



To trust, or not to trust? Individual differences in physiological reactivity predict trust under acute stress



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ABSTRACT

The stress response represents an evolutionarily ancient array of biological responses to challenge or threat that facilitate survival by promoting adaptive behaviors. ‘Adaptive’ in the evolutionary sense, however, does not easily translate to explain stress’ effect on human decisions. Much research demonstrates that acute stress alters decision-making, but outcomes are obscured by a range of methodological factors. Further, less is known about how stress affects decision-making in social contexts in which people so often act. This is of great importance in today’s increasingly complex social environment, replete with potential stressors, where cooperation and trust are critical. Here the aim was to explore acute stress’ effect on prosocial decision-making, while also controlling for methodological factors that may contribute to varied research outcomes. Ninety-six participants were exposed between-subjects to acute stressors with or without a significant social evaluative component, or a control procedure, after which they performed a variant of the Trust Game (a social decision-making task requiring cooperation and trust with a ‘partner’). Task performance occurred at different times with respect to exposure to examine the roles of temporally distinct biological stress pathways. Overall acute stress was associated with reduced trust, but a more complex pattern emerged when accounting for individual differences in physiological stress responses via multivariate analysis. In keeping with the complexity of stress itself, acute stress may enhance or reduce propensity to trust based on an individual’s unique pattern of physiological reactivity.

1. Introduction

In everyday conversation the term ‘stress’ often has a negative connotation, perhaps related to the unpleasantness of work or a difficult life experience. Despite its aversive nature, stress is of critical survival value – engaging evolutionarily ancient biological systems facilitating adaptive reactions in a changing environment (Dickerson and Kemeny, 2004). ‘Adaptive’ in the evolutionary sense, however, does not easily translate to describe stress’ effect on human decision-making. A growing literature on the topic has yielded variable outcomes, potentially related to divergent methodologies between-studies in operationalization of the stress construct. Further, focus on individual performance outside the social context in which many real-world decisions are made limits external validity. In today’s increasingly complex social environment, replete with diverse stressors, clarifying stress’ influence over social decision-making is of growing importance. Thus, the current study was designed to explore acute stress effects on decision-making in a social context while also controlling for a range of methodological

elements likely to contribute to variable outcomes in past research.

The ‘stress response’ represents coordinated activation of multiple biological systems by stimuli (stressors) signifying actual or perceived homeostatic disruption (Ulrich-Lai and Herman, 2009). While chronic exposure is associated with poor health outcomes (McEwen, 2007), acutely a ‘neuro-symphony’ of stress responses promotes modulation of behavioral, cognitive, and affective processes supporting an organism’s capability to adaptively react to, and learn from, a changing environment (Joëls and Baram, 2009). Examples associated with acute stress’ rapid engagement of the sympathetic-adrenal-medullary (SAM) axis and related catecholamine release include the classic ‘fight-or-flight’ response and, more recently proposed, a shift in neural processing from executive towards salience networks supporting vigilance and fear (Hermans et al., 2014). Concurrently, but at a slower pace, signals of homeostatic disruption originating in the brainstem can trigger the hypothalamic-pituitary-adrenal (HPA) axis and corticosteroid (e.g., cortisol) secretion, which may contribute both to synergistic potentiation of short-term SAM effects and their eventual reversal as

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homeostatic balance is restored.

While this simplified description is far from complete (for in-depth review, see Joëls and Baram, 2009; Ulrich-Lai and Herman, 2009), stress' SAM/HPA correlates are a sound starting point for isolating methodological elements likely to influence research outcomes. First, the degree to which a stressor involves processive versus systemic elements may influence the extent of SAM and/or HPA reactivity via engagement of partially dissociable neural circuits (Herman and Cullinan, 1997). Systemic stressors (i.e., representing an immediate physiologic threat) are brainstem-mediated and robustly engage SAM but not necessarily HPA, whereas processive stressors (i.e., involving psychological or psychosocial components such as social evaluative threat) require limbic engagement in threat appraisal to trigger HPA activation (and tend to do so strongly once engaged; Schwabe et al., 2008). Though a clean dissociation between the two is not possible in that techniques used in human laboratory-based stress research often unavoidably involve both, a useful methodological distinction is whether or not a significant processive component is present operationally. Second, interactions between SAM and HPA stress responses (e.g., catecholamines and corticosteroids respectively) may also vary depending on stress-to-task latency in that the temporal profile of SAM versus HPA reactivity differs post-exposure (SAM is rapid and quickly returns to baseline, HPA exhibits a slower rise peaking at 21–40 min.; Dickerson and Kemeny, 2004). Third, significant individual differences exist in reactivity among these same stress responses (and even when acquired they generally are not analyzed in conjunction with behavioral performance in a multivariate fashion).

The stress response's biological complexity manifests strongly in research on acute stress and decision-making. In studies examining non-social decision-making, which often employ financial risk-taking and/or reward seeking as a decision-making proxy, outcomes are mixed regarding both direction and existence of acute stress effects (Porcelli and Delgado, 2017). A recent meta-analysis involving laboratory-based acute stress induction and decision-making under uncertainty may be useful for delineating between-study methodological differences (Starcke and Brand, 2016). Among other factors the authors accounted for stressor type (primarily processive or systemic) while also exploring moderation of acute stress effects by HPA and/or SAM reactivity, and stress-to-task latency. In tasks where it could be considered disadvantageous, reward seeking and risk-taking were increased after exposure to acute stressors considered primarily processive (but not systemic) and no evidence of moderation by HPA/SAM reactivity or latency was observed. That said, few of the incorporated studies assessed HPA/SAM reactivity; contribution of individual differences along those lines could not be ruled out. Further, the need for experiments manipulating stress-to-task latency was highlighted because large between-study differences were evident (ranging from 0 to 55 min.; $M = 18.10$, $SD = 13.66$). While this preliminary evidence would seem to support the proposal that acute processive stress can influence decision-making, it is clear that research involving experimental manipulation and assessment of such factors at the within-study level is needed.

It is noteworthy that none of the studies examined above involved decision-making in a social context (in which real-life decision-making so often occurs). Social decision-making often engages prosocial behaviors, voluntary actions that benefit others (e.g., cooperation, altruism, trust; Eisenberg, 1982). While some offer return benefits (e.g., 'selfish' cooperation in expectation of reciprocity), others involve no expectation of reciprocation (e.g., 'unselfish' cooperation and altruism). As in the non-social decision-making literature, economics-inspired tasks have proven useful in exploring prosocial behavior in lab-based experimental research. For example, in the well-known Dictator Game (Kahneman et al., 1986) participants endowed with an amount of money are tasked with dividing it between themselves and another person in whatever way they see fit (the amount of money shared, if any, interpreted as a measure of unselfish behavior or altruism).

Similarly, the Ultimatum Game (Guth et al., 1982) assesses altruistic punishment (i.e., punishing others at a cost to oneself for violating social norms) by tasking participants with dividing an endowment as above with a partner who can either accept or reject it.

Recent research suggests that individuals exposed to acute stress differ in their propensity to engage in prosocial decisions as compared to controls. In the Dictator Game acutely stressed participants (via Trier Social Stress Task for groups; TSST-G) share significantly less than do their non-stressed counterparts (Vinkers et al., 2013). Further, such effects may be particularly sensitive to stress-to-task latency. In the Ultimatum Game acutely stressed participants reject unfair offers at rates similar to controls when deciding immediately after exposure, but are significantly more likely to accept them 75 min. after stress (Vinkers et al., 2013). Time-dependent acute stress effects have also been observed in the Dictator Game in social discounting (i.e., the inverse relationship between generosity and social 'distance'). Participant's exhibit increased sharing with socially 'close' but not 'distant' partners 20 min. after exposure, but no significant difference at 90 min. (Margittai et al., 2015). Such subtle stress-related shifts in prosocial behaviors may be linked with acute stress HPA reactivity (Margittai et al., 2018), which speaks to the importance of controlling for stress-to-task latency and stress-related physiological reactivity.

The current study focused on trust, a prosocial behavior in which an individual makes themselves vulnerable in expectation of the positive behavior of another (involving both risk and interdependence; Rousseau et al., 1998). In connection with non-social decisions trust may serve as a risk-taking analogue (Das and Teng, 2004), though behavioral and neurobiological evidence suggest they are not identical constructs (Fehr, 2009). As above trust can be modelled via economic paradigms such as the well-known investment or 'Trust Game' where participants are endowed with money they may decide to keep or invest with a partner entirely or in part in expectation of a reciprocal return (Berg et al., 1995). Any amount invested is multiplied by some factor to yield a new amount the partner may then keep or partially return. In satisfying the requirements of both risk (a reciprocal return may not be received) and interdependence (the outcome relies on another), decisions to invest and investment amount represent operationalization of trust (Camerer, 2003).

Few studies have examined Trust Game performance under acute stress, with mixed results as in non-social contexts. Both decreased (FeldmanHall et al., 2015; Steinbeis et al., 2015) and increased (von Dawans et al., 2012) trust after acute stress exposure have been reported, though previously discussed methodological elements may have influenced outcomes. Two involved stressors with a significant processive component via social evaluation (i.e., Trier Social Stress Task for Groups) and task performance at short stress-to-task latencies (Steinbeis et al., 2015; von Dawans et al., 2012), whereas the third primarily a systemic stressor at longer latency (i.e., cold pressor test; FeldmanHall et al., 2015). That said, the direction of reported acute stress effects does not cleanly map onto these distinctions and dissociation along those lines was not incorporated into their experimental methodology. Finally, while both SAM and HPA stress responses were assessed in all three studies they were not analyzed in conjunction with behavioral data in a multivariate fashion to assess the role of individual differences in physiological reactivity or interactive effects.

In a move towards integration, the current study examined acute stress effects on propensity to trust while also controlling for stressor type, stress-to-task latency, and individual differences in stress responses. Participants were exposed between-subjects to one of two acute stressors (without or with a significant processive component via social evaluative threat) or a control procedure, at different latencies with respect to performance of a Trust Game variant to capture SAM versus HPA-based stress effects. Individual differences in SAM and HPA stress responses were assessed via skin conductance and salivary cortisol, analyzed independent of Trust Game performance via traditional ANOVA-based approaches as well as in combination with it using a

multivariate approach novel in this context (logistic regression via the Generalized Estimating Equations method). It was hypothesized that acute stress would modulate decisions to trust, and that this effect would be most evident in participants who exhibited robust stress-related SAM and HPA reactivity (particularly at a delay with respect to exposure).

2. Methods

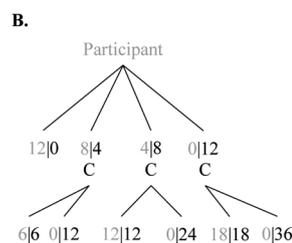
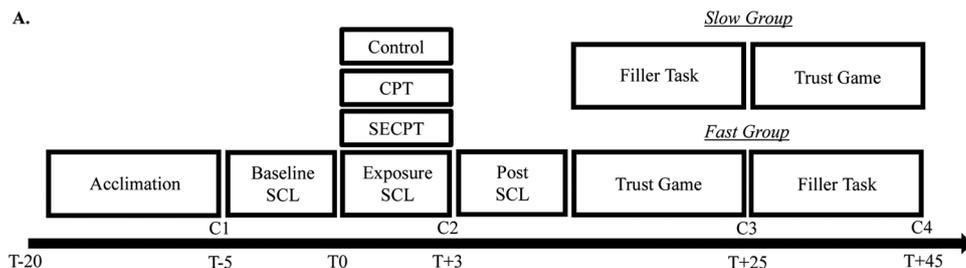
2.1. Participants

Ninety-six participants completed the current study (47 females, 49 males; mean age = 18.75, SD age = 0.98). Exclusionary criteria included history of psychiatric or neurological illness, nicotine use, or refusal to refrain from alcohol/caffeine use for 24 h prior to testing. Additional stress-related exclusions included cardiovascular illness, chronic rheumatologic disease, diabetes, Reynaud’s Disease, and Cold Urticaria. Female participants using hormonally active contraceptives were excluded and tasks of central interest were scheduled during female participant’s luteal menstrual phase (when stress-related free cortisol elevations are less likely to differ by sex; Kirschbaum et al., 1999). Study procedures were approved by the Marquette University Institutional Review Board (IRB) and all participants gave informed consent. Importantly, participants were told they would receive actual monetary compensation based on task performance.

2.2. Procedure

2.2.1. Overall experimental timeline

Study procedures took place over two days. On Day 1 participants gave informed consent, confirmed that exclusionary criteria did not apply, and completed a battery of surveys. These included the North American Adult Reading Test as a measure of premorbid intellectual ability (Uttl, 2002), the General Trust Scale (to assess participants’ propensity to trust based on beliefs about the honesty and trustworthiness of others; Yamagishi and Yamagishi, 1985), and a lottery-based financial survey assessing relative risk preferences (Holt and Laury, 2002). Day 2 sessions were conducted between 12 and 5 pm to control for circadian fluctuations in cortisol. To create the impression that Day 2 Trust Game interactions were with a real-life partner, study personnel escorted participants to a testing room after briefly introducing them to another individual in the waiting room as their ‘partner’ (in actuality, a confederate; see Fig. 1A for a Day 2 experimental timeline).



2.2.2. Acute stress induction

Participants were assigned between-subjects to one of three stress groups: traditional cold pressor test (CPT), socially evaluated cold pressor test (SECPT), or a no-stress control. CPT, primarily a systemic stressor, involved immersion of participants’ non-dominant hands in ice water between 2 and 4 °C for three minutes (Lovallo, 1975). Importantly, though investigators remained in the room during CPT exposure to assess compliance with hand immersion to minimize perceived social evaluation by study personnel investigators were trained to remain as unobtrusive as possible during the procedure (e.g., facing generally away from the participant, avoiding direct eye contact after the CPT began, maintaining a neutral demeanor). SECPT involved a variant of the traditional CPT incorporating social-evaluative elements (e.g., observation by investigators in white lab coats, video recording, and notetaking; Schwabe et al., 2008, 2012). Thus, SECPT represented a systemic stressor with the addition of a significant processive component. Finally, control participants simply immersed their hand in room temperature water for three minutes with no observation.

To dissociate stress-to-task latency effects of rapidly engaged SAM reactivity versus the slower activating HPA on performance, participants were randomly assigned between-subjects to a ‘fast’ or ‘slow’ group. For fast group participants, behavioral task performance occurred immediately following induction (i.e., in theory near peak SAM reactivity before HPA-based cortisol release could reach peak levels). In the slow group, participants performed a neutral filler task (i.e., reading emotionally neutral material) immediately after induction followed by task performance approximately 25 min. post-exposure (allowing time for cortisol to reach peak levels). Stress by latency group cell sizes of the final sample were as follows: control fast $n = 17$, control slow $n = 14$, CPT fast $n = 16$, CPT slow $n = 16$, SECPT fast $n = 17$, SECPT slow $n = 16$. At the end of Day 2 a post-experimental questionnaire was administered to assess perceptions of the experiments’ social context and ratings of the induction procedure to which participants had been exposed based on 7-point Likert scaled questions rating negative affect (how the procedure made them feel from ‘good’ to ‘bad’) and subjective stress (how much stress they experienced from ‘none’ to ‘very much’).

2.2.3. The Trust Game task

Participants were assigned the role of ‘investor’ in the Trust Game (this term was not used explicitly). They were endowed with \$12 on every new trial and instructed to invest either \$0, \$4, \$8, or \$12 in the ‘trustee’ (portrayed as the confederate, in actuality a preprogrammed series of responses). They then received feedback as to whether the trustee had decided to keep the money or return 50%. Any amount the investor decided to pass to the trustee was tripled in value to maximize

Fig. 1. Experimental timeline and Trust Game payoff structure. (A) Day 2 timeline including acute stress induction and Trust Game performance (T = time, C = cortisol sample). (B) Numbers in gray indicate the amount participants received for each decision option, black the amount the confederate/computer received. When the computer reciprocated after a participant invested, total winnings for a given trial would be the sum of both dollar amounts in red (i.e., the amount the participant did not invest initially and the amount returned, if any).

potential gains. Thus, participants could earn more money if they decided to trust *and* the trustee reciprocated than if they had not invested to begin with (see Fig. 1B for task payoff structure). If the trustee did not reciprocate, the participant/investor earned only the amount they had kept of the original endowment (if any).

Participants proceeded through 36 trials each with 4000 ms to make their investment decision and a 2000 ms fixation between their choice and receipt of feedback. When the participant decided to keep the endowment, that choice was confirmed (4000 ms) and the next trial began. To enhance the impression of a real social interaction when participants decided to invest a jittered interval ensued while the ‘partner’ made their decision (3000–7000 ms), after which the investor received feedback as to whether the trustee had reciprocated or kept the money (4000 ms). Each trial was followed by a 5000 ms intertrial interval. Total task time varied depending on the frequency of ‘invest’ to ‘keep’ decisions with a maximum of 13 min. and 20 s.

2.2.4. Stress response measures

To assess engagement of stress-related biology, during Day 2 skin conductance and salivary cortisol were measured (i.e., reflecting SAM and HPA stress correlates respectively). Skin conductance data were continuously recorded throughout exposure to the acute stress or control procedures using a BIOPAC MP150 system (BIOPAC Systems Inc., Goleta, CA). During analysis, skin conductance levels (SCL) were calculated as the average of the SCL waveform in microsiemens (μS) binned in three 3-min. segments surrounding exposure (i.e., Baseline, Exposure, Post-Exposure) for each stress group (i.e., Control, CPT, SECPT). Salivary cortisol samples were collected using a SalivaBio Oral Swabs (Salimetrics, LLC, State College, PA) placed in freezer storage at $-20\text{ }^{\circ}\text{C}$ prior to assaying. Samples were collected at four points during the experimental timeline: baseline (after acclimation to the lab environment prior to initiation of study procedures), immediately following completion of the randomly assigned stress induction procedure, 25-min. post-exposure, and 45-min. post-exposure. Both intra- and inter-assay coefficients were within acceptable limits (well below 10 and 15% respectively; Schultheiss and Stanton, 2009). Note, SAM and HPA ‘reactivity’ measures referred to in the results represent peak-to-baseline differences in stress response measures (for SAM the z-standardized difference in SCL between exposure and baseline measurements; for HPA the z-standardized difference in salivary cortisol between the 25-min. post-exposure and baseline samples).

2.3. Statistical analyses

2.3.1. ANOVA analyses

Initial analyses were conducted using IBM SPSS Statistics for Windows 24. Central study dependent measures (surveys, subjective stress measures, cortisol, skin conductance, Trust Game) were analyzed separately via a series of analyses-of-variance (ANOVAs). All results presented save one exception met ANOVA assumptions of independence, normality of dependent variable residuals, outliers, and homoscedasticity; some after application of an appropriate transformation (detailed in Sec. 3). Greenhouse-Geisser corrections were applied in any mixed ANOVA for which the sphericity assumption was violated (yielding adjusted *df*). To reduce Type I error inflation, a limited number of Bonferroni-Holm corrected planned t-tests were conducted to explore significant ANOVA interactions (Holm, 1979). Spearman correlations were performed between subjective ratings of negative affect and stress, and other measures (due to a computer error rating data were unavailable for 6 participants, leaving 90 for this purpose). The sole exception mentioned above involved a mixed ANOVA performed on the proportion of times participants chose to invest at different investment amounts during the Trust Game (e.g., \$0, \$4, \$8, and \$12); dependent variable residuals departed from normality in one subset of independent factor combinations. Thus, significant main effects and interactions associated with this analysis were verified

nonparametrically via aligned rank transform for factorial designs with repeated measures (ARTool; Feys, 2016).

2.3.2. Ordinal logistic regression via generalized estimating equations

Examination of individual differences in SAM and HPA stress responses in combination with Trust Game performance posed some thorny analysis issues in that (a) skin conductance and salivary cortisol variables were continuous, (b) Trust Game data were not independent (36 repeated measurements), and (c) the outcome variable was non-normal (decisions to invest \$0, \$4, \$8, or \$12 were ordinal or, if aggregated, proportional). Under strict distributional assumptions ANOVA can accommodate repeated-measures data, but not continuous covariates as predictors of interest to explore interactive effects (only as control variables, as in ANCOVA). Further, use of ANOVA given a categorical/proportional dependent variable may be invalid in some cases (for review, see Warton and Hui, 2011) and in this case would have required sacrificing important systematic variance by dichotomizing individual difference stress response variables (Fitzsimons, 2008).

The Generalized Estimating Equations (GEE; Zeger and Liang, 1986) method offers a viable analysis approach under these constraints. Commonly used in clinical and biomedical longitudinal research, GEE is a marginal (or, population-average) approach which extends many commonly used regression approaches to non-normal and non-independent data (e.g., linear regression, Poisson regression, logistic regression; Zeger and Liang, 1992). Thus, it is well-suited for analysis of experimental repeated-measures decision-making data (Agresti, 2012; Crockett et al., 2010). In the current study GEE with robust standard error estimation was employed to develop an ordinal logistic model predicting propensity to engage in trust (i.e., the investment amount dependent variable) by the independent variables of: (1) stress-to-task latency, (2) SAM reactivity, and (3) HPA reactivity (critically, stress group itself was not modelled). All GEE analyses were performed in R (version 3.2.5), using the *repolr* package to test the proportional odds assumption (Parsons et al., 2009) and the *multgee* package to perform the analysis (Touloumis, 2015). Note, additional information is included in article supplementary material.

GEE-based models and associated parameters are interpreted in the same way as any other regression-based analysis (save at the population rather than individual level). In logistic regression the dependent variable is not modelled directly, rather the odds of an outcome being a ‘success’ or ‘failure’ (O’Connell, 2006). As in the ordinal case the dependent variable has more than two inherently ordered levels, a cumulative logit link is used (Menard, 2002) and ordered splits/categories are iteratively fitted along the ordinal levels (each modelled separately yielding multiple sets of regression parameters and, thus, regression equations; O’Connell (2006). For example, based on the dependent variable here model parameters fell along three splits/categories: investing \$12 versus \$0, investing \$12 or \$8 versus \$0, the third investing at all (\$12, \$8, or \$4) versus \$0. Each can be likened to a single binary logistic regression where the binary event being predicted is category membership. Under proportional odds, intercepts associated with each equation vary but the slopes of estimated parameters do not (Williams, 2016). Thus, only one need be examined in detail (here, the third associated with investing \$12, \$8, or \$4 versus \$0; operationally, trust versus no trust). For model interpretation the resulting regression equation was used to generate predicted log odds at various independent variable values (see Sec. 3.4) and these were then converted directly into easily interpretable predicted probabilities with standard errors calculated via the delta method (Oehlert, 1992).

3. Results

3.1. Surveys and subjective stress ratings

To verify that participants did not differ between acute stress groups with respect to intellectual ability (NAART), propensity to trust

(General Trust Scale), or relative risk preference, three one-way ANOVAs were performed (Stress Group: Control vs. CPT vs. SECPT). No such differences were observed (NAART: $F(2, 93) = 1.02, p > .15, \eta_p^2 = .021$; General Trust Scale: $F(2, 92) = 2.34, p > .10, \eta_p^2 = .048$; relative risk preference: $F(2, 93) = 0.96, p > .15, \eta_p^2 = .02$). Note, the sample as a whole was risk-averse (60.42%; 11.5% risk-seeking, 28.12% risk-neutral). While these measures were included as covariates in initial analyses, they did not significantly contribute (neither direction nor significance of results was altered) and they were excluded from final analyses.

Two additional one-way ANOVAs with stress group as the independent variable examined participants' subjective reactions to their randomly assigned induction procedures. A significant effect of stress group on subjective stress levels was observed, $F(2, 87) = 53.42, p < .001, \eta_p^2 = .551$. Holm-Bonferroni corrected t-tests indicated that groups exposed to an acute stressor (i.e., CPT and SECPT) reported significantly higher stress after exposure than did controls, $t(46.016) = 9.90, p < .001, d = 2.55$ and $t(50.682) = 10.59, p < .001, d = 2.71$ respectively. The CPT and SECPT groups did not, however, significantly differ from one another, $t(61) = 0.22, p > .15, d = 0.06$. An identical pattern was observed with respect to negative affect, $F(2, 87) = 69.56, p < .001, \eta_p^2 = .615$. CPT and SECPT participants reported significantly greater negative affect over controls, $t(56) = 10.00, p < .001, d = 2.61$ and $t(57) = 9.80, p < .001, d = 2.53$ respectively. Again, the CPT and SECPT groups did not significantly differ, $t(61) = 0.49, p > .15, d = 0.13$. While five participants expressed uncertainty as to whether their financial interactions with the confederate during the Trust Game were 'real', when excluded from analyses direction and significance of results were unchanged. Therefore, final analyses included all participants.

3.2. Stress response measures

To examine acute stress effects on SAM engagement, a 3 (Stress Group: Control, CPT, SECPT) \times 3 (Exposure Phase: Baseline, Exposure, Post-Exposure) \times 2 (Latency: Fast, Slow) \times 2 (Sex: Female, Male) mixed ANOVA was performed on square-root transformed skin conductance levels (SCL). Confirming SAM engagement, a significant stress group by exposure phase interaction was observed, $F(3.69, 154.89) = 10.48, p < .001, \eta_p^2 = .200$. Holm-Bonferroni corrected t-tests indicated a significant SCL increase from baseline to exposure for CPT and SECPT participants only, $t(31) = 6.75, p < .001, d = 0.36$ and $t(33) = 5.69, p < .001, d = 0.36$ respectively. In contrast, controls exhibited a significant SCL decrease from baseline to exposure, $t(30) = -4.66, p < .001, d = -0.11$. Comparison of SCL reactivity (i.e., change from baseline to exposure) between the CPT and SECPT groups demonstrated no significant difference in SAM reactivity by stressor type, $t(63) = 1.43, p > .15, d = 0.36$, nor were sex effects observed. Notably, subjective ratings of both negative affect and stress positively correlated with SAM reactivity, $r_s(88) = 0.58, p < .001$ and $r_s(88) = 0.65, p < .001$ respectively.

To assess HPA acute stress responses, log transformed salivary cortisol data were subjected to a 3 (Stress Group: Control, CPT, SECPT) \times 4 (Sample Time: Baseline, Post-exposure, 25 min. post, 40 min. post) \times 2 (Sex: Female, Male) \times 2 (Latency: Fast, Slow) mixed ANOVA. Four participants were missing samples due to insufficient quantity of saliva (thus, analysis included 92 of the 96 participants). A significant stress group by sample time interaction was observed, $F(3.03, 121.36) = 7.44, p < .001, \eta_p^2 = .57$ (see Fig. 2). No significant results were observed involving latency or sex. Cortisol significantly increased from baseline to 25 min. post-exposure (i.e., allowing for slower HPA cortisol peak) only in the CPT and SECPT groups; $t(31) = 2.84, p < .01, d = 0.42$; $t(32) = 2.27, p < .05, d = 0.52$ respectively. Conversely, controls exhibited a significant decrease in salivary cortisol from baseline to 20-min.utes post-exposure, $t(29) = -3.75, p < .005, d = -0.59$. As above for SCL, cortisol reactivity (i.e., from baseline to

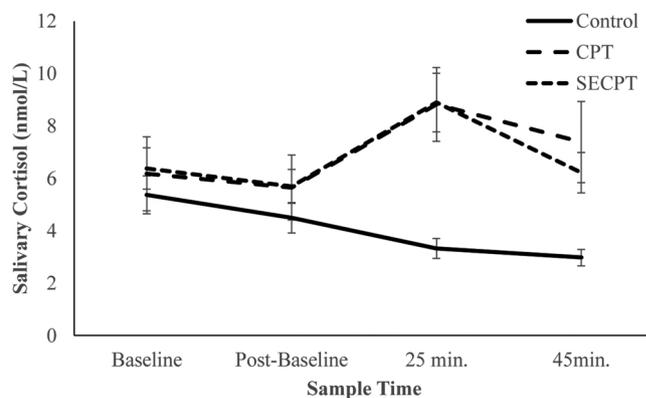


Fig. 2. Salivary cortisol data by stressor and sample. While both the CPT ($n = 32$) and SECPT ($n = 33$) groups exhibited significantly greater cortisol reactivity than controls ($n = 31$) from baseline to 25 min. post-exposure, they did not significantly differ from each other in terms of reactivity (figure uses raw cortisol data).

25 min post-exposure) between the CPT and SECPT groups did not significantly differ, $t(63) = 0.02, p > .15, d = -0.004$. Only 1 of the aforementioned 4 participants missing samples involved those required to calculate reactivity, thus all analyses involving cortisol reactivity (including the GEE analysis in Sec. 3.4) include 95 out of 96 participants. Again, subjective ratings of negative affect and stress positively correlated with HPA reactivity, $r_s(87) = 0.36, p < .001$ and $r_s(87) = 0.25, p < .05$ respectively.

Finally, CPT and SECPT participants were classified post-hoc as cortisol (non)responders based on a 1.5 nmol/l increase in cortisol reactivity (Miller et al., 2013). This yielded a relatively even split within each stress group (CPT, 50% responders; SECPT, 48.48% responders). Chi-square tests examining distribution of (non)responders by stress and latency groups were not significant, nonresponders $\chi^2(1, N = 33) = 0.036, p > .15$ and responders $\chi^2(1, N = 32) = 0.000, p > .15$. While two control participants (6.67%) exhibited a cortisol increase meeting this criterion, they were retained in analysis as exclusion did not alter direction or significance of results.

3.3. Trust Game ANOVA results

Over the 36 Trust Game trials participants invested an average of \$5.15 (± 2.36 SD) in their counterpart across stress group, latency, and sex. To examine the influence of those factors on decisions to invest (i.e., our operationalization of trust) a 3 (Stress Group: Control, CPT, SECPT) \times 2 (Latency: Fast, Slow) \times 2 (Sex: Female, Male) ANOVA was performed. While no significant interactions were observed, the main effect of sex was significant, $F(1, 84) = 5.39, p < .05, \eta_p^2 = .060$. Males invested more on average ($M = \$5.73, SD = 2.49$) than did females ($M = \$4.55, SD = 2.12$). Further, the main effect of stress group trended towards significance, $F(2, 84) = 2.60, p < .10, \eta_p^2 = .058$. CPT ($M = \$4.72, SD = 2.04$) and SECPT ($M = \$4.83, SD = 2.10$) participants did not significantly differ in amount invested, $t(63) = 0.21, p > .15, d = 0.05$, whereas both trended towards reduced investment compared to controls ($M = \$5.94, SD = 2.81$), $t(61) = -1.98, p < .10, d = -0.50$ and $t(62) = -1.80, p < .10, d = -0.49$ respectively.

Given no differential effects between the CPT and SECPT groups in either stress responses or amount invested, further analyses were conducted collapsing across those two groups (i.e., the between-subjects factor 'stress group' now represented exposure to acute stress of either type versus controls). An additional 2 (Stress Group: Control, Acute Stress) \times 2 (Latency: Fast, Slow) \times 2 (Sex: Female, Male) ANOVA yielded an identical main effect of sex but also a significant main effect of stress group, $F(1, 88) = 5.08, p < .05, \eta_p^2 = .055$. Participants exposed to

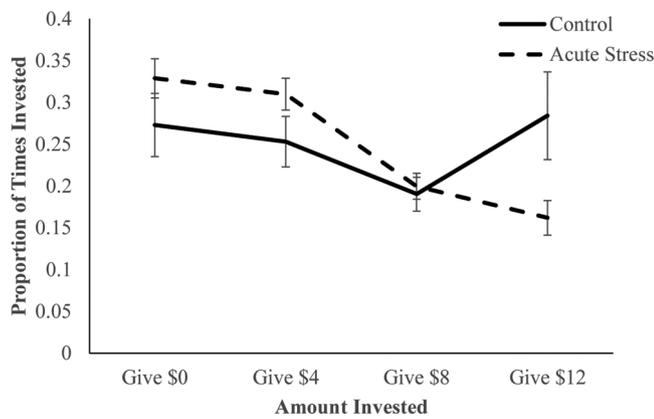


Fig. 3. Acute stress was associated with reduced investment/trust at high dollar amounts. Participants exposed to acute stress ($n = 65$) invested significantly less than controls ($n = 31$) at the highest (\$12) level (figure data are raw proportions).

acute stress invested significantly less than did controls, $t(94) = -2.24$, $p < .05$, $d = -0.46$, and investment negatively correlated with subjective ratings of stress intensity, $r_s(88) = -0.27$, $p < .01$.

Though average amount invested represented one operationalization of trust, it did not directly address how often participants chose to keep their entire endowment versus invest at various monetary amounts (i.e., \$0, \$4, \$8, or \$12). To explore this, proportions of trials participants invested at each level were calculated (accounting for null trials) and arcsine transformed (Cohen et al., 2003). A 4 (Amount: \$0, \$4, \$8, \$12) x 2 (Stress Group: Control, Acute Stress) x 2 (Latency: Fast, Slow) x 2 (Sex: Female, Male) mixed ANOVA was performed. As anticipated a significant main effect of investment amount was observed, $F(2.13, 187.53) = 4.23$, $p < .05$, $\eta_p^2 = .046$, characterized by decreased investment as dollar amount increased. Neither the four-way interaction nor any of the three-way interactions were statistically significant. Three significant two-way interactions manifested between (1) stress group and amount, $F(2.13, 187.53) = 4.13$, $p < .05$, $\eta_p^2 = .045$ (see Fig. 3), (2) sex and amount, $F(2.13, 187.53) = 4.34$, $p < .05$, $\eta_p^2 = .047$, and (3) latency and amount, $F(2.13, 187.53) = 3.29$, $p < .05$, $\eta_p^2 = .036$. As discussed in Sec. 2.3, main effects and interactions related to this analysis were verified non-parametrically via aligned rank transform for factorial designs with repeated measures using ARTool software (Feys, 2016). Level of statistical significance for all results remained the same save for the latency by amount interaction (thus, that interaction is not discussed).

Bonferroni-Holm corrected t-tests were used to examine significant two-way interactions (to limit the number of comparisons, at each amount along the between-subject factors involved in the interaction). In terms of the significant stress group by amount interaction, acutely stressed participants invested significantly less at the \$12 amount than did controls, $t(41.54) = -2.10$, $p < .05$, $d = -.49$, and subjective ratings of stress intensity negatively correlated with the proportion of times participants gave \$12, $r_s(88) = -0.28$, $p < .01$. In contrast, acutely stressed participants demonstrated increased investment over controls at \$4, $t(94) = 2.03$, $p < .05$, $d = .42$ (but this did not survive correction), and no significant differences were observed at the \$0 or \$8 amounts, $t(94) = 1.52$, $p > .10$, $d = .32$ and $t(94) = 0.24$, $p > .15$, $d = .05$ respectively. With respect to amount and sex, males invested \$4 significantly less often than did females, $t(94) = -2.75$, $p < .01$, $d = -.56$, but significantly more often at the \$12 level, $t(94) = 2.92$, $p < .01$, $d = .60$.

3.4. Exploratory analysis: ordinal logistic regression via GEE

A test of the proportional odds assumption central to ordinal logistic regression indicated the assumption was met, $\chi^2(14) = 10.19$,

$p > .15$. Further, missed trials were negligible (only 2.2%; Bennett, 2001). Examination of the within-subjects association pattern underlying repeated Trust Game decisions revealed a complicated structure (ϕ ranged from -0.03 to 0.80), thus a category exchangeability structure was employed (Touloumis, 2015). The model was clustered by participant and trial order with stress-to-task latency as a categorical factor (fast as the reference group), as continuous covariates z-standardized SCL and salivary cortisol reactivity (representing SAM and HPA reactivity respectively; zSCL and zCORT). Because the goal was to explore the influence only of physiological reactivity and latency on propensity to trust, the independent variables of stress group and sex were omitted. That said, both stress response variables were consistent with the experimental stress group manipulation descriptively (zSCL control: $M = -0.85$, $SD = 0.38$; zSCL acute stress: $M = 0.41$, $SD = 0.95$; zCORT control: $M = -0.50$, $SD = 0.51$; zCORT acute stress: $M = 0.23$, $SD = 1.08$). Finally, it should be noted that traditional goodness-of-fit statistics are not available for GEE ordinal models because they are based on quasilielihood estimation (Zorn, 2001), and those developed for GEE have not yet been extended to the ordinal case (Ballinger, 2004). That said, asymptotically equivalent Wald tests of overall significance against the null model are available and provided below.

Initially a main effects only model including latency, zCORT, and zSCL was performed but found to be nonsignificant, $Wald\ p > .15$. In contrast, a full factorial model involving those three variables was significant, $Wald\ p < .0001$. The factorial exhibited significantly better fit than the main effects model, $Wald\ \chi^2(4) = 23.79$, $p < .0001$. Further, the predicted three-way interaction among latency, zSCL, and zCORT was significant, $Wald\ \chi^2(1) = 19.86$, $p < .0001$ (see Table 1 for the full model results). This yielded the following equation:

$$\text{Logit}[P(Y \leq c)] = \theta_c + 0.065_{\text{Timing}} + 0.343_{\text{zCORT}} - 0.133_{\text{zSCL}} - 0.428_{\text{Timing} * \text{zCORT}} - 0.063_{\text{Timing} * \text{zSCL}} - 0.099_{\text{zCORT} * \text{zSCL}} + 0.983_{\text{Timing} * \text{zCORT} * \text{zSCL}}$$

Table 1
GEE Ordinal Logistic Model Predicting Trust by Stress-to-task Latency, Cortisol and Skin Conductance Level Reactivity ($N = 95$).

	B	SE(B)	95% CI	Wald χ^2	z	p-value	OR
Category Intercepts							
(1) Give \$12	-1.34 [†]	0.20	-1.72 -0.96	46.98	-6.85	< .0001	0.26
(2) Give \$8/\$12	-0.39 [†]	0.17	-0.72 -0.06	5.36	-2.31	.02	0.68
(3) Give \$4/\$8/\$12	0.81 [†]	0.15	0.51 1.11	27.93	5.29	< .0001	2.25
Variable							
Latency (Slow)	0.06	0.25	-0.43 0.56	0.07	0.26	.80	1.07
zCortisol	0.34 [*]	0.15	0.06 0.63	0.62	2.36	.02	1.41
zSCL	-0.13	0.17	-0.46 0.2	5.57	-0.79	.43	0.88
Latency x zCortisol	-0.06	0.30	-0.66 0.53	0.04	-0.21	.84	0.94
Latency x zSCL	-0.43 [*]	0.21	-0.84 -0.02	4.17	-2.04	.04	0.65
zCortisol x zSCL	-0.10	0.11	-0.31 0.11	0.83	-0.91	.36	0.91
Latency x zCortisol x zSCL	0.98 [†]	0.22	0.55 1.42	19.86	4.46	< .0001	2.67

Note: B = unstandardized coefficient; SE = standard error; OR = odds ratio; CI = confidence interval, zCortisol = z-standardized cortisol reactivity, zSCL = z-standardized SCL reactivity. Latency was presented with the fast group as the reference.

* $p < .05$.

† $p < .0001$.

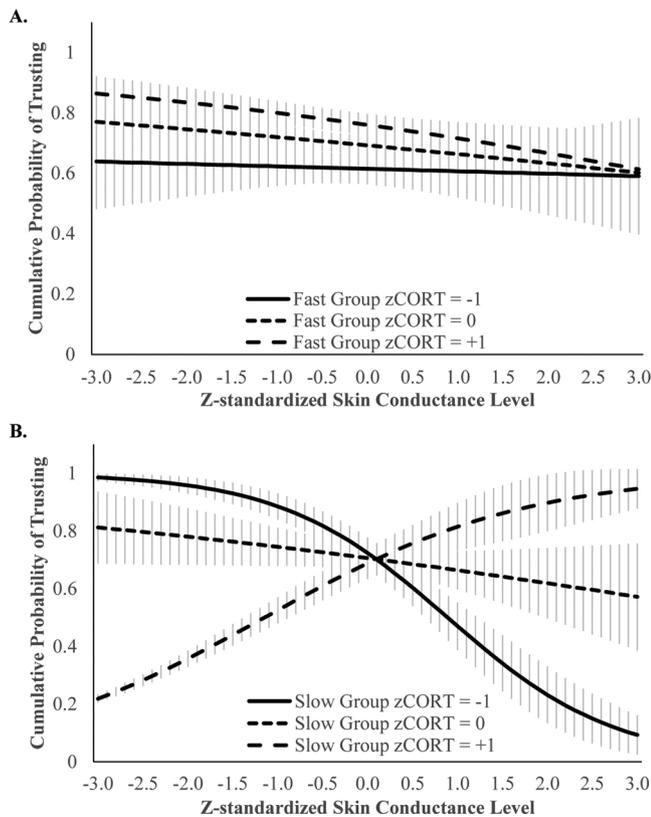


Fig. 4. GEE ordinal logistic regression predicted cumulative probability of trust. (A) Fast group ($n = 49$) cumulative probability of trust declines linearly with increasing SAM reactivity. (B) Slow group ($n = 46$) cumulative probability of trust declines linearly with increasing SAM reactivity for $zCORT = 0$, but diverges from this pattern for $zCORT \pm 1$. Bars represent prediction standard errors.

$zCORT * zSCL$

where $P(Y \leq c)$ is the cumulative probability of investing at or below a given category, and θ_c represents the three category intercepts. To explore the three-way interaction the equation above was used to generate predicted log odds for the third logit based on (a) latency group (0 or 1), cortisol reactivity ($zCORT$ held at -1 , 0 , and $+1$), and SCL reactivity (varying $zSCL$ between ± 3 in 0.1 increments). Predicted log odds were then converted to cumulative probabilities (Muller and MacLehose, 2014) and standard errors calculated via the delta method (Oehlert, 1992). Critically, the resulting cumulative probabilities represent predictions of propensity to trust at different stress-to-task latencies and varied levels of SAM and HPA reactivity.

Fast group cumulative probabilities (propensity to trust) decreased linearly as $zSCL$ reactivity increased, and closely overlapped at all three $zCORT$ levels (i.e., greater SAM engagement predicted reduced trust regardless of HPA activation as expected given fast group $zCORT$ levels could not have plausibly peaked; see Fig. 4A). In the slow group (where HPA reactivity could most plausibly be associated with a biologically active cortisol increase), the cumulative probability associated with no HPA reactivity ($zCORT = 0$) exhibited an identical pattern (see Fig. 4B). It was only at nonzero levels of $zCORT$ reactivity that slow group predictions significantly diverged from the prevalent inverse trust/ $zSCL$ relationship (see Fig. 5 for slow group predictions including 95% confidence intervals). At lower slow group HPA reactivity levels ($zCORT = -1$) the model predicted an exaggerated form of the aforementioned relationship. Only under robust elevation of both $zSCL$ and $zCORT$ was a new reversed pattern predicted, characterized by enhanced likelihood to engage in trust when both SAM and HPA were synergistically engaged.

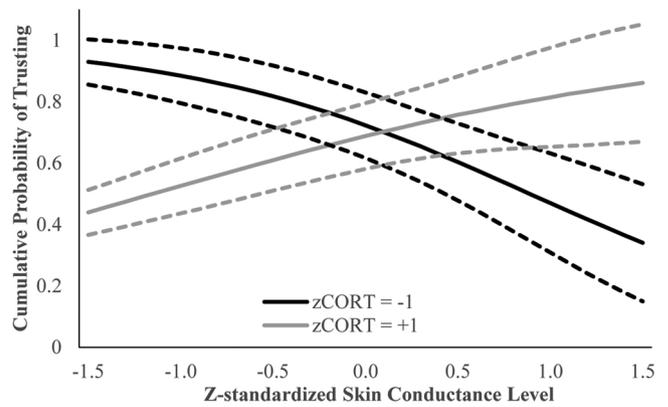


Fig. 5. Slow group predicted cumulative probability of trusting at $zCORT \pm 1$ only. Dotted lines represent 95% confidence intervals. At lower $zCORT$ increased trust was predicted if $zSCL$ was also low, the reverse when $zSCL$ was elevated. At higher $zCORT$ the opposite pattern is predicted, reduced trust at lower $zSCL$ but increased when both SAM and HPA are synergistically engaged.

4. Discussion

In this experiment we examined acute stress effects on trust in prosocial decision-making while controlling for stressor type, stress-to-task latency, and individual differences in physiological reactivity. Neither SAM nor HPA reactivity differed by stressor type; acute stressors with and without a significant processive component were associated with elevated physiological reactivity in both axes. Employing ANOVA-based analyses decisions during the Trust Game did not vary by stressor type or stress-to-task latency, though acute stress overall was associated with reduced trust. Modelling these data via a multivariate approach indicated the nature of this interaction was more complex, characterized by modulation of the direction and magnitude of predicted trust based on distinct and time-dependent profiles of SAM/HPA reactivity. All but one profile was associated with a decrease in trust with increasing acute SAM reactivity, but at longer stress-to-task latencies under robust SAM and HPA engagement an increase in trust was predicted.

Using analysis approaches comparable to most studies examining acute stress and decision-making our data would suggest a general acute stress-related decrease in trust. This is consistent with two of the aforementioned studies examining Trust Game performance under acute stress (FeldmanHall et al., 2015; Steinbeis et al., 2015) but not the third (von Dawans et al., 2012). That said, methodological differences in stressor type and latency obscure clear cross-study comparisons. Accounting for those factors here, acutely stressed participants did trust significantly less on average than controls. While largely consistent with ANOVA outcomes in that decreased trust was predicted in the majority of cases under biological (i.e., SAM) conditions typically classified as acutely stressful, the GEE ordinal logistic approach yielded a more parsimonious model consistent with all three aforementioned studies (and thus may be better-suited for characterizing variance in data comprised of both biological and behavioral measures). That acute stress or its SAM/HPA correlates might differentially influence behavior at varied latencies is biologically plausible, and has been reported before (e.g., Margittai et al., 2015; Pabst et al., 2013). Prediction of choice based purely on the temporal profile of physiological reactivity, however, represents a novel step forward in examining stress effects on decision-making.

The ordinal logistic model predicted reduced trust at both stress-to-task latencies and all stress response profiles save one (i.e., high SAM and HPA reactivity at long stress-to-task latencies). Namely, a decline in likelihood to trust as SAM engagement increased. Turning to the raw data for descriptive purposes, this is also evidenced by a negative correlation between SCL reactivity and the overall proportion of times

participants invested, $r(93) = -0.25, p < .05$. In that the opposite pattern was predicted under the long latency high SAM/HPA reactivity profile, at least two salient questions arise with respect to potential mechanisms. Exploring intersections between the neuroscientific stress literature and a growing body of social neuroscientific research may yield useful insights along these lines, though we acknowledge in that the current study did not incorporate neuroscientific measures we cannot fully address either question. Thus, we will limit our interpretation only to broad points useful for hypothesis generation and future directions.

First, why might increased SAM engagement be associated with reduced trust when HPA reactivity may not be involved (i.e., at short latencies before exposure-related changes in cortisol reactivity were biologically plausible, and at long latencies in the presence of a ‘flat’ cortisol reactivity profile)? One plausible explanation relates to the temporal dynamics of SAM and HPA associated stress responses and their interactions with neural circuits supporting social decision-making, which a range of research suggests is comprised of partially overlapping networks including (but necessarily not limited to) those involved in executive control, salience, reward processing, and social cognition (for review, see Adolphs, 2001; Bhanji and Delgado, 2014; Ruff and Fehr, 2014; Sanfey, 2007; Yoder and Decety, 2018). Acute stress-related SAM engagement has been associated with increased catecholamine release (e.g., norepinephrine and dopamine), impairing prefrontal neuronal firing and top-down control (characterized as promoting short-term engagement of salience over executive control networks, promoting fear and vigilance; Arnsten, 2015; Hermans et al., 2014). Thus, SAM-associated reduced trust may be linked to a shift towards salience network processing after acute stress exposure (though potential stress effects in neural circuits supporting social cognition and reward processing may also play a role). In that vein an alternative, but not mutually exclusive, interpretation is that greater SAM engagement modulates reward processing (Starcke and Brand, 2012) by reducing sensitivity to feedback associated with social rewards inherent to investing/trusting (Sanfey, 2007) and/or reciprocal returns after investment (Berghorst et al., 2013; Kumar et al., 2014; Porcelli et al., 2012).

Second, at longer latencies where stress-related cortisol increases could feasibly exert an influence why might reduced HPA reactivity predict *exaggeration* of an inverse SAM-trust relationship but greater HPA engagement a *reversal* of that relationship? Evidence suggests an acute stress-induced shift towards salience networks is temporary and, in fact, downregulated post-stress in proportion to HPA reactivity (Hermans et al., 2014). Thus, in the former case (i.e., an enhanced inverse SAM-trust relationship at long latencies under reduced HPA reactivity) individuals may not be mounting a cortisol response great enough to attenuate SAM reactivity experienced earlier. In the latter case (i.e., increased trust at long latencies with dual SAM/HPA engagement), it is noteworthy that overall participants were biased towards investing/trusting over keeping their endowment (i.e., investing in 68.92% of trials, keeping in only 31.08%). This pattern, commonly observed in Trust Game research, represents a violation of standard economic assumptions of rationality (which would predict the optimal strategy is never to invest; Berg et al., 1995). Much research suggests stress exposure can potentiate biases in decision-making (for review, see Yu, 2016); this converges with research in the context of memory (Roozendaal et al., 2009) and habit learning (Schwabe et al., 2012) highlighting the role of simultaneous glucocorticoid and noradrenergic engagement in promoting reliance on habitual over goal-directed processing (i.e., system 1 over system 2 in the economic sense; Kahneman, 2011). Thus, the ordinal model’s prediction of increased trust at long latencies under robust dual SAM and HPA engagement is consistent with acute stress-related exaggeration of an existing bias towards engaging in trust in the Trust Game.

Several limitations useful in plotting future directions exist with respect to the current study. That the traditional CPT and SECPT groups

exhibited comparable HPA engagement was unexpected. Primarily systemic stress (e.g., traditional CPT) has been characterized as less reliable in evoking robust HPA activation than stressors with a significant evocative component such as social evaluative threat (as in the SECPT; Dickerson and Kemeny, 2004). In two studies directly comparing CPT and SECPT, peak-to-baseline salivary cortisol increases were significantly greater for the former (Schwabe et al., 2008; Smeets et al., 2012). That said, though no systematic review or meta-analysis is available post-CPT cortisol increases comparable to those here have been reported in multiple studies (e.g., FeldmanHall et al., 2015; Lighthall et al., 2011; Otto et al., 2013; Schoofs et al., 2009). Further, a recent ten-year review of SECPT-based acute stress induction provides data suggesting that (while effective in reliably eliciting a cortisol secretory episode) significant variability exists in the extent of SECPT-evoked cortisol responses (Schwabe and Schachinger, 2018). Among the 21 studies examined SECPT was associated with a mean peak-to-baseline increase of 4.47 nmol/L, but said increase ranged from 1.81 to 8.11 nmol/L ($SD = 1.64$). The SECPT group increase observed here of 2.68 nmol/L was, in fact, only 1.09 standard deviations below the 21 study mean (greater than or equal to 19.05% of studies examined). In the review six ‘key ingredients’ to successful stress induction via SECPT were delineated: uncertainty, consistency, cold stress, continuous evaluation, self-monitoring, and lack of social support or reinforcement (Schwabe and Schachinger, 2018). We carefully attended to all save one: participants were not asked to self-monitor by viewing a real-time visual of their recording, though as described above even when this is included multiple studies yielded comparable reactivity post-SECPT. Results should be carefully interpreted against this backdrop as a different pattern of individual differences in stress responses at the sample level could feasibly yield different outcomes.

Another limitation is that our experimental design was not optimized to examine sex effects (cell sizes including the stress and latency groups in combination with sex were small, mean $n = 8$), reducing our ability to examine potential sex effects reported in some acute stress non-social decision-making research (e.g., Lighthall et al., 2011; van den Bos et al., 2009). A wealth of other neuroendocrine measures involved in social decision-making could further modulate acute stress effects (for review, see O’Connell and Hofmann, 2012); especially given the social nature of trust, oxytocin should be examined (Kosfeld et al., 2005; Kumsta and Heinrichs, 2013). Social decisions occurred within a highly staged environment, outside of a group context in which social decisions are often made. Thus, trust as operationalized here may not be fully generalizable to many real-world social interactions. Finally, there are likely other individual difference factors beyond those explored here which can influence an individual’s propensity to trust under acute stress. For example, social support (Cohen and Wills, 1985) and executive function capacity (Otto et al., 2013) which may attenuate acute stress effects.

In today’s increasingly interconnected modern society, prosocial behaviors like cooperation and trust play a crucial role in our daily decisions. With potential sources of stress increasing in step with technological advances, and decisions themselves often mediated by technology, developing a greater understanding of stress’ influence over social decision-making is progressively more important. Here we demonstrate that acute stress can reduce propensity to trust, an outcome that in and of itself has important implications. For example acute stress-related impairments of trust could lead to social isolation, associated with maladaptive physical and mental health outcomes (Cacioppo and Hawkey, 2003). Yet, a focus only on stress’ ability to harm belies a more complex reality. Acute stress can both impair and facilitate prosocial behaviors like trust based on specific profiles of physiological reactivity. While much research remains to more clearly delineate the methodological and individual difference factors that contribute to differential stress effects in this way, advances along these lines could move the field in an exciting applied direction.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:<https://doi.org/10.1016/j.psyneuen.2018.09.019>.

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