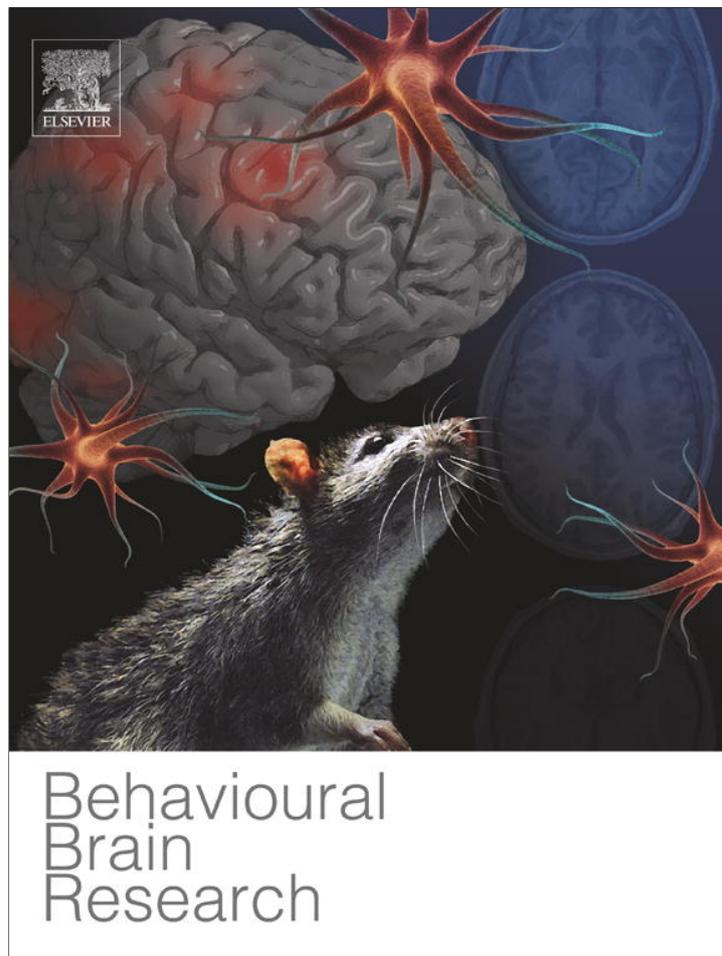


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Research report

Stress and decision making: A few minutes make all the difference

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HIGHLIGHTS

- The temporal dynamic of the effects of stress on decision making was tested.
- Five or 18 min of stress exposure caused less risky decision making.
- In contrast, 28 min after stress onset, decision making was more risky.
- Moderate increases of catecholamines may be beneficial for decisions under risk.
- Increasing cortisol concentrations may lead to riskier decision making.

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ABSTRACT

Stress has been shown to impair decision making. However the temporal development of this phenomenon remains poorly understood. We speculated that the rapid stress induced increase in norepinephrine and the delayed increase in cortisol might exert opposing effects on decision making under risk. Therefore, three different experimental groups underwent the Trier Social Stress Test (TSST) and performed the Game of Dice Task (GDT) at different time points in relation to the stressor, which lasted approximately 18 min. The first group performed the GDT 5 min after stress onset, the second and third group performed the GDT either 18 or 28 min after TSST onset. Decision-making performance of the control group was measured after a respective resting time. Results confirmed a rapid activation of the sympathetic nervous system and a somewhat slower response of the hypothalamic pituitary adrenal axis. In the GDT an improvement of decision-making performance in the 5 and 18 min stress groups compared to controls and the 28 min stress group occurred. Descriptively, decision making of the 28 min after stress group was more risky than decision making of the control group. Our findings are in line with the idea that a moderate increase in catecholamines enhances decision-making performance, while elevated cortisol concentrations may negatively affect decision making presumably via rapid nongenomic mechanisms.

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1. Introduction

In response to physical or psychological stress humans activate two main physiological pathways which have evolved to help the organism to restore homeostasis. The first consists of the fast activation of the sympathetic nervous system (SNS) causing an increase in catecholamines such as norepinephrine (NE) and dopamine (DA) [1]. The second somewhat slower pathway is manifested by the hypothalamic-pituitary-adrenal (HPA) axis [2]. Its activation leads to an enhanced release of the glucocorticoid cortisol from the adrenal cortex with concentrations reaching its peak approximately 20 to 40 min after stress onset [3].

As decision making often has to take place in stressful situations, research has focused on the effects of stress on decision making, specifically under conditions of risk, i.e. a situation where decision rules are explicit and stable and consequences can be estimated [4]. In the literature, stress has mostly been reported to have negative effects on decision making, e.g. more disadvantageous decisions manifested by riskier behavior [5–7]. Behavioral alterations may result from a stress evoked increase in catecholamines and/or cortisol, which affect neural correlates of decision making under risk, such as the prefrontal cortex (PFC) and the basal ganglia [8–12].

Most experimental stress studies measuring cortisol as an indicator of the stress response administered the decision-making task at times of approximately cortisol peak [for a review see 13], where strongest stress effects are expected. Glucocorticoid receptors play an important role at this specific period of the stress response and with regard to stress effects on decision making, as they can be found abundantly in the PFC [14,15]. These receptors

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may induce both, rapid non-genomic and delayed genomic effects [16,17]. While non-genomic effects may evoke a redistribution of neural resources away from higher order PFC functioning to more intuitive behavior, genomic effects are hypothesized to restore PFC functions in the long-run [17,18]. A recent study supports this time-dependent pattern in decision making, as participants acted more consistent during the late aftermath, 75 min after stress cessation, presumably due to the mentioned restoration of cognitive mechanisms [19].

Many stress studies have focused on stress effects of the somewhat slower stress response by the HPA axis, i.e. the rapid non-genomic effects of cortisol [5,7,13,20–22]. The rapid stress effects on decision making as evoked by the fast activation of the SNS has not received as much attention. Yet, the literature provides insights on effects of negative affect on decision making [23,24]. As increases in negative affect have been shown to be associated with increased SNS activity [25,26], such studies may help to understand the effects of SNS activity on decision making. However, results are contradictory. Some find that negative emotions bias people toward the high reward but also risky alternatives [27] other studies show less risky behavior of participants in a negative mood [23].

The opposing results and the fact that the mentioned studies did not directly measure SNS activity make it difficult to draw a conclusion of how stress evoked SNS activity may influence decision making. Another approach may be found in the effects of NE and DA on PFC functioning, which have been described as an inverted U-shaped curve indicating optimal performance at moderate catecholamine concentrations, described as a state of alertness [1]. Thus, a moderate increase in NE and DA in the absence of elevated cortisol concentrations, e.g. right after stress onset, may benefit decision making under risk.

Considering the different effects of catecholamines and cortisol over time [16,17,28], we investigated how decision making under risk is affected at different points in time in relation to the stressor. In doing so we aimed at characterizing the effects of SNS and HPA activation on decision making. To assess decision-making behavior, we used the Game of Dice Task (GDT) [29], a computerized decision-making task with stable and explicit information about gains and losses. We used three stress groups to assess GDT performance, starting time measurement at stress onset using a laboratory stressor lasting approximately 18 min [30]: (a) 5 min after stress onset, (b) 18 min (immediately after the cessation of the stressor), and (c) 28 min (10 min after cessation of the stressor) after onset of stress induction. The control group rested for 18 min.

In comparison to control participants, we hypothesized that the 5 min stress group would show improved decision-making performance, as they would be set into a state of alertness and benefit from the concomitant moderate increase in catecholamines [1,31]. Further, we hypothesized that participants of the 18 min stress group might show impaired decision-making performance, if noradrenergic activation is high and thus could impair PFC functioning based on the reported inverted U-shaped relationship between catecholamines and PFC functioning [8]. Due to the slow increase in cortisol and the concomitant effects on the PFC [32–34], we finally hypothesized an impaired decision-making performance for the 28 min stress group, at cortisol peak, as shown by previous studies of our groups [5,7].

2. Method

2.1. Participants

We recruited 40 healthy, male students aged 18–34, $M = 24.30$, $SD = 3.57$. They were randomly assigned to four different groups, one control group and three different stress groups, each one performing the decision-making task at a different point in time relative to the stressor (see Section 2.6). Before testing a standardized

telephone interview was conducted. Any potential participant was excluded, if one of the following criteria was met: a history of neurological or psychiatric disease, smoking, obesity (Body Mass Index (BMI in kg/m^2) > 30), drug abuse, or shift work. Also, anyone acquainted with the stressor or decision-making task and psychology students of higher semesters were excluded. Testing took place between 09:30 a.m. and 12:00. Participants were instructed not to exercise and to abstain from alcohol 24 h before the appointment. Additionally, they were asked to get up 2 h and not to eat or drink 1 hour before testing, except water. Each participant gave written informed consent and received €15 as compensation. The study was approved by the Ethic committee of the German Psychological Association (DGPs).

2.2. Stress induction

The Trier Social Stress Test (TSST) [30] was used to induce acute stress. Participants had a five-minute preparation period before they had to perform two consecutive tasks in front of a committee, which consisted of two confederates (a man and a woman), dressed in white coats acting neutral and distant. The task involved a free speech (fictitious job interview) and a mental arithmetic (counting backwards in steps of 17) after a preparation time (5 min each, with instructions leading to a total length of approximately 18 min). Also, participants were recorded on video and told that the footage would be used for further analyses after the test. The TSST has been shown to robustly activate the HPA axis [3] and the SNS [35,36]. Participants of the control group rested for 18 min reading neutral magazines. As we were interested in characterizing the impact of the SNS and the HPA axis on decision making, we were concerned that the SNS increases associated with possible control conditions, e.g. the placebo-TSST [37] or the friendly-TSST [38], would preclude an analysis of SNS effects in the current study. We therefore chose a pure resting control condition.

2.3. Measurement of stress response

An affect scale and two biological stress markers were used. The Positive and Negative Affect Schedule (PANAS) [39] was administered to assess negative affect. Participants were to answer 10 items for negative affect on a scale from 1 (very slightly or not at all) to 5 (extremely) how they felt at that very moment. We computed a score using the averaged rating. A higher score indicated higher negative affect.

To obtain endocrine stress markers salivary samples were collected at three different points in time (1 min before and 1 min and 10 min after either treatment; see Fig. 1) using salivette collection devices (Sarstedt, Nuembrecht, Germany). For the 5 min stress group, an additional fourth sample was collected after decision-making task completion, 10 min after TSST preparation, to measure the effect of stress anticipation on the endocrine markers.

We assessed salivary cortisol as an indicator of alterations in HPA axis activity [3] and salivary alpha-amylase (sAA) as an indirect marker of SNS activity [35,36]. Salivary samples were analyzed at the Laboratory of Professor Kirschbaum, Department of Biopsychology, Technical University Dresden, Germany. For free cortisol analysis a commercially available immunoassay was used (IBL, Hamburg, Germany). For sAA analysis a quantitative enzyme-kinetic method was used as described elsewhere [36]. Inter- and intra-assay variations were below 10%.

2.4. Executive functions

Previous studies have shown a relationship between performance of decision making under risk, reasoning abilities, and executive functions [40,41]. To assure that decision-making performance would not be affected by a group difference in reasoning abilities/executive functioning, we assessed logical and numerical thinking abilities using subtest 4 – reasoning – of the German intelligence test battery *Leistungsprüfungssystem* (LPS-4) [42] and the subtests calculative thinking (BIS-RD) and estimation (BIS-SC) of the *Berliner Intelligenzstruktur-Test* (BIS) [43]. For LPS-4, the number of correct responses was used for analyses, a higher score indicating a higher ability of logical thinking. Raw values of the BIS subtests were transformed into point values, with higher values indicating better performance.

2.5. Decision-making performance

We used the original Game of Dice Task (GDT) [29] to assess decision making under risk. The GDT is a computerized game of chance with stable and explicit rules. Participants started with a fictitious capital of €1,000 and were asked to pursue the goal of winning as much and losing as little money as possible. In each one of 18 trials a single die is thrown and participants must decide beforehand whether they choose a single number or a combination of two, three or four numbers, permanently shown on the screen. If the thrown number matches the single number or one of the numbers among the chosen combination, money is won. Else, money is lost. After each trial feedback is given about the outcome of the decision previously made. Every combination is associated with an explicit and stable gain or loss and a winning-losing probability. By choosing a single number €1,000 may be gained or lost (winning probability 1 out of 6). If a combination of two numbers is chosen €500 will be gained or lost (winning probability 2 out of 6). With three numbers a €200 gain-loss is associated (winning probability 3 out of 6) and a €100 gain-loss for four numbers (winning probability 4 out of 6).

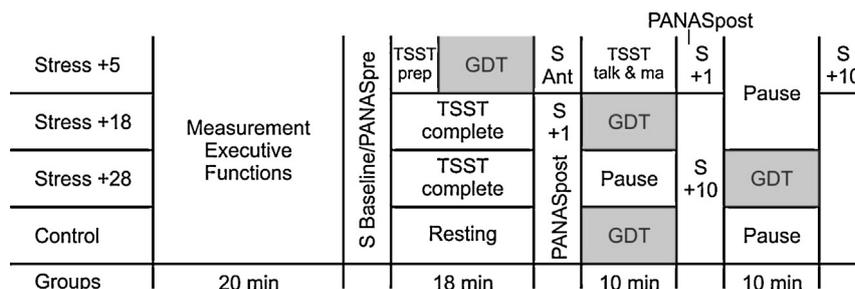


Fig. 1. The experimental procedure for the three stress groups, 5 (Stress +5), 18 (Stress +18), and 28 (Stress +28) minutes after stress onset and the control group (Control). After baseline measurement of endocrine markers and administration of the first Positive Affect Negative Affect Schedule (PANASpre), the Stress +18 and +28 groups completed the Trier Social Stress Test (TSSTcomplete), while Stress +5 only performed the TSST preparation (TSSTprep), followed by the Game of Dice Task (GDT) and a subsequent completion of the TSST by giving a talk and the mental arithmetic (TSSTtalk & ma). The Stress +18 and +28 groups gave a salivary sample 1 min (S +1) and 10 min (S +10) after stress cessation and again completed the PANAS (PANASpost). Stress +5 gave an additional salivary sample (S Ant) for anticipated stress effects right before returning to the TSST room and gave a +1 and +10 min sample right after and 10 min after TSST completion. PANASpost was administered with S +1 for Stress +5. The control group followed the pattern of the Stress +18 group. A more detailed description of the procedure can be found in the method section 2.6 Design and procedure.

Regarding probabilities the different alternatives can be grouped into higher risk and lower risk choices. With a winning probability below 34% the single number and two number combinations are understood to be disadvantageous or high risk choices. Yet, combinations of three or four numbers are advantageous or low risk choices, as the winning probability is 50% or higher. Trial sequences of the GDT are as follows: first, choosing one of the altogether 14 given alternatives (grouped into the mentioned four categories); second, the die is thrown; and third, feedback is given about any gain or loss, the current capital and the number of trials left. Performance is measured by a net score, computed by subtraction of disadvantageous from advantageous choices. A higher score indicated less risky performance [29,44].

2.6. Design and procedure

After participants' arrival a demographic questionnaire was answered followed by the LPS-4, BIS-RD and BIS-SC. The PANAS was administered and the first salivary sample was taken at baseline right before the TSST or resting condition, respectively. Participants of the control group did not leave the test room. Stress group participants were then brought to another room where they were introduced to the committee and TSST instructions were given. Participants of the 5 min stress group did not know beforehand that after preparation time, they would be asked to leave the room again to perform yet another task. Thus, 5 min stress group participants performed the GDT right after preparation time of the TSST. After GDT cessation the additional salivary sample was taken, as described above, and participants were brought back to complete the TSST. The two remaining stress groups performed the TSST without interruption. The second salivary sample was taken 1 min after TSST cessation or resting for 18 min in the control group and the PANAS was again administered. The 18 min stress group and the control group performed the GDT right after the second salivary sample was taken, whereas the 28 min stress group paused for 10 min reading neutral magazines. Ten minutes after TSST cessation or resting in the control group the third salivary sample was taken. The 28 min stress group performed the GDT at cortisol peak 10 min after TSST cessation and right after taking the third salivary sample. Fig. 1 illustrates the procedure.

2.7. Statistical analysis

All statistical analyses were conducted using SPSS 20.0. To compare groups for age, BMI, reasoning ability/executive functions, and decision-making performance, we used one-way analyses of variance (ANOVA). We used exploratory *t*-tests for independent samples for single comparisons of GDT performance of the four groups. Alterations in sAA and cortisol as well as changes in negative affect were determined by using an ANOVA with repeated measurement. For group comparisons of sAA and cortisol concentrations at different points in time of measurement *t*-tests for independent samples were computed. A *t*-test for dependent samples was used to assess cortisol and sAA alterations for the additional sample in the 5 min stress group. Two-tailed tests were performed with *p* set to .05 for all analyses.

3. Results

The one-way ANOVA showed no differences between the four groups for age (in years), BMI (in kg/m²), or reasoning ability/executive functions, as measured by LPS-4, BIS-RD, and BIS-SC. Table 1 shows detailed statistics.

3.1. Cortisol and autonomic stress responses

For sAA and cortisol, respectively, we first conducted a 2 (stress) × 3 (measurement time-points) repeated measurement ANOVA to evaluate stress effects between stress and control groups. Additionally, a 3 (stress groups) × 3 (measurement time-points) repeated measurement ANOVA was conducted for cortisol and sAA, respectively, to investigate whether the three stress groups showed a similar stress response. We found no significant main effect for the factors stress, $F(1, 38) = 1.09, p = .304, \eta^2 = .03$, or time, $F(1.68, 63.64) = 1.62, p = .208, \eta^2 = .04$. A significant interaction of Time × Stress, $F(1.68, 63.64) = 7.10, p = .003, \eta^2 = .16$, was found, reflecting an increase in sAA in the stress group compared to controls. Follow up analyses with *t*-tests for independent samples showed no significant sAA elevation in the stress compared to the control group at the different sampling points, all $p > .110$, all $d < 0.61$. Yet, we found a significant difference in increase of sAA concentration (sample 1 min after stress cessation minus baseline sample) between stress and control participants, $t(38) = -4.38, p < .001, d = 1.64$. Results of the stress group comparison showed an increase in sAA for all stress groups. We found no significant main effect for the factor stress groups, $F(2, 27) = 2.54, p = .098, \eta^2 = .16$, but a significant main effect for time, $F(1.65, 44.63) = 12.06, p < .001, \eta^2 = .31$. Analyses showed no Time × Stress Group interaction, $F(3.31, 44.63) = 1.01, p = .403, \eta^2 = .07$.

For cortisol, results showed an increase in the stress groups, but not in the control group, as indicated by a significant main effect for stress, $F(1, 37) = 13.46, p = .001, \eta^2 = .27$, and for time, $F(1.17, 43.88) = 5.24, p = .022, \eta^2 = .12$. We also found a significant interaction of Time × Stress, $F(1.17, 43.88) = 15.39, p < .001, \eta^2 = .29$. A follow up analysis with *t*-tests for independent samples indicated that stress and control groups differed in sampling points 1 min and 10 min after exposure to the TSST, all $p < .005$.

Stress group comparisons showed a cortisol increase in all stress groups and no differences between the groups. We found no significant main effect for the factor stress groups, $F(2, 26) = 2.11, p = .142, \eta^2 = .14$. The main effect for time was significant, $F(1.18, 30.55) = 29.10, p < .001, \eta^2 = .53$, but not the interaction of Time × Stress Groups, $F(2.35, 30.55) = 1.32, p = .285, \eta^2 = .09$. Results are illustrated in Fig. 2a and b.

For the additional salivary sample in the 5 min stress group, a *t*-test for dependent samples showed a non-significant trend of an increase for sAA, $t(9) = -1.95, p = .083$. Yet, there was no further increase after the TSST as measured by the 1 min after TSST sample, $t(9) = 0.38, p = .712$. There was no significant increase in cortisol between baseline and anticipated stress measurement 10 min after TSST preparation time, $t(9) = -0.46, p = .65$. In accord with the other

Table 1
Demographic variables and executive function values of the four test groups (for each group N = 10). No group differences for age, Body Mass Index (BMI) or executive functions were found.

	Stress Ant M (SD)	Stress +1 M (SD)	Stress +10 M (SD)	Control M (SD)	F	df	p
Age	23.90 (3.10)	25.50 (4.65)	24.40 (3.84)	24.30 (2.55)	0.615	3, 39	.610
BMI	22.26 (1.11)	23.13 (2.63)	22.50 (2.97)	22.00 (2.13)	0.435	3, 39	.729
Exec funct							
LPS-4	27.70 (4.22)	28.90 (2.56)	29.50 (3.89)	30.80 (4.32)	1.144	3, 39	.344
BIS-RD	99.40 (8.61)	93.10 (9.41)	95.6 (10.69)	95.30 (11.96)	0.652	3, 39	.587
BIS-SC	92.1 (10.81)	93.1 (11.72)	95.6 (10.14)	89.80 (6.81)	0.573	3, 39	.637

Note: Stress Ant = Anticipated stress group. Stress +1 = 1 min after stress group. Stress +10 = 10 min after stress group. Control = Control group. Exec funct = Executive Functions. LPS-4 = substest 4 – reasoning – of the German intelligence test battery Leistungsprüfysystem. BIS-RD = substests calculative thinking of the Berliner Intelligenzstruktur-Test. BIS-SC = substest estimation of the Berliner Intelligenzstruktur-Test.

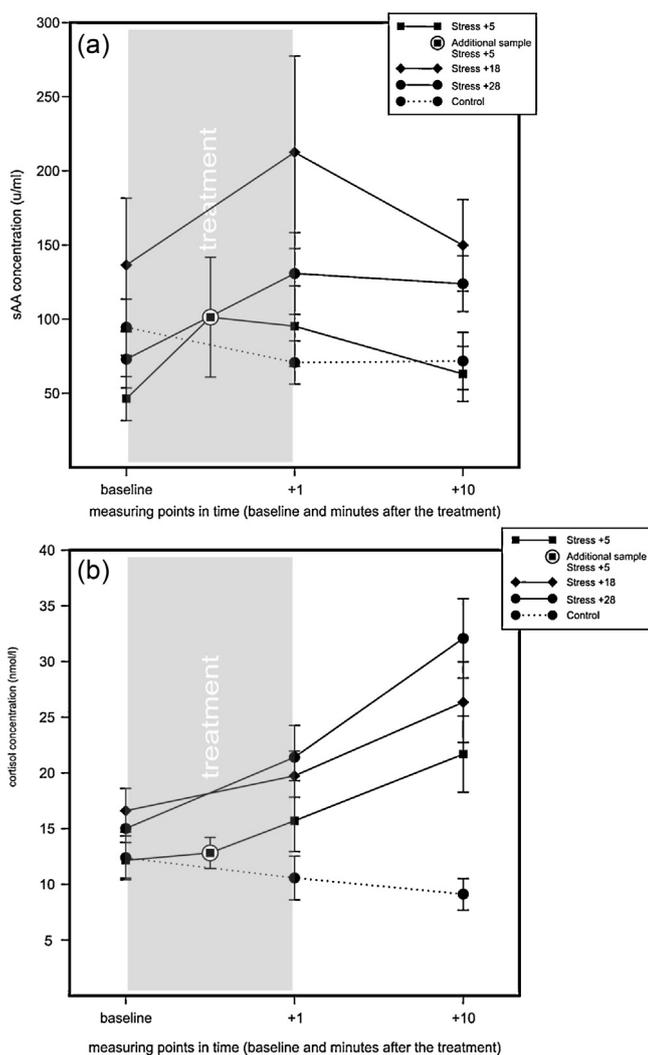


Fig. 2. (a) Results of the salivary alpha amylase (sAA) measures of the 5 (Stress +5), 18 (Stress +18), and 28 (Stress +28) minutes stress groups compared to the control group (Control) during the course of the experiment. Stress increased sAA concentrations in all stress groups. The additional salivary sample of Stress +5 is represented by a single point in the graph. It indicates the trend of an increase compared to baseline measurements. The sample was taken after GDT performance and right before the TSST speech (see Fig. 1). Data represent means and standard errors. (b) Results of the salivary cortisol measures of the 5 (Stress +5), 18 (Stress +18), and 28 (Stress +28) minutes stress groups compared to the control group (Control) during the course of the experiment. Stress increased cortisol concentrations within all stress groups. The additional salivary sample of Stress +5 is represented by a single point in the graph. There was no increase in cortisol concentration compared to baseline measurements. The sample was taken after GDT performance and right before the TSST speech (see Fig. 1). Data represent means and standard errors.

stress groups, the 5 min stress group reached cortisol peak 10 min after TSST cessation as shown in Fig. 2b.

Further, we compared the increase in sAA between the 5 min stress group (additional sample for anticipated stress minus baseline measurement) and the 18 min stress group (1 min after stress sample minus baseline measurement), to evaluate whether the sAA increase differed between the two groups and thus, could be interpreted as moderate or high. Using a *t*-test for independent samples, we observed no differences in sAA increase, $t(18) = -0.55$, $p = .590$. Increase of sAA in the 5 min stress group: $M = 54.95$ (U/ml), $SD = 89.21$, and the 18 min stress group: $M = 76.02$ (U/ml), $SD = 82.47$.

3.2. Negative affect

For negative affect, we first conducted a 2 (stress) \times 2 (measurement time-points) ANOVA with repeated measurement. Results showed a significant main effect for time, $F(1, 38) = 13.44$, $p = .001$, $\eta^2 = .26$, and also a significant interaction of Time \times Stress, $F(1, 38) = 11.63$, $p = .002$, $\eta^2 = .23$, indicating an increase in negative affect over time for the stress groups, but no increase in controls. Table 2 shows descriptive statistics.

To investigate, if the three stress groups differed in negative affect, we conducted a 3 (stress groups) \times 2 (measurement time-points) repeated measurement ANOVA. A main effect for time, $F(1, 27) = 39.86$, $p < .001$, $\eta^2 = .60$, was found, but no significant interaction of Time \times Stress Groups, $F(2, 27) = 0.72$, $p = .495$, $\eta^2 = .05$. These results indicate that all stress groups showed a similar change of negative affect over time.

3.3. Decision-making performance

As illustrated in Fig. 3 the 5 and 18 min stress groups decided more advantageously by choosing less risky alternatives than controls. In contrast, the 28 min stress group showed the riskiest behavior.

We conducted a one-way ANOVA with GDT net score as the dependent variable and the factor group as the independent variable. Results showed a significant main effect of group in GDT performance, $F(3, 36) = 6.42$, $p < .001$, $\eta^2 = .35$. As revealed by exploratory *t*-tests, compared to controls the 5 min stress group showed significantly less risky decisions, $t(18) = 2.16$, $p = .044$, $d = 1.02$, the 18 min stress group nearly reached significance, $t(18) = 1.98$, $p = .064$, $d = 0.93$, and the 28 min stress group showed more risky decisions in a descriptive manner only, $t(18) = -1.29$, $p = .214$, $d = 0.61$, but with a moderate effect. Compared to the 28 min stress group we found significantly less risky decision making for the 5 min stress group, $t(18) = 3.75$, $p = .001$, $d = 1.77$, and 18 min stress group, $t(18) = 3.55$, $p = .002$, $d = 1.67$.

We conducted a post hoc analysis to determine statistical power using G*Power 3, Version 3.1.6 [45]. Our previous study by Starcke,

Table 2

Results of the Positive Affect Negative Affect Schedule as an indicator of changes in negative affect in the stress and control groups.

	Stress Ant M (SD)	Stress +1 M (SD)	Stress +10 M (SD)	Control M (SD)
PANAS-NApre	13.70 (6.29)	13.40 (3.69)	15.40 (4.72)	12.90 (3.45)
PANAS-NApost	19.60 (8.88)	20.00 (4.85)	19.50 (5.23)	13.10 (3.99)

Note: For the three stress groups negative affect increased over time. Controls showed a stable negative affect. Stress Ant = Anticipated stress group. Stress +1 = 1 min after stress group. Stress +10 = 10 min after stress group. Control = Control group. PANAS = Positive Affect Negative Affect Schedule. NA = negative affect. pre = baseline measurement. post = measurement after stress cessation or resting, respectively.

Wolf et al. [5] suggests that effects of stress on decision making can be large (effects size: $d = 0.96$). The power of the current study to detect such an effect (two sided testing, alpha error = .05) was .52 (statistical power: 1-beta error).

4. Discussion

In this study we investigated, how decision making under risk is influenced by acute stress at different time points in relation to the stressor. As to be expected we found a fast increase in sAA and a slower increase in cortisol concentrations in participants of all the stress groups. The 5 min stress group showed a rapid stress reaction by elevated sAA, but not cortisol concentrations after preparation time, right before the stress situation. Thus, the induction of stress, by the TSST and the mere anticipation of it, has been successful. The stress effect is further supported by an increase in negative affect in all stress groups.

Results indicated less risky decision making of the 5 and 18 min stress group compared to controls and a descriptive trend of more risky decision making in the 28 min stress group. The 5 and 18 min stress group showed less risky decision making compared to the 28 min stress group. These differences are supported by rather strong effect sizes, as compared to medium and small effect sizes

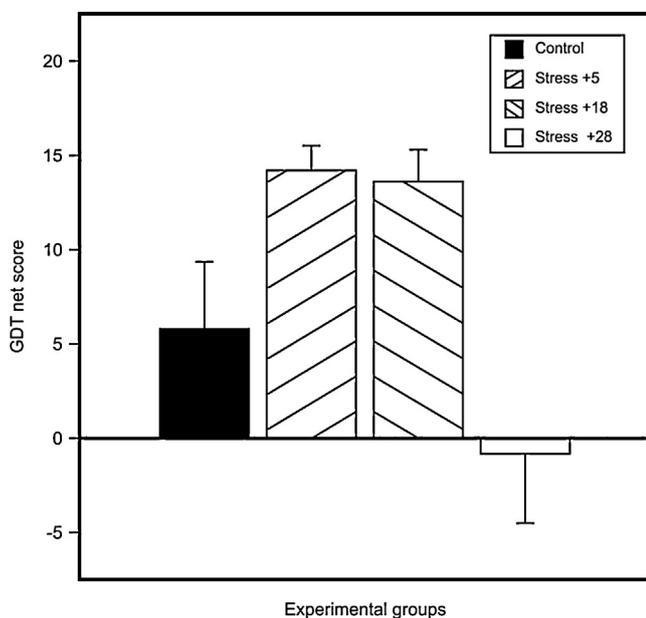


Fig. 3. Mean net score (number of advantageous choices minus number of disadvantageous choices) representing Game of Dice Task (GDT) performance for the control group (Control), and the groups 5 (Stress +5), 18 (Stress +18), and 28 (Stress +28) minutes after stress onset. Statistical analysis using an ANOVA revealed a significant effect for the factor group. The 5 and 18 min stress groups showed less risky behavior compared to controls. The 28 min stress group showed a trend of an impairment compared to controls. Also, the 5 and 18 min stress group showed significant less risky behavior compared to the 28 min stress group. Data represent means over participants with standard errors.

found by previous studies of our groups investigating stress effects on decisions at cortisol peak [5,7].

The least risky behavior in GDT performance was found in the 5 min stress group. The additional salivary sample showed a trend of an increase of SNS activity compared to baseline measurement. This increase suggests elevated catecholamines. As PFC functions are influenced by an inverted U-shaped curve of NE and DA, the improvement in decision-making performance may be due to an evoked state of alertness, being beneficial for PFC-dependent cognitive processes [1,31] and thus for decision making under risk.

Interestingly, we did not find a decision-making impairment in the 18 min stress group. Instead, performance was similar to the 5 min stress group with a trend of an improvement compared to controls. In the 5 min stress group, we did not find a further increase in sAA between the additional anticipated sample and the sample 1 min after TSST cessation, which indicates that the sAA peak was already reached at the time point of the additional sample right after GDT performance. A comparison of sAA increase in the 5 and 18 min stress groups showed no differences. We hypothesized that differences in decision-making performance of these two groups would result from higher sAA increases in the 18 min stress group. Such a difference was not observed and thus, a similar performance may be explained by a similar rather moderate increase in catecholamines.

Another possible explanation of less risky decision making in the 5 and 18 min stress group may be found in an increased activity of the SNS with or without an increase in cortisol concentration. Also, our data suggests that such an effect may not occur, if cortisol concentrations are elevated, only, but rather that mere increases in cortisol may lead to more risky decisions.

As induction of negative affect activates the SNS [25,26] our results may be compared with studies investigating effects of negative affective states on decision making. Although, findings have been inconsistent [23,27], our results are in accord with the notion that negative emotions lead to a perception of risk-related gains as more negatively [24]. Thus, higher risky decisions are avoided. This behavioral shift is further supported by the explanation that people in a negative affective state are more likely to perceive their environment as threatening and are consequently more cautious of potential losses [46].

In accordance with previous studies, we observed that participants exhibited the riskiest behavior when tested 10 min after stress exposure at times when cortisol concentrations have reached their peak [5,7]. Such behavioral shifts may be caused by stress evoked high cortisol concentrations leading to changes in neuronal structures associated with decision making, e.g. the PFC [33,47–49]. These changes may result into dysfunctional strategy use, insufficient adjustment from automatic response, altered feedback processing, or altered reward and punishment sensitivity [13]. A review by Kalenscher and Pennartz [50] illustrates that the PFC plays a vital role in controlling the desire for immediate small rewards for the benefit of a larger delayed reward. This hypothesis is further supported by a lack of self-control in making intertemporal decisions by disruption of the lateral PFC [51] implying a fast satisfaction of needs instead of gratification delay. As stress alters PFC-functioning a need for an immediate large reward, but

associated with a high risk of losing, may have been evoked in our participants. Hence, it may be postulated that elevated cortisol concentrations as evoked by acute psychosocial stress have a negative effect on decision making under risk. This conclusion is further supported by studies employing pharmacological cortisol administration [6]. Nevertheless, this effect may only be found at cortisol peak, as it has been shown that behavior becomes more consistent during the late aftermath of stress, which may indicate restored cognitive mechanisms mediated by slower genomic cortisol effects [19].

A limitation of our study may be the small sample size, as each of the four groups consisted of ten participants, only. Yet, we found significant effects of less risky performance in the 5 and 18 min stress group. Although the observed stress effects in the 28 min stress group are similar to results of two previous studies from our groups [5,7], the missing statistical significance precludes drawing the same conclusion as in the former studies. However, with the relatively small sample size statistical power was not optimal, which may in part be responsible for the missing significance between controls and the 28 min stress group (but note that we revealed moderate effects in terms of Cohen's *d*). Yet, the consistency of these three studies may provide some support for the overall conclusion of impaired decision making 10 min after stress exposure.

Another limitation may be the absence of a significant difference in sAA concentration between stress and control groups at sampling point 1 min after stress cessation, which may lead to the conclusion that it is not elevated SNS activity that may be responsible for altered decision making. Nevertheless, analyses investigating the increase in sAA concentrations showed a significant difference of the temporal progress in SNS activity between stress and control groups, indicating a higher increase in sAA in the stress groups. In this regard we would like to emphasize that sAA as a marker of SNS activity is not impeccable. Although sAA is proposed to be a sensitive biomarker for stress-related physical changes [35], it has also been discussed that not only sympathetic, but also parasympathetic activity plays a role in sAA secretion [52]. We refer to the review by Bosch, Veerman [52] for a critical discussion of this issue. Moreover, we acknowledge that an additional more sensitive SNS marker would have been helpful, such as heart rate or heart rate variability [53], as these markers may have been more sensitive to increases in SNS activity.

Further, it should be noticed that the sample consisted of men, only. Previous studies from our labs had shown no sex differences in GDT performance [5,7] and thus, we decided to include men, only. Nevertheless, the literature also consists of studies reporting sex differences in decision making under risk [21,54–56]. Future studies should test the presence of similar time dependent effects in women.

In summary, our results illustrate that acute stress has a rapid and time dependent effect on decision making. Time points characterized by increased SNS activation were associated with improved (less risky) decision making. In contrast the time point associated with the cortisol peak was associated with impaired (more risky) decision making, even though this was in the current study only apparent at a descriptive level. Future pharmacological and neuroimaging studies are needed to disentangle the underlying endocrine and neuronal mechanisms.

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