



# Cognitive reappraisal increases neuroendocrine reactivity to acute social stress and physical pain



Thomas F. Denson<sup>a,\*</sup>, J. David Creswell<sup>b</sup>, Matthew D. Terides<sup>a</sup>,  
Kate Blundell<sup>a</sup>

<sup>a</sup> School of Psychology, UNSW Australia, Sydney, New South Wales 2052, Australia

<sup>b</sup> Department of Psychology, Carnegie Mellon University, Baker Hall 342c, Pittsburgh, PA 15213, USA

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**Summary** Cognitive reappraisal can foster emotion regulation, yet less is known about whether cognitive reappraisal alters neuroendocrine stress reactivity. Some initial evidence suggests that although long-term training in cognitive behavioral therapy techniques (which include reappraisal as a primary training component) can reduce cortisol reactivity to stress, some studies also suggest that reappraisal is associated with heightened cortisol stress reactivity. To address this mixed evidence, the present report describes two experimental studies that randomly assigned young adult volunteers to use cognitive reappraisal while undergoing laboratory stressors. Relative to the control condition, participants in the reappraisal conditions showed greater peak cortisol reactivity in response to a socially evaluative speech task (Experiment 1,  $N=90$ ) and to a physical pain cold pressor task (Experiment 2,  $N=94$ ). Participants in the cognitive reappraisal group also reported enhanced anticipatory psychological appraisals of self-efficacy and control in Experiment 2 and greater post-stressor self-efficacy. There were no effects of the reappraisal manipulation on positive and negative subjective affect, pain, or heart rate in either experiment. These findings suggest that although cognitive reappraisal fosters psychological perceptions of self-efficacy and control under stress, this effortful emotion regulation strategy in the short-term may increase cortisol reactivity. Discussion focuses on promising psychological mechanisms for these cognitive reappraisal effects.

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Cognitive reappraisal is an emotion regulation strategy that entails mentally modifying the way a situation is evaluated typically prior to the elicitation of a full-scale, negative emotional response (for a review, see [Gross and Thompson, 2007](#)). For example, one could reframe an upcoming public

\* Corresponding author. Tel.: +61 2 93851305; fax: +61 2 93853641.  
E-mail address: [t.denson@unsw.edu.au](mailto:t.denson@unsw.edu.au) (T.F. Denson).

speaking engagement as a welcome opportunity to disseminate research findings rather than perceive it as a negative, socially evaluative event. Reappraisal typically includes mentally distancing oneself from a stressor, thinking about the stressor in objective, non-emotional terms, and positively re-evaluating the stressor. Classic work on stress and coping has long emphasized the effects of appraisal and reappraisal processes in supporting mental and physical well-being (Lazarus and Folkman, 1984; Folkman et al., 1986). Some studies suggest that habitual use of cognitive reappraisal, as an emotion regulation strategy, is associated with greater levels of positive affect, better interpersonal functioning, and well-being under some circumstances (Gross and John, 2003; John and Gross, 2004; Troy et al., 2013).

Some researchers have posited that reappraisal may be associated with lower physiological reactivity (Gross and John, 2003; John and Gross, 2004). Indeed, studies have shown that intensive training in cognitive-behavioral techniques (which emphasize reappraisal strategies) can reduce neuroendocrine stress reactivity in healthy volunteers (e.g., Gaab et al., 2003). However, this previous work implemented therapy that consisted of multiple components such as relaxation, cognitive restructuring, problem solving, and self-instruction (Gaab et al., 2003). In order to experimentally isolate the effects of cognitive reappraisal on cortisol responses to stress, we instructed participants to only use cognitive reappraisal. Furthermore, a recent study showed that reappraisal was associated with heightened neuroendocrine reactivity to acute stress. Specifically, this study showed that dispositional tendencies to use reappraisal (as measured by self-report) was associated with increased cortisol response to a modified version of the Trier Social Stress Task (TSST) (Kirschbaum et al., 1993; Lam et al., 2009; cf. Koh et al., 2006). These initial studies provide mixed evidence for the effects of cognitive reappraisal on acute stress reactivity, but did not experimentally test the use of cognitive reappraisal under stress. To address these issues, the present investigation experimentally manipulated the use of a cognitive reappraisal strategy on neuroendocrine responses to a stressful public speech (i.e., the TSST) (Experiment 1) and to a cold pressor task (Experiment 2).

Although no previous studies have tested whether manipulating cognitive reappraisal effects neuroendocrine stress reactivity, two initial studies suggest that experimental manipulations of cognitive reappraisal alter cardiovascular stress reactivity. In these experiments, participants were exposed to stressors and asked to reappraise their physiological arousal as performance enhancing. Relative to control participants, those who reappraised showed less vasoconstriction and heightened cardiac output (Jamieson et al., 2012). In a second experiment, participants who reappraised arousal due to problems from the Graduate Record Examination (GRE) showed increases on a measure of sympathetic nervous system arousal (i.e., salivary alpha amylase). They even performed better on the math section of the actual GRE (Jamieson et al., 2010). Moreover, this work argues that reappraising stressful situations can elicit challenge appraisals and physiological activation for effective coping (Jamieson et al., 2013). Thus, cognitive reappraisal during acute stressors may be challenging and effortful, yet

simultaneously enhance feelings of self-efficacy and control over the stressor.

The present research describes the first experimental investigations of cognitive reappraisal on cortisol reactivity to stress. We hypothesized that reappraisal would increase cortisol responses to public speaking (Experiment 1) and physical pain (Experiment 2). Given the emerging research showing that reappraisal increases sympathetic nervous system arousal (Jamieson et al., 2010), we also predicted that reappraisal would increase heart rate responses to the stressors.

## 1. Experiment 1

In Experiment 1, participants completed a modified TSST (Kirschbaum et al., 1993). The original TSST contains a 10-min preparation period followed by 10 min of the speech and mental arithmetic. Our modified version contained a 10-min preparation period and 5 min of speech. We intentionally used the same modified TSST as Lam et al. (2009) because that was the only study to date to examine reappraisal and cortisol. Participants in the reappraisal condition were given instructions on how to use cognitive reappraisal during the stressor. Participants in the control condition did not receive any instructions. Relative to the control condition, we expected to see a greater increase in cortisol reactivity to the stressor among participants in the reappraisal condition.

### 1.1. Method

#### 1.1.1. Participants and design

A total of 90 healthy undergraduates (47 women;  $M_{age} = 20.54$  years,  $SD = 3.62$ ) from UNSW Australia participated in the experiment for course credit or AUD\$25. Participants provided written informed consent and all procedures were conducted in accordance with the Declaration of Helsinki. We asked participants not to eat, exercise, or consume caffeine 2 h prior to the experiment. Exclusion criteria included smoking, regular recreational drug use, chronic infections, cancer, tumors, any immune, autoimmune, or metabolic disease, endocrine disorders, use of contraceptive medication, pregnancy, and breastfeeding. Participants were randomly assigned to either the cognitive reappraisal condition ( $n = 45$ ), or the control condition ( $n = 45$ ). Men and women were equally distributed across conditions,  $\chi^2(1, N = 90) = 0.05, p = .83$ , as was menstrual cycle phase,  $\chi^2(2, N = 47) = 3.85, p = .15$ .

#### 1.1.2. Materials and procedure

Research participation occurred between the hours of 1200 h and 1800 h. Participants were informed that the study was investigating the link between communication abilities and physiological responses. Participants were seated at a desk and fitted with a Polar™ Watch heart rate monitor. A 40-min passive rest period followed, during which participants completed questionnaires and read affectively neutral nature magazines in the remaining time. Following the rest period, the experimenter took the first of three saliva samples for cortisol with a cotton Salivette (Nümbrecht, Germany).

**1.1.2.1. Baseline affect.** Participants completed the state version of the 60-item Positive and Negative Affect Schedule (PANAS-X; Watson and Clark, 1994). We examined the general positive affect ( $\alpha = 0.90$ ; e.g., delighted, at ease), and negative affect scores ( $\alpha = 0.80$ ; e.g., shaky, upset). Participants rated the extent to which each emotion descriptor applied to them ‘‘right now’’ (1 = *not at all*; 5 = *extremely*).

**1.1.2.2. Cognitive reappraisal manipulation.** Following the rest period, participants were given written instructions for the speech task. The instructions were delivered in envelopes to keep the experimenter blind to the experimental condition. Participants in both the reappraisal and control conditions were then informed that they would have 10 min to prepare a 5-min presentation that described their suitability for an administrative position in the university’s psychology department. They were told that their speeches would be delivered to an evaluative panel and would be videotaped for later analysis. The panel consisted of the male experimenter, and a female confederate both of whom wore white laboratory coats.

Participants in the cognitive reappraisal condition received the following additional instructions in bold font (adapted from Gross, 1998, p. 227):

‘‘In preparation for your speech, try to think about some positive aspects of the task, such as lessons you may learn, or taking the task as an opportunity to develop skills and improve your abilities in this area, which may help performance in similar tasks that arise in the future. While performing the speech task, try to adopt a neutral and objective attitude toward your performance by thinking about your performance from a third-person perspective that is detached from the outcome of the task. In other words, feel as though you are indifferent and emotionally detached from your performance. When you perform the speech task try to think about the judgment of the panel in an objective and analytical manner, rather than personally, or in any way emotionally relevant to you. Try and detach yourself from what the panel thinks about your performance. When you have finished mentally preparing your speech, re-read the above paragraphs in bold so that these instructions are fresh in your mind when you begin performing the speech task.’’

After the 10-min preparation period, the experimenter entered the room with the female panel member, who was introduced as a Ph.D. student studying communication ability. The participant then delivered the 5 min speech in front of the panel. Panel members maintained serious faces and neutral body language throughout the speech, and pretended to make notes on the participant’s performance. Following the speech task, participants were given 30 min to complete the final questionnaires and read nature magazines.

**1.1.2.3. Post-stressor affect.** During the 30-min recovery period, participants completed a second state version of the PANAS-X to assess positive affect ( $\alpha = 0.92$ ) and negative affect ( $\alpha = 0.90$ ).

### 1.1.3. Physiological assessments

**1.1.3.1. Salivary cortisol.** In addition to the baseline assessment, cortisol was examined 15 min (peak) and 30 min

(recovery) following completion of the speech task. Cortisol samples were stored at  $-20^{\circ}\text{C}$  and analyzed by a professional reference laboratory in Dresden, Germany. After thawing, Salivettes were centrifuged at 3000 rpm for 5 min, which resulted in a clear supernatant of low viscosity. Salivary cortisol concentrations were measured using commercially available chemiluminescence-immuno-assays with high sensitivity (IBL International, Hamburg, Germany). Intra- and inter-assay coefficients of variations were below 10%.

**1.1.3.2. Heart rate.** Participants were affixed with the Polar Watch (model RS800CX) chest strap just below the chest muscles and the watch was placed face down so that the participant could not see it. The Polar Watch records heart rate time series to an accuracy of within the larger of either  $\pm 1\%$  or 1 bpm. Polar Watches are frequently used in psychophysiological research (e.g., Epstein et al., 2005; Sloan and Epstein, 2005; van der Meij et al., 2010), and heart rate assessed with the Polar Watch is very strongly correlated with heart rate assessed via traditional ECG ( $r_s = 0.97$  to 0.99; Goodie et al., 2000). Mean heart rate was taken during the 40 min relaxation period (baseline), the 10-min preparation period, the 5-min speech task, and the 30-min post-task recovery period.

### 1.1.4. Statistical analyses

Data were analyzed in SPSS Version 22 using condition  $\times$  time mixed ANCOVAs for each of the dependent variables. Data from five participants were removed due to baseline cortisol or cortisol reactivity  $> 3$  SDs from the sample mean. Time of experiment was included as a covariate. There were significant correlations between the number of hours participants had been awake and cortisol levels at baseline,  $r = -0.21$ ,  $p < 0.05$ . Therefore, hours awake were controlled for in the subsequent cortisol analyses. An ANCOVA controlling for hours awake showed that men had significantly greater mean cortisol levels at baseline ( $M = 9.44$ ,  $SD = 4.52$ ) than women ( $M = 7.84$ ,  $SD = 3.62$ ),  $F(1,86) = 4.24$ ,  $p = 0.042$ ,  $\eta^2 = 0.05$ . Therefore, gender was entered as a covariate in the cortisol analyses. There were no gender differences on heart rate, nor were there any differences between conditions on cortisol or heart rate at baseline.

## 1.2. Results

### 1.2.1. Affect

Separate 2 (time)  $\times$  2 (condition) mixed ANOVAs showed a decrease in positive affect from pre to post-stressor,  $F(1,89) = 44.41$ ,  $p < 0.001$ ,  $\eta^2 = 0.33$ , and an increase in negative affect,  $F(1,89) = 33.29$ ,  $p < 0.001$ ,  $\eta^2 = 0.27$ , suggesting stress-related emotional responses to the TSST. There was also a main effect of condition such that participants in the reappraisal condition reported less negative affect than participants in the control condition,  $F(1,88) = 4.98$ ,  $p = 0.03$ ,  $\eta^2 = 0.05$ . This main effect was due to a failure of randomization. At baseline, participants in the reappraisal condition reported lower levels of negative affect than participants in the control condition,  $F(1,88) = 8.67$ ,  $p = 0.004$ ,  $\eta^2 = 0.09$ . There was no main effect of condition on positive affect,  $F(1,88) = 0.49$ ,  $p = 0.49$ ,  $\eta^2 = 0.006$ . Furthermore, there were no time  $\times$  condition interactions

**Table 1** Self-reported state affect as a function of experimental condition in Experiment 1. Both groups showed decreased positive affect and increased negative affect as a result of the stress procedure in Experiment 1,  $p_s < 0.001$ .

	Cognitive reappraisal		Control condition	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Pre-stressor positive affect	2.68	0.89	2.57	0.75
Post-stressor positive affect	2.32	0.97	2.19	0.76
Pre-stressor negative affect	1.33	0.37	1.61	0.51
Post-stressor negative affect	1.69	0.60	1.91	0.81

for positive,  $F(1,88) = 0.03$ ,  $p = 0.86$ ,  $\eta^2 = 0.0004$ , or negative affect,  $F(1,88) = 0.26$ ,  $p = 0.61$ ,  $\eta^2 = 0.003$ , suggesting that the magnitude of the changes in affect were similar in both conditions (Table 1).

### 1.2.2. Cortisol

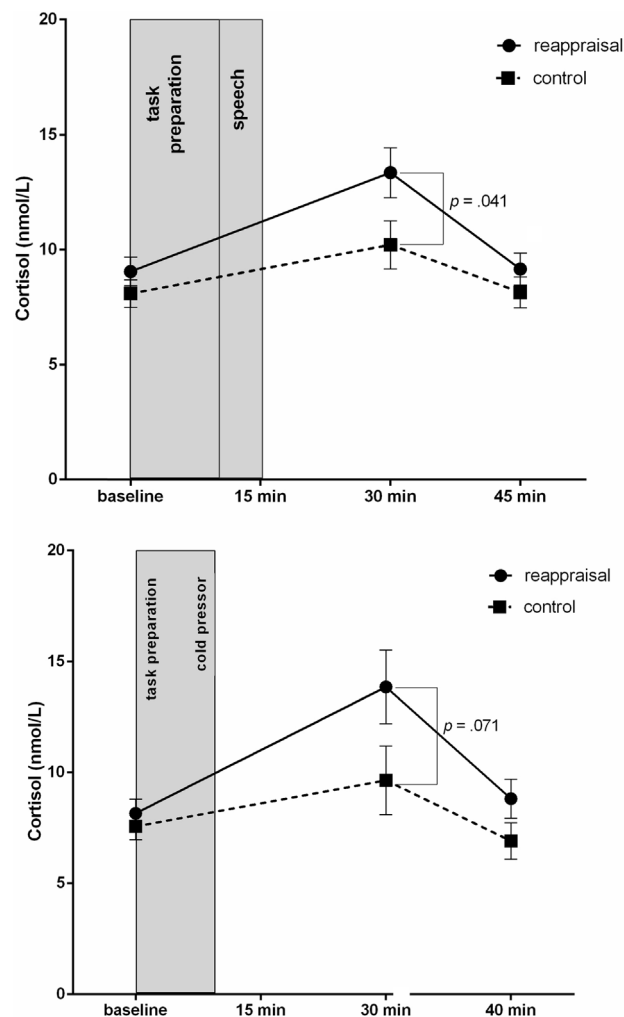
A 2 (condition)  $\times$  3 (time) mixed ANCOVA revealed a significant quadratic condition  $\times$  time interaction,  $F(1,80) = 4.51$ ,  $p = 0.037$ ,  $\eta^2 = 0.05$  (Fig. 1, top panel and Table 2). Follow-up tests between conditions showed that there was no significant difference in cortisol at baseline between the reappraisal condition and the control condition,  $F(1,80) = 1.24$ ,  $p = 0.27$ ,  $\eta^2 = 0.02$ , but at the post-stressor period, participants in the reappraisal condition had significantly higher cortisol levels than those in the control condition,  $F(1,80) = 4.33$ ,  $p = 0.041$ ,  $\eta^2 = 0.05$ . There was no significant difference between conditions in cortisol at recovery,  $F(1,80) = 1.10$ ,  $p = 0.30$ ,  $\eta^2 = 0.01$ .

### 1.2.3. Heart rate

A 2 (condition)  $\times$  4 (time) mixed ANOVA did not reveal a time  $\times$  condition interaction on HR,  $F(1.76, 154.65) = 2.77$ ,  $p = 0.07$ ,  $\eta^2 = 0.03$ . There was only a quadratic main effect of time,  $F(1,88) = 361.58$ ,  $p < 0.001$ ,  $\eta^2 = 0.80$ . Follow-up tests showed that HR increased from baseline to task preparation,  $F(1,89) = 111.24$ ,  $p < 0.001$ ,  $\eta^2 = 0.56$ . There was a further increase from preparation to the speech task,  $F(1,89) = 147.25$ ,  $p < 0.001$ ,  $\eta^2 = 0.62$ , which was followed by a decline during the recovery period,  $F(1,89) = 391.47$ ,  $p < 0.001$ ,  $\eta^2 = 0.82$  (Table 2).

## 1.3. Discussion

Within the context of a social-evaluative speech task, participants who engaged in cognitive reappraisal showed greater peak cortisol output than participants who did not receive instructions to reappraise. This is the first evidence that reappraisal increases neuroendocrine responses to stress. However, one limitation is that we did not evaluate whether reappraisal altered appraisals of challenge, threat, self-efficacy, and control. We conducted a second experiment to conceptually replicate and extend Experiment 1, by testing whether manipulating reappraisal altered psychological appraisals and cortisol reactivity to a physical pain stressor.



**Figure 1** Cortisol responses as a function of experimental condition across the duration of Experiment 1 (top panel) and Experiment 2 (bottom panel). Peak cortisol concentrations were significantly greater in the cognitive reappraisal condition than control condition in Experiment 1 and marginally greater in Experiment 2,  $p_s = 0.04$  and  $0.07$ , respectively. Values are estimated marginal means and standard error bars.

## 2. Experiment 2

The aim of Experiment 2 was to test the hypothesis that reappraisal would lower psychological threat perceptions and enhance feelings of challenge, self-efficacy, and



**Table 2** Cortisol and heart rate responses as a function of condition across the duration of Experiments 1 and 2. Participants in the reappraisal conditions had higher post-stressor cortisol than participants in the control conditions in both experiments,  $p$ s .04 and .07, respectively. The experimental conditions did not influence heart rate in either experiment.

	Cortisol (nmol/L)				Heart rate			
	Reappraisal		Control		Reappraisal		Control	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
<b>Experiment 1</b>								
Baseline	9.05	3.98	8.09	3.84	75.29	2.34	77.09	2.15
Task Preparation	—	—	—	—	84.76	1.77	83.96	1.81
Post-stressor	13.35*	6.94	10.22	6.94	95.58	2.52	99.69	2.65
Recovery	9.16	4.46	8.14	4.62	73.96	2.18	73.36	2.21
<b>Experiment 2</b>								
Baseline	8.15	0.64	7.56	0.60	75.74	1.36	77.66	1.63
During stressor	—	—	—	—	81.44	1.53	82.71	1.55
Post-stressor	13.86*	1.66	9.65	1.55	—	—	—	—
Recovery	8.82	0.88	6.91	0.82	73.68	1.68	72.35	1.15

\*  $p \leq 0.07$ .

control over the stressor. Within the context of a physical pain stressor, we also expected to conceptually replicate Experiment 1 by observing greater cortisol output in the reappraisal condition than the control group.

## 2.1. Method

### 2.1.1. Participants and design

A total of 94 undergraduates from UNSW Australia and nearby community members participated in exchange for course credit or AUD\$30. Participants provided written informed consent and all procedures were conducted in accordance with the Declaration of Helsinki. Data from 4 participants were removed for failure to follow instructions. The final sample consisted of 90 participants (52 women,  $M_{age} = 21.57$ ,  $SD = 4.20$ ). We asked participants not to eat, exercise, or consume caffeine 2 h prior to the experiment. Exclusion criteria were identical to Experiment 1. Participants were randomly assigned to either the cognitive reappraisal condition ( $n = 42$ ), or the control condition ( $n = 48$ ). Men and women were equally distributed across conditions,  $\chi^2(1, N = 88) = 0.01$ ,  $p = .91$ , as was menstrual cycle phase,  $\chi^2(2, N = 50) = 1.65$ ,  $p = .44$ .<sup>1</sup>

### 2.1.2. Materials and procedure

The experiment began between 1230 h and 1730 h. Participants rested for 40 min during which time they completed questionnaires and watched a nature video. At the end of the rest period, the participant provided a saliva sample with a cotton Salivette.

**2.1.2.1. Reappraisal manipulation.** All participants were then informed of what would happen during the cold pressor task and instructed to mentally prepare for it over the next

10 min. We used the same procedures as in Experiment 1, but provided additional instructions to increase the impact of the cognitive reappraisal manipulation. Specifically, participants in the cognitive reappraisal condition were given four additional instructions: (1) *adopt a neutral and objective attitude toward their performance*; (2) *think about it from a third person perspective*; (3) *think about the positive aspects of the task*; and (4) *detach themselves from any emotional experience or evaluation of their performance*. After reading these instructions, participants notified the experimenter, at which point, participants were asked to verbalize what they were asked to do.

**2.1.2.2. Anticipatory and post-stressor psychological stress appraisals.** The 16-item Primary Appraisal Secondary Appraisal Scale (PASA; Gaab et al., 2003) assessed appraisals about the upcoming cold pressor task. Specifically, after the instructions for cognitive reappraisal participants were asked to indicate how much they agreed or disagreed with each item *in relation to the upcoming cold pressor task* (1 = *totally disagree*; 6 = *totally agree*). The Primary Appraisal subscale assessed the extent to which participants expected the cold pressor to be challenging and threatening ( $\alpha = 0.59$ ). The Secondary Appraisal subscale assessed the extent to which participants felt able to handle the cold pressor and in control of the situation ( $\alpha = 0.71$ ). Although the PASA was originally designed as a measure of anticipatory stress appraisals, we included a post-stressor PASA on an exploratory basis. Specifically, participants completed a post-stressor PASA “in relation to the cold pressor task you just completed” (Primary Appraisal  $\alpha = 0.76$ ; Secondary Appraisals  $\alpha = 0.70$ ).

**2.1.2.3. Cold pressor stress task, pain, and affect.** Once participants finished the baseline questionnaires they were instructed to submerge their non-dominant hand in a bucket of ice-chilled water ( $7 \pm 1^\circ\text{C}$ ) for as long as they could, but for no longer than 2 min (Birnie et al., 2012). The experimenter reiterated to participants that they could stop the task at any time if they wanted, yet 92% of participants immersed their hands in the water for the full 2 min. At

<sup>1</sup> Saliva samples for an additional eight participants were mistakenly assayed in a separate batch and were therefore excluded from all study analyses.

15-s intervals during the task participants answered two rating scales: (1) "please indicate how you feel right now" (1 = *very bad*; 7 = *very good*), and (2) "please indicate how much pain you feel right now" (1 = *no pain*; 7 = *pain no longer tolerable*). To reduce potential noise due to social feedback, the experimenter faced away from the participant while notifying them when to complete each question set.

### 2.1.3. Physiological assessments

Cortisol was assessed at the end of the 40-min rest period (baseline), 30 min post-stressor (peak) and 40 min post-stressor (recovery). During wait times participants viewed a neutral video. Cortisol collection tubes and assays were identical to Experiment 1. Heart rate was recorded using a Polar heart rate monitor (model RS800CX) during the 40-min rest period (baseline), a 5-min period during the cold pressor (stressor), and 30-min post stressor.

### 2.1.4. Statistical analyses

Data were analyzed in SPSS Version 22 using condition  $\times$  time mixed ANCOVAs for each of the dependent variables. Data from two participants were removed due to baseline cortisol or cortisol reactivity  $> 3$  SDs from the sample mean. As in Experiment 1, time of experiment onset was used as a covariate in the cortisol analyses. Included in the initial questionnaires was a more comprehensive screening questionnaire than that used in Experiment 1 (similar to Schultheiss and Stanton, 2009). This measure assessed time since participants woke up, last consumed caffeine, ate, exercised, chewed gum, drank alcohol and days since last menstruation. Controlling for baseline cortisol, of these variables, only time since the last meal was correlated with cortisol reactivity ( $r_{\text{time } 2} = 0.24$ ,  $p = 0.03$ ;  $r_{\text{time } 3} = 0.24$ ,  $p = 0.03$ ), and was therefore included as a covariate. Gender was not included as a covariate in the cortisol or heart rate analyses because there were no gender differences or interactions with the experimental manipulation.

## 2.2. Results

### 2.2.1. Psychological appraisals

There was no effect of the reappraisal manipulation on primary anticipatory stress appraisals (i.e., combined feelings of challenge and threat) ( $M_{\text{reappraisal}} = 2.93$ ,  $SD = 0.54$ ;  $M_{\text{control}} = 2.72$ ,  $SD = 0.68$ ),  $F(1, 88) = 2.44$ ,  $p = 0.122$ ,  $\eta^2 = 0.03$ , or post-stressor primary appraisals, ( $M_{\text{reappraisal}} = 3.40$ ,  $SD = 0.73$ ;  $M_{\text{control}} = 3.35$ ,  $SD = 0.85$ ),  $F < 1$ . However, participants in the reappraisal condition reported greater anticipatory secondary appraisals (i.e., feeling in control and efficacious about the upcoming cold pressor task) than participants in the control condition ( $M_{\text{reappraisal}} = 4.38$ ,  $SD = 0.56$ ;  $M_{\text{control}} = 3.98$ ,  $SD = 0.72$ ),  $F(1, 88) = 8.15$ ,  $p = 0.005$ ,  $\eta^2 = 0.09$ . After the stressor, participants in the reappraisal condition also reported greater secondary appraisals than participants in the control condition ( $M_{\text{reappraisal}} = 4.23$ ,  $SD = 0.66$ ;  $M_{\text{control}} = 3.92$ ,  $SD = 0.67$ ),  $F(1, 88) = 4.94$ ,  $p = 0.029$ ,  $\eta^2 = 0.053$ . The data for the individual subscales are presented in Table 3. Cognitive reappraisal increased anticipatory appraisals of challenge, self-efficacy, and control but had no effect on threat appraisals. Furthermore, even after completing the stressor, participants in the reappraisal

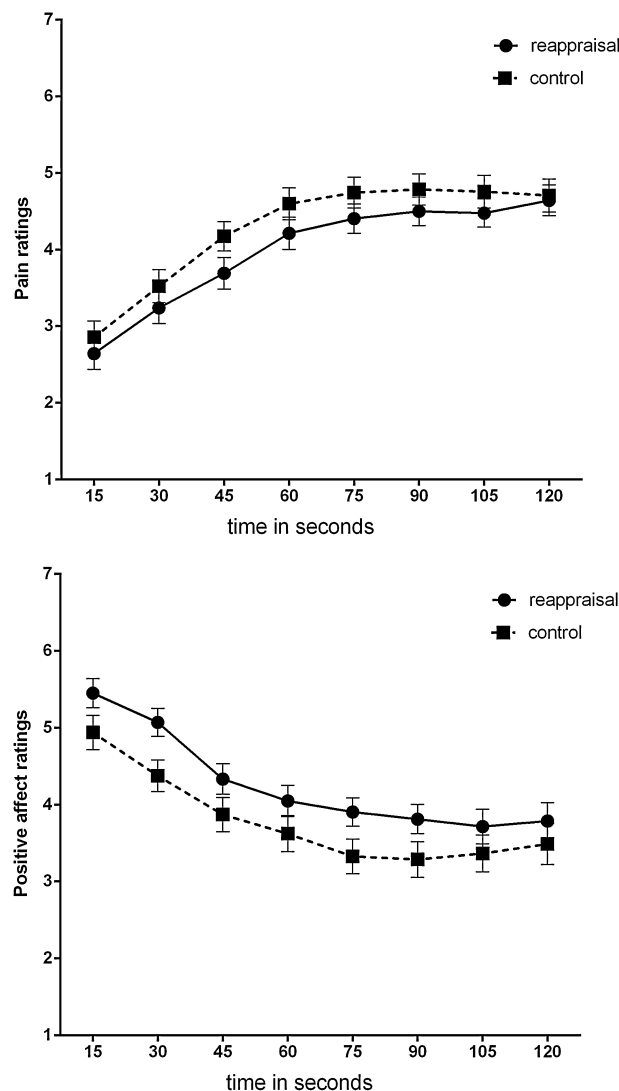


Figure 2 Pain ratings (top panel) and positive affect (bottom panel) during the cold pressor at 15 s intervals (Experiment 2).

condition reported feeling more efficacious than participants in the control condition.

### 2.2.2. Pain and affect during cold pressor

A 2 (condition)  $\times$  8 (time) mixed ANOVA on pain ratings during the stressor revealed a significant quadratic effect of time,  $F(1, 81) = 67.95$ ,  $p < 0.001$ ,  $\eta^2 = 0.46$ , but no main effect of condition or interaction,  $F_s < 1$ . A 2 (condition)  $\times$  8 (time) repeated measures ANOVA on positive affect ratings during the stressor revealed a significant quadratic effect of time,  $F(1, 81) = 64.07$ ,  $p < 0.001$ ,  $\eta^2 = 0.44$ , but no main effect of condition or interaction,  $F_s < 1$  (Fig. 2).

### 2.2.3. Cortisol

Fig. 1 (bottom panel) and Table 2 display the cortisol data. The 2 (condition)  $\times$  3 (time) ANCOVA revealed a marginally significant quadratic two-way interaction,  $F(1, 84) = 2.83$ ,  $p = 0.096$ ,  $\eta^2 = 0.03$ . Follow-up tests between conditions showed that there was no difference in cortisol at baseline between the reappraisal condition and the control

**Table 3** Anticipatory and post-stressor means and *SDs* for the PASA subscales as a function of experimental condition in Experiment 2. Relative to the control condition, participants in the reappraisal condition reported heightened anticipatory appraisals of challenge, self-efficacy, and control, and higher post-stressor self-efficacy.

	Cognitive reappraisal		Control condition	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Anticipatory threat	2.16	0.89	2.23	0.84
Anticipatory challenge	3.70**	0.72	3.21	0.81
Anticipatory self-efficacy	4.51**	0.61	4.11	0.79
Anticipatory control	4.24*	0.71	3.86	0.95
Post-stressor threat	2.65	0.98	2.81	1.08
Post-stressor challenge	4.16	0.85	3.90	0.95
Post-stressor self-efficacy	4.34**	0.77	3.85	0.83
Post-stressor control	3.99	0.89	4.12	0.79

\*  $p < 0.05$ .

\*\*  $p < 0.01$ .

condition,  $F < 1$ , but participants in the reappraisal condition had marginally significantly higher peak cortisol levels than those in the control condition,  $F(1,84) = 3.343$ ,  $p = 0.071$ ,  $\eta^2 = 0.04$ . There was no difference between the conditions in cortisol output at recovery,  $F(1,84) = 2.43$ ,  $p = 0.123$ ,  $\eta^2 = 0.03$ .

#### 2.2.4. Heart rate

A 2 (condition)  $\times$  3 (time) ANOVA revealed only a quadratic main effect of time,  $F(1,73) = 159.87$ ,  $p < 0.001$ ,  $\eta^2 = 0.69$ , such that heart rate increased from baseline during the stressor,  $F(1,74) = 67.77$ ,  $p < 0.001$ ,  $\eta^2 = 0.48$ , and declined to recovery,  $F(1,74) = 228.39$ ,  $p < 0.001$ ,  $\eta^2 = 0.76$ .<sup>2</sup> There was no condition  $\times$  time interaction (Table 2).

### 2.3. Discussion

Experiment 2 provided an initial indication that cognitive reappraisal increases cortisol reactivity to a physical pain stressor, although this effect was marginally significant. This finding conceptually replicates the findings of Experiment 1, but also extends them by showing that cognitive reappraisal increases secondary appraisals of coping during the cold pressor task.

### 3. General discussion

In two experiments healthy young adults were asked to cognitively reappraise the way they thought about a stressful public speaking task (Experiment 1) and a physical pain stressor (Experiment 2) in a detached and objective manner. Relative to participants who did not reappraise these stressors, cognitive reappraisal heightened cortisol reactivity. To the best of our knowledge, the two studies reported here are the first experiments to show a casual effect of an experimental manipulation of cognitive reappraisal on HPA-axis reactivity. These findings conceptually extend a

correlational study showing that dispositional reappraisal was associated with heightened cortisol reactivity to public speaking (Lam et al., 2009). More broadly, the present findings are consistent with a growing literature showing that cognitive reappraisal can increase physiological reactivity under stress (i.e., cardiovascular challenge responses and HPA-axis) (e.g., Jamieson et al., 2010; Mauss et al., 2007).

The present work suggests that a brief experimental manipulation of cognitive reappraisal may have dissociable effects from longer-term cognitive behavioral training programs, which have been shown to buffer neuroendocrine stress reactivity (Gaab et al., 2003; cf. Antoni et al., 2000). One interesting possibility, to be tested in future research, is that using cognitive reappraisal for the first time (such as that tested in the present study) is effortful during acute stressors, which results in greater sympathetic nervous system and HPA-axis activation. But over longer cognitive training intervals (spanning days or weeks), the use of cognitive reappraisal may become more automatic (and less effortful), resulting in less physiological stress reactivity (e.g., Gaab et al., 2003). Indeed, this explanation is consistent with an emerging set of studies showing that short-term psychological training such as mindfulness and attentional control training can increase neuroendocrine stress reactivity (Creswell et al., 2014; Pilgrim et al., 2014). If this short-term vs long-term account is correct, it offers a novel task effort mechanism that could be tested in future research. Specifically, the increase in peak cortisol we observed among those in the reappraisal condition in both studies may have been caused by increased task effort. Although we did not measure task effort directly in the present work, we did observe increased anticipatory secondary appraisals of control in the reappraisal group (in Experiment 2), suggesting that cognitive reappraisal may have mobilized greater active coping resources under stress. Future studies could identify how reappraisal affects neuroendocrine activity during the learning and implementation of the strategy.

Although we favor a task demand explanation for the present two experiments, we speculate that an alternative explanation can be offered for these findings. Cognitive reappraisal may have increased neuroendocrine stress

<sup>2</sup> Degrees of freedom differ because technical problems caused the loss of heart rate data from 15 participants.

reactivity by fostering greater self-consciousness. Indeed, relative to a control group, inducing self-consciousness can increase cortisol reactivity similarly to social evaluative audiences in the TSST (Denson et al., 2012). There is suggestive evidence for this possibility derived from neuroimaging studies of cognitive reappraisal. These studies indicate that reappraising negative emotions activates neural regions implicated in self-focused cognition such as the dorsal medial prefrontal cortex (PFC) (for a review, see Ochsner and Gross, 2008). Thus, cognitive reappraisal may increase self-awareness and self-consciousness via heightened dorsal medial PFC activity.

It is also noteworthy that cognitive reappraisal and stressful procedures activate shared neural networks to some extent. These shared networks include increased medial PFC and decreased hippocampal activity (Ochsner and Gross, 2008; Dedovic et al., 2009a; Buhle et al., 2013; Hermann et al., 2014). Although increased activity in the medial PFC is thought to down-regulate emotional and cortisol responses to stress, decreased hippocampal activation is thought to increase cortisol output via connectivity with the hypothalamus and peri-PVN (Dedovic et al., 2009b). For instance, in a recent study of social fear learning, dispositional reappraisal was inversely correlated with hippocampal activity (Hermann et al., 2014). Lower resting hippocampal activity is associated with increased HPA-axis activation (Kiem et al., 2013). Because the hippocampus inhibits the HPA-axis, reappraisal-induced deactivations in the hippocampus may account for the heightened cortisol output obtained here and in Lam et al.'s (2009) study of dispositional reappraisal. However, because the emotion regulation neuroimaging literature has not focused specifically on stress, more research examining the influence of reappraisal on increased medial PFC activation and decreased hippocampal activation during stress is needed. A promising next step would be to conduct a neuroimaging experiment of cognitive reappraisal that makes use of a standardized stress task known to elicit cortisol responses, such as the Montreal Imaging Stress Task (Dedovic et al., 2005).

The present research was limited in some aspects. Our samples consisted of healthy young adults and the findings may not generalize to populations enduring chronic stress. Another limitation is that the effect of cognitive reappraisal on cortisol was only marginally significant in Experiment 2. The marginal effect was probably due to greater variability in post-stressor cortisol responses in Experiment 2 than in Experiment 1. Moreover, although cortisol responses are typically smaller in response to cold pressor tasks than public speaking, we did not observe that pattern of responses (but did observe more variability) (e.g., McRae et al., 2006). Moreover, implicit experimenter demand may have influenced appraisals in Experiment 2. Demand is a problem for all studies of cognitive reappraisal; however, the use of theoretically grounded procedures, measures, and the pattern of results across two experiments strongly suggests an initial indication that reappraisal alters stress reactivity. Furthermore, a suggestion account might suggest that participants in the reappraisal condition would have lower threat appraisals and less negative affect, which they did not. Although we view the inclusion of two very different types of stressors as a strength of the present research, the stressors differ in key aspects such as the presence of

social-evaluative threat. However, across these two very different types of stressors, reappraisal heightened cortisol output. This conceptual replication suggests that cognitive reappraisal affects cortisol responses to both social evaluative and physical pain stressors. Nonetheless, future research could directly replicate and extend these findings to additional types of stressors. Another limitation is that our control condition did not include an active task to match the reappraisal instructions. We chose not to use a sham procedure in order to maintain comparability between our study and other studies using the TSST (especially Lam et al.'s (2009) study). Nonetheless, future research could incorporate well-matched active control conditions. We also did not examine longer-term training in cognitive reappraisal or exposure to repeated stressors. Future work could examine the extent to which cognitive reappraisal may lower HPA-axis reactivity over time.

In the present work, reappraisal did not affect heart rate or self-reported affect. These findings stand in contrast to some prior work on reappraising anger provocations and reappraising arousal as performance enhancing (e.g., Mauss et al., 2007; Ray et al., 2008; Jamieson et al., 2010; Memedovic et al., 2010; Denson et al., 2011). When provoked, this prior research found associations between dispositional and manipulated reappraisal and physiological responses such as salivary alpha amylase, heart rate variability, blood pressure, cardiac output, vasoconstriction, and total peripheral resistance. In contrast to provocation, the present research suggests that cognitive reappraisal may not influence heart rate or general positive and negative affective responses to public speaking or a brief period of physical pain. The reasons for these differences may be due to the non-anger inducing context in the present experiments and different instructions (i.e., reappraising the stressor vs reappraising stress-induced physiological arousal). Moreover, our measurement of affect at the end of the stressor in Experiment 1 and reliance on broad dimensions of positive and negative affect may have obscured finer aspects of the time course of affective downregulation.

## 4. Conclusion

The two experiments described here suggest that reappraising a public speaking task or a cold pressor pain task in a detached and objective manner increases cortisol reactivity. These experiments and related studies suggest that cognitive reappraisal may increase active coping and greater physiological reactivity to acute stress (e.g., Jamieson et al., 2013). Future research should evaluate whether these stress reactivity effects promote context-specific mobilization of effective coping under stress.

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## Conflict of interest

The authors declare no conflict of interest.

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## References

- Antoni, M.H., Cruess, S., Cruess, D.G., Kumar, M., Lutgendorf, S., Ironson, G., Dettmer, E., Williams, J., Klimas, N., Fletcher, M.A., 2000. Cognitive-behavioral stress management reduces distress and 24-h urinary free cortisol output among symptomatic HIV-infected gay men. *Ann. Behav. Med.* 22, 29–37.
- Birnie, K.A., Petter, M., Boerner, K.E., Noel, M., Chambers, C.T., 2012. Contemporary use of the cold pressor task in pediatric pain research: a systematic review of methods. *J. Pain* 13, 817–826.
- Buhle, J.T., Kober, H., Ochsner, K.N., Mende-Siedlecki, P., Weber, J., Hughes, B.L., Kross, E., Atlas, L.Y., McRae, K., Wager, T.D., 2013. Common representation of pain and negative emotion in the midbrain periaqueductal gray. *Soc. Cogn. Affect. Neurosci.* 8, 609–616.
- Creswell, J.D., Pacilio, L.E., Lindsay, E.K., Brown, K.W., 2014. Brief mindfulness meditation training alters psychological and neuroendocrine responses to social evaluative stress. *Psychoneuroendocrinology* 44, 1–12.
- Dedovic, K., D'Aguiar, C., Pruessner, J.C., 2009a. What stress does to your brain: a review of neuroimaging studies. *Can. J. Psychiatry* 54, 6–15.
- Dedovic, K., Duchesne, A., Andrews, J., Engert, V., Pruessner, J.C., 2009b. The brain and the stress axis: the neural correlates of cortisol regulation in response to stress. *Neuroimage* 47, 864–871.
- Dedovic, K., Renwick, R., Mahani, N.K., Engert, V., Lupien, S.J., Pruessner, J.C., 2005. The Montreal imaging stress task: using functional imaging to investigate the effects of perceiving and processing psychosocial stress in the human brain. *J. Psychiatry Neurosci.* 30, 319–325.
- Denson, T.F., Creswell, J.D., Granville-Smith, I., 2012. Self-focus and social evaluative threat increase salivary cortisol responses to acute stress in men. *J. Behav. Med.* 35, 624–633.
- Denson, T.F., Grisham, J.R., Moulds, M.L., 2011. Cognitive reappraisal increases heart rate variability in response to anger provocation. *Motiv. Emot.* 35, 14–22.
- Epstein, E.M., Sloan, D.M., Marx, B.P., 2005. Getting to the heart of the matter: written disclosure, gender, and heart rate. *Psychosom. Med.* 67, 413–419.
- Folkman, S., Lazarus, R.S., Dunkel-Schetter, C., DeLongis, A., Gruen, R.J., 1986. Dynamics of a stressful encounter: cognitive appraisal, coping, and encounter outcomes. *J. Pers. Soc. Psychol.* 50, 992–1003.
- Gaab, J., Blättler, N., Menzi, T., Pabst, B., Stoyer, S., Ehlert, U., 2003. Randomized controlled evaluation of the effects of cognitive-behavioral stress management on cortisol responses to acute stress in healthy subjects. *Psychoneuroendocrinology* 28, 767–779.
- Goodie, J.L., Larkin, K.T., Schauss, S., 2000. Validation of polar heart rate monitor for assessing heart rate during physical and mental stress. *J. Psychophysiol.* 14, 159–164.
- Gross, J.J., 1998. Antecedent-and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *J. Pers. Soc. Psychol.* 74, 224–237.
- Gross, J.J., Thompson, R.A., 2007. Emotion regulation: conceptual foundations. In: Gross, J.J. (Ed.), *Handbook of Emotion Regulation*. Guilford, New York, pp. 3–26.
- Gross, J.J., John, O.P., 2003. Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *J. Pers. Soc. Psychol.* 85, 348–362.
- Hermann, A., Keck, T., Stark, R., 2014. Dispositional cognitive reappraisal modulates the neural correlates of fear acquisition and extinction. *Neurobiol. Learn. Mem.* 113, 115–124.
- Jamieson, J.P., Mendes, W.B., Blackstock, E., Schmader, T., 2010. Turning the knots in your stomach into bows: reappraising arousal improves performance on the GRE. *J. Exp. Soc. Psychol.* 46, 208–212.
- Jamieson, J.P., Mendes, W.B., Nock, M.K., 2013. Improving acute stress responses the power of reappraisal. *Curr. Dir. Psychol. Sci.* 22, 51–56.
- Jamieson, J.P., Nock, M.K., Mendes, W.B., 2012. Mind over matter: reappraising arousal improves cardiovascular and cognitive responses to stress. *J. Exp. Psychol. Gen.* 141, 417–422.
- John, O.P., Gross, J.J., 2004. Healthy and unhealthy emotion regulation: personality processes, individual differences, and life span development. *J. Pers.* 72, 1301–1333.
- Kiem, S.A., Andrade, K.C., Spoormaker, V.I., Holsboer, F., Czisch, M., Sämann, P.G., 2013. Resting state functional MRI connectivity predicts hypothalamus-pituitary-axis status in healthy males. *Psychoneuroendocrinology* 38, 1338–1348.
- Kirschbaum, C., Pirke, K.M., Hellhammer, D.H., 1993. The 'trier social stress test' – a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology* 28, 76–81.
- Koh, K.B., Choe, E., Song, J.E., Lee, E.H., 2006. Effect of coping on endocrinimmune functions in different stress situations. *Psychiatry Res.* 143, 223–234.
- Lam, S., Dickerson, S.S., Zoccola, P.M., Zaldivar, F., 2009. Emotion regulation and cortisol reactivity to a social-evaluative speech task. *Psychoneuroendocrinology* 34, 1355–1362.
- Lazarus, R., Folkman, S., 1984. *Stress, Appraisal, and Coping*. Springer, New York.
- Mauss, I.B., Cook, C.L., Cheng, J.Y.J., Gross, J.J., 2007. Individual differences in cognitive reappraisal: experiential and physiological responses to an anger provocation. *Int. J. Psychophysiol.* 66, 116–124.
- McRae, A.L., Saladin, M.E., Brady, K.T., Upadhyaya, H., Back, S.E., Timmerman, M.A., 2006. Stress reactivity: biological and subjective responses to the cold pressor and trier social stressors. *Hum. Psychopharmacol.* 21, 377–385.
- Memedovic, S., Grisham, J.R., Denson, T.F., Moulds, M.L., 2010. The effects of trait reappraisal and suppression on anger and blood pressure in response to provocation. *J. Res. Pers.* 44, 540–543.
- Ochsner, K.N., Gross, J.J., 2008. Cognitive emotion regulation: insights from social cognitive and affective neuroscience. *Curr. Dir. Psychol. Sci.* 17, 153–158.
- Pilgrim, K., Ellenbogen, M.A., Paquin, K., 2014. The impact of attentional training on the salivary cortisol and alpha amylase response to psychosocial stress: importance of attentional control. *Psychoneuroendocrinology* 44, 88–99.
- Ray, R.D., Wilhelm, F.H., Gross, J.J., 2008. All in the mind's eye? Anger rumination and reappraisal. *J. Pers. Soc. Psychol.* 94, 133–145.
- Schultheiss, O.C., Stanton, S.J., 2009. Assessment of salivary hormones. In: Harmon-Jones, E., Beer, J.S. (Eds.), *Methods in Social Neuroscience*. Guilford, New York, NY, USA.

- Sloan, D.M., Epstein, E.M., 2005. Respiratory sinus arrhythmia predicts written disclosure outcome. *Psychophysiology* 42, 611–615.
- Troy, A.S., Shallcross, A.J., Mauss, I.B., 2013. A person-by-situation approach to emotion regulation cognitive reappraisal can either help or hurt, depending on the context. *Psychol. Sci.* 24, 2505–2514.
- van der Meij, L., Buunk, A.P., Almela, M., Salvador, A., 2010. Testosterone responses to competition: the opponent's psychological state makes it challenging. *Biol. Psychol.* 84, 330–335.
- Watson, D., Clark, L.A., 1994. *The PANAS-X: Manual for the Positive and Negative Affect Schedule-Expanded Form*. University of Iowa, Ames.