The Effect of a Primary Sexual Reward Manipulation on Cortisol Responses to Psychosocial Stress in Men

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Objective: Although previous research provides evidence for the role of rewarding activities in reducing hypothalamic-pituitary-adrenal axis responses to stress, no studies have tested whether rewards can buffer cortisol responses in humans undergoing social stressors. **Method:** This study experimentally investigated whether viewing appetitive rewarding pictures reduces cortisol responses to an acute stress challenge. Fifty-four heterosexual men were randomly assigned to view either mildly erotic (reward) or neutral images (control) of mixed-sex couples before completing the Trier Social Stress Test (TSST). **Results:** Participants in the reward condition had significantly lower area-under-the-curve cortisol reactivity to the TSST (mean [M] = 363.46) in comparison with participants in the control group (M = 807.06; $F(1,46) = 4.84$, $p = .033$, $\eta^2 = 0.095$). Reward participants also had improved cognitive performance on the math portion of the TSST (M = 20.74) in comparison with control participants (M = 13.82; $F(44) = 5.44$, $p = .024$, $\eta^2 = 0.11$). The stress-buffering effects of reward were specific to hypothalamic-pituitary-adrenal axis reactivity: the reward and control groups did not differ on psychological perceptions of anticipatory or poststress perceptions, heart rate, or blood pressure responses. **Conclusions:** This research provides the first evidence linking the experience of reward with reduced stress reactivity in humans and suggests a potential novel reward pathway for coping under stress. **Key Words:** reward, stress, Trier Social Stress Test, HPA axis.

SBP = systolic blood pressure; **DBP** = diastolic blood pressure; **MAP** = mean arterial pressure; **TSST** = Trier Social Stress Test; **RSE** = Rosenberg Self-Esteem Scale.

INTRODUCTION

Activation of the hypothalamic-pituitary-adrenal (HPA) axis during acute stress is considered to be important for mobilizing effective fight or flight responses, although studies indicate that lack of or excessive HPA-axis activation during laboratory stressors is associated with increased mental and physical health risks (1). Greater cortisol reactivity to laboratory stress has been linked to an increased risk for upper respiratory tract infections (2), central adiposity (3), and depressive symptoms (4). For example, Cohen and colleagues (2) showed that greater cortisol reactivity to a laboratory speech task was associated with a greater incidence of upper respiratory tract infections during a 12-week high-stress follow-up period. Despite these findings, little experimental work has considered psychosocial factors that can moderate cortisol responding to acute stressors. Most research to date has focused on examining how individual differences (e.g., perfectionism, mindfulness, and self-esteem) covary with cortisol reactivity in cross-sectional studies (e.g., Refs. (5–7)) or how anticipatory stress relates to salivary cortisol reactivity and recovery (8). Some promising initial evidence from experimental studies using random assignment shows that psychosocial factors may causally modulate cortisol responses. For example, thinking about an important personal value before acute stress or receiving social support during acute stress can reduce cortisol reactivity (9,10). One interesting possibility raised by this experimental work is that these psychosocial manipulations may reduce cortisol reactivity via a basic reward pathway, although no studies have tested whether rewards can buffer stress responses in humans.

The purpose of the present study was to experimentally evaluate whether a primary sexual reward can reduce cortisol reactivity in men. We elected to use a sexual reward manipulation, given that this image-viewing approach has well-defined psychological and physiological reward response properties in men (e.g., Refs. (11,12)) and erotic visual stimuli are robust activators of reward neurocircuitry (13). There are several lines of evidence that support a reward-cortisol stress-buffering effect. First, it is well known that rewarding activities increase endogeneous opioid release (14), and acute administration of opioids suppresses HPA-axis activity (15). Second, there are reciprocal links between stress and reward pathways—acute stress has been shown to blunt subsequent reward responding (16). Third, studies have shown that primary rewards promote analgesia to stimulated pain (17–19). Finally, perhaps the most suggestive evidence for this reward–stress-buffering link is a recent study in rodents showing that rewarding environments can buffer HPA-axis reactivity to restraint stress (20). Specifically, male rats exposed to 14 consecutive days of reward (consisting of access to a sexually receptive female or sucrose treats) showed blunted corticosterone responses to restraint stress on day 15 compared with control rats. Notably, these effects were shown even when using a calorie-free sweet saccharin drink, suggesting that the experience of a palatable reward is sufficient for stress-dampening effects (20).

Building on this emerging body of work suggesting that rewards can reduce HPA-axis reactivity, we hypothesized that a brief primary sexual reward activity (i.e., viewing erotic images) before an acute stress challenge (the Trier Social Stress Test, or TSST) (21) would reduce cortisol reactivity to this acute stress challenge task. Second, if rewards reduce cortisol responses to the TSST, they may also lead to improved task performance. Thus, a secondary aim of this study was to examine whether experiencing rewards before a stress challenge would improve math performance on the TSST.

METHODS

Participants

Fifty-four healthy heterosexual men, all undergraduate students, were recruited by paper flyers from the campus community. Inclusion criteria for

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Received for publication August 6, 2012; revision received January 27, 2013.
DOI: 10.1097/PSY.0b013e31828c4524

0033-3174/13/7504–0397

TABLE 1. Baseline Characteristics of Sample

Characteristic	Reward Condition (n = 22)	Control Condition (n = 28)	Difference Statistic
Age, M (SD), y	21.73 (3.28)	21.36 (2.41)	$t(48) = -0.46$, $p = .65$
Ethnicity			$\chi^2(4) = 4.49$, $p = .34$
African American	4	1	
Latino/Hispanic	2	4	
White	5	11	
Asian American	5	7	
Other	6	5	
Weight, M (SD), lb	162.23 (28.17)	162.86 (40.16)	$t(48) = 0.06$, $p = .95$
Experimental session time of day (2 PM = 0–7 PM = 5), M (SD)	1.89 (1.50)	1.56 (1.17)	$t(48) = -0.86$, $p = .40$
Do you smoke? (yes/no)			$\chi^2(1) = 0.64$, $p = .43$
Yes	1	3	
No	21	25	
So far today, how many cigarettes have you smoked? M (SD)	0.05 (0.21)	0.39 (1.89)	$t(48) = 0.86$, $p = .40$
So far today, how many cups of coffee did you have? M (SD)	0.36 (0.58)	0.61 (0.88)	$t(48) = 1.18$, $p = .25$
In the past hour, have you had a cup of coffee or other caffeinated drink? (yes/no)			$\chi^2(1) = 0.06$, $p = .80$
Yes	2	2	
No	20	26	
Today have you taken any prescription drugs? (yes/no)			$\chi^2(1) = 0.80$, $p = .37$
Yes	0	1	
No	22	27	
Did you eat breakfast today? (yes/no)			$\chi^2(1) = 0.41$, $p = .52$
Yes	9	14	
No	13	14	
Do you become easily sexually aroused? (1 = never to 5 = always), M (SD)	3.55 (0.74)	3.39 (0.50)	$t(48) = -0.83$, $p = .41$
Self-esteem (RSE) composite score, M (SD)	32.14 (5.09)	31.96 (5.28)	$t(47) = -0.12$, $p = .91$
Anhedonia (SHAPS composite) score, M (SD)	1.09 (1.85)	1.32 (1.63)	$t(48) = 0.47$, $p = .64$
Social support (ISEL composite), M (SD)	38.47 (4.43)	38.86 (5.54)	$t(48) = 0.27$, $p = .79$
Life event stress over the last 6 mo (overall LES score), M (SD)	15.00 (10.73)	11.46 (6.43)	$t(48) = -1.37$, $p = .18$

M = mean; SD = standard deviation; RSE = 10-item Rosenberg Self-Esteem Scale; SHAPS = Snaith-Hamilton Pleasure Scale; ISEL = 12-item Interpersonal Support Evaluation List; LES = Life Experiences Survey.
Independent-samples t tests and χ^2 tests determined whether there were significant differences in baseline characteristics between our reward and control groups.

study participation consisted of being a heterosexual man, between the ages of 18 and 30 years, mentally and physically healthy (i.e., no medical diagnosis of any ongoing disease), and willing to view erotic images one might see in an "R" rated film. We excluded four participants who did not complete the study because they either did not wish to view erotic imagery (n = 1) or did not feel comfortable participating in the stress task (n = 3). Study participants (n = 50) had a mean (M; [standard deviation]) age of 21.4 (2.8) years, and the ethnic breakdown consisted of 30% white, 11% African American, 27% Asian American, 11% Latino, and 21% other. All participants were heterosexual (not homosexual or bisexual). All study procedures were approved by the Carnegie Mellon Institutional Review board. All study data were collected between March and November 2011.

Procedure

All participants completed the experimental tasks between 2 and 7 PM, which provided a control for diurnal variation. Upon arriving, participants were informed that they would complete two unrelated studies (one was about understanding physiological responses to performance tasks and the other was about rating a new stimulus set of images). Participants then provided written informed consent, confirmed their study eligibility and willingness to view erotic images, completed individual difference measures, and were fitted with a blood pressure cuff on the brachial artery of their nondominant arm for baseline measurement of blood pressure and heart rate (HR). Thirty-five minutes after arriving for the experimental session, a baseline saliva sample was collected. Participants then heard prerecorded instructions explaining the upcoming speech performance activity and were given 5 minutes to mentally prepare. The participant was informed that the performance evaluators were running late and asked if they would be willing to complete the second unrelated pilot study (the images task) while waiting for the evaluators (all participants agreed). The purpose of this procedure was to minimize any demand characteristics or participant expectancies that could arise if participants made a link between the reward task and the subsequent stress task. Participants were randomly assigned to view either mildly erotic images (reward condition) or view neutral images of mixed-sex couples (control condition), and the experimenter remained blind to participant condition. As a manipulation check, participants were asked to provide ratings of their sexual arousal to each image (and were told that they were doing this so that the images could be evaluated for use in a future experiment). The images in both conditions consisted of 30 images of heterosexual couples that were presented in a random order to participants (each image was presented for 5 seconds). The images in the reward condition consisted of partially nude couples engaging in sexual behaviors, whereas the images in the control condition consisted of couples engaging in day-to-day behaviors (e.g., drinking wine together).

After viewing the set of images, participants completed an anticipatory stress appraisal measure, which took approximately 3 minutes to administer, and then completed a 5-minute speech addressing why they would be a good

administrative assistant for a hypothetical job in the psychology department, followed by 5 minutes of difficult mental arithmetic (i.e., counting backwards from 2083 by 17s) in front of two evaluators trained to be cold and nonaccepting. The evaluators interrupted participants during their speech to ask questions and pointed out mistakes during the arithmetic task, after which participants were instructed to restart counting from 2083. Participants were instructed on several occasions by the evaluators to sit as still as possible in the chair and to maintain eye contact with the evaluators throughout the speech and arithmetic tasks. Our stress challenge activities were designed to follow procedures used in the TSST (21), which robustly elicits cardiovascular and HPA-axis activation. To assess cognitive math performance, the evaluators (who were blind to participant study condition) recorded how many consecutive numbers the participant recited correctly without a mistake during the arithmetic task.

The participants completed a final set of questionnaires, including their poststress perceptions. Saliva samples were acquired at 25 and 35 minutes after the start of the performance task (22). Participants were then probed for suspicion of the evaluators and any connection between the images and performance tasks and were fully debriefed. A final saliva sample was taken at the conclusion of the study, 60 minutes after the start of the performance task.

Measures

Questionnaires

Anticipatory and posttask stress were assessed using the Primary Appraisal–Secondary Appraisal questionnaire (23). This 16-item measure assesses stress appraisals, including perceptions of threat (study α = .86), challenge (study α = .47), self-concept of abilities (study α = .79), control expectancies (study α = .45), primary appraisal (e.g., "I find this situation very unpleasant"; study α = .79), and secondary appraisal (e.g., "In this situation I know what I can do"; study α = .70), and a composite stress index.

State positive and negative affect scores were computed from participants' ratings on the 10-item positive (α = .85) and negative affect (α = .90) scales of the Positive and Negative Affect Schedule (24). As an additional test of our primary sexual reward manipulation, we also included an additional item embedded in this list of affect adjectives, "aroused sexually," which was scored separately as a single-item measure of sexual arousal. On the affect measures, participants rated the extent to which they were experiencing each feeling at the present moment from 1 (very slightly or not at all) to 5 (extremely).

To test for success of randomization, we included several individual difference measures. These included a measure of trait self-esteem using the Rosenberg Self-Esteem Scale (α = .87) (25), a measure of anhedonia (inability to experience pleasure) using the Snaith-Hamilton Pleasure Scale (α = .69) (26), a measure of social support using the Interpersonal Support Evaluation List (α = .60) (27), and life event stress using the Life Experiences Survey (total positive and [absolute valued] negative life event perceptions were summed into a composite life change score) (28).

Cortisol and Cardiovascular Measures

Salivary cortisol was collected using a Salivette (Rommelsdorf, Germany). All Salivettes were frozen at −20°C in a locked and secure laboratory freezer. Participants kept the Salivette under their tongue for 2 minutes during each collection period and did not touch the sample with their hands. At the conclusion of the experiment, the samples were shipped on dry ice to a professional laboratory in Dresden, Germany, specializing in cortisol measurement. At this laboratory, cortisol was measured using a chemoluminescence immunoassay with high sensitivity (IBL International, Hamburg, Germany). Intra-assay and interassay coefficients of variation were below 10%. A measure of oscillometric blood pressure was collected using an automatic sphygmomanometer (Dinamap Carescape V100; General Electric Company, GE, Helsinki, Finland). Systolic (SBP) and diastolic (DBP) blood pressure and HR were recorded with this device at 2-minute intervals. Participants remained seated throughout the collection of all physiological measures.

Statistical Analysis

All analyses of the present data were conducted using the SPSS 19.0 software package (IBM, Armonk, NY). The statistical tests used to analyze the data consisted of *t* tests, between-participant analyses of covariance (ANCOVAs) and mixed-model analyses of variance and ANCOVAs. For primary results, η^2 effect size statistics were calculated and reported. A total area under the curve with respect to increase (AUC-I) cortisol measure was calculated using the trapezoid formula of Pruessner et al. (29), using the following equation: AUC_I = ((Cort_T2 + Cort_T1)/2 * 45 + (Cort_T3 + Cort_T2)/2 * 25 + (Cort_T4 + Cort_T3)/2 * 10) − (Cort_T1 * (45 + 25 + 10)). Note that the AUC-I result subtracts out the baseline (or ground) cortisol level, leaving area under the curve from the TSST-driven cortisol increase.

RESULTS

Preliminary Analyses

To test the success of randomization, we compared our reward and control groups on measured characteristics before randomization. As shown in Table 1, our randomization was successful because there were no significant differences between the two groups. As a manipulation check, we tested whether participants rated the reward images as more sexually arousing compared with the control images, which was confirmed ($t(45) = -8.48$, $p < .001$; control M [standard error{SE} = 1.42 [0.11] of 5, reward M [SE] = 3.02 [0.16]). We also tested whether reward participants had increases in sexual arousal ratings from before to after viewing the images, compared with participants in the control condition. As expected, we found that participants in the reward condition had increases in self-reported sexual arousal before and after viewing of the images, compared with control participants (condition × time interaction: $F(1,47) = 20.29$ $p < .001$, $\eta^2 = 0.30$) (Table 2). The study manipulation effects were specific to sexual reward; we did not observe a significant condition × time interaction for change in general state positive ($p = .31$) or negative ($p = .52$) affect between the reward and the control groups (see Table 2).

Cortisol and Cardiovascular Measures

To control for diurnal variation, we ran all study sessions between 2 and 7 PM, and as an additional control, all cortisol analyses included time of day of the first saliva sample as a covariate. We first conducted a one-way ANCOVA testing for condition differences on total AUC-I cortisol responses,

TABLE 2. Sexual Arousal, Positive Affect, and Negative Affect ANOVA Means and SEs

	Mean Reward	SE	Mean Control	SE	*F*	*p*
Sexual arousal						
Preimages	1.57	0.201	1.57	0.174		
Postimages	2.62	0.225	1.50	0.195	$F(1,47) = 20.29$	<.001
Positive affect						
Preimages	29.38	1.779	27.82	1.541		
Postimages	28.33	1.908	25.21	1.652	$F(1,47) = 1.04$	.31
Negative affect						
Preimages	13.48	1.150	15.04	0.996		
Postimages	16.48	1.384	16.96	1.198	$F(1,47) = 0.42$	.52

ANOVA = analyses of variance; SE = standard error.
Scores on each item can range from 1(very slightly or not at all) to 5 (extremely). Positive and negative affect scores are derived from the Positive and Negative Affect Schedule.

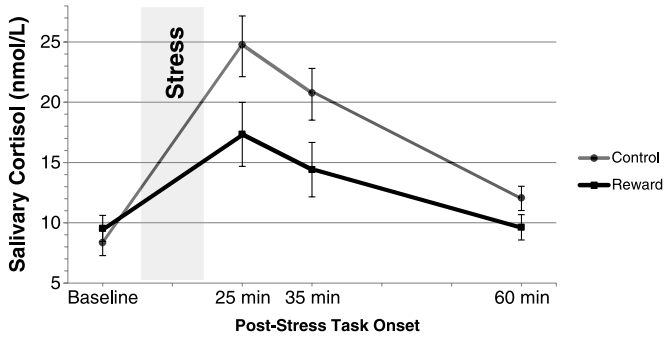

Figure 1. Salivary cortisol levels in the reward and control groups. Error bars reflect SEs around the mean. Pairwise differences between groups were significant at the $p = .057$ level at both the 25- and 35-minute sample time points. SE = standard error.

controlling for time of day. Consistent with our primary study prediction, we found that participants in the reward group (M [SE] = 363.46 [149.17] nM) had a significantly lower total cortisol response to the TSST compared with the control group participants (M [SE] = 807.06 [134.54] nM) ($F(1,46) = 4.84$, $p = .033$, $\eta^2 = 0.095$). We primarily used an all-female evaluative panel during the speech/math tasks, but there were instances where we used a mixed-sex panel. We note that the reward manipulation effect was also significant when we further controlled for the sex composition of our evaluative panel ($F(1,44) = 4.28$, $p = .045$, $\eta^2 = 0.09$). To further probe this cortisol effect, we conducted a repeated-measures ANCOVA, and as expected, we observed a significant experimental condition × time interaction on cortisol responses ($F(1,46) = 4.32$, $p = .006$, $\eta^2 = 0.086$) (Fig. 1). As shown in Figure 1, this interaction was driven by differences in peak cortisol reactivity to the TSST; planned pairwise comparisons showed that participants in the reward group had lower peak cortisol responses at 25 minutes ($M_{difference} = 7.01$ nM, $p = .057$) and at 35 minutes poststress task onset ($M_{difference} = 6.03$ nM, $p = .054$). Our randomization procedure equalized the reward and control groups, but as an additional test, we used each measured variable in Table 1 as a covariate in a series of repeated-measures ANCOVAs (as described previously), and their inclusion did not appreciably affect the observed experimental condition × time interaction on cortisol responses (all p values remained significant at <.05).

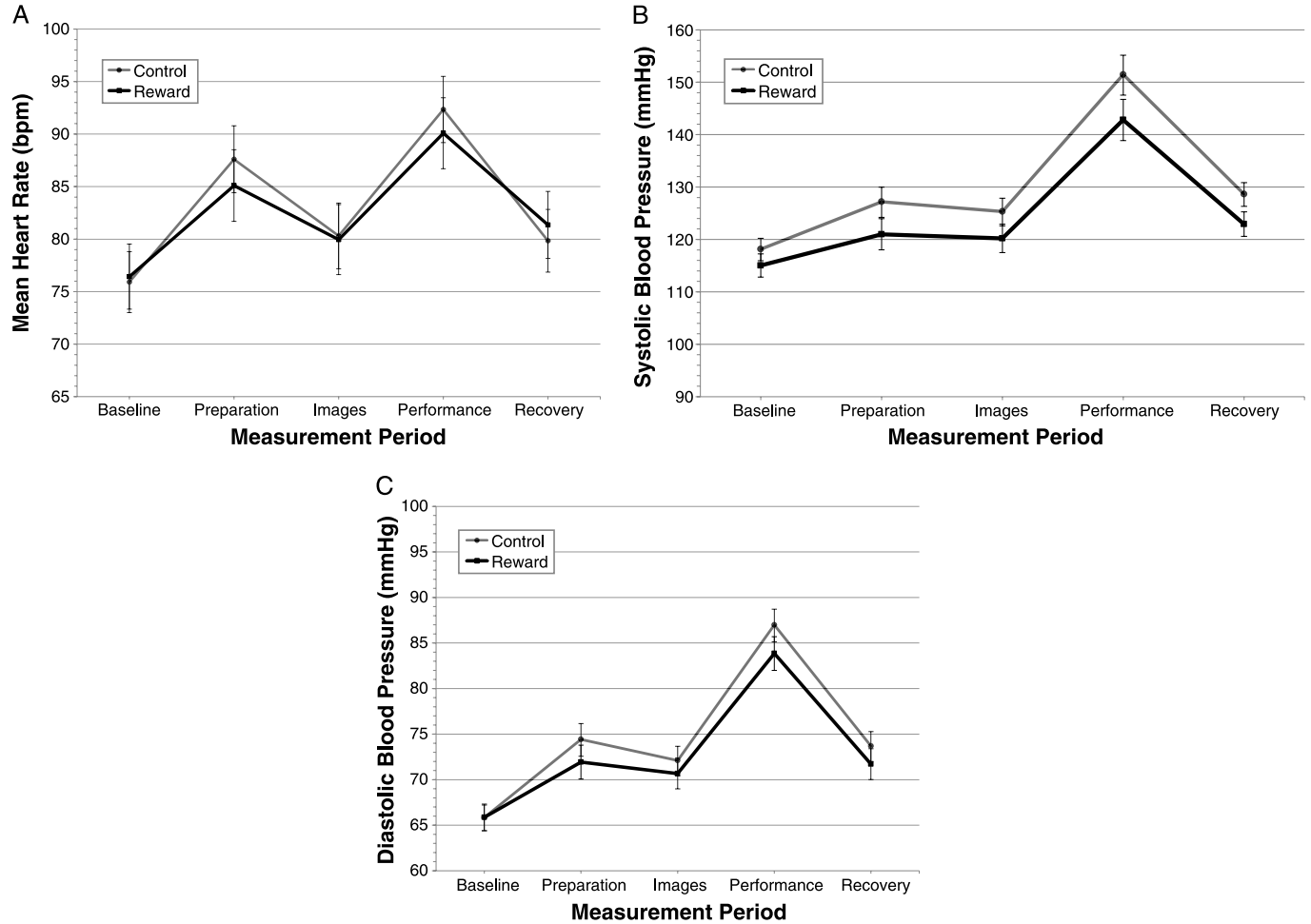

Figure 2. A, Average heart rate during the baseline, preparation, reward manipulation, stress, and recovery periods in the reward and control groups. Error bars reflect SEs around the mean. B, Average systolic blood pressure during the baseline, preparation, reward manipulation, stress, and recovery periods in the reward and control groups. Error bars reflect SEs around the mean. C, Average diastolic blood pressure during the baseline, preparation, reward manipulation, stress, and recovery periods in the reward and control groups. Error bars reflect SEs around the mean. SE = standard error; bpm = beats per minute.

TABLE 3. Anticipatory Stress Appraisal *t* Test Analyses

	Mean Reward	SE	Mean Control	SE	*t*	*p*
Threat						
Anticipatory Stress	12.95	1.09	13.36	0.869	$t(47) = 0.29$	.77
Poststress Perceptions	13.86	0.646	14.46	0.899	$t(48) = 0.54$	.59
Challenge						
Anticipatory Stress	14.33	0.698	14.79	0.530	$t(47) = 0.53$	.60
Poststress Perceptions	15.27	0.600	15.54	0.788	$t(48) = 0.27$	.79
Self-efficacy						
Anticipatory stress	15.33	0.922	15.21	0.643	$t(47) = -0.11$	.91
Poststress Perceptions	14.68	0.659	13.71	0.720	$t(48) = -0.97$	.34
Control expectancy						
Anticipatory Stress	16.52	0.572	15.75	0.536	$t(47) = -0.98$	.33
Poststress perceptions	15.09	0.699	13.64	0.841	$t(48) = -1.28$	.21
Primary appraisal						
Anticipatory stress	27.29	1.639	28.14	1.130	$t(47) = 0.45$	.66
Poststress perceptions	29.14	0.985	30.00	1.502	$t(48) = 0.48$	.63
Secondary appraisal						
Anticipatory Stress	31.86	1.098	30.96	1.042	$t(47) = -0.58$	.56
Poststress perceptions	29.77	1.164	27.36	1.304	$t(48) = -1.34$	.19
Stress index						
Anticipatory stress	−4.57	2.387	−2.82	1.679	$t(47) = 0.62$	.54
Poststress perceptions	−0.64	1.484	2.64	2.125	$t(48) = 1.20$	.24

SE = standard error.

We also tested whether the reward buffered HR, SBP, and DBP responses to stress. Although we observed significant increases in HR ($F(1,43) = 34.96$, $p = <.001$, $\eta^2 = 0.448$), SBP ($F(1,43) = 99.71$, $p = <.001$, $\eta^2 = 0.699$), and DBP ($F(1,43) = 102.13$, $p = <.001$, $\eta^2 = 0.70$) over time in both groups, these differences were not significant between conditions (see Fig. 2, A–C).

Anticipatory Stress Appraisal Analyses

It is possible that the effects of experimental rewards on cortisol responses may occur by modulating psychological appraisals in anticipation of, or in response to, the stress challenge task. To evaluate this, participants completed a measure of anticipatory stress appraisal after completing the reward task (and immediately before starting the stress challenge task), as well as a poststress perceptions measure after completing the stress challenge. As shown in Table 3, we found no evidence that the reward manipulation affected psychological appraisals of stress at either time point.

Stress Task Performance Analyses

Finally, to evaluate whether reward boosts cognitive performance under stress, we conducted a one-way ANCOVA comparing the number of digit responses recited correctly during the calculation portion of the math task, again controlling for time of day. Reward boosted cognitive performance. Specifically, the reward group (M [SE] = 20.74 [2.23] numbers recited) recited significantly more numbers correctly than did participants in the control group (M [SE] = 13.82 [1.92] numbers recited) ($F(44) = 5.44$, $p = .024$, $\eta^2 = 0.11$). We note that one participant whose arithmetic performance was more than 2 standard deviations above the mean was excluded from this analysis. (This participant was in the reward condition and would increase the magnitude of the reward performance effect if he were included.)

DISCUSSION

The present study indicated that a brief primary sexual reward task buffered cortisol responses and improved cognitive performance on a demanding social stress task. Although previous work suggests that rewarding environments can reduce HPA-axis activity to restraint stress in rodents (20), our work demonstrates this effect for the first time in human male volunteers. Moreover, our findings suggest that the cortisol-buffering effects of rewards can occur in response to even a brief experimental reward manipulation. One potential implication of these results is that rewarding activities may be a promising approach for proactively coping with upcoming stressors (30). However, it is important to note that we did not assess reward as a deliberate coping strategy for managing an upcoming stressor in this study; participants in the present study were not aware of any link between the reward task and the stress challenge performance tasks (they were told that the reward task was for a separate unrelated pilot study). We elected to use this procedure to eliminate a potential procedural confound in our study. Specifically, informing participants of an explicit link between the two tasks may have produced positive expectancies

for stress-buffering effects in the reward condition (e.g., Ref. (31)).

A notable strength of the present study was our experimental approach in evaluating the causal relationship between reward and subsequent cortisol reactivity to the TSST. We carefully matched our rewarding stimuli in both groups such that participants viewed the same number of couples in both picture sets; thus, it is unlikely that we inadvertently primed social support (a potential alternative explanation) (cf. Ref. (32)). In addition, it is possible that participants in the reward group simply were more distracted during the stress challenge tasks than participants in the control group. However, our performance data do not support this explanation. If participants in the reward condition were distracted, then they should have had worse performance on the math portion of the TSST, when, in fact, participants in the reward condition had better math performance than did those in the control condition. The improved cognitive (math) performance that we observed in our reward group suggests that our primary sexual reward activity may buffer social evaluative threat, reduce anxiety-related arousal, and boost problem-focused coping during the performance task, although we note that these hypotheses are speculative and should be tested in future studies.

An important future research direction will be to determine the biopsychosocial pathways linking rewards with reduced HPA-axis activation during acute stress. We did not observe evidence for a psychological appraisal or affect mechanism. Specifically, we observed no significant condition differences on anticipatory or poststress appraisals, nor did we find condition differences in state positive affect or negative affect. Rather, our effects were specific to a sexual reward pathway. In the present study, participants indicated being more sexually aroused in the reward condition compared with the control condition after viewing the images. This primary reward pathway suggests several candidate neurobiological pathways that may explain how rewards reduce cortisol reactivity to the TSST. First, it is possible that our reward task activated the HPA axis (33), resulting in negative feedback inhibition of the HPA axis during the subsequent TSST challenge tasks (34) (cf. Refs. (35,36)). Similarly, it may be that rewards trigger other neuroendocrine cascades that can inhibit HPA-axis activity. For example, rewarding stimuli can increase circulating levels of testosterone (37) and oxytocin (38), and both hormones have been linked with HPA-axis inhibition (39–42). We did not find strong evidence for reward effects on markers of autonomic nervous system activity (HR, blood pressure), but sympathetic nervous system pathways may be important mechanisms for reward effects on stress (e.g., Refs. (12,20)). Finally, it may be that rewards buffer cortisol responses via an endogenous opioid HPA-axis inhibition pathway. Rewarding activities can activate endogenous opioid neurotransmission, and opioids are known to suppress HPA-axis activity (15). Evaluating each of these candidate neurobiological reward-stress pathways experimentally is an exciting direction for new studies. For example, if these reward-cortisol effects are explained by an endogenous opioid pathway, then administering a nonspecific opioid receptor antagonist (e.g., naloxone) should eliminate the effects of rewards on buffering cortisol responses to acute stress.

Our study provides an initial demonstration of the stress-buffering effects of a primary sexual reward and suggests new questions for future research. First, we have offered some initial discussion about potential candidate pathways linking rewards with their stress-buffering effects, but more research is needed to test these mechanistic hypotheses. Second, it is currently unknown whether all rewards buffer cortisol responses to stress. Our work described here used a primary reward (sex), but more research is needed to test whether other primary rewards (e.g., appetitive foods) or secondary rewards (e.g., money) can produce comparable effects in human volunteers. (Consistent with the idea that secondary rewards buffer stress, a previous study showed that reflecting on an important personal value in a self-affirmation activity reduced cortisol responses to the TSST (9)). Also, it will be important to test whether the psychological perception of a reward stimulus (e.g., learning, wanting, or liking) is critical for stress-buffering effects (43). Our initial approach was to first to demonstrate this effect in men, who show robust reward responses to erotic visual stimuli (e.g., Ref. (44)). Future studies should determine whether these effects extend to women.

CONCLUSIONS

Our work provides an initial indication that rewarding activities can reduce cortisol reactivity and boost cognitive performance during acute stressors. This finding may have applied implications for helping people use rewarding activities to cope with upcoming stressors, and this research suggests new directions for exploring basic brain-reward pathways in modulating HPA-axis responses to stress.

We thank the research assistants in the Health and Human Performance Laboratory for their help and feedback.

Source of Funding and Conflicts of Interests: This research was supported by the Pittsburgh Life Sciences Greenhouse Opportunity Fund and National Science Foundation Grant #BCS-0924387. The authors report no conflicts of interests.

REFERENCES

1. Foley P, Kirschbaum C. Human hypothalamus-pituitary-adrenal axis responses to acute psychosocial stress in laboratory settings. Neurosci Biobehav Rev 2010;35:91–6.
2. Cohen S, Hamrick N, Rodriguez MS, Feldman PJ, Rabin BS, Manuck SB. Reactivity and vulnerability to stress-associated risk for upper respiratory illness. Psychosom Med 2002;64:302–10.
3. Epel ES, McEwen B, Seeman T, Matthews K, Castellazzo G, Brownell KD, Bell J, Ickovics JR. Stress and body shape: stress-induced cortisol secretion is consistently greater among women with central fat. Psychosom Med 2000;62:623–32.
4. Heim C, Newport DJ, Heit S, Graham YP, Wilcox M, Bonsall R, Miller AH, Nemeroff CB. Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. JAMA 2000;284:592–7.
5. Seeman TE, Berkman LF, Gulanski BI, Robbins RJ, Greenspan SL, Charpentier PA, Rowe JW. Self-esteem and neuroendocrine response to challenge: MacArthur studies of successful aging. J Psychosom Res 1995;39:69–84.

6. Wirtz PH, Elsenbruch S, Emini L, Rüdisüli K, Groessbauer S, Ehlert U. Perfectionism and the cortisol response to psychosocial stress in men. Psychosom Med 2007;69:249–55.
7. Brown KW, Weinstein N, Creswell JD. Trait mindfulness modulates neuroendocrine and affective responses to social evaluative threat. Psychoneuroendocrinology 2012;37:2037–41.
8. Juster R-P, Perna A, Marin M-F, Sindi S, Lupien SJ. Timing is everything: Anticipatory stress dynamics among cortisol and blood pressure reactivity and recovery in healthy adults. Stress (Amsterdam, Netherlands) [Internet]. March 14, 2012; Available at: http://www.ncbi.nlm.nih.gov/pubmed/22296506. Cited July 31, 2012.
9. Creswell JD, Welch WT, Taylor SE, Sherman DK, Gruenewald TL, Mann T. Affirmation of personal values buffers neuroendocrine and psychological stress responses. Psychol Sci 2005;16:846–51.
10. Ditzen B, Neumann ID, Bodenmann G, Von Dawans B, Turner RA, Ehlert U, Heinrichs M. Effects of different kinds of couple interaction on cortisol and heart rate responses to stress in women. Psychoneuroendocrinology 2007;32:565–74.
11. Rowland DL, Heiman JR, Gladue BA, Hatch JP, Doering CH, Weiler SJ. Endocrine, psychological and genital response to sexual arousal in men. Psychoneuroendocrinology 1987;12:149–58.
12. Exton NG, Chau Truong T, Exton MS, Wingenfeld SA, Leygraf N, Saller B, Hartmann U, Schedlowski M. Neuroendocrine response to film-induced sexual arousal in men and women. Psychoneuroendocrinology 2000;25: 187–99.
13. Hamann S, Herman RA, Nolan CL, Wallen K. Men and women differ in amygdala response to visual sexual stimuli. Nat Neurosci 2004;7:411–6.
14. Van Ree JM, Niesink RJ, Van Wolfswinkel L, Ramsey NF, Kornet MM, Van Furth WR, Vanderschuren LJ, Gerrits MA, Van den Berg CL. Endogenous opioids and reward. Eur J Pharmacol 2000;405:89–101.
15. Oswald LM, Wand GS. Opioids and alcoholism. Physiol Behav 2004;81: 339–58.
16. Bogdan R, Pizzagalli DA. Acute stress reduces reward responsiveness: implications for depression. Biol Psychiatry 2006;60:1147–54.
17. Szechtman H, Hershkowitz M, Simantov R. Sexual behavior decreases pain sensitivity and stimulates endogenous opioids in male rats. Eur J Pharmacol 1981;70:279–85.
18. Forsberg G, Wiesenfeld-Hallin Z, Eneroth P, Södersten P. Sexual behavior induces naloxone-reversible hypoalgesia in male rats. Neurosci Lett 1987;81:151–4.
19. Leknes S, Tracey I. A common neurobiology for pain and pleasure. Nat Rev Neurosci 2008;9:314–20.
20. Ulrich-Lai YM, Christiansen AM, Ostrander MM, Jones AA, Jones KR, Choi DC, Krause EG, Evanson NK, Furay AR, Davis JF, Solomon MB, de Kloet AD, Tamashiro KL, Sakai RR, Seeley RJ, Woods SC, Herman JP. Pleasurable behaviors reduce stress via brain reward pathways. Proc Natl Acad Sci U S A 2010;107:20529–34.
21. Kirschbaum C, Pirke KM, Hellhammer DH. The "Trier Social Stress Test"—a tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology 1993;28:76–81.
22. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychol Bull 2004;130:355–91.
23. Gaab J, Rohleder N, Nater UM, Ehlert U. Psychological determinants of the cortisol stress response: the role of anticipatory cognitive appraisal. Psychoneuroendocrinology 2005;30:599–610.
24. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS scales. J Pers Soc Psychol 1988;54:1063–70.
25. Rosenberg M. The measurement of self-esteem. In: Rosenberg M, editor. Society and the Adolescent Self Image. Princeton, NJ: Princeton University Press; 1965:297–307.
26. Snaith RP, Morley S, Humayan D, Hargreaves D, Trigwell P. A scale for the assessment of hedonic tone the Snaith-Hamilton Pleasure Scale. Br J Psychiat 1995;167:99–103.
27. Cohen S, Mermelstein R, Kamarck T, Hoberman H. Measuring the functional components of social support. In: Sarason IG, Sarason BR, editors. Social Support: Theory, Research and Applications. The Hague, Holland: Martinus Nijhoff; 1985:73–94.
28. Sarason IG, Johnson JH, Siegel JM. Assessing the impact of life changes: development of the life experiences survey. J Consult Clin Psychol 1978;46: 932–46.
29. Pruessner JC, Kirschbaum C, Meinlschmid G, Hellhammer DH. Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. Psychoneuroendocrinology 2003;28:916–31.
30. Aspinwall LG, Taylor SE. A stitch in time: self-regulation and proactive coping. Psychol Bull 1997;121:417–36.
31. Aslaksen PM, Flaten MA. The roles of physiological and subjective stress in the effectiveness of a placebo on experimentally induced pain. Psychosom Med 2008;70:811–8.
32. Master SL, Eisenberger NI, Taylor SE, Naliboff BD, Shirinyan D, Lieberman MD. A picture's worth partner photographs reduce experimentally induced pain. Psychol Sci 2009;20:1316–8.
33. Piazza PV, Moal ML. Glucocorticoids as a biological substrate of reward: physiological and pathophysiological implications. Brain Res Rev 1997;25: 359–72.
34. Herman JP, Cullinan WE. Neurocircuitry of stress: central control of the hypothalmo-pituitary-adrenocortical axis. Trends Neurosci 1997;20:78–84.
35. Henckens MJAG, Van Wingen GA, Joels M, Fernandez G. Time-dependent effect of corticosteroids on human amygdala processing. J Neurosci 2010;30: 12725–32.
36. Gear RW, Aley KO, Levine JD. Pain-induced analgesia mediated by mesolimbic reward circuits. J. Neurosci 1999;19:7175–81.
37. Hellhammer DH, Hubert W, Schürmeyer T. Changes in saliva testosterone after psychological stimulation in men. Psychoneuroendocrinology 1985; 10:77–81.
38. Carmichael MS, Warburton VL, Dixen J, Davidson JM. Relationships among cardiovascular, muscular, and oxytocin responses during human sexual activity. Arch Sex Behav 1994;23:59–79.
39. Viau V, Meaney MJ. The inhibitory effect of testosterone on hypothalamic-pituitary-adrenal responses to stress is mediated by the medial preoptic area. J Neurosci 1996;16:1866–76.
40. Ditzen B, Schaer M, Gabriel B, Bodenmann G, Ehlert U, Heinrichs M. Intranasal oxytocin increases positive communication and reduces cortisol levels during couple conflict. Biol Psychiat 2009;65:728–31.
41. Heinrichs M, Baumgartner T, Kirschbaum C, Ehlert U. Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. Biol Psychiat 2003;54:1389–98.
42. Windle RJ, Shanks N, Lightman SL, Ingram CD. Central oxytocin administration reduces stress-induced corticosterone release and anxiety behavior in rats. Endocrinology 1997;138:2829–34.
43. Berridge KC, Robinson TE. Parsing reward. Trends Neurosci 2003;26: 507–13.
44. Bradley MM, Codispoti M, Sabatinelli D, Lang PJ. Emotion and motivation II: sex differences in picture processing. Emotion 2001;1:300–19.