Stress and Risk Taking*

Extended Abstract

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Many economic decisions are made under stress. How does stress affect individual risk taking? Does a gambler go all-in or go home after a stressful provocation at the casino? How does a financial professional respond when bear market hits and her stress soars? How do individuals stressed by poverty take risk when opportunities present themselves? Understanding the impact of stress on risk taking helps to predict the respective behaviour in stressful situations. It also helps to reverse-engineer behaviour in the counterfactual stressless scenario for situations where stress prevails, and therefore has important implications for the efficiency loss caused by stress.

There are two types of stress which might influence risk taking decisions in different manners. One type is acute stress, i.e. the short-term response to a stressor as experienced in, for example, a traffic jam, an argument, a criticism or a job interview. The other is chronic stress, i.e. the stress caused by persistent experiences of acute stress, for example, in poverty, in stressful working environments, or in bad interpersonal relationships. This study investigates the impact of *acute* stress, *chronic* stress and their interaction on risk taking.

On acute stress, the existing studies on this topic mostly compare risk taking behavior between subjects, and the evidence shows a mix of positive, negative

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and null effects.[1] Our experiment features a *within* subject design: we elicit each subject's risk taking tendency both with and without exogenously induced acute stress. This design allows us to study the heterogeneous responses to acute stress in terms of their risk taking behavior. Among the dimensions along with heterogeneity might occur, we are particularly interested in the heterogeneity across individuals which different predispositions to take risk. With the *within* subject design, we can distinguish between four effects of acute stress on risk taking: amplification, moderation, higher risk aversion, and lower risk aversion. Amplification refers to the effect that stress amplifies individual risk taking predisposition: under acute stress risk averse individuals become more risk averse, and risk seeking individuals become more risk seeking. Moderation refers to the opposite of amplification. Higher and lower risk aversion refer to a homogeneous positive and negative effect of stress on risk aversion. Drawing on the Drift Diffusion Model (DDM) from cognitive neuroscience, we hypothesize that acute stress amplifies risk taking predisposition (for details see Section 1.1).To the best of our knowledge, we are among the first to study heterogeneous effects of acute stress on risk taking.[2] An other feature of our experimental design is that, unlike many previous studies, we investigate the effect of stress in both the loss and the gain domain. [3]

When it comes *chronic* stress, evidence on its role in risk taking has been scarce and inconclusive. Kandasamy et al. (2014) dose 36 adults with either hydrocortisone – the pharmaceutical form of the stress hormone cortisol – or a placebo for eight days and find that long-term hydrocortisone intake *decreases* risk taking. In contrast, Ceccato et al. (2016) measure endogenous chronic stress and find that self-assessed chronic stress *increases* risk taking. Our study provides further evidence on the effect of chronic stress on risk taking.

Regarding the interaction effect of actue and chronic stress on risk taking, we study how individuals under different chronic stress levels change their risk taking behaviour in response to an acute stressor. Our experiment has the following three key elements that make this investigation possible: *(i)* measure of subjects' chronic

[1]Previous experiments on the effect of acute stress on risk taking have found no effect (gain: Pabst et al., 2013, Clark et al., 2012; loss: Buckert et al., 2014), more risk seeking (gain: Buckert et al., 2014; loss: Porcelli and Delgado, 2009), and more risk aversion (gain: Porcelli and Delgado, 2009, Cahlíková and Cingl, 2017, Guiso et al., 2018; loss: Pabst et al., 2013, 2013,Clark et al., 2012).

[2]Lighthall et al. (2009) looked at heterogeneous effect between genders. Mather et al. (2009) found heterogeneous effect of risk for elderlies and younger adults.

[3]Excepstions are, for instance, Porcelli and Delgado (2009), Pabst et al. (2013), Buckert et al. (2014)

stress levels, *(ii)* exogenously induced acute stress and measure of the corresponding heart rate and cortisol elevation, and *(iii)* measure of individual risk taking both with and without exogeneous acute stressor. Combining these three elements, we first investigate the heterogeneity of the *acute stress elevation* caused by an exogenous acute stressor across individuals with different chronic stress levels.[4] Second, we study the heterogeneity of *the change of risk taking behaviour* in response to the acute stressor across individuals with different chronic stress levels. To the best of our knowledge, we are the first to study this interaction effect of chronic and acute stress on risk taking.

1 Hypotheses

1.1 The Effect of Acute Stress on Risk Taking

We take advantage of our within-subject design and investigate individuals' heterogeneous response to acute stress in terms of risk taking. One hypothesis that we are particularly interested in is that acute stress amplifies risk taking:

Amplification Hypothesis *Under acute stress, individuals with a predisposition to take risk will take even more risk, while individuals with a predisposition to avoid risk taking will take less risk.*

This hypothesis draws on the Drift Diffusion Model (DDM), which stems from cognitive neuroscience. In this model, an agent's decision between two options is modelled as evidence accumulation over time, which is in turn determined by a predisposition, a drift rate and a decision threshold of accumulated evidence for each option that has to be met before the decision maker decides for this option.

The Amplification Hypothesis shares a similar intuition as the one in Chen and Krajbich (2018), who show that time pressure amplifies social preferences. They explain the effect by a biased predisposition, and the narrowing of the decision thresholds. Companied by a biased predisposition, narrowed decision thresholds have disproportional effects on decisions for and against the predisposition: more decisions will succumb to the predisposition. Applying to risk taking under stress,

[4]Evidence on the effect of chronic stress on acute stress is scarce and inconclusive. Ockenfels et al. (1995) find no correlation between higher chronic stress and an individual's reaction to an acute stressor, while Matthews et al. (2001) and Kudielka et al. (2006) find that higher chronic stress correlates with less acute stress response to acute stressors.

it would mean that an agent with a predisposition for or against risk taking would become even more risk seeking or risk averse under acute stress, assuming that acute stress narrows the decision thresholds as time pressure does.

1.2 The Effect of Acute Stress on Risk Taking for Chronically Stressed Individuals

Differences in risk taking under acute stress with respect to individual chronic stress levels depend on two factors, *(i)* the potentially different response to acute stress related to different chronic stress levels, and *(ii)* the effect of chronic stress on the effect of acute stress on risk taking.

Since evidence on *(i)* is scarce and inconclusive and we are the first to study *(ii)*, the question as to how acute stress interacts with chronic stress to affect risk taking is of empirical nature and we take an explorative approach. If chronic stress led to a deregulation of mechanisms underlying cortisol secretion and resulted in excessive cortisol secretion in response to an acute stressor, highly chronically stressed individuals would response more strongly to acute stressors, compared to individuals under low chronic stress. On the contrary, if an inherently high cortisol level caused by chronic stress attenuated further cortisol secretion promoted by an acute stressor (Kudielka et al., 2006), those who have high chronic stress would exhibit smaller response to acute stressor than those who have low chronic stress.

2 The Experiment

In our laboratory experiment, we implement a within-subject design, in order to achieve within-subject comparisons of risk taking with and without exogenous acute stress. Each subject is invited to two laboratory sessions, in one of which she undergoes the Trier Social Stress Test for Groups, a laboratory acute stressor (TSST-G, von Dawans et al., 2011). We measure subjects' heart rate and collect their saliva samples to assess acute stress throughout the experiment. One hair sample is collected at the end of the experiment to assess our subjects' chronic stress. The remainder of this section outlines the details of our experimental set-up.

2.1 The Trier Social Stress Test for Groups (TSST-G)

The TSST-G is a modified version of Trier Social Stress Test (TSST, Kirschbaum et al., 1993), characterized by a faster procedure by treating subjects in groups of six (von Dawans et al., 2011). Various studies that used the TSST-G have successfully induced elevated cortisol levels - the primary stress hormone (e.g., Buckert et al., 2014, Cahlíková and Cingl, 2017).

The TSST-G has three phases: In Phase I, subjects are asked to prepare for a mock interview that is going to take place in ten minutes. In Phase II, the mock interview takes place, where subjects within groups of six take turns to give an interview speech in front of two experimenters and two video cameras for two minutes. In Phase III, subjects conduct a mental arithmetic task in front of the experimenters and receive instructions to restart whenever they made a mistake. We follow the standard TSST-G protocol of von Dawans et al. (2011).

2.2 Measure of Acute Stress Response

We use salivary cortisol to measure acute stress responses. Cortisol is a hormone released in response to stress when the hypothalamic-pituitary-adrenal axis (HPA), the major stress axis in human, is activated (Dedovic et al., 2009). It is widely used as a biomarker of stress in experimental studies (Foley and Kirschbaum, 2010). Saliva samples are taken throughout our experiment to monitor the salivary cortisol levels (see 2.5 for detail timing of the saliva collection).

We also measure the heart rate during the experiment with devices such as wrist or chest bands that do not require qualified medical personnel, in order to account for stress and arousal due to the situation (Hjortskov et al., 2004). von Dawans et al. (2011), for example, find a significant heart rate response in subjects who were treated by the TSST-G.

2.3 Measure of Chronic Stress

We use hair cortisol to measure chronic stress. The hair cortisol level in each 1cm segment of hair from the scalp outwards reflects the retrospective secretion of cortisol of approximately one month (Stalder and Kirschbaum, 2012). Compared to repeated salivary sampling, hair cortisol has the following two advantages: operational convenience compared to repeatedly collecting saliva samples over a prolonged pe-

riod on a daily basis, neutralization of daily cortisol fluctuations compared to saliva cortisol, which is sensitive to the exact sampling time of each day.[5]

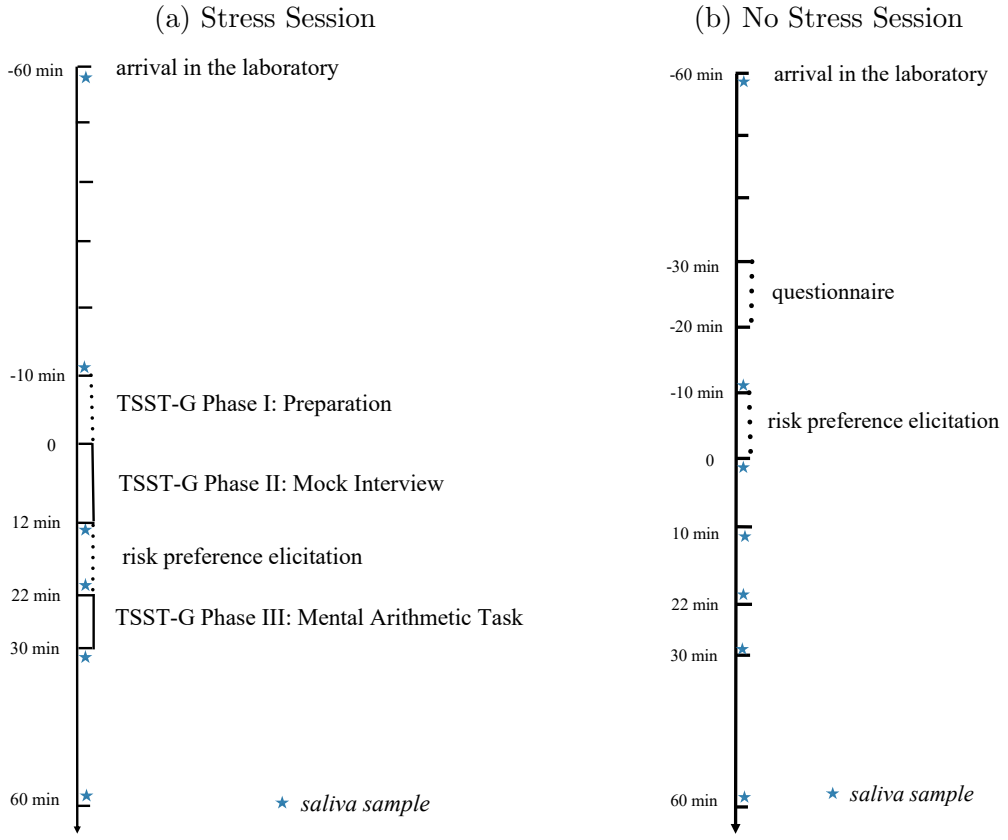
We also survey subjects' subjective chronic stress by means of the Trier Inventory for the Assessment of Chronic Stress (TICS, Schulz and Schlotz, 1999).

2.4 Measure Risk Taking

To measure subjects' risk taking, we use 30 lottery pairs similar to those in Hey and Orme (1994). Each lottery pair consists of a riskier lottery and a less risky lottery. In each lottery pair, subjects choose the preferred one. We collect data on both the subjects' choices and the response time for each subject to make a decision.

2.5 Experimental Design

Figure 1: Experiment Timeline



[5]See Stalder and Kirschbaum (2012, p. 1020) for an overview of the advantages and features of cortisol sampling via hair.

We conduct the experiment between 2pm and 5pm on weekdays, to control for circadian variation in cortisol (Kirschbaum and Hellhammer, 1989). In order to ensure comparable cortisol measures, we specify in the invitation message that we invite only subjects who do not suffer from somatic diseases, neurological or psychiatric disorders, heavy smoking, alcohol, or drug consumption. We also ask subjects to follow certain behavioural instruction, for instance, to restrain from alcohol consumption twelve hours, and from smoking and caffeine intake one hour before participation.

Each subject is invited to two laboratory sessions, with one week between the sessions. In the *Stress* session, we induce acute stress by the TSST-G; in the *No Stress* session we do not acutely stress the subjects. In both sessions, we elicit subjects' risk preferences. The order of the two sessions is randomized. Figure 1a summarizes the procedure of the *Stress* session, while Figure 1b summarizes the procedure of the *No Stress* session.

In both sessions, we eliminate potential pre-treatment acute stress and arousal because of the unusual situation of a laboratory experiment by allowing for a 50-minutes adaptation phase before the onset of the treatment intervention (Dohmen et al., 2017). During the adaptation period, subjects are allowed to read magazines or are asked to answer various questionnaires. In the *Stress* session, we then proceed with the standard TSST-G protocol immediately after the adaptation period. Between the public speaking task and the arithmetic task, individual risk taking is elicited. There is no break between the TSST-G and the risk elicitation. In the *No Stress* session, we elicit subjects' risk preferences after the adaptation period.

As shown in Figure 1, saliva samples are taken throughout the experiment. We use saliva cortisol elevation as the biomarker for successful acute stress induction in the *Stress* session. In order to measure chronic stress, we collect a strand of hair (thickness of a pencil or several smaller strands) below the subject's cranial bone close to their scalp. Hair sampling takes place at the end of the *Stress* session. The saliva samples are stored in the freezer, the hair strands are wrapped in aluminium foil. All samples are send to Dresden LabService GmbH for analysis (saliva samples are packaged on dry ice).

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