Eur. J. Soc. Psychol.*  
32 31, 477-497.
- 33 Navratilova, E., Xie, J. Y., Okun, A., Qu, C., Eyde, N., Ci, S., Ossipov, M. H., King, T.,  
34 Fields, H. L., and Porreca, F. (2012). Pain relief produces negative reinforcement  
35 through the activation of mesolimbic reward-valuation circuitry. *Proc. Natl. Acad.*  
36 *Sci.U.S.A.* 109, 20709-20713.
- 37 Nolen-Hoeksema, S., Hilt, L. (2009). Gender differences in depression. In I.H. Gotlib,  
38 C.L. Hammen (Eds.), *Handbook of Depression*, 2nd ed. New York: Guilford.
- 39 Ossewaarde, L., Qin, S., Van Marle, H.J., van Wingen, G.A., Fernández, G., Hermans,  
40 E.J. (2011). Stress-induced reduction in reward-related prefrontal cortex  
41 function. *Neuroimage* 55, 345-352.
- 42 Pizzagalli, D. A., Bogdan, R., Ratner, K. G., and Jahn, A. L. (2007). Increased perceived  
43 stress is associated with blunted hedonic capacity: potential implications for  
44 depression research. *Behav. Res. Ther.* 45, 2742-2753.

- 1 Porcelli, A.J., Lewis, A.H., and Delgado, M.R. (2012). Acute stress influences neural  
2 circuits of reward processing. *Front. Neurosci.* 2012;6:157. doi:  
3 10.3389/fnins.2012.00157
- 4 Robinson, O.J., Charney, D.R., Overstreet, C., Vytal, K., & Grillon, C. (2012). The  
5 adaptive threat bias in anxiety: Amygdala dorsomedial prefrontal cortex coupling  
6 and aversive amplification. *Neuroimage* 60, 523-529.
- 7 Robinson, O.J., Letkiewicz, A.M., Overstreet, C., Ernst, M., & Grillon, C. (2011). The  
8 effect of induced anxiety on cognition: threat of shock enhances aversive processing  
9 in healthy individuals. *Cog. Affect. Behav. Neurosci.* 11, 217-227.
- 10 Schackman, A.J., Sarinopoulos, I., Maxwell, J.S., Pizzagalli, D., Lavric, A., & Davidson,  
11 R. J. (2006). Anxiety selectively disrupts visuospatial working memory. *Emotion*  
12 6, 40-61.
- 13 Schmidt-Reinwald, A., Pruessner, J. C., Hellhammer, D. H., Federenko, I., Rohleder, N.,  
14 Schümeyer, T. H., and Kirschbaum, C. (1999). The cortisol response to awakening  
15 in relation to different challenge tests and a 12-hour cortisol rhythm. *Life Sci.* 64,  
16 1653-1660.
- 17 Segal, D. L., Coolidge, F. L., Cahill, B. S., and O’Riley, A. A. (2008). Psychometric  
18 properties of the Beck Depression Inventory-II (BDI-II) among community-dwelling  
19 older adults. *Behav. Modif.* 32, 3-20.
- 20 Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., and Jacobs, G. A. (1983).  
21 *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting  
22 Psychologists Press.
- 23 Steer, R. A., Rissmiller, D. J., and Beck, A. T. (2000). Use of the Beck Depression  
24 Inventory-II with depressed geriatric inpatients. *Behav. Res. Ther.* 38, 311-318.
- 25 Tennant, C. (2002). Life events, stress and depression: a review of recent findings.  
26 *Aust. N. Z. J. Psychiatry* 36, 173-182.
- 27 Townsend, S. S., Major, B., Gangi, C. E., and Mendes, W. B. (2011). From “In the Air”  
28 to “Under the Skin”: cortisol responses to social identity threat. *Pers. Soc. Psychol.*  
29 *Bull.* 37, 151-164.
- 30 Watson, D., Clark, L. A., and Tellegen, A. (1988). Development and validation of brief  
31 measures of positive and negative affect: the PANAS scales. *J. Pers. Soc. Psychol.*  
32 54, 1063-1070.
- 33 Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., and McCormick,  
34 R. A. (1995). Testing a tripartite model: I. evaluating the convergent and  
35 discriminant validity of anxiety and depression symptom scales. *J. Abnorm. Psychol.*  
36 104, 3-14.
- 37 Williams, Z. M., Bush, G., Rauch, S. L., Cosgrove, G. R., and Eskandar, E. N. (2004).  
38 Human anterior cingulate neurons and the integration of monetary reward with  
39 motor responses. *Nat. Neurosci.* 7, 1370-1375.
- 40

1 **Figure Legends**

2

3 **Figure 1:**

4 Schematic representation of the session timeline. CORT = collection of saliva sample to  
5 measure cortisol level; MSQ = mood state questionnaires (“in-the-moment” state self-  
6 report questionnaires); PSST = Probabilistic Stimulus Selection Task

7

8 **Figure 2:**

9 (A) Schematic representation of the *training* phase of the Probabilistic Stimulus Selection  
10 Task, which was performed under stress or no stress conditions. In the no-stress  
11 condition, every time a red border flashed, participants were instructed to press a foot  
12 pedal to indicate they were attending to the task. In the two stress conditions, participants  
13 were instructed that, every time the red border flashed, a shock might occur in the  
14 ensuing 15-30 sec. In the controllable stress condition, participants were further  
15 instructed that they could reduce (though not fully eliminate) the likelihood of the shock  
16 if they pressed the foot pedal when they saw the red border flashes. In contrast,  
17 participants in the ‘uncontrollable stress’ condition were instructed that they had no  
18 possibility of reducing the likelihood of the shock. (B) Schematic representation of the  
19 *test* phase of the Probabilistic Stimulus Selection Task. No stress was presented during  
20 this phase.

21

22 **Figure 3:**

23 Affective ratings in the no-stress (n = 27) and stress-reactive (n = 18) group both at  
24 baseline and during the Probabilistic Stimulus Selection Task. (A) State Trait Anxiety  
25 Inventory (STAI) scores; and (B) Negative Affect score on the Positive and Negative  
26 Affect Schedule (PANAS). For both scale, the state version was used.

27

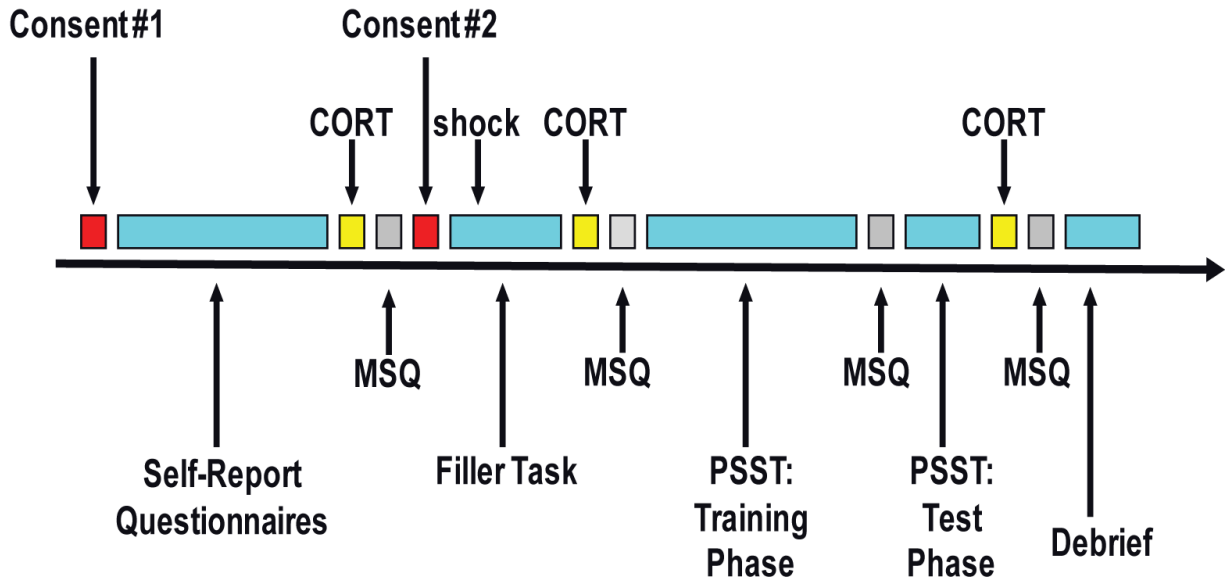
28 **Figure 4:**

29 Performance on “Choose A” and “Avoid B” Trials in Test Phase in the no-stress (n = 27)  
30 and stress-reactive (n = 18) group. (A) Accuracy; (B) Reaction Time (in ms).

31

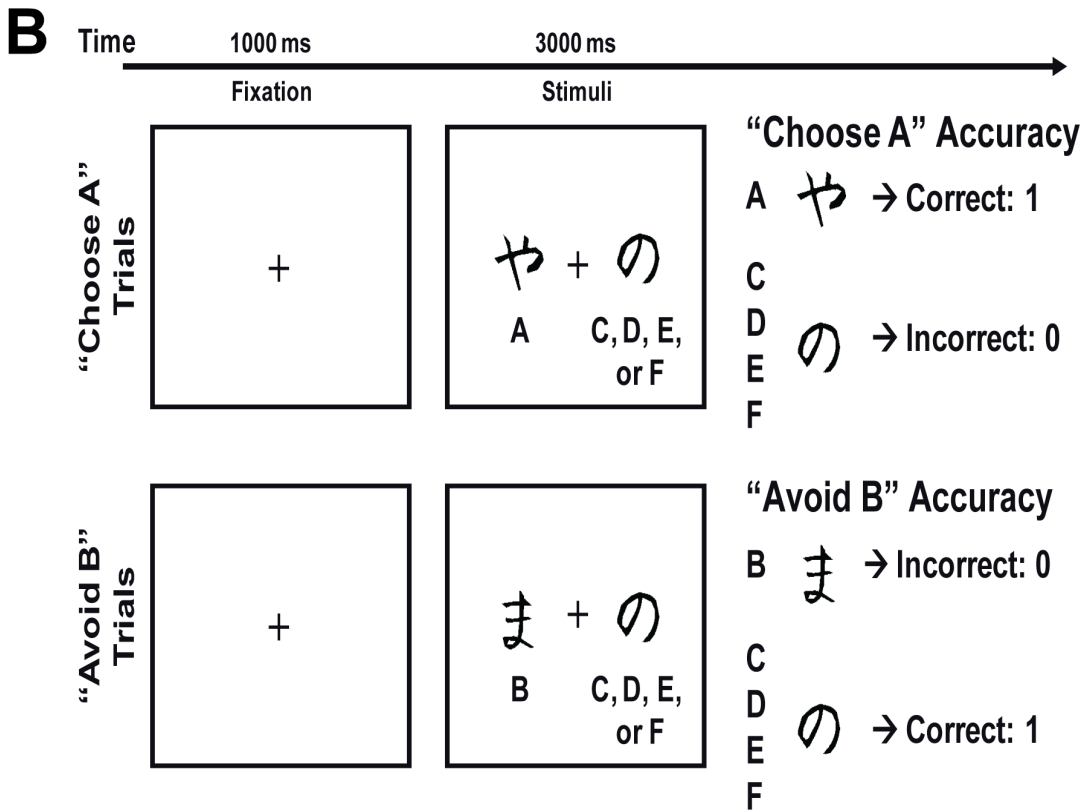
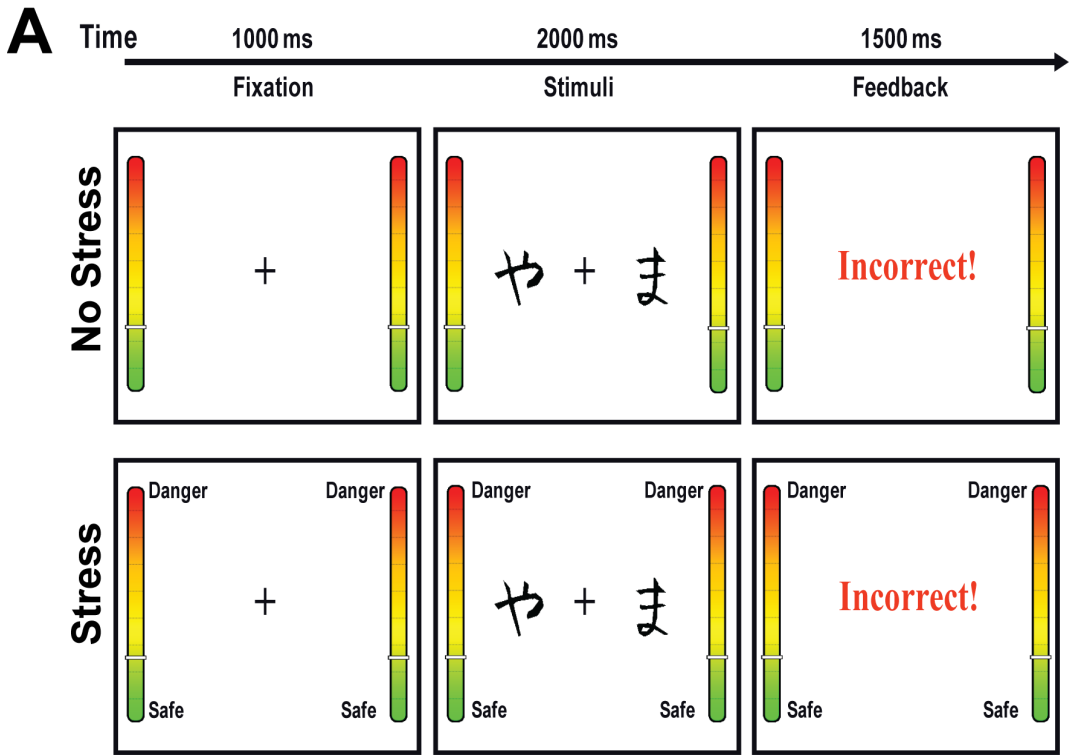
1 Figure 1

2

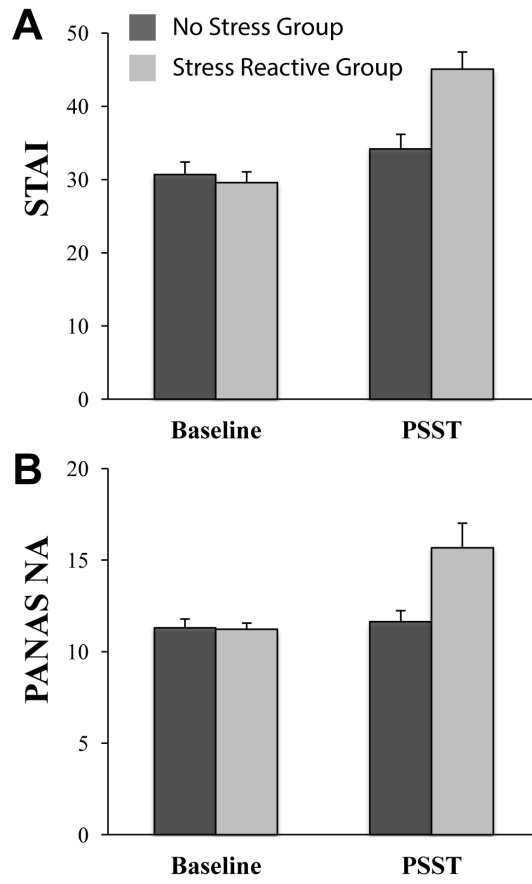


3

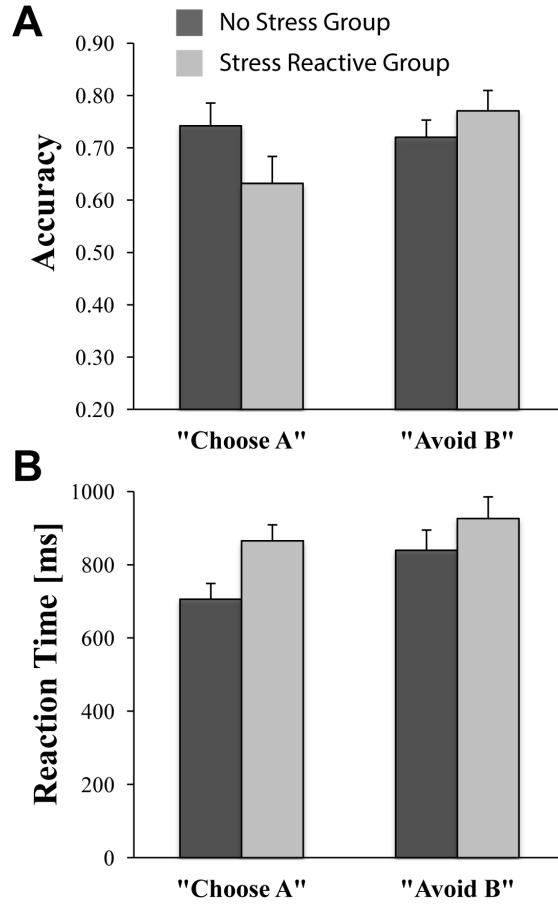
1 Figure 2



1 Figure 3



1 Figure 4



2  
3  
4  
5  
6

# Acute stress selectively reduces reward sensitivity

## Supplement

### Supplemental Description of Measures

#### ***Trait and dispositional self-report measures***

The Beck Depression Inventory-II (BDI-II; Beck *et al.*, 1996) is a 21-item questionnaire used to measure depressive symptoms over the past 2 weeks. It has strong internal reliability (.86-.92), high test-retest reliability over one-week (.93), and good convergent and discriminant validity (Beck *et al.*, 1996; Segal *et al.*, 2008; Steer *et al.*, 2000).

The Mood and Anxiety Symptom Questionnaire (MASQ-short) is a 62-item questionnaire used to assess symptoms of anxiety and depression over the past week with good convergent and discriminant validity in clinical and community samples (Watson *et al.*, 1995); it yields four subscales—general distress anxious, anxious arousal, general distress depressive, and anhedonic depressive.

The Perceived Stress Scale (PSS; Cohen *et al.*, 1983) is a 14-item measure used to assess the degree to which an individual appraises the situations in his or her life as stressful over the past month. Internal reliability coefficients for the PSS range from .84 to .86 with a test-retest reliability of .85 (over two days); the measure has been demonstrated to have strong convergent validity (Cohen *et al.*, 1983).

The Temporal Experience of Pleasure Scale (TEPS; Gard *et al.*, 2006) is a 14-item measure used to assess individual trait dispositions in anticipatory and consummatory experiences of pleasure. The scale has good internal consistency (.71-.79), high test-retest reliability over 5-7 weeks (.75-.81), and strong convergent and discriminant validity (Gard *et al.*, 2006).

The Behavioral Inhibition and Behavioral Activation Scales (BIS/BAS; Carver and White, 1994) are used to measure individual differences in sensitivity to two motivational systems purported to underlie behavior: a behavioral activation system and a behavioral inhibition system. It has good convergent and discriminant validity in community and clinical samples (Carver and White, 1994; Campbell-Sills *et al.*, 2004).

#### ***“In-the-moment” state self-report measures***

The state form of the State Trait Anxiety Inventory (STAI-S) includes 20 items used to quantify state anxiety levels. Internal consistency coefficients range from .86 to .95, while test-retest reliability coefficients (over 2 months) range from .65 to .75 (Spielberger *et al.*, 1983).

The state version of the Positive and Negative Affect Schedule (PANAS) is used to measure current levels of positive and negative affect. Internal consistency coefficients range from .86-.90 for the positive affect scale and .84-.87 for the negative affect scale; test-retest reliability coefficients (over 2 months) range from .47-.68 for the positive affect scale and .39-.71 for the negative affect scale (Watson *et al.*, 1988).

The Challenge-Threat Questionnaire (Mendes *et al.*, 2001) was designed to assess individuals' threat appraisals (perceived resources/demands) of a task, with pre-task and

1 post-task versions. Unfortunately, only 23 ‘controllable stress’ participants and 21  
 2 ‘uncontrollable stress’ participants completed this measure since it was added midway  
 3 through data collection. The pre-task version typically includes 11 statements (e.g., “The  
 4 upcoming task will take a lot of effort to complete,” “I have the abilities to perform the  
 5 upcoming task successfully”) that participants rate on a scale from 1 (“strongly disagree”) to 7  
 6 (“strongly agree”) to indicate how they are feeling about the task they are about to  
 7 complete. The pre-task version used in this study included two additional items to assess  
 8 participants’ perceived control over general task performance, and perceived control over  
 9 whether shocks would occur in the upcoming task. Participants completed the pre-task  
 10 form after receiving PSST instructions but prior to beginning the PSST. The post-task  
 11 version typically includes 9 statements (e.g., “The task was very demanding,” “I felt that  
 12 I had the abilities to perform well in the task”), which participants again rate on a scale  
 13 from 1 (“strongly disagree”) to 7 (“strongly agree”) to indicate how they feel about the  
 14 task they just completed. The post-task version used in this study also included two  
 15 additional items to assess participants’ perceived control over general task performance,  
 16 and perceived control over whether shocks occurred in the task. Participants completed  
 17 the post-task form after finishing the PSST.

18  
 19 **Supplemental Analyses**

20  
 21 All analyses parallel those reported in the main manuscript (*Trait and*  
 22 *dispositional self-report measures; “In-the-moment” state self-report measures; PSST*  
 23 *training phase; PSST test phase*) except they were computed using *Group* with three  
 24 levels (‘no stress,’ ‘controllable stress,’ uncontrollable stress’) in mixed ANOVAs.  
 25

26  
 27 **Supplemental Results**

28  
 29 **Table S1: Characteristics of Participants by the Original 3 Groups**  
 30

	No Stress Group (n = 27)	Controllable Stress Group (n = 34)	Uncontrollable Stress Group (n = 34)	Statistics	p
<b>Gender (% female)</b>	100%	100%	100%	N/A	N/A
<b>Age (years)</b>	21.43 (± 1.79)	21.33 (± 2.24)	21.32 (± 2.20)	F(2,94) = 0.02	0.98
<b>Education (years)</b>	14.81 (± 1.39)	14.44 (± 1.69)	14.26 (± 1.54)	F(2,94) = 0.96	0.39
<b>Marital Status (% single)</b>	100%	91%	94%	$\chi^2(1) = 5.37$	0.25
<b>Income (% &lt;\$50,000)</b>	90%	73%	74%	$\chi^2(1) = 2.29$	0.32
<b>Compensation Form (% monetary)</b>	85%	91%	88%	$\chi^2(1) = 0.53$	0.77

<b>Ethnicity (% Hispanic)</b>	7%	9%	6%	$\chi^2(1) = 0.22$	0.90
<b>Ethnicity (% Caucasian)</b>	85%	44%	74%	$\chi^2(1) = 12.60$	< 0.01
<b>BDI-II Score</b>	1.85 ( $\pm$ 2.38)	2.41 ( $\pm$ 2.52)	2.00 ( $\pm$ 2.16)	F(2,94) = 0.48	0.62
<b>MASQ: GDA</b>	15.52 ( $\pm$ 4.74)	15.50 ( $\pm$ 3.78)	15.82 ( $\pm$ 4.06)	F(2,94) = 0.06	0.94
<b>MASQ: GDD</b>	16.85 ( $\pm$ 5.25)	18.79 ( $\pm$ 5.59)	17.41 ( $\pm$ 4.59)	F(2,94) = 1.18	0.31
<b>MASQ: AA</b>	20.52 ( $\pm$ 4.82)	19.94 ( $\pm$ 4.32)	19.24 ( $\pm$ 2.76)	F(2,94) = 0.79	0.46
<b>MASQ: AD</b>	49.56 ( $\pm$ 10.90)	50.15 ( $\pm$ 10.15)	49.26 ( $\pm$ 11.32)	F(2,94) = 0.06	0.94
<b>Perceived Stress Scale</b>	19.67 ( $\pm$ 6.33)	21.65 ( $\pm$ 5.12)	19.71 ( $\pm$ 6.45)	F(2,94) = 1.18	0.31
<b>TEPS: Anticipatory</b>	64.67 ( $\pm$ 6.68)	65.12 ( $\pm$ 10.20)	64.18 ( $\pm$ 9.46)	F(2,94) = 0.09	0.91
<b>TEPS: Consummatory</b>	48.41 ( $\pm$ 5.56)	50.82 ( $\pm$ 6.04)	50.50 ( $\pm$ 6.17)	F(2,94) = 1.41	0.25
<b>BIS/BAS: Reward Responsiveness</b>	7.48 ( $\pm$ 1.67)	7.65 ( $\pm$ 2.71)	7.38 ( $\pm$ 1.50)	F(2,94) = 0.14	0.87
<b>BIS/BAS: Drive</b>	9.19 ( $\pm$ 1.96)	8.91 ( $\pm$ 2.14)	9.21 ( $\pm$ 2.14)	F(2,94) = 0.20	0.82
<b>BIS/BAS: Fun Seeking</b>	8.04 ( $\pm$ 2.16)	7.82 ( $\pm$ 2.36)	7.74 ( $\pm$ 2.12)	F(2,94) = 0.14	0.87
<b>BIS/BAS: Inhibition</b>	16.00 ( $\pm$ 2.82)	15.15 ( $\pm$ 2.81)	15.65 ( $\pm$ 2.87)	F(2,94) = 0.70	0.50

BDI-II = Beck Depression Inventory-II; MASQ = Mood and Anxiety Symptom Questionnaire; GDA = General Distress Anxious; GDD = General Distress Depressive; AA = Anxious Arousal; AD = Anhedonic Depression; TEPS = Temporal Experience of Pleasure Scale; BIS/BAS = Behavioral Inhibition and Behavioral Activation Scales

### Trait and dispositional self-report measures

There were no significant differences between groups on trait and dispositional self-report measures collected at baseline [all Fs < 2.09, ps > 0.13]; see Table S1.

### “In-the-moment” state self-report measures

**State anxiety.** The mixed ANOVA on STAI-S scores revealed a significant main effect of *Time* [F(1,92) = 65.68, p < 0.01] and, more critically, a *Time x Group* interaction [F(2,92) = 4.72, p = 0.01]; *Group* was not significant [F(2,92) = 2.71, p = 0.07]. Paired t-tests indicated that anxiety increased from baseline to PSST in the ‘controllable stress’ group [t(33) = 5.72, p < 0.01], the ‘uncontrollable stress’ group [t(33) = 6.29, p < 0.01], and the ‘no stress’ group [t(26) = 2.17, p = 0.04]. At baseline, there were no group differences [F(2,94) = 0.22, p = 0.81]. In line with hypotheses, anxiety levels during the

1 PSST were significantly different between groups [ $F(2,94) = 5.04, p < 0.01$ ]. Follow-up  
2 t-tests revealed that participants in both the ‘controllable stress’ [ $t(59) = 2.67, p = 0.01$ ]  
3 and uncontrollable group [ $t(59) = 3.00, p < 0.01$ ] reported significantly higher anxiety  
4 than participants in the ‘no-stress’ group. However, contrary to hypotheses, participants  
5 in the ‘controllable stress’ group did not differ from those in the ‘uncontrollable stress’  
6 group [ $t(66) = -0.24, p = 0.81$ ].

7 **State negative affect.** The mixed ANOVA on PANAS-NA scores also revealed a  
8 significant main effect of *Time* [ $F(1,92) = 16.87, p < 0.01$ ] and a *Time x Group*  
9 interaction [ $F(2,92) = 3.29, p = 0.04$ ]; *Group* was not significant [ $F(2,92) = 2.55, p =$   
10  $0.08$ ]. Paired t-tests indicated that negative affect increased significantly from baseline to  
11 PSST in the ‘controllable stress’ group [ $t(33) = 2.76, p < 0.01$ ] and the ‘uncontrollable  
12 stress’ group [ $t(33) = 3.50, p < 0.01$ ], but not in the ‘no stress’ group [ $t(26) = 0.62, p =$   
13  $0.54$ ]. At baseline, there were no group differences in negative affect [ $F(2,94) = 0.25, p =$   
14  $0.78$ ]. However, negative affect during the PSST was significantly different between  
15 groups [ $F(2,94) = 3.52, p = 0.03$ ]. Follow-up t-tests revealed that participants in both the  
16 ‘controllable stress’ [ $t(59) = 2.02, p < 0.05$ ] and ‘uncontrollable stress’ [ $t(59) = 2.61, p =$   
17  $0.01$ ] groups reported significantly higher negative affect than participants in the ‘no-  
18 stress’ group. However, again contrary to hypotheses, the two stress groups did not differ  
19 in their level of negative affect during the PSST [ $t(66) = -0.85, p = 0.40$ ].

20 **State positive affect.** The mixed ANOVA on PANAS-PA scores revealed a main  
21 effect of *Time* [ $F(1,92) = 18.37, p < 0.01$ ]; the *Time x Group* interaction [ $F(2,92) = 1.50,$   
22  $p = 0.23$ ] and the *Group* main effect [ $F(2,92) = 1.05, p = 0.36$ ] were not significant. All  
23 participants reported a reduction in positive affect from baseline to PSST.

24 **Challenge-threat questionnaire.** Contrary to hypotheses, the ‘controllable stress’  
25 and ‘uncontrollable stress’ groups were not significantly different in their pre-task [ $t(42)$   
26  $= 0.37, p = 0.71$ ] or post-task [ $t(42) = 0.28, p = 0.78$ ] threat appraisals. Moreover, the  
27 two stress groups did not differ in their ratings of control over performance in the task  
28 prior to task onset [ $t(42) = -0.03, p = 0.98$ ] or after completing the task [ $t(42) = 0.33, p =$   
29  $0.74$ ]. In both groups and at both assessments, these ratings were close to “neutral” but  
30 fell slightly on the “disagree” side of the scale ( $< 4$ ) with regard to having control over  
31 their performance.

32 A mixed ANOVA on ratings of perceived control over shock with *Group*  
33 (Uncontrollable Stress, Controllable Stress) as a between-subjects variable and *Time*  
34 (Pre-PSST, Post-PSST) as a within-subjects variable revealed a trend for a *Time x Group*  
35 interaction [ $F(1,42) = 3.42, p = 0.07$ ], with significant main effects of *Time* [ $F(1,42) =$   
36  $29.60, p < 0.01$ ] and *Group* [ $F(1,42) = 45.64, p < 0.01$ ]. On pre-task ratings of control  
37 over shock, the ‘controllable stress’ group was significantly higher than the  
38 ‘uncontrollable stress’ group [ $t(42) = 5.66, p < 0.01$ ], as predicted; however, importantly  
39 and contrary to expectations, both groups again fell in the “disagree” zone of the rating  
40 scale ( $< 4$ ). A paired t-test within the ‘controllable stress’ group indicated that they  
41 reported significantly more control over the shock at their post-task than pre-task rating  
42 [mean increased to  $5.39 \pm 1.62$ ;  $t(22) = 5.51, p < 0.01$ ]; interestingly, the ‘uncontrollable  
43 stress’ group also had a significant increase in level of perceived control over shock from  
44 pre-task to post-task [ $2.43 \pm 1.75$ ;  $t(20) = 2.38, p = 0.03$ ].

45 Overall, findings from the state measures indicate that the ‘threat-of-shock’ stress  
46 manipulation induced significantly higher levels of negative affect and anxiety in both

1 stress conditions than the no-stress condition, but no significant differences between the  
2 two stress groups. Further indications that the stress manipulation was only partially  
3 successful include the following: no significant differences between the two stress groups  
4 on pre-task threat appraisals or perceived control over general task performance, and pre-  
5 task ratings of control over shock were in the “disagree” zone of the scale for both  
6 groups.

### 8 **Cortisol levels**

9 The *Time* (T1 = Baseline, T2 = post-“filler” task/pre-PSST, T3 = post-PSST) x  
10 *Group* ANCOVA on cortisol levels, with “time since waking” as a covariate, revealed  
11 only a significant main effect of *Time* [ $F(2,176) = 11.37, p < 0.01$ ]. Consistent with  
12 cortisol’s diurnal pattern, cortisol levels dropped throughout the experiment [linear effect:  
13  $F(1,88) = 15.14, p < 0.01$ ]. Similarly, a one-way ANOVA comparing groups on cortisol  
14 reactivity scores at T2-T1, and a separate one-way ANOVA comparing groups on  
15 cortisol reactivity scores at T3-T1, yielded insignificant findings [all  $F < 1.78, p > 0.17$ ].  
16 The unpaired t-test comparing the ‘controllable stress’ group with the ‘uncontrollable  
17 stress’ group on cortisol reactivity scores at T3-T1 was not significant [ $t(64) = 0.36, p =$   
18  $0.72$ ], suggesting that both stress conditions yielded physiologically similar responses.

### 20 **PSST training phase**

21 Groups did not differ in the number of completed training blocks [ $F(2,94) = 0.49,$   
22  $p = 0.61$ ]; all groups took approximately 3 blocks to advance to the test phase [no-stress  
23 group:  $3.15 \pm 1.75$ ; controllable stress group:  $3.06 \pm 1.50$ ; uncontrollable stress group:  
24  $3.44 \pm 1.73$ ]. In the ANOVA for accuracy on the final training block with *Trial Type*  
25 (AB, CD, EF) and *Group* as factors, there was only a main effect of *Trial Type* [ $F(2,184)$   
26  $= 14.86, p < 0.01$ ; all other  $F_s < 1.30, p_s > 0.30$ ]; as expected, participants were most  
27 accurate on the AB trial type and least accurate on the EF trial type. No significant  
28 differences emerged from the ANOVA for RT on the final training block [all  $F_s < 1.91,$   
29  $p_s > 0.15$ ]. Altogether, these findings indicate that (1) the probabilistic contingencies  
30 elicited the intended behavioral effects, and (2) groups did not differ in performance  
31 during the training phase.

### 33 **PSST test phase**

34 The ANOVA comparing accuracy on AB trials (the “easiest” trial type) in the test  
35 phase with *Group* confirmed that there were no significant group differences in terms of  
36 participants learning the basic task [ $F(2,94) = 0.62, p = 0.54$ ]. For accuracy, contrary to  
37 hypotheses, the *Trial Type* (“Choose A,” “Avoid B”) x *Group* ANOVA revealed no  
38 significant effects [all  $F_s < 1.59, p_s > 0.21$ ].

39 For RT scores, the analogous *Trial Type* x *Group* ANOVA yielded a significant  
40 main effect of *Trial Type* [ $F(1,92) = 29.73, p < 0.01$ ] and a *Trial Type* x *Group* interaction  
41 [ $F(1,92) = 4.56, p = 0.01$ ]. Follow-up analyses indicated no significant group differences  
42 on “Choose-A” trials or “Avoid B” trials [all  $p_s > 0.058$ ]. Paired t-tests revealed that  
43 participants in the ‘no stress’ and ‘uncontrollable stress’ groups were slower on their  
44 “Avoid B” trials than their “Choose A” trials [no-stress group:  $t(26) = 4.47, p < 0.01$ ;  
45 uncontrollable stress group:  $t(33) = 4.49, p < 0.01$ ]. Participants in the ‘controllable

1 stress' condition, however, exhibited RTs that were not significantly different across trial  
2 types [ $t(33) = 0.72, p = 0.48$ ].

### 3 4 **Supplemental Discussion**

5 Inspired by non-human animal research documenting that uncontrollable stressors  
6 may be particular triggers of anhedonic-like behavior, we attempted to examine whether  
7 stressor controllability moderates the relationship between stress and reward processing  
8 dysfunction. Although the stress manipulation did induce significantly higher levels of  
9 negative affect and anxiety than the no-stress condition, the uncontrollable and  
10 controllable stress manipulations elicited similar affective and cortisol responses, which  
11 was contrary to hypotheses. Notably, these results echoed patterns with self-report  
12 measures indicating that the "controllable stress" group did not actually believe they had  
13 control over the stressor. Accordingly, due to an only partially successful stress  
14 manipulation, conclusions could not be drawn concerning the impact of perceived control  
15 over stress.

16 Contrary to expectations, the two stress groups ('controllable' and  
17 'uncontrollable') did not differ significantly from each other in their levels of anxiety or  
18 negative affect. Cortisol reactivity analyses similarly did not reveal differences between  
19 the 'controllable stress' and 'uncontrollable stress' groups. Moreover, there were no  
20 significant differences between the two stress groups on pre-task threat appraisals  
21 (perceived demands/personal resources) or perceived control over general task  
22 performance. Although pre-task ratings of control over shock were higher in the  
23 'controllable stress' group than the 'uncontrollable stress' group, both groups' ratings fell  
24 in the "disagree" zone of the scale, indicating that prior to task onset, subjects in the  
25 'controllable stress' group did not actually believe that they would have control over the  
26 stressor. This lack of believability may stem from the fact that participants in the  
27 'controllable stress' group were told they would be able to "significantly reduce" the  
28 likelihood of receiving shock by pressing down on the foot pedal, but could not  
29 completely eliminate the possibility of being shocked (i.e., they were not given  
30 "complete" control). Task instructions were outlined this way because of concerns that  
31 the latter set of instructions would not induce significantly more stress than the no-stress  
32 condition. Collectively, these data suggest that the stress manipulation was only partially  
33 successful: significantly more negative affect and anxiety was reported by participants in  
34 both stress groups relative to the 'no-stress' group, but the controllability manipulation  
35 was not successful.

36 Results from this aspect of the experiment serve to highlight key variables to  
37 consider in the design of future experiments. For example, the importance of  
38 administering an assessment of perceived control over stress *prior* to task onset and  
39 collecting data on a physiological index of stress (e.g., cortisol levels) to confirm the  
40 effects of any stress manipulation on participants. Moreover, given that participants in  
41 our 'controllable' stress condition (who were told they had 'partial' control over the  
42 stressor) did not report truly believing they had control over the stressor, future designs  
43 warrant including a 'controllable stress' condition in which participants are given  
44 perceived *full* control over the stressor.

45