Psychoneuroendocrinology 67 (2016) 51–60

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen

# Exposure to acute stress enhances decision-making competence: Evidence for the role of DHEA

Grant S. Shields*, Jovian C.W. Lam, Brian C. Trainor, Andrew P. Yonelinas

*Department of Psychology, University of California, Davis, CA 95616, United States*

ARTICLE INFO

*Article history:*
Received 21 November 2015
Received in revised form 30 January 2016
Accepted 31 January 2016

*Keywords:*
Stress
Decision-making competence
DHEA
Cortisol
Decision-making
Acute stress

ABSTRACT

Exposure to acute stress can impact performance on numerous cognitive abilities, but little is known about how acute stress affects real-world decision-making ability. In the present study, we induced acute stress with a standard laboratory task involving uncontrollable socio-evaluative stress and subsequently assessed decision-making ability using the Adult Decision Making Competence index. In addition, we took baseline and post-test saliva samples from participants to examine associations between decision-making competence and adrenal hormones. Participants in the stress induction group showed enhanced decision-making competence, relative to controls. Further, although both cortisol and dehydroepiandrosterone (DHEA) reactivity predicted decision-making competence when considered in isolation, DHEA was a significantly better predictor than cortisol when both hormones were considered simultaneously. Thus, our results show that exposure to acute stress can have beneficial effects on the cognitive ability underpinning real-world decision-making and that this effect relates to DHEA reactivity more than cortisol.



## 1. Introduction

Understanding the effects of stress on decision-making has important implications for society, the workplace, and family life, given the importance of decision-making in all of these domains (Parker et al., 2015). In this paper we sought to examine whether acute stress influenced the ability to make better real-world decisions—decision-making competence—and what the potential biological correlates of this effect might be.

Prior stress and decision-making research in humans has largely examined individual components of decision-making (for reviews, see Schwabe and Wolf, 2011; Starcke and Brand, 2012), such as goal-directedness or risk taking. Although understanding these effects is important, decision-making researchers have noted the poor ecological validity of investigating decision-making components in isolation (Bruine de Bruin et al., 2007). Actual decisions made in everyday life are a complex integration of multiple decision-making processes, and influences on one of these processes may influence other decision-making processes, leading to a different decision than might be expected by only examining individual processes. Thus, it is unknown how stress might influence ecologically valid measures of decision-making abilities.

The development of new, performance-based, measures of decision-making provides an approach for assessing "decision-making competence" or real-world decision-making ability (Bruine de Bruin et al., 2007). In the gold standard of these measures, the Adult Decision-Making Competence (Bruine de Bruin et al., 2007), participants make a number of decisions related to real-world situations (e.g., recognizing social norms, resistance to framing) that have an objectively correct choice, unlike other decision-making tasks where there is no "correct" choice, such as tasks assessing risk-taking or habit. Measures of decision-making competence require participants to make decisions that assess the processes (e.g., value assessment, metacognition) that contribute to better real-world decision-making. These decisions contribute to an overall score of decision-making competence.

The measure of decision-making competence we used in this study is ecologically valid, as it inversely predicts a host of poor decisions and resultant negative life events—such as having an unplanned pregnancy, quitting a job one has had for less than a week, or being incarcerated overnight (Parker et al., 2015). Although many factors play into adverse outcomes, decision-making competence remains a significant predictor of those decisions even when adjusting for factors such as socioeconomic

* Corresponding author.
*E-mail address:* gsshields@ucdavis.edu (G.S. Shields).

http://dx.doi.org/10.1016/j.psyneuen.2016.01.031
0306-4530/

status (Parker et al., 2015). Thus, the relatively new ability to assess decision-making competence now allows us to examine what effects, if any, stress has on the decision-making ability underpinning decisions made in everyday life.

How should stress influence decision-making? Introspection might suggest that stress impairs decision-making, and there is some evidence supporting this idea. For example, acute stress increases habitual behaviors and correspondingly reduces goal-directed actions (Schwabe and Wolf, 2011), which could suggest a diminished capacity to make beneficial decisions. Second, although this does not necessarily imply worse decision-making, acute stress increases risky decision-making (Starcke and Brand, 2012), which could suggest a decrease in error monitoring processes.

In contrast, alternate lines of research suggest that acute stress may enhance decision-making competence. First, stress induces negative affect, and negative affect promotes an analytical style of information processing (Moons and Mackie, 2007); analytic information processing in turn promotes better decision-making competence (Finucane and Gullion, 2010). Second, acute stress enhances inhibitory control (Schwabe et al., 2013), which is an executive function partially underpinning decision-making competence (Del Missier et al., 2012); thus, acute stress may enhance decision-making competence by improving inhibition. However, like evidence suggesting stress may impair decision-making competence, the above is limited to studies examining relatively restricted cognitive processes, and it is not known how stress impacts decision-making competence as a whole.

To elucidate the biological correlates of potential stress effects on decision-making competence, we examined the hormones cortisol and dehydroepiandrosterone (DHEA). We chose to examine these hormones because they both increase in response to the stressor employed in this study (Dickerson and Kemeny, 2004; Lennartsson et al., 2012b) and modulate receptors on neurons (e.g., GR, $GABA_A$, $\sigma_1$) that are expressed in brain circuits supporting decision-making (Butts et al., 2011; Pérez-Neri et al., 2008). In addition, both of these hormones causally influence decision-making processes (Ohana et al., 2015; Putman et al., 2010). Thus, given the neural and behavioral evidence suggesting that these hormones should exert important modulatory effects on decision-making, we chose to examine the relation of these hormones with decision-making competence.

DHEA and cortisol act through different pathways to influence neural and cognitive processes. For example, DHEA can influence neural activity by binding to $GABA_A$ receptors (Majewska et al., 1990), whereas cortisol influences neural activity through actions at glucocorticoid and mineralocorticoid receptors (Patel et al., 2008). DHEA is also a neurosteroid present within brain regions supportive of decision-making (Kancheva et al., 2011; Maninger et al., 2009). These different mechanisms of action produce different cognitive effects (e.g., Davis et al., 2008; Shields et al., 2015); for example, DHEA administration reduces risk-taking in decision-making in individuals enrolled in an addiction recovery program (Ohana et al., 2015), whereas cortisol administration increases risk-taking in decision-making in healthy individuals (Putman et al., 2010).

Determining whether the effects of stress on decision-making are related to cortisol or DHEA could provide important insights into the underlying mechanisms of these effects. Because of their high covariance with stress, reactivity of both hormones should associate with decision-making competence. However, because cortisol tends to impair cognitive processes, if acute stress decreases decision-making competence, we might expect that cortisol reactivity would predict decision-making competence better than DHEA reactivity. Conversely, because DHEA tends to enhance cognitive processes, if acute stress increases decision-making competence, we might expect that DHEA reactivity would predict decision-making competence better than cortisol reactivity.

### 1.1. Current research

To elucidate the effects of acute stress on decision-making ability underpinning better real-world decision-making, we assigned a large sample of young adults to well-validated stress induction or control conditions. This was followed by a decision-making index designed to measure real world decision-making competence. In addition, we collected baseline and post-manipulation saliva samples to examine levels of cortisol and DHEA, focusing on participants in the stress group, in order to assess the hormonal responses underlying these behavioral effects.

## 2. Method

### 2.1. Participants

Participants were 124 healthy young adults attending the University of California, Davis. Five participants were excluded from analyses due to misunderstanding the instructions. We did not invite participants who had a current illness, diabetes, history of stroke, neurological disorders, current or former diagnosis of post-traumatic stress disorder, hospitalization for a psychiatric disorder within the past year, current injury or illness within the past week, major sleep disturbances within the past six weeks, or consumption of more than eight caffeinated beverages a day. Similarly, individuals who were pregnant, nursing, on any form of medication (including hormonal birth control or asthma medication) or illegal drugs, had taken any mood-altering medications within the past two months, or had taken oral or injected corticosteroids within the past three months were not invited to participate. Participants were instructed not to eat, drink anything besides water, use tobacco, brush their teeth or floss, or engage in any exercise for two hours prior to the start of the study. Compliance with these instructions and inclusion criteria (i.e., no drug or hormonal contraceptive use) was assessed using a questionnaire at the beginning of the study; women also reported the date of the first day of their last menstrual period using that questionnaire. Menstrual cycle phase was approximated by days since preceding cycle had begun (i.e., 5 or less days: menstrual period; 6–13 days: follicular phase; 14+ days: luteal phase). The study protocol was approved by the institutional review board and all subjects gave written informed consent.

Of these 119 participants, 61 individuals (37 women) were randomly assigned to the stress induction condition and 58 individuals (39 women) were randomized to the non-stressful control condition. 16.7% of women were tested during their menstrual period, 20% during the follicular phase, and 63% during the luteal phase of their menstrual cycle. Participants ranged in age from 18 to 33 years-old ($M = 19.98$, $SD = 2.0$), and the sample was diverse, with 1.7% self-reporting as American Indian or Alaskan Native, 55.5% as Asian, 2.5% as Native Hawaiian or Pacific Islander, 3.4% as Black or African American, 16.8% as White, 14.3% as Hispanic, and 7.6% declined to state. Similarly, 5.9% of participants had parents whose mother or father completed only elementary or junior high school, 7.6% who completed some high school, 12.6% who graduated from high school, 11.8% who completed some college, 16.8% who graduated college, 25.2% who completed post-graduate or professional schooling, and 21.8% whose highest level of education was either unknown or not reported. Importantly, participants in the stress induction versus control conditions did not differ with respect to sex, race, menstrual cycle phase, socioeconomic status, or age ($ps > .17$, uncorrected).

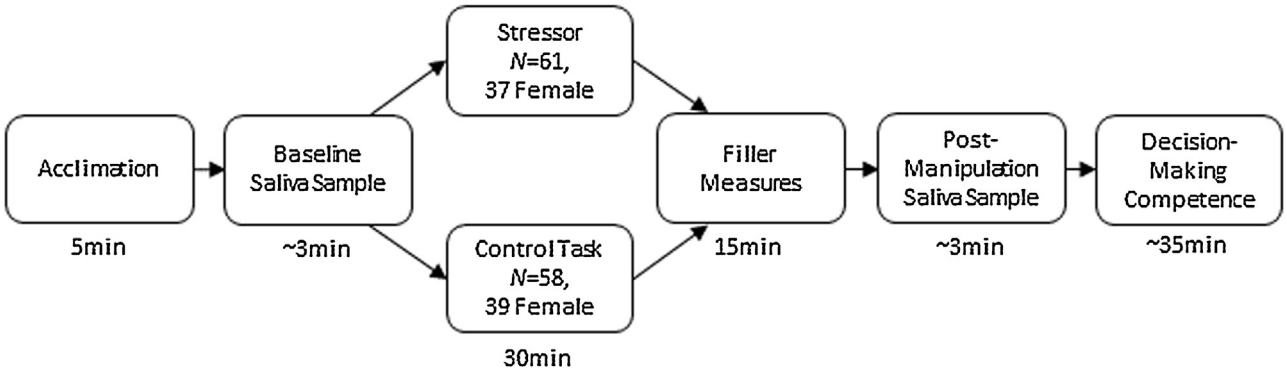


**Fig. 1.** Illustration of study procedure.

### 2.2. Materials and procedure

Participants came to the laboratory at either 12 pm or 3 pm for four-participant group sessions. Fig. 1 illustrates the study procedure. Upon arrival, an experimenter immediately greeted each participant and brought the participant into a cubicle in order to prevent the participants from interacting with each other. Once in the cubicle, each participant provided informed consent and completed miscellaneous measures—including a baseline measure of current affect (see below)—for approximately five minutes to allow acclimation to the testing environment. Participants' computers then reached a password-protected screen that instructed them to wait for instructions from the experimenters. Participants waited until all other participants for the session completed the initial measures, upon which time the first (baseline) saliva sample was taken.

Next, participants completed the laboratory-based stressor or control task, depending upon their time slot's assigned condition. An experience of acute stress was induced using the Trier Social Stress Test for Groups (TSST-G; von Dawans et al., 2011). This task includes two conditions: a stress induction condition and a non-stressful control condition. This task involves motivated performance and employs social evaluation and uncontrollability (Dickerson and Kemeny, 2004). In brief, participants in the stress induction condition were conspicuously recorded while they spoke on their real qualifications for the job they would like to have in front of a live panel of trained, stern evaluators, and afterwards were evaluated as they completed a difficult math task and told various threatening statements. In contrast, participants in the control condition quietly read aloud a scientific article and subsequently completed a math task without any social evaluation.

The TSST-G lasted approximately 30 min (including anticipation), after which time participants immediately completed in a randomized order the following stress appraisal questionnaire and measure of current affect. To assess appraisals of stress, participants in both the stress and non-stress TSST-G conditions used an unmarked scale ranging from 1 (*Strongly Disagree*) to 9 (*Strongly Agree*), to indicate the extent to which they agreed with seven randomly-ordered statements that assessed the stressfulness of the stressor/control task, such as, "The speech and mathematics tasks were very stressful." Reliability for this measure was excellent ($\alpha = .90$).

To assess changes in negative affect as a function of the stressor, prior to and after the stressor participants indicated on an unmarked 1–7 scale, ranging from 1 (*Not at All*) to 7 (*Very Much*), to what extent they currently felt 11 negative emotions, such as "scared." To avoid demand characteristics that might have arisen by only assessing negative affect, participants also indicated the extent to which they currently felt 11 positive emotions. Participants responded to the emotions in a randomized order. Self-reports of the negative emotions were averaged at each time point to create indices of negative affect. Reliabilities for negative affect assessed at baseline ($\alpha = .90$) and after the stress manipulation ($\alpha = .94$) were excellent.

To allow time for hormone reactivity to reach detectable levels in saliva, participants then completed filler personality questionnaires for 15 min. This delay was chosen based upon prior research showing that cortisol and DHEA are both significantly elevated at this time post-stressor (Dickerson and Kemeny, 2004; Lennartsson et al., 2012b). Following this delay, participants provided the second saliva sample (post-manipulation). Participants then completed the decision-making competence inventory.

Decision-making competence was indexed using the Adult Decision-Making Competence (Bruine de Bruin et al., 2007). This performance-based measure of decision-making has high internal consistency and test–retest reliability (Bruine de Bruin et al., 2007) as well as strong associations to other cognitive processes (Del Missier et al., 2012) and ecological validity (Bruine de Bruin et al., 2007; Parker et al., 2015). This measure requires participants to make a number of decisions, answers to which assess four fundamental decision-making skills. These fundamental decision-making skills are value assessment (i.e., appropriately evaluating the value of an outcome), belief assessment (i.e., appropriately evaluating the likelihood of an outcome), integration (i.e., combining beliefs and values when making decisions), and metacognition (i.e., being accurate in knowing one's own limitations and abilities). Decisions assessed in this inventory are made using multiple choice, 1–6 rating, binary (e.g., yes/no, true/false), and continuous (i.e., 0–100%) response scales. The criterion for better performance is either consistency or accuracy, depending upon the decision made (e.g., consistency between answers when the same question is presented highlighting either benefits or drawbacks of a decision would indicate better decision-making, whereas accuracy would indicate better decision-making competence when properly answering a question involves accurately assessing one's abilities). The overall score on this measure is thus our dependent variable of interest, as it reflects better real-world decision-making ability. Higher scores on thus indicate a better ability to make good decisions in everyday life. For ease of interpretability as well as graphing our results, we scaled the scores so that the control group's mean was 100 and standard deviation was 15 (as in IQ scores).

Finally, participants completed the demographics questionnaire before being debriefed, thanked, and dismissed.

### 2.3. Saliva samples

Participants provided two saliva samples (baseline and post-manipulation) using a passive drool method. Immediately after

collection, the saliva vials were placed in a freezer kept at −20 °C until assayed.

#### 2.3.1. Cortisol

Saliva samples were assayed in duplicate for cortisol using high-sensitivity Salivary Cortisol ELISA Kits (Salimetrics LLC, State College, PA) according to the manufacturer instructions. The inter-assay CV was 7.45% and the average intra-assay CV was 2.68%. Sensitivity for these assays was .012 μg/dL. All controls were in the expected ranges. Cortisol concentrations were converted from μg/dL to nmol/L for consistency with most human stress literature.

#### 2.3.2. DHEA

Saliva samples were assayed in duplicate for DHEA using the Salivary DHEA ELISA Kit (Salimetrics LLC, State College, PA) according to the manufacturer instructions. The inter-assay CV was 2.67% and the average intra-assay CV was 2.59%. Sensitivity for these assays was 10.2 pg/mL. All controls were in the expected ranges. Values are in the units of pg/mL.

### 2.4. Data reduction and analysis

All variables were inspected for conformity to a normal distribution and the natural logarithm transformation was applied when variables evidenced significant skew (i.e., DHEA and cortisol, both baseline and post-stressor). One participant was excluded from cortisol analyses due to excessively high baseline cortisol (|Value| > 3 SDs × Mean after log transformation). Importantly, however, including the outlier did not significantly alter any parameter estimate. There were no other outliers present in any other variable. In graphs and analyses, we discuss "reactivity" of hormones. By "reactivity" we mean residuals from regressing post-manipulation values (i.e., post-stressor or control task) on baseline values—that is, changes in these hormones from pre- to post-stressor.

Because the acute stress manipulation necessitated randomization of participant sessions to conditions (i.e., rather than participants), analyses required a multilevel model to account for shared variability within sessions. Thus, all analyses were linear mixed models with participants nested within session. We used a mixed model ANOVA nesting measurement occasions within participants and further nesting participants within session to assess changes in cortisol and DHEA from baseline to post-stressor. Bayesian parameter estimation was conducted when additional information could be gleaned from these analyses, accounting for the random effect of session if possible.[1] Aside from parameters estimated by Bayesian methods, all reported means and standard errors were least-squares means and standard errors. Degrees of freedom for all mixed models and their follow-up analyses were estimated using the Satterthwaite approximation, which relaxes the assumption of homogeneity of variance, but entails that the degrees of freedom contain non-integer numbers. Because of the importance of exploring sex differences (Cahill, 2012), we examined sex as a potential moderator of all effects; however, there were no significant interactions with sex.

Bayesian parameter estimation was employed in addition to traditional significance testing because it provides much richer information than null hypothesis significance testing (NHST); moreover, it is robust against violations of normality, heterogeneity between groups, and outliers (Kruschke, 2013), unlike NHST. Although Bayesian estimation estimates population parameters, we have retained the traditional use of $B$ to represent unstandardized slope estimates and $\beta$ to represent standardized slope estimates for clarity.

All analyses were conducted in R, version 3.2.1. Mixed models were fit using the lmerTest package. Least-squares means and their corresponding standard errors were derived using the lsmeans package. Bayesian between-groups parameter estimation was conducted with the package BEST using uninformative priors. Bayesian regression model parameter estimation was conducted with the packages MCMCpack and coda using informative priors derived from the previously fit linear mixed model.

## 3. Results

### 3.1. Preliminary analyses

#### 3.1.1. Self-reports

*3.1.1.1. Stress appraisals.* We first assessed whether participants in the stress induction condition perceived the stress manipulation to be more stressful than did participants in the control condition. As hypothesized, participants in the stress induction condition rated the stress manipulation to be significantly more stressful than participants in the control condition, $F(1,119.0) = 71.95$, $p < .001$ (Fig. 2(A)).

*3.1.1.2. Negative affect.* We next assessed whether participants in the stress induction condition evidenced an increase in negative affect relative to participants in the control condition as a function of the stress manipulation. As hypothesized, the Time × Condition interaction was significant, $F(1,117.0) = 15.45$, $p < .001$. At baseline, participants in the stress induction ($M = 2.28$, SE = .14) and control ($M = 2.29$, SE = .14) conditions did not differ in negative affect, $t(194.4) = -.05$, $p = .961$. However, following the acute stress or control manipulation, participants in the stress induction condition ($M = 2.68$, SE = .14) evidenced significantly greater negative affect than did participants in the control condition ($M = 1.89$, SE = .14), $t(194.4) = 4.07$, $p < .001$.

#### 3.1.2. Cortisol

We examined cortisol reactivity over time for the stress induction group and a randomly-selected 10 participants in the control group to confirm the success of both our stress manipulation and our control condition. As hypothesized, the Time × Condition interaction was significant, $F(1,64.7) = 17.57$, $p < .001$. Participants in the stress induction group significantly increased from pre- to post-manipulation, $t(67.0) = 5.97$, $p < .001$, whereas—as expected from a natural diurnal decline in cortisol—participants in the control group decreased from pre- to post-manipulation, $t(66.1) = -2.07$, $p = .042$ (Fig. 2(B)). Thus, the stress manipulation successfully increased cortisol in only the stress induction group.

#### 3.1.3. DHEA

We examined DHEA reactivity over time for the stress induction group and 10 participants in the control group. As hypothesized, the Time × Condition interaction was significant, $F(1,66.4) = 9.55$, $p = .003$. Participants in the stress induction group significantly increased from pre- to post-manipulation, $t(67.5) = 3.75$, $p < .001$, whereas—as expected from a natural diurnal decline in DHEA—participants in the control group tended to decrease from pre- to post-manipulation, $t(67.1) = -1.80$, $p = .076$ (Fig. 2(C)). Thus, the stress manipulation successfully increased DHEA in only the stress induction group.

[1] The method of Bayesian estimation used for between-groups analysis cannot incorporate random effects. However, the random effect of session contributed essentially nothing to that model ($\chi^2 \leq .001$, $p \geq .99$), so the parameter estimation proceeded without incorporating the random effect.

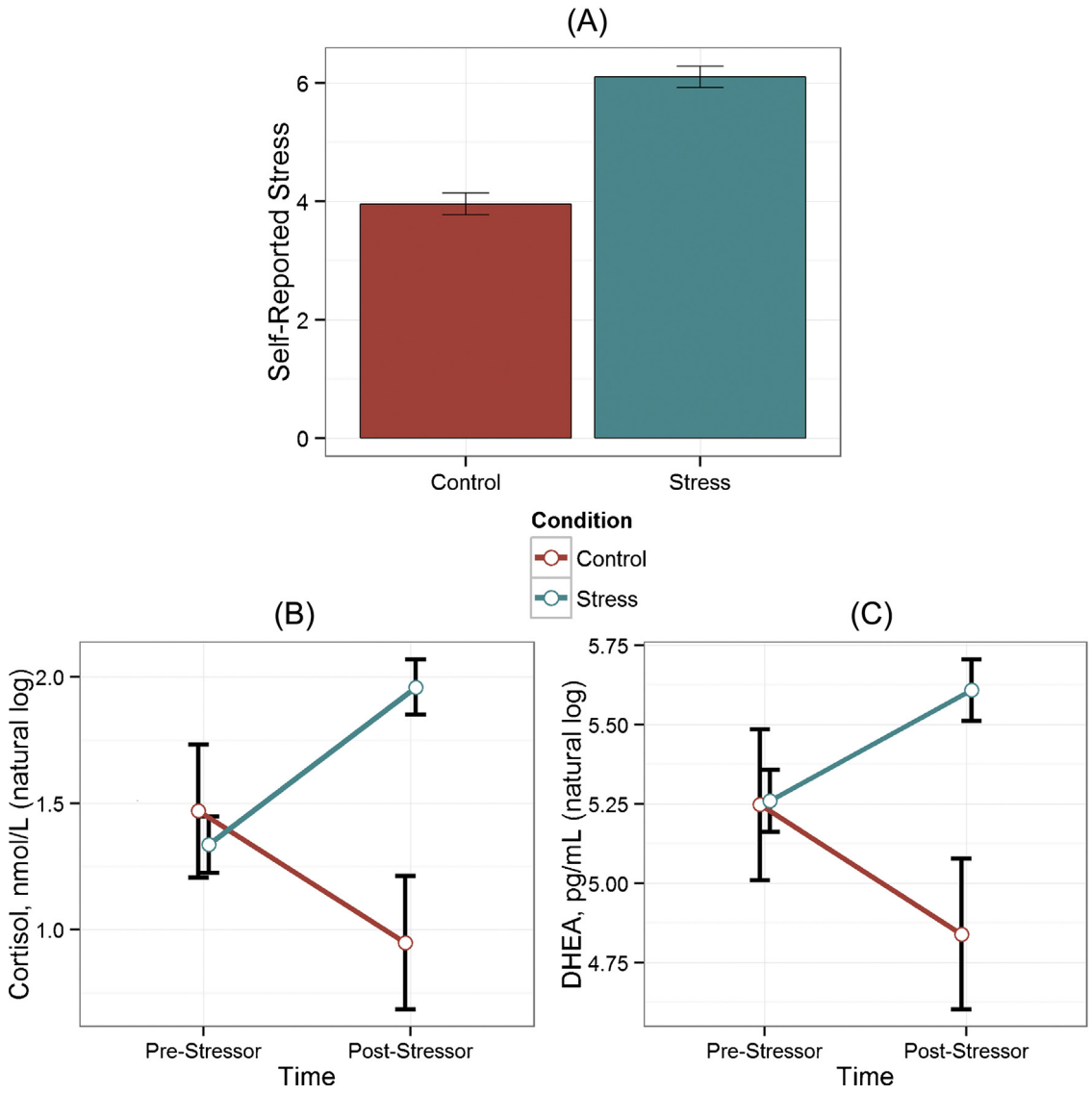


**Fig. 2.** Manipulation check. (A) Participants in the stress induction condition reported that significantly greater amounts of stress from the stress-induction task than did participants assigned to the control condition and task. (B) Participants in the stress induction condition evidenced a significant increase in cortisol, whereas the 10 participants assayed in the control condition evidenced a significant decrease. (C) Participants in the stress induction condition evidenced a significant increase in DHEA, whereas the 10 participants assayed in the control condition evidenced a marginal decrease.

### 3.2. Primary analyses

#### 3.2.1. Stress effects

Participants in the stress induction condition evidenced significantly better decision-making competence than participants in the control condition, $t(119.0) = 2.10$, $p = .038$ (Fig. 3). Thus, stress significantly enhanced decision-making competence.

To better elucidate the effect of stress on decision-making competence, we turn to Bayesian parameter estimation. Bayesian parameter estimation confirmed the results of the linear mixed model, as it showed the 95% Highest Density Interval (HDI; somewhat analogous to a more robust confidence interval that provides distributional information) of the difference between means did not include zero, $\mu_{diff} = 5.95$, 95% HDI$\mu_{diff}$ [.42, 11.33]. That is, participants in the stress induction condition ($\mu = 105.89$, $\sigma = 16.62$) evidenced better decision-making competence than participants in the control condition ($\mu = 99.94$, $\sigma = 12.26$). The magnitude of this effect was approximately moderate, $\theta = .41$, and similarly the 95% HDI of the effect size did not include zero, 95% HDI$_\theta$ [.028, .789]. Thus, not only did stress enhance decision-making competence, this enhancement was approximately equivalent in magnitude to the difference observed between individuals in high-level leadership positions and healthy controls (Carnevale et al., 2011). As such, the effect of stress on decision-making competence could be relevant to success in one's career or other aspects of everyday life.

Interestingly, the stress induction also appeared to increase variability in decision-making competence, as the 95% HDI of the difference between the standard deviations of each group did not include zero, $\sigma_{diff} = 4.36$, 95% HDI [.35, 8.47]. Although this heterogeneity between groups could pose a problem for assessing mean differences using traditional statistical tests, it does not pose a problem for a Bayesian estimation of the difference between means or the effect size. Thus, not only did stress enhance decision-making competence, stress also increased variability in decision-making competence. Thus, the decision-making ability of some people presumably benefits more from stress than that ability of other people.

#### 3.2.2. Associations with hormones

*3.2.2.1. DHEA.* To elucidate the biological mechanisms related to the effects of stress on decision-making competence, we

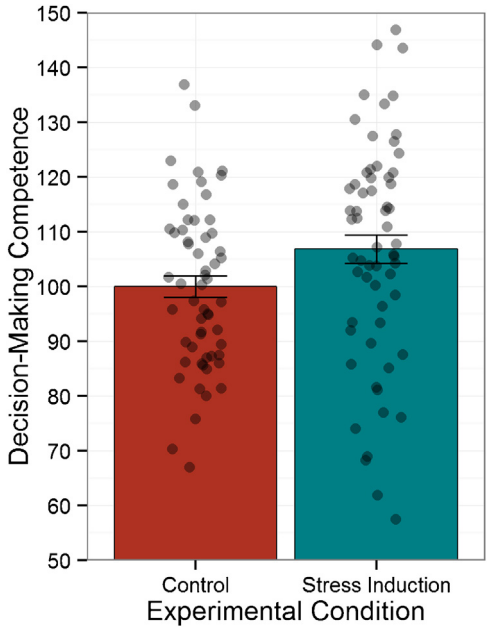


**Fig. 3.** Effects of acute stress on decision-making competence. Participants in the stress induction condition ($M = 105.91$, SD = 21.62) evidenced significantly better decision-making competence than participants in the control condition ($M = 100$, SD = 15), $p = .038$.

examined the association between post-manipulation DHEA and decision-making competence for participants in the stress induction condition, controlling for baseline DHEA. As hypothesized, the association between DHEA reactivity and decision-making competence was significant, $\beta = .40$, $t(59.0) = 2.58$, $p = .012$. Thus, DHEA reactivity was a strong predictor of decision-making competence; greater stress-induced increases in DHEA predicted better decision-making competence.

*3.2.2.2. Cortisol.* We next examined the association between post-manipulation cortisol and decision-making competence for participants in the stress induction condition, controlling for baseline cortisol. As hypothesized, the association between cortisol reactivity and decision-making competence was significant, $\beta = .30$, $t(58.0) = 2.07$, $p = .043$. Thus, cortisol reactivity was a strong predictor of decision-making competence; greater stress-induced increases in cortisol predicted better decision-making competence.

*3.2.2.3. Comparing DHEA with cortisol.* Although both cortisol and DHEA were associated with decision-making competence, this association may be due to their shared association with stress. That is, DHEA and cortisol reactivity strongly covaried, $r = .477$, $p < .001$, which could produce spurious correlations between those variables and a third variable affected by stress if this covariation is not taken into account. Thus, to examine whether cortisol or DHEA selectively accounted for the effects of stress on decision-making, we examined the associations between post-manipulation cortisol, post-manipulation DHEA, and decision-making competence, controlling for baseline cortisol and baseline DHEA. In this model, DHEA reactivity remained a significant predictor of decision-making competence, $\beta = .35$, $t(57.0) = 2.05$, $p = .045$ (Fig. 4a), whereas cortisol reactivity was no longer a significant predictor of decision-making competence, $\beta = .15$, $t(57.0) = .97$, $p = .335$ (Fig. 4(B)).

Bayesian parameter estimation further elucidated these results. In particular, DHEA was a significantly better predictor of decision-making competence than cortisol, because the upper bound of the 95% HDI of the slope predicting decision-making competence from cortisol, 95% HDI$\beta$ [−.059, .345], did not include the Bayesian estimate of the slope predicting decision-making competence from DHEA, $\beta = .349$. Thus, not only was post-manipulation DHEA more strongly associated with decision-making competence, it was a significantly better predictor than post-manipulation cortisol.

### 3.3. Exploratory analyses

Although we did not have a priori reason to hypothesize interactive effects, we nonetheless explored them. Experimental condition did not interact with age, sex, menstrual cycle phase, race, or changes in negative affect to influence decision-making competence, all ps > .250, uncorrected. Similarly, neither cortisol nor DHEA reactivity interacted with any of the aforementioned variables to predict decision-making competence, all ps > .147, uncorrected. This lack of interactions indicates that observed effects were robust across our sample.

Based upon prior observations suggesting that the DHEA/cortisol ratio might relate to different psychological stress responses (Shirotsuki et al., 2009), we regressed decision-making competence on this ratio. Neither the post-stressor DHEA/cortisol ratio ($p = .338$) nor the changes in this ratio ($p = .833$) were predictive of decision-making competence. Thus, the effects of cortisol and DHEA on decision-making competence are additive (as tested in analyses within Section 3.2.2), rather than interactive (as tested in the ratio).

Finally, we also conducted the analyses presented in Section 3.2.2 with the only difference being that we included the 10 randomly selected participants in the control group who were assayed for both DHEA and cortisol. In these analyses, all of the results were similar—including participants in the control group had no effect on associations of DHEA or cortisol with decision-making competence.

## 4. Discussion

Little is known about how acute stress influences the ability to make better decisions in everyday life. We addressed that gap in the present study by using a gold-standard laboratory manipulation of acute stress and subsequently assessed decision-making competence in stress and control groups while also collecting saliva samples to assay stress-reactive hormones with cognitive effects. We found, perhaps counterintuitively, that acute stress actually enhanced the ability to make better real-world decisions—decision-making competence. Moreover, we showed that although stress-induced increases in both cortisol and DHEA predicted decision-making competence, DHEA evidenced a significantly stronger association when both hormones were considered simultaneously. In addition, these associations were robust, with a lack of interactions with age, sex, and other variables. These results therefore show for the first time that acute stress can exert beneficial effects on the decision-making ability that appears to support better decision-making in everyday life.

Our study's findings provide empirical support for the hypothesis that acute stress enhances decision-making competence. Further, these findings also provide empirical support for one pathway by which acute stress may influence decision-making. In particular, after accounting for the covariance between cortisol and DHEA, only post-stressor DHEA predicted decision-making competence. Moreover, Bayesian parameter estimation confirmed that post-stressor DHEA was a significantly better predictor of decision-making competence than post-stressor cortisol.

It is also worth noting that potential confounds such as subjective experience of stress did not play a role in producing a stronger association between DHEA and decision-making competence than cortisol. That is, the effect of stress relative to the control condition on cortisol reactivity was greater than that effect on DHEA

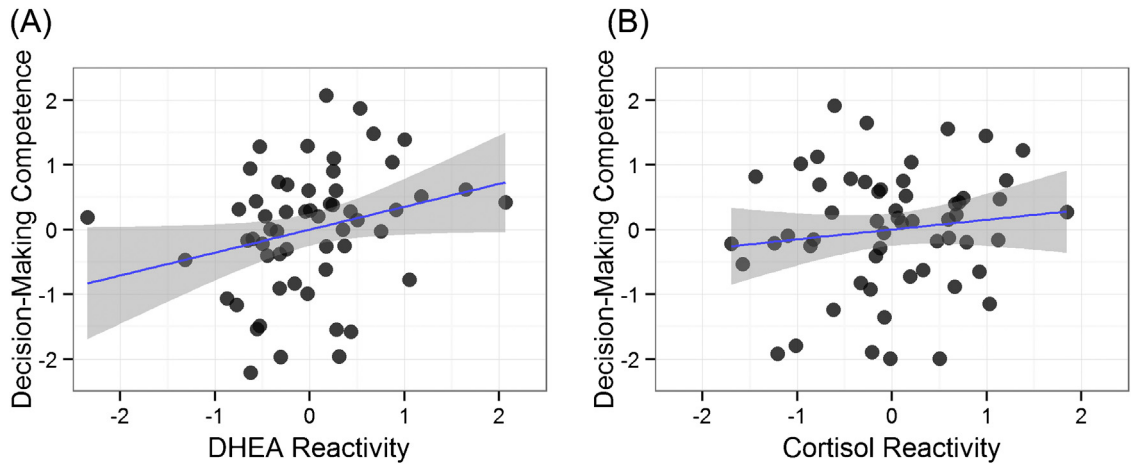


**Fig. 4.** Standardized associations of DHEA and cortisol (both natural log transformed) with decision-making competence, controlling for the other hormone as well as baseline values of these hormones. Post-manipulation DHEA ($\beta = .35$) was a significantly better predictor of decision-making competence than post-manipulation cortisol ($\beta = .15$).

reactivity. Thus, our data strongly suggest that DHEA is a better predictor of stress-induced alterations in decision-making competence than is cortisol.

The neural mechanisms underpinning potential effects of DHEA on decision-making competence are currently unclear. At a neurobiological level, some of DHEA's actions result in increased dopaminergic activity in the prefrontal cortex (Dong et al., 2007; Pérez-Neri et al., 2008), and increased dopaminergic activity can both enhance cognitive functions (Arnsten, 2009; Puig and Miller, 2015) and alter decision-making (Shafiei et al., 2012). Thus, our results may add to a growing list of stress effects due to alterations in dopaminergic activity (Trainor, 2011). It should be noted, though, that the mechanisms by which DHEA contributes to increased dopaminergic activity are still unclear, and there is some evidence that glucocorticoids can also increase dopaminergic activity within the prefrontal cortex (Butts et al., 2011; though see Inoue and Koyama, 1996). DHEA is also a neurosteroid that can modulate the function $GABA_A$, NMDA, and $\sigma_1$ receptors (Maninger et al., 2009; Pérez-Neri et al., 2008; Yabuki et al., 2015; Yadid et al., 2010). Additionally, most DHEA is converted to its sulfate ester—DHEA-S—and DHEA-S can exert neurobiological effects (Zajda et al., 2012). Thus, it is unknown whether DHEA exerts potential effects on decision-making directly through interacting with the aforementioned receptors or indirectly through the actions of one of its metabolites. As such, future research should attempt to determine the mechanism of action through which DHEA exerts effects on decision-making competence if it indeed plays a causal role in the effects observed here.

Given the common experience of making a decision one later regrets while stressed, how is it that stress can enhance decision-making competence? Although we can only speculate, one possible answer may lie in how stress influences emotions and reward salience. In particular, stress can make rewarding stimuli more appetizing, and it increases the intensity of negative affect (Heatherton and Wagner, 2011). As such, while stressed, one might be more tempted to indulge in a pleasurable activity one later regrets or be more tempted to make poor decisions based upon one's current mood. This explanation also fits with the fact that most of our poor decisions made during stress were made even when we "knew better"—or, had the decision-making competence to know that it was a bad decision. Thus, although stress enhances decision-making competence, benefits of this enhanced decision-making capability might not always be readily apparent. Future research should attempt to determine if this is indeed the case.

Because decision-making competence plays such an important role in everyday life, the potential for enhancing it has important implications. For example, because greater decision-making competence predicts better decision-making in interpersonal and financial situations, greater decision-making competence is associated with a more positive social environment and higher socioeconomic status (Bruine de Bruin et al., 2007; Parker and Fischhoff, 2005), though it should be noted that many factors contribute to these circumstances. In addition, people with greater decision-making competence are more likely to hold high-level leadership positions (Carnevale et al., 2011). As such, improving decision-making competence could potentially enhance a person's interpersonal quality of life and socioeconomic status. Thus, understanding the biological mechanisms underpinning the effects of stress on decision-making competence could provide enormous societal benefits. Future research should thus attempt to determine if experimentally manipulating DHEA using a pharmacological approach enhances decision-making competence.

DHEA may enhance cognitive function (Morgan et al., 2004, 2009; Rasmusson et al., 2004), and more importantly to this paper, DHEA administration may enhance decision-making. That is, individuals who were administered DHEA during rehabilitation from drug addiction were significantly less likely to choose to use drugs during the time they were given DHEA than were individuals given a placebo (Ohana et al., 2015). Although it is unclear whether this choice is due to enhanced decision-making competence, decision-making competence is a strong, inverse predictor of poor decisions regarding use and abuse of intoxicating substances (Parker et al., 2015), lending credence to the idea that administration of DHEA enhances decision-making competence. Thus, given the associations between DHEA, cortisol, and decision-making competence, a factorial manipulation of DHEA and cortisol could provide important insight into how stress enhances decision-making competence and potentially elucidate a mechanism through which to enhance decision-making competence.

Although we did not observe any effects of menstrual cycle on decision-making, gonadal hormones such as progesterone, testosterone, and estradiol all play a role in the biological stress response (Childs and De Wit, 2009; Lennartsson et al., 2012a) and could thus exert important effects on cognitive processes (Barros et al., 2015). We did not examine these hormones in this study because we wanted to constrain our analyses to those in which we had strong a priori expectations; because DHEA and cortisol had been linked to decision-making processes previously (Section 1), we selected these two hormones to examine. However, it is

possible that gonadal hormones modulated our effects. Nonetheless, not controlling for gonadal hormones does not invalidate our obtained results (McCarthy, 2015). As such, future research could attempt to extend our findings by examining the associations of gonadal or other hormones with decision-making competence following stress.

An additional extension of these results could examine personality traits that influence decision-making. Because our primary aim was to determine what effect, if any, acute stress had on decision-making competence, our goal was not to elucidate personality traits that might moderate the effect of stress on decision-making competence. Thus, examining the interplay of traits with acute effects of stress on decision-making competence is an important avenue for future research.

It is important to consider that chronic stress likely has different effects on decision-making competence. Although the acute effects of adrenal hormone responses are generally considered beneficial (McEwen, 2007; Schwabe et al., 2013), chronic or cumulative adrenal activation can induce glucocorticoid receptor resistance and is associated with more detrimental effects on health and cognitive functioning (Cohen et al., 2012; Jones and Moller, 2011; Silverman and Sternberg, 2012). Indeed, prior work reported that chronic stress impaired decision-making ability (Dias-Ferreira et al., 2009). Thus, the enhancing effects of acute stress we observed are unlikely to generalize to chronic stress.

Several limitations of this study are worth noting. First, although decision-making competence as measured in the current study did benefit from stress, future studies will be necessary to determine whether these effects generalize to other measures of decision-making. Second, although participants reported a lack of use of prescription or nonprescription drugs, we could not verify this abstinence directly, and it is possible that self-report data contained inaccuracies. Similarly, womens' menstrual cycle phases were approximated by self-reports rather than determined through hormone assessment, and these self-reports may contain inaccuracies. However, these self-report limitations would likely have affected the randomly-assigned experimental and control groups equally, so they are unlikely to have produced our obtained results. Third, although our stress manipulation was experimental, the analyses of hormones to decision-making competence were correlational—thus we cannot infer causation. Relatedly, factors affecting both cognitive function and DHEA or cortisol responses to stress (i.e., burnout; Lennartsson et al., 2015) may be important contributors to the effects we observed, but we are unable to test this. Fourth, we examined effects of stress in the afternoon, when DHEA and cortisol levels are both relatively lower than they are in the morning due to diurnal rhythms (Hucklebridge et al., 2005); as such, increases in these hormones may influence decision-making competence differently when baseline levels of these hormones are high. Fifth, although the measure of decision-making competence we used in this study is considered the current gold standard, some research suggests that the predictive utility of this measure could be improved by considering social skills and time orientation as well (Geisler and Allwood, 2015). Sixth, although the percentage of women was equivalent across our stress and control groups, because our sample was approximately two-thirds women we may have lacked statistical power to detect subtle sex differences in stress effects on decision-making competence. Nonetheless, given our sample size we achieved .77 power to detect a medium-sized sex by stress condition interaction effect ($f = .25$). Thus, at this time there is no evidence for strong sex differences in the effect of acute stress on decision-making competence. Finally, all research requires replication and extension. For example, using a pretest–posttest-control group design could replicate our finding that acute stress enhances decision-making competence and would require far less power than a purely between-groups design. Similarly, additional physiological parameters reflecting a stress response—i.e., salivary α-amylase—could help to further elucidate factors potentially involved in stress effects on decision-making competence.

Several strengths of this study are notable. First, our use of gold-standard methodology, such as the TSST-G or Adult Decision-Making Competence index, ensures that we have accurately manipulated and assessed what we intended to manipulate and assess. Second, the excellent coefficients of variation coupled with the consistency of all of our control values within expected ranges provides confidence that the hormone effects we observed are genuine and robust. Third, the large sample size ensured that this study evidenced sufficient power to detect a true effect and made it unlikely that sampling error produced the effects observed here—as can happen with smaller sample sizes. Finally, the use of advanced statistical techniques, such as Bayesian parameter estimation, allowed for a rich analysis of the observed data and provided security against potential violations of assumptions related to models used in null hypothesis significance testing.

### 4.1. Conclusion

In conclusion, in a large sample of healthy young adults we found that exposure to acute stress enhanced the decision-making ability that appears to underpin better decision-making in everyday life—decision-making competence. Moreover, we provided correlational support for the idea that DHEA may have contributed to this effect, because although stress increased both DHEA and cortisol, stress-induced increases in DHEA were a significantly better predictor of decision-making competence than cortisol. The correlational nature of our results cannot establish causation but suggest that a factorial manipulation of DHEA and cortisol should be a fruitful avenue for future research, especially considering the important implications of enhancing decision-making competence.

## Conflict of interest

The authors declare no conflict of interest in this work.

## Funding

The funding sources were in no way a part of this research.

## Contributors

GSS designed the study, conducted half of the cortisol and DHEA assays, analyzed the data, and wrote and revised the manuscript. JCWL conducted half of the cortisol and DHEA assays and provided minor revisions to the manuscript. BCT supervised the cortisol and DHEA assays and provided critical revisions to the manuscript. APY provided critical revisions to the manuscript.

## Acknowledgments

This research was supported by a University of California, Davis Psychology Department Summer Grant in Aid of Research to Grant S. Shields, NIH MH103322 to Brian C. Trainor, a University of California, Davis Provost's Undergraduate Fellowship to Jovian C. W. Lam, and NIH MH059352 to Andrew Yonelinas. The authors wish to thank numerous research assistants for assistance with data collection.

## References


Arnsten, A.F.T., 2009. Stress signalling pathways that impair prefrontal cortex structure and function. Nat. Rev. Neurosci. 10, 410–422, http://dx.doi.org/10.1038/nrn2648.

Barros, L.A., Tufik, S., Andersen, M.L., 2015. The role of progesterone in memory: an overview of three decades. Neurosci. Biobehav. Rev. 49, 193–204, http://dx.doi.org/10.1016/j.neubiorev.2014.11.015.

Bruine de Bruin, W., Parker, A.M., Fischhoff, B., 2007. Individual differences in adult decision-making competence. J. Personal. Soc. Psychol. 92, 938–956, http://dx.doi.org/10.1037/0022-3514.92.5.938.

Butts, K.A., Weinberg, J., Young, A.H., Phillips, A.G., 2011. Glucocorticoid receptors in the prefrontal cortex regulate stress-evoked dopamine efflux and aspects of executive function. Proc. Natl. Acad. Sci. U. S. A. 108, 18459–18464, http://dx.doi.org/10.1073/pnas.1111746108.

Cahill, L., 2012. A half-truth is a whole lie: on the necessity of investigating sex influences on the brain. Endocrinology 153, 2541–2543, http://dx.doi.org/10.1210/en.2011-2167.

Carnevale, J.J., Inbar, Y., Lerner, J.S., 2011. Individual differences in need for cognition and decision-making competence among leaders. Personal. Individ. Differ. 51, 274–278, http://dx.doi.org/10.1016/j.paid.2010.07.002.

Childs, E., De Wit, H., 2009. Hormonal, cardiovascular, and subjective responses to acute stress in smokers. Psychopharmacology (Berl.) 203, 1–12, http://dx.doi.org/10.1007/s00213-008-1359-5.

Cohen, S., Janicki-Deverts, D., Doyle, W.J., Miller, G.E., Frank, E., Rabin, B.S., Turner, R.B., 2012. Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. Proc. Natl. Acad. Sci. U. S. A. 109, 5995–5999, http://dx.doi.org/10.1073/pnas.1118355109.

Davis, S.R., Shah, S.M., McKenzie, D.P., Kulkarni, J., Davison, S.L., Bell, R.J., 2008. Dehydroepiandrosterone sulfate levels are associated with more favorable cognitive function in women. J. Clin. Endocrinol. Metab. 93, 801–808, http://dx.doi.org/10.1210/jc.2007-2128.

Del Missier, F., Mantyla, T., Bruine de Bruin, W., 2012. Decision-making competence, executive functioning, and general cognitive abilities. J. Behav. Decis. Mak. 25, 331–351, http://dx.doi.org/10.1002/bdm.731.

Dias-Ferreira, E., Sousa, J.C., Melo, I., Morgado, P., Mesquita, A.R., Cerqueira, J.J., Costa, R.M., Sousa, N., 2009. Chronic stress causes frontostriatal reorganization and affects decision-making. Science 325, 621–625, http://dx.doi.org/10.1126/science.1171203.

Dickerson, S.S., Kemeny, M.E., 2004. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychol. Bull. 130, 355–391, http://dx.doi.org/10.1037/0033-2909.130.3.355.

Dong, L.-Y., Cheng, Z.-X., Fu, Y.-M., Wang, Z.-M., Zhu, Y.-H., Sun, J.-L., Dong, Y., Zheng, P., 2007. Neurosteroid dehydroepiandrosterone sulfate enhances spontaneous glutamate release in rat prelimbic cortex through activation of dopamine D1 and sigma-1 receptor. Neuropharmacology 52, 966–974, http://dx.doi.org/10.1016/j.neuropharm.2006.10.015.

Finucane, M.L., Gullion, C.M., 2010. Developing a tool for measuring the decision-making competence of older adults. Psychol. Aging 25, 271–288, http://dx.doi.org/10.1037/a0019106.

Geisler, M., Allwood, C.M., 2015. Competence and quality in real-life decision making. PLoS One 10, e0142178, http://dx.doi.org/10.1371/journal.pone.0142178.

Heatherton, T.F., Wagner, D.D., 2011. Cognitive neuroscience of self-regulation failure. Trends Cogn. Sci. 15, 132–139, http://dx.doi.org/10.1016/j.tics.2010.12.005.

Hucklebridge, F., Hussain, T., Evans, P., Clow, A., 2005. The diurnal patterns of the adrenal steroids cortisol and dehydroepiandrosterone (DHEA) in relation to awakening. Psychoneuroendocrinology 30, 51–57, http://dx.doi.org/10.1016/j.psyneuen.2004.04.007.

Inoue, T., Koyama, T., 1996. Effects of acute and chronic administration of high-dose corticosterone and dexamethasone on regional brain dopamine and serotonin metabolism in rats. Prog. Neuropsychopharmacol. Biol. Psychiatry 20, 147–156, http://dx.doi.org/10.1016/0278-5846(95)00299-5.

Jones, T., Moller, M.D., 2011. Implications of hypothalamic-pituitary-adrenal axis functioning in posttraumatic stress disorder. J. Am. Psychiatr. Nurses Assoc. 17, 393–403, http://dx.doi.org/10.1177/1078390311420564.

Kancheva, R., Hill, M., Novák, Z., Chrastina, J., Kancheva, L., Stárka, L., 2011. Neuroactive steroids in periphery and cerebrospinal fluid. Neuroscience 191, 22–27, http://dx.doi.org/10.1016/j.neuroscience.2011.05.054.

Lennartsson, A.-K., Kushnir, M.M., Bergquist, J., Billig, H., Jonsdottir, I.H., 2012a. Sex steroid levels temporarily increase in response to acute psychosocial stress in healthy men and women. Int. J. Psychophysiol. 84, 246–253, http://dx.doi.org/10.1016/j.ijpsycho.2012.03.001.

Lennartsson, A.-K., Kushnir, M.M., Bergquist, J., Jonsdottir, I.H., 2012b. DHEA and DHEA-S response to acute psychosocial stress in healthy men and women. Biol. Psychol. 90, 143–149, http://dx.doi.org/10.1016/j.biopsycho.2012.03.003.

Lennartsson, A.-K., Sjörs, A., Jonsdottir, I.H., 2015. Indication of attenuated DHEA-s response during acute psychosocial stress in patients with clinical burnout. J. Psychosom. Res. 79, 107–111, http://dx.doi.org/10.1016/j.jpsychores.2015.05.011.

Majewska, M.D., Demirgören, S., Spivak, C.E., London, E.D., 1990. The neurosteroid dehydroepiandrosterone sulfate is an allosteric antagonist of the GABAA receptor. Brain Res. 526, 143–146, http://dx.doi.org/10.1016/0006-8993(90)90261-9.

Maninger, N., Wolkowitz, O.M., Reus, V.I., Epel, E.S., Mellon, S.H., 2009. Neurobiological and neuropsychiatric effects of dehydroepiandrosterone (DHEA) and DHEA sulfate (DHEAS). Front. Neuroendocrinol. 30, 65–91, http://dx.doi.org/10.1016/j.yfrne.2008.11.002.

McCarthy, M.M., 2015. Incorporating sex as a variable in preclinical neuropsychiatric research. Schizophr. Bull. 41, 1016–1020, http://dx.doi.org/10.1093/schbul/sbv077.

McEwen, B.S., 2007. Physiology and neurobiology of stress and adaptation: central role of the brain. Physiol. Rev. 87, 873–904, http://dx.doi.org/10.1152/physrev.00041.2006.

Moons, W.G., Mackie, D.M., 2007. Thinking straight while seeing red: the influence of anger on information processing. Personal. Soc. Psychol. Bull. 33, 706–720, http://dx.doi.org/10.1177/0146167206298566.

Morgan, C.A., Southwick, S., Hazlett, G., Rasmusson, A., Hoyt, G., Zimolo, Z., Charney, D., 2004. Relationships among plasma dehydroepiandrosterone sulfate and cortisol levels, symptoms of dissociation, and objective performance in humans exposed to acute stress. Arch. Gen. Psychiatry 61, 819–825, http://dx.doi.org/10.1001/archpsyc.61.8.819.

Morgan, C.A., Rasmusson, A., Pietrzak, R.H., Coric, V., Southwick, S.M., 2009. Relationships among plasma dehydroepiandrosterone and dehydroepiandrosterone sulfate, cortisol, symptoms of dissociation, and objective performance in humans exposed to underwater navigation stress. Biol. Psychiatry 66, 334–340, http://dx.doi.org/10.1016/j.biopsych.2009.04.004.

Ohana, D., Maayan, R., Delayahu, Y., Roska, P., Ponizovsky, A.M., Weizman, A., Yadid, G., Yechiam, E., 2015. Effect of dehydroepiandrosterone add-on therapy on mood, decision making and subsequent relapse of polydrug users. Addict. Biol., http://dx.doi.org/10.1111/adb.12241.

Parker, A.M., Bruine de Bruin, W., Fischhoff, B., 2015. Negative decision outcomes are more common among people with lower decision-making competence: an item-level analysis of the Decision Outcome Inventory (DOI). Front. Psychol. 6, 363, http://dx.doi.org/10.3389/fpsyg.2015.00363.

Parker, A.M., Fischhoff, B., 2005. Decision-making competence: external validation through an individual-differences approach. J. Behav. Decis. Mak. 18, 1–28, http://dx.doi.org/10.1002/bdm.481.

Patel, P.D., Katz, M., Karssen, A.M., Lyons, D.M., 2008. Stress-induced changes in corticosteroid receptor expression in primate hippocampus and prefrontal cortex. Psychoneuroendocrinology 33, 360–367, http://dx.doi.org/10.1016/j.psyneuen.2007.12.003.

Pérez-Neri, I., Montes, S., Ojeda-López, C., Ramírez-Bermúdez, J., Ríos, C., 2008. Modulation of neurotransmitter systems by dehydroepiandrosterone and dehydroepiandrosterone sulfate: mechanism of action and relevance to psychiatric disorders. Prog. Neuropsychopharmacol. Biol. Psychiatry 32, 1118–1130, http://dx.doi.org/10.1016/j.pnpbp.2007.12.001.

Puig, M.V., Miller, E.K., 2015. Neural substrates of dopamine D2 receptor modulated executive functions in the monkey prefrontal cortex. Cereb. Cortex 25, 2980–2987, http://dx.doi.org/10.1093/cercor/bhu096.

Putman, P., Antypa, N., Crysovergi, P., van der Does, W.A.J., 2010. Exogenous cortisol acutely influences motivated decision making in healthy young men. Psychopharmacology (Berl.) 208, 257–263, http://dx.doi.org/10.1007/s00213-009-1725-y.

Rasmusson, A.M., Vasek, J., Lipschitz, D.S., Vojvoda, D., Mustone, M.E., Shi, Q., Gudmundsen, G., Morgan, C.A., Wolfe, J., Charney, D.S., 2004. An increased capacity for adrenal DHEA release is associated with decreased avoidance and negative mood symptoms in women with PTSD. Neuropsychopharmacology 29, 1546–1557, http://dx.doi.org/10.1038/sj.npp.1300432.

Schwabe, L., Höffken, O., Tegenthoff, M., Wolf, O.T., 2013. Stress-induced enhancement of response inhibition depends on mineralocorticoid receptor activation. Psychoneuroendocrinology 38, 2319–2326, http://dx.doi.org/10.1016/j.psyneuen.2013.05.001.

Schwabe, L., Wolf, O.T., 2011. Stress-induced modulation of instrumental behavior: from goal-directed to habitual control of action. Behav. Brain Res. 219, 321–328, http://dx.doi.org/10.1016/j.bbr.2010.12.038.

Shafiei, N., Gray, M., Viau, V., Floresco, S.B., 2012. Acute stress induces selective alterations in cost/benefit decision-making. Neuropsychopharmacology 37, 2194–2209, http://dx.doi.org/10.1038/npp.2012.69.

Shields, G.S., Bonner, J.C., Moons, W.G., 2015. Does cortisol influence core executive functions? A meta-analysis of acute cortisol administration effects on working memory, inhibition, and set-shifting. Psychoneuroendocrinology 58, 91–103, http://dx.doi.org/10.1016/j.psyneuen.2015.04.017.

Shirotsuki, K., Izawa, S., Sugaya, N., Yamada, K.C., Ogawa, N., Ouchi, Y., Nagano, Y., Nomura, S., 2009. Salivary cortisol and DHEA reactivity to psychosocial stress in socially anxious males. Int. J. Psychophysiol. 72, 198–203, http://dx.doi.org/10.1016/j.ijpsycho.2008.12.010.

Silverman, M.N., Sternberg, E.M., 2012. Glucocorticoid regulation of inflammation and its functional correlates: from HPA axis to glucocorticoid receptor dysfunction. Ann. N. Y. Acad. Sci. 1261, 55–63, http://dx.doi.org/10.1111/j.1749-6632.2012.06633.x.

Starcke, K., Brand, M., 2012. Decision making under stress: a selective review. Neurosci. Biobehav. Rev. 36, 1228–1248, http://dx.doi.org/10.1016/j.neubiorev.2012.02.003.

Trainor, B.C., 2011. Stress responses and the mesolimbic dopamine system: social contexts and sex differences. Horm. Behav. 60, 457–469, http://dx.doi.org/10.1016/j.yhbeh.2011.08.013.

von Dawans, B., Kirschbaum, C., Heinrichs, M., 2011. The Trier Social Stress Test for Groups (TSST-G): a new research tool for controlled simultaneous social stress

exposure in a group format. Psychoneuroendocrinology 36, 514–522, http://dx.doi.org/10.1016/j.psyneuen.2010.08.004.
Yabuki, Y., Shinoda, Y., Izumi, H., Ikuno, T., Shioda, N., Fukunaga, K., 2015. Dehydroepiandrosterone administration improves memory deficits following transient brain ischemia through sigma-1 receptor stimulation. Brain Res. 1622, 102–113, http://dx.doi.org/10.1016/j.brainres.2015.05.006.
Yadid, G., Sudai, E., Maayan, R., Gispan, I., Weizman, A., 2010. The role of dehydroepiandrosterone (DHEA) in drug-seeking behavior. Neurosci. Biobehav. Rev. 35, 303–314, http://dx.doi.org/10.1016/j.neubiorev.2010.03.003.
Zajda, M.E., Krzascik, P., Hill, M., Majewska, M.D., 2012. Psychomotor and rewarding properties of the neurosteroids dehydroepiandrosterone sulphate and androsterone: effects on monoamine and steroid metabolism. Acta Neurobiol. Exp. (Wars) 72, 65–79.