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Risk Preferences under Acute Stress

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Abstract:
Many important decisions are made under stress and they often involve risky alternatives. There has been ample evidence that stress influences decision making in cognitive as well as in affective domains, but still very little is known about whether individual attitudes to risk change with exposure to acute stress. To directly evaluate the causal effect of stress on risk attitudes, we adopt an experimental approach in which we randomly expose participants to a psychosocial stressor in the form of a standard laboratory stress-induction procedure: the Trier Social Stress Test for Groups. Risk preferences are elicited using an incentive compatible task, which has been previously shown to predict risk-oriented behavior out of the laboratory. Using three different measures (salivary cortisol levels, heart rate and multidimensional mood questionnaire scores), we show that stress was successfully induced on the treatment group. Our main result is that acute psychosocial stress significantly increases risk aversion. The effect is mainly driven by males; men in our control group are less risk-averse than women, which is a

standard result in the literature, but this difference almost disappears when under psychosocial stress.

Keywords: risk preferences, stress, Trier Social Stress Test, cortisol

JEL: C90, C91, D03, D81, D87

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1 Introduction

Daily decision making often involves risky choices made under severe pressure or even stress, such as giving diagnoses of patients in emergency rooms, trading stocks during a market crash or speeding in a car when coming late for an important meeting. Stress is an instinctive physiological reaction to perceived threats and as such it cannot be controlled by the human will. Understanding behavioral changes under stress, including attitudes towards risk, is vital for setting appropriate guidelines and procedures for times of emergency and construction of decision-making support systems in order to increase the efficiency of individual decisions in such situations.

Behavioral changes under stress have been studied extensively in the psychological literature, mainly looking on the effects of stress on memory and performance, but also on various other aspects of decision making (see review in Starcke and Brand, 2012). However, due to the methodological limitations of previously published studies, only little is known about the effect of stress on risk preferences.

To identify the causal effect of stress on risk preferences, we combine an effective laboratory stressor with random assignment of subjects into the treatment and control groups and an externally validated task for the elicitation of risk preferences. Our stress-inducing procedure, the Trier Social Stress Test (Kirschbaum et al., 1993) in the group modification (TSST-G, von Dawans et al., 2011), is well-established in the literature and has been shown to be one of the most efficient laboratory stressors in terms of physiological as well as psychological reactions (Dickerson and Kemeny, 2004). We use three different measures to validate the efficiency of the TSST-G procedure: two physiological (heart-rate and salivary cortisol concentration) and one psychological (multi-dimensional mood questionnaire[1] scores). To elicit risk-preferences we use the task of Dohmen et al. (2010), which is easily comprehensible to subjects, involves neither feedback processing nor learning which itself may be affected by stress, is incentive compatible, and has been shown to predict risk-taking behavior outside of the laboratory.

[1] Further-on abbreviated as MDMQ

The stress-inducing procedure TSST-G was successful in our case. All three measures were significantly different in the expected direction[2] for the group exposed to the stressor than for the control group during or after the TSST-G procedure, but not before the procedure. On an individual level, when we focus on the increase of salivary cortisol as an indicator of whether or not the subject was stressed, we show that the compliance rate i.e. the correct physiological response to either the TSST-G stress or control procedure is almost 90%.

Our main result is that acute psychosocial stress increases risk aversion when controlling for personal characteristics. The estimated magnitude of the effect is comparable to the gender difference in risk-attitudes. Since not all subjects exposed to the stress-inducing procedure were actually stressed and vice-versa, we need to face the problem of imperfect compliance. Therefore in the analysis we distinguish between the intention-to-treat effects (ITT - effect of random exposure to the stressor on risk preferences) and the average treatment effect on treated (ATT, effect of being stressed on risk preferences). The ATT effect is estimated using a a two-stage instrumental variable regression, with random exposure to the stressor used as an instrument for the physiological state of stress. Both ITT and ATT effects of stress on risk preferences are significant at the 10% level when controlling for gender, age and personality traits, showing that stress increases risk-aversion. This effect is in our study mostly driven by men: females in the control group are more risk-averse than males, which confirms results from previous literature (Charness and Gneezy, 2012), but this difference almost disappears when under stress.

Our study differs from the previous literature by carefully identifying the causal effect of stress on risk preferences using an efficient stressor and a validated risk task. Several studies have already been published on the topic of stress and risk-preferences, but overall they do not provide conclusive results, which may be due to some of their methodological limitations. Some studies point to increased risk-taking under stress (Starcke et al., 2008); (Putman et al., 2010, also found with

[2]An increase in case of cortisol and heart-rate, a change in the MDMQ scores in the good-bad and calm-nervous dimensions towards the bad and nervous mood states, respectively.

direct administration of cortisol instead of stress), others find men take more risks under stress, while women take less (van den Bos et al., 2009; Lighthall et al., 2009; Starcke et al., 2008), or conclude on no change in risk preferences under stress (von Dawans et al., 2012). Pabst et al. (2013a) found a time trend in risk-taking behavior with respect to the time elapsed from the onset of the stressor. Porcelli and Delgado (2009) obtain increased risk-aversion for gain domains, but increased risk-seeking for loss domains. However, these studies either do not show a causal relationship, are unable to effectively induce stress in the majority of subjects, or use tasks for elicitation of risk-preferences that include feedback-processing which itself can be affected by stress (Starcke et al., 2008).

There is an exception. Von Dawans et al. (2012) included a risk-game as a control task into their framework for studying social preferences under successfully induced psychosocial stress in men. The risk game consisted of a repeated choice between high-risk and low-risk lotteries and was executed in the middle and right after the end of the TSST-G protocol. Contrary to our results, no difference was found between the treatment and control groups in terms of risk-preferences. This may have been caused by several factors. First, our task was administered relatively later. As suggested in Pabst et al. (2013a), there may be opposing effects of catecholamines[3] which are released immediately after the onset of the stressor and the somewhat delayed increase in cortisol. Second, their task combined with positive and negative pay-offs and it is possible that the effect of stress on risk preferences is heterogeneous over the gain and the loss domains [4] As risk preferences in Von Dawans et al. (2012) were measured just by the number of risky choices made (the task does not allow for direct computation of a risk-aversion parameter), it is possible that the effects in the gain domain and loss domain cancelled each other

[3]Most prominent catecholamines adrenaline and nor-adrenaline are released immediately after the stress exposure from the adrenal medulla. Their release leads to increase in blood pressure, heart-rate and electrodermal activity. These effects usually stop 10 mins after the cessation of the stressor. See Starcke and Brand (2012) for more details.

[4]This can be expected from the results of Porcelli and Delgado (2009) and generally from the vast amount of literature of different risk attitudes in the gain and loss domain originating in Kahneman and Tversky (1983). A promising study in this respect is Pabst et al. (2013b).

out. Another difference of our risk-elicitation protocol was that subjects made their choices between a risky lottery and a safe payment, whereas in all related literature subjects faced two different lotteries. In addition, the recruited subjects in von Dawans et al. (2012) anticipated the stress procedure since it was literally stated in the advertisement, which may have led to self-selection into the experiment, possibly directly linked to risk attitudes.

We show on a sample of both men and women that stress reduces risk-taking in the gain domain using an easily comprehensible task involving decisions between a safe payment and a risky lottery which was administered 15-20 minutes after the cessation of the stressor. Moreover, participants of our study did not know the purpose of the experiment in advance and therefore the self-selection effect was minimized.

2 Methodology

2.1 Sample

30 female (mean age 22.8, SD = 2.2 years) and 51 male subjects (mean age 23.1, SD = 3.5 years)[5] were recruited via the standard online recruitment-system used for economic experiments at the Laboratory of Experimental Economics in Prague. Participants were mostly students of economics or related disciplines (84%). The participants had no prior knowledge of the purpose of the experiment in order to minimize the sample self-selection effect.[6]

In the e-mail invitation, participants were instructed to abstain from heavy food, nicotine intake and heavy exercise at least two hours prior to the experiment. Before the start of the experiment, the participants underwent questionnaire screening in order to find out if there were any circumstances that would interfere with the cortisol measurement. With one exception the participants were all normal bodyweight and twelve women indicated taking oral contraceptives.[7] All participants were unfamiliar with the stress-inducing procedure and they mostly did not know other participants. They were required to sign an informed consent form, which included an option to leave at any point of the experiment. Out of the 81 participants, none decided to leave, but three were dropped from the analysis due to inconsistent answers in the risk-preferences task (see below), so we were left with 40 observations in the treatment group and 38 observations in the control group.

[5]Since potential gender differences were not the original aim of this experiment, we did not recruit exactly equal shares of males and females and this composition thus reflects the gender composition in the on-line recruitment database.

[6]The name of the experiment in the invitation email was "decision making experiment under unusual conditions". Through the experiment, the stress task was referred to as the "challenge task".

[7]Above-normal weight (BMI above 25) and the intake of hormonal contraceptives may affect cortisol response to stress (Kudielka et al., 2009). Out of the twelve women indicating intake of oral contraceptives, five were assigned to the treatment group and two of them did not show the expected cortisol reaction.

2.2 Experimental Procedure

Six experimental sessions were conducted, all of them between 4:30 PM and 7:00 PM to control for the impact of the circadian variability in cortisol levels. Each session lasted on average a little less than two and a half hours. The average payment was 500 CZK (about EUR 20), including the fixed show-up fee of 150 CZK (about EUR 6). Throughout the experiment, all payoffs were denominated in experimental currency units (ECU).[8] The whole experiment was run in English [9] and no communication among the participants was allowed. The study was approved by the Internal Review Board of the Laboratory of Experimental Economics.

Before entering the laboratory, subjects randomly drew a number that assigned them into either control or treatment group.[10] The instructions explaining the general procedure of the experiment were read aloud and subjects were then asked to sign an informed consent form. The heart-rate monitors were attached and subjects were asked to fill-out a questionnaire to measure their personality traits. They were then given instructions on a task studying Bayesian updating and completed two trial and five real rounds of this task.[11] Next, the first saliva samples were collected and participants filled in the first part of the MDMQ questionnaire.

Afterwards, instructions to either the TSST-G stress-inducing treatment or TSST-G stress-free control procedure were distributed. Subjects read the instructions quietly and had five minutes for preparation. Then the groups were taken to two separate rooms where they completed the TSST-G treatment or control procedure, which lasted about 30 minutes.

[8] The conversion rate was set to 32 ECU = 1 CZK.

[9] The standard language of experiments performed in this laboratory is English. Participants were informed about the language of the experiment in the invitation email. Moreover, they had previously indicated in the options of the on-line recruitment system that they were willing to participate in experiments in English.

[10] We made sure that women who were taking and not taking oral contraceptives were evenly assigned to treatment and control groups – i.e. stratified random assignment to treatment was applied.

[11] Results from the Bayesian updating task are not reported in this paper. Subjects learned about their payment from the Bayesian updating task only at the end of the experiment. The task consisted of stating beliefs about which of two states of the world occurred based on varying number of different types of signals. The task is standard in the literature and was based on Anderson and Holt (1997).

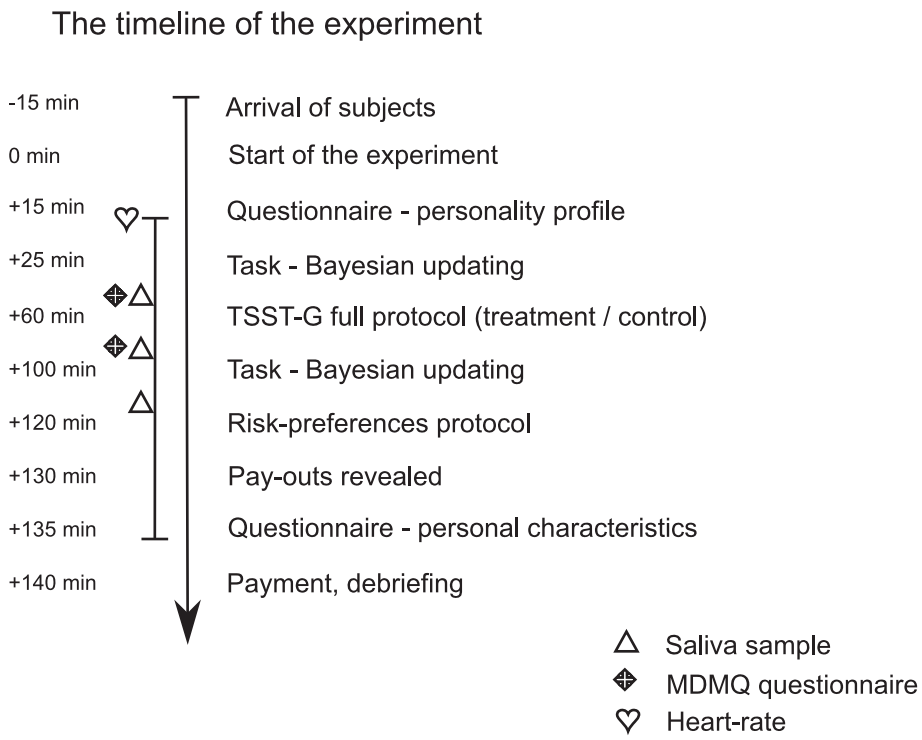


Figure 1: Timeline of the experiment.

When finished, the participants arrived back to the laboratory, gave the second sample of their saliva, filled in the second part of the MDMQ questionnaire and continued in the task aimed at Bayesian updating for the following 15 minutes. Afterwards, the third saliva sample was collected and the risk-preferences task was run, which did not last more than five minutes. Then the payments for the whole experiment were revealed, subjects were asked to fill-out a questionnaire regarding their personal characteristics, returned the heart-rate monitors and proceeded to payments. After the participants from the control group had left, a thorough debriefing about the TSST-G treatment procedure was conducted.

2.3 Measurement of Risk Preferences

Risk preferences were elicited using a simple task after Dohmen et al. (2010), where participants repeatedly chose between a lottery and different safe payments. Subjects had to fill in a table of 10 rows, where in each row the lottery stayed the same paying either 4000 ECU or 0 ECU with 50% probability each, but the safe payment

gradually increased from 0 ECU by steps of 300 ECU up to 2700 ECU.[12] Subjects knew that one row would be randomly determined for payment and that they would be paid according to their choices in that row. We allowed for inconsistent behavior; subjects filled in all 10 rows and were not in any way guided to a single switching point.

If the individual's behavior is consistent, then the row where the subject switches preferences indicates the individual certainty equivalent, i.e. the safe amount which makes the individual indifferent to choosing or not choosing the lottery.[13] As the expected value of the lottery is 2000 ECU, risk neutral subjects should start with preferring the lottery up to the safe amount equal to 1800 ECU (row 7) and then switch to preferring the safe amounts. Risk-averse subjects may switch to preferring safe amounts earlier, with the switching row depending on their degree of risk-aversion (the more risk-averse they are, the earlier they switch). Only risk-loving subjects should choose lottery for the safe amounts bigger or equal to 2100 ECU.

2.4 Trier Social Stress Test for Groups

Stress was induced by a standard validated stress procedure the Trier Social Stress Test for groups (TSST-G, von Dawans et al., 2011) which is a modified version of an individual TSST originally developed in Kirschbaum et al. (1993).[14] The TSST-G provides a combination of a social-evaluative threat and uncontrollable elements, which are the key attributes of an efficient psychosocial stressor (Dickerson and

[12] Detailed instructions and a copy of the decision-making task can be found in the Appendix. The experiment was programmed and conducted with the software Z-TREE (Fischbacher, 2007).

[13] For the descriptive statistics, the individual certainty equivalent is determined as the central point of the switching interval e.g. if the participant preferred the lottery up to row 6 (safe amount=1500 ECU) and switched to preferring the safe amount starting in row 7 (safe amount=1800 ECU), 1650 ECU is taken as the certainty equivalent. For interval regressions, the certainty equivalent is specified as lying in the interval between the two safe amounts where the switch occurred – i.e. in the example above that would mean that certainty equivalent lies between 1500 and 1800 ECU.

[14] To conform to the standards of experimental economics, we modified the original protocol so that it did not contain any deception or false information. These modifications concerned mainly the information given to participant in the treatment condition; they were not told that the panel members were trained in behavioral analysis, and we did not tell them that the video recordings would later be analyzed. The detailed instructions and protocol script can be provided on a request.

Kemeny, 2004). Specifically, the TSST-G treatment (i.e. stress-inducing) protocol consists of two parts – a public speaking task and a mental arithmetic task that are performed in front of an evaluation committee.

In our case, during the public speaking task each participant was asked to perform her best at a fictive job interview for two minutes. In the second part during the mental arithmetic task participants were one by one asked to serially subtract 17 from 4878 for two minutes. Participants were called one-by-one in a random order, were separated by cardboard curtains and wore headphones so that they would not hear or see the other participants. The two committee members wore white coats and had two video cameras by side that recorded performance of the participants. The committee was trained not to give any feedback on the subjects' performance, neither verbally or physically. The full TSST-G control protocol was applied to the control group, which mirrors the activities of the treatment protocol (i.e. participants go through a speaking task and a mental arithmetic task), but without the stressing attributes.

2.5 Measurement of Stress Response

To measure individual stress response, we combine two physiological measures, salivary cortisol concentration and heart rate, and one psychological measure of stress reaction.

First, cortisol is the final hormone of the major endocrine stress axis of the human body (hypothalamic-pituitary-adrenal axis, Dedovic et al., 2009) and Foley and Kirschbaum (2010) show that it is highly predictive of psychosocial stress, while being the most commonly used measure of stress in general. The cortisol concentration peaks in the interval approximately 20 to 40 minutes after the onset of the stressor (Dickerson and Kemeny, 2004). Saliva sample 1 was collected right before the TSST-G procedure, sample 2 was collected right after the stress procedure, and sample 3 was gathered before the risk-preferences protocol, approximately 15

minutes after the cessation of the stressor.[15] Saliva samples were collected using a standard sampling device Salivette.[16] The samples were frozen to -20°C after each experimental session and the salivary cortisol concentration was analyzed by the laboratory of the Biopsychology department at TU Dresden.

Second, as shown in Kirschbaum et al. (1993), heart rate increases are correlated with endured psychosocial stress. Heart-rate of participants was measured by heart-rate monitors of types Polar RS400 and Polar S725X which are composed of a wireless chest transmitter and a wrist monitor. The recording precision was 1s (Polar RS400) or 5s (Polar S725X). The individual difference between the average heart-rate during the TSST-G procedure and the average baseline level can be used as one measure of the induced stress.

Third, Multidimensional Mood State Questionnaire (MDMQ, Steyer et al., 1997) was used to assess the effects of the TSST-G procedure on the mood state of the participants.[17] The mood state is measured in three dimensions: good-bad, awake-tired, and calm-nervous. The MDMQ questionnaire has two parts, left and right, which can be used together or separately. Each part is composed of 15 items, giving a final score for each dimension. In our case, participants filled one part of the MDMQ right before the TSST-G procedure and the other part right after the TSST-G procedure, where the order of the two parts was randomized across sessions. We expected that the stress response would be associated with scores closer to the "bad" and "nervous" poles of the respective dimensions.

[15] We decided to use three samples in order to be able to show that (i) the groups did not differ in the cortisol levels before the TSST-G protocol, (ii) the TSST-G administration was successful and (iii) the reaction lasted similarly as in the comparable experiments. Prior to the experiment we conducted a separate pilot session where only the TSST-G procedure was administered and five saliva samples were collected and analyzed. The dynamics of the cortisol elevation in the pilot session followed the trajectory common in the literature (e.g. in von Dawans et al., 2011) including the recovery phase and therefore we assume the same trajectory in our subjects.

[16] Commercially available from Sarstedt, Germany.

[17] An English version of the MDMQ was used. Available at: http://www.metheval.uni-jena.de/mdbf.php

2.6 Measurement of Personality Traits

Apart from basic observable characteristics, such us gender or age, personality traits can also explain individual differences in risk attitudes (Borghans et al., 2008; Heckman, 2011). To capture the personality profile of participants, we used a battery of 50 questions to construct the Big Five factors that are Openness to Experience, Conscientiousness, Extraversion, Agreeableness and Neuroticism (Goldberg, 2010). The Big Five factors are the most commonly used measure of personality traits, where each factor represents a summary of a large number of specific personality characteristics (Costa and McCrae, 1992).

3 Results

3.1 Stress Response

The induced stress response is summarized in Figures 2 to 4. All three measures, salivary cortisol, heart rate and the MDMQ questionnaire, indicate that we were successful in externally manipulating the stress reaction of the participants.

Figure 2 presents the cortisol reaction. The two-sample Wilcoxon rank-sum test reveals that the treatment and control group do not differ in cortisol levels before the stress procedure (sample 1: $z = -1.430, p = 0.153, d = -0.27$), but that the cortisol level is significantly higher for the treatment group both immediately after the TSST-G stress-induction procedure (sample 2: $z = -6.846, p < 0.001, d = -1.66$) and 15-20 minutes after its end, right before the risk-preferences task (sample 3: $z = -6.618, p < 0.001, d = -1.45$).[18] Therefore, we are able to clearly show that the stress manipulation was successful. Cortisol levels showed a significant increase for the treatment group, while they decreased over time for the control group.

Figure 3 summarizes the heart rates of subjects before, during and after the TSST-G stress-induction procedure.[19] Heart-rate is a standard measure of the im-

[18] Reported effect sizes are Cohen's d, corrected for uneven groups.

[19] We have data on the heart rates of 73 participants; data for the remaining 5 subjects (2 in the treatment group, 3 in the control group) were not obtained during the whole TSST-G procedure

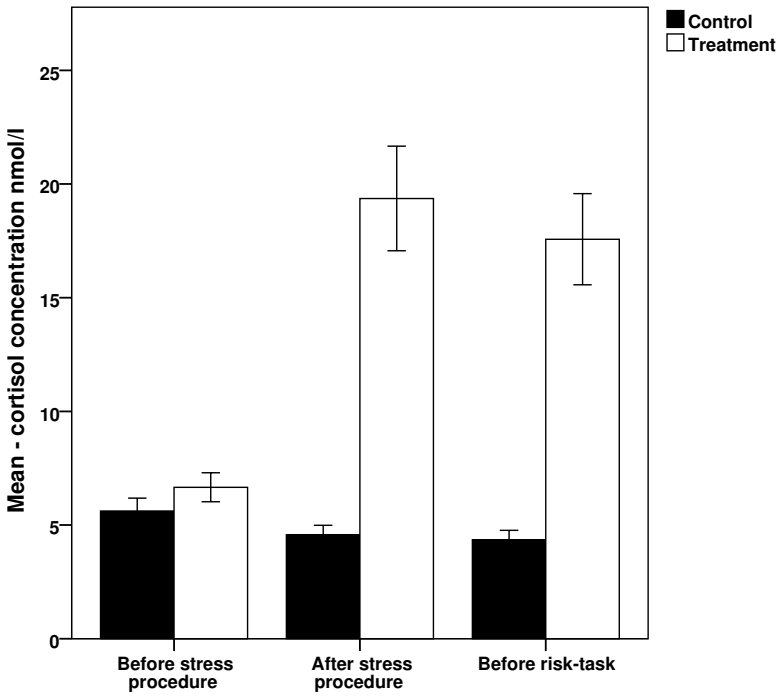


Figure 2: Induced Stress Reaction: Mean levels of free salivary cortisol. Sample 1 was collected before the TSST-G stress-induction procedure, sample 2 after the TSST-G procedure and sample 3 before the risk task. Error bars indicate standard errors of the mean.

mediate response to stress (McEwen and Gianaros, 2010). Together with a measure of endocrinal response (i.e. cortisol concentration), it provides a clear picture of the real-time physiological response to the stressor. Using the two-sample Wilcoxon rank-sum test, we show that the heart-rate of subjects undergoing the TSST-G stress procedure is significantly higher than the heart-rate of subjects undergoing the control procedure ($z = -1.988, p = 0.047, d = -0.55$), but this difference disappears shortly after the cessation of the stressor.[20] Additionally, we analyse the heart rate using two-way analysis of variance (ANOVA) with repeated measures. The factors included were treatment condition (TSST-G stress and control procedures) and time (repeated factor, 35). Since the covariance was heterogenous as indicated by the Mauchly's test of sphericity($\chi^2(594) = 2381.3, p < 0.001$), we report the significance of the results using Greenhouse-Geisser corrections ($\epsilon = 0.175$). The results

due to technical problems. The heart-rate measurement of another six participants was incomplete during the protocol and we are missing three to seven minutes of heart-rate data.

[20] When we look on the increase of heart rate before the TSST-G procedure and during the procedure, it is significantly higher for the treatment group ($z = -2.628, p = 0.009, d = -0.66$).

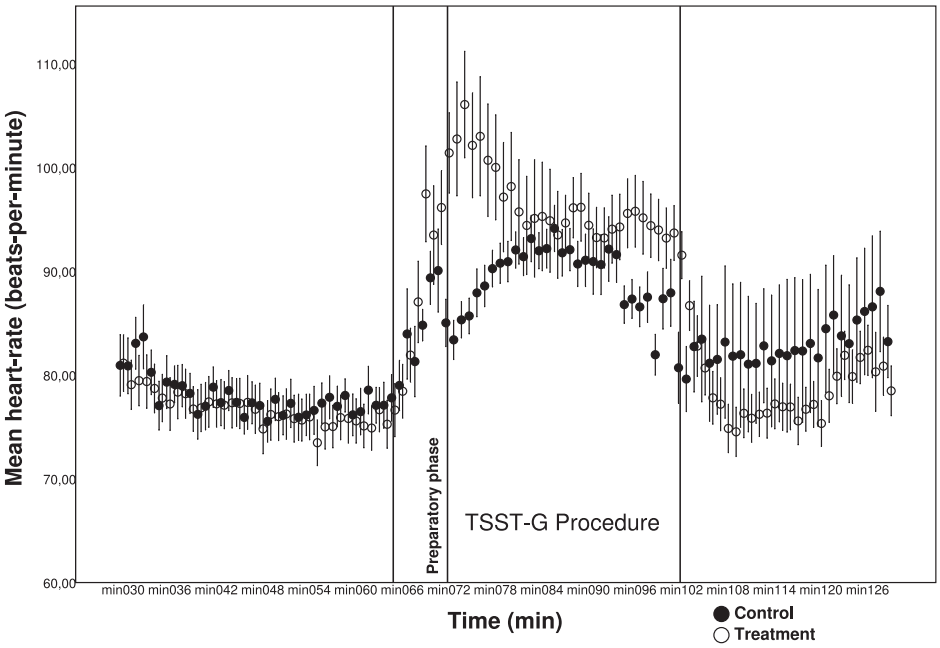
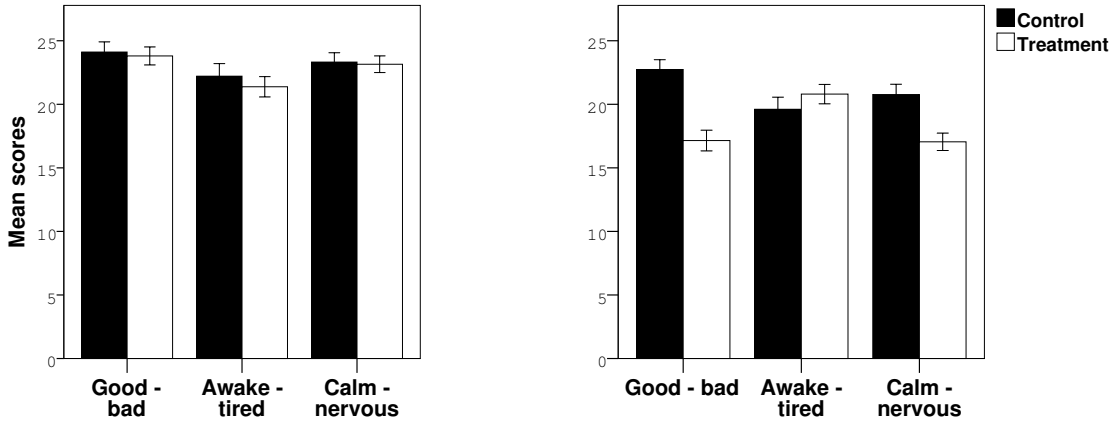


Figure 3: Induced Stress Reaction: Mean heart rate before, during and after the TSST-G stress-induction or control procedure. Points are averages over minute intervals. Error bars indicate standard errors of the mean.

indicate that the effect of time($F(5.961, 387.471) = 19.363, p < 0.001, \eta_p^2 = 0.23$), treatment condition ($F(1, 65) = 3.951, p = 0.051, \eta_p^2 = 0.057$) as well as the interaction of time and condition ($F(5.961, 387.471) = 4.948, p < 0.001, \eta_p^2 = 0.071$) was significant and the treatment condition was higher, which indicates that the manipulation was successful.

To measure the psychological response to stress, we test the effect of the TSST-G stress-induction procedure on the mood state of the participants using a Two-sample Wilcoxon rank-sum test. Figure 4 provides a summary that the treatment and control group score similarly in all three dimensions before the TSST-G procedure: good-bad ($z = 0.411, p = 0.681, d = 0.07$), ($z = 0.576, p = 0.565, d = 0.15$), and calm-nervous ($z = 0.336, p = 0.737, d = 0.04$). Comparison of the two groups after the stress procedure shows that the treatment group has a significantly lower score in the good-bad dimension ($z = 4.356, p < 0.001, d = 1.14$), and in the calm-nervous dimensions ($z = 3.338, p < 0.001, d = 0.81$). This shows that the stressed subjects scored more in the directions "bad" and "nervous". The scores in the awake-tired dimension are not significantly different across the two groups



(a) Before stress procedure

(b) After stress procedure

Figure 4: Induced Stress Reaction: Mood state - scores from the Multidimensional Mood State Questionnaire (MDMQ, Steyer et al., 1997) before and after the TSST-G stress-induction procedure. Error bars indicate standard errors of the mean.

$(z = -1.218, p = 0.223, d = -0.26)$.

Associations between the three measures of stress response are tested using Spearman's rank correlations. The two physiological measures of stress – cortisol response[21] and heart-rate response[22] – are significantly correlated ($\rho = 0.381, p < 0.001$). As for the associations between the physiological and psychological measures, there is a significant correlation between the cortisol response and the psychological response in the good-bad dimension ($\rho = -0.456, p < 0.001$) and in the calm-nervous dimension ($\rho = -0.313, p < 0.01$). The association between the heart-rate response and the psychological response is weaker, significant at the 10% level for the good-bad dimension ($\rho = -0.226, p = 0.055$), but insignificant for the calm-nervous dimension ($\rho = -0.170, p = 0.1500$).

[21] Calculated as the maximum difference between the cortisol concentration in the baseline sample (sample 1, collected before the TSST-G stress induction procedure) and concentration in samples collected after the TSST-G procedure (samples 2 or 3, collected immediately after the TSST-G procedure and with a delay of 15-20 minutes, respectively).

[22] Calculated as the difference between the baseline heart rate (average heart rate before the TSST-G stress induction procedure) and the average heart rate during the TSST-G stress or control procedure.

3.1.1 Compliance

We have shown that the manipulation of the stress condition was successful on the aggregate level. The average maximum cortisol response, calculated as the maximum difference between the baseline sample (sample 1) and samples taken after the stress-inducing procedure (sample 2 or 3), was an increase of 14.15 nmol/l in the treatment group (SD=12.66) and a decrease of -0.86 nmol/l in the control group (SD=2.04). To analyze compliance on an individual level, we also focus on the cortisol response.

We define that a participant is stressed if her maximum cortisol response is bigger than 2.5 nmol/l. Kirschbaum et al. (1993) summarize results from the five original studies of the TSST procedure (n=155), finding that in each study, above 70% of subjects responded to the stressor with an increase of cortisol of at least 2.5 nmol/l above baseline (p. 78). Following our classification, 35 out of 40 subjects in the treatment group are stressed and 35 out of 38 subjects in the control group are not stressed, so the compliance rate is high, at 90%.[23]

3.2 Risk Preferences

Starting with the descriptive statistics of the elicited risk attitudes, we see that inconsistent behavior, i.e. multiple switches between preferring lottery and safe payment in the risk task occurred in three cases (two in the control group, one in the treatment group). These subjects were dropped from the analysis, as their certainty equivalent could not be inferred.[24] In the remaining 78 observations (40 in

[23] This approach is necessarily a simplification as stress reaction is complex and cortisol reactivity individual. However, we still think this is a useful simplification. It enables us to distinguish the effect of random exposure to the stressor (TSST-G stress procedure) on risk preferences from the effect of stress (physiological state of the body) on risk preferences, see below.

[24] We perform two robustness checks of our results, in which we do not drop the multiple switchers from the analysis. In the first robustness check, risk preferences are measured not using the elicited certainty equivalent, but using the number of risky choices made. We then estimate the intention-to-treat effect of stress on risk preferences using ordered probit. As a second robustness check, we treat the inconsistent subjects as indifferent between the safe amounts and the lottery for the entire interval in which multiple switches occur, as suggested by Andersen et al. (2006). This means that certainty equivalent of these subjects is elicited in a wider interval than the certainty equivalent of subjects who switch just once. The intention-to-treat effect of stress on risk preferences is then estimated using interval regression. The results of both robustness checks are reported in Table

the treatment group and 38 in the control group), the modal certainty equivalent is 1,950 ECU, the median is 1,650 ECU and 85.9% of subjects are weakly risk-averse.[25]

To talk about the effect of stress on risk preferences, we need to distinguish the effect of random exposure to the stressor (the TSST-G stress procedure) on risk preferences from the effect of stress (a physiological state of the body) on risk preferences. The problem of imperfect compliance does not usually arise in economic experiments performed in the laboratory, but it is a relevant issue when estimating the effects of laboratory-induced stress.

We start the analysis by presenting the differences in risk attitudes between the TSST-G treatment and control group, to estimate the effect of random exposure to the psychosocial stressor on risk preferences (intention-to-treat effect). To estimate the causal effect of stress (physiological state of the body) on risk preferences, we first look on the difference in risk attitudes between subjects who underwent the stress reaction and those who did not (using cortisol response as a measure of induced stress). The average treatment effect on the treated is then estimated using a two-stage instrumental variable regression, with random exposure to the stressor used as an instrument for the physiological state of stress.

3.2.1 Effect of Exposure to Stressor

Differences between the elicited certainty equivalents of the TSST-G stress and control group are summarized in Figure 5. The two-sample Wilcoxon rank-sum test shows that the difference between the treatment and the control group approaches significance at the 10% level ($z = 1.560, p = 0.119, d = 0.31$). However, there is important gender heterogeneity. The effect of exposure to the stressor on risk preferences is significant at the 10% level for men ($n = 51, z = 1.683, p = 0.092, d = 0.43$), while it is insignificant for women ($n = 27, z = 0.575, p = 0.565, d = 0.14$). We should note that females in our sample are in general more risk-averse than

4 in the Appendix and show that results presented in the main text are robust to including the multiple switchers.

[25] Certainty equivalent below 2,000 ECU indicates that the subject switches to preferring the safe amount at row 8 or earlier.

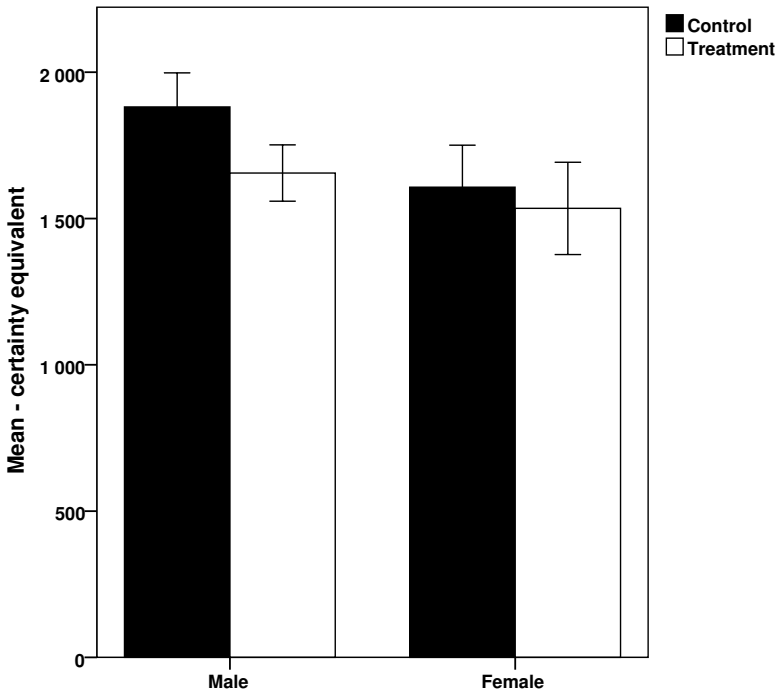


Figure 5: Elicited Certainty Equivalent by Treatment. Error bars indicate standard errors of the mean.

males ($z = 1.726, p = 0.084, d = 0.35$), which is driven by the gender-difference in the control group ($z = 1.613, p = 0.107, d = 0.5$); the gender-difference in the stress treatment is much smaller and insignificant ($z = 0.979, p = 0.327, d = 0.24$). Overall, these results show that random exposure to a stressor leads to increased risk-aversion for men, but not for women, suggesting that the gender gap in risk preferences becomes much less important under stress.

We next extend our analysis and regress the elicited certainty equivalent on the treatment status *Exposed_to_stressor* and additional controls: gender, age, and personality traits (Big Five – openness to experience, conscientiousness, extraversion, agreeableness and neuroticism), which have been found to be important determinants of risk preferences in the literature (Dohmen et al., 2010, 2011; Borghans et al., 2008). We also allow for different responses to treatment across gender by including an interaction term *Exposed_to_stressor*Female*, with the limitation that we have a relatively low number of female subjects. Effects are estimated using an interval regression, to account for the fact that certainty equivalents were elicited in intervals. The results are reported in columns 1-3 of Table 1. Controlling for

additional characteristics, the effect of assignment to treatment on risk attitudes is significant at the 10% level. Results of an ordered probit regression are used as robustness checks and confirm the result of the interval regression, see columns 3-6 of Table 1. This further reinforces our finding that exposure to the psychosocial stressor makes people more risk averse.

3.2.2 Induced Stress and Risk Preferences

Before we identify the effect of the physiological state of stress on risk preferences, we first need to evaluate the differences across participants who are stressed and who are not stressed, independent of treatment. A participant is considered as stressed if the cortisol increase is bigger than 2.5 nmol/L, as explained above. Results of an interval regression with the indicator variable *Stressed* are presented in the first column of Table 2.

There is a clear correlation between the cortisol response and the certainty equivalent. Participants under stress are more risk averse with the effect being significant at the 5% level. Controlling for gender, age and personality traits (the second and third columns of Table 2), the difference is statistically significant at the 1% level and economically important; the size of the coefficient suggests that participants under stress switch to preferring the safe amount about 1.5 rows before participants not under stress, on the scale of 10 rows. The results are confirmed by the ordered-probit estimation, see columns 4-6 of Table 2. The effect of gender is negative and significant at the 5% level; the effects of age and of the personality trait neuroticity are in some specifications also significant at the 10% level.

However, the observed correlation between stress and risk preferences can be driven both by the effect of stress on risk preferences and by different underlying preferences of compliers and non-compliers.[26] This is why we next look at which part of the effect is due to the random assignment to treatment and analyze the

[26]Subjects that get stressed going through the TSST-G control procedure are most likely different from subjects who do not get stressed going through the TSST-G stress procedure.

Table 1: Effects of random exposure to stressor (TSST-G stress procedure) on risk preferences

	(1)	(2)	(3)	(4)	(5)	(6)
	Interval regression			Ordered probit		
Dependent variable	Certainty equivalent			Certainty equivalent		
Exposed to stressor	-192.75	-264.34	-274.59*	-0.38	-0.55*	-0.59*
	(134.00)	(183.62)	(161.97)	(0.23)	(0.32)	(0.30)
Female		-334.13	-304.24		-0.66*	-0.61
		(204.31)	(202.62)		(0.36)	(0.39)
Exposed to stressor*Female		207.62	249.59		0.38	0.47
		(282.26)	(271.12)		(0.50)	(0.50)
Age		223.54	284.18		0.37	0.47
		(157.88)	(183.17)		(0.30)	(0.33)
Age squared		-4.02	-5.05		-0.01	-0.01
		(2.74)	(3.27)		(0.01)	(0.01)
Big Five Personality Traits:						
Openess to experience			-15.69			-0.03
			(14.15)			(0.03)
Concientiousness			-5.25			-0.01
			(12.68)			(0.02)
Extraversion			9.55			0.01
			(9.38)			(0.02)
Agreableness			-6.40			-0.01
			(12.92)			(0.02)
Neuroticity			15.99			0.03
			(10.80)			(0.02)
Constant	1803.69***	-1052.94	-1742.84			
	(102.89)	(2219.80)	(3376.78)			
chi2	2.07	6.53	13.15	2.70	6.54	16.48
Observations	78	78	78	78	78	78

Robust standard errors in parentheses

$*p < 0.10, **p < 0.05, ***p < 0.01$

Notes: The dependent variable is the certainty equivalent calculated from the binary choices in the Risk preferences task. Exposed to stressor = subject was randomly exposed to the TSST-G stress procedure. The reported coefficients in columns 1-3 are marginal effects, estimated using interval regressions to correct for the fact that the dependent variable is elicited in intervals. The reported coefficients in columns 4-6 are estimated using ordered probit regressions.

Table 2: Risk preferences by induced stress (measured by cortisol response)

	(1)	(2)	(3)	(4)	(5)	(6)
	Interval regression			Ordered probit		
Dependent variable	Certainty equivalent			Certainty equivalent		
Under stress	-266.98**	-477.26***	-493.43***	-0.51**	-0.91***	-1.00***
	(133.35)	(165.27)	(150.99)	(0.23)	(0.29)	(0.28)
Under stress*Female		367.97	423.48*		0.53	0.67
		(246.28)	(254.20)		(0.46)	(0.49)
Female		-475.64**	-436.26**		-0.89**	-0.85**
		(189.81)	(194.93)		(0.36)	(0.39)
Age		241.91	295.05*		0.42	0.51
		(151.81)	(174.68)		(0.30)	(0.33)
Age squared		-4.52*	-5.42*		-0.01	-0.01
		(2.69)	(3.18)		(0.01)	(0.01)
Personality Traits:						
Openess to experience			-17.46			-0.04
			(13.69)			(0.02)
Concientiousness			-8.19			-0.02
			(11.79)			(0.02)
Extraversion			7.85			0.01
			(9.14)			(0.02)
Agreableness			-6.33			-0.01
			(12.84)			(0.02)
Neuroticity			16.37			0.03*
			(10.43)			(0.02)
Constant	1834.78***	-1066.15	-1443.21			
	(94.55)	(2051.54)	(3074.02)			
chi2	4.01	13.95	19.32	4.99	17.81	27.30
Observations	78	78	78	78	78	78

Robust standard errors in parentheses
$*p < 0.10, **p < 0.05, ***p < 0.01$
Notes: The dependent variable is the certainty equivalent calculated from the binary choices in the Risk preferences task. Under stress = dummy variable equal to one if the difference in cortisol levels between baseline (sample 1) and sample 2 or sample 3 is bigger than 2.5 nmol/L. The reported coefficients in columns 1-3 are marginal effects, estimated using interval regressions to correct for the fact that the dependent variable is elicited in intervals. The reported coefficients in columns 4-6 are estimated using ordered probit regressions.

data using an instrumental variable interval regression.[27] Results of this regression without and with additional controls are presented in Table 3. The first stages show that the assignment to treatment is strongly correlated with the stress response and therefore confirm that assignment to treatment is a strong instrument. The second-stage results reveal that the causal effect of stress on risk preferences is significant at the 10% level when controlling for other characteristics.[28]

3.3 Gender-specific Response

Our results show that both the exposure to the stressor and the physiological state of stress increase risk aversion when controlling for other characteristics. However, the identified effect of increased risk-aversion under stress is driven mostly by men. Using a nonparametric Wilcoxon rank-sum test, we have identified a different intention-to-treat effect for men and women: the change in risk aversion was significant only for men. We are not able to reject the zero hypothesis of the same treatment effect in the regression analysis, but this is probably due to the relatively low number of women in our sample (n=27).[29] The estimated size of the treatment effect is close to the estimated gender difference in risk preferences under control condition. As a result of the heterogonous treatment effect, the gender gap in risk-aversion almost disappears under stress.

3.4 Physiological vs. Psychological Explanation

As to the mechanism by which stress affects risk-preferences, we cannot clearly distinguish whether the effect is caused just by the physiological reaction or by

[27] Calculated using the cmp module in Stata (Roodman, 2012). The first stages are fitted using an OLS model and second stage is fitted using an interval regression. IV is an asymptotic estimator, so applying it in small samples leads in general to biased estimates. However, this should not be a problem in our case as the instruments are very strong – random assignment to treatment (TSST-G stress or control procedure) predicts the stress response in almost 90% of the cases.

[28] Results of an instrumental variable ordered-probit regression are provided as a robustness check in Table 5 in the Appendix. However, IV ordered-probit can potentially be problematic, as consistency of the second-stage estimates depends on the correct specification of the functional form in the first stage.

[29] Out of which 13 are in the stress treatment, 14 are in the control group.

Table 3: Effect of stress on risk preferences: IV interval regression

	(1)	(2)	(3)
	IV interval regression		
	Second stage: Certainty equivalent		
Under stress	-241.57	-303.43	-315.86*
	(166.79)	(204.69)	(179.31)
Under stress*Female		211.94	271.31
		(403.74)	(390.61)
Female		-374.03*	-341.76
		(217.04)	(207.90)
Age		236.14	290.32
		(153.85)	(177.90)
Age squared		-4.29	-5.19
		(2.68)	(3.18)
Big Five Personality Traits:	No	No	Yes
Constant	1822.31***	-1163.37	-1589.74
	(110.39)	(2157.25)	(3238.45)
	First Stage: Under stress		
Exposed to stressor	0.80***	0.87***	0.87***
	(0.07)	(0.08)	(0.07)
Exposed to stressor*Female		-0.25	-0.27*
		(0.16)	(0.15)
	First Stage: Under stress*Female		
Exposed to stressor		0.01	0.00
		(0.01)	(0.01)
Exposed to stressor*Female		0.61***	0.62***
		(0.14)	(0.14)
chi2	2.10	6.84	13.24
Observations	78	78	78

Robust standard errors in parentheses
$*p < 0.10, **p < 0.05, ***p < 0.01$
Notes: The dependent variable is the certainty equivalent calculated from the binary choices in the Risk preferences task. Under stress = dummy variable equal to one if the difference in cortisol levels between the baseline (sample 1) and sample 2 or sample 3 is bigger than 2.5 nmol/L. Exposed to stressor = subject was randomly exposed to the TSST-G stress procedure. IV interval regression is calculated as a mixed-process regression using the cmp module in Stata (Roodman, 2012), where the first stages are fitted using a linear probability model and the second stage is fitted using an interval regression

the psychological reaction to the stressor. This is because the cortisol response is strongly correlated with the heart-rate response and the mood-state response in the good-bad and calm-nervous dimension, as shown above. However, we can provide some suggestive evidence.

We have shown above that when we measure stress using the cortisol response only, the correlation with the certainty equivalent is highly significant (Table 2), and we get a significant effect of stress on risk preferences using the instrumental variable approach (Table 3). When focusing just on the heart-rate response, the correlation between the heart-rate response and elicited risk-preferences is weaker, but still statistically significant at the 5% level when controlling for other observable characteristics.[30] But if we focus just on the mood-state after stress or change in mood-state, we do not find any significant correlations between mood and elicited certainty equivalent.[31] This suggests that the physiological response to stress is the main driver of the observed behavioral change in risk preferences.

[30] Results are presented in Table 6 in the Appendix.
[31] Results are presented in Table 7 in the Appendix.

4 Conclusion

In this paper we contribute to the literature by studying the effect of acute psychosocial stress on individual risk attitudes. We induce stress with an effective laboratory stressor Trier Social Stress Test for Groups (Von Dawans et al., 2011). Subjects are divided randomly to experience either the treatment "stress" procedure, or the control "no-stress" procedure. Individual risk-preferences are elicited using the task of Dohmen et al. (2010) which is an easily comprehensible, incentive compatible and externally validated measure of risk attitudes. By using three different measures (salivary cortisol concentration, heart rate and multi-dimensional mood questionnaire scores) we show that subjects exposed to the stressor were indeed stressed, while the subjects in the control group were not, with the compliance rate close to 90%. Our main result is that stress increases risk aversion and that this effect is mostly driven by men. The magnitude of the effect of stress is comparable to the gender differences in risk-taking under normal conditions: while in the control group the women are more risk averse than men, this difference almost disappears in the treatment group.

This study is among the first to study the causal effect of stress on individual decision-making and to the best of our knowledge is the first to correctly identify the causal effect of stress on risk preferences. Even though several studies have aimed at this question, all of them experience various methodological limitations. Only the study of Von Dawans et al. (2012) has so far studied this effect using an efficient stress-inducing procedure and a measure of risk preferences not including feedback processing or other potential confounds. Focusing primarily on social preferences, Von Dawans et al. (2012) found no effect of stress on risk preferences. However, this can be due to several reasons: First, their measure of risk preferences included choices both from the gain and the loss domain and effects of stress in these two domains could have cancelled out. Risk attitudes can differ substantially in the gain and in the loss domain, as illustrated by the reflection effect (Kahneman and Tversky, 1979; Laury and Holt, 2008), so the effect of stress on risk preferences could also

be domain-specific as suggested by Pabst et al. (2013b). In our study, we focused on gain domain only, and we identified increased risk-aversion under stress. The effects of stress on risk preferences in the loss domain are yet to be investigated and we think this is an important topic for further research. Second, in our experiment the task was administered relatively later (15-20 min) than in case of von Dawans et al. (2012). As Pabst et al. (2013a) argue, catecholamines, hormones released immediately after the onset of stressor as part of the excitatory reaction, may have opposing effect on risk preferences than cortisol, hormone released relatively later as part of the accommodative reaction to stressor. Third, the task for elicitation of risk attitudes we used consists of repeated decisions between a safe payment and a risky lottery, whereas in the rest of the literature two or more different lotteries have been typically used.

The present study also contributes to the discussion about gender heterogeneity in risk preferences. Women are usually found to be more risk averse than men (Croson and Gneezy, 2009; Charness and Gneezy, 2012), which is confirmed in our results. In addition, our results suggest that the identified effect of increased risk-aversion under stress is driven primarily by males. As a consequence, the gender difference in risk preferences almost disappears under stress. Our study gives just a first insight into the possibility of a gender-specific effect of stress on risk preferences and we suggest it should be tested further using a larger sample. But it leads us to a general note: most of the laboratory research on behavioral decisions under stress has focused on men,[32] mainly because their cortisol response is less affected by other factors, such as the use of hormonal contraceptives or the phase of menstrual cycle in the case of women (Kirschbaum et al., 1999). But since gender differences in preferences and decision-making can be large (Croson and Gneezy, 2009), studying the effects of stress on men only gives half of the story.

When trying to disentangle the channel through which stress affects risk preferences, it is difficult to separate the effects of the physiological and psychological

[32]Including the above mentioned study of Von Dawans et al. (2012).

reactions, since these are highly correlated. Still, we have suggested that the physiological reaction is the main driving force, since the increase in cortisol or heart rate predicts risk-preferences much better than the change in mood states. The increased risk aversion under stress seems to be a part of the famous "fight-or-flight" response to stressors (Cannon, 1932). When exposed to life-threatening danger, the rational instinctive response is to avoid the danger in the way with the highest chance of survival. Being risk-averse in general helps to increase the probability of survival when compared to risk-neutral or risk-seeking behavior. It therefore seems only natural that we find this attitude reinforced under stress.

Our finding of a gender-specific effect of stress on risk preferences can also be linked to a gender-specific response to stress. As reviewed in Kajantie and Phillips (2006), female physiological reaction to stress is typically of a smaller magnitude than the reaction of men of same age, including secretion of cortisol. Therefore, if the main channel causing the effect we observe is the increase in cortisol, women should be less affected than men. Moreover, recent studies suggests that the "fight-or-flight" behavioral response is a rather male reaction to acute stress, while the typical female reaction may be characterized as "tend-and-befriend" (Taylor et al., 2000). In brief, the "tend-and-befriend" reaction means that females under stress show tendencies to maximize the chance of survival themselves and their offspring by seeking help in social networks or groups. An evolutionary perspective can help to explain both the fact that women are found to be more risk-averse under normal conditions and that stress increases risk-aversion, especially in men. In human history, the division of gender roles has typically been such that men had to expose themselves to riskier conditions then women, for example while hunting. In this sense, males needed to be generally more risk-seeking than women, but this tendency had to be regulated when facing immediate threat.

It is worth pointing out that this study concerns only immediate reactions to acute stressors. The physiological effects of long-lasting or chronic stress are different from the physiological effects of the acute stress (Goldstein and McEwen,

2002). Therefore, our findings of the effects of acute stress cannot be generalized to situations with long-lasting or chronic stress, such as the effect of war or traumatic experience. As documented recently in Voors et al. (2012), these events can, on the contrary, increase risk-seeking behavior. Apart from that, we acknowledge the fact that our findings concerning women are limited due to the small sample size and to the fact that we did not ask for the phase of a menstrual cycle, since the stress-reaction may depend on it (Kajantie and Phillips, 2006). However, there is an emerging evidence that risk-preferences are stable over the cycle (Schipper, 2012) so we believe that the overall results are not affected.

Our findings of increased risk-aversion under stress can help to explain many real-life phenomena. For example, risk profiles of investors may change in times of high volatility in the financial markets, the higher probability of losing money acting as a stressor. The investors may thus overreact and readjust their portfolio toward more conservative investment strategies than necessary, as is already documented to happen under time pressure in Dror et al. (1999). Generally, during periods of market stress, there is a high demand for "safe-haven assets", such as safe government bonds (Upper, 2000) safe currencies (Kaul and Sapp, 2006), and gold (Baur and McDermott, 2010). Another example could be that the decisions of doctors in emergency rooms may be biased toward older and verified, but sometimes less efficient medical treatments. Overall, if there is indeed increased risk-aversion under stress, it may have important consequences for creating guidelines and policies for times of stress and panic. It also highlights the necessity of training and simulations, since the physiological reaction to a specific stressor diminishes with training (Kudielka et al., 2009).

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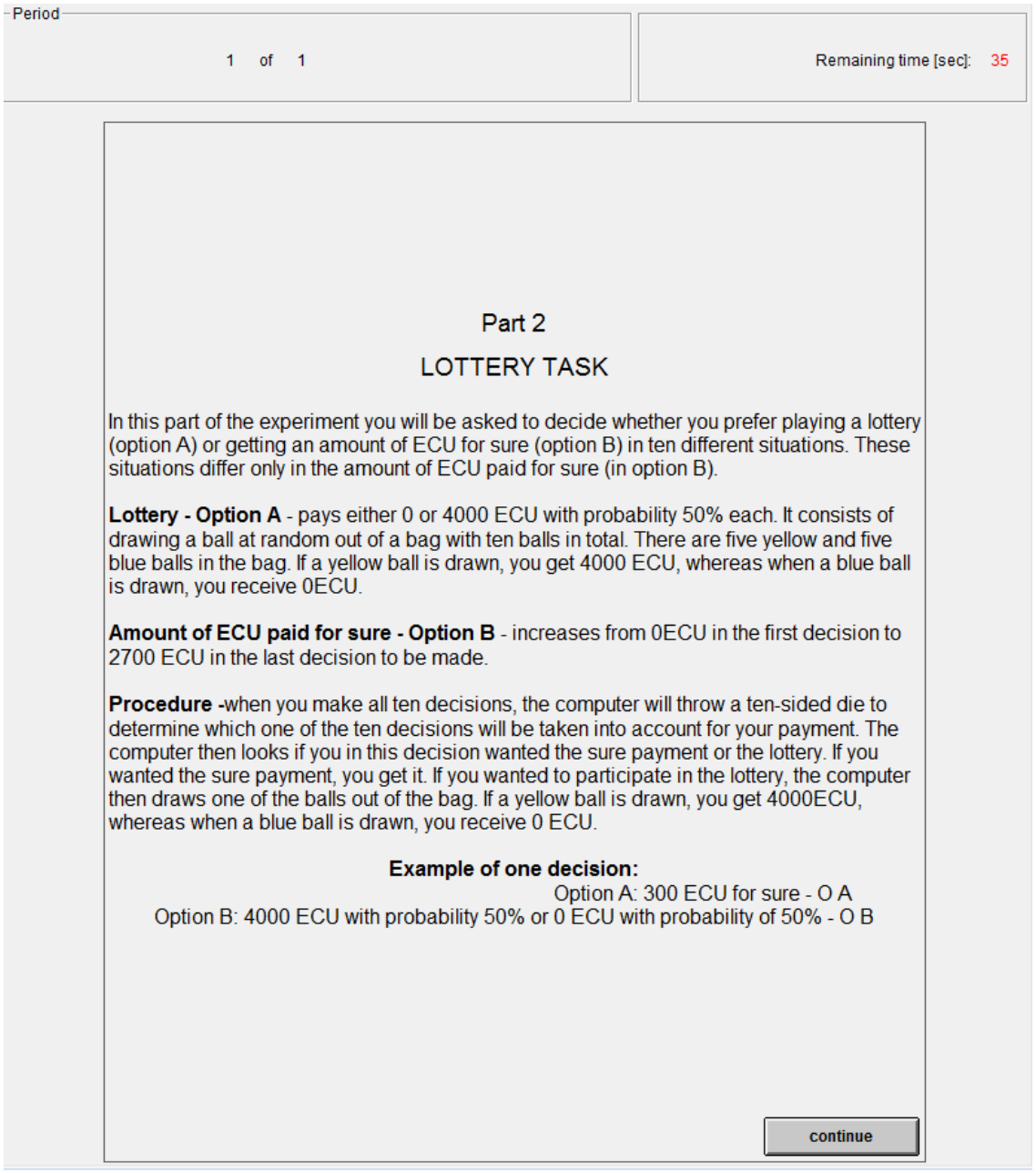
Appendix A - Instructions: Risk task




Period
1 of 1
Remaining time [sec]: 23
Choose one of the two options for each row.
Option A: 0ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 300ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 600ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 900ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 1200ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 1500ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 1800ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 2100ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 2400ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
Option A: 2700ECU for sure
Option B: 4000ECU with probability 50%
or0ECU with probability of 50%
A
B
OK

Appendix B - Additional results

Table 4: Effects of random exposure to stressor (TSST-G stress procedure) on risk preferences - including subjects with multiple switches in the risk task

	(1)	(2)	(3)	(4)	(5)	(6)
	Ordered probit			Interval regression		
Dependent variable	Number of risky choices			Certainty equivalent		
Exposed to stressor	-0.33	-0.55*	-0.61**	-173.49	-267.65	-283.30*
	(0.23)	(0.32)	(0.30)	(133.39)	(183.86)	(162.18)
Exposed to stressor*Female		0.43	0.55		246.66	291.99
		(0.50)	(0.50)		(279.03)	(270.81)
Female		-0.79**	-0.72*		-382.99*	-336.99
		(0.36)	(0.39)		(203.43)	(205.96)
Age		0.32	0.49		203.11	281.95
		(0.30)	(0.33)		(156.70)	(183.00)
Age squared		-0.01	-0.01		-3.68	-5.05
		(0.01)	(0.01)		(2.72)	(3.26)
Personality Traits:						
Openess to experience			-0.03			-13.89
			(0.02)			(14.02)
Concientiousness			-0.02			-7.83
			(0.02)			(12.49)
Extraversion			0.01			8.64
			(0.02)			(9.38)
Agreableness			-0.00			-4.38
			(0.02)			(12.71)
Neuroticity			0.04**			18.35*
			(0.02)			(10.35)
Constant				1780.71***	-767.42	-1783.18
				(103.22)	(2200.47)	(3346.78)
chi2	2.11	7.63	21.24	1.69	6.87	15.17
Observations	81	81	81	81	81	81

Robust standard errors in parentheses

$*p < 0.10, **p < 0.05, ***p < 0.01$

*Notes:*The dependent variable in columns 1-3 is the number of risky choices in the Risk preferences task. The dependent variable in columns 4-6 is the certainty equivalent calculated from the binary choices in the Risk preferences task. Subjects with multiple switches between lottery and safe amount are not dropped as inconsistent, as through the paper, but considered indifferent for the entire switching interval. Exposed to stressor = subject was randomly exposed to the TSST-G stress procedure. Columns 1-3 are estimated using ordered probit, columns 4-6 are estimated using interval regressions to account for the fact that the dependent variable was elicited in intervals.

Table 5: Effect of stress on risk preferences: IV ordered probit

	(1)	(2)	(3)
	IV ordered probit		
	Second stage: Certainty equivalent		
Under stress	-0.49*	-0.65*	-0.70**
	(0.29)	(0.37)	(0.35)
Under stress*Female		0.37	0.49
		(0.75)	(0.75)
Female		-0.76*	-0.72*
		(0.39)	(0.41)
Age		0.41	0.51
		(0.30)	(0.34)
Age squared		-0.01	-0.01
		(0.01)	(0.01)
Big Five Personality Traits:	No	No	Yes
	First Stage: Under stress		
Exposed to stressor	0.80***	0.87***	0.87***
	(0.07)	(0.08)	(0.07)
Exposed to stressor*Female		-0.25	-0.27*
		(0.16)	(0.15)
	First Stage: Under stress*Female		
Exposed to stressor		0.01	0.00
		(0.01)	(0.01)
Exposed to stressor*Female		0.61***	0.62***
		(0.14)	(0.14)
chi2	2.85	7.03	16.81
Observations	78	78	78

Robust standard errors in parentheses
$*p < 0.10, **p < 0.05, ***p < 0.01$
Notes: The dependent variable is the certainty equivalent calculated from the binary choices in the Risk preferences task. Under stress = dummy variable equal to one if the difference in cortisol levels between baseline (sample 1) and sample 2 or sample 3 is bigger than 2.5 nmol/L. Exposed to stressor = subject was randomly exposed to the TSST-G stress procedure. IV ordered probit is calculated as a mixed-process regression using the cmp module in Stata (Roodman, 2012), where the first stages are fitted using a linear probability model and the second stage is fitted using an ordered probit.

Table 6: Risk preferences by induced stress (measured by heart-rate response)

	(1)	(2)	(3)	(4)	(5)	(6)
	Interval regression			Ordered probit		
Dependent variable	Certainty equivalent			Certainty equivalent		
Heart-rate response	-4.84	-15.73*	-15.97**	-0.01	-0.03**	-0.03**
	(3.77)	(8.04)	(7.52)	(0.01)	(0.01)	(0.01)
Heart-rate response*Female		19.51**	24.24**		0.03*	0.04**
		(9.59)	(9.69)		(0.02)	(0.02)
Female		-520.59**	-533.64**		-0.93**	-1.01**
		(235.42)	(239.52)		(0.43)	(0.46)
Age		235.63	289.60		0.39	0.48
		(171.25)	(191.58)		(0.31)	(0.34)
Age squared		-4.19	-5.08		-0.01	-0.01
		(2.98)	(3.44)		(0.01)	(0.01)
Big Five Personality Traits:						
Openess to experience			-23.62			-0.05*
			(15.32)			(0.03)
Concientiousness			-8.55			-0.02
			(12.75)			(0.02)
Extraversion			8.07			0.01
			(10.16)			(0.02)
Agreableness			-5.85			-0.01
			(12.72)			(0.02)
Neuroticity			18.32			0.03
			(12.82)			(0.02)
Constant	1781.79***	-1167.54	-1427.90			
	(103.02)	(2359.93)	(3479.77)			
chi2	1.65	7.67	17.60	2.08	7.76	24.91
Observations	73	73	73	73	73	73

Robust standard errors in parentheses

$*p < 0.10, **p < 0.05, ***p < 0.01$

Notes: The dependent variable is the certainty equivalent calculated from the binary choices in the Risk preferences task. Heart-rate response = individual difference between the baseline heart rate (average heart rate before the TSST-G stress induction procedure) and the average heart rate during the TSST-G stress or control procedure. The reported coefficients in columns 1-3 are marginal effects, estimated using interval regressions to correct for the fact that the dependent variable is elicited in intervals. Reported coefficients in columns 4-6 are estimated using the ordered probit regressions.

Table 7: Risk preferences by induced stress (measured by the change in mood state - MDMQ scores)

	(1)	(2)	(3)	(4)	(5)	(6)
	Interval regression			Ordered probit		
Dependent variable	Certainty equivalent			Certainty equivalent		
Change in Mood State:						
Good-Bad	24.54	29.71	26.64	0.05*	0.05	0.05
	(19.30)	(24.81)	(22.74)	(0.03)	(0.04)	(0.04)
Awake-Tired	3.82	-6.99	-6.39	0.00	-0.01	-0.01
	(13.26)	(11.36)	(11.79)	(0.02)	(0.02)	(0.02)
Calm-Nervous	-21.37	-22.49	-18.41	-0.04	-0.04	-0.04
	(16.22)	(19.41)	(19.52)	(0.03)	(0.04)	(0.04)
Good-Bad*Female		-20.56	-23.79		-0.03	-0.03
		(28.43)	(28.42)		(0.05)	(0.05)
Awake-Tired*Female		43.67**	45.68**		0.07*	0.07*
		(20.33)	(20.09)		(0.04)	(0.04)
Calm-Nervous*Female		11.48	8.38		0.03	0.03
		(26.02)	(26.42)		(0.05)	(0.05)
Female		-212.21	-210.66		-0.32	-0.32
		(157.16)	(170.02)		(0.34)	(0.34)
Age		257.21	340.83*		0.56	0.56
		(159.76)	(191.47)		(0.35)	(0.35)
Age squared		-4.43	-5.95*		-0.01	-0.01
		(2.90)	(3.55)		(0.01)	(0.01)
Big Five Personality Traits:						
Openess to experience			-5.61		-0.01	-0.01
			(14.49)		(0.03)	(0.03)
Concientiousness			0.52		-0.00	-0.00
			(12.96)		(0.03)	(0.03)
Extraversion			7.95		0.01	0.01
			(8.80)		(0.02)	(0.02)
Agreableness			-1.01		-0.00	-0.00
			(12.07)		(0.02)	(0.02)
Neuroticity			14.95		0.03	0.03
			(10.70)		(0.02)	(0.02)
Constant	1716.94***	-1727.48	-3346.62			
	(81.93)	(2180.86)	(3415.53)			
chi2	2.73	12.38	20.56	3.88	21.45	21.45
Observations	78	78	78	78	78	78

Robust standard errors in parentheses

$*p < 0.10, **p < 0.05, ***p < 0.01$

Notes: The dependent variable is the certainty equivalent calculated from the binary choices in the Risk preferences task. Change in Mood State = change between the Multidimensional-mood-state-questionnaire (MDMQ) scores before and after the TSST-G stress induction procedure. All three MDMQ dimensions are considered: good-bad, awake-tired and calm-nervous. The reported coefficients in columns 1-3 are marginal effects, estimated using interval regressions to correct for the fact that the dependent variable is elicited in intervals. The deported coefficients in columns 4-6 are estimated using ordered probit regressions.

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