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**Investigating the impact of individual differences and stress on decision-making  
performance under threat**

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## Abstract

Quality of life largely depends on the outcomes of our decisions. A common model for understanding decision-making is reinforcement learning. Reinforcement learning involves deciding how to behave based on the appraisal of the situation and the post-evaluation of the positive or negative outcomes of decisions. In reinforcement learning, the difference in appraised value between expectations and actual outcomes is referred to as reward prediction error (RPE). Dopaminergic neuronal firing activity in the midbrain has been shown to encode RPE. RPE is used as a signal to guide decisions; for example, when decision outcomes are worse than expected (negative RPE), then those decisions are subsequently avoided. In contrast, when decision outcomes are better than expected (positive RPE), then those decisions are likely to be repeated. There is limited research explaining how individual differences such as age, gender, years of education, history of acute and chronic stress, and personality might impact decision-making performance under threat. For example, although a certain level of stress can be adaptive and improve cognitive and physical performance, including decision-making, prolonged and repeated exposure to stress has been negatively associated with both mental and physical health and longevity. As such, a history of acute or chronic stress might impact decision-making under threat; however, the interrelationship between individual differences, stress and decision-making under threat is still poorly understood. This thesis attempts to synthesise and expand existing knowledge regarding the relationship between decision-making performance, individual differences and stress. Hence, a novel decision-making task was designed and deployed in order to test the ability to learn from positive and negative RPE during safe and threatening conditions. The decision-making task, along with self-rated surveys associated with individual differences in demographics, personality, and history of acute and chronic stress, were delivered both online ( $N=109$ ,  $M=$

37.09,  $SD= 10.9$  years), using a crowd sourcing platform, and within a laboratory setting ( $N=107$ ,  $M= 19.42$ ,  $SD= 3.77$  years). In the online experiment we identified several significant linear regression models predicting the overall average of win-stay (i.e. correctly staying with a choice following a positive RPE) and lose-switch (i.e. correctly switching a choice following a negative RPE) performance across both safe and threat conditions. One of such models having age, gender, years of education, personality, acute and chronic stress factors as predictive variables, explained 34.7% of the variance in overall average win-stay performance across safe and threat conditions, and 30.8% of the overall average lose-switch performance across safe and threat conditions. In the lab experiment, we identified significant linear regression models predicting the difference in mean win-stay and lose-switch performance between threat and safe conditions. One of such models having age, gender and years of education as predictive variables, explained 10.2% of the variance in the difference of lose-switch performance between threat and safe conditions. Another model having age, gender, years of education, personality, acute and chronic stress factors, as well as the difference in heart rate between threat and safe conditions, as predictive variables, explained 22.0% of the variance in the difference of win-stay performance between threat and safe conditions. Such findings contribute to the body of knowledge regarding the impact of individual differences and stress on decision-making performance under threat, and could guide the design and development of stress management prevention and intervention decision support systems.

## **Declaration**

I certify that this work contains no material which has been accepted for the award of any other degree or diploma in my name, in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. In addition, I certify that no part of this work will, in the future, be used in a submission in my name, for any other degree or diploma in any university or other tertiary institution without the prior approval of the University of Adelaide and where applicable, any partner institution responsible for the joint award of this degree.

I give permission for the digital version of my thesis to be made available on the web, via the University's digital research repository, the Library Search and also through web search engines, unless permission has been granted by the University to restrict access for a period of time.

I acknowledge the support I have received for my research through the provision of a Medical School Scholarship.

Name: Manuel Salazar

Signature:

Date: 21 March 2024



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## Structure of the thesis

The thesis is structured as follows. In [Chapters 1-3](#), we present a thorough exploration of the literature on the neural mechanisms of decision-making, with a particular focus on the role of dopamine in this process, as well as a consideration of how this may be affected by both stress and individual factors, such as age, sex and personality variables. Given the extensive literature reviewed in [Chapters 1-3](#) and the gaps identified as part of this work, [Chapters 4 and 5](#) then explore the effect of threat and stress on decision-making in both an online and lab-based study, while [Chapter 6](#) looks at how personality variables may affect these relationships. Specific research questions or hypotheses are presented at the start of each data chapter (i.e. [Chapters 4-6](#)), prior to presentation of the methods and results, but an individual introduction is not provided for these chapters, in order to avoid redundancy with the first three chapters. At the end of [Chapters 4 and 5](#), a brief summary of the results is presented. However, given the inter-related nature of the three data chapters, a full discussion of results is reserved until the end of [Chapter 6](#).

## **Thesis contributions**

The novelty of this thesis, in comparison to previous decision-making research, lies in its comprehensive assessment of various individual factors, such as age, gender, education, personality, and stress history, as well as physiological factors, like heart rate stress reactivity, on decision-making performance under both safe and threatening contexts. By considering multiple factors simultaneously, the study provides a more holistic understanding of decision-making and enhances ecological validity. Furthermore, the use of linear models allows for a more sophisticated analysis of the relationships between individual factors, past history of stress, and decision-making performance.

At the core of our methodological innovation lies the development of an open-source and scalable decision-making task, which can be deployed on-line and off-line and fosters transparency, collaboration, and reproducibility within the scientific community. Its scalability empowers researchers to tailor experimental paradigms to suit specific research objectives, thereby facilitating the exploration of additional nuances in decision-making performance.

In contributing to the broader literature, our research not only expands our understanding of decision-making, but also lays the groundwork for future investigations in this area. By illuminating the intricate relationship between individual differences, stress, and decision-making performance, we provide valuable insights that can inform theoretical models and practical interventions aimed at improving decision-making outcomes across diverse domains. Ultimately, our work aims to pave the way for more holistic and nuanced approaches to decision-making research that better reflect the complexities of human behavior in real-world settings.

## **Chapter 1: Neurobiology of decision-making based on the dopamine Reward Prediction Error (RPE) theory**

### **1.1 Decision-making and reinforcement learning**

Human decision-making is an adaptive and pervasive cognitive process essential for survival. It involves integrating sensorial experiences with previously learnt positive or negative outcomes, in order to choose between alternative actions (Cox and Witten, 2019). Actions which lead to negative outcomes are more likely to be avoided, replaced or decreased. Conversely, those leading to positive outcomes are more likely to be repeated. As such, reinforcement learning (RL) provides a valuable framework to model how decision-making is guided by evaluating and learning the likely outcomes of a selected action (Sutton and Barto, 2018).

### **1.2 Reward prediction error**

In the context of RL, reward prediction error (RPE) is defined as the difference between the value of an actual outcome that follows a given response to contextual stimuli or cues, and the value of the outcome that was expected (Schultz, Dayan and Montague, 1997; Sutton and Barto 1981). The concept of RPE emerged from early learning experiments and theories (Kamin 1969, Rescorla & Wagner 1972) and was later incorporated into RL algorithms (Sutton and Barto 1981). RPE can be conceptualised as a signal which guides new learning and influences subsequent decisions to select or avoid potential actions (Schultz, 2015). For example, if RPE is positive (i.e. the outcome was better than expected), then responses to stimuli are more likely to be repeated in the future. However, if RPE is negative (i.e. the outcome was worse than expected), then current responses to stimuli are less likely to be repeated and more likely to be replaced with other actions, or the stimuli avoided. Finally, when the expected and actual

outcome are close or equal to each other, RPE no longer influences updating the expected value (Sutton and Barto, 2018). This condition could mean that the stimulus-action-outcome associations have been learnt (Horvitz, 2009).

### **1.3 Cortico-basal ganglia circuits and reward prediction error**

As discussed earlier, RPE involves comparing expected and actual decision outcomes. In animal studies, representations of expected outcomes are observed as increased brain activity within the orbitofrontal and prefrontal cortex, as well as the amygdala (Amemori and Sawaguchi, 2006; Frank and Claus, 2006). In contrast, neural activity corresponding to the actual outcome value of a choice is widespread in the brain and has been found in the orbitofrontal cortex (Padoa-Schioppa and Assad, 2006; Sul et al., 2010), medial frontal cortex (Sul et al., 2010), dorsolateral prefrontal cortex (Kim and Lee, 2011) and dorsal striatum (i.e. caudate and putamen; Lau and Glimcher, 2008; Kim et al., 2009; Cai, Kim and Lee, 2011). Importantly, various animal and human studies have identified cortico-basal ganglia circuits as being essential for RPE (the comparison between expected and actual outcome value), RL and decision-making (for reviews see Lee, Seo and Jung, 2012; Klaus, Alves da Silva and Costa, 2019).

RL-driven decision-making relies on circuits linking the cortex to the basal ganglia. Cortical activity precedes striatal activity of the basal ganglia during movement initiation (Schultz and Romo, 1992; Seo, Lee and Averbach, 2012). The dorsal striatum, the input nucleus of the basal ganglia, integrates cortical sensory and cognitive information, and relays motor plans from the motor cortex to the thalamus, via the basal ganglia's direct and indirect pathways (Li et al., 2015, Pidoux et al., 2011; Reig and Silberberg, 2014; Gremel and Costa, 2013; Stalnaker et al., 2016). The dorsal striatum includes topographically aligned motor and somatosensory cortical neuron projections that converge from multiple cortical areas (including frontal cortex)

(Hintiryan et al., 2016; Hooks et al., 2018). Such cortical neuron projections between the cortex and the striatum are essential for converting specific cognitive states and plans from the cortex into specific motor commands that initiate movement (Li et al., 2015). This process is critical for coordinating and executing actions and is an integral part of the brain's control over voluntary movements (Li et al., 2015).

Despite the cortex selecting a specific motor plan (Seo, Lee and Averbeck, 2012), the basal ganglia must evaluate the plan against the specific contextual information before it commits to executing the movement (Thura and Cisek, 2017). As such, the basal ganglia play a central role in processing cortical information concerning internal and external states, and enabling the evaluation and selection of motor plans (Reig and Silberberg, 2014, Stalnaker et al., 2016). These motor plans aim to execute motor actions that either pursue positive outcomes or avoid negative ones. Evaluation of planned actions is based on factors like expected outcomes, rewards, and costs (Gremel and Costa, 2013). Once the most suitable action is determined, signals are sent to brainstem motor centers to execute the chosen motor program (Li et al., 2015).

The basal ganglia contribute to learning from outcomes by adjusting action selection based on positive and negative RPE feedback (for review see Calabresi et al., 2014). The basal ganglia inhibit inappropriate motor actions via suppression, and enable selected actions through disinhibition of the thalamus (for review see Gerfen et al., 2011). Action suppression follows from the activation of the basal ganglia's indirect pathway, whilst action disinhibition follows from the activation of its direct pathway (for review see Gerfen et al., 2011). Both the direct and indirect pathways originate in the striatum, but they project to different structures within the basal ganglia. The direct or striatonigral pathway consists of approximately half of the striatal medium spiny neurons (MSNs) that innervate the basal ganglia output nuclei (Gerfen et al., 1990). The direct pathway is more sensitive to positive RPE, and includes the following

nuclei: striatal MSNs that express D1 dopamine receptors, the substantia nigra *Pars* reticulata (SNr), and the globus pallidus internal (GPi) (see Figure 1) (Gerfen et al., 1990). In contrast, the remaining striatal MSNs are part of the indirect or striatopallidal pathway. The indirect pathway is sensitive to negative RPE, and includes the following nuclei: MSNs in the striatum that express D2 dopamine receptors, the globus pallidus external (GPe), the subthalamic nucleus (STN), the SNr, and GPi (see Figure 1) (Gerfen et al., 1990).

Figure 1 below summarises the brain nuclei and the type of neuronal connections between them which have been associated with decision-making in cortico-basal ganglia circuits.

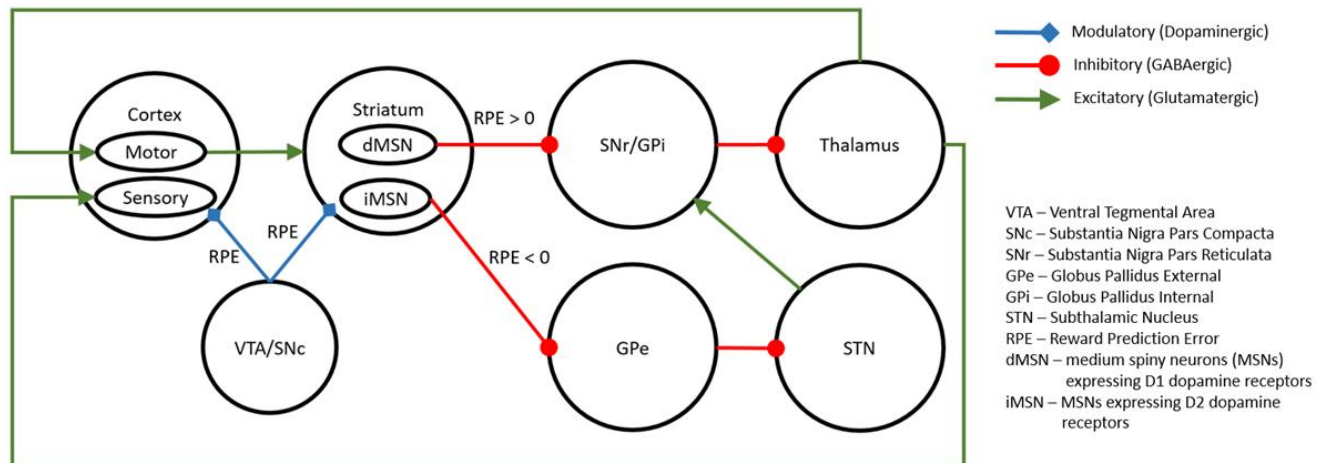


Figure 1 Basal ganglia nuclei involved in the classic reinforcement learning model of decision-making. The circles of the cortico-basal ganglia circuit diagram above, represent brain nuclei, and the lines represent neuron pathway connections, between nuclei, that either modulate, inhibit, or excite connected nuclei. The red lines, terminated with solid circles, represent inhibitory connections, which consist of GABAergic neurons. The green arrows, represent excitatory connections, which consist of glutamatergic neurons. The blue lines, terminated with solid squares, represent modulatory connections, which consist of dopaminergic medium spiny neurons (MSNs). The neural activity of the direct pathway (labelled dMSN) is modulated by dopaminergic MSNs from the SNc, which activate postsynaptic MSNs in the striatum, that express D1 dopamine receptors. When there is a positive reward prediction error (RPE), a dopamine burst triggers the striatum to disinhibit the thalamus via the direct pathway, which results in the execution of cortex motor plans. In contrast, the neural activity of the indirect pathway (labelled iMSN) is modulated by dopaminergic MSNs, from the SNc, which activate postsynaptic MSNs in the striatum, that express D2 dopamine receptors. When there is a negative RPE, a dopamine dip triggers the striatum to further inhibit the thalamus via the indirect pathway, which results in halting cortex motor plans. Such mechanism of inhibiting and disinhibiting the thalamus, which is modulated by dopaminergic MSNs, encoding RPE, from the SNc, allows for fine tuned control of cortex motor plans. The dopaminergic MSNs within the SNc are sensitive to dopamine bursts and dips associated with changes in RPE, which arise from evaluating and appraising the outcomes of motor actions.

#### 1.4 The ventral tegmental area and reward prediction error

As shown in [Figure 1](#), striatal activity is influenced by RPE signals arising from, not only dopaminergic activity of the substantia nigra *Pars compacta* (SNc), but also dopaminergic projections arising from the ventral tegmental area (VTA) ([Montague, Dayan and Sejnowski, 1996](#); [Schultz, Dayan and Montague, 1997](#); [Watabe-Uchida, Eshel and Uchida, 2017](#)). The RPE function of these dopaminergic neurons, and their role in RL, has been shown to involve phasic dopamine release in reinforcement-based learning (for review, see [Watabe-Uchida, Eshel and Uchida, 2017](#)). A causal link between prediction errors, VTA dopamine neurons, and learning was observed in rats by using optogenetic manipulations ([Steinberg et al., 2013](#)). The optogenetic manipulations artificially activated VTA dopamine neurons to mimic a positive RPE signal and facilitate learning of a redundant cue that would have otherwise been ignored in a blocking learning experiment ([Steinberg et al., 2013](#)). This demonstrates that RPE signals from the VTA are a sufficient condition to induce new learning.

Single-cell recordings in the VTA of macaque monkeys have also confirmed that the phasic activity of dopaminergic neurons encodes RPE ([Schultz, Dayan and Montague, 1997](#)). When a reward is first experienced after a cue, dopaminergic neurons in the VTA and SNc produce high phasic activity beyond baseline tonic activity ([Schultz, Dayan and Montague, 1997](#)). Such phasic activity occurs very close to the time the reward was experienced. At this stage, the activity can be represented as a large positive RPE, due to the unexpected reward. After consecutively experiencing the cue and reward, however, the phasic response gradually reduces to baseline tonic activity. This is presumably because, as the reward becomes more frequently expected after the onset of the cue, the RPE begins to approach zero; that is, the reward is no longer surprising. Once RPE reaches zero, the phasic activity now occurs at the time when the cue occurred (signalling the expected outcome generated by the cue), instead of occurring at the time of reward ([Schultz, Dayan and Montague, 1997](#)). Conversely, if the



reward is not experienced following the cue, then the activity drops below baseline activity at the time when the reward was expected, which arguably represents a negative RPE to signal the surprising absence of the reward (Schultz, Dayan and Montague, 1997). These neurons can therefore signal both positive and negative RPEs in order to learn which actions are likely to be followed by positive or negative outcomes and guide decisions (see Figure 2).

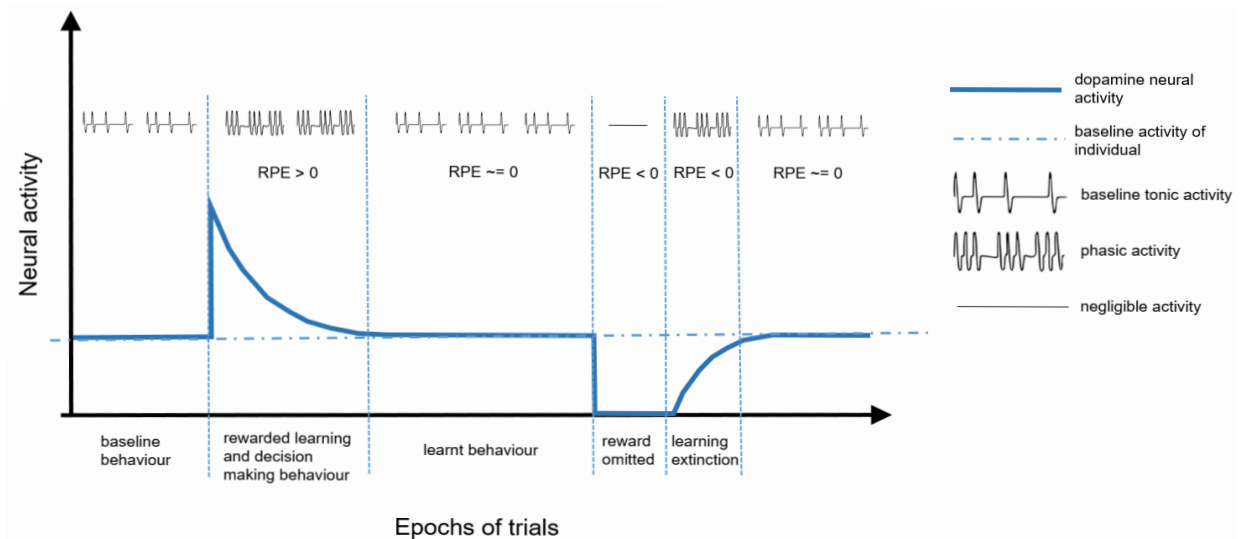


Figure 2 Dopaminergic activity of reinforcement learning. The vertical axis represents neuronal firing activity of dopaminergic MSNs of the VTA and SNc. The horizontal axis represents epochs of time elapsed across a hypothetical reinforcement learning process. In the beginning, the dopaminergic MSNs are firing at tonic baseline levels, reflecting baseline behaviour. An unexpected reward is first experienced following a certain action, and this event causes a large dopamine burst of phasic activity, which is significantly higher than tonic baseline activity, and that occurs close to the time of the unexpected outcome. The experienced dopamine burst represents a large RPE. As such, the action that resulted in the positive outcome is likely to be repeated again. This begins rewarded learning and decision-making behaviour to repeat the action. Each time the action-outcome events occur the phasic activity of the neurons gradually approaches baseline activity, or zero RPE. At this stage, the association between action and positive outcome results in a new learnt behaviour. When the same action occurs and there is no expected reward, then a sudden dopamine dip is experienced. The dopamine dip is associated with a halt in neurons firing that is significantly less than tonic baseline. Such event, signals a large negative RPE. When the action following no expected reward is repeated, the frequency of performing the action diminishes causing learning extinction, and neuron activity gradually returns back to tonic baseline, or zero RPE. At this stage, the previously learnt association between action and positive outcome has been extinguished.

## 1.5 Dopaminergic pathways and reward prediction error

Dopaminergic neurons from the VTA and SNc project both positive and negative RPE signals to multiple locations in the striatum (Ikemoto, 2007; Beier et al., 2015). Dopaminergic neurons from the VTA project to the ventral striatum, and those from the SNc to the dorsal striatum. Classically, the dorsal striatum is the origin of the two major pathways known as the direct and indirect pathway, which respond differently to dopamine, and therefore may play distinct roles in RPE and RL.

Direct pathway MSNs (D1-MSNs) express D1-type dopamine receptors, and indirect pathway MSNs (D2-MSNs) express D2-type dopamine receptors (Gerfen et al., 1990). The profile of dopamine receptor expression makes D1-MSNs more excitable to dopamine bursts compared to D2-MSNs (Gerfen et al., 2011; Planert, Berger and Silberberg, 2013). In contrast, D2-MSNs are more excitable to dopamine dips in comparison to D1-MSNs. Such observations lead to the classical model characterised by pro- versus anti- kinetic functions associated with the direct and indirect pathways, respectively (Gerfen et al., 2011; Planert, Berger and Silberberg, 2013). In the classic model, the activity of D1-MSNs facilitates movement, or action selection, and they are highly active during movement production, whereas the activity of D2-MSNs inhibits movement, or action selection, and therefore they exhibit lower activity during movement (Gerfen et al., 2011; Planert, Berger and Silberberg, 2013). Conversely, during immobility, activity in D2-MSNs is higher and activity in D1-MSNs is lower. This model is supported by both optogenetic (Kravitz et al., 2010) and pharmacogenetic (Alcacer et al., 2017) manipulation experiments, in which the activation of many D1-MSNs leads to more movement and activation of many D2-MSNs inhibits movement. Notably, many of these features are modulated by dopamine, which can shape the balance between the direct and indirect pathways to influence motor behaviour (Planert, Berger and Silberberg, 2013; Dobbs et al., 2016; Parker et al., 2018). Indeed, accumulating evidence suggests that self-paced

movement initiation is regulated by dopamine in a very dynamic fashion (Klaus, Alves da Silva and Costa, 2019). The classic model described can aid in understanding individual differences in decision-making actions that are guided by dopamine dynamics associated with negative or positive RPE.

D1 and D2 receptors have relatively low and high affinities for dopamine, respectively (Creese et al., 1983; Yin and Knowlton, 2006). D1 stimulation is therefore hypothesized to depend on phasic dopamine bursts, with larger bursts producing greater stimulation (Shen et al., 2008). Burst magnitude is therefore crucial for D1-mediated long-term potentiation (LTP) as a result of positive RPE (Calabresi et al., 2007). D2 receptors, in contrast, are hypothesized to be stimulated tonically by low baseline dopamine levels (Shen et al., 2008). The effect of pauses in dopaminergic neuron firing on D2 receptors, therefore, depends on dopamine reuptake, with longer pauses allowing greater reuptake and therefore producing a larger dip in dopamine concentration (Maia and Frank, 2011). Pause duration is therefore crucial for D2-mediated LTP as a result of negative RPE (Calabresi et al., 2007). Long-term depression (LTD) mechanisms are also consistent with a key role for magnitudes and durations in coding positive and negative prediction errors, respectively (Calabresi et al., 2007). Because D2 receptors have a high affinity for dopamine, positive RPE stimulates D2 receptors directly, further suppressing the indirect pathway, which consequently leads to LTD in the indirect pathway (Calabresi et al., 2007). Negative RPE, however, may not strongly affect D1 receptors, because D1 receptors may not be substantially stimulated by tonic dopamine (Maia and Frank, 2011).

Therefore, because unexpected rewards cause positive RPEs, which are represented by bursts in dopamine activity from the VTA to the striatum, these events are more likely to induce D1-mediated neuroplasticity in direct-pathway MSNs. Such neuroplasticity in the direct pathway ensures the action that immediately preceded the reward is more likely to be

repeated in the future, since this action plan will activate the direct pathway more strongly, which will make it more likely to be selected. In contrast, the absence of expected reward or punishment typically elicits a negative RPE that is coded by VTA neurons as a drop in baseline dopaminergic activity. This drop is more likely to affect indirect pathway neurons in the striatum that express D2 receptors. This D2-mediated neuroplasticity in the indirect pathway will make the action plan less likely to be repeated in the future by preventing its action selection.

Figure 3 below summarises hypothetical dopamine dynamics as a result of RPE that could trigger LTD or LTP in the direct or indirect pathways. Such a model, give insights into decision-making behaviour guided by positive or negative RPE.

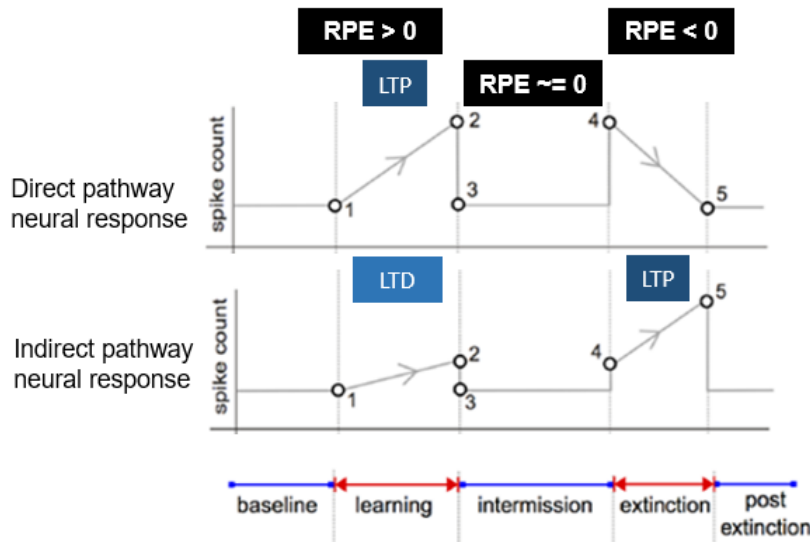


Figure 3 Dopamine activity of direct and indirect pathways during reinforcement learning. The direct and indirect pathways play a central role in decision-making: actions that are followed by negative consequences (that trigger a negative RPE – coded by a phasic dip in dopamine release) will strengthen the indirect pathway via D2-mediated neuroplasticity, making it less likely that this action will be selected in the future, whereas actions that are followed by positive consequences (that trigger a positive RPE – coded by a phasic burst of dopamine release) will strengthen the direct pathway via D1-mediated neuroplasticity, making it more likely that this action will be selected in the future. The diagram highlights whether long term potentiation (LTP) or long term depression (LTD) neuroplasticity occurs within the direct and indirect pathways based on a hypothetical reinforcement learning process. The horizontal axis represents the stages of the reinforcement learning process. The vertical axis represents the neuron spike count of activity within the dorsal striatum which expresses D1 and D2 receptors. Initially, baseline tonic activity is associated with baseline behaviour. Learning follows a positive prediction error and increases neuron activity, from the corresponding dopamine burst in the direct pathway, which leads to LTP in the direct pathway and LTD in the indirect pathway. Once learning occurs, RPE is near zero, and tonic baseline activity returns through the intermission stage. When a negative prediction error occurs, the indirect pathway is activated by a dopamine dip, and the previous learnt behaviour gradually reaches extinction, through LTP in the indirect pathway, as tonic baseline is reached at post extinction of the previously learnt behaviour.

## Chapter 2: Decision-making under threat

### 2.1 Research motivations

Given the findings above, the evidence for how RPE dopamine dynamics modulate decision-making in cortico-basal ganglia circuits is compelling. However, what is less clear is how the performance of these circuits is altered by factors that might influence decision-making ability, such as individual differences in threat appraisal and stress reactivity. Such research is currently underdeveloped and is important for two main reasons. Firstly, understanding decision-making competence (Weller et al, 2018), and the individual factors that may influence it, is crucial to survive and thrive in both safe, and more importantly, in threatening environments (for reviews see Woody and Szechtman, 2011; LeDoux, 2018; Levy and Schiller, 2021). Secondly, decision-making under threat might cause stress, which may negatively interfere with and influence decision-making performance (for review see Starcke and Brand, 2012). Furthermore, if threatening conditions continue over long periods of time, repeated stress may result in negative impacts to physical and mental health (Cohen et al., 2007; McEwen, 2008; Schneiderman et al., 2005). For example, repeated stress may pose a risk to developing chronic stress, which has been associated with inflammation and disturbances to homeostasis that can negatively impact longevity, physical and mental health (Chen and Miller, 2007; Kivimäki et al., 2006).

Understanding the mechanisms by which decision-making performance under threat varies as a result of individual differences, and both acute and chronic stress, could assist in the design of decision support systems incorporating health monitoring functionality. Such decision support systems could attempt to optimise the decision-making performance of individuals under threatening and stressful conditions. They could also monitor anomalies in decision-making performance. These anomalies could be used, in conjunction with predictive models

of stress, to 1) highlight any vulnerabilities or risk factors that might lead to poor decision-making under threat and 2) detect improvements in resilience as a result of stress management interventions.

## **2.2 Definition of threat and stress**

There are a variety of ways to define a threat (for reviews see [Woody and Szechtman, 2011](#); [LeDoux, 2018](#); [Levy and Schiller, 2021](#)). However, for the purpose of this thesis, we use the term threat to describe improbable, unpredictable and/or uncontrollable stimuli or situations with the potential to harm, damage, or cause significant loss. Threats might be experienced as stressors, i.e., stimuli or situations that elicit stress. Although the stress response is essential for adaptation and survival, the term ‘stress’ is today generally associated with a negative experience ([McEwen, 2013a, 2013b](#)). Work-related stress is common and chronic exposure to stress is linked to various neuropsychiatric disorders, such as major depressive disorder ([Calabrese et al., 2009](#); [Kessler, 1997](#)).

For the purpose of this thesis, we align to the definition that stress is “an actual or anticipated disruption for homeostasis, or an anticipated threat to well-being” ([Ulrich-Lai and Herman, 2009](#)), and similarly, that stress is “a bodily reaction to a perceived threat (i.e., a stressor) to homeostasis” ([Sapolsky, 1994](#)). Homeostasis is the ability of an organism to maintain the internal environment of the body within limits that allow it to survive ([Chrousos, 2009](#)). Homeostatic mechanisms, including the stress response, usually exert their effects in an inverted U-shaped dose–response curve ([Chrousos, 2009](#)). As such, it can be speculated that, within range limits, a stress response of intermediate levels might be adaptive and result in near-optimal decision-making performance. However, at lower or higher range limits, the stress response could be maladaptive and significantly impair performance.

A stress response which has been studied in laboratory conditions has been characterised by the activation of the autonomic nervous system, the endocrine system, the hypothalamus pituitary adrenal axis (HPA-axis) (Selye, 1956) and/or the sympathetic adrenomedullary system (SAM-system) (Cannon, 1914). This stress response has been termed the ‘fight or flight’ response (Cannon, 1914). For example, in human stress research, the Trier Social Stress Test (TSST), which simulates a 15-min job interview and judged public speaking, is a commonly used stress protocol, and is known to activate both the autonomic nervous system and the HPA-axis, in a similar way to the ‘fight or flight’ response (Kirschbaum et al., 1993). Common experimental measures for physiological responses to the TSST include heart rate, breathing rate, blood pressure and salivary glucocorticoids levels (e.g. cortisol) (van den Bos et al, 2009; Starcke et al, 2011; Youssef et al, 2012; Kirschbaum et al., 1993).

Experimental data demonstrate that stress can have both immediate (acute) and long-lasting (chronic) effects on brain and behaviour (Duckworth et al., 2011; Kandasamy et al., 2014; Lewis et al., 2014; McEwen and Morrison, 2013; Schwabe and Wolf, 2010), and even relatively moderate and acute stressors have been shown to affect decision-making (Gathmann et al., 2014; Lempert et al., 2012; Porcelli and Delgado, 2009; Porcelli et al., 2012; Schwabe et al., 2012; Schwabe and Wolf, 2009; Starcke et al., 2008).

In addition to DA, other neurochemical messengers including adrenaline, noradrenaline, serotonin, glutamate (Moore and Lariviere, 1964) and glucocorticoids (Carini and Nephew, 2013; Corum and Thurmond, 1977) have also been shown to be involved in the stress response (for review see Vaessen et al., 2015). However, this thesis specifically focusses on the effects of stress on dopamine dynamics because they are best described in the context of decision-making. As discussed earlier, it is known that dopamine neurons encode RPE in the midbrain, striatum and frontal cortex, which form basal-cortical circuits essential for decision-making. Hence, it follows that dopaminergic changes, as a result of stress, might significantly impact



decision-making performance under threat. Importantly, we distinguish here between acute (temporary) and chronic stress, as they have been shown to impact the DA system differently and might hence have different effects on decision-making.

### **2.3 Acute stress**

Acute, temporary, stress has been observed to impair prefrontal functions, such as directing attention and inhibiting task-inappropriate actions, which are fundamental for goal-based action control (for reviews see [Arnsten, 2009](#); [Starcke and Brand, 2012](#)). At the same time, acute stress has been reported to amplify craving or wanting signals that might bias an individual toward choosing immediately rewarding options ([Adam and Epel, 2007](#); [Pruessner et al., 2004](#); [Sinha et al., 1999](#)).

Human studies that used functional magnetic resonance imaging (fMRI) or positron emission tomography (PET) have found that the exposure to acute stressors leads to increased brain activity within different regions, including the prefrontal cortex, basal ganglia and thalamus (for review see [Dedovic et al., 2009a](#)). However, mixed results (showing either a decrease in or an increase in activity) were found for the amygdala, the thalamus and the insular cortex ([Dedovic et al., 2009b](#); [Pruessner et al., 2008](#); [Tillfors et al., 2002](#); [Wang et al., 2005](#)). These mixed results might be explained by individual differences in stress reactivity (for review see [Kudielka et al., 2009](#)).

For instance, [Pruessner et al. \(2008\)](#) highlights individual differences between stress responders and non-responders after experimentally-induced acute stress using the Montreal Imaging Stress Task ([Dedovic et al., 2005](#)), where subjects are exposed to challenging mental arithmetic presented on a computer screen. The authors divided their participants into cortisol responders and non-responders based on whether their cortisol level increased following the laboratory stress manipulation; and demonstrated that the responders showed a deactivation of

specific parts of the limbic system, including the hypothalamus, hippocampus, amygdala, and medial-orbitofrontal cortex. In contrast, the cortisol non-responders did not show this deactivation pattern. The authors concluded that the limbic system has a high basal activity that can serve as an alarm system. Once an alarm has been given after exposure to a stressor, the activity is curtailed. The question of why some participants respond to a challenge with a cortisol response while others do not, has previously been investigated (for a review see [Kudielka et al., 2009](#)). The review by [Kudielka et al., 2009](#) identified a variety of individual differences, including age and gender, that influence the cortisol response to acute stress.

In line with this, there are various individual differences that could influence HPA-axis reactivity, including age, gender, personality, genetic factors and early life stress ([Starcke and Brand, 2012](#)). For example, early life stress ([Luecken and Appelhans, 2006](#)) and some personality variables, such as low self-esteem and low locus of control ([Pruessner et al., 2005](#)), are thought to increase the reactivity of the HPA-axis. Additionally, a habituation effect occurs in most participants after repeated exposure to the same stressor ([Schommer et al., 2003](#)), and therefore prior experience with the respective challenge may be a confounding factor. However, it is not clear how such individual differences in stress reactivity influence decision-making performance under threat.

Although human studies investigating how acute and chronic stress impact RPE signals and decision-making are scarce, current knowledge of the impact of stress on these neural circuits, especially the VTA, can help us make predictions regarding the effects of stress on decision-making performance under threat. For example, in animal studies, acute or short-term life stressors can induce a change in the activity of DA neurons within the VTA (for review see [Baik, 2020](#)). Such change in activity appears to promote reward-related neural connectivity by, for example, enhancing learning of cue-reward associations ([Stelly et al, 2020](#)).

Acute stress increases DA neurotransmission within the cortex, nucleus accumbens (NAc) and striatum, which are regions anatomically connected to the VTA (Belujon and Grace, 2015; Piazza and Le Moal, 1998; Holly and Miczek, 2016; Lammel, Lim and Malenka, 2014a). Such changes increase awareness, attention, arousal and information processing (Berridge, 1998; Salamone, Cousins, Snyder, 1997). Furthermore, phasic DA activity in the NAc supports active coping strategies, goal-directed behaviour, and motivational arousal, while a diminished stress-induced DA is linked to passive coping in situations that are uncontrollable (Cabib and Puglisi-Allegra, 2012; Fiore et al., 2015).

Phasic activity is differentially expressed in VTA-DA neuron subpopulations at onset and termination of acute stressors (Douma and de Kloet, 2020). For example, previous animal microdialysis studies have observed acute stress to result in increases to tonic activity, which led to facilitated phasic VTA-DA activity (Belujon and Grace, 2015). Two, rather than one, distinct VTA-DA neuron subpopulations have been proposed as being sensitive to acute stress (Lammel et al., 2014 a,b; Ungless et al., 2010). One is a dopaminergic neuron population found in the dorsolateral VTA region, which is mainly inhibited by acute stress (Guarraci and Kapp, 1999; Mantz et al., 1989; Mirenowicz and Schultz, 1996; Schultz and Romo, 1987; Ungless, 2004); however, these neurons show phasic excitation upon termination of the stressor (Brischoux et al., 2009; Navratilova et al., 2012; Tanimoto et al., 2004). In contrast, a second dopaminergic neuron population experiences rapid and potent phasic bursts at the onset of stressor exposure, which occurs in the ventromedial region of the VTA (Anstrom et al., 2009; Anstrom and Woodward, 2005; Cohen et al., 2012; Lammel et al., 2014 a, b; Zweifel et al., 2011). As such, it could be speculated that there might be variations in decision-making performance before or after experiencing acute stressors.

Other animal studies have shown that the activation of VTA-DA neurons upon acute stress exposure can alter VTA-DA activity responses to later stimulation (Holly and Miczek, 2016;

Valenti et al., 2012). Importantly, these alterations in VTA-DA neurons are shown in both tonic and phasic firing patterns, but appear to depend on the experimental conditions. For example, mild or intermittent stress protocols generally tend to increase VTA-DA population activity, while exposure to prolonged, more severe and uncontrollable/inescapable stress paradigms (i.e., paradigms expected to generate chronic stress) tend to blunt tonic firing in VTA-DA neurons (Chang and Grace, 2014; Rincón-Cortés and Grace, 2017; Kaufling, 2019). These differences in tonic firing may lead to transitions in the use of active and passive coping strategies in response to acute stressors (Cabib and Puglisi-Allegra, 2012; Lloyd and Dayan, 2016; Tye et al., 2013). Furthermore, when an animal is subsequently exposed to stressors that differ in nature to previous stressor(s) used in the stress protocol – the phasic responses are generally sensitized or amplified (Cuadra, 2001; Cuadra et al., 1999; Di Chiara et al., 1999; Finlay et al., 1995; Gresch et al., 2002; Murphy et al., 2003; Tidey and Miczek, 1997, 1996; Watt et al., 2014). It is somewhat difficult to understand how these results can be translated to humans, however, it can be argued that acute stress in the absence of chronic stress should temporarily enhance learning from rewards. It is also clear that individual differences of previous history of acute or chronic stress and differences associated with learning from negative or positive RPE needs to be investigated.

A focused review by Starcke and Brand (2012) highlighted diverse effects of acute laboratory stress on decision-making in humans, including dysfunctional decision strategies, inadequate adjustment from automatic responses, altered feedback processing, and modified reward and punishment sensitivity. However, the connection between these effects and reward prediction error (RPE) dopamine dynamics in the ventral tegmental area (VTA) remains unclear. The review encompassed various decision-making tasks and stress protocols, posing challenges in comparing and explaining decision-making performance variations. Notably, the studies also lacked consideration of individual differences, particularly in assessing past

experiences with stressors, which could elucidate decision-making variances and align with the proposed inverted U-shape relationship between stress and performance, as well as differences in dopamine dynamics linked to positive and negative RPE.

Taken together, evidence suggests that acute stress alters different forms of cognitive functioning, in a manner dependent on a variety of factors, including the type of task, the specific brain circuitry recruited by these tasks, and the severity of the stressor (Shors et al, 1992; Stillman et al, 1998; Cordero et al, 2003; Joe's et al, 2006; Shansky et al, 2006; Luethi et al, 2008). Based on the aforementioned findings, a hypothesis could be that a healthy acute stress response leads to increased VTA dopamine neural excitability that could potentially enhance the activity of the direct pathway of the basal ganglia. As such, participants without a history of chronic stress, but under mild acute stress conditions, might display improved reward learning, since the direct pathway might be more sensitive to positive RPE if acute stress increases the DAergic output of the VTA. Furthermore, decision-making may benefit from an increased ability to initiate and execute voluntary actions, which could lead to quicker, more decisive responses to choices and opportunities. In contrast, an increase in phasic activity and baseline levels of dopamine might make it more difficult to learn from negative RPE, because the indirect pathway is mainly sensitive to changes of activity below tonic baseline. For example, acute stress was shown to impair decision-making under uncertainty by biasing choices towards riskier options when the chances of losing were higher (Miu et al, 2008; Porcelli and Delgado, 2009; Putman et al, 2010). Such risky behaviour supports the finding that VTA-DA output is enhanced under acute stress, and consequently enhances learning from larger positive RPEs. It is important to note, however, that such behaviour may not always be adaptive. For example, in a similar experiment, participants who were stressed due to anticipating a speech performance, frequently made choices that anticipated higher rewards; however, those choices led to punishment more often (Starcke et al., 2008). This suggests that,

in such conditions, learning from rewards was enhanced while learning from punishment was diminished, consistent with the findings from animal studies that acute stress increases dopaminergic output of the VTA, potentially enhancing positive RPEs but blunting negative RPEs. Such differences in decision-making behaviour require further investigation, including how they might be influenced by individual differences in dopamine dynamics associated with previous experiences with acute or chronic stressors.

## **2.4 Chronic stress**

In contrast to acute stressors, chronic stress can have the opposite effect, and decrease the activity of VTA DAergic neurons (for review see [Douma and de Kloet, 2020](#)). Perhaps a sign of these different biological changes is that acute stressful events do not typically induce depressive behaviour, while chronic repeated stress may result in depressive behaviour ([Krishnan and Nestler, 2011](#)). Furthermore, previous history with chronic stress exposure can additionally result in permanent changes and excessive loss of dopaminergic neurons in the VTA ([Sugama and Kakinuma, 2016](#)) compared to healthy baselines.

For ethical reasons, chronic stress protocols have been designed exclusively for animal studies. For example, several validated animal models of chronic stress include chronic restraint stress (CRS), chronic unpredictable stress (CUS) or chronic mild stress (CMS), and chronic social defeat stress (CSDS) ([Christoffel et al., 2011](#); [Krishnan et al., 2008a](#); [Krishnan and Nestler, 2011](#)).

CRS involves restraining animals for a minimum of three weeks, 1–6 hours a day. Although CRS is inescapable and relatively mild, animals habituate over time, resulting in attenuated HPA-axis activation ([Radley et al., 2006](#); [Stetler and Miller, 2011](#); [Watanabe et al., 1992](#)). [Sugama and Kakinuma \(2016\)](#) utilized immunohistochemical and in situ hybridization techniques to demonstrate that a 16-week CRS leads to dopaminergic neurodegeneration in

male Wistar rats. After the CRS protocol, rodents displayed depressive-like behaviour, as validated by different panels of behavioural tests, such as the sucrose preference test, the forced swim test, and the tail suspension test (Xu et al., 2012; Cryan and Mombereau, 2004; Castagne et al., 2011).

Similarly, CUS or CMS employs various stressors (e.g., tail pinch, overnight illumination, cage tilt, damp bedding, unpleasant noises, home cage changes, food/water deprivation, etc.) in a semi-random or unpredictable order for several days or weeks, inducing numerous changes in brain and behaviour, including decreased reward responsivity (Willner, 2017). CUS or CMS also induces persistent depressive behaviours and seems to mimic the stress-induced depression observed in depressed patients (Willner, 1997; Willner, 2017).

This is similar in action to CSDS, in which male animals are exposed to a single bout of social defeat followed by separation with visual and olfactory contact. CSDS induces anhedonia, anxiety, and social-avoidance behaviours. The intermittent variant (ISDS) involves four exposures in 10 days, while the consecutive variant (CSDS) spans ten days. A variation of the CSDS was conducted by Lim et al. (2012) using 3–4 hours per day for 7–8 days. Their findings indicated a decrease in the strength of excitatory synapses on D1-MSNs but not on D2-MSNs in the core region of the NAc. This suggests that the observed reduction in excitatory transmission is specific to D1-MSNs, which are predominantly found in the direct pathway and are involved in learning to repeat rewarded actions. Importantly, the study proposed that this D1-MSN-specific alteration in excitatory transmission might be a contributing factor to the development of anhedonia, a condition characterised by a reduced ability to experience pleasure.

Beyond DA, 10 days of either CUS or CRS increased the global expression of receptors in the VTA, including Glutamate Receptor 1 and N-Methyl-D-aspartate Receptor (NMDAR) Subunit 1 (Fitzgerald et al., 1996). However, a study by Toth et al. (2008) found no evidence

for such changes after 4 weeks of CUS. Other studies using CSDS and social isolation stress paradigms demonstrated chronic stress-enhanced long-term potentiation (LTP) of NMDAR-mediated glutamatergic synaptic plasticity in the VTA (Stelly et al., 2016; Whitaker et al., 2013).

Importantly, such changes can be long-lasting. In line with this, chronic stress exposure can induce morphological changes in VTA-DA neurons, as demonstrated in a study by Kaska et al. (2017) who reported that in mice susceptible to CSDS, the VTA-DA neuron soma size decreased (Kaska et al., 2017). In this study, western blot analysis on micro-dissected VTA tissue revealed a decreased level of phosphorylated cofilin – a protein which can disassemble cytoskeletal actin filaments. Therefore, the authors hypothesized that chronic stress may alter – amongst many other effects – the cytoskeleton of VTA-DA neurons. Moreover, the ability of chronic stress to induce shrinkage of neuronal soma sizes may be related to the diminished availability of neurotrophic factors (Chu et al., 2007; Stockmeier et al., 2004).

Studies with rats subjected to CRS revealed basal-like levels of nucleus accumbens (NAc) DA activity. Interestingly, upon release from the restraining apparatus, there was a rapid and substantial increase in DA levels (Imperato et al., 1991, 1992). Subsequent experiences of the same stressor, however, did not reduce the activation of NAc DA upon release, supporting the notion that the response is linked to the positive experience of ending a still-aversive condition. This suggests that the novelty and relief associated with the end of a stressor contribute to the observed NAc DA release (Imperato et al., 1991, 1992).

Alterations in both tonic VTA DA projection targets and the phasic dopamine response in the NAc and medial prefrontal cortex (mPFC) occur in response to subsequent stressors (Imperato et al., 1992; Imperato et al., 1993). Habituation of the extracellular dopamine response in the NAc is observed during daily restraint stress for six consecutive days (Imperato et al., 1992; Imperato et al., 1993). However, when restraint is repeated after a 72-hour interval,



the phasic dopamine response in the NAc becomes equivalent to the response on the first day (Imperato et al., 1992; Imperato et al., 1993). In contrast, repeated footshock stress (Young, 2004) and intermittent social defeat stress (Holly et al., 2015) do not induce habituation in the phasic extracellular NAc dopamine response. Instead, a sensitised response is observed after milder stress manipulations, such as repeated tail pinch (Naef et al., 2013) or forced swim (Jordan et al., 1994; Petty et al., 1997).

Importantly, prolonged elevations of DA with repeated stress may lead to impairments of cellular functioning (Belujon and Grace, 2015; Kulak et al., 2013; Alghasham and Rasheed, 2014). Furthermore, many cellular and molecular changes have been described in the development of neurochemical and behavioural sensitisation following chronic stress (Cabib and Puglisi-Allegra, 2012; Lucas et al., 2004; Arnsten, 2011; Marinelli and Piazza, 2002; Muir et al., 2018; Tye et al., 2013).

Taken together, the above findings suggest long-term changes in neuroplasticity as a result of chronic stress. In general, chronic stress seemed to reduce the ability of the VTA to release DA, and to reduce the excitability of D1-MSNs found in the direct pathway. Chronic stress might therefore reduce the ability of the VTA to signal RPEs, which is necessary for learning to repeat rewarded actions. However, it is difficult to generalise such changes. For example, each aforementioned chronic stress protocol results in various distinct changes in neuroplasticity. Furthermore, such protocols only rely on animal models. As such, we shall consider the possibility that there may be individual differences in life experiences associated with a variety of acute and chronic stressors that might result in differences in neuroplasticity within the basal ganglia in people. Such differences might impact dopamine dynamics within the VTA, LTD or LTP within the direct and indirect pathways, and consequently on decision-making performance under threat.

In summary, we hypothesise that participants with a history of chronic stress may have a less active VTA circuit. Reduced VTA circuit activity means a reduction in dopaminergic input into the direct pathway of the basal ganglia, which is responsible for initiating voluntary movements and learning to repeat rewarded actions. As such, decision-making may be affected by a decreased ability to initiate and execute voluntary actions, as well as a reduced tendency to repeat successful actions, and slower and less decisive responses to choices and opportunities. Thus, if individuals with a history of severe life stressors or chronic stress are more likely to have less active VTA circuits compared to healthy individuals, then their baseline dopamine levels might be lower, such that the direct pathway might not respond as much to rewards and learn less to repeat rewarded actions. However, since the indirect pathway is more sensitive to phasic drops in dopamine release, then it might not be as affected when learning to avoid losses under an acute stress compared to healthy individuals.

## Chapter 3: Decision-making, stress and individual differences

Stress elicits psychological, physiological and behavioural responses and there is evidence for large inter-individual differences in stress reactions (for reviews see [Kudielka et al., 2009](#); [Starcke and Brand, 2012](#)). Many factors may impact reactivity to an acute stress manipulation, such as genetic factors, a history of early life stress, or personality. One may assume that individual factors determine the thresholds for hormonal responses to stress exposure ([Starcke and Brand, 2012](#)). Individual factors, such as susceptibility to stress, age, gender or personality variables, might therefore also influence decision-making performance under threat ([Kudielka et al., 2009](#)). However, these relationships have not yet been systematically examined. The following section discusses how age, gender, and personality might influence decision-making performance under threat.

### 3.1 Age differences

Age has been associated with a volume decline in prefrontal brain regions and declines in DA transmission effectiveness ([Raz, 2000](#); [Volkow et al., 2000](#)). For example, age-related grey matter volume decline within decision-making regions, including the lateral orbitofrontal cortex and dorsolateral prefrontal cortex, have been observed using cross-sectional ([Driscoll et al., 2009](#); [Good et al., 2001](#); [Kennedy et al., 2009](#)) and longitudinal ([Pfefferbaum et al., 1998](#); [Resnick et al., 2003](#)) grey matter volumetric analyses. Additionally, significant reductions in the volume of ventral and dorsal striatal regions have been observed, surpassing the decline seen in structures like the amygdala and VTA/SNc, which tend to be better preserved during the aging process ([Raz et al., 2005](#); [Raz et al., 2010](#); [Walhovd et al., 2011](#)).

In terms of age-related declines in DA transmission effectiveness, [Karrer et al. \(2017\)](#) conducted a meta-analysis revealing a significant decline in DA transporters and receptors, with D1-like receptors being more affected than D2-like receptors. On average, the entire DA

system (e.g., receptor subtypes, transporters, synthesis capacity) was found to decline between 3.7% and 14.0% per decade. However, no age-related impact was observed on DA synthesis capacity, and age-related decline in dopamine receptor binding was most prominent in frontal brain regions, with relatively less decline in striatal regions (Karrer et al., 2017). Furthermore, D2-like binding potential in the VTA or SNc has been observed to have negligible age-related changes, and in some cases, increased binding potential was observed (Matuskey et al., 2016; Nakajima et al., 2015).

Despite the age-related changes discussed above, it is not clear how such changes impact decision-making performance. For example, there is conflicting evidence regarding age-related sensitivity to feedback during learning. Some studies suggest that older adults may be more sensitive to positive than negative feedback compared to younger adults (Denburg et al., 2006; Wood et al., 2005), while others propose the opposite, indicating that older adults might be relatively more sensitive to negative than positive feedback (Eppinger et al., 2013; Hämmerer et al., 2011; Simon et al., 2010a). If there is a shift toward negative-feedback sensitivity, it likely occurs later in old age (Frank and Kong, 2008; Simon et al., 2010a). However, some studies find no evidence for age differences in valence effects during learning (Lighthall et al., 2013; Samanez-Larkin et al., 2007, 2014). Across reward-learning tasks, the average effect reported is a main effect of age, without an age by valence interaction (Eppinger et al., 2011). This aligns with a meta-analysis demonstrating a lack of consistent age differences in valence effects on decision tasks not dependent on learning (Mata et al., 2011), despite limited findings and theories suggesting an age-related valence difference (Depping and Freund, 2011).

It is thus possible that age differences in reward-learning and decision-making tasks might not be specifically attributed to the differential processing of gains or losses but rather other effects such as individual differences including past stress experiences, accumulated over an

individual's lifetime. For example, as discussed in [Chapter 2](#), acute stress has been shown to induce dopamine release in the mesolimbic system and influence dopamine-dependent learning behaviour ([Abercrombie et al., 1989](#); [Anstrom & Woodward, 2005](#); [Imperato et al., 1991](#); [Cavanagh et al., 2011](#); [Lighthall et al., 2013](#); [Mather & Lighthall, 2012](#)). Limited evidence suggests that the effects of stressful experiences on dopamine-dependent learning are similar in both younger and older adults ([Lighthall et al., 2013](#)). Neuroimaging studies investigating responses to primary reinforcers yield mixed results, with some indicating an age-related increase in neural responses to aversive experiences (e.g., taste) and others showing an age-related reduction (e.g., pain) ([Jacobson et al., 2010](#); [Tseng et al., 2013](#)). As such, given individual differences associated with past experiences with acute and chronic stress, which, as discussed earlier, impact dopaminergic neurons in different ways, it is uncertain of how age modulates the impact of stress on decision-making performance under threat ([Starcke and Brand, 2012](#)).

### **3.2 Biological sex differences**

Studies on biological sex differences in stress reactivity and decision-making have shown mixed and disparate results. A review by [Starcke and Brand \(2012\)](#) notes differences in neural and endocrine stress reactivity, behaviour, and the neurobiology of decision-making amongst men and women. For example, across the lifespan, there is evidence showing that males consistently score higher than females in self-rated reward sensitivity, as indicated by questionnaires ([Cardoso Melo et al., 2023](#)). Both genders show an exponential decline in such scores with age; however, males experience a nonlinear peak in self-rated reward sensitivity between 18 to 20 years, followed by a decline aligning with the rate observed in females beyond this age range ([Cardoso Melo et al., 2023](#)). This increase in reward sensitivity in men during young adulthood ([Pagliaccio et al., 2016](#); [Schreuders et al., 2018](#); [Urosevic et al., 2012](#);

Windsor et al., 2012) may be driven by the peak in testosterone level during that period (Harden et al., 2018).

Both behavioural and neuroimaging studies have also shown higher reward sensitivity in males than females (Eneva et al., 2017; Georgiou et al., 2018; Soutschek et al., 2017), however, not all studies support this (Colder & O'Connor, 2004; Scheres & Sanfey, 2006). For example, women show higher neural and physiological responses than men to social reward (Borland et al., 2019) and men show higher responses than women to monetary reward (Dhingra et al., 2021; Warthen et al., 2020). Similarly, striatal responses to reward differed between males and females, with males showing higher ventral striatum response only to monetary reward and females to both monetary and social rewards (Spreckelmeyer et al., 2009).

In a reward go/no-go task, males showed higher physiological arousal relative to females, as reflected in skin conductance response (SCR), in response to “go” action, and the SCR predicted go success rate in males but not females (Le et al., 2019). In contrast, women were better than men at learning from positive (but not negative) feedback in a probabilistic selection task (Evans & Hampson, 2015). In another study analysing the Human Connectome Project data, women showed more suppression of the default mode circuit and higher activation of the dorsal attention circuit during exposure to both reward and punishment, suggesting enhanced saliency of both reward and punishment in women (Dumais et al., 2018).

In line with this, women also exhibit greater sensitivity to punishment, as evidenced by studies on delay gratification, gambling tasks, and reinforcement learning which consistently demonstrate these differences (Silverman, 2003; Byrnes, Miller & Schafer, 1999; Evans and Hampson, 2015; Ding et al., 2017). For example, sensitivity to Punishment (SP) and Sensitivity to Reward (SR) scores reveal sex differences, with women scoring higher in SP and men scoring higher in SR (Dhingra, 2021). Men exhibit increased neural responses in

specific brain regions to monetary rewards, while women show more pronounced modulation by SP in response to wins (Dhingra, 2021).

Women, in particular, are more likely to show risk-avoiding responses, whereas men are more likely to show risk-seeking responses (Taylor et al., 2000). The findings suggest that, under threatening conditions, men might make riskier decisions compared to women. As such, they might perform more exploration of choices than necessary. Such behaviour could either be adaptive or detrimental. For example, risk taking behaviour is adaptive in the case of outcome contingency reversals, where choices need to be changed. However, in circumstances where action-outcome contingencies do not change, it might be more adaptive to make choices with known positive outcomes. Furthermore, in decisions that were made under uncertainty, the right prefrontal areas were observed to be more strongly involved in men than in women (Bolla et al., 2004; Tranel et al., 2005). This finding might be relevant to decision-making under stressful conditions, since previous animal (Stalnaker et al., 2009) and human studies (Lueken et al., 2009) showed right prefrontal areas to be more sensitive to cortisol (Starcke and Brand, 2012). There is also evidence that individuals with stronger activation in the right prefrontal cortex exhibit better self-control (Knock and Fehr, 2007). Given these findings, it is possible that men might have the ability to better control their decision-making performance under threat, though direct evidence for this is lacking.

Another factor that may account for differences in decision-making under threat is variation in hormone release, which may interact with the dopaminergic system. Of particular relevance, males and females differ with respect to reactivity of the HPA axis, including levels of adrenocorticotrophic hormone (ACTH), glucocorticoid and cortisol release in response to stressors. For example, Stroud et al. (2002) uncovered distinct patterns in cortisol and Adrenocorticotrophic Hormone (ACTH) responses during achievement and social rejection

challenges. Men exhibited significantly greater cortisol responses to achievement tasks, while women displayed heightened cortisol responses to social rejection challenges.

In the realm of cognitive challenges, [Bale and Epperson \(2015\)](#) noted that older women exhibited an accentuated cortisol response compared to both age-matched men and younger individuals. [Seeman et al. \(2001\)](#) further demonstrated that among younger adults, men displayed a greater percentage increase in cortisol in response to cognitive challenges, whereas this pattern reversed among older adults, with women exhibiting greater increases.

Interestingly, pharmacological studies, as discussed by [Bangasser and Valentino \(2014\)](#), indicated inconsistent directions in cortisol level differences following stress between men and women. However, women, both healthy and depressed, displayed a more robust hormonal response to corticotropin-releasing factor (CRF) following dexamethasone pre-treatment compared to men. [Otte et al. \(2005\)](#) highlighted that aging increased cortisol responses to challenges, with this effect being almost three-fold stronger in women than in men.

[Kirschbaum et al. \(1999\)](#) delved into pharmacological and psychological stress, revealing that men exhibited higher ACTH responses to the Trier Social Stress Test (TSST) compared to women using oral contraception and those in different menstrual phases. Salivary cortisol responses varied based on hormonal status, with men showing similarities to women in the luteal phase, but differences compared to those in the follicular phase or using oral contraception.

Psychological stress studies consistently indicated that men respond to psychological stress with greater increases in cortisol compared to women ([Kudielka and Kirschbaum, 2005](#)). [Wang et al. \(2007\)](#) explored neural responses, noting that prefrontal activity in males correlated with salivary cortisol, while females exhibited a lower degree of correlation between limbic activation and cortisol.



Examining psychosocial stress via the Trier Social Stress Test (TSST), [Kumsta et al. \(2007a, 2007b\)](#) found that women with higher corticosteroid-binding globulin (CBG) levels showed reduced ACTH and salivary cortisol responses to stress but increased total cortisol levels. In contrast, men displayed greater ACTH increases, and higher CBG levels were linked to elevated ACTH responses during the TSST. [Rohleder et al. \(2001\)](#) added to this understanding by revealing marked increases in glucocorticoid sensitivity in men one hour after psychosocial stress, while women experienced a significant decrease.

Men under achievement and psychological stressors generally exhibit larger cortisol responses to stress, suggesting increased stress sensitivity ([Stroud et al., 2002](#); [Kudielka et al., 2007b](#); [Kudielka and Kirschbaum, 2005](#); [Wang et al., 2007](#)). In contrast, cortisol responses in women are smaller than in men during mental stress ([Lovallo et al., 2006](#)). As such, women might display a lower sensitivity to reward and punishment. This reduced sensitivity might lead to more stable DA dynamics within the indirect pathway, enabling better inhibition of unwanted actions. Smaller cortisol responses to stressors, may exhibit a more risk-averse decision-making pattern. The stable DA dynamics within the indirect pathway could lead to a cautious approach, avoiding risky behaviours and favouring risk-averse choices under threat. Women's hormonal responses to threat may result in a slower adaptation to threat-related cues. Their stable DA dynamics within the indirect pathway might lead to a more persistent decision-making strategy, potentially making them more cautious in the face of potential threats.

The sex differences observed in HPA axis responses to stress may be due to sexual dimorphisms in brain structure and function ([Patchev et al., 1995](#); [Rhodes and Rubin, 1999](#); [Cahill et al., 2001](#); [Killgore and Yurgelun-Todd, 2001](#); [Shors et al., 2001](#); [Wang et al., 2007](#)). Beside the impact of circulating corticosteroid binding globulin (CBG) levels ([Kirschbaum et al., 1999](#); [Kumsta et al., 2007a](#)), further prime candidates, for explaining such observations are

differences in the secretion of central arginine vasopressin (AVP) levels, or circulating gonadal steroids, with their complex effects on glucocorticoid and mineralocorticoid receptor regulation and functioning across men and women (for reviews and a meta-analysis see (Kudielka and Kirschbaum, 2005; Otte et al., 2005; Kajantie and Phillips, 2006; Kudielka et al., 2007b).

Previous studies suggest that there is a biological sex difference in glucocorticoid feedback (Kudielka and Kirschbaum, 2005; Goel et al., 2014; Zorn et al., 2017) and in glucocorticoid action. For example, Duma et al. (2010) showed profound sex differences in genome wide transcriptional response to dexamethasone in the liver of rat models. Glucocorticoid responsive genes were over represented in male versus female rats, suggesting that male rats are more susceptible to the anti-inflammatory actions of dexamethasone. In fact, according to Cidlowski, (2010), such profound sexual dimorphism in glucocorticoid action is a common phenomenon, and is also observed in the rodent brain (Duma et al., 2010). Such variations may be attributed to genetic differences and organisational actions of sex hormones. Genetic differences manifest in terms of differences in brain receptors for the sex hormone oestrogen and progesterone, which may modulate stress reactivity and also decision-making.

Sex differences in the responses of the mesocorticolimbic system to stress and stress hormones, in particular glucocorticoids, during adulthood or development, represents a mechanism which may contribute to sex biases in common DA-dependent-associated disorders (Gillies et al., 2014), as well as deficits in decision-making (Georgiou et al., 2018), risk assessment, and resilience (Wellman et al., 2018).

Wu<sup>st</sup> and colleagues (Wu<sup>st</sup> et al., 2004b) investigated for the first time whether variants of the glucocorticoid or mineralocorticoid receptor gene might contribute to the large inter-individual variability of HPA axis stress reactivity and they documented a sex-specific association between different glucocorticoid gene polymorphisms and salivary cortisol

responses to acute psychosocial stress (Wüst et al., 2004b; DeRijk et al., 2006; Kumsta et al., 2007b). Such findings may explain observations from previous studies which have shown a larger salivary cortisol response in healthy adult men compared to women following short-term laboratory stress (Stephens et al., 1996; Nicolson et al., 1997; Earle et al., 1999; Seeman et al., 2001; Lovallo et al., 2006; Kudielka, Hellhammer and Wüst, 2009). Salivary cortisol increases in men are up to twice as high as in women. The typical mean response magnitude in men ranges from 200 to 400% increase from baseline, whereas in women 50–150% changes are usually found. Moreover, in men the sole anticipation of an upcoming psychosocial stress task led to a significant saliva cortisol response even when they were not actually confronted with the stressor. A similar anticipatory endocrine response was absent in women (Kirschbaum et al., 1992b). Such sex differences in adrenocortical responsivity have also been observed in more than a dozen studies (for reviews and meta-analysis see Kudielka and Kirschbaum, 2005; Otte et al., 2005; Kajantie and Phillips, 2006; Kudielka et al., 2007b).

Furthermore, different stress protocols may cause stressor-specific salivary cortisol responses that might differ between men and women. For example, Stroud et al. (2002) reported that men, but not women, had significant saliva cortisol increases after confrontation with an achievement challenge (mathematical and verbal tasks), whereas women showed significant salivary cortisol responses to a social rejection challenge.

Finally, stress responses in women are sensitive to their menstrual cycle. Women in the luteal phase had saliva cortisol stress responses comparable to those of men whereas women in the follicular phase or women taking oral contraceptives showed significantly lower salivary cortisol responses (Kirschbaum et al., 1999). Other studies replicated the findings of comparably high salivary cortisol stress responses in men and women during the luteal phase (Rohleder et al., 2001; Wolf et al., 2001) as well as higher salivary cortisol responses in women during the luteal phase versus women taking oral contraceptives (Rohleder et al., 2003). Note

that, such results underline the importance of strictly distinguishing between the total cortisol secretion and the levels of bioavailable free cortisol, as can be measured in saliva (Kudielka et al., 2009).

Taken together, differences in sex hormone receptors, afferent connectivity and sexual dimorphic function of the brain, along with glucocorticoid actions and differences in HPA axis reactivity, might account for the differences in behaviour and psychological states of stress and threat appraisals that have been observed between men and women. For example, the preferred coping strategy of males is the well-known ‘fight-or-flight’ response in attempts to gain control. In contrast, females rely on a more passive strategy that can be characterized by ‘tend-and-befriend’ (Taylor et al., 2000). Such findings warrant further research into any sexual dimorphic functions in brain regions associated with decision-making during stress, and suggest that sex differences might be a mediator of the relationship between acute stress and decision-making performance.

### 3.3 Personality

Individual differences in personality traits might represent an internal predisposition to react negatively toward stress and highlight greater risks to developing psychiatric illnesses (Bolger and Schilling, 1991; De Jong et al., 1999; Kendler et al., 2004; Larsen and Ketelaar, 1991; Weinstock and Whisman, 2006). Personality differences affect the stress experience, and also influence how people cope with stress (Bolger and Schilling, 1991). Such differences play an important role in identifying, responding, and approaching stressful events (Dumitru and Cozman, 2012). Vulnerability to stress is thus based on a person's appraisal and response preferences to stressful situations, which are influenced by personality types (Kaur, Chodagiri and Reddi, 2013).

Types of personality can have powerful traits, which, over time, facilitate resilience to stress and psychological support against the toughest of life events (Dumitru and Cozman, 2012). Such personality traits are also important as they highlight predispositions for mental disorders (Huh et al., 2013; Na et al., 2011) and non-optimal behaviours. For example, individuals with a higher score on Openness to Experience are more likely to engage in unconventional behaviours (Suridjan et al., 2012). Other studies have also shown that personality traits may highlight vulnerabilities to environmental stressors (Cussen and Mench, 2015; Cockrem, 2007).

The construct of personality has also been found to be associated with physiological traits. As such, personality has been proposed as a potential moderator (Roger and Najarian, 1998) that could influence decision-making performance under threat. For example, the ventral striatal dopaminergic stress response has been associated with personality traits (Suridjan et al., 2012) which consequently could impact the encoding of RPE in guiding decisions towards expected outcomes.

The “big five” or “five-factor” model is an accepted construct that describes the diversity of personality across five dimensions: Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness. Within the five-factor model of personality, various correlations between personality traits and physiological responses to stress have been reported.

Table 1 summarises findings from a previous review by [Soliemanifar, Soleymanifar and Afrisham, 2018](#) which shows how physiological stress measures are related to several personality traits as described by the five-factor model of personality.

Table 1 Literature findings regarding personality and the stress response

Reference	General Findings	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
<b>Russell (2017)</b>	High neuroticism was associated with higher levels of cortisol.	+ Cortisol				
<b>Ouanes et al. (2017)</b>	High extraversion was negatively associated with cortisol area under the curve. High openness was associated with higher cortisol. No correlation was found between neuroticism and cortisol levels.		- Cortisol	+ Cortisol		
<b>Sadegh-Nejadi et al. (2017)</b>	A positive correlation was found between neuroticism and salivary cortisol. Openness has a negative correlation with salivary cortisol response.	+ Cortisol		- Cortisol		
<b>Evans et al. (2016)</b>	Higher levels of extraversion showed lower cortisol reactivity. Higher level of neuroticism showed higher cortisol pre-ejection period reactivity.	+ Cortisol	- Cortisol			
<b>Afrisham et al. (2016)</b>	Openness was positively correlated with salivary testosterone.	- IgA	- Testosterone			

Reference	General Findings	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
	A negative correlation was found between extraversion and salivary testosterone. A negative correlation was found between neuroticism and salivary (immunoglobulin A) IgA. Openness was positively correlated with salivary IgA.			+ Testosterone  + IgA		
<b>Parent-Lamarche et al. (2015)</b>	Agreeableness was associated with lower cortisol levels at awakening.				- Cortisol	
<b>Bogg et al. (2015)</b>	High conscientiousness is associated with reductions in diurnal cortisol concentrations.					- Cortisol
<b>Laceulle et al. (2015)</b>	Neuroticism, extraversion and conscientiousness were related to low level of basal cortisol.	- Cortisol	- Cortisol			- Cortisol
<b>Afrisham et al. (2015)</b>	Neuroticism and agreeableness were positively correlated with salivary alpha-amylase.	+ sAA			+ sAA	
<b>Chu et al. (2015)</b>	Interaction of stressors (health, family, social, work) and both agreeableness and extraversion negatively predict physiological stress response.		- Cortisol		- Cortisol	



Reference	General Findings	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
<b>Bibbey et al. (2013)</b>	Higher neuroticism was related to lower cortisol and cardiovascular stress reactions. Low agreeableness and low openness had shown lower cortisol and cardiac reactions to stress.	- Cortisol - Heart rate			+ Cortisol + Heart rate	
<b>Agrigoroaei et al. (2011)</b>	Individuals with higher levels of conscientiousness showed lower cortisol reactivity. Neuroticism, agreeableness and extraversion were positively related to greater cortisol reactivity.	+ Cortisol	+ Cortisol		+ Cortisol	- Cortisol
<b>van Santen et al. (2011)</b>	Individuals with higher levels of extraversion have tended to a lower cortisol awakening response. No significant associations were found for neuroticism, conscientiousness, openness, agreeableness.		- Cortisol			
<b>Inukai et al. (2010)</b>	Positive correlation between neuroticism and Salivary Alpha-Amylase (sAA) were observed. Agreeableness was positively correlated with sAA.	+ sAA	- sAA	- sAA	- sAA	

Reference	General Findings	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
	Extraversion, agreeableness, openness were negatively related to sAA after controlling for age.					
<b>Nater et al. (2010)</b>	Neuroticism was positively associated with cortisol levels during all periods of measurement. Individuals with high levels of conscientiousness showed reductions in diurnal cortisol concentrations.	+ Cortisol				- Cortisol
<b>Hauner et al (2008)</b>	Higher introversion was associated with a lower cortisol awakening response. In interaction with gender, higher levels of introversion among males were associated with the increased cortisol levels at the time of wakeup. A flatter cortisol rhythm was observed across the waking day among male participants with higher Neuroticism.	- Cortisol (males)	+ Cortisol  - Cortisol (males)			
<b>Oswald et al. (2006)</b>	Low levels of openness were associated with lower cortisol responses		+ Cortisol (males)	+ Cortisol		

Reference	General Findings	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
	In the interaction of gender, the low cortisol responses were associated with higher neuroticism in women and with lower extraversion in men.	- Cortisol  (females)				
<b>Zobel et al. (2004)</b>	Participants with high neuroticism had higher levels of cortisol.	+ Cortisol				
<b>Schwebel et al. (1999)</b>	Blood pressure reactivity to hand grip task reported in highly neurotic individuals.	+ Systolic  Blood Pressure				
<b>Miller et al. (1999)</b>	Low agreeableness has showed higher levels of systolic blood pressure, diastolic blood pressure, and urinary epinephrine. Low extroversion was related to higher levels of epinephrine, blood pressure, norepinephrine, and natural killer cell cytotoxicity. Neuroticism was not associated with physiological outcomes.		- Blood Pressure  - Norepinephrine  - Epinephrine		- Systolic Blood Pressure  - Diastolic Blood Pressure  - Epinephrine	
<b>Vickers et al. (1991)</b>	Agreeableness was associated to higher cortisol. Conscientiousness was related to lower cortisol.				+ Cortisol	- Cortisol

Among the five personality traits, neuroticism seems to exhibit the strongest relationship with stress responses. Neuroticism, which appears to account for most of the variances in several other extensively studied individual difference variables in social psychology (such as self-esteem, locus of control, and generalized self-efficacy; Judge et al., 2002), has been associated with exaggerated appraisals of stimuli as stressful (Bishop, 2008). A positive correlation between neuroticism with level of psychological stress has been observed (Afshar et al., 2015; Cabarkapa, Korica and Rodjenkov, 2011; Vollrath and Torgersen, 2000). In line with this, low levels of neuroticism and high conscientiousness have shown a favourable personality profile to the coping with stress (Vollrath and Torgersen, 2000). Similarly, higher levels of neuroticism were associated with lower diastolic blood pressure and total peripheral resistance index (TPRI) reactivity during mental arithmetic tasks, but higher TPRI reactivity during anger recall questionnaires (Jonassaint et al., 2009). Furthermore, the three facets, anxiety, angry hostility and depression, which are the strongest loadings on the neuroticism factor, have shown negative associations with general cardiovascular health (e.g., Smith and MacKenzie, 2006; Suls and Bunde, 2005; Hughes et al., 2011).

Comparisons of reactivity curves suggest blunted initial stress responses among persons with high neuroticism, followed by enhanced stress responses. In contrast, persons with low neuroticism levels show higher initial responses followed by greater decreases in their stress responses (Hughes et al., 2011). Neuroticism might be associated with blunted initial stress responding because participants high in neuroticism might lack the motivation to fully engage with task demands (Dobson, 2000; Hughes et al., 2011). Neuroticism and other anxiety related traits are also associated with poorer cardiovascular recovery to laboratory stressors (Chida & Hamer, 2008; Contrada and Baum, 2010).

In-vivo positron emission tomography (PET) and single photon emission computed tomography (SPECT) studies reported associations between the D2 receptors availability in

the striatum and different personality traits such as Detachment (Farde et al., 1997; Laakso et al., 2003; Wacker et al., 2005), Extraversion (Depue and Collins, 1999), Novelty-/Sensation-Seeking (Kaasinen et al., 2004; Suhara et al., 2001; Zald et al., 2008) and Neuroticism (Kestler et al., 2000; Lee et al., 2005; Wacker et al., 2005). Other studies have also shown personality traits like irritability detachment, psychasthenia and somatic anxiety, extraversion, and high scores on the lie scale to be associated with reactivity to stress (Kaur, Chodagiri and Reddi, 2013; Flaa et al., 2007; Desa et al., 2014).

Furthermore, there is some evidence that individual differences in personality traits are associated with DA response to psychosocial stress (Suridjan et al., 2012). For example, a previous study has found that Neuroticism-related traits, such as Angry-Hostility, Vulnerability and Depression, were related to a blunted DA response to a psychosocial stress in the D2-rich and D2/3-mixed regions; while the trait related to Openness to Experience was associated with a blunted DA response in the D3-rich region (Suridjan et al., 2012). Although there are other PET studies reporting the associations between the dopamine response to stress and several variables (Pruessner et al., 2004; Soliman et al., 2008), this was the first study that examined the inter-individual variability of the brain dopamine stress response in the context of personality variations in healthy individuals (Suridjan et al., 2012; Soliemanifar, Soleymanifar and Afrisham, 2018). Furthermore, an association study between the dopamine D3 receptor gene (DRD3) variants and personality traits revealed links between DRD3 and traits related to the Novelty-Seeking and Openness to Experience (Jonsson et al., 2003).

Biological and cognitive responses to stress suggest that physiological mechanisms are influenced by personality-related mediator and/or moderating factors. For example, hope (Snyder et al., 1991), optimism (Scheier and Carver, 1987), hardiness (Kobasa, 1979), constructive thinking (Epstein and Meier, 1989), learned resourcefulness (Rosenbaum, 1989), self-efficacy (Bandura, 1982), and sense of coherence (Antonovsky, 1993) have been

identified as personality-related traits that predict positive appraisal, resilience, effective coping, or even growth in the stress process (Soliemanifar, Soleymanifar and Afrisham, 2018). Psychological factors, such as low self-esteem and low locus of control, are also thought to relate to increases in the reactivity of the HPA-axis (Pruessner et al., 2005). Self-esteem has been found to be negatively associated with cortisol and adrenocorticotrophic hormone responses (Seeman et al., 1995). Therefore, individuals with low self-esteem and low locus of control may be more vulnerable to stress and have difficulties regulating their stress response, which may then impact decision-making performance under threat.

In summary, elevated blood pressure, heart reactivity, and cortisol responses are physiological responses to stress that differ with personality. These responses can affect the neural activity in regions involved in decision-making, including the VTA. Higher blood pressure and heart reactivity might lead to altered dopamine release and influence reward processing, potentially impacting decision-making strategies. Also, stress triggers the release of cortisol, which also varies with personality traits. Cortisol can modulate neural activity, including activity within the VTA, through its interaction with glucocorticoid receptors. Elevated cortisol levels have been associated with increases in dopamine signalling which may improve reward learning, but impair avoidance learning (Lighthall et al., 2013), consequently affecting decision-making. In contrast, moderate levels of acute stress-related cortisol may facilitate learning (Abercrombie et al., 2003; Luksys & Sandi, 2011; Wolf, 2009) and consequently improve decision-making that depends on learning from positive or negative RPE (Mather & Lighthall, 2012).

Based on the observations above, it seems that humans might have a biological basis for personality that influences thinking, behaviour and how to interact with their environment (Soliemanifar, Soleymanifar and Afrisham, 2018). Numerous researchers have stated that the structure of the five-factor model is a genetically-based human universal personality that is

independent of language and other cultural differences (Bouchard and Loehlin, 2001; Wiggins and Trapnell, 1997; Yamagata et al., 2006; McCrae and Costa, 1997). Nevertheless, some studies have suggested that extraversion and agreeableness may be more dependent on cultural context (Rolland, 2002). Also, based on the five-factor model, some researchers have presented the evolutionary approach which states that personality diversity may not be constant among human communities (Gurven et al., 2013; Buss, 2009). The question whether a biological basis personality is derived from heredity (nature) or learning (nurture) or an interaction between them requires further research. Such research into the origins of personality could help us understand how personality might moderate and/or mediate the relationship between acute stress and decision-making performance under threat.

## **Chapter 4: Relationships Between Age, Gender, Years of Education, History of Stress and Decision-making Performance Under Threat – Online Experiment**

### **4.1 Research questions**

As reviewed in the first three chapters of this thesis, chronic and acute stress may alter dopaminergic basal ganglia circuits, which might influence RPE; yet, evidence for specific decision-making changes (e.g., altered learning from rewards vs punishment) in humans is very limited. We therefore present a human decision-making experiment in which we systematically analyse individual difference factors that may impact decision-making performance under threat. Specifically, we focus on investigating the relationships between age, gender, years of education, previous experiences with acute and chronic stressors and decision-making performance in both safe and threat conditions. Our primary aims are to answer the following questions:

- 1) Will participants perform better or worse in a decision-making task under threatening or safe conditions?
- 2) Will participants with past acute or chronic stress experiences perform better or worse when making decisions under threat compared to safe conditions?

The decision-making task we have used consists of learning by trial and error which of two alternative choices (choosing between two different coloured doors) is more likely to lead to a win rather than a loss. Unbeknownst to the participants, the likelihood of winning associated with some of the doors is reversed after blocks of trials, so participants have to adapt to these changing contingencies. We quantify decision-making performance in a way that allows us to disentangle the potential influences of the direct and indirect pathways on performance, i.e., learning from positive versus negative RPE.



In the task, win-stay choices are those when a participant consistently repeats the same choice that leads to a winning outcome. Such decisions are most likely to engage the direct pathway based on learning from a positive reward prediction error (Ikemoto, 2007; Beier et al., 2015). In contrast, lose-switch choices happen when a participant correctly switches to an alternative choice after losing. Such decisions are most likely to engage the indirect pathway based on learning from a negative reward prediction error (Ikemoto, 2007; Beier et al., 2015).

In order to study decision-making performance under safe and threat conditions, we implement a mild threat manipulation whereby participants experience a series of safe and threat trials. On threat trials participants learn that they might lose a large amount of their accumulated winnings, whereas this does not happen on safe trials.

It is expected that participants with a low history of either acute or chronic stressors might display higher win-stay performance under threatening conditions compared to safe conditions. This is because a threatening condition might result in a higher increase in DA baseline levels (Bromberg-Martin, 2010), which might improve attention and arousal. Also, any increases in dopamine bursts experienced in the direct pathway should consequently facilitate learning from positive reward prediction errors (Ikemoto, 2007; Beier et al., 2015). In contrast, based on the current state of the literature, it is unclear whether such participants might display increased or decreased lose-switch performance during safe conditions. Indeed, there are two possible outcomes. First, the presence of a threat might make an individual biased (Maier, Makwana and Hare, 2015) and inflexible to change their choice when encountering a winning door reversal, as they attempt to secure their winning choice in anticipation of threats that are not actually there. Alternatively, the flexibility to explore alternatives might be higher during safe conditions compared to threat conditions (Porcelli and Delgado, 2017), hence increasing the chance

of being flexible in behaviour to correctly try different door alternatives after experiencing winning door outcome reversals.

Furthermore, during threat conditions, such individuals might be more impulsive or have varying levels of risk aversion (Porcelli and Delgado, 2009), whether they stay with a winning choice or not, which consequently might lead to more inconsistent and impulsive decisions. Such impulsive or inconsistent behaviour, however, might be beneficial for lose-switch performance during threat conditions. In contrast, such participants might, by default, be in an alert mode (Bishop, 2008), which would make them more likely to stay with their winning choices during safe conditions. Such behaviour would lead to lower lose-switch performance, as they become reluctant to switch choices after winning reversals that occur during safe conditions. Alternatively, they might behave more consistently and carefully during a safe condition compared to the threat condition (LeDoux & Daw, 2018). Conversely, they might have higher lose-switch performance under threat conditions, because the threatening condition might increase the chances of them engaging in more explorative behaviour as they attempt to perform actions that might be perceived as actions with the potential of avoiding the threat (Wang, Jackson and Cai, 2016).

It is expected that participants with history of high acute and chronic stressors might display higher win-stay performance under safe conditions compared to threat conditions. Such individuals might have less sensitivity to dopamine bursts associated with positive prediction error. A reason for this might be due to negative changes in the VTA/SNc associated with past chronic stress experiences (Douma and de Kloet, 2020). An acute stress manipulation might exacerbate this loss of sensitivity to dopamine bursts rather than enhance it. As such, they may not learn equally well from positive prediction error in the threat condition compared to the safe condition. It could also be the case that

there may not be a change in win-stay or lose-switch performance between threat and safe conditions, given that dopamine changes in dips and bursts might be less dynamic overall, due to any negative changes in the VTA/SNc associated with history of high chronic stressors. In any case, we expect these individuals to not show the enhanced win-stay performance under threat that individuals with low acute and chronic stress are expected to show.

In addition to history of acute and chronic stress, individual differences in age, gender and years of education might also predict win-stay and lose-switch performance. As such, we explored linear regression models in order to test our predictions in win-stay and lose-switch performance. Note that, due to the limited literature on this topic, no predictions were made regarding win-stay and lose-switch performance for participants with previous experiences of low acute stressors and high chronic stressors, and those having past experiences with high acute stressors and low chronic stressors. As such, linear regression models that include interactions between acute and chronic stress experiences were also tested, since it is possible that a history of chronic stress might modulate the effect of acute stress.

## **4.2 Method**

### **4.2.1 Participants**

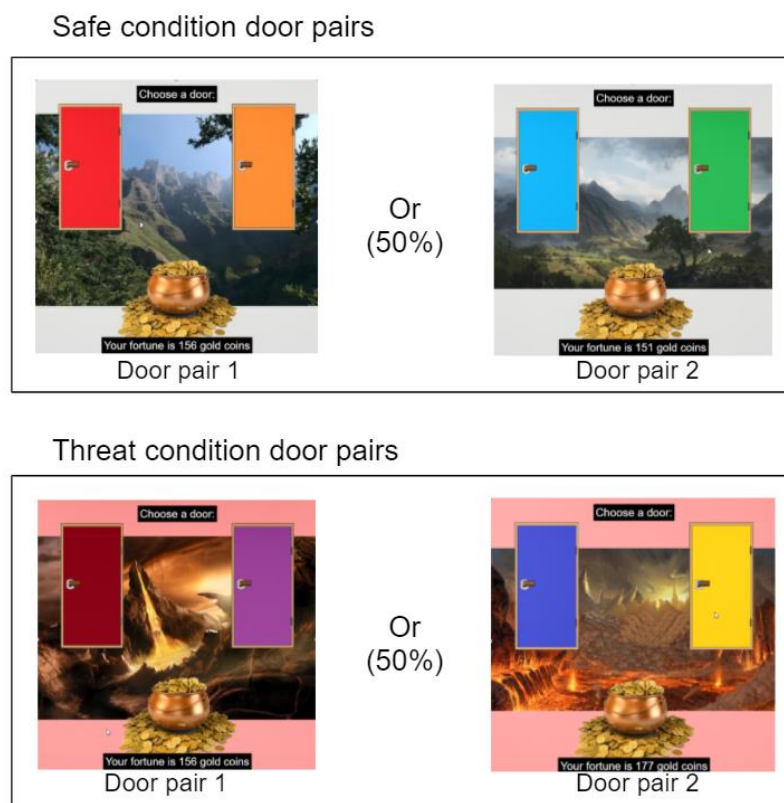
$N=109$  participants (53 females) were recruited from Amazon's Mechanical Turk crowdsourcing online platform (<https://www.mturk.com/>), with ages ranging between 18 and 71 years ( $M= 37.09$ ,  $SD= 10.9$  years). Eligibility requirements included being aged 18 years or over and being fluent in the English language.

With ethical approval from the Human Research Ethics Committee of the University of Adelaide (H-2021-124) and participant consent, measures related to

decision-making performance, demographics and individual differences in personality, impulsivity, and history of stress experiences were collected (see [Appendix 1](#)).

#### 4.2.2 Stimuli

In order to investigate the impact of a threat manipulation on decision-making, participants played the Dracoin Doors Game and then completed a series of questionnaires. In brief, four different pairs of doors were included in the game as shown below. Two pairs were presented during safe trials and the other two during threat trials.



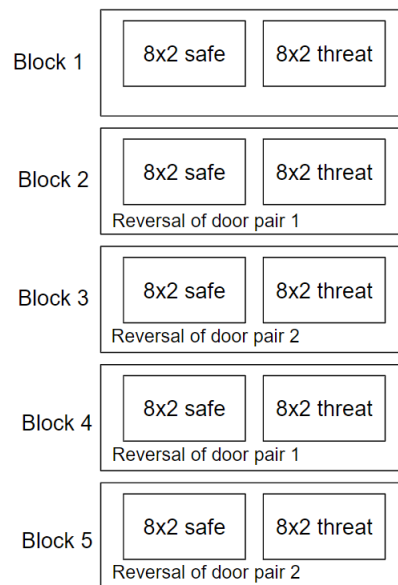
On each trial, participants were asked to select one of two doors, only one of which was rewarded. The participants' goal was to learn by trial and error which choices would help them accrue the highest monetary reward. Each pair had a unique background image and music, a pleasant and calm landscape and music for safe condition trials, and a chaotic, fiery, hostile landscape with suspenseful music, for threat condition trials. The doors were

distinguished by distinct arbitrary colours. A pot of gold symbolised the participant's accrued wealth and included a text label that provided feedback regarding accrued wealth.

The doors had a 50% chance of being displayed on the left or the right such that participants needed to base their decisions on the colour of the doors rather than their location. Selecting the correct coloured door was guaranteed to result in a fixed monetary reward. Selecting an incorrect coloured door resulted in no monetary reward. Participants used a pointing device (preferably a computer mouse) to move towards the desired door and click the door. The reward event represented winning 5 coins and consisted of an animation of a coin descending into their pot of gold.

The background images included in the safe condition provided a safety cue and those under a threat condition provided the cue that an unpredictable and uncontrollable threat event might occur. The threat was represented by an event that resulted in a proportion of accrued coins being stolen. When the threat event occurred, an animation and sound of a dragon were presented to resemble the dragon roaring and moving towards the pot of gold and stealing their coins. The threat occurred a total of 5 times during the task. It occurred once after the first trial of block 1, and then once again at an arbitrary interval occurring for a single instance of each door pair, and each left/right door position combination. This was an attempt to signal a threat condition, and to signal that the dragon event was not related to the colour or position of the doors. The loss due to a threat occurring was 30% of the total reward accrued. These threat events were unpredictable and not contingent on the participants' choices. In fact, they occurred during inter-trial intervals when participants were not making a choice. Participants did not know the probability of outcomes for each door, the probability of threat occurring, the loss factor incurred when a threat occurred and that door probabilities were reversed on consecutive blocks. They learnt these probabilities by trial and error, and their performance allowed us to determine how fast they learnt to

adjust their behaviour (for example, by repeating choices that were rewarded, or avoiding choices that were not). Participants were instructed to make a choice as fast as they could and to accrue as many gold coins as they could. The sequence of trials was organised into five sequential blocks, each consisting of 8 trials for each door pair 1 and 2, for each safe and threat condition, as shown below.



*Figure 4 Experiment trial sequence*

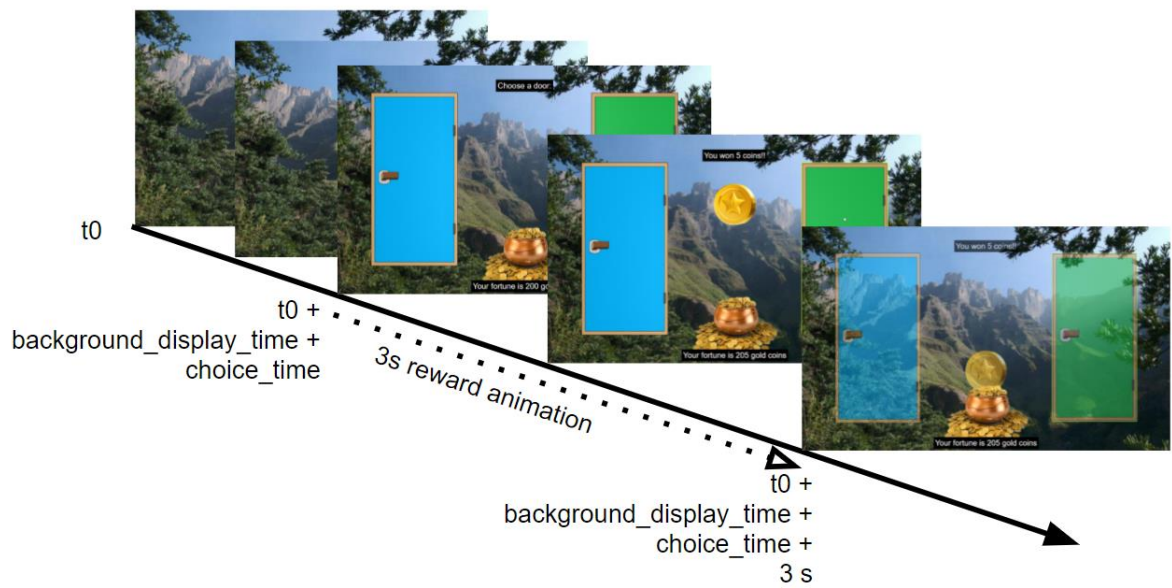
The trials were randomised to eliminate any type of false learning associations between the position or colours of doors and the choice outcomes (see [Appendix 2](#)). A random sequence of 8 trials randomly containing either pair 1 or 2, for each safe and threat conditions, was also counterbalanced to eliminate any type of ordering effects due to experiencing either a safe or threat trial sequence first (see [Appendix 2](#)). Therefore, each block consisted of a total of 32 trials, i.e. two sequences of 8 trials for each safe and threat condition.

Following completion of the first block, consecutive blocks included a contingency reversal for one door pair in both safe and threat conditions. That is, the door that was previously rewarded was no longer rewarded, and vice versa for the other choice.

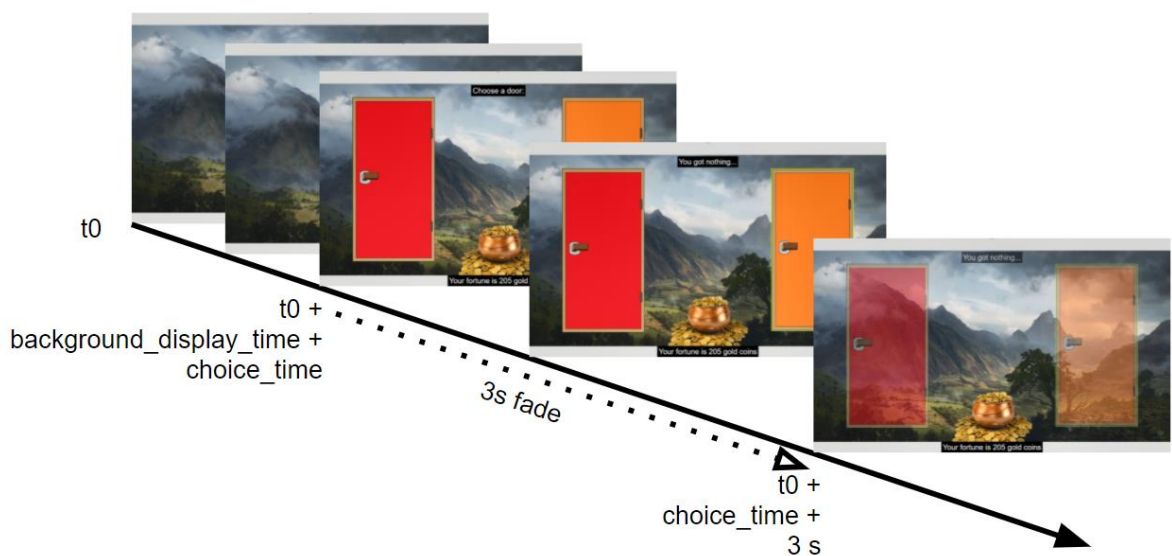
Contingency reversals provided more opportunities to assess the speed of learning and allowed us to study behavioural adaptation in a changing environment.

The different events that could be experienced during safe and threat conditions are displayed in Figure 5 below.

## Safe stimuli with reward

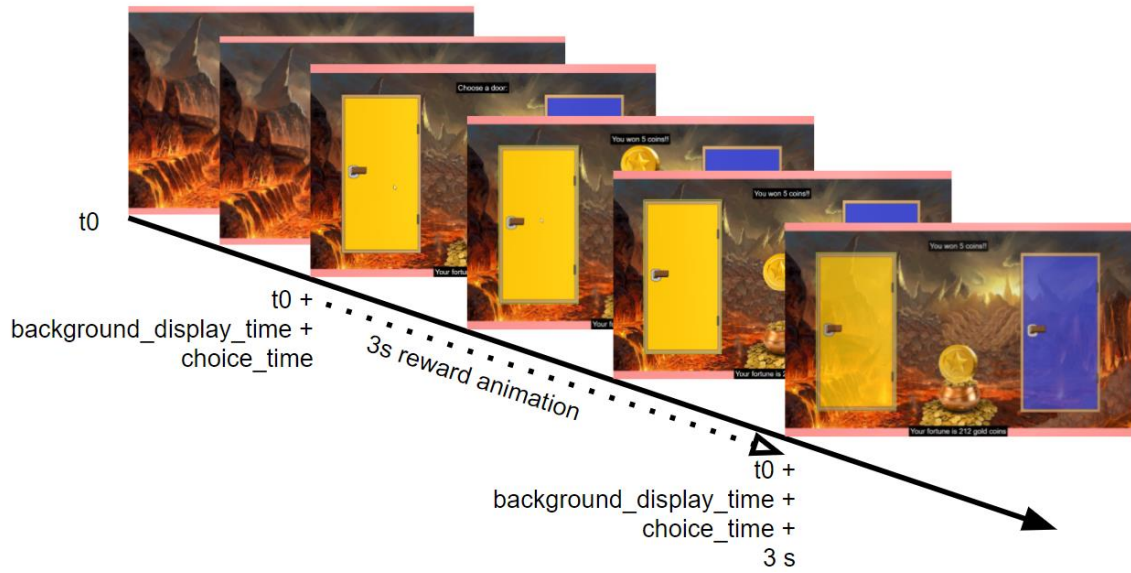


## Safe stimuli with no reward

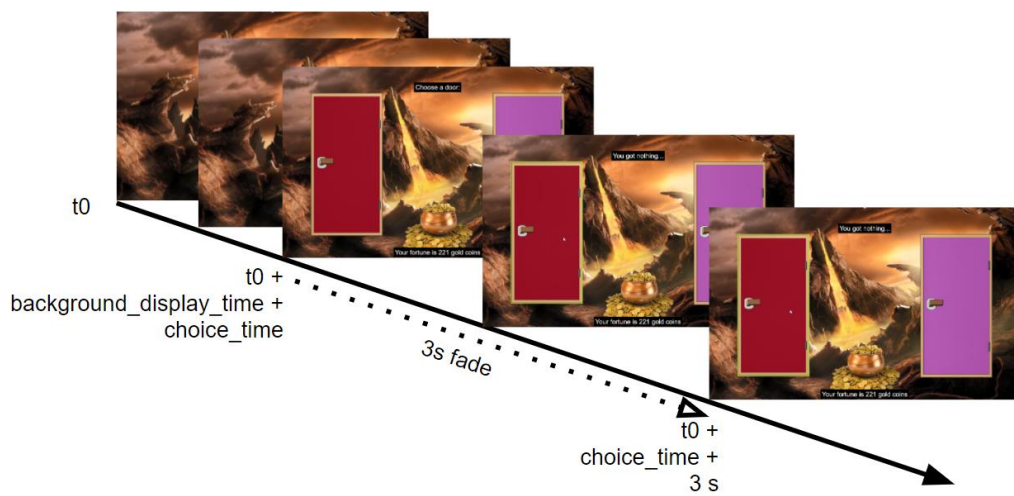




# Threat stimuli with reward

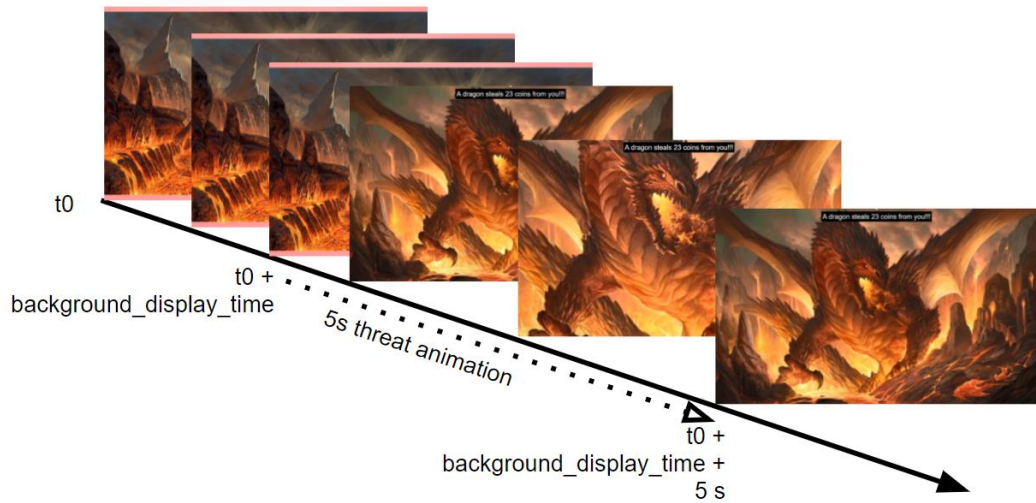


# Threat stimuli with no reward





# Threat occurrence



*Figure 5 From top to bottom are five sets of image snapshots representing animation sequences and timings that highlight the different events that could be experienced whilst playing the Dracoin Doors Game. Events include either reward or no reward events during safe and threat conditions, and a threat event occurring only during threat conditions.*

Upon completion of the Dracoin Doors Game described above, win-stay and lose-switch measures were collected. Win-stay and lose-switch are measures used to test Reward Prediction Error (RPE) dynamics in decision-making performance. These measures are often used in behavioural and cognitive psychology research to understand how individuals adapt their choices based on the outcomes of previous decisions. The measures were selected to assess the accuracy of learning from positive or negative outcomes, in line with testing reward prediction error dynamics within the direct and direct pathways of the basal ganglia.

### 4.2.3 Survey measures

To investigate how individual differences in age, gender, years of education and history of acute and chronic stressors impact decision-making performance under safe and threat conditions, the following measures were collected after the game (refer to [Appendix 1](#)). In addition to demographic variables, we administered surveys that reflect experiences with acute (recent) stress (DASS, PSS, PCL-5), as well as experiences with chronic stressors (LEC-5, MOSS-21).

#### a. Demographics

Physical and mental health, stress and well-being are related to demographic factors such as age, gender, education, employment, income, relationships and living conditions. For example, performance in cognitive tasks may vary with age and levels of education. Adequate income, living conditions and healthy relationships can reduce levels of stress. As such, the following demographic measures were collected: age, gender, education, employment status, household income, relationship status, country of birth, country of residence and living environment. Note, however, that we only analysed age, gender and education level.

#### b. Ten-Item Personality Inventory-(TIPI)

Longitudinal studies have been instrumental in showing how differences in the Big Five personality traits (openness, conscientiousness, extraversion, agreeableness and neuroticism) relate to mental health, mental disorders, job success and marriage satisfaction, all of which influence stress sensitivity ([Ozer and Benet-Martínez, 2006](#); [Roberts, 2007](#)). As explained in [Chapter 3](#), the trait of neuroticism positively correlates with levels of

psychological stress (Afshar et al., 2015), while low levels of neuroticism and high conscientiousness positively influences coping with stress (Vollrath and Torgersen, 2000). As such, personality might influence threat and stress sensitivity. The TIPI is a 10-item measure of the Big Five personality traits (Gosling, Rentfrow and Swann, 2003) and is proposed to assess the personality of participants. Note that an analysis of this data is presented in Chapter 6.

c. 4-Item Perceived Stress Scale (PSS),

The PSS is a commonly used instrument to measure the degree to which situations in life are appraised as stressful (Cohen et al., 1983; Cohen, Kessler and Gordon, 1997). The items were designed to assess how unpredictable, uncontrollable, and overloaded respondents find their lives (Cohen et al., 1983; Cohen, Kessler and Gordon, 1997). The scale also includes a number of direct queries about levels of experienced stress over the last month. As such, this measure is proposed to capture individual differences in threat and stress sensitivity.

d. 21-item Depression Anxiety and Stress (DASS-21),

The DASS-21 is designed to measure states of depression, anxiety and stress (Lovibond and Lovibond, 1996). The scales of the DASS have been shown to have high internal consistency and to yield meaningful discriminations (Lovibond and Lovibond, 1996). The stress scale is sensitive to levels of stress. As such, it is expected that items from the DASS-21 will highlight individual differences in decision-making performance associated with variances in levels of stress over the last week.

e. Post-traumatic stress disorder (PTSD) DSM-5 checklist (PCL-5)

Early life adversities are pervasive across social and cultural settings (Kessler et al., 2010). The Post Traumatic Stress Disorder (PTSD) checklist (PCL-5) is a 20-item self-report measure that assesses 20 symptoms of PTSD as per the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5) (Weathers et al, 2013). Specifically, the PCL-5 was used to assess levels of stress experienced over the last month as a result of previous traumatic life events. Such previous experiences might show different threat and stress sensitivities, which might be reflected in their decision-making performance.

f. Life Events Checklist for DSM-5 (LEC-5)

The LEC-5 was used to assess participants' history of stressful life events. Such experiences might have resulted in dopamine system changes which might reflect decision-making performance differences.

g. Medical Outcomes Study 20-Item Short Form Survey Instrument (MOSS-20),

As discussed earlier, previous experiences with distress or chronic stress can have a negative impact on physical and mental health. Poor physical and mental health are also stressors, which can in turn contribute to chronic stress. To understand how individual differences in mental and physical health stressors could impact decision-making performance, validated survey instruments can be used to assess an individual's mental and physical health. The Medical Outcomes Study 20-Item Short Form Survey Instrument (MOSS-20) has been reported to have adequate reliability and validity (Stewart, Hays and Ware, 1988). As such,

the MOSS-20 was used to assess the state of mental and physical health stressors of participants. The MOSS-20 provides additional history of chronic stress to that of any stress induced as a result of life events from the LEC-5. The MOSS-20 includes items that assess the following: physical functioning, physical limitations and capacities, mobility, and self-care, limitations in role functioning due to poor health, limitations in social activities due to health, general mental health across the following dimensions: anxiety, depression, loss of behavioural-emotional control and psychological well-being; and finally health and pain perception. Note that, items associated with mental health were not included in scores since other measures such as the PSS and DASS addressed measures of mental health.

#### 4.2.4 Procedure

The protocol was registered as a crowdsourcing task via [https://requester.mturk.com/signin\\_options](https://requester.mturk.com/signin_options). The Dracoin Doors Game (file Mturk\_study\_v57\_integrate.html from the attached Supplement Material) was uploaded to the Amazon Mechanical Turk Platform, with crowdsourcing task details as specified in [Appendix 1](#).

#### 4.3 Results

[Table 2](#) shows that scores on the acute stress measures (PSS-4, DASS-21 and PCL-5) positively correlated. As such, these scores were combined into a single factor using the principal components analysis R function ‘principal()’ (Revelle, 2022). The participants’ scores on the first component is henceforth referred to as the ‘acute stress factor’. The acute stress factor accounted for 79% of the variance in the DASS-21, PSS-4 and PCL-5 scores, and their respective loadings were 0.94, 0.80, and 0.91.

Participants also completed two questionnaires that we assumed would reflect experiences with chronic stress, the MOSS-21 and the LEC-5. The MOSS-21 includes items associated with depression, anxiety and stress. Therefore, these items were omitted from the total scoring, with the assumption that such factors are already covered by using the PSS-4, DASS-21 and PCL-5 scores. [Table 2](#) shows that the scores on the modified version of the MOSS-21 and the LEC-5 have a strong significant negative correlation (i.e., lower health scores on the MOSS-21 were associated with more adverse events reported in the LEC-5). As such, these scores were also combined into a single factor using the aforementioned ‘principal()’ function. Such scores are based on experiences likely to have persisted for longer than three months; as such, they are assumed to be chronic stressors, hence the combined factor is referred to as the ‘chronic stress factor’. The chronic stress factor accounted for 71% of the variance in the

modified MOSS-21 and LEC-5 scores, and their respective loadings were -0.84, and 0.84 (note that higher scores on the MOSS-21 indicate better health, hence fewer causes for chronic stress, thus its loading on the chronic stress factor is negative).

Before investigating individual differences in decision-making performance, we tested whether the acute stress manipulation had an effect on performance. Paired-sample *t*-tests showed that differences in mean win-stay ( $t(108) = 0.40, p = 0.69, CI_{95\%} \text{ Cohen's } d [-0.016, 0.025]$ ) and lose-switch performance ( $t(108) = -1.12, p = 0.27, CI_{95\%} \text{ Cohen's } d [-0.048, 0.013]$ ) between threat and safe conditions were not significant. As such, in addition to including linear regression models predicting the difference in win-stay and lose-switch between threat and safe conditions, we also included models predicting the average win-stay and lose-switch across both threat and safe conditions to test whether the chronic and acute stress self-report measures could predict differences in learning from rewards and punishment in general.

Table 2 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for scores on the PSS-4, depression, anxiety and stress of the DASS-21, DSM-5 PTSD checklist (PCL-5) with life events checklist (LEC-5), and modified MOSS-21

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6
1. PSS-4	6.77	3.02						
2. Depression	17.39	10.69	.58** [.44, .69]					
3. Anxiety	17.53	10.54	.65** [.52, .74]	.96** [.95, .97]				
4. Stress	16.86	10.74	.62** [.49, .73]	.96** [.94, .97]	.97** [.95, .98]			
5. PCL-5	34.40	21.65	.55** [.40, .67]	.85** [.79, .89]	.85** [.78, .89]	.84** [.78, .89]		
6. LEC-5	0.57	0.36	.32** [.14, .48]	.35** [.17, .50]	.32** [.14, .48]	.36** [.18, .51]	.47** [.30, .60]	
7. MOSS-21 (modified)	29.24	5.55	-.43** [-.57, -.26]	-.58** [-.69, -.44]	-.56** [-.68, -.41]	-.58** [-.70, -.44]	-.50** [-.63, -.35]	-.41** [-.56, -.24]

*Note.* *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .



Table 3 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for age, years of education, acute and chronic stress factors, average win-stay and lose-switch performance across both safe and threat conditions, and the differences in mean win-stay and lose-switch performance between threat and safe conditions.

Variable	<i>M</i>	<i>SD</i>	1	2	3	4
1. Age	37.09	10.90				
2. Years of education	15.30	2.31	.14 [-.05, .32]			
3. Acute stress factor	-0.00	1.00	-.37** [-.52, -.20]	-.09 [-.28, .10]		
4. Chronic stress factor	0.00	1.00	-.14 [-.32, .05]	.04 [-.15, .22]	.59** [.46, .70]	
5. Win-stay average	0.66	0.20	.26** [.08, .43]	.13 [-.06, .31]	-.48** [-.61, -.32]	-.32** [-.48, -.14]
6. Lose-switch average	0.60	0.17	.28** [.09, .44]	-.04 [-.23, .15]	-.42** [-.56, -.25]	-.29** [-.45, -.10]
7. Win-stay difference	0.00	0.11	.02 [-.17, .21]	-.05 [-.24, .14]	.06 [-.13, .24]	-.05 [-.23, .14]
8. Lose-switch difference	-0.02	0.16	-.06 [-.24, .13]	-.16 [-.34, .03]	.07 [-.12, .26]	-.00 [-.19, .19]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .

### 4.3.1 Age, gender, years of education, acute and chronic stress factors

Two regression models predicting the average win-stay performance across threat and safe conditions were tested (refer to Table 4). The first model included age, gender, years of education, the chronic stress factor and the acute stress factor as predictors of the average of win-stay performance across both safe and threat conditions. The second model included the same predictors as the first, however, it also included an interaction between the acute and chronic stress factors as an additional predictor.

The first model was significant ( $F(5, 103) = 7.48, p < 0.001$ ) and the predictors accounted for 26.6% of the variance, see Table 4. Only the acute stress factor significantly predicted lower win-stay scores. The second model was also significant ( $F(6, 102) = 6.27, p < 0.001$ ), and its predictors accounted for 26.9% of the variance in average win-stay across threat and safe conditions. The second model also showed a significant effect for the acute stress factor, however, the interaction was not significant.

Table 4 Linear regression models predicting average win-stay performance across safe and threat conditions

Average win-stay across safe and threat conditions	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.46	0.20 – 0.71	<b>0.001</b>	0.46	0.20 – 0.71	<b>0.001</b>
Age	0.00	-0.00 – 0.01	0.269	0.00	-0.00 – 0.01	0.289
Gender [female]	0.05	-0.02 – 0.12	0.126	0.05	-0.01 – 0.12	0.122
Years of education	0.01	-0.01 – 0.02	0.355	0.01	-0.01 – 0.02	0.321
Chronic stress factor	-0.02	-0.06 – 0.02	0.365	-0.02	-0.07 – 0.02	0.291

Average win-stay across safe and threat conditions	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI
Acute stress factor	-0.08	-0.12 – -0.03	<b>0.001</b>	-0.08	-0.13 – -0.03	<b>0.001</b>
Interaction between the acute and chronic stress factors				-0.01	-0.05 – 0.03	0.517
Observations			109			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.266 / 0.231			0.269 / 0.227	
Significance		$F(5, 103) = 7.48, p < \mathbf{0.001}$			$F(6, 102) = 6.27, p < \mathbf{0.001}$	

Two similar regression models predicting the average lose-switch performance across threat and safe conditions were tested (refer to [Table 5](#)). As before, the first model included the predictors age, gender, years of education, chronic stress factor and acute stress factor, and the second model additionally included an interaction term between the acute and chronic stress factors.

The first model was significant ( $F(5, 103) = 5.58, p < 0.001$ ), and the predictors accounted for 21.3% of the variance. Only the acute stress factor significantly predicted lower lose-switch scores. The second model was also significant ( $F(6, 102) = 4.65, p < 0.001$ ), and its predictors accounted for 21.5% of the variance in average lose-switch across threat and safe conditions. The second model also showed a significant effect of the acute stress factor, but not an interaction between acute and chronic stress factors.

Table 5 Linear regression models predicting average lose-switch performance across both safe and threat conditions

Average lose-switch across safe and threat conditions	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.60	0.38 – 0.82	< <b>0.001</b>	0.60	0.38 – 0.82	< <b>0.001</b>
Age	0.00	-0.00 – 0.01	0.095	0.00	-0.00 – 0.01	0.092
Gender [female]	0.04	-0.02 – 0.10	0.236	0.04	-0.02 – 0.10	0.244
Years of education	-0.01	-0.02 – 0.01	0.285	-0.01	-0.02 – 0.01	0.266
Chronic stress factor	-0.01	-0.05 – 0.02	0.471	-0.01	-0.05 – 0.03	0.584
Acute stress factor	-0.05	-0.09 – -0.01	<b>0.008</b>	-0.05	-0.09 – -0.01	<b>0.017</b>
Interaction between the acute and chronic stress factors				0.01	-0.03 – 0.04	0.647
Observations			109			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.213 / 0.175			0.215 / 0.169	
Significance		$F(5, 103) = 5.58, p < \mathbf{0.001}$			$F(6, 102) = 4.65, p < \mathbf{0.001}$	

We further ran two similar regression models predicting the difference in mean win-stay performance between threat and conditions, one with and one without the interaction between the acute and chronic stress factors (refer to Table 6). Neither model was significant (refer to Table 6), as the predictors accounted for only 1.8% and 2.8% of the variance, respectively.

Table 6 Linear regression models predicting the difference in mean win-stay performance between threat and safe conditions

Mean win-stay difference between threat and safe	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.01	-0.15 – 0.16	0.918	0.01	-0.15 – 0.16	0.913
Age	0.00	-0.00 – 0.00	0.535	0.00	-0.00 – 0.00	0.576
Gender [female]	-0.00	-0.04 – 0.04	0.987	0.00	-0.04 – 0.04	0.986
Years of education	-0.00	-0.01 – 0.01	0.694	-0.00	-0.01 – 0.01	0.788
Chronic stress factor	-0.01	-0.04 – 0.01	0.312	-0.02	-0.05 – 0.01	0.207
Acute stress factor	0.02	-0.01 – 0.04	0.245	0.01	-0.02 – 0.04	0.408
Interaction between the acute and chronic stress factors				-0.01	-0.04 – 0.01	0.318
Observations			109			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.018 / -0.030			0.028 / -0.030	
Significance		F(5, 103) = 0.38, p = 0.86			F(6, 102) = 0.48, p = 0.82	

Finally, we ran two more regression models predicting the difference in mean lose-switch performance between threat and safe conditions, again with one having the interaction between the acute and chronic stress factors and one not including the interaction (refer to Table 7). Again, neither model was significant (refer to Table 7), accounting for only 3.4% and 3.6% of the variance, respectively.

Table 7 Linear regression models predicting the difference in mean lose-switch performance between threat and safe conditions

Mean lose-switch difference between threat and safe	Model without interaction			Model with interaction			
	Predictors	Estimates	CI	p	Estimates	CI	p
(Intercept)		0.16	-0.07 – 0.39	0.178	0.16	-0.07 – 0.39	0.178
Age		-0.00	-0.00 – 0.00	0.888	-0.00	-0.00 – 0.00	0.866
Gender [female]		-0.02	-0.08 – 0.05	0.628	-0.01	-0.08 – 0.05	0.641
Years of education		-0.01	-0.02 – 0.00	0.133	-0.01	-0.02 – 0.00	0.153
Chronic Stress Factor		-0.01	-0.05 – 0.03	0.748	-0.01	-0.05 – 0.03	0.655
Acute Stress Factor		0.01	-0.03 – 0.05	0.562	0.01	-0.03 – 0.05	0.677
Interaction between the acute and chronic stress factors					-0.01	-0.05 – 0.03	0.636
Observations				109			
R <sup>2</sup> / R <sup>2</sup> adjusted			0.034 / -0.013			0.036 / -0.021	
Significance			$F(5, 103) = 0.72, p = 0.61$			$F(6, 102) = 0.63, p = 0.70$	

#### 4.4 Discussion

The results revealed insights into the relationships between age, gender, years of education, past acute and chronic stress experiences and decision-making performance under safe and threat conditions. Taken together, the results suggest that there were no statistically significant differences in win-stay, and lose-switch performance between threat and safe conditions. Interestingly, however, while linear regression models predicting the differences in win-stay and lose-switch performance between threat and safe conditions were not statistically significant (refer to [Table 6](#) and [Table 7](#)), models predicting the average win-stay and lose-switch performance across both threat and safe conditions were statistically significant (refer to [Table 4](#) and [Table 5](#)). These models also accounted for a moderate amount of the variance in average win-stay (~27%) and lose-switch (~22%) performance across both safe and threat conditions. Of note, the only significant predictor in both of these models was the acute stress factor, which showed a negative relationship with both average win-stay performance and lose-switch performance. That is, those individuals with higher past acute stress experiences were more likely to show poorer win-stay and lose-switch performance. The other predictors included in these models, i.e. age, gender, and years of education, did not show any significant effects.

It is important to note that a limitation of the online experiment was that there was not a statistically significant difference in decision-making performance between the threat and safe conditions. This suggests that our mild stress manipulation was not aversive enough to participants to have an effect on decision-making performance, perhaps due to variations in the conditions in which the participants completed the task (e.g. whether they had their computer audio on or at what volume). In line with this, none of the linear regression models attempting to predict differences in performance between threat and safe conditions were significant, and the models poorly fitted the data.

Therefore, in the subsequent chapter, we describe an experiment that attempted to test our hypotheses using a more precise and rigorous stressor and stress measurement approach. We

administered a more aversive stressor (bursts of loud white noise) in a laboratory experiment, and we also collected a physiological measure (heart rate) that might reflect the extent to which participants were stressed by this manipulation.



## **Chapter 5: Relationships Between Age, Gender, History of Stress and Decision-making Performance Under Threat – Lab Experiment**

### **5.1 Research questions**

To further build on the results obtained from the online experiment discussed in [Chapter 4](#), and to have more control over experimental conditions, the online experiment was converted to a lab-based study. The following chapter describes the lab-based study conducted to investigate the relationship between decision-making performance, age, gender, acute and chronic stress factors. The hypotheses investigated and result expectations remained the same as described in [Chapter 4](#).

### **5.2 Method**

#### **5.2.1 Participants**

$N=107$  participants (84 females, 21 males, 2 non-binary) were recruited using The University of Adelaide School of Psychology web-based research participation system, hosted by Sona Systems. The participants were comprised of 1<sup>st</sup> year students with ages ranging between 18 and 50 years ( $M= 19.42$ ,  $SD= 3.77$  years). Eligibility requirements included 1) being aged 18 years or over, 2) being fluent in the English language, and 3) not having acute illnesses with antibiotics treatment or being on anti-inflammatory therapy.

With ethical approval (H-2021-124) and participant consent, measures related to decision-making performance, demographics and individual differences in personality, impulsivity and history of stress experiences were collected (see [Appendix 1](#)). Upon completion of the task, participants received course credits.

### 5.2.2 Measures

We used the same measures as [Chapter 4](#) to investigate how individual differences in age, gender, and history of stressors impact decision-making performance under safe and threat conditions (refer to [Appendix 1](#)); however, they were delivered in a laboratory setting, which allowed for the following additional measure to be included:

#### a. Physiological measures of stress

As mentioned in [Chapter 2](#), heart rate is a measure that may correlate with physiological responses to stress. Thus, pulse from the left index was measured continuously throughout the task using a Bionomadix wireless transmitter-receiver system (Biopac Systems Inc., Goleta, CA, USA). The heart rate signals and data from the heart pulse sensor was processed by the AcqKnowledge version 4.3 (Biopac Systems Inc., Goleta, CA, USA) to generate a data file of voltage readings which were then converted offline using the ‘event\_timeseries.py’ script (refer to [Appendix 1 and 2](#)) into beats-per-minute. The beats-per-minute data was averaged across all safe trials and compared to the average heart beats-per-minute across all threat trials in order to test whether the threat manipulation had an effect. Note that, blood pressure was also collected before and after, however, it was not analysed for the purposes of this thesis.

### 5.2.3 Procedure

The experiment was completed in the following order: participants were requested to make themselves comfortable and familiar with their computer task workstation, asked to carefully read instructions and provide consent. Before commencing the game, blood pressure was measured using the Jianzhikang JZK-002R Wrist Style Electronic Blood Pressure Monitor. The Wireless Photo Plethysmogram (PPG), connected to a BioNomadix Transmitter, was placed and adjusted on participants’ non-working idle hand, in order to record their heart rate as they played the lab-based

version of the Dracoin Doors Game (file Mturk\_study\_v62\_lab.html from the attached Supplement Material). Headphones were then placed on the participants, and they were instructed to begin playing the game (as described in [Appendix 1](#)). Following completion of the game, another blood pressure reading was collected, the pulse recording was stopped and sensor removed. Headphones were also removed, and participants were requested to proceed to complete the computer-based demographics questionnaires, including the TIPI, BIS-11, MOSS-20, PSS, DASS-21, PCL-5 and LEC-5 (see [Appendix 1](#)). Note that, unlike the online study in which we could not control the sound level, the dragon roar experienced under the threatening condition was set to be approximately 85dB, in order to elicit a more heightened acute stress response when experiencing the threatening stimuli.

### 5.3 Results

[Table 8](#) shows that scores on the acute stress measures (PSS-4, depression, anxiety, and stress from the DASS-21, and PCL-5) were positively correlated. As in the previous experiment, these scores were combined into a single factor, using the principal components analysis R function ‘principal()’ ([Revelle, 2022](#)). The participants’ scores on the first component is henceforth referred to as the ‘acute stress factor’, which accounted for 77.1% of the variance in the DASS-21, PSS-4 and PCL-5 scores, and their respective loadings were 0.92, 0.86, and 0.86.

Participants also completed two questionnaires that we assumed would reflect experiences with chronic stress, the MOSS-21 (as before, we removed the items assessing recent experiences with depression, anxiety, and stress) and the LEC-5. [Table 8](#) shows that the scores on the modified version of the MOSS-21 and the LEC-5 have a moderate negative correlation (i.e., lower health scores on the MOSS-21 were associated with more adverse events reported in the LEC-5 ). Although the correlation was not significant, for consistency with the method of analysis used in the online experiment, these scores were also combined into a single factor using the aforementioned ‘principal()’ function. Such scores are based on experiences likely to have persisted for longer than three months; as such, they are assumed to be chronic stressors and hence the combined factor is referred to as the ‘chronic stress

factor'. The chronic stress factor accounted for 56% of the variance in the modified MOSS-21 and LEC-5 scores, and their respective loadings were -0.75, and 0.75.

Before investigating individual differences in decision-making performance, we tested whether the acute stress manipulation had an effect on performance. Paired-sample  $t$ -tests showed that differences in mean win-stay ( $t(106) = -1.341, p = 0.183, CI_{95\%} \text{ Cohen's } d [-0.033, 0.006]$ ) and lose-switch performance ( $t(106) = -1.898, p = 0.06, CI_{95\%} \text{ Cohen's } d [-0.061, 0.001]$ ) between threat and safe conditions were not significant. However, a paired-sample  $t$ -test showed that the difference in heart beats per minute (BPM) between threat and safe conditions was significant ( $t(106) = 4.171, p < 0.001, CI_{95\%} \text{ Cohen's } d [0.232, 0.652]$ ). This difference can be attributed to the acute stress manipulation in the Dracoin Doors Game, i.e. the change between safe and threat conditions in the game. The change in BPM between safe and threat conditions is assumed to be a measure of physiological reactivity to the acute stress manipulation in the Dracoin Doors Game. Again, for consistency with the online experiment method of analysis, in addition to including linear regression models predicting the difference in win-stay and lose-switch between threat and safe conditions, we also included models predicting the average win-stay and lose-switch across both threat and safe conditions.

Table 8 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for scores on the PSS-4, depression, anxiety and stress of the DASS-21, DSM-5 PTSD checklist (PCL-5) with life events checklist (LEC-5), and modified MOSS-21

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6
1. PSS-4	7.10	2.81						
2. Depression	12.94	6.84	.59** [.45, .70]					
3. Anxiety	12.88	7.00	.72** [.62, .80]	.82** [.74, .87]				
4. Stress	10.33	6.35	.70** [.59, .79]	.86** [.80, .90]	.86** [.80, .90]			
5. PCL-5	23.04	16.73	.56** [.41, .68]	.66** [.54, .75]	.64** [.51, .74]	.69** [.58, .78]		
6. LEC-5	0.57	0.24	.00 [-.19, .19]	.12 [-.07, .30]	.03 [-.16, .22]	.09 [-.10, .28]	.08 [-.12, .26]	
7. MOSS-21 (modified)	34.42	4.10	-.47** [-.61, -.31]	-.56** [-.68, -.41]	-.51** [-.63, -.35]	-.59** [-.70, -.45]	-.46** [-.60, -.29]	-.12 [-.31, .07]

*Note.* *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .

Table 9 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for age, years of education, acute and chronic stress factors, heart rate difference between threat and safe conditions, average win-stay and lose-switch performance across both safe and threat conditions, and the differences in mean win-stay and lose-switch performance between threat and safe conditions.

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5
1. Age	19.42	3.77					
2. Years of education	13.52	1.35	.28** [.09, .45]				
3. Acute stress factor	0.00	1.00	-.07 [-.26, .12]	-.12 [-.31, .07]			
4. Chronic stress factor	-0.00	1.00	-.01 [-.20, .18]	.03 [-.16, .22]	.43** [.26, .57]		
5. Heart rate difference	0.44	1.10	-.08 [-.27, .11]	.05 [-.14, .24]	.05 [-.14, .24]	.01 [-.18, .20]	
6. Win-stay average	0.83	0.15	-.00 [-.19, .19]	-.05 [-.24, .14]	-.07 [-.26, .12]	-.04 [-.22, .16]	.27** [.09, .44]
7. Lose-switch average	0.72	0.16	.01 [-.18, .20]	-.12 [-.30, .08]	.03 [-.16, .22]	-.07 [-.26, .12]	.23* [.04, .40]
8. Win-stay difference	-0.01	0.10	-.23* [-.40, -.04]	-.00 [-.19, .19]	-.13 [-.32, .06]	-.16 [-.34, .03]	.19* [.00, .37]
9. Lose-switch difference	-0.03	0.16	-.21* [-.38, -.02]	.03 [-.16, .22]	-.01 [-.20, .18]	-.07 [-.25, .13]	.09 [-.10, .28]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .

### 5.3.1 Age, gender, years of education, acute and chronic stress

Two regression models predicting the average win-stay performance across threat and safe conditions were tested (Table 10). The first model included age, gender, years of education, chronic stress factor and acute stress factor as predictors of the average of win-stay performance across both safe and threat conditions. The second model included the same predictors as the first, however, it also included an interaction between the acute and chronic stress factors as an additional predictor. Neither of the models or their coefficients were statistically significant (Table 10) as the predictors accounted for only 1.0% and 1.5% of the variance, respectively.

Table 10 Linear regression models predicting average win-stay across safe and threat conditions

Average win-stay across safe and threat conditions	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.92	0.59 – 1.25	<0.001	0.91	0.58 – 1.24	<0.001
Age	0.00	-0.01 – 0.01	0.909	0.00	-0.01 – 0.01	0.959
Gender [female]	-0.00	-0.08 – 0.07	0.904	-0.00	-0.08 – 0.08	0.932
Gender [other]	0.04	-0.21 – 0.28	0.772	0.05	-0.20 – 0.30	0.681
Years of education	-0.01	-0.03 – 0.02	0.557	-0.01	-0.03 – 0.02	0.628
Chronic stress factor	-0.00	-0.04 – 0.03	0.939	0.00	-0.04 – 0.04	0.951
Acute stress factor	-0.01	-0.05 – 0.02	0.493	-0.01	-0.05 – 0.02	0.488

Average win-stay across safe and threat conditions	Model without interaction			Model with interaction			
	Predictors	Estimates	CI	p	Estimates	CI	p
Interaction between the acute and chronic stress factors					-0.01	-0.04 – 0.02	0.482
Observations				107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.010 / -0.049			0.015 / -0.055		
Significance		F(6, 100) = 0.17, p = 0.98			F(7, 99) = 0.22, p = 0.98		

Two similar regression models predicting the average lose-switch performance across threat and safe conditions were tested (refer to Table 11). As before, the first model included the predictors age, gender, years of education, chronic stress factor and acute stress factor, and the second model additionally included an interaction term between the acute and chronic stress factors. Again, neither of these models or their coefficients were statistically significant (Table 11). Both predictors accounted for only 2.6% of the variance.

Table 11 Linear regression models predicting average lose-switch across both safe and threat conditions

Average lose-switch across safe and threat conditions	Model without interaction			Model with interaction			
	Predictors	Estimates	CI	p	Estimates	CI	p
(Intercept)		0.87	0.52 – 1.22	<0.001	0.87	0.52 – 1.22	<0.001
Age		0.00	-0.01 – 0.01	0.619	0.00	-0.01 – 0.01	0.624
Gender [female]		0.00	-0.08 – 0.09	0.920	0.00	-0.08 – 0.09	0.919



Average lose-switch across safe and threat conditions	Model without interaction			Model with interaction		
	<i>Predictors</i>	<i>Estimates</i>	<i>CI</i>	<i>p</i>	<i>Estimates</i>	<i>CI</i>
Gender [other]	0.08	-0.18 – 0.34	0.559	0.08	-0.19 – 0.34	0.565
Years of education	-0.01	-0.04 – 0.01	0.256	-0.01	-0.04 – 0.01	0.264
Chronic stress factor	-0.02	-0.06 – 0.02	0.333	-0.02	-0.06 – 0.02	0.347
Acute stress factor	0.01	-0.03 – 0.05	0.623	0.01	-0.03 – 0.05	0.625
Interaction between the acute and chronic stress factors				-0.00	-0.03 – 0.03	0.983
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.026 / -0.032			0.026 / -0.043	
Significance		$F(6, 100) = 0.17, p = 0.98$			$F(7, 99) = 0.38, p = 0.91$	

We further ran two similar regression models predicting the differences in mean win-stay performance between threat and safe conditions, one with and one without the interaction between the acute and chronic stress factors (refer to Table 12). The first model was significant ( $F(6, 100) = 2.73, p < 0.05$ ), and the predictors accounted for 14.1% of the variance. Only age significantly predicted lower differences in mean win-stay performance between threat and safe conditions. The second model was also significant ( $F(7, 99) = 3.27, p < 0.01$ ), and its predictors accounted for 18.8% of the variance in the differences in mean win-stay performance between threat and safe conditions.

The second model additionally showed a significant effect for the interaction between acute and chronic stress factors, which is illustrated in [Figure 6](#).

Table 12 Linear regression models predicting the difference in mean win-stay performance between threat and safe conditions

Mean win-stay difference between threat and safe <i>Predictors</i>	Model without interaction			Model with interaction		
	<i>Estimates</i>	<i>CI</i>	<i>p</i>	<i>Estimates</i>	<i>CI</i>	<i>p</i>
(Intercept)	0.05	-0.16 – 0.26	0.634	0.03	-0.17 – 0.24	0.734
Age	-0.01	-0.01 – -0.00	<b>0.007</b>	-0.01	-0.01 – -0.00	<b>0.003</b>
Gender [female]	0.02	-0.03 – 0.07	0.398	0.02	-0.02 – 0.07	0.326
Gender [other]	-0.15	-0.30 – 0.00	0.057	-0.12	-0.27 – 0.04	0.135
Years of education	0.00	-0.01 – 0.02	0.515	0.01	-0.01 – 0.02	0.323
Chronic stress factor	-0.01	-0.03 – 0.01	0.523	-0.00	-0.02 – 0.02	0.858
Acute stress factor	-0.01	-0.03 – 0.01	0.309	-0.01	-0.03 – 0.01	0.282
Interaction between the acute and chronic stress factors				-0.02	-0.04 – -0.00	<b>0.018</b>
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted	0.141 / 0.089			0.188 / 0.131		
Significance	$F(6, 100) = 2.73, p < 0.05$			$F(7, 99) = 3.27, p < 0.01$		

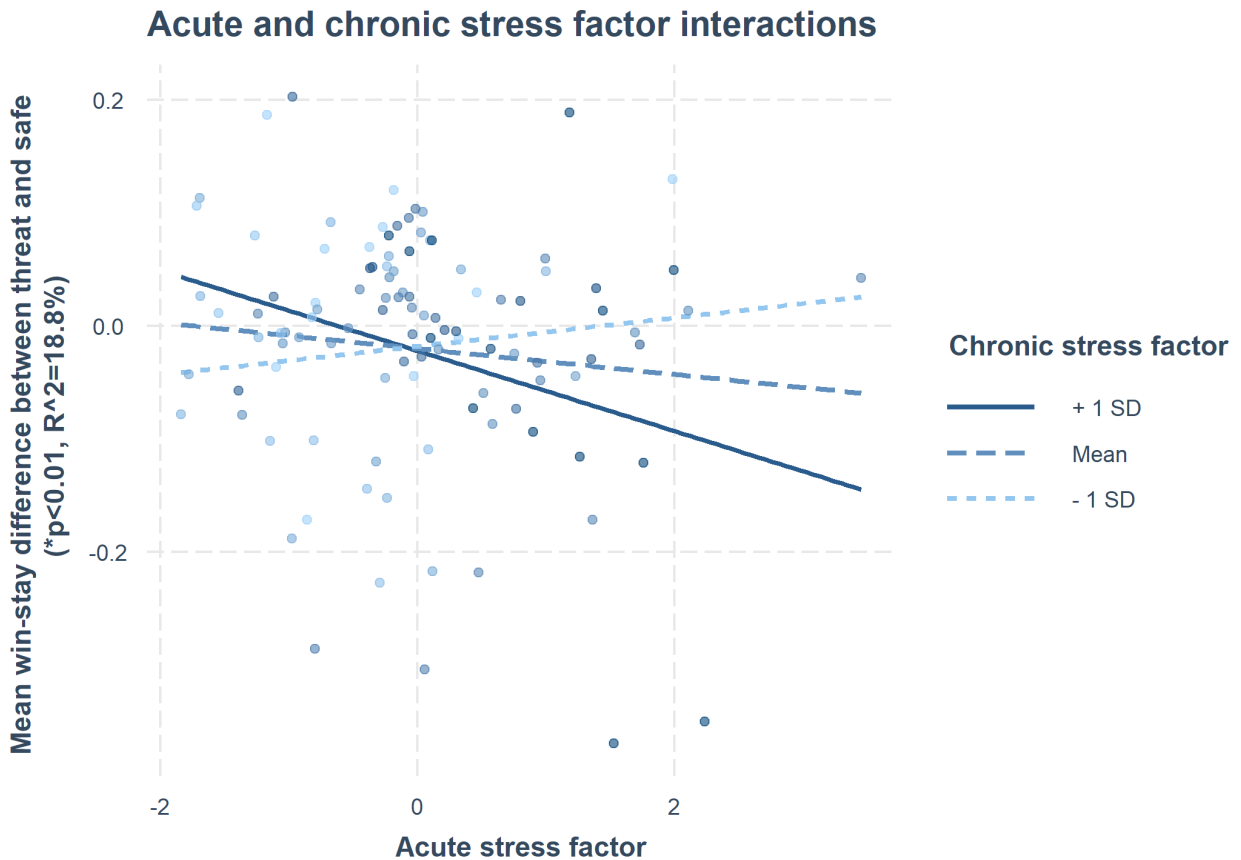


Figure 6 The plot illustrates the interaction between the acute and chronic stress factors from the linear regression model from Table 12, which predicted the mean win-stay differences between threat and safe conditions. Note that both chronic and acute stress factors were included in the regression model as continuous measures. Participants were split into three groups according to their chronic stress factor scores for illustration purposes only. The plot suggests that acute stress impairs win-stay performance under threat (relative to the safe condition), but only in those with a history of chronic stress. That is, the effect of our stress manipulation (shown as more negative win-stay difference scores - i.e., lower performance in the threat condition relative to the safe condition) is only visible in those participants with both high acute and high chronic stress.

We ran another two similar regression models predicting the differences in mean lose-switch performance between threat and safe conditions, again with one featuring the interaction between the acute and chronic stress factors and the other not including this interaction (refer to Table 13). Only age significantly predicted lower differences in mean lose-switch performance between threat and safe conditions across both models. However, neither model was significant (refer to Table 13) and the predictors accounted for 10.3% and 10.9% of the variance, respectively.

Table 13 Linear regression models predicting the difference in mean lose-switch performance between threat and safe conditions

Mean lose-switch difference between threat and safe <i>Predictors</i>	Model without interaction			Model with interaction		
	<i>Estimates</i>	<i>CI</i>	<i>p</i>	<i>Estimates</i>	<i>CI</i>	<i>p</i>
(Intercept)	-0.03	-0.35 – 0.30	0.879	-0.02	-0.35 – 0.31	0.918
Age	-0.01	-0.02 – -0.00	<b>0.014</b>	-0.01	-0.02 – -0.00	<b>0.017</b>
Gender [female]	0.04	-0.04 – 0.12	0.287	0.04	-0.04 – 0.12	0.307
Gender [other]	-0.20	-0.44 – 0.05	0.112	-0.22	-0.47 – 0.03	0.089
Years of education	0.01	-0.01 – 0.04	0.289	0.01	-0.01 – 0.04	0.348
Chronic stress factor	-0.01	-0.04 – 0.03	0.742	-0.01	-0.04 – 0.03	0.633
Acute stress factor	0.00	-0.03 – 0.03	0.973	0.00	-0.03 – 0.04	0.964
Interaction between the acute and chronic stress factors				0.01	-0.02 – 0.05	0.424
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.103 / 0.050			0.109 / 0.046	
Significance		$F(6, 100) = 1.92, p = 0.08$			$F(7, 99) = 1.73, p = 0.11$	

The models above replicated the analyses we performed for the online experiment data presented in [Chapter 4](#). However, we also collected heart rate in this experiment, which allowed us to measure a physiological response to our stress manipulation, and this could be used as an index of stress reactivity. We therefore repeated the analyses, replacing our self-report measure of acute stress with this new stress reactivity measure, which might better reflect an individual’s current physiological response to acute stress. Two regression models predicting the average win-stay performance across threat and safe conditions were tested ([Table 14](#)). The first model included age, gender, years of education, chronic stress factor, and the heart rate difference between threat and safe conditions (which may be considered a measure for heart rate reactivity to acute stress), as predictors of the average of win-stay performance across both safe and threat conditions. The second model included the same predictors as the first, however, it also included an interaction between the heart rate difference and the chronic stress factor as an additional predictor. Neither of the models were statistically significant ([Table 14](#)). However, the heart rate difference had a significant effect in both models, and predictors accounted for 8.5% and 10.7% of the variance, respectively.

*Table 14 Linear regression models predicting average win-stay across safe and threat conditions*

<b>Average win-stay across safe and threat conditions</b>	<b>Model without interaction</b>			<b>Model with interaction</b>		
	<i>Predictors</i>	<i>Estimates</i>	<i>CI</i>	<i>p</i>	<i>Estimates</i>	<i>CI</i>
(Intercept)	0.90	0.58 – 1.21	< <b>0.001</b>	0.87	0.55 – 1.18	< <b>0.001</b>
Age	0.00	-0.01 – 0.01	0.650	0.00	-0.01 – 0.01	0.643
Gender [female]	-0.01	-0.08 – 0.07	0.837	-0.01	-0.08 – 0.07	0.798
Gender [other]	0.05	-0.18 – 0.29	0.654	0.07	-0.17 – 0.31	0.552

Average win-stay across safe and threat conditions	Model without interaction			Model with interaction		
	<i>Predictors</i>	<i>Estimates</i>	<i>CI</i>	<i>p</i>	<i>Estimates</i>	<i>CI</i>
Years of education	-0.01	-0.03 – 0.01	0.448	-0.01	-0.03 – 0.02	0.577
Chronic stress factor	-0.01	-0.04 – 0.02	0.650	-0.02	-0.06 – 0.01	0.256
Heart rate difference between threat and safe	0.04	0.01 – 0.07	<b>0.004</b>	0.04	0.02 – 0.07	<b>0.002</b>
Interaction between the chronic stress factor and heart rate difference				0.02	-0.01 – 0.05	0.121
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.085 / 0.030			0.107 / 0.044	
Significance		$F(6, 100) = 1.54, p = 0.17$			$F(7, 99) = 1.69, p = 0.12$	

Two similar regression models predicting the average lose-switch performance across threat and safe conditions were tested (refer to [Table 15](#)). Again, neither of these models or their coefficients were statistically significant ([Table 15](#)), with the heart rate difference showing a significant effect in both models, and predictors accounting for 8.2% and 11.3% of the variance, respectively.

Table 15 Linear regression models predicting average lose-switch across safe and threat conditions

Average lose-switch across safe and threat conditions	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.87	0.54 – 1.21	< <b>0.001</b>	0.83	0.50 – 1.17	< <b>0.001</b>
Age	0.00	-0.01 – 0.01	0.450	0.00	-0.01 – 0.01	0.440
Gender [female]	0.01	-0.07 – 0.09	0.854	0.01	-0.07 – 0.08	0.899
Gender [other]	0.10	-0.16 – 0.35	0.449	0.12	-0.13 – 0.37	0.350
Years of education	-0.02	-0.04 – 0.01	0.150	-0.01	-0.04 – 0.01	0.226
Chronic stress factor	-0.02	-0.05 – 0.02	0.359	-0.03	-0.07 – 0.01	0.093
Heart rate difference between threat and safe	0.04	0.01 – 0.07	<b>0.013</b>	0.04	0.01 – 0.07	<b>0.006</b>
Interaction between the chronic stress factor and heart rate difference				0.03	-0.00 – 0.06	0.067
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.082 / 0.027			0.113 / 0.050	
Significance		F(6, 100) = 1.49, p = 0.19			F(7, 99) = 1.80, p = 0.10	

We further ran two similar regression models predicting the difference in mean win-stay performance between threat and safe conditions, one with the interaction between the chronic stress

factor and heart rate difference and one without this interaction (refer to Table 16). The first model was significant ( $F(6, 100) = 3.08, p < 0.05$ ), and the predictors accounted for 15.6% of the variance. The second model was also significant ( $F(7, 99) = 2.82, p < 0.05$ ), and its predictors accounted for 16.6% of the variance in the differences between win-stay scores in threat and safe conditions. In both models, age significantly predicted lower differences between win-stay scores in threat and safe conditions.

Table 16 Linear regression models predicting the difference in mean win-stay performance between threat and safe conditions

Mean win-stay difference between threat and safe	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.03	-0.17 – 0.24	0.734	0.05	-0.16 – 0.25	0.633
Age	-0.01	-0.01 – -0.00	<b>0.012</b>	-0.01	-0.01 – -0.00	<b>0.012</b>
Gender [female]	0.02	-0.03 – 0.07	0.462	0.02	-0.03 – 0.07	0.441
Gender [other]	-0.14	-0.30 – 0.01	0.064	-0.15	-0.31 – 0.00	0.052
Years of education	0.00	-0.01 – 0.02	0.513	0.00	-0.01 – 0.02	0.610
Chronic stress factor	-0.01	-0.03 – 0.01	0.238	-0.01	-0.03 – 0.02	0.596
Heart rate difference between threat and safe	0.01	-0.00 – 0.03	0.093	0.01	-0.00 – 0.03	0.130
Interaction between the chronic stress factor and heart rate difference				-0.01	-0.03 – 0.01	0.276
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.156 / 0.106			0.166 / 0.107	
Significance		$F(6, 100) = 3.08, p < \mathbf{0.05}$			$F(7, 99) = 2.82, p < \mathbf{0.05}$	



Finally, we ran another two similar regression models predicting the difference in mean lose-switch performance between threat and safe conditions, again with one of these including the interaction between the chronic stress factor and heart rate difference (refer to Table 17). Neither model was significant (refer to Table 17) and the predictors accounted for 10.7% and 11.7% of the variance, respectively. In both models, age was a significant predictor of lower differences between lose-switch scores in threat and safe conditions.

Table 17 Linear regression models predicting the difference in mean lose-switch performance between threat and safe conditions

Mean lose-switch difference between threat and safe	Model without interaction			Model with interaction		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	-0.03	-0.35 – 0.30	0.874	-0.00	-0.33 – 0.33	0.984
Age	-0.01	-0.02 – -0.00	<b>0.017</b>	-0.01	-0.02 – -0.00	<b>0.016</b>
Gender [female]	0.04	-0.04 – 0.12	0.279	0.04	-0.03 – 0.12	0.264
Gender [other]	-0.19	-0.44 – 0.05	0.120	-0.21	-0.45 – 0.04	0.100
Years of education	0.01	-0.01 – 0.04	0.310	0.01	-0.01 – 0.03	0.383
Chronic stress factor	-0.01	-0.04 – 0.03	0.722	0.00	-0.03 – 0.04	0.853
Heart rate difference between threat and safe	0.01	-0.02 – 0.04	0.548	0.01	-0.02 – 0.03	0.656
Interaction between the chronic stress factor and heart rate difference				-0.02	-0.05 – 0.01	0.290
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.107 / 0.053			0.117 / 0.054	
Significance		F(6, 100) = 1.99, p = 0.07			F(7, 99) = 1.87, p = 0.08	

## 5.4 Discussion

The results revealed interesting insights into the relationships between age, gender, years of education, past acute and chronic stress experiences and decision-making performance under safe and threat conditions. First, there were no statistically significant differences between win-stay and lose-switch performance between threat and safe conditions. However, there was a significant difference between conditions for heart rate, which suggests that the acute stress manipulation had a physiological effect, and that this difference can therefore be considered a measure of the participants' stress reactivity to the experimental threat condition. Interestingly, from [Table 9](#), the mean stress reactivity was positive, suggesting that, on average, heart rate increased during threat conditions. Given that there was a significant moderate positive correlation between stress reactivity and the average win-stay and lose-shift across conditions, the findings imply that a heart rate increase associated with acute stress might have a positive effect on improving win-stay or lose-switch in both safe and threat conditions, perhaps by heightening participants' focus and attention, subsequently improving task performance.

In contrast to the online experiment, the linear models for prediction of average mean-stay or lose-shift performance were not significant, even when accounting for the interaction between the acute and chronic stress factor. Conversely, linear models predicting differences in win-stay performance were significant. As shown in [Table 12](#), for win-stay performance, for the model without the interaction, only age significantly predicted lower differences in mean win-stay performance between threat and safe conditions differences, with 14.1% of the total variance explained by the overall model. Adding in the interaction between the acute and chronic stress factor increased the portion of the variance accounted for to 18.8%, and further suggested that, in addition to age, the interaction between acute and chronic stress factors was significant (see [Table 12](#)). When this interaction was probed further, it was demonstrated that individuals with low acute and chronic stress factors, relative to the mean, are on average more likely to have higher win-stay performance in safe conditions (see [Figure 6](#)). However, those with a low chronic stress factor and higher acute

stress factor are more likely to have higher win-stay performance in threat conditions. In contrast, participants who score highly for both the acute and chronic stress factors are more likely to have higher win-stay performance in safe conditions, while those with a low acute stress factor and high chronic stress factor are more likely, on average, to have higher win-stay performance during threat conditions. The model also suggested that, on average, participants are more likely to have higher win-stay performance during safe conditions compared to threatening conditions. It should also be noted that age was a significant predictor of lower differences in mean win-stay performance between threat and safe conditions in both models assessed, suggesting that differences in performance might reduce with age.

Interestingly, this same pattern wasn't seen for prediction of differences in lose-shift performance in either model tested. While age was a significant predictor of lower differences in mean lose-switch performance between threat and safe conditions across both models, neither model itself reached statistical significance and each explained only a relatively low percentage of the variance.

A unique aspect of the current lab-based experiment, compared to our earlier online experiment, was our ability to use heart rate difference as an index of stress reactivity. Interestingly, while none of the regression models formulated to predict either average win-stay or lose-shift performance were significant, heart rate difference had a significant effect in all models. This was in contrast to the models formulated to predict differences in mean win-stay performance, which did reach significance (with inclusion of the interaction between the chronic stress factor and heart rate difference slightly increasing the amount of variance predicted from 15.6% to 16.6%), but where only age, and not heart rate difference, acted as a significant predictor. It is worth noting, however, that the same pattern was not seen in the models investigating differences in mean lose-shift performance between threat and safe conditions, where despite age acting as a significant predictor, neither model formulated reached statistical significance.

## **Chapter 6: Individual Differences in Age, Gender, Years of Education, Personality, History of Stress, and Decision-making Performance Under Threat**

### **6.1 Hypotheses**

Chapters 4 and 5 formulated significant linear models predicting decision-making performance using age, gender, years of educations, and history of acute and chronic stress as predictors. In contrast, Chapter 6 explores whether personality might explain additional individual differences in decision-making performance. Given that individuals high in openness to experience are more likely to engage in unconventional behaviours (Suridjan et al., 2012), it is expected that the personality trait of openness to experience might show a positive correlation with lose-switch scores. For example, such individuals might be more likely to explore new choices during a door reversal, compared to those with low openness to experience. In other words, they might be more liberal in switching between exploring new alternatives, instead of exploiting the outcomes of previous ones.

Further, it is expected that neuroticism and conscientiousness will positively correlate with decision-making performance overall. This is because neurotic or conscientious individuals are more likely to pay more attention and engage cognitive resources to improve their performance. However, the trait of extraversion is expected to negatively correlate with decision-making performance overall. This is because such individuals are, in general, less conscientious, and/or neurotic; and are more prone to seeking positive rewards. Hence, such individuals are less likely to switch doors during door reversals. To test these hypotheses, correlations and linear regression models are investigated.

### **6.2 Method**

The same data that was collected for Chapters 4 and 5 is analysed here; however, the focus of the analyses is on the relationship between the big five personality factors, as measured by the

TIPI and decision-making performance under threat (refer to the method sections of [Chapter 4](#) and [5](#) for details).

### **6.3 Results**

Relevant to our hypotheses, [Table 18](#) shows that openness, and conscientiousness and agreeableness all had both have a significant positive correlation with average win-stay and lose-switch performance across threat and safe conditions in the online experiment. Conversely, in the lab-based experiment, only openness had a significant positive correlation with either average win-stay or lose-shift performance ([Table 19](#)). A more complex pattern emerged for extraversion and neuroticism. However, only neuroticism had a positive correlation with average win-stay across threat and safe conditions. In contrast, extraversion had a significant negative correlation with both average win-stay and lose-switch across threat and safe conditions in the online experiment. In contrast, neuroticism was only correlated with average win-stay performance. Neither showed any relationship with average win-stay or lose-shift performance in the lab-based experiment.

When it came to difference in performance across safe and threat conditions, only agreeableness displayed any significant correlation for the online experiment. Specifically, there was a negative correlation between this variable and the difference in lose-shift performance between threat and safe conditions. Conversely, for the lab experiment, no significant correlations were observed for any of the personality variables and difference in either win-stay or lose-shift performance. In fact, only age had a significant negative correlation with differences in either of these aspects of performance under threat versus safe conditions.

In terms of relationships between personality and stress factors, [Table 20](#) shows that openness, conscientiousness, agreeableness, and neuroticism all had a significant negative correlation with both the acute and chronic stress factors. In contrast, [Table 21](#) shows only neuroticism had a significant negative correlation with both the acute and chronic stress factors, and conscientiousness just with the acute stress factor.

Table 18 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for age, years of education, personality, average win-stay and lose-switch performance across both safe and threat conditions, and the differences in mean win-stay and lose-switch performance between threat and safe conditions. Data is based on the results from the online experiment described in Chapter 4

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7
1. Age	37.09	10.90							
2. Years of education	15.30	2.31	.14 [-.05, .32]						
3. Openness	4.56	1.22	.09 [-.10, .28]	.21* [.03, .39]					
4. Conscientiousness	4.83	1.31	.32** [.14, .48]	.10 [-.09, .28]	.31** [.12, .47]				
5. Extraversion	3.94	1.25	-.01 [-.20, .18]	-.01 [-.19, .18]	.08 [-.11, .26]	.01 [-.18, .20]			
6. Agreeableness	4.54	1.37	.25** [.07, .42]	.18 [-.01, .36]	.34** [.16, .50]	.54** [.39, .66]	-.14 [-.32, .05]		
7. Neuroticism	4.59	1.23	.37** [.20, .52]	.17 [-.02, .35]	.28** [.10, .44]	.53** [.38, .65]	.25* [.06, .41]	.43** [.26, .57]	
8. Win-stay average	0.66	0.20	.26** [.08, .43]	.13 [-.06, .31]	.33** [.15, .49]	.40** [.23, .54]	-.21* [-.38, -.02]	.43** [.26, .57]	.24* [.05, .41]
9. Lose-switch average	0.60	0.17	.28** [.09, .44]	-.04 [-.23, .15]	.24* [.06, .41]	.39** [.22, .54]	-.20* [-.37, -.01]	.41** [.24, .55]	.17 [-.02, .35]
10. Win-stay difference	0.00	0.11	.02 [-.17, .21]	-.05 [-.24, .14]	-.07 [-.25, .12]	-.08 [-.27, .11]	.07 [-.12, .25]	-.06 [-.25, .13]	-.04 [-.23, .15]
11. Lose-switch difference	-0.02	0.16	-.06 [-.24, .13]	-.16 [-.34, .03]	-.05 [-.24, .14]	.01 [-.18, .20]	.04 [-.15, .22]	-.22* [-.39, -.03]	-.13 [-.31, .06]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .

Table 19 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for age, years of education, personality, average win-stay and lose-switch performance across both safe and threat conditions, and the differences in mean win-stay and lose-switch performance between threat and safe conditions. Data is based on the results from the laboratory experiment described in Chapter 5

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7
1. Age	19.42	3.77							
2. Years of education	13.52	1.35	.28** [.09, .45]						
3. Openness	5.24	0.98	.11 [-.08, .29]	.16 [-.04, .34]					
4. Conscientiousness	5.12	1.24	.02 [-.17, .20]	.09 [-.10, .28]	.05 [-.14, .24]				
5. Extraversion	3.99	1.54	.00 [-.19, .19]	.13 [-.06, .31]	.22* [.04, .40]	-.07 [-.26, .12]			
6. Agreeableness	4.76	0.97	.22* [.03, .39]	.20* [.01, .37]	.17 [-.02, .35]	.16 [-.03, .34]	-.08 [-.27, .11]		
7. Neuroticism	4.00	1.38	.10 [-.10, .28]	.20* [.01, .37]	.26** [.07, .43]	.24* [.05, .41]	.20* [.01, .38]	.11 [-.08, .29]	
8. Win-stay average	0.83	0.15	-.00 [-.19, .19]	-.05 [-.24, .14]	.21* [.03, .39]	.14 [-.05, .32]	.05 [-.14, .23]	-.06 [-.25, .13]	.15 [-.05, .33]
9. Lose-switch average	0.72	0.16	.01 [-.18, .20]	-.12 [-.30, .08]	.25** [.07, .42]	.05 [-.14, .24]	.09 [-.10, .28]	-.06 [-.24, .13]	.09 [-.10, .28]
10. Win-stay difference	-0.01	0.10	-.23* [-.40, -.04]	-.00 [-.19, .19]	.09 [-.10, .27]	.18 [-.02, .35]	.10 [-.09, .29]	-.07 [-.26, .12]	.16 [-.03, .34]
11. Lose-switch difference	-0.03	0.16	-.21* [-.38, -.02]	.03 [-.16, .22]	.08 [-.11, .27]	-.01 [-.20, .18]	-.03 [-.22, .16]	.07 [-.12, .26]	-.04 [-.22, .15]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .

Table 20 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for age, personality, and stress. Data is based on the results from the online experiment described in Chapter 4

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7
1. Age	37.09	10.90							
2. Years of education	15.30	2.31	.14 [-.05, .32]						
3. Openness	4.56	1.22	.09 [-.10, .28]	.21* [.03, .39]					
4. Conscientiousness	4.83	1.31	.32** [.14, .48]	.10 [-.09, .28]	.31** [.12, .47]				
5. Extraversion	3.94	1.25	-.01 [-.20, .18]	-.01 [-.19, .18]	.08 [-.11, .26]	.01 [-.18, .20]			
6. Agreeableness	4.54	1.37	.25** [.07, .42]	.18 [-.01, .36]	.34** [.16, .50]	.54** [.39, .66]	-.14 [-.32, .05]		
7. Neuroticism	4.59	1.23	.37** [.20, .52]	.17 [-.02, .35]	.28** [.10, .44]	.53** [.38, .65]	.25* [.06, .41]	.43** [.26, .57]	
8. Chronic stress factor	0.00	1.00	-.14 [-.32, .05]	.04 [-.15, .22]	-.28** [-.45, -.10]	-.57** [-.68, -.43]	.01 [-.18, .20]	-.43** [-.58, -.27]	-.33** [-.49, -.15]
9. Acute stress factor	-0.00	1.00	-.37** [-.52, -.20]	-.09 [-.28, .10]	-.41** [-.55, -.24]	-.74** [-.82, -.65]	.02 [-.17, .21]	-.60** [-.71, -.47]	-.65** [-.74, -.52]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .



Table 21 Measure means, standard deviations, and Pearson's product-moment correlations with confidence intervals for age, personality, health and stress. Data is based on the results from the online experiment described in Chapter 5

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7
1. Age	19.42	3.77							
2. Years of education	13.52	1.35	.28** [.09, .45]						
3. Openness	5.24	0.98	.11 [-.08, .29]	.16 [-.04, .34]					
4. Conscientiousness	5.12	1.24	.02 [-.17, .20]	.09 [-.10, .28]	.05 [-.14, .24]				
5. Extraversion	3.99	1.54	.00 [-.19, .19]	.13 [-.06, .31]	.22* [.04, .40]	-.07 [-.26, .12]			
6. Agreeableness	4.76	0.97	.22* [.03, .39]	.20* [.01, .37]	.17 [-.02, .35]	.16 [-.03, .34]	-.08 [-.27, .11]		
7. Neuroticism	4.00	1.38	.10 [-.10, .28]	.20* [.01, .37]	.26** [.07, .43]	.24* [.05, .41]	.20* [.01, .38]	.11 [-.08, .29]	
8. Chronic stress factor	-0.00	1.00	-.01 [-.20, .18]	.03 [-.16, .22]	-.02 [-.21, .17]	-.15 [-.33, .04]	.04 [-.15, .23]	.11 [-.08, .29]	-.29** [-.45, -.11]
9. Acute stress factor	0.00	1.00	-.07 [-.26, .12]	-.12 [-.31, .07]	-.18 [-.35, .02]	-.36** [-.51, -.18]	-.11 [-.29, .08]	-.14 [-.32, .05]	-.57** [-.69, -.43]
10. Heart rate difference	0.44	1.10	-.08 [-.27, .11]	.05 [-.14, .24]	-.04 [-.22, .16]	.09 [-.10, .27]	-.09 [-.27, .10]	.09 [-.10, .27]	-.01 [-.20, .18]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .

### 6.3.1 Age, gender, years of education and personality

We first analysed the data from the online experiment described in Chapter 4. Two regression models predicting the average win-stay performance across threat and safe conditions were tested (Table 22). The first model included age, gender, and years of education as predictors of the average of win-stay performance across both safe and threat conditions. The second model included the same predictors as the first, however, it also included neuroticism, openness, extraversion, agreeableness and conscientiousness, in order to test whether the addition of the personality measures to the list of predictors improves the performance of the model. The first model was significant ( $F(3, 105) = 3.81$ ,  $p < 0.05$ ) and the predictors accounted for 9.8% of the variance (see Table 22), with only age significantly predicting slightly higher win-stay scores. The second model was also significant ( $F(8, 100) = 5.92$ ,  $p < 0.001$ ), and its predictors accounted for 32.1% of the variance in average win-stay performance across threat and safe conditions; thus, the addition of the personality measures increased the amount of variance explained by 22.3%. The second model also showed a significant effect for openness and extraversion, with openness predicting higher average win-stay performance across threat and safe conditions, and extraversion predicting lower performance.

Table 22 Linear regression models, predicting average win-stay across safe and threat conditions, generated using the online experiment results from Chapter 4

Average win-stay across safe and threat conditions	Model without personality factors			Model with personality factors			
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI	<i>p</i>
(Intercept)		0.34	0.07 – 0.61	0.015	0.24	-0.04 – 0.52	0.092
Age		0.00	0.00 – 0.01	<b>0.008</b>	0.00	-0.00 – 0.01	0.138
Gender [female]		0.06	-0.02 – 0.13	0.147	0.05	-0.01 – 0.12	0.126
Years of education		0.01	-0.01 – 0.02	0.339	0.00	-0.01 – 0.02	0.910

Average win-stay across safe and threat conditions	Model without personality factors			Model with personality factors			
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI	<i>p</i>
Neuroticism					-0.00	-0.04 – 0.04	0.994
Openness					0.03	0.00 – 0.06	<b>0.027</b>
Extraversion					-0.03	-0.06 – -0.00	<b>0.022</b>
Agreeableness					0.03	-0.00 – 0.06	0.083
Conscientiousness					0.03	-0.00 – 0.06	0.083
Observations				109			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.098 / 0.072			0.321 / 0.267		
Significance		$F(3, 105) = 3.81, p < \mathbf{0.05}$			$F(8, 100) = 5.92, p < \mathbf{0.001}$		

Two similar regression models predicting the average lose-switch performance across threat and safe conditions were tested (refer to [Table 23](#)). As before, the first model included the predictors age, gender, years of education, and the second model additionally included neuroticism, openness, extraversion, agreeableness and conscientiousness. The first model was significant ( $F(3, 105) = 3.62, p < 0.05$ ) and the predictors accounted for 9.4% of the variance, with only age significantly predicting higher win-stay scores. The second model was also significant ( $F(8, 100) = 5.34, p < 0.001$ ), and its predictors accounted for 29.9% of the variance in average win-stay across threat and safe conditions (i.e., including the personality measures resulted in a 20.5% increase in explained variance). Similarly, to the first model, age was significant, however, the second model showed a significant effect for agreeableness and conscientiousness, with both predicting higher average win-stay performance across safe and threat conditions.

Table 23 Linear regression models, predicting average lose-switch across safe and threat conditions, generated using the online experiment results from Chapter 4

Average lose-switch across safe and threat conditions	Model without personality factors			Model with personality factors		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.51	0.29 – 0.74	< <b>0.001</b>	0.43	0.19 – 0.67	<b>0.001</b>
Age	0.00	0.00 – 0.01	<b>0.003</b>	0.00	0.00 – 0.01	<b>0.047</b>
Gender [female]	0.04	-0.03 – 0.10	0.245	0.03	-0.02 – 0.09	0.252
Years of education	-0.01	-0.02 – 0.01	0.358	-0.01	-0.02 – 0.00	0.088
Neuroticism				-0.01	-0.04 – 0.02	0.399
Openness				0.02	-0.01 – 0.05	0.129
Extraversion				-0.02	-0.05 – 0.00	0.076
Agreeableness				0.03	0.00 – 0.05	<b>0.041</b>
Conscientiousness				0.03	0.00 – 0.06	<b>0.036</b>
Observations			109			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.094 / 0.068			0.299 / 0.243	
Significance		$F(3, 105) = 3.62, p < \mathbf{0.05}$			$F(8, 100) = 5.34, p < \mathbf{0.001}$	

We ran another two similar regression models predicting the difference in mean win-stay performance between threat and safe conditions (refer to Table 24). Neither model was significant and the predictors accounted for only 0.3% and 1.9% of the variance, respectively.

Table 24 Linear regression models predicting the difference in mean win-stay performance between threat and safe conditions. The models were generated from the data set of the online experiment discussed in Chapter 4.

Mean win-stay difference between threat and safe	Model without personality factors			Model with personality factors		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.03	-0.12 – 0.18	0.670	0.04	-0.14 – 0.21	0.681
Age	0.00	-0.00 – 0.00	0.769	0.00	-0.00 – 0.00	0.546
Gender [female]	-0.00	-0.04 – 0.04	0.887	-0.00	-0.05 – 0.04	0.855
Years of education	-0.00	-0.01 – 0.01	0.598	-0.00	-0.01 – 0.01	0.729
Neuroticism				-0.00	-0.03 – 0.02	0.792
Openness				-0.00	-0.02 – 0.02	0.704
Extraversion				0.01	-0.01 – 0.03	0.441
Agreeableness				0.00	-0.02 – 0.02	0.940
Conscientiousness				-0.01	-0.03 – 0.01	0.560
Observations			109			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.003 / -0.025			0.019 / -0.059	
Significance		$F(3, 105) = 0.12, p = 0.95$			$F(8, 100) = 0.25, p = 0.98$	

Another two regression models tested whether the demographic and personality variables could predict the difference in mean lose-switch performance between threat and safe conditions (refer to Table 25). Neither model was significant and the predictors accounted for only 3.1% and 9.8% of the variance, respectively. However, the second model revealed that agreeableness predicted significantly lower difference scores in mean lose-switch performance between threat and safe conditions.

Table 25 Linear regression models predicting the difference in mean lose-switch performance between threat and safe conditions. The models were generated from the data set of the online experiment discussed in Chapter 4.

Mean lose-switch difference between threat and safe	Model without personality factors			Model with personality factors		
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI
(Intercept)	0.18	-0.04 – 0.40	0.116	0.17	-0.08 – 0.42	0.188
Age	-0.00	-0.00 – 0.00	0.707	-0.00	-0.00 – 0.00	0.994
Gender [female]	-0.02	-0.08 – 0.04	0.590	-0.01	-0.08 – 0.05	0.649
Years of education	-0.01	-0.02 – 0.00	0.115	-0.01	-0.02 – 0.01	0.231
Neuroticism				-0.02	-0.05 – 0.02	0.299
Openness				0.00	-0.02 – 0.03	0.759
Extraversion				0.00	-0.02 – 0.03	0.766
Agreeableness				-0.03	-0.06 – -0.00	<b>0.033</b>
Conscientiousness				0.03	-0.00 – 0.06	0.074
Observations			109			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.031 / 0.003			0.098 / 0.026	
Significance		$F(3, 105) = 1.11, p = 0.35$			$F(8, 100) = 1.36, p = 0.22$	

We ran similar regression models for the laboratory experiment described in Chapter 5. Two regression models predicting the average win-stay performance across threat and safe conditions were tested (refer to Table 26). The first model included age, gender, and years of education, as predictors of the average of win-stay performance across both safe and threat conditions. The second model included the same predictors as the first, however, it also included neuroticism, openness, extraversion, agreeableness and conscientiousness. Neither model was significant and the predictors

accounted for only 0.4% and 9.2% of the variance, respectively. However, in the second model openness significantly predicted an increase in the average win-stay performance across safe and threat conditions.

Table 26 Linear regression models, predicting average win-stay across safe and threat conditions, generated using the lab experiment results from Chapter 5

Average win-stay across safe and threat conditions	Model without personality factors			Model with personality factors		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.91	0.59 – 1.24	<0.001	0.73	0.38 – 1.09	<0.001
Age	0.00	-0.01 – 0.01	0.884	0.00	-0.01 – 0.01	0.877
Gender [female]	-0.01	-0.09 – 0.07	0.772	0.01	-0.07 – 0.09	0.770
Gender [other]	0.02	-0.21 – 0.25	0.875	0.05	-0.19 – 0.28	0.681
Years of education	-0.01	-0.03 – 0.02	0.598	-0.01	-0.03 – 0.01	0.379
Neuroticism				0.01	-0.01 – 0.04	0.366
Openness				0.03	0.00 – 0.07	<b>0.044</b>
Extraversion				-0.00	-0.02 – 0.02	0.928
Agreeableness				-0.02	-0.05 – 0.01	0.244
Conscientiousness				0.02	-0.01 – 0.04	0.182
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.004 / -0.035			0.092 / 0.007	
Significance		$F(4, 102) = 0.10, p = 0.98$			$F(9, 97) = 1.09, p = 0.38$	

Two similar regression models predicting the average lose-switch performance across threat and safe conditions were tested (refer to Table 27). Neither model was significant and the predictors

accounted for only 1.7% and 10.5% of the variance, respectively. However, the second model revealed that openness significantly increases the average lose-switch performance across safe and threat conditions.

Table 27 Linear regression models, predicting average lose-switch across safe and threat conditions, generated using the lab experiment results from Chapter 5

Average lose-switch across safe and threat conditions	Model without personality factors			Model with personality factors		
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI
(Intercept)	0.89	0.55 – 1.23	<0.001	0.71	0.33 – 1.08	<0.001
Age	0.00	-0.01 – 0.01	0.622	0.00	-0.01 – 0.01	0.651
Gender [female]	0.00	-0.08 – 0.08	0.993	0.01	-0.07 – 0.10	0.737
Gender [other]	0.04	-0.20 – 0.29	0.730	0.05	-0.20 – 0.29	0.716
Years of education	-0.02	-0.04 – 0.01	0.211	-0.02	-0.05 – 0.00	0.102
Neuroticism				0.01	-0.02 – 0.03	0.615
Openness				0.04	0.01 – 0.08	<b>0.014</b>
Extraversion				0.00	-0.02 – 0.03	0.686
Agreeableness				-0.02	-0.05 – 0.02	0.362
Conscientiousness				0.01	-0.02 – 0.04	0.515
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.017 / -0.022			0.105 / 0.022	
Significance		$F(4, 102) = 0.43, p = 0.79$			$F(9, 97) = 1.27, p = 0.26$	

We ran another two similar regression models predicting the difference in mean win-stay performance between threat and safe conditions (refer to Table 28). The first model was significant



( $F(4, 102) = 3.50, p < 0.05$ ) and the predictors accounted for 12.1% of the variance (see Table 28).

The second model was also significant ( $F(9, 97) = 2.53, p < 0.05$ ), and its predictors accounted for 19.0% of the variance in the difference in mean win-stay performance between threat and safe conditions. In both models, age and a non-binary gender significantly predicted lower differences in mean win-stay performance between threat and safe conditions.

Table 28 Linear regression models predicting the difference in mean win-stay performance between threat and safe conditions. The models were generated from the data set of the lab experiment discussed in Chapter 5

Mean win-stay difference between threat and safe	Model without personality factors			Model with personality factors		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.05	-0.16 – 0.25	0.655	-0.06	-0.29 – 0.16	0.590
Age	-0.01	-0.01 – -0.00	<b>0.008</b>	-0.01	-0.01 – -0.00	<b>0.008</b>
Gender [female]	0.01	-0.04 – 0.06	0.606	0.03	-0.02 – 0.09	0.193
Gender [other]	-0.18	-0.33 – -0.03	<b>0.016</b>	-0.15	-0.30 – -0.00	<b>0.050</b>
Years of education	0.01	-0.01 – 0.02	0.463	0.00	-0.01 – 0.02	0.822
Neuroticism				0.01	-0.00 – 0.03	0.156
Openness				0.01	-0.01 – 0.03	0.442
Extraversion				0.01	-0.01 – 0.02	0.431
Agreeableness				-0.01	-0.03 – 0.01	0.522
Conscientiousness				0.01	-0.00 – 0.03	0.128
Observations			107			
R <sup>2</sup> / R <sup>2</sup> adjusted		0.121 / 0.086			0.190 / 0.115	
Significance		$F(4, 102) = 3.50, p < \mathbf{0.05}$			$F(9, 97) = 2.53, p < \mathbf{0.05}$	

Finally, another two regression models were tested each predicting the difference in mean lose-switch performance between threat and safe conditions (refer to Table 29). The first model was

significant ( $F(4, 102) = 2.91, p < 0.05$ ) and the predictors accounted for 10.2% of the variance (see Table 29). The second model was not significant, however, age and a non-binary gender significantly predict lower differences in mean lose-switch performance between threat and safe conditions.

Table 29 Linear regression models predicting the difference in mean lose-switch performance between threat and safe conditions. The models were generated from the data set of the lab experiment discussed in Chapter 5

Mean lose-switch difference between threat and safe	Model without personality factors			Model with personality factors			
	Predictors	Estimates	CI	p	Estimates	CI	p
(Intercept)		-0.02	-0.34 – 0.30	0.901	-0.08	-0.45 – 0.28	0.646
Age		-0.01	-0.02 – -0.00	<b>0.013</b>	-0.01	-0.02 – -0.00	<b>0.007</b>
Gender [female]		0.04	-0.04 – 0.12	0.297	0.03	-0.05 – 0.11	0.496
Gender [other]		-0.21	-0.44 – 0.02	0.070	-0.24	-0.48 – -0.00	<b>0.047</b>
Years of education		0.01	-0.01 – 0.04	0.289	0.01	-0.01 – 0.04	0.388
Neuroticism					-0.00	-0.03 – 0.02	0.716
Openness					0.02	-0.01 – 0.05	0.244
Extraversion					-0.00	-0.03 – 0.02	0.680
Agreeableness					0.02	-0.02 – 0.05	0.297
Conscientiousness					-0.01	-0.03 – 0.02	0.668
Observations				109			
R <sup>2</sup> / R <sup>2</sup> adjusted			0.102 / 0.067			0.130 / 0.049	
Significance			$F(4, 102) = 2.91, p < \mathbf{0.05}$			$F(9, 97) = 1.60, p = 0.12$	

### 6.3.2 Age, gender, years of education and personality and stress factors

Finally, we compared our results across the two experiments by running regression models that included the demographic variables, acute and chronic self-report measures, stress reactivity in the case of the laboratory experiment, and the personality measures. Our aim was to test the extent to which decision-making performance could be predicted by the variables we measured (quantifying it in terms of explained variance) and to test whether the two experiments revealed similar patterns. Two regression models predicting the average win-stay performance across threat and safe conditions were tested (refer to Table 30). Both models included age, gender, years of education, personality, chronic and acute stress factors as predictors of the average of win-stay performance across both safe and threat conditions. However, the second model, which was generated based on laboratory conditions, also included the heart rate differences between threat and safe conditions. The first model was significant ( $F(10, 98) = 5.22, p < 0.001$ ) and the predictors accounted for 34.7% of the variance (see Table 30), with only extraversion significantly predicting lower average win-stay performance across threat and safe conditions. The second model was not significant and the predictors accounted for 17.8% of the variance, however, openness and the heart rate difference between conditions significantly predicted a higher average in win-stay performance across threat and safe conditions.

Table 30 Linear regression models, predicting average win-stay across safe and threat conditions, generated using the experiment results from Chapters 4 and 5

Average win-stay across safe and threat conditions	Online Experiment			Lab Experiment		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.44	0.10 – 0.78	<b>0.013</b>	0.73	0.37 – 1.09	<b>&lt;0.001</b>
Age	0.00	-0.00 – 0.01	0.191	0.00	-0.01 – 0.01	0.595
Gender [female]	0.05	-0.01 – 0.12	0.119	0.01	-0.07 – 0.09	0.745

Average win-stay across safe and threat conditions	Online Experiment			Lab Experiment		
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI
Gender [other]	N/A	N/A	N/A	0.06	-0.17 – 0.30	0.609
Years of education	0.00	-0.01 – 0.02	0.676	-0.01	-0.04 – 0.01	0.246
Neuroticism	-0.01	-0.05 – 0.03	0.474	0.01	-0.01 – 0.04	0.327
Openness	0.03	-0.00 – 0.06	0.089	0.04	0.00 – 0.07	<b>0.030</b>
Extraversion	-0.03	-0.06 – -0.00	<b>0.042</b>	0.00	-0.02 – 0.02	0.907
Agreeableness	0.02	-0.01 – 0.05	0.238	-0.02	-0.06 – 0.01	0.145
Conscientiousness	0.01	-0.03 – 0.05	0.689	0.02	-0.01 – 0.04	0.232
Chronic stress factor	-0.01	-0.05 – 0.03	0.631	0.00	-0.03 – 0.04	0.895
Acute stress factor	-0.05	-0.12 – 0.01	0.091	0.00	-0.04 – 0.04	0.888
Heart rate difference between threat and safe	N/A	N/A	N/A	0.04	0.02 – 0.07	<b>0.002</b>
Observations		109			107	
R <sup>2</sup> / R <sup>2</sup> adjusted		0.347 / 0.281			0.178 / 0.074	
Significance		$F(10, 98) = 5.22, p < \mathbf{0.001}$			$F(12, 94) = 1.70, p = 0.08$	

Two similar regression models predicting the average lose-switch performance across threat and safe conditions were tested (refer to Table 31). The first model was significant ( $F(10, 98) = 4.36, p < 0.001$ ) and the predictors accounted for 30.8% of the variance. The second model was not significant and the predictors accounted for 19.1% of the variance, however, openness and the heart rate difference between conditions significantly predicted a higher average in lose-switch performance across threat and safe conditions.

Table 31 Linear regression models, predicting average lose-switch across safe and threat conditions, generated using the experiment results from Chapters 4 and 5

Average lose-switch across safe and threat conditions	Online Experiment			Lab Experiment		
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI
(Intercept)	0.53	0.23 – 0.83	<b>0.001</b>	0.63	0.25 – 1.01	<b>0.002</b>
Age	0.00	-0.00 – 0.01	0.066	0.00	-0.01 – 0.01	0.440
Gender [female]	0.03	-0.03 – 0.09	0.264	0.02	-0.06 – 0.10	0.626
Gender [other]	N/A	N/A	N/A	0.09	-0.16 – 0.33	0.496
Years of education	0.01	-0.01 – 0.02	0.339	-0.02	-0.05 – 0.00	0.064
Neuroticism	-0.01	-0.02 – 0.00	0.126	0.01	-0.02 – 0.04	0.340
Openness	-0.02	-0.05 – 0.01	0.229	0.05	0.01 – 0.08	<b>0.008</b>
Extraversion	0.02	-0.01 – 0.04	0.228	0.01	-0.01 – 0.03	0.460
Agreeableness	-0.02	-0.05 – 0.00	0.112	-0.02	-0.05 – 0.02	0.353
Conscientiousness	0.02	-0.00 – 0.05	0.088	0.01	-0.02 – 0.04	0.437
Chronic stress factor	0.02	-0.01 – 0.06	0.233	-0.02	-0.05 – 0.02	0.347
Acute stress factor	-0.00	-0.04 – 0.04	0.982	0.03	-0.01 – 0.07	0.198
Heart rate difference between threat and safe	N/A	N/A	N/A	0.04	0.01 – 0.07	<b>0.008</b>
Observations		109			107	
R <sup>2</sup> / R <sup>2</sup> adjusted		0.308 / 0.238			0.191 / 0.087	
Significance		$F(10, 98) = 4.36, p < \mathbf{0.001}$			$F(12, 94) = 1.85, p = 0.05$	

We ran another two similar regression models predicting the difference in mean win-stay performance between threat and safe conditions (refer to Table 32). Unlike the first model, the second model was significant ( $F(12, 94) = 2.2, p < 0.05$ ), and its predictors accounted for 22.0% of the variance in the difference in mean win-stay performance between threat and safe conditions, with age significantly predicting lower differences in mean win-stay performance between threat and safe conditions.

Table 32 Linear regression models predicting the difference in mean win-stay performance between threat and safe conditions. The models were generated from the data set of the experiment discussed in Chapters 4 and 5.

Mean win-stay difference between threat and safe	Online Experiment			Lab Experiment		
	Predictors	Estimates	CI	p	Estimates	CI
(Intercept)	0.06	-0.16 – 0.28	0.598	-0.07	-0.31 – 0.17	0.574
Age	0.00	-0.00 – 0.00	0.496	-0.01	-0.01 – -0.00	<b>0.015</b>
Gender [female]	-0.00	-0.04 – 0.04	0.971	0.04	-0.02 – 0.09	0.172
Gender [other]	N/A	N/A	N/A	-0.13	-0.28 – 0.02	0.099
Years of education	-0.00	-0.01 – 0.01	0.872	0.00	-0.01 – 0.02	0.902
Neuroticism	-0.00	-0.03 – 0.02	0.827	0.01	-0.01 – 0.03	0.229
Openness	-0.00	-0.02 – 0.01	0.620	0.01	-0.01 – 0.03	0.417
Extraversion	0.01	-0.01 – 0.03	0.463	0.01	-0.01 – 0.02	0.335
Agreeableness	-0.00	-0.02 – 0.02	0.900	-0.01	-0.03 – 0.01	0.492
Conscientiousness	-0.01	-0.04 – 0.01	0.372	0.01	-0.01 – 0.03	0.178
Chronic stress factor	-0.02	-0.05 – 0.01	0.229	-0.01	-0.03 – 0.02	0.556
Acute stress factor	0.00	-0.04 – 0.04	0.918	0.00	-0.02 – 0.03	0.906

Mean win-stay difference between threat and safe	Online Experiment			Lab Experiment			
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI	<i>p</i>
Heart rate difference between threat and safe	N/A	N/A	N/A	0.02	-0.00 – 0.03	0.077	
Observations		109			107		
R <sup>2</sup> / R <sup>2</sup> adjusted		0.034 / -0.064			0.220 / 0.120		
Significance		$F(10, 98) = 0.35, p = 0.96$		$F(12, 94) = 2.20, p < \mathbf{0.05}$			

Finally, another two regression models were tested each predicting the difference in mean lose-switch performance between threat and safe conditions (refer to Table 33). Neither of the models was significant, however, in the first model agreeableness was associated with significantly lower difference scores in mean lose-switch performance between threat and safe conditions. In contrast, in the second model age significantly predicted lower mean lose-switch performance between threat and safe conditions.

Table 33 Linear regression models predicting the difference in mean lose-switch performance between threat and safe conditions. The models were generated from the data set of the online experiment discussed in Chapters 4 and 5.

Mean lose-switch difference between threat and safe	Online Experiment			Lab Experiment			
	Predictors	Estimates	CI	<i>p</i>	Estimates	CI	<i>p</i>
(Intercept)	0.18	-0.14 – 0.50	0.272	-0.09	-0.48 – 0.30	0.663	
Age	0.00	-0.00 – 0.00	0.998	-0.01	-0.02 – -0.00	<b>0.010</b>	
Gender [female]	-0.01	-0.08 – 0.05	0.671	0.03	-0.05 – 0.12	0.478	
Gender [other]	N/A	N/A	N/A	-0.22	-0.47 – 0.03	0.084	
Years of education	0.01	-0.01 – 0.02	0.256	0.01	-0.01 – 0.04	0.400	

Mean lose-switch difference between threat and safe	Online Experiment			Lab Experiment		
	<i>Predictors</i>	<i>Estimates</i>	<i>CI</i>	<i>p</i>	<i>Estimates</i>	<i>CI</i>
Neuroticism	-0.01	-0.02 – 0.01	0.343	-0.01	-0.04 – 0.02	0.660
Openness	-0.02	-0.05 – 0.02	0.789	0.02	-0.01 – 0.05	0.249
Extraversion	0.00	-0.02 – 0.03	0.770	-0.00	-0.02 – 0.02	0.746
Agreeableness	0.00	-0.02 – 0.03	<b>0.038</b>	0.02	-0.02 – 0.05	0.302
Conscientiousness	-0.03	-0.06 – -0.00	0.162	-0.01	-0.03 – 0.02	0.625
Chronic stress factor	0.03	-0.01 – 0.06	0.851	-0.01	-0.05 – 0.03	0.619
Acute stress factor	-0.00	-0.04 – 0.04	0.990	-0.00	-0.04 – 0.04	0.967
Heart rate difference between threat and safe	N/A	N/A	N/A	0.01	-0.02 – 0.04	0.606
Observations		109			107	
R <sup>2</sup> / R <sup>2</sup> adjusted		0.099 / 0.007			0.135 / 0.024	
Significance		$F(10, 98) = 1.07, p = 0.39$			$F(12, 94) = 1.22, p = 0.28$	



### 6.3.3 Overall Discussion (Chapters 4-6)

The online experiment revealed two significant linear models, one that included age, gender, years of education, and history of acute and chronic stress and another with the same predictors, however, also including interactions between the acute and chronic stress factor as predictors of average win-stay and lose-switch performance across safe and threat conditions. For each of these models, only the acute stress factor significantly predicted lower average win-stay (see [Table 4](#)) and lose-switch (see [Table 5](#)) performance across safe and threat conditions. The results are also consistent with the observation that the acute stress factor is significantly negatively correlated with average win-stay and lose-switch performance across safe and threat conditions (see [Table 3](#)). The linear regression models and correlations suggest that a higher acute stress factor significantly lowers average win-stay and lose-switch performance across safe and threat conditions.

The stress response has been observed to result in an inverted U-shaped curve ([Chrousos, 2009](#)), which suggests that at lower or higher stress limits, the stress response is maladaptive and significantly impairs performance. Therefore, for individuals in the online experiment who self-report a high amount of current stress, it may be that this results in a level of stress outside the optimal window for performance. In line with this, stress is known to negatively affect many areas of decision-making performance (see [Chapter 2](#) for review; also, [Porcelli and Delgado, 2017](#)). Of particular relevance to the Dracoin Doors Game, acute stress has been shown to reduce sensitivity to reward ([Berghorst et al., 2013](#); [Bogdan & Pizzagalli, 2006](#)), and indeed even to alter activity in neural networks that are important for reward processing, such as the striatum, medial prefrontal cortex and orbitofrontal cortex ([Porcelli et al., 2012](#); [Ossewaarde et al., 2011](#)). Thus, this may impact the participant's ability to engage with the task, which relies, at least to some extent, on the participant being motivated to win coins through correct responses. Interestingly, despite the fact that the chronic stress factor significantly negatively correlated with average win-stay and lose-switch performance across safe and threat conditions (see [Table 3](#)), it was not a

significant predictor of performance in either factor. Further research is needed to probe this, as the literature suggests that chronic stress typically leads to a shift in responses from being more goal-directed to being more reliant on habit, with concomitant reorganisation of frontostriatal circuitry (Dias-Ferreira et al., 2009).

Of note, while acute stress was a predictor of average win-stay and lose-shift performance, it did not act as a significant predictor of the differences in win-stay (refer to Table 6) and lose-switch (refer to Table 7) performance between threat and safe conditions. Indeed, none of the models that we specified for difference in either win-stay or lose-shift performance reached statistical significance. This appears to be because the manipulation itself was not perceived as threatening by the participants, leading to minimal differences between the two conditions for the models to actually predict. In support of this, there was no statistically significant difference in performance between threat and safe conditions. This may be a reflection of the fact that it is difficult to control the experimental conditions of the online decision-making task. For example, we had limited control on the type of workstation used by participants, including their monitor or screen size, headset usage and or sound level. There was also no control over how much attention or effort the individual exerted while performing the task. It is worth noting, however, that recent research has suggested that participants using the MTurk crowdsourcing platform actually display increased effort across a variety of indicators compared to demographically matched peers (Anson, 2018). Similarly, Hauser and Schwartz (2016) reported that MTurk workers actually perform better on online attention checks than participants recruited from undergraduate participant pools, perhaps to improve their chances of receiving compensation (Hauser & Schwarz, 2016). Thus, it is likely that this effect may be due to the ineffectiveness of the threat manipulation itself, rather than a lack of inattentiveness or effort by participants.

Given this, we repeated the experiment under controlled laboratory conditions, enhanced the saliency of the threat experience, and included the collection of heart rate measurements, in order to provide insights into the physiological response to the threat manipulation. Although the

lab experiment results, once again, revealed no statistically significant differences in the differences in mean win-stay and lose-switch performance between safe and threat conditions; there was indeed a statistically significant difference in heart beats-per-minute between threat and safe conditions, which suggests the threat manipulation produced a significant effect as an acute stressor. A review by [Kim and colleagues \(2018\)](#) of 37 studies supports the use of heart rate variation as an indicator of psychological stress, with heart rate varying as a result of stress manipulations of various types across most studies ([Kim et al., 2018](#)). Interestingly, a meta-analysis of neuroimaging studies has suggested that such heart rate variation may be linked to regions such as the ventromedial prefrontal cortex, which is involved in threat perception ([Thayer, et al., 2012](#)). This was in line with our results from [Chapter 5](#), where the difference in heart rate between threat and safe conditions was a significant predictor of average performance in both safe and threat conditions, despite the overall models themselves not reaching statistical significance. Further, there was a significant positive correlation between the acute stress reactivity index and both average win-stay and average lose-switch performance across safe and threat conditions (refer to [Table 9](#)), and also for the differences in mean win-stay performance between threat and safe conditions.

For the lab-based experiment, none of the models specified significantly predicted mean win-stay or lose-shift performance, nor did they predict the difference in mean lose-shift performance between threat and safe conditions. This may indicate that factors beyond those specified in the models are responsible for performance in these aspects of decision-making, but it may also be a result of limitations around study design, such as the nature of the threat condition or the potentially biased nature of a sample of participants drawn from a first-year psychology course participant pool (e.g. restricted age range and years of education). Nevertheless, for difference in mean win-stay performance between the threat and safe conditions, there were several significant linear regression models specified, two with age, gender, years of education, history of chronic stress and either acute stress factor or heart rate variation between threat and

safe condition as predictors, and two others with the same predictors, however, also including the interaction between either the acute and chronic stress factors or heart rate variation and the chronic stress factor as a predictor of the differences in mean win-stay performance between threat and safe conditions (refer to [Table 12](#) and [Table 16](#)). In all of these models, age significantly predicted lower differences in mean win-stay performance between threat and safe conditions. Of note, while the overall models failed to reach statistical significance, age was also a significant predictor of the difference in mean lose-switch performance between threat and safe conditions. This age effect is also consistent with the observed significant negative correlations between age and the difference in mean win-stay performance between threat and safe conditions (refer to [Table 9](#)).

Furthermore, the results show that the interaction between the acute and chronic stress factors was a significant predictor of difference in mean-stay performance between individuals. When the interaction was plotted, it was revealed that acute stress impairs win-stay performance under threat (relative to the safe condition), but only in those with a history of chronic stress. That is, the effect of our stress manipulation (shown as more negative win-stay difference scores, i.e. lower performance in the threat condition relative to the safe condition) is only visible in participants with both high acute and high chronic stress.

It may be that, under threat, such participants become biased toward choosing immediately rewarding options ([Adam and Epel, 2007](#); [Pruessner et al., 2004](#); [Sinha et al., 1999](#)). Hence, the threat condition may be causing interference in learning from negative RPE, making these participants reluctant to switch between doors upon experiencing door reversals. This is in line with our hypothesis discussed in [Chapter 2](#) that acute stress increases dopaminergic output of the VTA, potentially enhancing positive RPEs, but blunting negative RPEs. As such, during threat conditions, such participants may not be learning from punishments in pursuit for rewards, or they might be biased towards riskier options since the chances of losing are perceived as higher ([Miu et al, 2008](#); [Porcelli and Delgado, 2009](#); [Putman et al, 2010](#)). Alternatively, for such participants,

the threat condition might result in impaired attention and goal-action control (for reviews see [Arnsten, 2009](#); [Starcke and Brand, 2012](#)). As noted by [Starcke and Brand \(2012\)](#), under threat, such participants might be engaging in dysfunctional decision strategies, inadequate adjustment from automatic responses and modified reward and punishment sensitivity. Importantly, individuals having both a high acute and chronic stress factor might experience the threat condition differently. Such individuals might not respond as much to rewards and learn less to repeat rewarded actions, which is supported by the fact that such individuals show higher impairments during the threat condition. Conversely, individuals with high acute and low chronic stress factors, and those with low acute and high chronic stress factors, actually show improvements in win-stay performance in the threat condition. It might be that such individuals may be classified as stress responders ([Pruessner et al., 2008](#)), and may have a threat bias that leads them to perform better under threat conditions ([Mogg & Bradley, 1999](#); [Bishop, 2008](#)).

One thing that is interesting to note is that when the acute stress factor was replaced by a more objective measure of heart rate variation between the threat and safe conditions, this more objective measure was a significant predictor of both average mean-stay and average lose-shift performance (although the overall models were not significant), an effect not seen for the self-report acute stress factors. This highlights potential differences between self-reported measures of stress and physiological measures, and emphasises the need to perhaps include other measures in future studies. In line with this, a recent systematic review of 37 studies by [Noushad and colleagues \(2021\)](#) suggested that cortisol, adrenocorticotrophic hormone (ACTH), brain-derived neurotrophic factor (BDNF), catecholamines, glucose, HbA1c, triglycerides, cholesterol, prolactin, oxytocin, dehydroepiandrosterone sulfate (DHEA-S), C-reactive protein (CRP), and interleukins- 6 and 8 may all be potential diagnostic biomarkers of chronic stress. While these have potentially interesting utility, limitations around real-time collection of biomarkers, and the often invasive nature of collection of such biomarkers, must be acknowledged.

In terms of personality differences, the results supported our main hypotheses. For example, openness to experience showed a significant positive correlation with average lose-switch across threat and safe conditions for both the online (refer to [Table 18](#)) and lab (refer to [Table 19](#)) experiments. Similarly, the online experiment results show that conscientiousness had a significant positive correlation with average win-stay and lose-switch performance across threat and safe conditions (refer to [Table 18](#)), and also that neuroticism had a significant positive correlation with average win-stay performance across threat and safe conditions. Furthermore, extraversion had a significant negative correlation with average win-stay and lose-switch performance across threat and safe conditions.

Linear regression models also revealed some support for our hypotheses. For example, a linear regression model (refer to [Table 22](#)) with age, gender, years of education and personality as predictors was significant and explained 32.1% of the variance in average win-stay performance across threat and safe conditions. This model revealed a significant positive coefficient for the openness variable, and a negative one for extraversion. A similar model (refer to [Table 23](#)), predicting average lose-switch explained 29.9% of the variance, with both agreeableness and conscientiousness being significant positive coefficients.

Note that, agreeableness was not considered in our hypotheses, as there was no previous literature we could use to formulate hypotheses. However, [Table 18](#) shows a significant positive correlation between both average win-stay and lose-switch performance across safe and threat conditions and agreeableness. This might be because agreeableness correlates with other personality measures that might affect decision-making. For example, agreeableness correlates with openness, conscientiousness, and extraversion, all of which have shown associations with decision-making performance. Given such limitations, and constraints in the sample size and diversity and confounds in personality scores, further experiments with larger sample sizes are needed to disentangle the individual effects of each personality trait.

Taken together, including personality factors as predictors of win-stay and lose-switch performance considerably increased the variance explained in the models compared to simpler models that only included the demographic variables. For example, in the online experiment, an additional 22.3% of variance in average win-stay performance across safe and threat conditions can be explained by including personality factors as predictors beyond age, gender and years of education (refer to [Table 22](#)). Similarly, an additional 20.5% of the variance in average lose-switch performance is explained by including personality factors as predictors (refer to [Table 23](#)). However, in the online experiment, no significant linear models were identified that explained the variance in the difference in win-stay and lose-switch performance between conditions. Conversely, the lab experiment did not identify significant similar linear regression models that explained variance in average win-stay and lose-switch performance across conditions. Instead, the lab experiment identified that an additional 6.9% of the variance in the difference in mean win-stay performance between threat and safe conditions can be explained by including personality factors in the model (refer to [Table 28](#)). Furthermore, including personality, acute and chronic stress factors, in addition to demographic, in the online experiment increased the explained variance in average win-stay performance by 24.9% (see [Table 30](#)), and the explained variance in average lose-switch performance by 21.4% (refer to [Table 31](#)). In contrast, the lab experiment identified an additional 9.9% of the variance in the difference in mean win-stay performance between threat and safe conditions (see [Table 30](#)). Once again, both the online and lab experiment did not identify significant linear models predicting variances in the difference in mean lose-switch performance across threat and safe conditions. As such, further investigations into additional factors that may explain such individual differences in decision-making performance under threat are required, such as impulsivity.

Thus, personality certainly contributed to explaining variance in decision-making, at least the average win-stay and lose-shift performance. Most notably, openness and agreeableness positively correlated with both average win-stay and lose-switch performance across safe and

threat conditions. In contrast, extraversion showed a significant negative correlation. The direction of these relationships is at least partially consistent with the literature in terms of correlations between cortisol responses and personality factors. [Table 1](#) summarises these studies, which generally report that higher openness and agreeableness are associated with an increase in cortisol release during acute stress, while higher extraversion is associated with a decrease. Elevated cortisol levels have been associated with increases in dopamine signalling which may improve reward learning, but impair avoidance learning ([Lighthall et al., 2013](#)), and consequently improve average win-stay and impair lose-switch performance across safe and threat conditions. In contrast, moderate levels of acute stress-related cortisol may facilitate learning ([Abercrombie et al., 2003](#); [Luksys & Sandi, 2011](#); [Wolf, 2009](#)) and consequently improve both average win-stay and lose-switch performance ([Mather & Lighthall, 2012](#)). Our results suggest that perhaps our participants who scored high in neuroticism and openness might have experienced moderate levels of cortisol increase (given the relatively mild nature of the task and stress manipulation), which might have improved their performance overall, including lose-shift performance.

In the lab experiment, age was another significant factor that predicted lower differences in mean win-stay and lose-switch performance between threat and safe conditions. The results suggest that as individuals age, they might respond similarly during threat and safe conditions. However, there is limited evidence to support this and further investigations are required. In contrast, in the online experiment, age showed significant positive correlations with average win-stay and lose-switch performance in both threat and safe conditions. In comparison, the literature reports that older adults may be more sensitive to positive than negative feedback compared to younger adults ([Denburg et al., 2006](#); [Wood et al., 2005](#)), while others propose the opposite, indicating that older adults might be relatively more sensitive to negative than positive feedback ([Eppinger et al., 2013](#); [Hämmerer et al., 2011](#); [Simon et al., 2010a](#)). However, if there is a shift toward negative-feedback sensitivity, it likely occurs later in old age ([Frank and Kong, 2008](#); [Simon et al., 2010a](#)). Furthermore, as discussed in [Chapter 3](#), on average, the entire DA system



was found to decline between 3.7% and 14.0% per decade, as such it would suggest that age should negatively correlate with average win-stay and lose-switch performance across threat and safe conditions. Our results are therefore inconsistent with these findings, since we found that age positively correlated with performance overall. It is possible that this may be due to other confounding factors, such as motivation to perform or differences in executive function, such as working memory. For example, working memory (WM) facilitates the learning of stimulus-response associations, enabling faster learning and more adaptable association updates than RPE-based learning (van de Vijver & Ligneul 2020). The duration between stimuli, action, and outcome may affect whether WM or reinforcement learning (RL) is engaged (Baddeley 2012). Since the Dracoin Doors Game does not impose time limits for choosing between doors, individual differences in working memory is a factor to consider.

Individual differences in the utilisation of WM and dopamine (DA) function can impact RL (Rmus, McDougle & Collins, 2021). The influence of WM on RL closely correlates with the processing of RPEs in both the striatum and frontoparietal regions (Collins et al., 2017; Collins & Frank, 2018; Collins, 2018). For example, individuals who predominantly rely on WM, compared to RL, exhibit more pronounced effects of set size (i.e., the number of decision options) on RPE signalling. Unexpectedly, simpler tasks, with fewer decision options, tend to show stronger indications of interference between WM and RL processes. This implies that under such circumstances WM might impact RL computations, potentially through competitive or cooperative interactions, influencing reward expectations and attenuating RPE signals (Badre, 2020; Badre & Desrochers, 2019; Collins & Frank, 2018; Collins, 2018). For instance, in straightforward learning environments, information retained in WM elicits quicker reward expectations than the RL system, thus diminishing RPEs (Collins & Frank, 2018; Collins, 2018).

Animal studies support the inverted U relationship between stress-induced DA activation and working memory (Arnsten and Wang, 2016; Zahrt et al., 1997; Goldman-Rakic et al., 2004) and in human pharmacological studies of dopamine, such inverted U relationship varies within

and between individuals depending on task demands (for a review see [Cools & D'Esposito, 2011](#)). For example, both dopamine drug improvements and impairments have been observed across different individuals who perform the same task or within the same individual across different tasks ([Mehta et al., 2004](#); [Frank et al., 2004](#); [Cools et al., 2001](#)). Such findings are relevant to our experiment because in addition to testing win-stay performance, the Dracoin Doors Game includes a winning reversal feature that tests lose-switch performance. The door reversal feature may be considered a different task hence follow a different inverted U performance vs dopamine profile. As such, any increases in dopamine associated with an acute stress manipulation, such as the threat condition in the Dracoin Doors Game, may improve, or degrade, win-stay and lose-switch performance depending on the individual differences in WM and DA function baselines.

Individual differences in baseline dopamine (DA) function have revealed that subjects with low WM capacity exhibit significantly lower DA synthesis capacity in the striatum compared to those with high working memory capacity ([Cools et al., 2008](#)). This pattern persists in older individuals, where striatal DA synthesis capacity not only correlates with WM capacity but also with prefrontal cortex activity during working memory tasks ([Landau et al., 2009](#)). These findings provide direct evidence supporting the hypothesis that dopaminergic effects depend on baseline WM capacity, reflecting differential levels of DA function. Moreover, distinct working memory functions necessitate varying levels of DA across different brain regions. Higher DA levels in the prefrontal cortex might enhance WM stabilisation (which is relevant to win-stay performance) but hinder flexible WM updating (which is relevant to lose-switch performance) ([Meyer-Lindenberg et al., 2005](#); [Pycock, Kerwin, & Carter, 1980](#); [Akil et al., 2003](#)). Conversely, elevated DA levels in the striatum might facilitate flexible WM updating but impede WM stabilisation ([Meyer-Lindenberg et al., 2005](#); [Pycock, Kerwin, & Carter, 1980](#); [Akil et al., 2003](#)). This evidence suggests that individual differences in WM capacity baselines could serve as predictors to understand how dopamine dynamics associated with the threat condition of the

Dracoin Doors Game affect win-stay or lose-switch performance. Taken together, incorporating a measure of WM capacity baseline in future studies might help account for additional variances in win-stay and lose-switch performance.

It's worth noting that, much like dopamine function, WM also changes with age (Blasiman & Was, 2018; Barbey, Koenigs, & Grafman, 2013; Braver & West, 2015; Funahashi, 2017). For instance, aging alone can account for up to 30% of the variations seen in WM (Salthouse, 1994). Young adults tend to engage WM more in short-delay conditions compared to other age groups. Depending on the learning timescale, age-related changes in RL may not only stem from reduced dopaminergic RPE signaling but also from WM decline (Rmus, McDougle & Collins, 2021). These changes in aging are often attributed to alterations in striatal and dopaminergic RPE signaling (Chowdhury et al., 2013; Eppinger, Hämmerer, & Li, 2011; Eppinger et al., 2013). However, the effects of aging extend beyond these systems, particularly impacting the frontal cortex, one of the brain regions most affected by aging (Bennett et al., 2010; Burzynska et al., 2010; Raz et al., 2005; Salat et al., 2009). Age-related changes influence various cognitive functions, including WM and executive functioning, which also rely on dopamine signaling (Berry, Jagust & Hsu 2019, Burzynska et al., 2012; Charlton et al., 2010; Cools & D'Esposito, 2011; Grieve et al., 2007; Madden et al., 2010; Ziegler et al., 2010). Given this evidence which highlights age-related impacts on both RL and WM, and recognizing the interrelationship between RL and WM, it's evident that the construct of WM merits consideration in future studies.

In terms of gender, there was only a significant negative coefficient identified in the lab experiment, which predicted lower differences in mean win-stay performance amongst participants identifying as binary. Since there were only two non-binary participants in the lab study it is difficult to interpret this effect, furthermore there are limited studies that include the construct of gender from multiple perspectives socially, psychologically and biologically. As such further investigation is required with a better representation of non-binary individuals. This statement may also apply for all the factors investigated as part of this thesis, given the restricted

individual differences represented in the participants of both the online and lab experiments, which do not necessarily capture enough diversity of the general population and limit our ability to generalise our findings. For example, in the online experiment participants mainly comprised of US citizens between 25 and 35 years, and most had completed a bachelor's degree. In contrast, the sample of the lab experiment mainly comprised of 18 and 19 year olds, and most were females that had completed high school and were in their first year at university. Given the lack of variance in these demographic variables, it is not surprising that other factors with more variance such as personality, acute and chronic stress, better explained individual differences in win-stay and lose-switch performance. There may also be additional interactions worth exploring that would require a larger and more diverse sample of participants that can then allow for investigating more complex models involving mediators (eg. reward and punishment sensitivity) and moderators (eg. appraisal of challenge and threat) between the relationship of acute stress and decision-making performance under threat.

## Chapter 7: Conclusion, limitations and future works

### 7.1 Summary of findings of the thesis

Within this thesis, we have attempted to synthesise and extend knowledge on the effects of individual differences and stress on decision-making performance under threat. In terms of synthesis of knowledge, we firstly discussed the neurobiology of decision-making based on the dopamine RPE theory. Next, we introduced the concepts of threat and stress and discussed the different perspectives on understanding the stress response, with a particular focus on the impacts on dopaminergic activity within the VTA, which consequently alters RPE. As discussed in [Chapter 2](#), stress can elicit biological disturbances or changes within the body. The body then responds in a way that regulates the disturbance or change, which, in turn, can produce other internal disturbances, hence, initiating a complex cascade of parallel or coupled feedback biological mechanisms until steady state or homeostasis is resolved ([Chrousos, 2009](#); [Sapolsky, Krey, & McEwen, 1986](#)). Such changes can influence the brain and consequently influence learning, particularly through changes on midbrain DA mechanisms, as reviewed in [Chapter 2](#). It is important to note, however, that this process may not impact all individuals in the same manner. For instance, [Pruessner et al. \(2008\)](#) highlights individual differences between stress responders and non-responders after experimentally-induced acute stress using the Montreal Imaging Stress Task ([Dedovic et al., 2005](#)), where subjects are exposed to challenging mental arithmetic presented on a computer screen. Thus, in [Chapter 3](#), we identified how specific individual differences might influence the stress response. Such factors, including age, gender and personality variables, are included since they are major factors with the potential to mediate or moderate the relationship between acute stress and decision-making performance under threat and may interact in complex ways. This is not, however, an exhaustive list, with multiple other factors, such as socioeconomic status ([Roberts et al., 2007](#)), prior life experiences ([Kobasa, 1979](#)) and a history of a diagnosed psychological disorder ([Kendler et al., 2004](#); [Lammel et al., 2014b](#)), such as a mood or anxiety disorder, potentially also influencing the relationship between stress and decision-making.

Following an attempt at synthesising knowledge on the potential impact of individual differences and stress on decision-making under threat within the first three chapters of the thesis, we then designed the Dracoin Doors Game (see [Appendix 1 and 2](#)), and deployed it to conduct both an online (see [Chapter 4](#)), and a lab-based experiment (see [Chapter 5](#)), in order to further understand the impact of individual differences and stress on decision-making performance under threat. In [Chapter 6](#), we extended upon this work by looking at how individual personality factors may impact this performance. Overall, as discussed in [Chapter 4, 5 and 6](#), the results showed significant correlations and linear regression models that provide insights into the relationships between individual differences associated with age, gender, years of education, personality, acute and chronic stress factors; and their impacts on average win-stay and lose-switch performance across threat and safe conditions, and also differences in mean win-stay and lose-switch performance between threat and safe conditions. The results suggest that individual factors associated with personality, acute stress and chronic stress can predict significant variances in decision-making performance under threat in addition to age, gender and years of education. However, it should be noted that the observed correlations and linear models and their coefficients', significance, magnitudes and directions, were not all consistent between the online and lab experiments. However, an observation from the online and lab experiment that does appear to generalise, and which has been described in the literature previously ([Cohen et al., 2007](#); [McEwen, 2008](#); [Schneiderman et al., 2005](#)), is the significant negative correlation between stress and health. For example, [Table 2](#) of the online experiment, shows significant negative correlations between health and stress measures. Similarly, [Table 3](#) of the lab experiment shows the same significant negative correlation, with the exception of the stress measure related to life events, which also has a negative correlation, however, it is not significant.

Based on the results of the lab experiment, the interaction between the acute and chronic stress factor in explaining variances in differences in mean win-stay performance between threat and safe condition illustrate the suggested U-shaped performance relationship as previously referred to in the literature ([Chrousos, 2009](#)). That is, a certain level of stress experiences is necessary for an individual's

optimal decision-making performance under threat. For example, [Figure 6](#) suggests that win-stay performance is impaired in threat conditions when both acute and chronic stress are either high or low. However, when the acute and chronic stress factor are either high or low then win-stay performance is improved.

Of interest, across all models, the factor which most consistently predicted either average performance or difference in performance between threat and safe conditions was age, even in [Chapter 5](#) where the age was relatively restricted, given that the participant pool was drawn from first year Psychology students. This result aligns with the observation that, on average, the entire DA system declines between 3.7% and 14.0% per decade (for meta-analysis see [Karrer et al., 2017](#)). For example, from the lab experiment, age significantly predicted a reduction in the differences between threat and safe win-stay performance. Also, in the online study, age and the acute stress factor were significantly negatively correlated, and the acute stress factor was a significant predictor of lower scores in average win-stay and lose-switch performance across safe and threat conditions. In the online experiment, the age range of participants spanned 5 decades; however, the coefficient of age was relatively small across most linear regression models. As such, the effects of age should be probed further in the future, ideally using a sample drawn from the community with a wider range of age that is more representative of the general population.

## **7.2 Limitations and Future Works**

As clear from the review of the literature presented in the first three chapters of this thesis, the field as a whole currently does a poor job of capturing the complex neurobiological, limbic and endocrine molecular mechanisms that are triggered by specific threatening stimuli, and subsequently how these may affect decision-making performance. Importantly, the current literature also does not include studies which have systematically integrated the topics of individual differences, stress, and decision-making across multiple levels of analysis, including genetic, epigenetic, molecular, neural, through to behavioural, psychological and social. Studies have mostly looked at the topics of individual

differences, decision-making and stress on an individual basis and across disparate levels of analysis. For example, some studies have looked at individual differences in decision-making (Brand et al., 2005; Georgiou et al., 2018; Glicksohn & Zilberman, 2010) and individual differences in the stress response (Agrigoroaei, Polito, & Lachman; Hughes et al., 2011; Lempert et al., 2012) separately. Only one review highlights the need to systematically test moderators and mediators of individual differences between acute stress and decision-making performance (Starcke and Brand, 2012). Furthermore, the literature has yet to standardise a means to consistently and accurately quantify both the stress response and decision-making performance associated with individual differences across multiple levels of analysis.

While this thesis has attempted to address at least some of these shortcomings, it nevertheless has several limitations that must be acknowledged. First, our methods for measuring the stress response were based entirely on either self-report or through a relatively crude measurement based on differences in heart rate. Future work could improve upon this by including more complex measurement of this response, including blood pressure (which we collected data for, but did not analyse), cortisol levels, and other stress-related biomarkers. For example, a systematic review of 37 studies by Noushad and colleagues (2021) suggested that cortisol, adrenocorticotropic hormone (ACTH), brain-derived neurotrophic factor (BDNF), catecholamines, glucose, HbA1c, triglycerides, cholesterol, prolactin, oxytocin, dehydroepiandrosterone sulfate (DHEA-S), C-reactive protein (CRP), and interleukins- 6 and 8 may all be potential diagnostic biomarkers of chronic stress. While this has the potential to result in a more accurate characterisation of the physiological stress response, however, it must be noted that it is difficult to measure such biomarkers during task performance and the invasive nature of measurement may itself impact the stress response. It is also difficult to detect real-time alterations in these markers, at least with currently available technology.

Further, the threat manipulation used in this thesis may not have been sufficient to induce a stress response. This is demonstrated by the fact that there was no difference in performance in either win-stay or lose-shift performance in either experiment. Thus, this may impact the models formulated



in this thesis, making it potentially difficult to draw definitive conclusion about effects on mean win-stay and lose-shift performance during the so-called “threat” versus “safe” conditions. This may be particularly relevant for the models formulated in [Chapter 4](#), which is supported by the fact that no factors were able to predict differences in performance between threat and safe conditions for either win-stay or lose-shift, with all models accounting for negligible percentages of the variance. It may simply be that there was not sufficient variance in performance between the two conditions to allow for these effects to be probed. This could be for several reasons, including that the participants in the online experiment performed the task under uncontrolled conditions, and may in fact have had their computer audio off or playing at a very low volume. Conversely, in the lab-based experiment, where the tone was played directly into headphones, there was a significant difference in heart beats-per-minute (although interestingly not in task performance) between conditions, suggesting at least some effect of the manipulation, and, in turn, the models formulated to predict difference reached significance, at least for differences in win-stay performance between threat and safe conditions; explaining a greater percentage of the variance. Further, while the overall models themselves did not reach significance, the difference in heart rate between threat and safe conditions was able to significantly predict higher average performance in both win-stay and lose-shift across safe and threat conditions when replacing the self-report derived acute stress factor with heart rate difference as a proxy measure of stress reactivity, again, supporting that the manipulation had an effect. There were also significant positive correlations between the differences in heart rate between the threat and safe conditions (or the acute stress reactivity index) and both average win-stay and lose-switch performance across the safe and threat conditions (refer to [Table 9](#)). There was also a significant positive correlation between the acute stress reactivity index and the difference in mean win-stay between threat and safe conditions. Thus, in order to further probe these effects, it may be beneficial to include a more significant stress manipulation, although this may be difficult, given ethical constraints. Given this, future research should employ preclinical models (for review see [Vaessen et al., 2015](#)), which may allow for these complex interactions to be untangled.

It should be noted that the reported  $p$ -values for the linear models and their coefficients have not been adjusted to help control the overall Type I error rate when conducting multiple hypothesis tests. This could have potentially led us to identify some false positives regarding specific predictors. Conversely, a formal power analysis was not conducted prior to conducting this work, with sample size for both the the online and lab-based experiments dictated by cost, time pressure and participant availability constraints. Thus, it needed to be determined if the sample size of the current work was sufficiently powered to detect all potentially significant relationships.

Further, the original analyses were not pre-registered and while the majority of analyses were planned *a priori* in order to allow us to test specific hypotheses that followed from review of the literature, it also must be acknowledged that some analyses evolved through trial and error and by examining trade-offs associated with analytical complexity, sample and schedule constraints. For example, the literature reviewed identified the need to test moderators and mediators between the relationship of stress and decision-making. As such, in the early stages of the study, several hypothetical structured equation models were considered *a priori*; however, these were ultimately not included in the final statistical analysis, due to their complexity and worries that the study would not be sufficiently powered to conduct these, given the variability of the sample.

Finally, future studies can improve on validity and reliability. For example, although the Dracoin Doors Game has been carefully designed to focus on testing the reinforcement learning model of decision-making (as described in [Chapter 1](#)), this model arguably depends on other confounding latent factors such as working memory capacity, executive functioning, reward sensitivity, risk preferences, or loss aversion. Hence, using multiple measures of latent factors related to win-stay and lose-switch performance can enhance the validity and reliability of assessments by providing converging evidence and reducing the impact of measurement error on the correlation between variables.

### 7.3 Overall conclusions

Overall, while this thesis presents corroborating evidence that stress, particularly either currently experiencing acute stress or having a history of chronic stress, has the ability to impact upon decision-making performance. The thesis highlights the need for more consistent, impactful controlled stress protocols in decision-making research, in order to understand how performance may differ between conditions, and what mediating/moderating effect individual difference may have on this. Some aspects to consider include, for example, standardising the nature of the threat (e.g. mortality salience, fearful faces, emotional stimuli, uncertainty, uncontrollable events, etc.), as well as the stressor's duration, intensity and frequency and the context features of the environment in which the threat manipulation is administered. Groups must also do significant pilot work in order to ensure that the manipulation is sufficiently perceived as threatening by their cohort of interest. For example, threat characteristics may impact attention (Bishop, 2008), behavioural responses (LeDoux & Daw, 2018), reinforcement learning (Gao et al., 2020) or emotional responses (Lipka, Miltner, & Straube, 2011). We have made the Dracoin Doors Game open-source, so that it can provide a starting point for other groups to modify for their own investigations. Coupled with this, it is also critical to accurately measure the stress response. Interestingly, this thesis provided evidence that self-report of both acute and chronic stress may have utility for predicting either average performance or differences in performance between threat and safe conditions; however, while encouraging, this may be improved by incorporating more objective measurement of biomarkers associated with either acute or chronic stress.

Finally, this thesis highlights the importance of taking into account individual measures when considering the effect of stress on decision-making. In line with this, age was a consistently significant predictor of either average performance or differences in performance between threat and safe conditions, with personality factors also having an impact. For example, in the online experiment an additional 22.3% variances in average win-stay performance across safe and threat conditions, can be explained by including personality factors as predictors beyond age, gender and years of education

(refer to [Table 22](#)). Similarly, an additional 20.5% of the variance in average lose-switch performance is explained by including personality factors as predictors (refer to [Table 23](#)).

Given how many factors have the potential to vary between individuals, including genetics, life history, and both personality and demographic variables, among others too numerous to name, our work provides a rich basis for future research into the impact of stress on decision-making and how individual factors can influence this. Such foundational research is a critical first step in guiding the design and development of stress management prevention, as well as intervention decision support systems.

## References

- Abercrombie, E. D. et al. (1989) 'Differential Effect of Stress on In Vivo Dopamine Release in Striatum, Nucleus Accumbens, and Medial Frontal Cortex', *Journal of Neurochemistry*, pp. 1655–1658. doi: 10.1111/j.1471-4159.1989.tb09224.x.
- Abercrombie, H. C., Kalin, N. H., Thurow, M. E., Rosenkranz, M. A., & Davidson, R. J. (2003). Cortisol variation in humans affects memory for emotionally laden and neutral information. *Behavioral Neuroscience*, 117, 505–516.
- Adam, T.C. and Epel, E.S. (2007) 'Stress, eating and the reward system', *Physiology & Behavior*, pp. 449–458. doi:10.1016/j.physbeh.2007.04.011.
- Ader, R., Felten, D. L., & Cohen, N. (Eds.). (2001). *Psychoneuroimmunology* (3rd ed.). New York: Academic Press.
- Adler, C.M., Elman, I., Weisenfeld, N., Kestler, L., Pickar, D., Breier, A., (2000). Effects of acute metabolic stress on striatal dopamine release in healthy volunteers. *Neuropsychopharmacol. Off. Publ. Am. Coll. Neuropsychopharmacol.* 22,545–550.
- Afrisham R, Aberomand M, SoliemaniFar O, Kooti W, Ashtary-Larky D, Alamiri F, et al. (2016) Levels of salivary immunoglobulin A under psychological stress and its relationship with rumination and five personality traits in medical students. *Eur J Psychiatry* 2016;30:41-53.
- Afrisham R, Sadegh-Nejadi S, SoliemaniFar O, Kooti W, Ashtary-Larky D, Alamiri F, et al. (2016) Salivary testosterone levels under psychological stress and its relationship with rumination and five personality traits in medical students. *Psychiatry Investig*;13:637-643.
- Afrisham R, Sadegh-Nejadi S, SoliemaniFar O, Abromand M, Kooti W, Najjar Asl S, et al. (2015) Evaluating the salivary alpha-amylase level under psychological stress and its

relationship with rumination and the five personality traits. *J Mazandaran Univ Med Sc*;25:22-33.

Afshar, H. et al. (2015) 'The association of personality traits and coping styles according to stress level', *Journal of research in medical sciences: the official journal of Isfahan University of Medical Sciences*, 20(4), pp. 353–358.

Agrigoroaei S, Polito M, Lachman M. Individual differences in stress reactivity: The role of personality. 12th Annual Meeting of the Society for Personality and Social Psychology at San Antonio, Texas. Available at:

<http://www.brandeis.edu/departments/psych/lachman/pdfs/SPSP.202011>. 2011.

Accessed June 19, 2017.

Akil, M., Kolachana, B. S., Rothmond, D. A., Hyde, T. M., Weinberger, D. R., Kleinman, J. E., et al. (2003). Catechol-O-methyltransferase genotype and dopamine regulation in the human brain. *Journal of Neuroscience*, 23, 2008–2013.

Alcacer, C. et al. (2017) 'Chemogenetic stimulation of striatal projection neurons modulates responses to Parkinson's disease therapy', *Journal of Clinical Investigation*, pp. 720–734. doi: 10.1172/jci90132.

Alexander, G. E. and Crutcher, M. D. (1990) 'Functional architecture of basal ganglia circuits: neural substrates of parallel processing', *Trends in neurosciences*, 13(7), pp. 266–271.

Alghasham, A. and Rasheed, N. (2014) 'Stress-mediated modulations in dopaminergic system and their subsequent impact on behavioural and oxidative alterations: an update', *Pharmaceutical biology*, 52(3), pp. 368–377.

Alvarez, G.A. and Cavanagh, P. (2004) 'The capacity of visual short-term memory is set both by visual information load and by number of objects', *Psychological science*, 15(2), pp. 106–111.

Anson, IG 2018, 'Taking the time? Explaining effortful participation among low-cost online survey participants', *Research & Politics*, vol. 5, no. 3, pp. 205316801878548-.

- Anstrom, K.K., Miczek, K.A., Budygin, E.A. (2009). "Increased phasic dopamine signaling in the mesolimbic pathway during social defeat in rats." *Neuroscience*, 161, 3–12.  
<https://doi.org/10.1016/j.neuroscience.2009.03.023>.
- Anstrom, K.K., Woodward, D.J. (2005). "Restraint increases dopaminergic burst firing in awake rats." *Neuropsychopharmacology*, 30, 1832–1840.  
<https://doi.org/10.1038/sj.npp.1300730>.
- Amemori, K.-I. and Sawaguchi, T. (2006) 'Contrasting effects of reward expectation on sensory and motor memories in primate prefrontal neurons', *Cerebral cortex*, 16(7), pp. 1002–1015.
- Antonovsky, A. (1993). The structure and properties of the sense of coherence scale. *Social Science & Medicine*, 36(6):725–733.
- Arend, I & Botella, J (2002), 'Emotional stimuli reduce the attentional blink in sub-clinical anxious subjects', *Psicothema*, vol. 14, no. 2.
- Arnsten, A. F. T. (2011) 'Prefrontal cortical network connections: key site of vulnerability in stress and schizophrenia', *International journal of developmental neuroscience: the official journal of the International Society for Developmental Neuroscience*, 29(3), pp. 215–223.
- Arnsten, A.F.T. (2009) 'Stress signalling pathways that impair prefrontal cortex structure and function', *Nature reviews. Neuroscience*, 10(6), pp. 410–422.
- Arnsten, A.F.T. and Wang, M. (2016) 'Targeting Prefrontal Cortical Systems for Drug Development: Potential Therapies for Cognitive Disorders', *Annual review of pharmacology and toxicology*, 56, pp. 339–360.
- Ashcraft, M. H. and Kirk, E. P. (2001) 'The relationships among working memory, math anxiety, and performance', *Journal of experimental psychology. General*, 130(2), pp. 224–237.

- Ator, N. A. and Griffiths, R. R. (2003) 'Principles of drug abuse liability assessment in laboratory animals', *Drug and alcohol dependence*, 70(3 Suppl), pp. S55–72.
- Baddeley, A. (2012), 'Working Memory: Theories, Models, and Controversies', *Annual Review of Psychology*, vol. 63, no. 1, pp. 1–29.
- Badre, D. (2020). Brain networks for cognitive control: Four unresolved questions. In P. W. Kalivas & M. P. Paulus (Eds.), *Intrusive Thinking across Neuropsychiatric Disorders: From Molecules to Free Will (Strüngmann Forum Reports, vol. 30)*. Cambridge, MA: MIT Press.
- Badre, D., & Desrochers, T. M. (2019). Chapter 9—Hierarchical cognitive control and the frontal lobes. In M. D'Esposito & J. H. Grafman (Eds.), *Handbook of Clinical Neurology* (vol. 163, pp. 165–177). Elsevier. <http://dx.doi.org/10.1016/B978-0-12-804281-6.00009-4>
- Baik, J.-H. (2020) 'Stress and the dopaminergic reward system', *Experimental & molecular medicine*, 52(12), pp. 1879–1890.
- Bale, T.L., Epperson, C.N., 2015. Sex differences and stress across the lifespan. *Nat. Neurosci.* 18, 1413–1420. <https://doi.org/10.1038/nn.4112>.
- Bale, T.L., Epperson, C.N., 2017. Sex as a biological variable: who, what, when, why, and how. *Neuropsychopharmacology* 42, 386–396. <https://doi.org/10.1038/npp.2016.215>.
- Bandura, A. (1993) 'Perceived Self-Efficacy in Cognitive Development and Functioning', *Educational Psychologist*, pp. 117–148. doi: 10.1207/s15326985ep2802\_3.
- Bandura. (1982). Self-efficacy mechanism in human agency. *The American Psychologist*, 37(2), 122–147. <https://doi.org/10.1037/0003-066X.37.2.122>
- Bangasser, D.A., Valentino, R.J., 2014. Sex differences in stress-related psychiatric disorders: neurobiological perspectives. *Front. Neuroendocrinol.* 35, 303–319. <https://doi.org/10.1016/j.yfrne.2014.03.008>.



- Barbey, A.K., Koenigs, M., & Grafman, J. (2013). Dorsolateral prefrontal contributions to human working memory. *Cortex*, 49, 1195–1205. doi: 10.1016/j.cortex.2012.05.022
- Beaulieu, J.-M. and Gainetdinov, R. R. (2011) ‘The Physiology, Signaling, and Pharmacology of Dopamine Receptors’, *Pharmacological Reviews*, pp. 182–217. doi: 10.1124/pr.110.002642.
- Beier, K. T. et al. (2015) ‘Circuit Architecture of VTA Dopamine Neurons Revealed by Systematic Input-Output Mapping’, *Cell*, 162(3), pp. 622–634.
- Belujon, P., Grace, A.A., (2015). Regulation of dopamine system responsivity and its adaptive and pathological response to stress. In: *Proceedings of Biological Sciences*. The Royal Society, p. 282.
- Berg, M. T. et al. (2017) ‘Childhood/Adolescent stressors and allostatic load in adulthood: Support for a calibration model’, *Social science & medicine*, 193, pp. 130–139.
- Berggren, N. and Derakshan, N. (2013) ‘Trait anxiety reduces implicit expectancy during target spatial probability cueing’, *Emotion*, 13(2), pp. 345–349.
- Berghorst, L. H., Bogdan, R., Frank, M. J., & Pizzagalli, D. A. (2013). Acute stress selectively reduces reward sensitivity. *Frontiers in Human Neuroscience*, 7, 133.
- Berridge, K. C., & Robinson, T. E. (1998). What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? *Brain Research. Brain Research Reviews*, 28, 309–369. [https://doi.org/10.1016/S0165-0173\(98\)00019-8](https://doi.org/10.1016/S0165-0173(98)00019-8).
- Berry, AS, Jagust, WJ & Hsu, M (2019), Age-related variability in decision-making: Insights from neurochemistry, *Cognitive, Affective, & Behavioral Neuroscience*, vol. 19, no. 3, pp. 415–434.
- Bibbey A, Carroll D, Roseboom TJ, Phillips AC, de Rooij SR. (2013) Personality and physiological reactions to acute psychological stress. *Int J Psychophysiol*; 90:28-36.
- Bishop, S. J. (2008) ‘Neural mechanisms underlying selective attention to threat’, *Annals of the New York Academy of Sciences*, 1129, pp. 141–152.

- Bishop, S.J. (2009) 'Trait anxiety and impoverished prefrontal control of attention', *Nature neuroscience*, 12(1), pp. 92–98.
- Blanchard, R. J., & Blanchard, D. C. (1990). Anti-predator defense as models of animal fear and anxiety. In P. F. Brain, S. Parmigiani, R. J. Blanchard, & D. Mainardi (Eds.), *Fear and defence* (pp. 89–108). Harwood Academic Publishers.
- Blasiman, R.N., & Was, C.A. (2018). Why is working memory performance unstable? A review of 21 factors. *Europe's Journal of Psychology*, 14, 188–231. doi: 10.5964/ejop.v14i1.1472.
- Block, J. P. et al. (2009) 'Psychosocial stress and change in weight among US adults', *American journal of epidemiology*, 170(2), pp. 181–192.
- Bogdan, R., & Pizzagalli, D. A. (2006). Acute stress reduces reward responsiveness: implications for depression. *Biological Psychiatry*, 60(10), 1147–1154.
- Bogg T, Slatcher RB. (2015) Activity mediates conscientiousness' relationship to diurnal cortisol slope in a national sample. *Health Psychol*; 34:1195-1199.
- Bolger, N., Shilling, E.A., (1991). Personality and the problems of everyday life: the role of neuroticism in exposure and reactivity to daily stressors. *J. Pers.* 59, 355–386.
- Bolla, K.I., Eldreth, D.A., Matochik, J.A., Cadet, J.L., (2004). Sex-related differences in a gambling task and its neurological correlates. *Cereb. Cortex* 14, 1226–1232.
- Borland, J. M., Aiani, L. M., Norvelle, A., Grantham, K. N., O'Laughlin, K., Terranova, J. I., Frantz, K. J., Albers, H. E. (2019). Sex-dependent Regulation of Social Reward by Oxytocin Receptors in the Ventral Tegmental Area, *Neuropsychopharmacology*, 44, 785–792.
- Bosker, W. M., Neuner, I. and Shah, N. J. (2017) 'The role of impulsivity in psychostimulant- and stress-induced dopamine release: Review of human imaging studies', *Neuroscience and biobehavioral reviews*, 78, pp. 82–90.

- Bouchard Jr, T.J. & Loehlin, J.C. (2001), 'Genes, Evolution, and Personality', *Behavior Genetics*, vol. 31, no. 3, pp. 243–273.
- Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, 60, 113–124.
- Brand, Heinze, K., Labudda, K., & Markowitsch, H. J. (2008). The role of strategies in deciding advantageously in ambiguous and risky situations. *Cognitive Processing*, 9(3), 159–173. <https://doi.org/10.1007/s10339-008-0204-4>
- Brand, Kalbe, E., Labudda, K., Fujiwara, E., Kessler, J., & Markowitsch, H. J. (2005). Decision-making impairments in patients with pathological gambling. *Psychiatry Research*, 133(1), 91–99. <https://doi.org/10.1016/j.psychres.2004.10.003>
- Braver, T.S., & West, R. (2015). Working memory, executive control, and aging. In F.I.M. Craik & T.A. Salthouse (Eds.), *The handbook of aging and cognition*. London: Routledge.
- Brischoux, F., Chakraborty, S., Brierley, D.I., Ungless, M.A. (2009). "Phasic excitation of dopamine neurons in ventral VTA by noxious stimuli." *Proc. Natl. Acad. Sci. U. S. A.*, 106, 4894–4899. <https://doi.org/10.1073/pnas.0811507106>.
- Bromberg-Martin, E. S., Matsumoto, M. & Hikosaka, O. (2010). Dopamine in motivational control: rewarding, aversive, and alerting. *Neuron* 68, 815–834
- Burghardt, P.R., Love, T.M., Stohler, C.S., Hodgkinson, C., Shen, P.H., Enoch, M.A., Goldman, D., Zubieta, J.K., (2012). Leptin regulates dopamine responses to sustained stress in humans. *J. Neurosci.: Off. J. Soc. Neurosci.* 32, 15369–15376.
- Burke, A. R. and Miczek, K. A. (2014) 'Stress in adolescence and drugs of abuse in rodent models: role of dopamine, CRF, and HPA axis', *Psychopharmacology*, 231(8), pp. 1557–1580.

Burzynska, A. Z., Nagel, I. E., Preuschhof, C., Gluth, S., Bäckman, L., Li, S.-C., . . .

Heekeren, H. R. (2012). Cortical thickness is linked to executive functioning in adulthood and aging. *Human Brain Mapping, 33*(7), 1607–1620.

doi:10.1002/hbm.21311

Buss, D. M. (2009) ‘How Can Evolutionary Psychology Successfully Explain Personality and Individual Differences?’, *Perspectives on psychological science: a journal of the Association for Psychological Science, 4*(4), pp. 359–366.

Butler, E. A., Wilhelm, F. H., & Gross, J. J. (2006). Respiratory sinus arrhythmia, emotion, and emotion regulation during social interaction. *Psychophysiology, 43*, 612–622.

Byrnes, J. P., Miller, D. C., & Schafer, W. D. (1999). Gender differences in risk-taking: a meta-analysis. *Psychological Bulletin, 125*(3), 367–383.

Bywaters, M., Andrade, J. and Turpin, G. (2004) ‘Determinants of the vividness of visual imagery: the effects of delayed recall, stimulus affect and individual differences’, *Memory, 12*(4), pp. 479–488.

Cabarkapa, M., Korica, V. and Rodjenkov, S. (2011) ‘Personal traits and a sense of job-related stress in a military aviation crew’, *Vojnosanitetski pregled, vol. 68, no. 2*, pp. 143–149.  
doi: 10.2298/vsp1102143c.

Cabib, S. and Puglisi-Allegra, S. (2012) ‘The mesoaccumbens dopamine in coping with stress’, *Neuroscience & Biobehavioral Reviews, pp. 79–89*. doi:  
10.1016/j.neubiorev.2011.04.012.

Cahill, L. et al. (2001) ‘Sex-related difference in amygdala activity during emotionally influenced memory storage’, *Neurobiology of learning and memory, 75*(1), pp. 1–9.

Cai, X., Kim, S. and Lee, D. (2011) ‘Heterogeneous coding of temporally discounted values in the dorsal and ventral striatum during intertemporal choice’, *Neuron, 69*(1), pp. 170–182.

- Calabresi, P, Picconi, B, Tozzi, A, Ghiglieri, V & Di Filippo, M (2014). 'Direct and indirect pathways of basal ganglia: a critical reappraisal', *Nature Neuroscience*, vol. 17, no. 8, pp. 1022–1030.
- Calabresi, P. et al. (2007) 'Dopamine-mediated regulation of corticostriatal synaptic plasticity', *Trends in neurosciences*, 30(5), pp. 211–219.
- Carini, L.M., Nephew, B.C., (2013). Effects of early life social stress on endocrinology, maternal behavior, and lactation in rats. *Horm. Behav.* 64, 634–641.
- Castagne, V., Moser, P., Roux, S., & Porsolt, R. D. (2011). Rodent models of depression: forced swim and tail suspension behavioral despair tests in rats and mice. In *Current Protocols in Neuroscience* (Eds. Enna, S. J. & Williams, M.), Vol 55, pp 8.10A.1–8.10A.14. Wiley. <https://doi.org/10.1002/0471142301.ns0810as55>
- Cannon, W.B., (1914). The emergency function of the adrenal medulla in pain and the major emotions. *Am. J. Physiol.* 33, 356–372.
- Cavanagh, J.F., Frank, M.J., Allen, J.J.B., (2011). Social stress reactivity alters reward and punishment learning. *Soc. Cogn. Affect. Neurosci.* 6, 311–320.
- Chang, M. S. et al. (2000) 'Increased transcription of the tyrosine hydroxylase gene in individual locus coeruleus neurons following footshock stress', *Neuroscience*, 101(1), pp. 131–139.
- Chang, C., Grace, A.A. (2014). "Amygdala-ventral pallidum pathway decreases dopamine activity after chronic mild stress in rats." *Biol. Psychiatry*, 76, 223–230.  
<https://doi.org/10.1016/j.biopsych.2013.09.020>.
- Charlton, R. A., Barrick, T. R., Lawes, I. N. C., Markus, H. S., & Morris, R. G. (2010). White matter pathways associated with working memory in normal aging. *Cortex*, 46(4), 474–489. doi:10.1016/J.CORTEX.2009.07.005.
- Chen, E. and Miller, G. E. (2007) 'Stress and inflammation in exacerbations of asthma', *Brain, Behavior, and Immunity*, pp. 993–999. doi: 10.1016/j.bbi.2007.03.009.

- Chida, Y. and Hamer, M. (2008) 'Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: a quantitative review of 30 years of investigations', *Psychological bulletin*, 134(6), pp. 829–885.
- Chowdhury, R., Guitart-Masip, M., Lambert, C., Dayan, P., Huys, Q., Düzel, E., & Dolan, R. J. (2013). Dopamine restores reward prediction errors in old age. *Nature Neuroscience*, 16(5), 648–653. doi:10.103.
- Christoffel, D.J., Golden, S.A., Russo, S.J., (2011). Structural and synaptic plasticity in stress-related disorders. *Rev. Neurosci.* 22, 535–549. <https://doi.org/10.1515/RNS.2011.044>.
- Chrousos, GP, (2009), 'Stress and disorders of the stress system', *Nature Reviews. Endocrinology*, vol. 5, no. 7, pp. 374–381.
- Chu X, Ma Z, Li Y, Han J. (2015) Agreeableness, extraversion, stressor and physiological stress response. *Intl J Soc Sci Stud*; 3:79-86.
- Chu, N., Zuo, Y., Meng, L., Lee, D. Y.-W., Han, J., & Cui, C. (2007). Peripheral electrical stimulation reversed the cell size reduction and increased BDNF level in the ventral tegmental area in chronic morphine-treated rats. *Brain Res.*, 1182, 90–98. <https://doi.org/10.1016/j.brainres.2007.08.086>.
- Cockrem, J. F. (2007) 'Stress, corticosterone responses and avian personalities', *Journal of Ornithology*, pp. 169–178. doi: 10.1007/s10336-007-0175-8.
- Cohen, J.Y., Haesler, S., Vong, L., Lowell, B.B., Uchida, N. (2012). "Neuron-type-specific signals for reward and punishment in the ventral tegmental area." *Nature*, 482, 85–88. <https://doi.org/10.1038/nature10754>.
- Cohen, S., Janicki-Deverts, D., Miller, G.E. (2007). "Psychological stress and disease." *JAMA*, 298(14), 1685–1687.
- Cohen, S., Kamarck, T. and Mermelstein, R. (1983) 'A global measure of perceived stress', *Journal of health and social behavior*, 24(4), pp. 385–396.

- Cohen, S., Kessler, R. C. and Gordon, L. U. (1997) *Measuring Stress: A Guide for Health and Social Scientists*. Oxford University Press on Demand.
- Colder, C. R., & O'Connor, R. M. (2004). Gray's reinforcement sensitivity model and child psychopathology: Laboratory and questionnaire assessment of the BAS and BIS. *Journal of Abnormal Child Psychology*, 32(4), 435–451.  
<https://doi.org/JACP.0000030296.54122.b6>
- Collins, AG (2018), 'The Tortoise and the Hare: Interactions between Reinforcement Learning and Working Memory', *Journal of Cognitive Neuroscience*
- Collins, A.G.E. & Frank, M.J. (2018), 'Within- and across-trial dynamics of human EEG reveal cooperative interplay between reinforcement learning and working memory', *Proceedings of the National Academy of Sciences - PNAS*, vol. 115, no. 10, pp. 2502–2507.
- Collins, A.G.E., Ciullo, B., Frank, M.J. & Badre, D. (2017), 'Working Memory Load Strengthens Reward Prediction Errors', *The Journal of Neuroscience*, vol. 37, no. 16, pp. 4332–4342.
- Cools, R., & D'Esposito, M. (2011). Inverted-U-shaped dopamine actions on human working memory and cognitive control. *Biological Psychiatry*, 69(12), e113–e125.  
[doi:10.1016/j.biopsych.2011.03.028](https://doi.org/10.1016/j.biopsych.2011.03.028)
- Cools, R, Barker, RA, Sahakian, BJ & Robbins, TW (2001), 'Enhanced or impaired cognitive function in Parkinson's disease as a function of dopaminergic medication and task demands', *Cereb Cortex*, vol. 11, pp. 1136–1143.
- Cools, R., Gibbs, S., Miyakawa, A., Jagust, W., & D'Esposito, M. (2008). Working memory capacity predicts dopamine synthesis capacity in the human striatum. *Journal of Neuroscience*, 28, 1208–1212.
- Contrada, R. J. and Baum, A. (2010) *The Handbook of Stress Science: Biology, Psychology, and Health*. Springer Publishing Company.

- Cordero, M.I., Venero, C., Kruyt, N.D., Sandi, C. (2003). "Prior exposure to a single stress session facilitates subsequent contextual fear conditioning in rats: evidence for a role of corticosterone." *Horm Behav*, 44, 338–345.
- Corum, C.R., Thurmond, J.B., (1977). Effects of acute exposure to stress on subsequent aggression and locomotion performance. *Psychosom. Med.* 39, 436–443.
- Cox, J. and Witten, I. B. (2019) ‘Striatal circuits for reward learning and decision-making’, *Nature reviews. Neuroscience*, 20(8), pp. 482–494.
- Creese, I. et al. (1983) ‘The classification of dopamine receptors: relationship to radioligand binding’, *Annual review of neuroscience*, 6, pp. 43–71.
- Cryan, J., & Mombereau, C. (2004). In search of a depressed mouse: utility of models for studying depression-related behavior in genetically modified mice. *Mol. Psychiatry*, 9, 326–357.
- Cuadra, G. (2001). "Influence of different antidepressant drugs on the effect of chronic variable stress on restraint-induced dopamine release in frontal cortex." *Neuropsychopharmacology*, 25, 384–394. [https://doi.org/10.1016/S0893-133X\(01\)00234-2](https://doi.org/10.1016/S0893-133X(01)00234-2).
- Cuadra, G., Zurita, A., Lacerra, C., Molina, V. (1999). "Chronic stress sensitizes frontal cortex dopamine release in response to a subsequent novel stressor: reversal by naloxone." *Brain Res. Bull.*, 48, 303–308. [https://doi.org/10.1016/S0361-9230\(98\)00179-8](https://doi.org/10.1016/S0361-9230(98)00179-8).
- Cussen, V. A. and Mench, J. A. (2015) ‘The Relationship between Personality Dimensions and Resiliency to Environmental Stress in Orange-Winged Amazon Parrots (*Amazona amazonica*), as Indicated by the Development of Abnormal Behaviors’, *PloS one*, 10(6), p. e0126170.
- Dedovic, K., D’Aguiar, C., Pruessner, J.C., (2009a). What stress does to your brain: a review of neuroimaging studies. *Can. J. Psychiatry* 54, 6–15.



- Dedovic, K., Rexroth, M., Wolff, E., Duchesne, A., Scherling, C., Beaudry, T., Lue, S.D., Lord, C., Engert, V., Pruessner, J., (2009b). Neural correlates of processing stressful information: an event related fMRI study. *Brain Res.* 1293, 49–60.
- Dedovic, K., Renwick, R., Mahani, N. K., Engert, V., Lupien, S. J., & Pruessner, J. C. (2005). The Montreal Imaging Stress Task: Using functional imaging to investigate the effects of perceiving and processing psychosocial stress in the human brain. *Journal of Psychiatry and Neuroscience*, 30(5), 319–325.
- De Jong, G. M., van Sonderen, E. and Emmelkamp, P. M. (1999) ‘A comprehensive model of stress. the roles of experienced stress and neuroticism in explaining the stress-distress relationship’, *Psychotherapy and psychosomatics*, 68(6), pp. 290–298.
- De Kloet, E.R., Sutanto, W., Rots, N., van Haarst, A., van den Berg, D., Oitzl, M., van Eekelen, A., Voorhuis, D., (1991). Plasticity and function of brain corticosteroid receptors during aging. *Acta Endocrinol. (Copenh)* 125 (Suppl. 1), 65—72.
- De Kloet, E.R., Vreugdenhil, E., Oitzl, M.S., Joels, M., (1998). Brain corticosteroid receptor balance in health and disease. *Endocr. Rev.* 19, 269—301.
- Denburg, N.L., Weller, J.A., Yamada, B.A., Shivapour, D.M., Kaup, A.R., LaLoggia, A., Cole, C.A., Tranel, D., Bechara, A., (2009). Poor decision-making among older adults is related to elevated levels of neuroticism. *Ann. Behav. Med.* 37, 164–172.
- Denson, T. F., Spanovic, M. and Miller, N. (2009) ‘Cognitive appraisals and emotions predict cortisol and immune responses: a meta-analysis of acute laboratory social stressors and emotion inductions’, *Psychological bulletin*, 135(6), pp. 823–853.
- Depping, M. K., & Freund, A. M. (2011). Normal aging and decision making: The role of motivation. *Human Development*, 54(6), 349–367.
- Depue, R. A. and Collins, P. F. (1999) ‘Neurobiology of the structure of personality: dopamine, facilitation of incentive motivation, and extraversion’, *The Behavioral and brain sciences*, 22(3), pp. 491–517; discussion 518–69.

- Deutch, A. Y. and Roth, R. H. (1990) 'The determinants of stress-induced activation of the prefrontal cortical dopamine system', *Progress in brain research*, 85, pp. 367–402; discussion 402–3.
- DeRijk, R.H., Wu<sup>st</sup>, S., Meijer, O.C., Zennaro, M.C., Federenko, I.S., Hellhammer, D.H., Giacchetti, G., Vreugdenhil, E., Zitman, F.G., de Kloet, E.R., (2006). A common polymorphism in the mineralocorticoid receptor modulates stress responsiveness. *J. Clin. Endocrinol. Metab.* 91, 5083—5089.
- Desa, A. et al. (2014) 'A study of the Relationship and Influence of Personality on Job Stress among Academic Administrators at a University', *Procedia - Social and Behavioral Sciences*, pp. 355–359. doi:10.1016/j.sbspro.2013.12.711.
- Dhingra, I., Zhang, S., Zhornitsky, S., Wang, W., Le, T. M., Li, C. R. (2021). Sex differences in neural responses to reward and the influences of individual reward and punishment sensitivity. *BMC Neuroscience*, 22, 12.
- Dias-Ferreira, E., Sousa, J.C., Melo, I., Morgado, P., Mesquita, A.R., Cerqueira, J.J., et al. (2009). Chronic stress causes frontostriatal reorganization and affects decision-making. *Science*, 325(5940), 621–625.
- Di Chiara, G., Loddo, P., Tanda, G. (1999). "Reciprocal changes in prefrontal and limbic dopamine responsiveness to aversive and rewarding stimuli after chronic mild stress: implications for the psychobiology of depression." *Biol. Psychiatry*, 46, 1624–1633. [https://doi.org/10.1016/S0006-3223\(99\)00236-X](https://doi.org/10.1016/S0006-3223(99)00236-X).
- Dickerson, S.S., Kemeny, M.E., (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol. Bull.* 130, 355–391.
- Ding, Y., Wang, E., Zou, Y., Song, Y., Xiao, X., Huang, W., et al. (2017). Gender differences in reward and punishment for monetary and social feedback in children: an ERP study. *PLoS ONE*, 12(3), e0174100.

- Dobbs, L. K. et al. (2016) 'Dopamine Regulation of Lateral Inhibition between Striatal Neurons Gates the Stimulant Actions of Cocaine', *Neuron*, 90(5), pp. 1100–1113.
- Dobson, P. (2000) 'An Investigation into the Relationship between Neuroticism, Extraversion and Cognitive Test Performance in Selection', *International Journal of Selection and Assessment*, pp. 99–109. doi: 10.1111/1468-2389.00140.
- Douma, E. H. and de Kloet, E. R. (2020) 'Stress-induced plasticity and functioning of ventral tegmental dopamine neurons', *Neuroscience and biobehavioral reviews*, 108, pp. 48–77.
- Driscoll, I., Davatzikos, C., An, Y., Wu, X., Shen, D., Kraut, M., & Resnick, S. M. (2009). 'Longitudinal pattern of regional brain volume change differentiates normal aging from MCI.' *Neurology*, 72(22), 1906–1913.
- Driskell, J.E., Salas, E., (1991). Group decision-making under stress. *J. Appl. Psychol.* 76, 473–478.
- Duckworth, AL 2011, 'The significance of self-control', *Proceedings of the National Academy of Sciences - PNAS*, vol. 108, no. 7, pp. 2639–2640.
- Duma, D., Collins, J.B., Chou, J.W., Cidlowski, J.A., 2010. Sexually dimorphic actions of glucocorticoids provide a link to inflammatory diseases with gender differences in prevalence. *Sci. Signal.* 3, ra74. <https://doi.org/10.1126/scisignal.2001077>.
- Dumitru, V M & Cozman, Doina (2012), 'The relationship between stress and personality factors', *Human & Veterinary Medicine*, vol. 4, no. 1, pp. 34–39.
- Earle, T.L., Linden, W., Weinberg, J., 1999. Differential effects of harassment on cardiovascular and salivary cortisol stress reactivity and recovery in women and men. *J. Psychosom. Res.* 46, 125—141.
- Eneva, K. T., Murray, S., O'Garro-Moore, J., Yiu, A., Alloy, L. B., Avena, N. M., & Chen, E. Y. (2017). Reward and punishment sensitivity and disordered eating behaviors in men

- and women. *Journal of Eating Disorders*, 5(1), 6. <https://doi.org/10.1186/s40337-017-0138-2>
- Eng, H.Y., Chen, D. and Jiang, Y. (2010) 'Visual working memory for simple and complex visual stimuli', *Journal of Vision*, pp. 611–611. doi:10.1167/5.8.611.
- Epel, E. S. et al. (2004) 'Accelerated telomere shortening in response to life stress', *Proceedings of the National Academy of Sciences of the United States of America*, 101(49), pp. 17312–17315.
- Eppinger, B., Schuck, N. W., Nystrom, L. E., & Cohen, J. D. (2013). 'Reduced striatal responses to reward prediction errors in older compared with younger adults.' *The Journal of Neuroscience*, 33(24), 9905–9912.  
<http://dx.doi.org/10.1523/JNEUROSCI.2942-12.2013>.
- Eppinger, B., Hämmerer, D., & Li, S.-C. (2011). Neuromodulation of reward-based learning and decision making in human aging. *Annals of the New York Academy of Sciences*, 1235(1), 1–17. <http://dx.doi.org/10.1111/j.1749-6632.2011.06230.x>.
- Eppinger, B., & Kray, J. (2011). To choose or to avoid: age differences in learning from positive and negative feedback. *Journal of Cognitive Neuroscience*, 23(1), 41–52.  
<http://dx.doi.org/10.1162/jocn.2009.21364>.
- Epstein, S. and Meier, P. (1989). Constructive thinking: a broad coping variable with specific components. *Journal of personality and social psychology*, 57(2):332–350.
- Eriega EG, Chidozie IG, Tunde OT, Adebunmi WA (2014). Personality and demographic factors as correlates of post-traumatic stress disorder (ptsd) among flood victims. *Br J Educ*; 2:82-88.
- Ernst, M., Romeo, R. D. and Andersen, S. L. (2009) 'Neurobiology of the development of motivated behaviors in adolescence: a window into a neural systems model', *Pharmacology, biochemistry, and behavior*, 93(3), pp. 199–211.

- Ershler, W. B., Sun, W. H. and Binkley, N. (1994) 'The role of interleukin-6 in certain age-related diseases', *Drugs & aging*, 5(5), pp. 358–365.
- Etkin, A. and Wager, T. D. (2007) 'Functional neuroimaging of anxiety: a meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia', *The American journal of psychiatry*, 164(10), pp. 1476–1488.
- Evans, K. L., & Hampson, E. (2015). Sex-dependent effects on tasks assessing reinforcement learning and interference inhibition. *Frontiers in Psychology*, 6, 1044.
- Evans W. (1986) Personality and stress. *Pers Individ Dif*, 7:251-253.
- Eysenck, M.W. et al. (2007) 'Anxiety and cognitive performance: Attentional control theory', *Emotion*, pp. 336–353. doi:10.1037/1528-3542.7.2.336.
- van der Feltz-Cornelis, C. M. et al. (2019) 'Adverse Childhood Experiences (ACE) in outpatients with anxiety and depressive disorders and their association with psychiatric and somatic comorbidity and revictimization. Cross-sectional observational study', *Journal of affective disorders*, 246, pp. 458–464.
- Fanselow, M. S., & Lester, L. S. (1988). A functional behavioristic approach to aversively motivated behavior: Predatory imminence as a determinant of the topography of defensive behavior. In R. C. Bolles & M. D. Beecher (Eds.), *Evolution and learning* (pp. 185–212). Hillsdale, NJ: Erlbaum.
- Farde, L., Petter Gustavsson, J. and Jönsson, E. (1997) 'D2 dopamine receptors and personality traits', *Nature*, pp. 590–590. doi: 10.1038/385590a0.
- Fienberg, A. A. et al. (1998) 'DARPP-32: regulator of the efficacy of dopaminergic neurotransmission', *Science*, 281(5378), pp. 838–842.
- Fink, G. (2019) *Stress: Physiology, Biochemistry, and Pathology: Handbook of Stress Series, Volume 3*. Academic Press.
- Fink, G. (2007). *The Encyclopedia of Stress*. Academic Press, San Diego, CA, USA, 2nd ed edition.

- Finlay, J. M. and Zigmond, M. J. (1997) 'The effects of stress on central dopaminergic neurons: possible clinical implications', *Neurochemical research*, 22(11), pp. 1387–1394.
- Finlay, J.M., Zigmond, M.J., Abercrombie, E.D. (1995). "Increased dopamine and norepinephrine release in medial prefrontal cortex induced by acute and chronic stress: effects of diazepam." *Neuroscience*, 64, 619–628. [https://doi.org/10.1016/0306-4522\(94\)00331-X](https://doi.org/10.1016/0306-4522(94)00331-X).
- Fiore, V. G., Mannella, F., Mirolli, M., Latagliata, E. C., Valzania, A., Cabib, S., Dolan, R. J., Puglisi-Allegra, S., & Baldassarre, G. (2015). Corticolimbic catecholamines in stress: a computational model of the appraisal of controllability. *Brain Structure & Function*, 220, 1339–1353. <https://doi.org/10.1007/s00429-014-0727-7>.
- Fitzgerald, L., Ortiz, J., Hamedani, A., & Nestler, E. (1996). Drugs of abuse and stress increase the expression of GluR1 and NMDAR1 glutamate receptor subunits in the rat ventral tegmental area: common adaptations among cross-sensitizing agents. *The Journal of Neuroscience*, 16(1), 274–282.
- Flaa, A. et al. (2007) 'Personality may influence reactivity to stress', *BioPsychoSocial Medicine*, p. 5. doi:10.1186/1751-0759-1-5.
- Francis, T. C. and Lobo, M. K. (2017) 'Emerging Role for Nucleus Accumbens Medium Spiny Neuron Subtypes in Depression', *Biological psychiatry*, 81(8), pp. 645–653.
- Frank, M. J. and Claus, E. D. (2006) 'Anatomy of a decision: striato-orbitofrontal interactions in reinforcement learning, decision-making, and reversal', *Psychological review*, 113(2), pp. 300–326.
- Frank, M. J., & Kong, L. (2008). Learning to avoid in older age. *Psychology and Aging*, 23(2), 392–398. doi:10.1037/0882-7974.23.2.392.
- Frank, MJ, Seeberger, LC & O'Reilly, RC (2004), 'By carrot or by stick: Cognitive reinforcement learning in parkinsonism', *Science*, vol. 306, pp. 1940–1943.

- Frederick, A. L. and Stanwood, G. D. (2009) 'Drugs, biogenic amine targets and the developing brain', *Developmental neuroscience*, 31(1-2), pp. 7–22.
- Friston, K. (2005) 'A theory of cortical responses', *Philosophical Transactions of the Royal Society B: Biological Sciences*, pp. 815–836. doi:10.1098/rstb.2005.1622.
- Funahashi, S. (2017). Working memory in the prefrontal cortex. *Brain Sci.* 7:49. doi: 10.3390/brainsci7050049
- Gaab, J. et al. (2005) 'Psychological determinants of the cortisol stress response: the role of anticipatory cognitive appraisal', *Psychoneuroendocrinology*, 30(6), pp. 599–610.
- Gathmann, B. et al. (2014) 'Stress and decision-making: neural correlates of the interaction between stress, executive functions, and decision-making under risk', *Experimental brain research. Experimentelle Hirnforschung. Experimentation cerebrale*, 232(3), pp. 957–973.
- Gao, T. et al. (2020) 'Neural mechanisms of reinforcement learning under mortality threat', *Social neuroscience*, 15(2), pp. 170–185.
- Gerfen CR, Engber TM, Mahan LC, Susel Z, Chase TN, et al. (1990) 'D1 and D2 dopamine receptor-regulated gene expression of striatonigral and striatopallidal neurons', *Science* 250(4986):1429–32.
- Gerfen, C. R. and Surmeier, D. J. (2011) 'Modulation of striatal projection systems by dopamine', *Annual review of neuroscience*, 34, pp. 441–466.
- Georgiou, P., Zanos, P., Bhat, S., Tracy, J.K., Merchant, I.J., McCarthy, M.M., Gould, T.D., 2018. Dopamine and stress system modulation of sex differences in decision-making. *Neuropsychopharmacology* 43, 313–324.  
<https://doi.org/10.1038/npp.2017.161>.
- Gillies, G.E., Virdee, K., McArthur, S., Dalley, J.W., 2014. Sex-dependent diversity in ventral tegmental dopaminergic neurons and developmental programming: a molecular, cellular

and behavioral analysis. *Neuroscience* 282, 69–85.

<https://doi.org/10.1016/j.neuroscience.2014.05.033>.

- Glicksohn, J. and Zilberman, N. (2010) ‘Gambling on individual differences in decision-making’, *Personality and Individual Differences*, 48(5), pp. 557–562.
- Glimcher, P. W. (2011) ‘Understanding dopamine and reinforcement learning: the dopamine reward prediction error hypothesis’, *Proceedings of the National Academy of Sciences of the United States of America*, 108 Suppl 3, pp. 15647–15654.
- Goel, N., Workman, J.L., Lee, T.T., Innala, L., Viau, V., 2014. Sex differences in the HPA axis. *Comprehensive Physiology*. John Wiley & Sons, Inc., Hoboken, NJ, USA, pp. 1121–1155. <https://doi.org/10.1002/cphy.c130054>.
- Golden, S.A., Covington, H.E., Berton, O., Russo, S.J., (2011). A standardized protocol for repeated social defeat stress in mice. *Nat. Protoc.* 6, 1183–1191. <https://doi.org/10.1038/nprot.2011.361>.
- Goldman-Rakic, P. S. et al. (2004) ‘Targeting the dopamine D1 receptor in schizophrenia: insights for cognitive dysfunction’, *Psychopharmacology*, 174(1), pp. 3–16.
- Good, C. D., Johnsrude, I. S., Ashburner, J., Henson, R. N., Friston, K. J., & Frackowiak, R. S. (2001). 'A voxel-based morphometric study of ageing in 465 normal adult human brains.' *NeuroImage*, 14(1 Pt 1), 21–36.
- Gosling, S. D., Rentfrow, P. J. and Swann, W. B. (2003) ‘A very brief measure of the Big-Five personality domains’, *Journal of Research in Personality*, 37(6), pp. 504–528.
- Gremel, C. M. and Costa, R. M. (2013) ‘Orbitofrontal and striatal circuits dynamically encode the shift between goal-directed and habitual actions’, *Nature Communications*. doi:10.1038/ncomms3264.
- Gresch, P.J., Sved, A.F., Zigmond, M.J., Finlay, J.M. (2002). "Stress-induced sensitization of dopamine and norepinephrine efflux in medial prefrontal cortex of the rat." *J. Neurochem.*, 63, 575–583. <https://doi.org/10.1046/j.1471-4159.1994.63020575.x>.



- Grieve, S. M., Williams, L. M., Paul, R. H., Clark, C. R., & Gordon, E. (2007). Cognitive aging, executive function, and fractional anisotropy: A diffusion tensor MR imaging study. *American Journal of Neuroradiology*, 28(2), 226–235. Retrieved from [http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list\\_uids=17296985](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=17296985)
- Grillon, C. and Charney, D.R. (2011) 'In the face of fear: anxiety sensitizes defensive responses to fearful faces', *Psychophysiology*, 48(12), pp. 1745–1752.
- Guarraci, F.A., Kapp, B.S. (1999). "An electrophysiological characterization of ventral tegmental area dopaminergic neurons during differential pavlovian fear conditioning in the awake rabbit." *Behav. Brain Res.*, 99, 169–179. [https://doi.org/10.1016/S0166-4328\(98\)00102-8](https://doi.org/10.1016/S0166-4328(98)00102-8).
- Gurven M, Von Rueden C, Massenkoff M, Kaplan H, Lero Vie M (2013). How universal is the Big Five? Testing the five-factor model of personality variation among forager-farmers in the Bolivian Amazon. *J Pers Soc Psychol*; 104:354-370.
- Hämmerer, D., Li, S.-C., Müller, V., & Lindenberger, U. (2011). 'Life span differences in electrophysiological correlates of monitoring gains and losses during probabilistic reinforcement learning.' *Journal of Cognitive Neuroscience*, 23(3), 579-592.
- Harden, K. P., Mann, F. D., Grotzinger, A. D., Patterson, M. W., Steinberg, L., Tackett, J. L., & Tucker-Drob, E. M. (2018). Developmental differences in reward sensitivity and sensation seeking in adolescence: Testing sex-specific associations with gonadal hormones and pubertal development. *Journal of Personality and Social Psychology*, 115(1), 161–178. <https://doi.org/10.1037/pspp0000172>
- Harvard Health Publishing (2020) Understanding the stress response. Available at: <https://www.health.harvard.edu/staying-healthy/understanding-the-stress-response> (Accessed: 06 02 2022).

- Hauner KK, Adam EK, Mineka S, Doane LD, DeSantis AS, Zinbarg R, et al. (2008) Neuroticism and introversion are associated with salivary cortisol patterns in adolescents. *Psychoneuroendocrinology*;33:1344-1356.
- Hauser DJ, Norbert Schwarz (2016) Attentive Turkers: MTurk participants perform better on online attention checks than do subject pool participants. *Behavior Research Methods* 48(1): 400–407.
- Hays, R. D. et al. (1995) User's Manual for the Medical Outcomes Study (MOS) Core Measures of Health-related Quality of Life. RAND Corporation.
- Hensler, J. G. et al. (2013) 'Catecholamine/Serotonin interactions: systems thinking for brain function and disease', *Advances in pharmacology*, 68, pp. 167–197.
- Herbison, C. E. et al. (2017) 'The impact of life stress on adult depression and anxiety is dependent on gender and timing of exposure', *Development and psychopathology*, 29(4), pp. 1443–1454.
- Hernaus, D., Collip, D., Lataster, J., Ceccarini, J., Kenis, G., Booij, L., Pruessner, J., Van Laere, K., van Winkel, R., van Os, J., Myin-Germeys, I., 2013. COMT Val158Met genotype selectively alters prefrontal [<sup>18</sup>F]fallypride displacement and subjective feelings of stress in response to a psychosocial stress challenge. *PLOS ONE* 8, e65662.
- Hernaus, D., Mehta, M.A., 2015. Prefrontal cortex dopamine release measured in vivo with positron emission tomography: Implications for the stimulant paradigm. *NeuroImage*.
- Hikosaka, O., Takikawa, Y. and Kawagoe, R. (2000) 'Role of the basal ganglia in the control of purposive saccadic eye movements', *Physiological reviews*, 80(3), pp. 953–978.
- Hines, E.A., Brown, G.E., (1932). A standard stimulus for measuring vasomotor reactions: its application in the study of hypertension. *Proc. Staff Meet. Mayo Clin.* 7, 332.
- Hintiryan, H. et al. (2016) 'The mouse cortico-striatal projectome', *Nature neuroscience*, 19(8), pp. 1100–1114.

- Holly, E. N. and Miczek, K. A. (2016) 'Ventral tegmental area dopamine revisited: effects of acute and repeated stress', *Psychopharmacology*, 233(2), pp. 163–186.
- Holly, E. N., DeBold, J. F., & Miczek, K. A. (2015). Increased mesocorticolimbic dopamine during acute and repeated social defeat stress: modulation by corticotropin-releasing factor receptors in the ventral tegmental area. *Psychopharmacology (Berl)*, 232(24), 4469–4479.
- Hooks, B. M. et al. (2018) 'Topographic precision in sensory and motor corticostriatal projections varies across cell type and cortical area', *Nature communications*, 9(1), p. 3549.
- Horger, B. A. and Roth, R. H. (1996) 'The role of mesoprefrontal dopamine neurons in stress', *Critical reviews in neurobiology*, 10(3-4), pp. 395–418.
- Horowitz, M., Wilmer, N., Alvarez, W., 1979. Impact for Event Scale: a measure of subjective stress. *Psychosom. Med.* 41, 209–218.
- Horvitz, J. C. (2009) 'Stimulus-response and response-outcome learning mechanisms in the striatum', *Behavioural brain research*, 199(1), pp. 129–140.
- Hughes, B. M. et al. (2011) 'Individual differences in adaptation of cardiovascular responses to stress', *Biological psychology*, 86(2), pp. 129–136.
- Huh, M. J. et al. (2013) 'The impact of personality traits on ratings of obsessive-compulsive symptoms', *Psychiatry investigation*, 10(3), pp. 259–265.
- Ikemoto, S. (2007) 'Dopamine reward circuitry: two projection systems from the ventral midbrain to the nucleus accumbens-olfactory tubercle complex', *Brain research reviews*, 56(1), pp. 27–78.
- Imperato, A., Puglisi-Allegra, S., Casolini, P., & Angelucci, L. (1991). Changes in brain dopamine and acetylcholine release during and following stress are independent of the pituitary-adrenocortical axis. *Brain Res.*, 538, 111–117. [https://doi.org/10.1016/0006-8993\(91\)90384-8](https://doi.org/10.1016/0006-8993(91)90384-8).

- Imperato, A., Angelucci, L., Casolini, P., Zocchi, A., & Puglisi-Allegra, S. (1992). Repeated stressful experiences differently affect limbic dopamine release during and following stress. *Brain Res*, 577, 194–199.
- Imperato, A., Cabib, S., & Puglisi-Allegra, S. (1993). Repeated stressful experiences differently affect the time-dependent responses of the mesolimbic dopamine system to the stressor. *Brain Res*, 601, 333–336.
- Inukai K, Shinada M, Tanida S, Takahashi C, Mifune N, Takagishi H, et al. (2010) Salivary alpha-amylase levels and big five personality factors in adults. *Neuroendocrinol Lett*;31:771-774.
- Jackson, M.C., Linden, D.E.J. and Raymond, J.E. (2014) ‘Angry expressions strengthen the encoding and maintenance of face identity representations in visual working memory’, *Cognition & emotion*, 28(2), pp. 278–297.
- Jacobson, A., Green, E., & Murphy, C. (2010). Age-related functional changes in gustatory and reward processing regions: An fMRI study. *NeuroImage*, 53(2), 602–610.
- Joels, M., Baram, T.Z., (2009). The neuro-symphony of stress. *Nat. Rev. Neurosci.* 10, 459–466.
- Joels, M., Pu, Z., Wiegert, O., Oitzl, M.S., Krugers, H.J. (2006). "Learning under stress: How does it work?" *Trends Cogn Sci*, 10, 152–158.
- Jonassaint, C. R. et al. (2009) ‘The effects of Neuroticism and Extraversion on cardiovascular reactivity during a mental and an emotional stress task’, *International Journal of Psychophysiology*, pp. 274–279. doi: 10.1016/j.ijpsycho.2009.09.012.
- Jordan, S., Kramer, G. L., Zukas, P. K., & Petty, F. (1994). Previous stress increases in vivo biogenic amine response to swim stress. *Neurochemical Research*, 19(12), 1521–1525.
- Judge, T. A. et al. (2002) ‘Are measures of self-esteem, neuroticism, locus of control, and generalized self-efficacy indicators of a common core construct?’, *Journal of personality and social psychology*, 83(3), pp. 693–710.

- Kaasinen, V. et al. (2004) 'Insular dopamine D2 receptors and novelty seeking personality in Parkinson's disease', *Movement disorders: official journal of the Movement Disorder Society*, 19(11), pp. 1348–1351.
- Kajantie, E. and Phillips, D. I. W. (2006) 'The effects of sex and hormonal status on the physiological response to acute psychosocial stress', *Psychoneuroendocrinology*, 31(2), pp. 151–178.
- Kamin LJ. (1969) 'Selective association and conditioning', *Fundamental Issues in Associative Learning*, ed. NJ Mackintosh, WK Honig, pp. 42–64. Halifax, NS: Dalhousie Univ. Press
- Kandasamy, N. et al. (2014) 'Cortisol shifts financial risk preferences', *Proceedings of the National Academy of Sciences of the United States of America*, 111(9), pp. 3608–3613.
- Kanner, A. D. et al. (1981) 'Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events', *Journal of Behavioral Medicine*, pp. 1–39. doi: 10.1007/bf00844845.
- Karrer, T. M., Josef, A. K., Mata, R., Morris, E. D., & Samanez-Larkin, G. R. (2017). 'Reduced dopamine receptors and transporters but not synthesis capacity in normal aging adults: A meta-analysis.' *Neurobiology of Aging*, 57, 36–46.
- Kaska, S., Brunk, R., Kechner, M., & Mazei-Robison, M. (2017). Regulation of cytoskeletal remodeling proteins in the ventral tegmental area by morphine, stress, and TORC2. *FASEB J.*, 31, 985.12.
- Kaur, R., Chodagiri, V. K. and Reddi, N. K. (2013) 'A psychological study of stress, personality and coping in police personnel', *Indian journal of psychological medicine*, 35(2), pp. 141–147.

- Kaufling, J. (2019). "Alterations and adaptation of ventral tegmental area dopaminergic neurons in animal models of depression." *Cell Tissue Res.*, 377(1), 59–71.  
<https://doi.org/10.1007/s00441-019-03007-9>.
- Kavaliers, M., & Choleris, E. (2001). Antipredator responses and defensive behavior: Ecological and ethological approaches for the neurosciences. *Neuroscience & Biobehavioral Reviews*, 25, 577–586.
- Keinan, G., 1987. Decision-making under stress: scanning of alternatives under controllable and uncontrollable threats. *J. Pers. Soc. Psychol.* 52, 639–644.
- Kendler, K. S., Kuhn, J. and Prescott, C. A. (2004) 'The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression', *The American journal of psychiatry*, 161(4), pp. 631–636.
- Kennedy, K. M., Erickson, K. I., Rodrigue, K. M., Voss, M. W., Colcombe, S. J., Kramer, A. F., ... Raz, N. (2009). 'Age-related differences in regional brain volumes: A comparison of optimized voxel-based morphometry to manual volumetry.' *Neurobiology of Aging*, 30(10), 1657–1676.
- Kessler, R. C. et al. (2010) 'Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys', *The British journal of psychiatry: the journal of mental science*, 197(5), pp. 378–385.
- Kessler, R.C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K.R., Rush, A.J., Walters, E.E., Wang, P.S., 2003. The epidemiology of major depressive disorder. *Am. Med. Assoc.* 289, 3095–3105.
- Kestler, L. P. et al. (2000) 'The relation between dopamine D2 receptor density and personality: preliminary evidence from the NEO personality inventory-revised', *Neuropsychiatry, neuropsychology, and behavioral neurology*, 13(1), pp. 48–52.
- Killgore, W. D. and Yurgelun-Todd, D. A. (2001) 'Sex differences in amygdala activation during the perception of facial affect', *Neuroreport*, 12(11), pp. 2543–2547.

- Kim, EJ & Kim, JJ. (2023), 'Neurocognitive effects of stress: a metaparadigm perspective', *Molecular Psychiatry*, vol. 28, no. 7, pp. 2750–2763.
- Kim, K. M. et al. (2012) 'Optogenetic mimicry of the transient activation of dopamine neurons by natural reward is sufficient for operant reinforcement', *PloS one*, 7(4), p. e33612.
- Kim, S. and Lee, D. (2011) 'Prefrontal cortex and impulsive decision-making', *Biological psychiatry*, 69(12), pp. 1140–1146.
- Kim, H. et al. (2009) 'Role of striatum in updating values of chosen actions', *The Journal of neuroscience: the official journal of the Society for Neuroscience*, 29(47), pp. 14701–14712.
- Kim, H.-G., Cheon, E.-J., Bai, D.-S., Lee, Y. H., & Koo, B.-H. (2018). Stress and Heart Rate Variability: A Meta-Analysis and Review of the Literature. *Psychiatry Investigation*, 15(3), 235–245.
- Kirschbaum, C., Wuist, S., Faig, H.G., Hellhammer, D.H., (1992b). Heritability of cortisol responses to human corticotropin-releasing hormone, ergometry, and psychological stress in humans. *J. Clin. Endocrinol. Metab.* 75, 1526—1530.
- Kirschbaum, C., Hellhammer, D.H., (1994). Salivary cortisol in psychoneuroendocrine research: recent developments and applications. *Psychoneuroendocrinology* 19, 313–333.
- Kirschbaum, C., Pirke, K.M., Hellhammer, D.H., (1993). The 'Trier Social Stress Test'—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology* 28, 76–81.
- Kirschbaum, Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, 61(2), 154–162.  
<https://doi.org/10.1097/00006842-199903000-00006>

- Kivimäki, M. et al. (2006) 'Work stress in the etiology of coronary heart disease—a meta-analysis', *Scandinavian Journal of Work, Environment & Health*, pp. 431–442. doi: 10.5271/sjweh.1049.
- Klaus, A., Alves da Silva, J. and Costa, R. M. (2019) 'What, If, and When to Move: Basal Ganglia Circuits and Self-Paced Action Initiation', *Annual review of neuroscience*, 42, pp. 459–483.
- Knock, & Fehr, E. (2007). Resisting the Power of Temptations: The Right Prefrontal Cortex and Self-Control. *Annals of the New York Academy of Sciences*, 1104(1), 123–134. <https://doi.org/10.1196/annals.1390.004>
- Kobasa, S. C. (1979). Stressful life events, personality, and health: an inquiry into hardiness. *Journal of personality and social psychology*, 37(1):1–11.
- Kollack-Walker, S., Watson, S. J. and Akil, H. (1997) 'Social stress in hamsters: defeat activates specific neurocircuits within the brain', *The Journal of neuroscience: the official journal of the Society for Neuroscience*, 17(22), pp. 8842–8855.
- Koizumi, A, Mobbs, D & Lau, H (2016), 'Is fear perception special? Evidence at the level of decision-making and subjective confidence', *Social Cognitive and Affective Neuroscience*, vol. 11, no. 11, pp. 1772–1782.
- Kravitz, A. V. et al. (2010) 'Regulation of parkinsonian motor behaviours by optogenetic control of basal ganglia circuitry', *Nature*, 466(7306), pp. 622–626.
- Krishnan, V., Nestler, E.J., (2011). Animal models of depression: molecular perspectives. In: Hagan, J.J. (Ed.), *Molecular and Functional Models in Neuropsychiatry*. Springer, pp. 121–147. [https://doi.org/10.1007/7854\\_2010\\_108](https://doi.org/10.1007/7854_2010_108).
- Krishnan, V., Berton, O., & Nestler, E.J. (2008a). The use of animal models in psychiatric research and treatment. *American Journal of Psychiatry*, 165, 1109. <https://doi.org/10.1176/appi.ajp.2008.08071076>.



- Kudielka, B.M., Hellhammer, D.H., Wüst, S., (2009). Why do we respond so differentially? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology* 34, 2–18.
- Kudielka, B.M., Hellhammer, D.H., Kirschbaum, C., 2007b. Sex differences in human stress response. In: Fink, G. (Ed.), *Encyclopedia of Stress*, 2nd revised ed., vol. 3. Academic Press, Oxford, pp. 469—473.
- Kudielka, B. M. and Kirschbaum, C. (2005) ‘Sex differences in HPA axis responses to stress: a review’, *Biological Psychology*, pp. 113–132. doi: 10.1016/j.biopsycho.2004.11.009.
- Kulak, A. et al. (2013) ‘Redox dysregulation in the pathophysiology of schizophrenia and bipolar disorder: insights from animal models’, *Antioxidants & redox signaling*, 18(12), pp. 1428–1443.
- Kumsta, R. et al. (2007a) ‘Cortisol and ACTH responses to psychosocial stress are modulated by corticosteroid binding globulin levels’, *Psychoneuroendocrinology*, 32(8-10), pp. 1153–1157.
- Kumsta, R., Entringer, S., Koper, J.W., van Rossum, E.F., Hellhammer, D.H., Wüst, S., 2007b. Sex specific associations between common glucocorticoid receptor gene variants and hypothalamus—pituitary—adrenal axis responses to psychosocial stress. *Biol. Psychiatry* 62, 863—869.
- Laakso, A. et al. (2003) ‘Personality traits and striatal dopamine synthesis capacity in healthy subjects’, *The American journal of psychiatry*, 160(5), pp. 904–910
- Laceulle OM, Nederhof E, van Aken MA, Ormel J. (2015) Adolescent personality: Associations with basal, awakening, and stress-induced cortisol responses. *J Pers*;83:262-273.
- La Fratta, I., Tatangelo, R., Campagna, G., Rizzuto, A., Franceschelli, S., Ferrone, A., et al., 2018. The plasmatic and salivary levels of IL-1 $\beta$ , IL-18 and IL-6 are associated to

- emotional difference during stress in young male. *Sci. Rep.*  
<https://doi.org/10.1038/S41598-018-21474-y>.
- Lammel, S., Lim, B. K. and Malenka, R. C. (2014a) 'Reward and aversion in a heterogeneous midbrain dopamine system', *Neuropharmacology*, pp. 351–359. doi: 10.1016/j.neuropharm.2013.03.019.
- Lammel, S., Tye, K.M., Warden, M.R. (2014b). "Progress in understanding mood disorders: optogenetic dissection of neural circuits." *Genes Brain Behav.*, 13, 38–51.  
<https://doi.org/10.1111/gbb.12049>.
- Landau, S. M., Lal, R., O'Neil, J. P., Baker, S., & Jagust, W. J. (2009). Striatal dopamine and working memory. *Cerebral Cortex*, 19, 445–454.
- Lane, R. D., Reiman, E. M., Ahern, G. L., & Thayer, J. F. (2001). Activity in medial prefrontal cortex correlates with vagal component of heart rate variability during emotion. *Brain and Cognition*, 47, 97–100.
- Larsen, RJ & Ketelaar, T 1991, 'Personality and Susceptibility to Positive and Negative Emotional States', *Journal of Personality and Social Psychology*, vol. 61, no. 1, pp. 132–140.
- Lataster, J., Collip, D., Ceccarini, J., Haas, D., Booij, L., van Os, J., Pruessner, J., VanLaere, K., Myin-Germeys, I., 2011. Psychosocial stress is associated with in vivo dopamine release in human ventromedial prefrontal cortex: a positron emission tomography study using [(1)(8)F]fallypride. *NeuroImage* 58,1081–1089.
- Lau, B. and Glimcher, P. W. (2008) 'Value representations in the primate striatum during matching behavior', *Neuron*, 58(3), pp. 451–463.
- Lazarus, R. S. (1993) From psychological stress to the emotions: a history of changing outlooks. *Annual review of psychology*, 44:1–21.
- Lazarus, RS (1966), *Psychological stress and the coping process*, McGraw-Hill, New York.
- Lazarus, RS (1999), *Stress and emotion a new synthesis*, Springer, New York.

- Lazarus, R.S. & Folkman, S. (1984), *Stress, appraisal, and coping*, Springer Pub. Co., New York.
- Le, T. M., Wang, W., Zhornitsky, S., Dhingra, I., Zhang, S., Li, C.-S. R. (2019). Reward sensitivity and electrodermal responses to actions and outcomes in a go/no-go task. *PLoS One*, 14, e0219147.
- LeDoux, J. and Daw, N. D. (2018) ‘Surviving threats: neural circuit and computational implications of a new taxonomy of defensive behaviour’, *Nature reviews. Neuroscience*, 19(5), pp. 269–282.
- Lee-Baggley, D., Preece, M., & DeLongis, A. (2005). Coping with interpersonal stress: Role of big five traits. *Journal of Personality*, 73, 1141–1180.
- Lee, I. H. et al. (2005) ‘Correlation between striatal dopamine D2 receptor density and neuroticism in community volunteers’, *Psychiatry research*, 138(3), pp. 259–264.
- Lee, D., Seo, H. and Jung, M. W. (2012) ‘Neural Basis of Reinforcement Learning and Decision-making’, *Annual Review of Neuroscience*, pp. 287–308. doi: 10.1146/annurev-neuro-062111-150512.
- Levi, L. (1972) ‘Stress and Distress in Response to Psychosocial Stimuli’, Pergamon Press, New York.
- Levy, I & Schiller, D (2021) ‘Neural Computations of Threat’, *Trends in Cognitive Sciences*, vol. 25, no. 2, pp. 151–171.
- Lempert, K.M. et al. (2012) ‘Individual differences in delay discounting under acute stress: the role of trait perceived stress’, *Frontiers in psychology*, 3, p. 251.
- Lewis, A.H., Porcelli, A.J. and Delgado, M.R. (2014) ‘The effects of acute stress exposure on striatal activity during Pavlovian conditioning with monetary gains and losses’, *Frontiers in behavioral neuroscience*, 8, p. 179.
- Li, N. et al. (2015) ‘A motor cortex circuit for motor planning and movement’, *Nature*, 519(7541), pp. 51–56.

- Lighthall, N. R., Gorlick, M. A., Schoeke, A., Frank, M. J., & Mather, M. (2013). Stress modulates reinforcement learning in younger and older adults. *Psychology and Aging*, 28(1), 35–46. <http://dx.doi.org/10.1037/a0029823>.
- Lim, B. K., Huang, K. W., Grueter, B. A., Rothwell, P. E., & Malenka, R. C. (2012). Anhedonia requires MC4R-mediated synaptic adaptations in nucleus accumbens. *Nature*, 487, 183–189.
- Lipka, J., Miltner, W. H. R. and Straube, T. (2011) ‘Vigilance for threat interacts with amygdala responses to subliminal threat cues in specific phobia’, *Biological psychiatry*, 70(5), pp. 472–478.
- Lloyd, K., Dayan, P. (2016). "Safety out of control: dopamine and defence." *Behav. Brain Funct.*, 12, 15. <https://doi.org/10.1186/s12993-016-0099-7>.
- Lovaglio, W. R. and Pincomb, G. (1990) Heart rate reactivity as a predictor of neuroendocrine responses to aversive and appetitive challenges. *Psychosomatic medicine*, 52(1):17–26.
- Lovaglio, W. R. and Thomas, T. L. (2000). Stress hormones in psychophysiological research. In Cacioppo, J. T., Tassinary, L. G., and Berntson, G. G., editors, *Handbook of Psychophysiology*, pages 342–367. Cambridge University Press, 2nd edition.
- Lovaglio, W.R., Farag, N.H., Vincent, A.S., Thomas, T.L., Wilson, M.F., 2006. Cortisol responses to mental stress, exercise, and meals following caffeine intake in men and women. *Pharmacol. Biochem. Behav.* 83, 441—447.
- Love, T.M., Enoch, M.A., Hodgkinson, C.A., Pecina, M., Mickey, B., Koeppe, R.A., Stohler, C.S., Goldman, D., Zubieta, J.K., (2012). Oxytocin gene polymorphisms influence human dopaminergic function in a sex-dependent manner. *Biol. Psychiatry* 72, 198–206.
- Lovibond, S. H. and Lovibond, P. F. (1996) *Manual for the Depression Anxiety Stress Scales*.

- Lucas, L. R. et al. (2004) 'Repeated exposure to social stress has long-term effects on indirect markers of dopaminergic activity in brain regions associated with motivated behavior', *Neuroscience*, 124(2), pp. 449–457.
- Luecken, L.J., Appelhans, B.M., (2006). Early parental loss and salivary cortisol in young adulthood: the moderating role of family environment. *Dev. Psychopathol.* 18, 295–308.
- Lueken, U. et al. (2009) 'Altered tonic and phasic cortisol secretion following unilateral stroke', *Psychoneuroendocrinology*, 34(3), pp. 402–412.
- Luethi, M., Merier, B., Sandi, C. (2008). "Stress effects on working memory, explicit memory, and implicit memory for neutral and emotional stimuli in healthy men." *Frontiers in Behav Neurosci*, 2, 1–9.
- Luksys, G., & Sandi, C. (2011). Neural mechanisms and computations underlying stress effects on learning and memory. *Current Opinion in Neurobiology*, 21, 502–508.
- Lupien, S.J., Maheu, F., Tu, M., Fiocco, A., Schramek, T.E., 2007. The effects of stress and stress hormones on human cognition: implications for the field of brain and cognition. *Brain Cogn.* 65, 209–237.
- Madden, D. J., Costello, M. C., Dennis, N. A., Davis, S. W., Shepler, A. M., Spaniol, J., . . . Cabeza, R. (2010). Adult age differences in functional connectivity during executive control. *Neuroimage*, 52(2), 643–657. doi:10.1016/j.neuroimage.2010.04.249.
- Maia, T. V. and Frank, M. J. (2011) 'From reinforcement learning models to psychiatric and neurological disorders', *Nature Neuroscience*, pp. 154–162. doi: 10.1038/nn.2723.
- Maier, SU, Makwana, AB & Hare, TA (2015), 'Acute Stress Impairs Self-Control in Goal-Directed Choice by Altering Multiple Functional Connections within the Brain's Decision Circuits', *Neuron* (Cambridge, Mass.), vol. 87, no. 3, pp. 621–631.

- Mazei, M. S. et al. (2002) 'Effects of catecholamine uptake blockers in the caudate-putamen and subregions of the medial prefrontal cortex of the rat', *Brain Research*, pp. 58–67. doi: 10.1016/s0006-8993(02)02542-8.
- Makino, S., Smith, M. A. and Gold, P. W. (2002) 'Regulatory role of glucocorticoids and glucocorticoid receptor mRNA levels on tyrosine hydroxylase gene expression in the locus coeruleus during repeated immobilization stress', *Brain research*, 943(2), pp. 216–223.
- Mantz, J., Thierry, A.M., Glowinski, J. (1989). "Effect of noxious tail pinch on the discharge rate of mesocortical and mesolimbic dopamine neurons: selective activation of the mesocortical system." *Brain Res.*, 476, 377–381. [https://doi.org/10.1016/0006-8993\(89\)91263-8](https://doi.org/10.1016/0006-8993(89)91263-8).
- Marinelli, M. and Piazza, P. V. (2002) 'Interaction between glucocorticoid hormones, stress and psychostimulant drugs', *The European journal of neuroscience*, 16(3), pp. 387–394.
- Mason, J. W. (1968) A Review of Psychoendocrine Research on the Pituitary-Adrenal Cortical System. *Psychosomatic medicine*, 30(5):576–607.
- Mata, R., Josef, A. K., Samanez-Larkin, G. R., & Hertwig, R. (2011). Age differences in risky choice: A meta-analysis. *Annals of the New York Academy of Sciences*, 1235(1), 18–29. <http://dx.doi.org/10.1111/j.1749-6632.2011.06200.x>.
- Mather, M., Gorlick, M.A., Lighthall, N.R., (2009). To brake or accelerate when the light turns yellow? Stress reduces older adults' risk taking in a driving game. *Psychol. Sci.* 20, 174–176.
- Mather, M., & Lighthall, N. R. (2012). Risk and reward are processed differently in decisions made under stress. *Current Directions in Psychological Science*, 21(1), 36–41.

- Mather, M. and Sutherland, M.R. (2011) 'Arousal-Biased Competition in Perception and Memory', *Perspectives on psychological science: a journal of the Association for Psychological Science*, 6(2), pp. 114–133.
- Matuskey, D., Worhunksy, P., Correa, E., Pittman, B., Gallezot, J. D., Nabulsi, N., ... Cosgrove, K. (2016). 'Age-related changes in binding of the D2/3 receptor radioligand 11C PHNO in healthy volunteers.' *NeuroImage*, 130, 241–247.
- Meyer-Lindenberg, A., Kohn, P. D., Kolachana, B., Kippenhan, S., McInerney-Leo, A., Nussbaum, R., et al. (2005). Midbrain dopamine and prefrontal function in humans: Interaction and modulation by COMT genotype. *Nature Neuroscience*, 8, 594–596.
- McEwen, B.S. (2008). "Central effects of stress hormones in health and disease, Understanding the protective and damaging effects of stress and stress mediators." *Eur. J. Pharmacol.*, 583, 174–185.
- McEwen, B.S., Sapolsky, R.M., (1995). Stress and cognitive function. *Curr. Opin. Neurobiol.* 5, 205–216.
- McEwen, B.S. and Morrison, J.H. (2013) 'The brain on stress: vulnerability and plasticity of the prefrontal cortex over the life course', *Neuron*, 79(1), pp. 16–29.
- McCrae, R. R. and Costa, P. T., Jr (1997) 'Personality trait structure as a human universal', *The American psychologist*, 52(5), pp. 509–516.
- Meaney, M. J., Bhatnagar, S., Larocque, S., McCormick, C., Shanks, N., Sharma, S., Smythe, J., Viau, V., and Plotsky, P. M. (1993). Individual differences in the hypothalamic-pituitary-adrenal stress response and the hypothalamic CRF system. *Annals of the New York Academy of Sciences*, 697:70–85.
- Mehta, MA, Manes, FF, Magnolfi, G, Sahakian, BJ & Robbins, TW (2004), 'Impaired set-shifting and dissociable effects on tests of spatial working memory following the dopamine D2 receptor antagonist sulpiride in human volunteers', *Psychopharmacology (Berl)*, vol. 176, pp. 331–342.

- Mickey, B.J., Sanford, B.J., Love, T.M., Shen, P.H., Hodgkinson, C.A., Stohler, C.S., Goldman, D., Zubieta, J.K., 2012. Striatal dopamine release and genetic variation of the serotonin 2C receptor in humans. *J. Neurosci.: Off. J. Soc. Neurosci.* 32, 9344–9350.
- Miller, G. E. et al. (2008) 'A functional genomic fingerprint of chronic stress in humans: blunted glucocorticoid and increased NF-kappaB signaling', *Biological psychiatry*, 64(4), pp. 266–272.
- Miller, Cohen, S., Rabin, B. S., Skoner, D. P., & Doyle, W. J. (1999). Personality and Tonic Cardiovascular, Neuroendocrine, and Immune Parameters. *Brain, Behavior, and Immunity*, 13(2), 109–123. <https://doi.org/10.1006/brbi.1998.0545>
- Mirenowicz, J., Schultz, W. (1996). "Preferential activation of midbrain dopamine neurons by appetitive rather than aversive stimuli." *Nature*, 379, 449–451. <https://doi.org/10.1038/379449a0>.
- Miu, A.C., Heilman, R.M., Houser, D., (2008). Anxiety impairs decision-making: psychophysiological evidence from an Iowa gambling task. *Biol. Psychol.* 77, 353–358.
- Mizoguchi, K., Ishige, A., Takeda, S., Aburada, M., & Tabira, T. (2004). Endogenous glucocorticoids are essential for maintaining prefrontal cortical cognitive function. *Journal of Neuroscience*, 24, 5492–5499.
- Mobbs, D., Hagan, C. C., Dalgleish, T., Silston, B., & Prévost, C. (2015). The ecology of human fear: Survival optimization and the nervous system. *Frontiers in Neuroscience*, 9, 55.
- Mogg, K. and Bradley, B. P. (1999) 'Orienting of Attention to Threatening Facial Expressions Presented under Conditions of Restricted Awareness', *Cognition & Emotion*, pp. 713–740. doi: 10.1080/026999399379050.



- Moghaddam, B & Jackson, M (2004), 'Effect of stress on prefrontal cortex function', *Neurotoxicity Research*, vol. 6, no. 1, pp. 73–78.
- Moghaddam, B. (2002) 'Stress activation of glutamate neurotransmission in the prefrontal cortex: implications for dopamine-associated psychiatric disorders', *Biological psychiatry*, 51(10), pp. 775–787.
- Monroe, S. M. et al. (2007) 'Major life events and major chronic difficulties are differentially associated with history of major depressive episodes', *Journal of abnormal psychology*, 116(1), pp. 116–124.
- Montague, P. R., Dayan, P. and Sejnowski, T. J. (1996) 'A framework for mesencephalic dopamine systems based on predictive Hebbian learning', *The Journal of neuroscience: the official journal of the Society for Neuroscience*, 16(5), pp. 1936–1947.
- Montgomery, A.J., Mehta, M.A., Grasby, P.M., 2006. Is psychological stress in man associated with increased striatal dopamine levels? A [<sup>11</sup>C]raclopride PET study. *Synapse (New York, N.Y.)* 60, 124–131.
- Moore, K.E., Lariviere, E.W., 1964. Effects of stress and d-amphetamine on rat brain catecholamines. *Biochem. Pharmacol.* 13, 1098–1100.
- Mosely, J. V., & Linden, W. (2006). Predicting blood pressure and heart rate change with cardiovascular reactivity and recovery: Results from a 3-year and 10-year follow-up. *Psychosomatic Medicine*, 68, 833–843.
- Muir, J. et al. (2018) 'In Vivo Fiber Photometry Reveals Signature of Future Stress Susceptibility in Nucleus Accumbens', *Neuropsychopharmacology: official publication of the American College of Neuropsychopharmacology*, 43(2), pp. 255–263.
- Murphy, E., Sved, A., Finlay, J. (2003). "Corticotropin-releasing hormone receptor blockade fails to alter stress-evoked catecholamine release in prefrontal cortex of control or

- chronically stressed rats." *Neuroscience*, 116, 1081–1087.  
[https://doi.org/10.1016/S0306-4522\(02\)00565-1](https://doi.org/10.1016/S0306-4522(02)00565-1).
- Naef, L., Gratton, A., & Walker, C. D. (2013). Exposure to high fat during early development impairs adaptations in dopamine and neuroendocrine responses to repeated stress. *Stress*, 16(5), 540–548.
- Nagano-Saito, A., Dagher, A., Booij, L., Gravel, P., Welfeld, K., Casey, K.F., Leyton, M., Benkelfat, C., (2013). Stress-induced dopamine release in human medial prefrontal cortex – 18F-fallypride/PET study in healthy volunteers. *Synapse*(New York, N.Y.) 67, 821–830.
- Nakajima, S., Caravaggio, F., Boileau, I., Chung, J. K., Plitman, E., Gerretsen, P., ... Graff-Guerrero, A. (2015). 'Lack of age-dependent decrease in dopamine D3 receptor availability: A [11C]-(+)-PHNO and [11C]-raclopride positron emission tomography study.' *Journal of Cerebral Blood Flow & Metabolism*, 35(11), 1812–1818.
- Nater, U.M., Rohleder, N., (2009). Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: current state of research. *Psychoneuroendocrinology* 34, 486–496.
- Nater, Hoppmann, C., & Klumb, P. L. (2010). Neuroticism and conscientiousness are associated with cortisol diurnal profiles in adults—Role of positive and negative affect. *Psychoneuroendocrinology*, 35(10), 1573–1577.  
<https://doi.org/10.1016/j.psyneuen.2010.02.017>
- Navratilova, E., Xie, J.Y., Okun, A., Qu, C., Eyde, N., Ci, S., Ossipov, M.H., King, T., Fields, H.L., Porreca, F. (2012). "Pain relief produces negative reinforcement through activation of mesolimbic reward-valuation circuitry." *Proc. Natl. Acad. Sci. U. S. A.*, 109, 20709–20713. <https://doi.org/10.1073/pnas.1214605109>.
- Nicolson, N., Storms, C., Ponds, R., Sulon, J., 1997. Salivary cortisol levels and stress reactivity in human aging. *J. Gerontol. A: Biol. Sci. Med. Sci.* 52, M68—M75.

- Norbeck, J., (1984). Modification of life events questionnaires for use with female respondents. *Res. Nurs. Health* 7, 61–71.
- Noushad, S., Ahmed, S., Ansari, B., Mustafa, U-H., Saleem, Y., & Hazrat, H. (2021). Physiological biomarkers of chronic stress: A systematic review. *International Journal of Health Sciences*, 15(5), 46–59.
- Ossewaarde, L., Qin, S., Van Marle, H. J., van Wingen, G. A., Fernandez, G., & Hermans, E. J. (2011). Stress-induced reduction in reward-related prefrontal cortex function. *Neuroimage*, 55(1), 345–352.
- Otte, C. et al. (2005) ‘A meta-analysis of cortisol response to challenge in human aging: importance of gender’, *Psychoneuroendocrinology*, 30(1), pp. 80–91.
- Oliver, G., Wardle, J., Gibson, E.L., 2000. Stress and food choice: a laboratory study. *Psychosom. Med.* 62, 853–865.
- Oswald LM, Zandi P, Nestadt G, Potash JB, Kalaydjian AE, Wand GS. (2006) Relationship between cortisol responses to stress and personality. *Neuropsychopharmacology*;31:15831591.
- Ouanes S, Castelao E, von Gunten A, Vidal M, Preisig M, Popp J. (2017) Personality, cortisol, and cognition in non-demented elderly subjects: results from a population-based study. *Front Aging Neuroscience*
- Ozer, D. J. and Benet-Martínez, V. (2006) ‘Personality and the prediction of consequential outcomes’, *Annual review of psychology*, 57, pp. 401–421.
- Pacheco-Unguetti, A. P. et al. (2010) ‘Attention and anxiety: different attentional functioning under state and trait anxiety’, *Psychological science*, 21(2), pp. 298–304.
- Padoa-Schioppa, C. and Assad, J. A. (2006) ‘Neurons in the orbitofrontal cortex encode economic value’, *Nature*, 441(7090), pp. 223–226.
- Pagliaccio, D., Luking, K. R., Anokhin, A. P., Gotlib, I. H., Hayden, E. P., Olino, T. M., Peng, C., Hajcak, G., & Barch, D. M. (2016). Revising the BIS/BAS scale to study

- development: Measurement invariance and normative effects of age and sex from childhood through adulthood. *Psychological Assessment*, 28(4), 429–442.  
<https://doi.org/10.1037/pas0000186>
- Parent-Lamarche A, Marchand A. (2015) The moderating role of personality traits in the relationship between work and salivary cortisol: a cross-sectional study of 401 employees in 34 Canadian companies. *BMC Psychol*;3:45.
- Patchev, V.K. et al. (1995) ‘Implications of estrogen-dependent brain organization for gender differences in hypothalamo-pituitary-adrenal regulation’, *FASEB journal: official publication of the Federation of American Societies for Experimental Biology*, 9(5), pp. 419–423.
- Patten, S. B. et al. (2015) ‘Retrospective and prospectively assessed childhood adversity in association with major depression, alcohol consumption and painful conditions’, *Epidemiology and psychiatric sciences*, 24(2), pp. 158–165.
- Patton, J. H., Stanford, M.S. and Barratt, E. S. (1995) ‘Factor structure of the barratt impulsiveness scale’, *Journal of Clinical Psychology*, 51(6), pp. 768–774.
- Parker, J. G. et al. (2018) ‘Diametric neural ensemble dynamics in parkinsonian and dyskinetic states’, *Nature*, 557(7704), pp. 177–182.
- Peciña, Love, T., Stohler, C. S., Goldman, D., & Zubieta, J.-K. (2015). Effects of the Mu opioid receptor polymorphism (OPRM1 A118G) on pain regulation, placebo effects and associated personality trait measures. *Neuropsychopharmacology (New York, N.Y.)*, 40(4), 957–965. <https://doi.org/10.1038/npp.2014.272>
- Petty, F., Jordan, S., Kramer, G. L., Zukas, P. K., & Wu, J. (1997). Benzodiazepine prevention of swim stress-induced sensitization of cortical biogenic amines: an in vivo microdialysis study. *Neurochemical Research*, 22(9), 1101–1104.
- Pezze, M. A. and Feldon, J. (2004) ‘Mesolimbic dopaminergic pathways in fear conditioning’, *Progress in neurobiology*, 74(5), pp. 301–320.

- Pfefferbaum, A., Sullivan, E. V., Rosenbloom, M. J., Mathalon, D. H., & Lim, K. O. (1998). 'A controlled study of cortical gray matter and ventricular changes in alcoholic men over a 5-year interval.' *Archives of General Psychiatry*, 55(10), 905–912.
- Piazza, P. V. and Le Moal, M. (1998) 'The role of stress in drug self-administration', *Trends in Pharmacological Sciences*, pp. 67–74. doi: 10.1016/s0165-6147(97)01115-2.
- Pidoux, M. et al. (2011) 'Integration and propagation of somatosensory responses in the corticostriatal pathway: an intracellular study in vivo', *The Journal of physiology*, 589(Pt 2), pp. 263–281.
- Pieper, S., & Brosschot, J. F. (2005). Prolonged stress-related cardiovascular activation: Is there any? *Annals of Behavioral Medicine*, 30, 91–103.
- Planert, H., Berger, T. K. and Silberberg, G. (2013) 'Membrane properties of striatal direct and indirect pathway neurons in mouse and rat slices and their modulation by dopamine', *PloS one*, 8(3), p. e57054.
- Porcelli, A. J. and Delgado, M. R. (2017) 'Stress and Decision-making: Effects on Valuation, Learning, and Risk-taking', *Current opinion in behavioral sciences*, 14, pp. 33–39.
- Porcelli, A.J., Delgado, M.R., (2009). Acute stress modulates risk taking in financial decision-making. *Psychol. Sci.* 20, 278–283.
- Porcelli, A.J., Lewis, A.H. and Delgado, M.R. (2012) 'Acute stress influences neural circuits of reward processing', *Frontiers in neuroscience*, 6, p. 157.
- Pourtois, G., Schettino, A. and Vuilleumier, P. (2013) 'Brain mechanisms for emotional influences on perception and attention: what is magic and what is not', *Biological psychology*, 92(3), pp. 492–512.
- Preston, S.D., Buchanan, T.W., Stansfield, R.B., Bechara, A., 2007. Effects of anticipatory stress on decision-making in a gambling task. *Behav. Neurosci.* 121, 257–263.

- Pruessner, J., Baldwin, M.W., Dedovic, K., Renwick, R., Mahani, N.K., Lord, C., Meaney, M., Lupien, S.J., (2005). Self-esteem, locus of control, hippocampal volume, and cortisol regulation in young and old adulthood. *Neuroimage* 28, 815–826.
- Pruessner, J., Champagne, F., Meanes, M.J., Dagher, A., (2004). Dopamine release in response to a psychological stress in humans and its relationship to early life maternal care: a positron emission tomography study using [11C] raclopride. *J. Neurosci.* 24, 2825–2831.
- Pruessner, J.C., Dedovic, K., Khalili-Mahani, N., Engert, V., Pruessner, M., Buss, C., et al., (2008). Deactivation of the limbic system during acute psychosocial stress: evidence from positron emission tomography and functional magnetic resonance imaging studies. *Biol. Psychiatry*, 63.
- Pruessner, JC, Dedovic, K, Pruessner, M, Lord, C, Buss, C, Collins, L, ... Lupien, SJ, (2010), ‘Stress regulation in the central nervous system: evidence from structural and functional neuroimaging studies in human populations - 2008 Curt Richter Award Winner’, *Psychoneuroendocrinology*, vol. 35, no. 1, pp. 179–191.
- Putman, P., Antypa, N., Crysovergi, P., van der Does, W.A.J., (2010) Exogenous cortisol acutely influences motivated decision-making in healthy young men. *Psychopharmacology (Berl.)* 208, 257–263.
- Pycock, C. J., Kerwin, R. W., & Carter, C. J. (1980). Effect of lesion of cortical dopamine terminals on sub-cortical dopamine-receptors in rats. *Nature*, 286, 74–77.
- Qi, Song (2020) Decision-making Under Threat: An Ecological Framework. Dissertation (Ph.D.), California Institute of Technology. doi:10.7907/p7j5-bw96.  
<https://resolver.caltech.edu/CaltechTHESIS:04242020-001419374>
- Radley, J.J., Rocher, A.B., Miller, M., Janssen, W.G.M., Liston, C., Hof, P.R., McEwen, B.S., Morrison, J.H., (2006). Repeated stress induces dendritic spine loss in the rat medial prefrontal cortex. *Cereb. Cortex* 16, 313–320. <https://doi.org/10.1093/cercor/bhi104>.

- Raz, N., (2000). Aging of the brain and its impact on cognitive performance: integration of structural and functional findings. In: Craik, F.I.M., Salthouse, T.A. (Eds.), *The Handbook of Aging and Cognition*. Lawrence Erlbaum Associates, Mahwah, NJ, pp. 1–90.
- Raz, N., Ghisletta, P., Rodrigue, K. M., Kennedy, K. M., & Lindenberger, U. (2010). 'Trajectories of brain aging in middle-aged and older adults: Regional and individual differences.' *NeuroImage*, 51(2), 501–511.
- Raz, N., Lindenberger, U., Rodrigue, K. M., Kennedy, K. M., Head, D., Williamson, A., ... Acker, J. D. (2005). 'Regional brain changes in aging healthy adults: General trends, individual differences and modifiers.' *Cerebral Cortex*, 15(11), 1676–1689.
- Reig, R. and Silberberg, G. (2014) 'Multisensory integration in the mouse striatum', *Neuron*, 83(5), pp. 1200–1212.
- Rescorla R.A., Wagner A. (1972). A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. In *Classical Conditioning II: Current Research and Theory*, ed. AH Black, WF Prokasy, pp. 64–99. New York: Appleton-Century-Crofts
- Resnick, S. M., Pham, D. L., Kraut, M. A., Zonderman, A. B., & Davatzikos, C. (2003). 'Longitudinal magnetic resonance imaging studies of older adults: A shrinking brain.' *Journal of Neuroscience*, 23(8), 3295–3301.
- Revelle, W. (2022) *psych: Procedures for Personality and Psychological Research*, Northwestern University, Evanston, Illinois, USA, <https://CRAN.R-project.org/package=psych> Version = 2.2.5.
- Rhodes, M. E. and Rubin, R. T. (1999) 'Functional sex differences ('sexual diergism') of central nervous system cholinergic systems, vasopressin, and hypothalamic–pituitary–

- adrenal axis activity in mammals: a selective review', *Brain research reviews*, 30(2), pp. 135–152.
- Ridker PM, Rifai N, Stampfer MJ, Hennekens CH. Plasma Concentration of Interleukin-6 and the Risk of Future Myocardial Infarction Among Apparently Healthy Men. *Circulation*. (2000); 101(15):1767–72. <https://doi.org/10.1161/01.Cir.101.15.1767>. PMID: 10769275
- Rincón-Cortés, M., Grace, A.A. (2017). "Sex-dependent effects of stress on immobility behavior and VTA dopamine neuron activity: modulation by ketamine." *Int. J. Neuropsychopharmacol.*, 20, 823–832. <https://doi.org/10.1093/ijnp/pyx048>.
- Rmus, M, McDougle, SD & Collins, AG (2021), 'The role of executive function in shaping reinforcement learning', *Current Opinion in Behavioral Sciences*, vol. 38, pp. 66–73
- Robbins, S. B., Lauver, K., Le, H., Davis, D., Langley, R., and Carlstrom, A. (2004). Do psychosocial and study skill factors predict college outcomes? A meta-analysis. *Psychological bulletin*, 130(2), 261–288. doi: 10.1037/0033-2909.130.2.261
- Roberts, B. W. et al. (2007) 'The Power of Personality: The Comparative Validity of Personality Traits, Socioeconomic Status, and Cognitive Ability for Predicting Important Life Outcomes', *Perspectives on psychological science: a journal of the Association for Psychological Science*, 2(4), pp. 313–345.
- Roger, D & Najarian, B 1998, 'The relationship between emotional rumination and cortisol secretion under stress', *Personality and Individual Differences*, vol. 24, no. 4, pp. 531–538.
- Rohleder, N. (2014) 'Stimulation of systemic low-grade inflammation by psychosocial stress', *Psychosomatic medicine*, 76(3), pp. 181–189.
- Rohleder, N., Nater, U.M., (2009). Determinants of salivary alpha-amylase in humans and methodological considerations. *Psychoneuroendocrinology* 34, 469–485.



- Rohleder, N., Schommer, N.C., Hellhammer, D.H., Engel, R., Kirschbaum, C., 2001. Sex differences in glucocorticoid sensitivity of pro-inflammatory cytokine production after psychosocial stress. *Psychosom. Med.* 63, 966—972.
- Rohleder, N., Wolf, J.M., Piel, M., Kirschbaum, C., 2003. Impact of oral contraceptive use on glucocorticoid sensitivity of pro-inflammatory cytokine production after psychosocial stress. *Psychoneuroendocrinology* 28, 261—273.
- Rolland, J.-P. (2002). The Cross-Cultural Generalizability of the Five-Factor Model of Personality. *The Five-Factor Model of Personality Across Cultures*, 7–28.  
doi:10.1007/978-1-4615-0763-5\_2
- Roosendaal, B., McEwen, B.S., Chattarji, S., 2009. Stress, memory and the amygdala. *Nat. Rev. Neurosci.* 10, 423–433.
- Rosenbaum, M. (1989). Self-control under stress: The role of learned resourcefulness. *Advances in Behaviour Research and Therapy*, 11(4):249–258.
- Russell WM. (2017) *Leadership Skills and Stress* (Doctoral dissertation). Industrial/Organizational Psychology. San Angelo, Texas: Angelo State University.
- Sadegh-Nejadi S, Afrisham R, Soliemanifar O, Kooti W, Aberomand M, Najjar-Asl S, et al. (2017) Alteration of the level of salivary cortisol under psychological stress and its relationship with rumination and personality traits. *J Gorgan Univ Med Sci*
- Salamone, J. D., Cousins, M. S., & Snyder, B. J. (1997). Behavioral functions of nucleus accumbens dopamine: empirical and conceptual problems with the anhedonia hypothesis. *Neuroscience and Biobehavioral Reviews*, 21, 341–359.  
[https://doi.org/10.1016/S0149-7634\(96\)00017-6](https://doi.org/10.1016/S0149-7634(96)00017-6).
- Salleh, Mohd Razali. (2008). “Life Event, Stress and Illness.” *The Malaysian Journal of Medical Sciences: MJMS* 15 (4): 9–18.
- Salthouse, T.A. 1994, 'The Aging of Working Memory', *Neuropsychology*, vol. 8, no. 4, pp. 535–543.

- Samanez-Larkin, G. R., Gibbs, S. E. B., Khanna, K., Nielsen, L., Carstensen, L. L., & Knutson, B. (2007). Anticipation of monetary gain but not loss in healthy older adults. *Nature Neuroscience*, 10(6), 787–791. <http://dx.doi.org/10.1038/nn1894>.
- Samanez-Larkin, G. R., Worthy, D. A., Mata, R., McClure, S. M., & Knutson, B. (2014). Adult age differences in frontostriatal representation of prediction error but not reward outcome. *Cognitive, Affective, & Behavioral Neuroscience*, 14(2), 672–682. <http://dx.doi.org/10.3758/s13415-014-0297-4>.
- Santiago, P. H., Nielsen Lisa, T., Smithers, G., & Roberts, R. (2020). Measuring stress in Australia: validation of the perceived stress scale (PSS-14) in a national sample. *Health and Quality of Life Outcomes*, 18-100. doi:<https://doi.org/10.1186/s12955-020-01343-x>
- Sapolsky, R. M. (1994). Individual differences and the stress response. *Seminars in Neuroscience*, 6(4):261–269.
- Sapolsky, R.M., 1994. *Why Zebra's Don't Get Ulcers: A Guide to Stress, Stress-related Diseases and Coping*. W.H. Freeman and Company, New York.
- Sapolsky, R.M., Krey, L.C., McEwen, B.S., 1986. The neuroendocrinology of stress and aging: the glucocorticoid cascade hypothesis. *Endocr. Rev.* 7, 284—301.
- Sapolsky, R. M. (1996). Why stress is bad for your brain. *Science*, 273, 749–750.
- Schneiderman, N., Ironson, G., Siegel, S.D. (2005). "Stress and health: psychological, behavioral, and biological determinants." *Annu. Rev. Clin. Psychol.*, 1, 607–628.
- Schreuders, E., Braams, B. R., Blankenstein, N. E., Peper, J. S., Guroglu, B., & Crone, E. A. (2018). Contributions of reward sensitivity to ventral striatum activity across adolescence and early adulthood. *Child Development*, 89(3), 797–810. <https://doi.org/10.1111/cdev.13056>
- Seeman, T.E., Singer, B., Wilkinson, C.W., McEwen, B., 2001. Gender differences in age-related changes in HPA axis reactivity. *Psychoneuroendocrinology* 26, 225—240.

- Seeman, T. E. et al. (1995) 'Self-esteem and neuroendocrine response to challenge: MacArthur studies of successful aging', *Journal of psychosomatic research*, 39(1), pp. 69–84.
- Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, 130, 601– 630.
- Segerstrom, S. C., & Solberg-Nes, L. (2007). Heart rate variability reflects self-regulatory strength, effort, and fatigue. *Psychological Science*, 18, 275–281.
- Selye, H. (1956) *The Stress of Life*. New York, McGraw-Hill Book Company, Inc. 1956. \$5.95', *The Journal of Bone & Joint Surgery*, p. 479. doi: 10.2106/00004623-195739020-00034.
- Seo, M., Lee, E. and Averbeck, B. B. (2012) 'Action selection and action value in frontal-striatal circuits', *Neuron*, 74(5), pp. 947–960.
- Shackman, A. J. et al. (2006) 'Anxiety selectively disrupts visuospatial working memory', *Emotion* , 6(1), pp. 40–61.
- Shalev, I. et al. (2013) 'Stress and telomere biology: a lifespan perspective', *Psychoneuroendocrinology*, 38(9), pp. 1835–1842.
- Shansky, R.M., Rubinow, K., Brennan, A., Arnsten, A.F.T. (2006). "The effects of sex and hormonal status on restraint-stress-induced working memory impairment." *Behav Brain Funct*, 2, 8.
- Shen, W. et al. (2008) 'Dichotomous dopaminergic control of striatal synaptic plasticity', *Science*, 321(5890), pp. 848–851.
- Shonkoff, J. P. et al. (2012) 'The lifelong effects of early childhood adversity and toxic stress', *Pediatrics*, 129(1), pp. e232–46.
- Shors, T.J., Chua, C. and Falduto, J. (2001) 'Sex differences and opposite effects of stress on dendritic spine density in the male versus female hippocampus', *The Journal of*

- neuroscience: the official journal of the Society for Neuroscience, 21(16), pp. 6292–6297.
- Shors, T.J., Weiss, C., Thompson, R.F. (1992). "Stress-induced facilitation of classical conditioning." *Science*, 257, 537–539.
- Simon, J. R., Howard, J. H., & Howard, D. V. (2010a). 'Adult age differences in learning from positive and negative probabilistic feedback.' *Neuropsychology*, 24(4), 534-541.
- Sinha, R., Catapano, D. and O'Malley, S. (1999) 'Stress-induced craving and stress response in cocaine dependent individuals', *Psychopharmacology*, 142(4), pp. 343–351.
- Scheier, M. F. and Carver, C. S. (1985) 'Optimism, coping, and health: assessment and implications of generalized outcome expectancies', *Health psychology: official journal of the Division of Health Psychology, American Psychological Association*, 4(3), pp. 219–247.
- Scheier, M. F. and Carver, C. S. (1987). Dispositional optimism and physical well-being: the influence of generalized outcome expectancies on health. *Journal of personality*, 55(2):169–210.
- Scheres, A., & Sanfey, A. G. (2006). Individual differences in decision making: Drive and reward responsiveness affect strategic bargaining in economic games. *Behavioral and Brain Functions: BBF*, 2(1), 35. <https://doi.org/10.1186/1744-9081-2-35>
- Schommer, N.C., Hellhammer, D.H., Kirschbaum, C., (2003). Dissociation between reactivity of the hypothalamus–pituitary–adrenal axis and the sympathetic–adrenal–medullary system to repeated psychosocial stress. *Psychosom. Med.* 65, 450–460.
- Schultz, W. (2015) 'Neuronal Reward and Decision Signals: From Theories to Data', *Physiological reviews*, 95(3), pp. 853–951.
- Schultz, W., Dayan, P. and Montague, P. R. (1997) 'A neural substrate of prediction and reward', *Science*, 275(5306), pp. 1593–1599.

- Schultz, W. and Romo, R. (1992) 'Role of primate basal ganglia and frontal cortex in the internal generation of movements. I. Preparatory activity in the anterior striatum', *Experimental brain research. Experimentelle Hirnforschung. Experimentation cerebrale*, 91(3), pp. 363–384.
- Schultz, W., Romo, R. (1987). "Responses of nigrostriatal dopamine neurons to high-intensity somatosensory stimulation in the anesthetized monkey." *J. Neurophysiol.*, 57, 201–217.
- Schwabe, L., Haddad, L., Schachinger, H., (2008). HPA axis activation by a socially evaluated cold-pressor test. *Psychoneuroendocrinology* 33, 890–895.
- Schwabe, L. et al. (2012) 'Simultaneous Glucocorticoid and Noradrenergic Activity Disrupts the Neural Basis of Goal-Directed Action in the Human Brain', *Journal of Neuroscience*, pp. 10146–10155. doi:10.1523/jneurosci.1304-12.2012.
- Schwabe, L. and Wolf, O.T. (2009) 'Stress Prompts Habit Behavior in Humans', *Journal of Neuroscience*, pp. 7191–7198. doi:10.1523/jneurosci.0979-09.2009.
- Schwabe, L. and Wolf, O.T. (2010) 'Socially evaluated cold pressor stress after instrumental learning favors habits over goal-directed action', *Psychoneuroendocrinology*, 35(7), pp. 977–986.
- Schwartz, A. R., Gerin, W., Davidson, K. W., Pickering, T. G., Brosschot, J. F., Thayer, J. F., et al. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine*, 65, 22–35.
- Schwebel DC, Suls J. (1999) Cardiovascular reactivity and neuroticism: Results from a laboratory and controlled ambulatory stress protocol. *J Pers*;67:67-92.
- Scott, D.J., Heitzeg, M.M., Koeppe, R.A., Stohler, C.S., Zubieta, J.K., 2006. Variations in the human pain stress experience mediated by ventral and dorsal basal ganglia dopamine activity. *J. Neurosci.: Off. J. Soc. Neurosci.* 26, 10789–10795.

- Shin, L.M. et al. (2005) 'A functional magnetic resonance imaging study of amygdala and medial prefrontal cortex responses to overtly presented fearful faces in posttraumatic stress disorder', *Archives of general psychiatry*, 62(3), pp. 273–281.
- Smith, T. W. and MacKenzie, J. (2006) 'Personality and Risk of Physical Illness', *Annual Review of Clinical Psychology*, pp. 435–467. doi: 10.1146/annurev.clinpsy.2.022305.095257.
- Snyder, C. R., Harris, C., Anderson, J. R., Holleran, S. a., Irving, L. M., Sigmon, S. T., Yoshinobu, L., Gibb, J., Langelle, C., and Harney, P. (1991). The will and the ways: development and validation of an individual-differences measure of hope. *Journal of personality and social psychology*, 60(4):570–585.
- Solimanifar, O., Soleymanifar, A. and Afrisham, R. (2018) 'Relationship between Personality and Biological Reactivity to Stress: A Review', *Psychiatry Investigation*, pp. 1100–1114. doi:10.30773/pi.2018.10.14.2.
- Soliman, A., O'Driscoll, G.A., Pruessner, J., Holahan, A.L., Boileau, I., Gagnon, D., Dagher, A., 2008. Stress-induced dopamine release in humans at risk of psychosis: a [11C]raclopride PET study. *Neuropsychopharmacol.: Off. Publ. Am. Coll. Neuropsychopharmacol.* 33, 2033–2041.
- Soutschek, A., Burke, C. J., Raja Beharelle, A., Schreiber, R., Weber, S. C., Karipidis, I. I., ten Velden, J., Weber, B., Haker, H., Kalenscher, T., & Tobler, P. N. (2017). The dopaminergic reward system underpins gender differences in social preferences. *Nature Human Behaviour*, 1(11), 819–827. <https://doi.org/10.1038/s41562-017-0226-y>
- Spielberger, C. D. (2012) 'State-Trait Anxiety Inventory for Adults', *PsycTESTS Dataset*. doi: 10.1037/t06496-000.
- Spreckelmeyer, K. N., Krach, S., Kohls, G., Rademacher, L., Irmak, A., Konrad, K., Kircher, T., Gründer, G. (2009). Anticipation of monetary and social reward differently

- activates mesolimbic brain structures in men and women. *Social Cognitive and Affective Neuroscience*, 4, 158–165.
- Sreenivasan, K.K., Sambhara, D. and Jha, A.P. (2011) ‘Working memory templates are maintained as feature-specific perceptual codes’, *Journal of neurophysiology*, 106(1), pp. 115–121.
- Stalnaker, T. A. et al. (2016) ‘Cholinergic Interneurons Use Orbitofrontal Input to Track Beliefs about Current State’, *The Journal of neuroscience: the official journal of the Society for Neuroscience*, 36(23), pp. 6242–6257.
- Stalnaker, T.A., Espana, R.A., Berridge, C.W., (2009). Coping behavior causes asymmetric changes in neuronal activation in the prefrontal cortex and amygdala. *Synapse* 63, 82–85.
- Stanwood, G.D. (2019) 'Dopamine and Stress', in: Fink, G. (ed.) *Stress: Physiology, Biochemistry, and Pathology*, Academic Press, pp. 105-114. Available at: <https://doi.org/10.1016/B978-0-12-813146-6.00009-6>.
- Starcke, K., Wolf, O. T., Markowitsch, H. J., & Brand, M. (2008). Anticipatory stress influences decision-making under explicit risk conditions, *Behavioral Neuroscience*, 122, 1352–1360.
- Starcke, K. and Brand, M. (2012) ‘Decision-making under stress: A selective review’, *Neuroscience & Biobehavioral Reviews*, pp. 1228–1248. doi: 10.1016/j.neubiorev.2012.02.003.
- Starcke, K., Polzer, C., Wolf, O.T., Brand, M., (2011). Does stress alter everyday moral decision-making? *Psychoneuroendocrinology* 36, 210–219.
- Steinberg, E. E. et al. (2013) ‘A causal link between prediction errors, dopamine neurons and learning’, *Nature neuroscience*, 16(7), pp. 966–973.

- Stelly, CE, Tritley, SC, Rafati, Y & Wanat, MJ, (2020). 'Acute Stress Enhances Associative Learning via Dopamine Signaling in the Ventral Lateral Striatum', *The Journal of Neuroscience*, vol. 40, no. 22, pp. 4391–4400.
- Stelly, C. E., Pomrenze, M. B., Cook, J. B., & Morikawa, H. (2016). Repeated social defeat stress enhances glutamatergic synaptic plasticity in the VTA and cocaine place conditioning. *eLife*, 5, 1–18. <https://doi.org/10.7554/eLife.15448>.
- Steptoe, A., Fieldman, G., Evans, O., Perry, L., 1996. Cardiovascular risk and responsivity to mental stress: the influence of age, gender and risk factors. *J. Cardiovasc. Risk* 3, 83–93.
- Stetler, C., Miller, G.E., (2011). Depression and hypothalamic-pituitary-adrenal activation: a quantitative summary of four decades of research. *Psychosom. Med.* 73, 114–126. <https://doi.org/10.1097/PSY.0b013e31820ad12b>.
- Stewart, A. L., Hays, R. D. and Ware, J. E. (1988), 'The MOS Short-Form General Health Survey: Reliability and Validity in a Patient Population', *Medical Care*, vol. 26, no. 7, pp. 724–735.
- Stewart, J. C., Janicki, D. L., & Kamarck, T. W. (2006). Cardiovascular reactivity and recovery from psychological challenge as predictors of 3-year change in blood pressure. *Health Psychology*, 25, 111–118.
- Stillman, M.J., Shukitt-Hale, B., Levy, A., Lieberman, R. (1998). "Spatial memory under acute cold and restraint stress." *Physiol Behav*, 64, 605–609.
- Stockmeier, C. A., Mahajan, G. J., Konick, L. C., Overholser, J. C., Jurjus, G. J., Meltzer, H. Y., Uylings, H. B. M., Friedman, L., & Rajkowska, G. (2004). Cellular changes in the postmortem hippocampus in major depression. *Biol. Psychiatry*, 56, 640–650. <https://doi.org/10.1016/j.biopsych.2004.08.022>.
- Stout, D.M. et al. (2015) 'Worry is associated with impaired gating of threat from working memory', *Emotion* , 15(1), pp. 6–11.



- Stout, D.M., Shackman, A.J. and Larson, C.L. (2013) 'Failure to filter: anxious individuals show inefficient gating of threat from working memory', *Frontiers in human neuroscience*, 7, p. 58.
- Stroud, L.R., Salovey, P., Epel, E.S., 2002. Sex differences in stress responses: social rejection versus achievement stress. *Biol. Psychiatry* 52, 318—327.
- Straube, T., Mentzel, H.-J. and Miltner, W.H.R. (2005) 'Common and distinct brain activation to threat and safety signals in social phobia', *Neuropsychobiology*, 52(3), pp. 163–168.
- Sugama, S. and Kakinuma, Y. (2016) 'Loss of dopaminergic neurons occurs in the ventral tegmental area and hypothalamus of rats following chronic stress: Possible pathogenetic loci for depression involved in Parkinson's disease', *Neuroscience research*, 111, pp. 48–55.
- Suhara, T, Yasuno, F, Sudo, Y, Yamamoto, M, Inoue, M, Okubo, Y & Suzuki, K (2001), 'Dopamine D2 Receptors in the Insular Cortex and the Personality Trait of Novelty Seeking', *NeuroImage (Orlando, Fla.)*, vol. 13, no. 5, pp. 891–895.
- Sul, J. H. et al. (2010) 'Distinct roles of rodent orbitofrontal and medial prefrontal cortex in decision-making', *Neuron*, 66(3), pp. 449–460.
- Suls, J. and Bunde, J. (2005) 'Anger, anxiety, and depression as risk factors for cardiovascular disease: the problems and implications of overlapping affective dispositions', *Psychological bulletin*, 131(2), pp. 260–300.
- Summerfield, C. et al. (2006) 'Predictive codes for forthcoming perception in the frontal cortex', *Science*, 314(5803), pp. 1311–1314.
- Summerfield, C. and de Lange, F.P. (2014) 'Expectation in perceptual decision-making: neural and computational mechanisms', *Nature reviews. Neuroscience*, 15(11), pp. 745–756.

- Suridjan, I., Boileau, I., Bagby, M., Rusjan, P.M., Wilson, A.A., Houle, S., Mizrahi, R.,(2012). Dopamine response to psychosocial stress in humans and its relationship to individual differences in personality traits. *J. Psychiatr. Res.* 46, 890–897.
- Sussman, T.J. et al. (2016) ‘It’s all in the anticipation: How perception of threat is enhanced in anxiety’, *Emotion*, pp. 320–327. doi:10.1037/emo0000098.
- Sutton, R. S. and Barto, A. G. (2018) *Reinforcement Learning: An Introduction*. A Bradford Book.
- Sutton, R. S. and Barto, A. G. (1981) ‘Toward a modern theory of adaptive networks: expectation and prediction’, *Psychological review*, 88(2), pp. 135–170.
- Svenningsson, P. et al. (2004) ‘DARPP-32: an integrator of neurotransmission’, *Annual review of pharmacology and toxicology*, 44, pp. 269–296.
- Takahashi, T., Ikeda, K., Hasegawa, T., 2007. Social evaluation-induced amylase elevation and economic decision-making in the dictator game in humans. *Neuroendocrinol. Lett.* 28, 662–665.
- Tamás, N. (2015) *Psychophysiological Responses to Distress and Eustress*.
- Tanimoto, H., Heisenberg, M., Gerber, B. (2004). "Experimental psychology: event timing turns punishment to reward." *Nature*, 430, 983. <https://doi.org/10.1038/430983a>.
- Taylor, S.E., Klein, L.C., Lewis, B.P., Gruenewald, T.L., Gurung, R.A., Updegraff, J.A., (2000). Bio-behavioral responses to stress in females: tend-and-befriend, not fight-or-flight. *Psychol. Rev.* 107, 411–429. <https://doi.org/10.1037/0033-295X.107.3.411>.
- Teicher, M. H. et al. (2003) ‘The neurobiological consequences of early stress and childhood maltreatment’, *Neuroscience and biobehavioral reviews*, 27(1-2), pp. 33–44.
- Thayer, J. F., & Lane, R. D. (2007). The role of vagal function in the risk for cardiovascular disease and mortality. *Biological Psychology*, 74, 224–242.

- Thayer, J. F., & Lane, R. D. (2009). Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience & Biobehavioral Reviews*, 33, 81–88.
- Thompson, B. L., Levitt, P. and Stanwood, G. D. (2009) ‘Prenatal exposure to drugs: effects on brain development and implications for policy and education’, *Nature reviews. Neuroscience*, 10(4), pp. 303–312.
- Thura, D. and Cisek, P. (2017) ‘The Basal Ganglia Do Not Select Reach Targets but Control the Urgency of Commitment’, *Neuron*, pp. 1160–1170.e5. doi: 10.1016/j.neuron.2017.07.039.
- Tidey, J.W., Miczek, K.A. (1997). "Acquisition of cocaine self-administration after social stress: role of accumbens dopamine." *Psychopharmacology (Berl.)*, 130, 203–212. <https://doi.org/10.1007/s002130050230>.
- Tidey, J.W., Miczek, K.A. (1996). "Social defeat stress selectively alters mesocorticolimbic dopamine release: an in vivo microdialysis study." *Brain Res.*, 721, 140–149. [https://doi.org/10.1016/0006-8993\(96\)00159-X](https://doi.org/10.1016/0006-8993(96)00159-X).
- Tillfors, M., Furmark, T., Marteinsdottir, I., Fredrikson, M., (2002). Cerebral blood flow during anticipation of public speaking in social phobia: a PET study. *Biol. Psychiatry* 52, 1113–1119.
- Toth, E., Gersner, R., Wilf-Yarkoni, A., Raizel, H., Dar, D. E., Richter-Levin, G., Levit, O., & Zangen, A. (2008). Age-dependent effects of chronic stress on brain plasticity and depressive behavior. *Journal of Neurochemistry*, 107, 522–532. <https://doi.org/10.1111/j.1471-4159.2008.05642.x>.
- Trainor, B. C. (2011) ‘Stress responses and the mesolimbic dopamine system: social contexts and sex differences’, *Hormones and behavior*, 60(5), pp. 457–469.
- Tranel, D., Damasio, H., Denburg, N.L., Bechara, A., 2005. Does gender play a role in functional asymmetry of ventromedial prefrontal cortex? *Brain* 128, 2872–2881.

- Tseng, M. T., Chiang, M. C., Yazhuo, K., Chao, C. C., Tseng, W. Y. I., & Hsieh, S. T. (2013). Effect of aging on the cerebral processing of thermal pain in the human brain. *Pain*, 154(10), 2120–2129.
- Tye, K.M., Mirzabekov, J.J., Warden, M.R., Ferenczi, E.A., Tsai, H.-C., Finkelstein, J., Kim, S.-Y., Adhikari, A., Thompson, K.R., Andalman, A.S., Gunaydin, L.A., Witten, I.B., Deisseroth, K. (2013). Dopamine neurons modulate neural encoding and expression of depression-related behaviour. *Nature*, 493, 537–541.  
<https://doi.org/10.1038/nature11740>.
- Uchino, B. N., Smith, T. W., Holt-Lunstead, J., Campo, R. A., & Reblin, M. (2007). Stress and illness. In J. T. Cacioppo, L. G. Tassinary, & G. G. Bertson (Eds.), *Handbook of psychophysiology* (pp. 608–632). New York: Cambridge University Press.
- Ulrich-Lai, Y. M. and Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature reviews. Neuroscience*, 10(6):397–409.
- Ungless, M.A. (2004). Uniform inhibition of dopamine neurons in the ventral tegmental area by aversive stimuli. *Science (80-.)*, 303, 2040–2042.  
<https://doi.org/10.1126/science.1093360>.
- Urosevic, S., Collins, P., Muetzel, R., Lim, K., & Luciana, M. (2012). Longitudinal changes in behavioral approach system sensitivity and brain structures involved in reward processing during adolescence. *Developmental Psychology*, 48(5), 1488–1500.  
<https://doi.org/10.1037/a0027502>
- Vaessen T, Hernaus D, Myin-Germeys I, van Amelsvoort T. (2015) The dopaminergic response to acute stress in health and psychopathology: a systematic review. *Neurosci Biobehav Rev.*; 56:241e251. <https://doi.org/10.1016/j.neubiorev.2015.07.008>.
- Valenti, O., Gill, K.M., Grace, A.A. (2012). "Different stressors produce excitation or inhibition of mesolimbic dopamine neuron activity: response alteration by stress

- preexposure." *Eur. J. Neurosci.*, 35, 1312–1321. <https://doi.org/10.1111/j.1460-9568.2012.08038.x>.
- van den Bos, R., Harteveld, M., Stoop, H., 2009. Stress and decision-making in humans: performance is related to cortisol reactivity, albeit differently in men and women. *Psychoneuroendocrinology* 34, 1449–1458.
- Van de Vijver, I & Ligneul, R (2020), Relevance of working memory for reinforcement learning in older adults varies with timescale of learning, *Aging, Neuropsychology and Cognition*, vol. 27, no. 5, pp. 654–676.
- van Eck, M.M., Berkhof, H., Nicolson, N., Sulon, J., (1996). The effects of perceived stress, traits, mood states and stressful daily events on salivary cortisol. *Psychosom. Med.* 58, 447–458.
- van Santen A, Vreeburg SA, Van der Does AW, Spinhoven P, Zitman FG, Penninx BW. (2011) Psychological traits and the cortisol awakening response: results from the Netherlands Study of Depression and Anxiety. *Psychoneuroendocrinology*;36:240-248.
- Vickers Jr RR. (1991) *Stress Reactivity: Five-Factor Representation of a Psychobiological Typology*. San Diego, CA: Naval Health Research Center.
- Volkow, N.D., Logan, J., Fowler, J.S., Wang, G.-J., Gur, R.C., Wong, C., Felder, C., Gatley, S.J., Ding, Y.S., Hitzemann, R., Pappas, N., 2000. Association between age-related decline in brain dopamine activity and impairment in frontal and cingulate metabolism. *Am. J. Psychiatry* 157, 75–80.
- Vollrath, M. and Torgersen, S. (2000) ‘Personality types and coping’, *Personality and Individual Differences*, 29(2), pp. 367–378.
- Wacker, J. et al. (2005) ‘Sexually dimorphic link between dopamine D2 receptor gene and neuroticism-anxiety’, *Neuroreport*, 16(6), pp. 611–614.

- Walther. (1969). Flight Behaviour and Avoidance of Predators in Thomson's Gazelle (*Gazella Thomsoni* Guenther 1884). *Behaviour*, 34(3), 184–220.  
<https://doi.org/10.1163/156853969X00053>
- Wang, Y., Jackson, T. and Cai, L. (2016) 'Causal effects of threat and challenge appraisals on coping and pain perception', *European journal of pain*, 20(7), pp. 1111–1120.
- Wang, J. et al. (2007) 'Gender difference in neural response to psychological stress', *Social Cognitive and Affective Neuroscience*, pp. 227–239. doi: 10.1093/scan/nsm018.
- Wang, J., Rao, H., Wetmore, G.S., Furlan, P.M., Korczykowski, M., Dinges, D.F.al.e., (2005). Perfusion functional MRI reveals cerebral blood flow pattern under psychological stress. *Proc. Natl. Acad. Sci. U.S.A.* 102, 17804–17809.
- Warthen, K. G., Boyse-Peacor, A., Jones, K. G., Sanford, B., Love, T. M., Mickey, B. J. (2020). Sex differences in the human reward system: convergent behavioral, autonomic, and neural evidence. *Social Cognitive and Affective Neuroscience*, 15, 789–801.
- Watabe-Uchida, M., Eshel, N. and Uchida, N. (2017) 'Neural Circuitry of Reward Prediction Error', *Annual review of neuroscience*, 40, pp. 373–394.
- Watanabe, Y., Gould, E., McEwen, B.S., 1992. Stress induces atrophy of apical dendrites of hippocampal CA3 pyramidal neurons. *Brain Res.* 588, 341–345.  
[https://doi.org/10.1016/0006-8993\(92\)91597-8](https://doi.org/10.1016/0006-8993(92)91597-8).
- Watson, D., Clark, L. A. and Tellegen, A. (1988) 'Development and validation of brief measures of positive and negative affect: the PANAS scales', *Journal of personality and social psychology*, 54(6), pp. 1063–1070.
- Watt, M.J., Roberts, C.L., Scholl, J.L., Meyer, D.L., Miiller, L.C., Barr, J.L., Novick, A.M., Renner, K.J., Forster, G.L. (2014). "Decreased prefrontal cortex dopamine activity following adolescent social defeat in male rats: role of dopamine D2 receptors."

- Psychopharmacology (Berl.), 231, 1627–1636. <https://doi.org/10.1007/s00213-013-3353-9>.
- Weathers, F.W., Litz, B.T., Keane, T.M., Palmieri, P.A., Marx, B.P., & Schnurr, P.P. (2013). The PTSD Checklist for DSM-5 (PCL-5). Scale available from the National Center for PTSD at [www.ptsd.va.gov](http://www.ptsd.va.gov).
- Weinstock, L. M. and Whisman, M. A. (2006) ‘Neuroticism as a common feature of the depressive and anxiety disorders: A test of the revised integrative hierarchical model in a national sample’, *Journal of Abnormal Psychology*, pp. 68–74. doi: 10.1037/0021-843x.115.1.68.
- Weller, J. et al. (2018) ‘Accounting for Individual Differences in Decision-Making Competence: Personality and Gender Differences’, *Frontiers in psychology*, 9, p. 2258.
- Wellman, C. L. et al. (2018) ‘Sex Differences in Risk and Resilience: Stress Effects on the Neural Substrates of Emotion and Motivation’, *The Journal of neuroscience: the official journal of the Society for Neuroscience*, 38(44), pp. 9423–9432.
- Whitaker, L. R., Degoulet, M., & Morikawa, H. (2013). Social deprivation enhances VTA synaptic plasticity and drug-induced contextual learning. *Neuron*, 77, 335–345. <https://doi.org/10.1016/j.neuron.2012.11.022>.
- Wiggins, JS & Trapnell, PD 1997, ‘Chapter 28 - Personality Structure: The Return of the Big Five’, in *Handbook of Personality Psychology*, Academic Press, pp. 737–765.
- Williams, J. M. G., Mathews, A. and MacLeod, C. (1996) ‘The emotional Stroop task and psychopathology’, *Psychological Bulletin*, pp. 3–24. doi: 10.1037/0033-2909.120.1.3.
- Willner, P (2017), ‘Reliability of the chronic mild stress model of depression: A user survey’, *Neurobiology of Stress*, vol. 6, no. C, pp. 68–77. Wolf, O.T., Schommer, N.C., Hellhammer, D.H., McEwen, B.S., Kirschbaum, C., 2001. The relationship between stress induced cortisol levels and memory differs between men and women. *Psychoneuroendocrinology* 26, 711—720.

- Willner, P. (1997). Validity, reliability, utility of the chronic mild stress model of depression: a 10-year review, evaluation. *Psychopharmacology*, 134, 319–329.
- Wolf, O. T. (2009). Stress and memory in humans: Twelve years of progress? *Brain Research*, 1293, 142–154.
- Wood, P.B., Schweinhardt, P., Jaeger, E., Dagher, A., Hakyemez, H., Rabiner, E.A., Bushnell, M.C., Chizh, B.A. (2007). Fibromyalgia patients show an abnormal dopamine response to pain. *Eur. J. Neurosci.* 25, 3576–3582.
- Wood, S., Busemeyer, J., Koling, A., Cox, C. R., & Davis, H. (2005). 'Older adults as adaptive decision makers: Evidence from the Iowa Gambling Task.' *Psychology and Aging*, 20(2), 220–225.
- Woody, E.Z. and Szechtman, H. (2011) 'Adaptation to potential threat: the evolution, neurobiology, and psychopathology of the security motivation system', *Neuroscience and biobehavioral reviews*, 35(4), pp. 1019–1033.
- World Health Organization. (2018) Adverse Childhood Experiences International Questionnaire. In Adverse Childhood Experiences International Questionnaire (ACE-IQ). [website]: Geneva: WHO, 2018.
- Wu<sup>st</sup>, S., Van Rossum, E.F., Federenko, I.S., Koper, J.W., Kumsta, R., Hellhammer, D.H., (2004b). Common polymorphisms in the glucocorticoid receptor gene are associated with adrenocortical responses to psychosocial stress. *J. Clin. Endocrinol. Metab.* 89, 565—573.
- Xing, B., Li, Y.-C. and Gao, W.-J. (2016) 'Norepinephrine versus dopamine and their interaction in modulating synaptic function in the prefrontal cortex', *Brain research*, 1641(Pt B), pp. 217–233.
- Xu, Y., Barish, P. A., Pan, J., Ogle, W. O., & O'Donnell, J. M. (2012). Animal models of depression and neuroplasticity: assessing drug action in relation to behavior and neurogenesis. *Methods Mol. Biol.*, 829, 103–124.



- Yamagata, S. et al. (2006) 'Is the genetic structure of human personality universal? A cross-cultural twin study from North America, Europe, and Asia', *Journal of personality and social psychology*, 90(6), pp. 987–998.
- Yamamoto, B. K. and Novotney, S. (2002) 'Regulation of Extracellular Dopamine by the Norepinephrine Transporter', *Journal of Neurochemistry*, pp. 274–280. doi: 10.1046/j.1471-4159.1998.71010274.x.
- Yin, H. H. and Knowlton, B. J. (2006) 'The role of the basal ganglia in habit formation', *Nature reviews. Neuroscience*, 7(6), pp. 464–476.
- Young, A. M. (2004). Increased extracellular dopamine in the nucleus accumbens in response to unconditioned and conditioned aversive stimuli: studies using 1 min microdialysis in rats. *Journal of Neuroscience Methods*, 138, 57–63.
- Youssef, F.F., Dookeeram, K., Basdeo, V., Francis, E., Doman, M., Mamed, D., Maloo, S., Degannes, J., Dobo, L., Ditshotlo, P., Legall, G. Stress alters personal moral decision-making. *Psychoneuroendocrinology*, in press.
- Zahrt J, Taylor JR, Mathew RG, Arnsten AF. (1997) Supranormal stimulation of D1 dopamine receptors in the rodent prefrontal cortex impairs spatial working memory performance. *J Neurosci.*;17:8528e8535.
- Zald, D. H. et al. (2008) 'Midbrain dopamine receptor availability is inversely associated with novelty-seeking traits in humans', *The Journal of neuroscience: the official journal of the Society for Neuroscience*, 28(53), pp. 14372–14378.
- Zellner, D.A., Loaiza, S., Gonzalez, Z., Pita, J., Morales, J., Pecora, D., Wolf, A., (2006). Food selection changes under stress. *Physiol. Behav.* 87, 789–793.
- Zellner, D.A., Saito, S., Gonzalez, J., (2007). The effect of stress on men's food selection. *Appetite* 49, 696–699.
- Ziegler, D. A., Piguet, O., Salat, D. H., Prince, K., Connally, E., & Corkin, S. (2010). Cognition in healthy aging is related to regional white matter integrity, but not cortical

thickness. *Neurobiology of Aging*, 31(11), 1912–1926. Retrieved from  
<https://doi.org/10.1016/j.neurobiolaging.2008.10.015>

Zobel A, Barkow K, Schulze-Rauschenbach S, Von Widdern O, Metten M, Pfeiffer U, et al.  
(2004) High neuroticism and depressive temperament are associated with  
dysfunctional regulation of the hypothalamic-pituitary-adrenocortical system in  
healthy volunteers. *Acta Psychiatr Scand*;109:392-399.

Zorn, J.V., Schür, R.R., Boks, M.P., Kahn, R.S., Joëls, M., Vinkers, C.H., 2017. Cortisol stress  
reactivity across psychiatric disorders: a systematic review and meta-analysis.  
*Psychoneuroendocrinology* 77, 25–36. <https://doi.org/10.1016/j.psyneuen.2016.11.036>.

Zweifel, L.S., Fadok, J.P., Argilli, E., Garelick, M.G., Jones, G.L., Dickerson, T.M.K., Allen,  
J.M., Mizumori, S.J.Y., Bonci, A., Palmiter, R.D. (2011). "Activation of dopamine  
neurons is critical for aversive conditioning and prevention of generalized anxiety."  
*Nat. Neurosci.*, 14, 620–626. <https://doi.org/10.1038/nn.2808>.

## Appendix 1

### A1.1 Decision-making experiment data dictionary

*Table 34 The measures used for the investigations of Chapters 4 and 5*

Software variable name	Description	Type	Values	Relation
age	The chronological age provided by the participant	Integer	>=18	Age differences
gender	Self-identified gender of a participant	Category	male, female, other	Gender differences
health_score	Total Score from the 20 item Medical Outcome Study Scale (MOSS-20)	Integer	0 to 100	Health differences
pss_score	Total Score from the 4 Item Perceived Stress Scale (PSS-4) (Santiago, Nielsen Lisa, Smithers, & Roberts, 2020)	Integer	0 to 16	Stress perceptions over the last month
DASS_score	Total Score from the 21 Item Depression Anxiety and Stress Scale (DASS-21), it is the sum of scores for depression, anxiety and stress scores.	Integer	0 to 126	Stress perceptions over the last week
part1_score	Tally of aversive events from the PTSD Checklist for DSM-5, which participants have recalled to have experienced or witnessed over their life span.	Integer	0 to 56	Aversive life events
part2_score	Stress perceptions, experienced within the last month, regarding the worst aversive life event recalled	Integer	0 to 60	Stress perceptions over the last month

Software variable name	Description	Type	Values	Relation
PCA_stress_combined	This is a value representing an underlying latent variable of stress perceptions over the last week or month. The value is created using the principal() function from the R psych package to combine pss_score, DASS_score and part2_score. The value is a number representing the standard deviation difference from the mean. Negative values mean less stress and positive values more stress.	Double	-Inf to +Inf	Stress perceptions over the last week/month
stress_react_bpm	This is the difference in the mean heart beats per minutes (BPM) between threat and safe conditions (mean BPM threat minus safe). The measure is interpreted as a measure of reactivity to the difference between safe and threat conditions	Double	-40 to +40	Acute stress reactivity
stress_react_rmssd	This is the difference between threat and safe conditions of the average Root Mean Square of Successive Differences between normal heart beats (RMSSD), which is a measure of hear rate variability (mean RMSSD threat minus safe). The measure is interpreted as a measure of reactivity to the difference between safe and threat conditions	Double	-Inf to +Inf	Acute stress reactivity
stress_react_br	This is the difference in the mean breathing rate (BR) between threat and safe conditions (mean BR threat minus safe). The measure is interpreted as a measure of reactivity to the difference between safe and threat conditions	Double	-Inf to +Inf	Acute stress reactivity
mean_stay_0	Mean correct choice stays in safe condition	Double	0 to 1	Decision-making performance

Software variable name	Description	Type	Values	Relation
mean_stay_1	Mean correct choice stays in threat condition	Double	0 to 1	Decision-making performance
mean_stay_diff	Difference in mean correct choice stays between conditions (mean_stay_1 - mean_stay_0)	Double	-1 to 1	Decision-making performance
mean_stay	Mean correct choice stays overall ((mean_stay_0 + mean_stay_1)/2)	Double	0 to 1	Decision-making performance
mean_switch_0	Mean correct choice switch in safe condition	Double	0 to 1	Decision-making performance
mean_switch_1	Mean correct choice switch in threat condition	Double	0 to 1	Decision-making performance
mean_switch_diff	Difference in mean correct choice switches between conditions (mean_switch_1 - mean_switch_0)	Double	-1 to 1	Decision-making performance
mean_switch	Mean correct choice switches overall ((mean_switch_0 + mean_switch_1)/2)	Double	0 to 1	Decision-making performance
mean_stay_switch_diff	Difference between mean stay and switch performance overall	Double	-1 to 1	Decision-making performance
mean_stay_switch_diff_0	Difference between mean stay and switch performance during safe conditions	Double	-1 to 1	Decision-making performance
mean_stay_switch_diff_1	Difference between mean stay and switch performance during threat conditions	Double	-1 to 1	Decision-making performance

Notes:

BPM, RMSSD and BR were estimated using the HeartPy library referenced in <https://python-heart-rate-analysis-toolkit.readthedocs.io/en/latest/index.html#>

## A1.2 Dracoin Door Game instructions, questionnaires, consent form, and information for participant

### Game Instructions

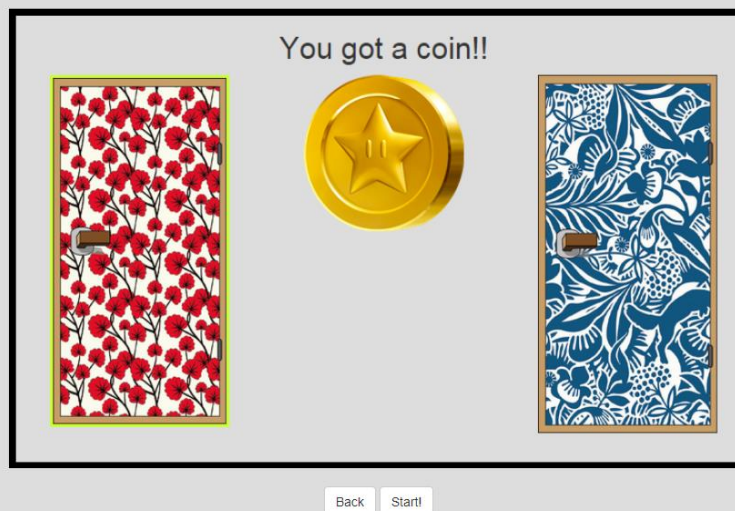
You have discovered an alchemist paint formula that turns ordinary doors into Dracoin Doors. Dracoin Doors teleport you to another realm called the Island of Dracoinia where treasure awaits! On your teleporting journey you will have to choose between two distinctly colored Dracoin Doors. You choose a door by clicking on it with your mouse. Please click the NEXT button below to proceed



### Game Instructions

You will need to pay attention and decide which Dracoin Doors brings you most fortune. When you decide which door to open, you will either receive golden coins as shown in the example image below, or you may receive nothing. On some unforeseen occasions, you will encounter dragons whom are notorious for being unpredictable, uncontrollable and impulsive thieves. Unfortunately, resistance against them is futile, they are indomitable!

Choose swiftly and wisely and good luck on your intrepid journey to fortune!



# PARTICIPANT INFORMATION SHEET

**PROJECT TITLE: Understanding individual differences in decision-making under mild stress**

**HUMAN RESEARCH ETHICS COMMITTEE APPROVAL NUMBER: H-2019-35142**

**PRINCIPAL INVESTIGATOR: Assoc. Prof. Lyndsey Collins-Praino.**

**STUDENT RESEARCHER: Manuel Salazar**

**STUDENT'S DEGREE: Master of Philosophy (Medical Science)**

Dear Mturkers! [For online experiment]

Dear Students! [For lab experiment]

You are invited to participate in the research project described below.

## **What is the project about?**

We rely on our decision-making skills to improve our quality of life. However, throughout our life we sometimes experience non-ideal conditions that are unpredictable or uncontrollable. Such experiences might seem like challenges to some, whilst to others, they might seem as threatening or stressful. These individual differences can influence our judgements and decision-making skills. We want to understand how individual differences in demographics, health, personality, impulsivity and previous experiences with stress influence our decision-making under non-ideal conditions.

## **Who is undertaking the project?**

This project is being conducted by Assoc. Prof. Lyndsey Collins-Praino, Dr. Irina Baetu and Mr. Manuel Salazar. If relevant add the following: This research will form the basis for the degree of Master of Philosophy (Medical Science) at the University of Adelaide under the supervision of Assoc. Prof. Lyndsey Collins-Praino and Dr. Irina Baetu.

## **Why am I being invited to participate?**

You are being invited as you are 18 years of age or over, are fluent in the English language.

## **What am I being invited to do?**

You are being invited to play a decision-making game and answer questionnaires about your demographics, physical and mental health, personality, impulsivity, life events and stress.

## **How much time will my involvement in the project take?**

Playing the game and completing the questionnaires is estimated to take approximately thirty minutes to an hour.

## **Are there any risks associated with participating in this project?**

We understand that some of the questionnaires included in this study might cause feelings of distress or might remind you of events or circumstances that cause you to feel anxious. Should you need to speak to someone immediately regarding your psychological difficulties, please contact your GP or health professional.

The following telephone numbers are for services that you can access to help you with any difficulties you might experience:

[For the online study]

The following telephone numbers are for services, from different nations, that you can access to help you with any difficulties you might experience:

- Free call Samaritans: 116 123 (Ireland)
- Infoline: 0300 123 3393 (United Kingdom)
- Toll-free help line: 1 855 242 3310 (Canada)
- Free call: 1737 (New Zealand)
- National Helpline: 1-800-662-HELP (4357) (United States)
- Lifeline: 13 11 14 (Australia)

[For the lab study]

The following telephone numbers are for services that you can access to help you with any difficulties you might experience:

- Lifeline: 13 11 14
- Lifeline: 13 11 14
- Beyond Blue: 1300 22 4636
- Headspace: 1800 650 890
- SANE Australia: 1800 18 7263
- MindSpot: 1800 61 44 34
- Blue Knot Foundation Helpline: 1300 657 380

### **What are the potential benefits of the research project?**

Studying such individual differences in decision-making will assist in predicting the likely decision-making patterns that might be observed in individuals who frequently experience higher levels and exposure to threats, such as emergency response and disaster relief workers, police, ambulance workers or military personnel.

### **Can I withdraw from the project?**

[For the online study]

As you are a volunteer for this research project, participation in this project is completely voluntary. If you agree to participate, you can withdraw from the study by stopping the task and returning to your HITs dashboard at any time. At no time must you feel pressured to participate or to continue if you do not wish to do so. As data is anonymous, once you submit your responses, it is not possible to withdraw them from



the study. However, any incomplete data will be deleted and will not be included in the data analysis or made available as open data and stored publicly. Please note, if you decide to withdraw from the study, you will not be paid through Amazon Mechanical Turk.

[For the lab study]

As you are a volunteer for this research project, participation in this project is completely voluntary. If you agree to participate, you can withdraw from the study at any time. At no time must you feel pressured to participate or to continue if you do not wish to do so.

### **What will happen to my information?**

The data you provide will be released after the study is completed but in a de-identified form, thus it will not be possible to identify you by name from any aspect of documentation or reporting for this research study. Anonymity will be preserved in reports or published articles.

Once the current study has been completed, the data will become open data. This means the data will be made available, free of charge, to anyone interested in the research, or who wishes to conduct their own analysis of the data. We will therefore have no control over how the data are used, however, prior to becoming open data, all data will be anonymised and therefore all participants will maintain confidentiality and anonymity.

No personal participant information will be kept or maintained.

Your information will only be used as described in this participant information sheet and it will only be disclosed according to the consent provided, except as required by law.

### **Who do I contact if I have questions about the project?**

You may contact either: Associate Professor Dr. Lyndsey Collins-Praino on +61 8 8313 5488, Senior Lecturer

Dr. Irina Baetu on +61 8 8313 6102 or Master student Mr. Manuel Salazar on

manuel.salazar@student.adelaide.edu.au

### **What if I have a complaint or any concerns?**

The study has been approved by the Human Research Ethics Committee at the University of Adelaide

(approval number H-2019-35142). This research project will be conducted according to the NHMRC National Statement on Ethical Conduct in Human Research 2007 (Updated 2018). If you have questions or problems

associated with the practical aspects of your participation in the project, or wish to raise a concern or complaint about the project, then you should consult the Principal Investigator. If you wish to speak with an independent person regarding concerns or a complaint, the University's policy on research involving human participants, or your rights as a participant, please contact the Human Research Ethics Committee's

Secretariat on:

Phone: +61 8 8313 6028

Email: [hrec@adelaide.edu.au](mailto:hrec@adelaide.edu.au)

Post: Level 4, Rundle Mall Plaza, 50 Rundle Mall, ADELAIDE SA 5000

Any complaint or concern will be treated in confidence and fully investigated. You will be informed of the outcome.

For participants outside Australia please consult the relevant bodies on the conduct of human experiments as per the following link: <https://www.hhs.gov/ohrp/sites/default/files/2020-international-compilation-of-human-research-standards.pdf>

**If I want to participate, what do I do?**

Before participating please ensure you understand what you will be doing and why. If you decide to participate, then you remain free to withdraw from this study at any time, without prejudice and without the need to provide reason or justification. If you wish to withdraw, simply click the NEXT button located at the bottom and stop completing the online surveys and tasks.

Yours sincerely,  
Assoc. Prof. Lyndsey Collins-Praino  
Dr. Irina Baetu  
Mr. Manuel Salazar

Consent to volunteer in the study

Please carefully read and understand the following:

I understand that:

I have had explained to me the aims of the study, how it will be conducted and my role in it,

I understand the risks involved as described in the volunteer information section,

The information I provide will be kept anonymous and we will not record your worker ID.

The worker ID will only be used to process payment if you decide to accept payment for your participation,

Participating means that you answer the questions and complete the decision-making game in an honest and accurate manner,

Completion of the questionnaire and decision-making game will be considered implied consent,

There is no obligation to take part in this research study,

I am free to withdraw at any time prior to submitting my responses,

I have carefully read the information above,

I know taking part in the research study is voluntary and I can stop at any moment,

I give consent for my data to be used in future research projects that are an extension of, or closely related to, this project under consideration,

I give permission for the data to be reported in aggregate,

I want to partake in this research study

No

Yes, I have carefully read and understood the information above and I consent to participate in the study

## Human Research Ethics Committee (HREC)

### CONSENT FORM [For the Lab Study]

1. I have read the attached Information Sheet and agree to take part in the following research project:

<b>Title:</b>	<b>Understanding individual differences in decision-making under mild stress</b>
<b>Ethics Approval</b>	<b>H-xxxx-xxx</b>

2. I have had the project, so far as it affects me, and the potential risks and burdens fully explained to my satisfaction by the research worker. I have had the opportunity to ask any questions I may have about the project and my participation. My consent is given freely.
3. Although I understand the purpose of the research project, it has also been explained that my involvement may not be of any benefit to me.
4. I agree to play a decision-making game and answer questionnaires about my demographics, physical and mental health, personality, impulsivity, life events and stress, as outlined in the participant information sheet.
5. I understand that as my participation is anonymous, I can withdraw any time up until submission of my questionnaire answers.
6. I have been informed that the information gained in the project may be published in a journal article, thesis or conference presentations.
7. I have been informed that in the published materials I will not be identified and my personal results will not be divulged. not give consent to be named in the published materials.
8. I hereby provide 'extended' consent for the use of my data in future research projects that are:
- (i) an extension of, or closely related to, the original project: Yes  No
  - (ii) in the same general area of research (for example, genealogical, ethnographical, epidemiological, or chronic illness research): Yes  No
  - (iii) I hereby provide 'unspecified' consent for the use of my data in any future research: Yes  No
9. I agree to be contacted with information about future research studies: Yes  No
10. I understand my information will only be disclosed according to the consent provided, except where disclosure is required by law.

11. I am aware that I should keep a copy of this Consent Form, when completed, and the attached Information Sheet.

**Participant to complete:**

Name: \_\_\_\_\_ Signature: \_\_\_\_\_ Date: \_\_\_\_\_

**Researcher/Witness to complete:**

I have described the nature of the research to \_\_\_\_\_

*(print name of participant)*

and in my opinion she/he understood the explanation.

Signature: \_\_\_\_\_ Position: \_\_\_\_\_ Date: \_\_\_\_\_

Welcome and thank you for your participation!

You may proceed to playing the decision-making game and completing the questionnaires.

It is very important that you be honest and accurate in your answers.

Please read and follow the instructions carefully.

Once again thank you for contributing and supporting the research.

## Questionnaire 1 of 8

Age

## Gender

- Male
- Female
- Other

If selected other then please specify gender:

## Full-time equivalent years of education to date

## Highest Education Level Attained

- Less than high school
- High school graduate
- Some college
- Technical or trade school
- Associate degree or equivalent
- Bachelor degree or equivalent
- Graduate certificate or diploma
- Masters
- Doctorate

## Current employment status

- Employed full time
- Employed part time
- Student
- Unemployed looking for work
- Unemployed not looking for work
- Retired
- Disabled

## Annual household income (US dollar equivalent)

You may use the [XE website](#) website for currency conversion

- Less than \$10,000
- \$10,000 - \$29,999
- \$30,000 - \$49,999
- \$50,000 - \$69,999
- \$70,000 - \$89,999
- \$90,000 - \$149,999
- More than or equal to \$150,000

## Relationship status

- De facto
- Married
- Widowed
- Divorced
- Separated
- Never married

## Country or countries of citizenship

- Australia
- New Zealand
- United Kingdom of Great Britain
- United States of America
- Canada
- Ireland
- Other

If selected other then please specify country(ies) of citizenship:



## Country of primary residence

- Australia
- New Zealand
- United Kingdom of Great Britain
- United States of America
- Canada
- Ireland
- Other

If selected other then please specify country of residence:

## Living environment surrounding primary residence

- Rural area more than 1 hour flight to nearest city or town and having less than or equal to 10 thousand residents
- Rural area more than 1 hour flight to nearest city or town and having greater than 10 thousand residents
- Suburb more than 1 hour drive to nearest city or town and having less than or equal to 50 thousand residents
- Suburb more than 1 hour drive to nearest city or town and having more than 50 thousand residents
- Suburb less than 1 hour drive to nearest city or town and having less than or equal to 50 thousand residents

- Suburb less than 1 hour drive to nearest city or town and having greater than 50 thousand residents
- City living and having less than or equal to 1 million residents
- City living and having greater than 1 million residents

[For the online study]

## Computing device used to complete the study

- Desktop Computer
- Laptop
- Tablet
- Smartphone
- Other

If selected other then please specify the computing device:

## Input devices used to complete the study

- Keyboard
- Mouse
- Touchpad
- Touchscreen (including Ipads, tablets, and smart phones)
- Other

If selected other then please specify the input device(s):

# Operating system of the computing device used to complete the study

- Windows
- MacOS/iOS
- Android
- Linux
- Other

If selected other then please specify the operating system:

# Questionnaire 2 of 8

Here are a number of personality traits that may or may not apply to you. Please indicate the extent to which you agree or disagree with the statements. You should rate the extent

to which the pair of traits applies to you, even if one characteristic applies more strongly than the other.

1 = Disagree strongly

2 = Disagree moderately

3 = Disagree a little

4 = Neither agree nor disagree

5 = Agree a little

6 = Agree moderately

7 = Agree strongly

	<b>Disagree strongly</b>	<b>Disagree moderately</b>	<b>Disagree a little</b>	<b>Neither agree nor disagree</b>	<b>Agree a little</b>	<b>Agree moderately</b>	<b>Agree strongly</b>
Extraverted, enthusiastic	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Critical, quarrelsome	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Dependable, self-disciplined	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Anxious, easily upset	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Open to new experiences, complex	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Reserved, quiet	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Sympathetic, warm	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Disorganized, careless	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

Calm, emotionally stable	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Conventional, uncreative	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

# Questionnaire 3 of 8

People differ in the ways they act and think in different situations. This is a test to measure some of the ways in which you act and think.

Select the appropriate circle. Do not spend too much time on any statement. Answer quickly and honestly.

	Rarely/Neve r	Occasionall y	Often	Almost Always/Always
I plan tasks carefully	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I do things without thinking	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I make-up my mind quickly	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am happy-go-lucky	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I don't "pay attention"	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I have "racing" thoughts	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

I plan trips well ahead of time	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am self-controlled	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I concentrate easily	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I save regularly	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I "squirm" at plays or lectures	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am a careful thinker	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I plan for job security	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I say things without thinking	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I like to think about complex problems	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I change jobs	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I act "on impulse"	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I get easily bored when solving thought problems	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I act on the spur of the moment	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>



I am a steady thinker	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I change residences	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I buy things on impulse	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I can only think about one thing at a time	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I change hobbies	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I spend or charge more than I earn	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I often have extraneous thoughts when thinking	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am more interested in the present than the future	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am restless at the theatre or lectures"	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I like puzzles	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am future oriented	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

## Questionnaire 4 of 8

For how long (if at all) has your physical health limited you in each of the following activities?

	Limited for more than 3 months	Limited for 3 months or less	Not limited at all
The kinds or amounts of vigorous activities you can do, like lifting heavy objects, running or participating in strenuous sports	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
The kinds or amounts of moderate activities you can do, like moving a table, carrying groceries, or bowling	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Walking uphill or climbing a few flights of stairs	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Bending, lifting, or stooping	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Walking one block	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Eating, dressing, bathing, or using the toilet	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

For each of the following questions, please mark the circle for the one answer that comes closest to the way you have been feeling during the PAST MONTH.

	All of the time	Most of the time	A good bit of the time	Some of the time	A little of the time	None of the time
How much of the time, during the past month, has your health limited your social activities (like visiting with friends or close relatives)?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
How much of the time, during the past month, have you been a very nervous person?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

During the past month, how much of the time have you felt calm and peaceful?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
How much of the time, during the past month, have you felt downhearted and blue?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
During the past month, how much of the time have you been a happy person?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
How often, during the past month, have you felt so down in the dumps that nothing could cheer you up?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

Please mark the circle that best describes whether each of the following statements is true or false with respect to both your physical and mental health CURRENTLY.

	<b>Definitely true</b>	<b>Mostly true</b>	<b>Mostly false</b>	<b>Definitely false</b>
I am somewhat ill	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am as healthy as anybody I know	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
My health is excellent	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I have been feeling bad lately	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

Has there been any change to your physical health amidst the COVID-19 pandemic?

- Much better
- Moderately better
- Slightly better

- About the same
- Slightly worse
- Moderately worse
- Much worse

Has there been any change to your mental health amidst the COVID-19 pandemic?

- Much better
- Moderately better
- Slightly better
- About the same
- Slightly worse
- Moderately worse
- Much worse

## Questionnaire 5 of 8

The questions in this scale ask you about your feelings and thoughts during the LAST MONTH. In each case, please indicate with a check how often you felt or thought a certain way

	Never	Almost never	Sometimes	Fairly often	Very often
In the last month, how often have you felt that you were unable to control the important things in your life?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

In the last month, how often have you felt confident about your ability to handle your personal problems?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
In the last month, how often have you felt that things were going your way?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

## Questionnaire 6 of 8

The DASS-21 is the short form of the DASS-42, a self-report scale designed to measure the negative emotional states of depression, anxiety and stress.



Please read each statement and choose an option that indicates how much the statement applied to you over the PAST WEEK. There are no right or wrong answers. Do not spend too much time on any statement.

	Never	Sometimes	Often	Always
I found it hard to wind down	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I was aware of dryness of my mouth	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I couldn't seem to experience any positive feeling at all	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I found it difficult to work up the initiative to do things	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

I tended to over-react to situations	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I experienced trembling (e.g., in the hands)	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt that I was using a lot of nervous energy	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I was worried about situations in which I might panic and make a fool of myself	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt that I had nothing to look forward to	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I found myself getting agitated	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I found it difficult to relax	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt down-hearted and blue	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I was intolerant of anything that kept me from getting on with what I was doing	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt I was close to panic	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

I was unable to become enthusiastic about anything	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt I wasn't worth much as a person	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt that I was rather touchy	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt scared without any good reason	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I felt that life was meaningless	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

## Questionnaire 7 of 8

**Instructions:** Listed below are a number of difficult or stressful things that sometimes happen to people. For each event check one or more of the boxes to the right to indicate that: (a) it happened to you personally; (b) you witnessed it happen to someone else; (c)

you learned about it happening to a close family member or close friend; (d) you were exposed to it as part of your job (for example, paramedic, police, military, or other first responder); (e) you're not sure if it fits; or (f) it doesn't apply to you.

If any of these questions cause you to feel distressed or anxious, please consult your GP or health professional.

[For the online study] Alternatively, the following website links reference services from different nations that you may access to help you with any difficulties you might experience:

- Free call Samaritans: 116 123 (Ireland)
- Infoline: 0300 123 3393 (United Kingdom)
- Toll-free help line: 1 855 242 3310 (Canada)
- Free call: 1737 (New Zealand)
- National Helpline: 1-800-662-HELP (4357) (United States)
- Lifeline: 13 11 14 (Australia)

[For the lab study] Alternatively, the following website links reference services that you may access to help you with any difficulties you might experience:

- Lifeline: 13 11 14
- Beyond Blue: 1300 22 4636
- Headspace: 1800 650 890
- SANE Australia: 1800 18 7263
- MindSpot: 1800 61 44 34
- Blue Knot Foundation Helpline: 1300 657 380

Be sure to consider your entire life (growing up as well as adulthood) as you go through the list of events.

	<b>Happened to me</b>	<b>Witnessed it</b>	<b>Learned about it</b>	<b>Part of my job</b>	<b>Not sure</b>	<b>Doesn't apply</b>
Natural disaster (for example, flood, hurricane, tornado, earthquake)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Fire or explosion	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Transportation accident (for example, car accident, boat accident, train wreck, plane crash)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Serious accident at work, home, or during recreational activity	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Exposure to toxic substance (for example, dangerous chemicals, radiation)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Physical assault (for example, being attacked, hit, slapped, kicked, beaten up)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Assault with a weapon (for example, being shot, stabbed, threatened with a knife, gun, bomb)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sexual assault (rape, attempted rape, made to perform any type of sexual act through force or threat of harm)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Other unwanted or uncomfortable sexual experience	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Combat or exposure to a war-zone (in the military or as a civilian)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Captivity (for example, being kidnapped, abducted, held hostage, prisoner of war)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Life-threatening illness or injury	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Severe human suffering	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Sudden violent death (for example, homicide, suicide)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sudden accidental death	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Serious injury, harm, or death you caused to someone else	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Any other very stressful event or experience	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

## Questionnaire 8 of 8

Below is a list of problems that people sometimes have in response to a very stressful experience. Keeping your worst event in mind, please read each problem carefully and then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the past month.

In the PAST MONTH, how much were you bothered by:

	<b>Not at all</b>	<b>A little bit</b>	<b>Moderately</b>	<b>Quite a bit</b>	<b>Extremely</b>
Repeated, disturbing, and unwanted memories of the stressful experience?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Repeated, disturbing dreams of the stressful experience?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Suddenly feeling or acting as if the stressful experience were actually happening again (as if you were actually back there reliving it)?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Feeling very upset when something reminded you of the stressful experience?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>



Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble breathing, sweating)?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Avoiding memories, thoughts, or feelings related to the stressful experience?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Avoiding external reminders of the stressful experience (for example, people, places, conversations, activities, objects, or situations)?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Trouble remembering important parts of the stressful experience?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Having strong negative beliefs about yourself, other people, or the world (for example, having thoughts such as: I am bad, there is something seriously wrong	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

with me, no one can be trusted, the world is completely dangerous)?					
Blaming yourself or someone else for the stressful experience or what happened after it?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Having strong negative feelings such as fear, horror, anger, guilt, or shame?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Loss of interest in activities that you used to enjoy?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Feeling distant or cut off from other people?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Trouble experiencing positive feelings (for example, being unable to feel happiness or have loving feelings for people close to you)?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Irritable behavior, angry outbursts, or acting aggressively?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

Taking too many risks or doing things that could cause you harm?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Being “superalert” or watchful or on guard?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Feeling jumpy or easily startled?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Having difficulty concentrating?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Trouble falling or staying asleep?	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

### A1.3 Amazon Mechanical Turk Platform crowdsourcing task details

<b>Project Name</b>	Dracoin Doors Decision Game
<b>Title</b>	Decision-making Research - (Pay will be between 3 and 5 US dollars depending on performance, expected task duration is 30mins)
<b>Description</b>	Understanding individual differences in decision-making under non-ideal conditions (Required: quiet work place with uninterrupted internet connection, enable computer sound, monitor size larger than 1366 x 768, browser: Chrome, Firefox, Edge, Safari)
<b>Keywords</b>	decision, game, survey, research, study
<b>Reward per response</b>	\$5 US
<b>Number of respondents</b>	109
<b>Time allotted per Worker</b>	1 hours
<b>Survey expires in</b>	14 days
<b>Auto-approve and pay Workers in</b>	30 days
<b>Require that Workers be Masters to do your tasks</b>	No
<b>Location is one of</b>	Australia, Canada, Ireland, New Zealand, United Kingdom, United States,
<b>HIT Approval Rate (%) for all Requesters' HITs greater than or equal to</b>	95
<b>Number of HITs Approved greater than</b>	1000

<b>Fresh workers only</b>	<p>has not been granted</p> <p>This qualification was created to ensure that participants do not repeat the task, however, with technologies such as virtual private networks and virtual machines it is difficult to know if a single participant can register to do the same task multiple times</p>
<b>Gender - Female/Male</b>	<p>True</p> <p>This was used to ensure an even number of males and females, however, there were some occasions that the qualification matched and the participant entered a different gender in the Demographics questionnaire.</p>
<b>Project contains adult content</b>	No
<b>Task Visibility</b>	Hidden - Only Workers that meet my Qualification requirements can see and preview my tasks

## Appendix 2

### A2.1 Dracoin Doors Decision Game Software Description

The Dracoin Doors Game was written as a single HTML file using CSS, HTML, and JavaScript. The software was written in a way that allows dynamic pages to be rendered in the browser and to then wait for the player to interact with the web page. It should be noted that user interaction constraints were enabled to prevent the user from unwanted interactions such as dragging and dropping, unnecessary double clicking, etc. Each web page was rendered in a sequential manner. The web page rendering sequence is as described below.

Sequence	Web page displayed	Controls
1	Participant information sheet	Next button
2	Consent form	Radio button
2a	Reconsider alert if 'no' was selected	None
2b	Greeting page if yes was selected	Next button
2c	Exit message if no was selected following the reconsider alert	Amazon Turk Submit button
3	Instruction page 1	Next button
4	Instruction page 2	Back, and Start buttons
4a	Back to instruction page 1 if Back button was pressed	Next button
5	Start cue count to 3	None

Sequence	Web page displayed	Controls
6	<p>Decision-making game begins by randomly choosing 1 of 4 pre-determined randomly generated trial sequences as defined in the file TrialsCheckV8.xlsx</p> <p>Game trials are displayed following the selected random trial sequence</p> <p>Note: Condition background is first displayed prior to each game trial</p>	Each trial allows the choice between two colored doors
6a	<p>Dragon animation if a threat event has occurred in the trial sequence</p> <p>Monetary loss displayed</p>	None
6b	<p>Coin drop animation occurs if the right door was selected within a trial</p> <p>Monetary gain displayed</p>	None
6c	No reward message displayed in the case of the wrong door selected	None

Sequence	Web page displayed	Controls
7	Game end notice is displayed following completion of all the game trials	Next button
8	Demographics questionnaire	See Appendix 1
9	Personality questionnaire	See Appendix 1
10	Impulsivity questionnaire	See Appendix 1
11	Health questionnaire	See Appendix 1
12	Perceived Stress Scale	See Appendix 1
13	Depression Anxiety and Stress Scale	See Appendix 1
14	Post-Traumatic Stress Checklist – 1 <sup>st</sup> part	See Appendix 1
15	Post-Traumatic Stress Checklist – 2 <sup>nd</sup> part	See Appendix 1
16	Task completion page	Amazon Turk Submit button for online study  Submit button for lab study

Open science framework link:

[https://osf.io/4dsr7/?view\\_only=abc42c3a173a4d5288239946cf565663](https://osf.io/4dsr7/?view_only=abc42c3a173a4d5288239946cf565663)



The software made use of the following JavaScript references:

Library name	Source
crowd-html-elements	<a href="https://assets.crowd.aws/crowd-html-elements.js">https://assets.crowd.aws/crowd-html-elements.js</a>
JQuery version 1.10.2	<a href="https://ajax.googleapis.com/ajax/libs/jquery/1.10.2/jquery.min.js">https://ajax.googleapis.com/ajax/libs/jquery/1.10.2/jquery.min.js</a>
Bootstrap cascade style sheet	<a href="https://maxcdn.bootstrapcdn.com/bootstrap/3.3.4/css/bootstrap.min.css">https://maxcdn.bootstrapcdn.com/bootstrap/3.3.4/css/bootstrap.min.css</a>

Royalty free images and sound resources were obtained from the internet and images edited, were necessary, using Microsoft Paint. The following images were used:

Image	Storage location
Door 1	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_395gIzQcHi2awSh">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_395gIzQcHi2awSh</a>
Door 2	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6Gavf6AEtnvEiQ5">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6Gavf6AEtnvEiQ5</a>
Door 3	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_eX6AnLTG6fKZYLb">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_eX6AnLTG6fKZYLb</a>
Door 4	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_3sWePPL9BSNxsvV">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_3sWePPL9BSNxsvV</a>
Door 5	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6YFYTI18fksPjSJ">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6YFYTI18fksPjSJ</a>
Door 6	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_cIb7C8qhW6YWwOF">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_cIb7C8qhW6YWwOF</a>
Door 7	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6yP7q6PL2pkR3uJ">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6yP7q6PL2pkR3uJ</a>
Door 8	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_9WyERAprao1CK5D">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_9WyERAprao1CK5D</a>
Gold pot	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_0qRO6kOe9zVfb7v">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_0qRO6kOe9zVfb7v</a>
Coin reward	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_bPbWG3cBXA4Ecct">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_bPbWG3cBXA4Ecct</a>
Dragon	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_cGbj8jdTW7MN7Cd">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_cGbj8jdTW7MN7Cd</a>
Threat condition	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_7R5SAW5QkK3xVf7">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_7R5SAW5QkK3xVf7</a>

Pair 1 background	
Threat condition Pair 2 background	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6Rafnpt7J00Io97">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_6Rafnpt7J00Io97</a>
Safe condition Pair 1 background	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_4So84ZJFXAhXxel">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_4So84ZJFXAhXxel</a>
Safe condition Pair 2 background	<a href="https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_9pJf6p3TGzVAyup">https://adelaideunisop.au1.qualtrics.com/ControlPanel/Graphic.php?IM=IM_9pJf6p3TGzVAyup</a>
Instruction 1 image	Obtained from a decision-making game previously implemented (reference <a href="https://github.com/socialdecisionlab/JStutorial">https://github.com/socialdecisionlab/JStutorial</a> ), the image was then converted to Base 64 using a free online conversion application and embedded within the HTML file
Instruction 2 image	Obtained from a decision-making game previously implemented (reference <a href="https://github.com/socialdecisionlab/JStutorial">https://github.com/socialdecisionlab/JStutorial</a> ), the image was then converted to Base 64 using a free online conversion application and embedded within the HTML file

The following royalty free music and sound files were obtained from the internet: Dragon roar, safe condition music, and threat condition music. The files were converted to Base 64 using a free online conversion application and then embedded as variables within the HTML source file. These files are in ‘..\Appendix2\Dracoin\_Doors\_Game\_Lab\_Version\references\resources’

## A2.2 Dracoin Doors Game Trial Sequences

The game trial sequences were generated by the TrialRandomisation14.m Matlab script which was created to generate 4 randomised trials to accommodate counterbalancing sequences that

begin with a safe condition or threat condition and begin with either Pair 1 or Pair 2. As such, the script generates 4 CSV files containing a random sequence of game trials. Each of the generated sequences were then copied and used as variables in the Dracoin Doors Game software code and one of the four sequence is selected at random at the beginning of the game.

The TrialsCheckV8.xlsx spreadsheet was created from the generated trials to check the 4 sequences and as a source of ground truth to compare participants' choices. An example of the contents of TrialsCheckV8.xlsx, for the first 5 trials of 1 of 4 random trial sequences is shown below.

Trial	Block	Condition	Pair	Position	Win	Loss	Reversal	Threat	Condition	Left	Right
1	1	0	0	0	1	0	none	0	safe	turquoise	green
2	1	0	1	1	1	0	none	0	safe	red	orange
3	1	0	1	0	1	0	none	0	safe	orange	red
4	1	0	1	1	1	0	none	0	safe	red	orange
5	1	0	0	1	1	0	none	0	safe	green	turquoise

Notes:

Colors and pairs:

- Door 1 is red (starts off as left and winning door in block 1) Pair1
- Door 2 is orange
- Door 3 is green (starts off as left and winning door in block 1) Pair2
- Door 4 is turquoise
- Door 5 is magenta (starts off as left and winning door in block 1) Pair1
- Door 6 is maroon
- Door 7 is yellow (starts off as left and winning door in block 1) Pair2
- Door 8 is blue

Value enumerations:

- 1 represents pair 1 and 0 represents pair 2
- 1 is left/right and 0 is right/left
- 1 represents a win 0 a loss

*Note-* File paths within the supplementary material: ..\Appendix2\Randomisation\

### A2.3 Dracoin Doors Game Data Collected

The Dracoin Doors Game retrieves experimental data in JSON format. For the lab study, additional files were used to extract heart rate information. The following data was collected.

Data label	Description	Example
IP_address	The IP address collected from using XMLHttpRequest()	73.171.98.42 (only collected for online study)
date	Time and date, in ISO 8601 format, when the experiment began	2021-11-02T11:18:55.937Z
group	Number enumeration representing one of four random trial sequences used as a way of counterbalancing the experiment	4
IP_details	A set of information related to the IP address, which is collected from querying <a href="https://extreme-ip-lookup.com/json/">https://extreme-ip-lookup.com/json/</a> (this information is collected only for the online study, note that, an API key is required to access this information, otherwise it retrieves none)	<pre>{   "IP_details": {     "businessName": "",     "businessWebsite": "",     "city": "Bluefield",     "continent": "North America",     "country": "United States",     "countryCode": "US",     "ipName": "c-73-171-98-42.hsd1.va.comcast.net",     "ipType": "Residential",     "isp": "Comcast Cable Communications, LLC",     "lat": "37.25262",     "lon": "-81.27121",     "message": "Important: API Key required, please get your API Key at https://extreme-ip-lookup.com",     "org": "Comcast Cable Communications, LLC",     "query": "73.171.98.42",</pre>

Data label	Description	Example
		<pre> "region": "Virginia", "status": "success" } }, </pre>
agree	A yes or no string representing consent	“yes”
Trial Information	A data set containing information about the trial conditions. Which includes the block number the trial belongs to, the trial number, type of condition (1 represents a threatening, and 0 represents a safe condition), a number specifying if the threat occurred (1 means it occurs, 0 means it doesn't occur), the frequency of threat occurrence, the variable name and color for the doors in the trial	<pre> { "block_num": 1, "trial_id": 1, "threat_condition": 1, "threat_occurred": 1, "threat_prob": "0.06", "left_door": "#Door8", "left_color": "blue", "right_door": "#Door7", "right_color": "yellow" }, </pre>
mouseBehaviour	This is a data set containing a system count representing time elapsed and the vertical and horizontal screen coordinates, in pixels, representing the location of the pointing device cursor displayed on the computer monitor. The time stamp and coordinates are periodically collected based on mouse movements.	<pre> { "mouseBehaviour": { "t": 1635852766125, "x": 702, "y": 351 } }, </pre>
Choice information	This is a data set containing information on the participant's choice within a trial,	<pre> { "door_choice": "#Door7", </pre>

Data label	Description	Example
	including the door type and color, the probability of the winning door, and the time taken to make a choice in milliseconds	<pre> "door_choice_color": "yellow", "door_choice_win_prob": "0.75", "decisionTime": 3436 } </pre>
Outcome information	This is a data set containing information about the outcome of a trial choice, which includes a monetary value of the reward, the total money accrued, the duration of the outcome animation, any money lost if a threat occurred, and the number of door selection clicks	<pre> { "reward_value": 5, "reward_balance": 185, "outcome_duration": 1805, "threat_loss": 0, "click_count": 1 } </pre>

Demographics	This is a data set which collects information regarding a player's demographic (refer to Appendix 1)	<pre> {   "age": "34",   "gender": "female",   "gendero": "",   "ed_years": "16",   "ed_level": "ed6",   "employed": "em1",   "income": "in2",   "relations": "re2",   "citizen": [     "US"   ],   "citizeno": "",   "residence": "rUS",   "residenceo": "",   "environment": "en2",   "device": "LT",   "deviceo": "",   "inputs": [     "KB",     "TP"   ],   "inputso": "",   "os": "AND",   "oso": "" </pre>
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Data label	Description	Example
		},
Personality	This is a data set which collects information regarding a player's self-rated personality (refer to Appendix 1)	<pre> {   "tp1": "2",   "tp2": "1",   "tp3": "7",   "tp4": "1",   "tp5": "3",   "tp6": "7",   "tp7": "2",   "tp8": "2",   "tp9": "6",   "tp10": "1" } </pre>



Impulsivity	This is a data set which collects information regarding a player's self-rated impulsiveness (refer to Appendix 1)	<pre> {   "imp1": "4",   "imp2": "1",   "imp3": "4",   "imp4": "4",   "imp5": "4",   "imp6": "4",   "imp7": "4",   "imp8": "4",   "imp9": "4",   "imp10": "4",   "imp11": "1",   "imp12": "4",   "imp13": "4",   "imp14": "1",   "imp15": "4",   "imp16": "1",   "imp17": "1",   "imp18": "1",   "imp19": "1",   "imp20": "4",   "imp21": "1",   "imp22": "1",   "imp23": "1",   "imp24": "1", </pre>
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Data label	Description	Example
		<pre>"imp25": "2", "imp26": "1", "imp27": "1", "imp28": "1", "imp29": "4", "imp30": "4" }</pre>

Data label	Description	Example
Health	This is a data set which collects information regarding a player's self-assessed health (refer to Appendix 1)	<pre> {   "htha1": "3",   "htha2": "3",   "htha3": "3",   "htha4": "3",   "htha5": "3",   "htha6": "3",   "hthb1": "6",   "hthb2": "6",   "hthb3": "2",   "hthb4": "6",   "hthb5": "1",   "hthb6": "6",   "hthc1": "4",   "hthc2": "1",   "hthc3": "1",   "hthc4": "4",   "hthd1": "5",   "hthd2": "4" } </pre>

Data label	Description	Example
Perceived stress	This is a data set which collects information regarding a player's self-assessed perceptions of their stress levels (refer to Appendix 1)	<pre> {   "ps1": "3",   "ps2": "4",   "ps3": "4",   "ps4": "2" } </pre>

Data label	Description	Example
Depression, anxiety and stress	This is a data set which collects information regarding a player's self-assessed perceptions of their mental health levels (refer to Appendix 1)	<pre> {   "ds1": "1",   "ds2": "1",   "ds3": "1",   "ds4": "1",   "ds5": "1",   "ds6": "1",   "ds7": "1",   "ds8": "1",   "ds9": "1",   "ds10": "1",   "ds11": "1",   "ds12": "1",   "ds13": "1",   "ds14": "1",   "ds15": "1",   "ds16": "1",   "ds17": "1",   "ds18": "1",   "ds19": "1",   "ds20": "1",   "ds21": "1" } </pre>

<p>PTSD Checklist for DSM-5 with Life Events Checklist</p>	<p>This is a data set which collects information regarding a player's experience with adverse events (refer to Appendix 1)</p>	<pre>{   "pc1": [     "witness"   ],   "pc2": [     "na"   ],   "pc3": [     "na"   ],   "pc4": [     "na"   ],   "pc5": [     "na"   ],   "pc6": [     "na"   ],   "pc7": [     "na"   ],   "pc8": [     "na"   ], }</pre>
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		<pre>"pc9": [   "na" ], "pc10": [   "na" ], "pc11": [   "na" ], "pc12": [   "na" ], "pc13": [   "na" ], "pc14": [   "na" ], "pc15": [   "na" ], "pc16": [   "na" ], "pc17": [ </pre>
--	--	--

Data label	Description	Example
		"na"  ]  }



Data label	Description	Example
PTSD Checklist for DSM-5	This is a data set which collects information regarding a player's stressful thoughts or experiences (refer to Appendix 1)	<pre> {   "p3s1": "1",   "p3s2": "1",   "p3s3": "1",   "p3s4": "1",   "p3s5": "1",   "p3s6": "1",   "p3s7": "1",   "p3s8": "1",   "p3s9": "1",   "p3s10": "1",   "p3s11": "1",   "p3s12": "1",   "p3s13": "1",   "p3s14": "1",   "p3s15": "1",   "p3s16": "1",   "p3s17": "1",   "p3s18": "1",   "p3s19": "1",   "p3s20": "1" } </pre>

Data label	Description	Example
task_duration	This is the time spent, in minutes, to complete the whole experiment including the Dracoin Doors game and questionnaires	32.52
task_ended	This is a string representing the local time and date at completion of the experiment	Tue Nov 02 2021 07:51:27 GMT-0400 (Eastern Daylight Time)
Heart rate data	For the lab study, heart activity data was collected from a PPG sensor. The sensor connects to a DAQ and the data files can be generated using the DAQ's proprietary software. Heart rate sensor time series of sensor voltages and BPM estimates was collected in a comma delimited text file	.csv file content example:  113716_1681363529202_1_1.acq 1 msec/sample 2 channels Pulse - PPG, X, PPGED-R Volts Rate BPM min,CH1,CH2, ,907112,907112, 0,0.00183105,0, 1.66667E-05,0.00152588,0, 3.33333E-05,0.00213623,0, 5E-05,0.00213623,0, 6.66667E-05,0.00152588,0, .....

Data label	Description	Example
Start time of heart rate sensor recording	For the lab study, heart activity data was collected from a PPG sensor. The sensor connects to a DAQ and the data files can be generated using the DAQ's proprietary software. Heart events and the time of recording was collected in a .xls file	Extracted from the .xls file Start recording Thu Apr 13 2023 15:05:20

#### **A2.4 Data Processing Script Description**

For the online study, the JSON data collected (as described above) was extracted from a comma-separated value (CSV) file using the Python script 'read\_csv\_v1.py'. The CSV file was created and stored by the Amazon Turk Crowdsourcing platform following the submit button click event after completion of the experiment. The read\_csv\_v1.py script was created to parse through the CSV file and generate JSON files for each participant, and also a temporary summary CSV file (task\_summary.csv) that includes information regarding the participant, including when the task was created, the time it was accepted, the time it was submitted and the total duration of the task. Note participant information was de-identified for inclusion in the data analysis. However, it was stored internally within Adelaide University Drop Box platform. It was then deleted upon completion of the project.

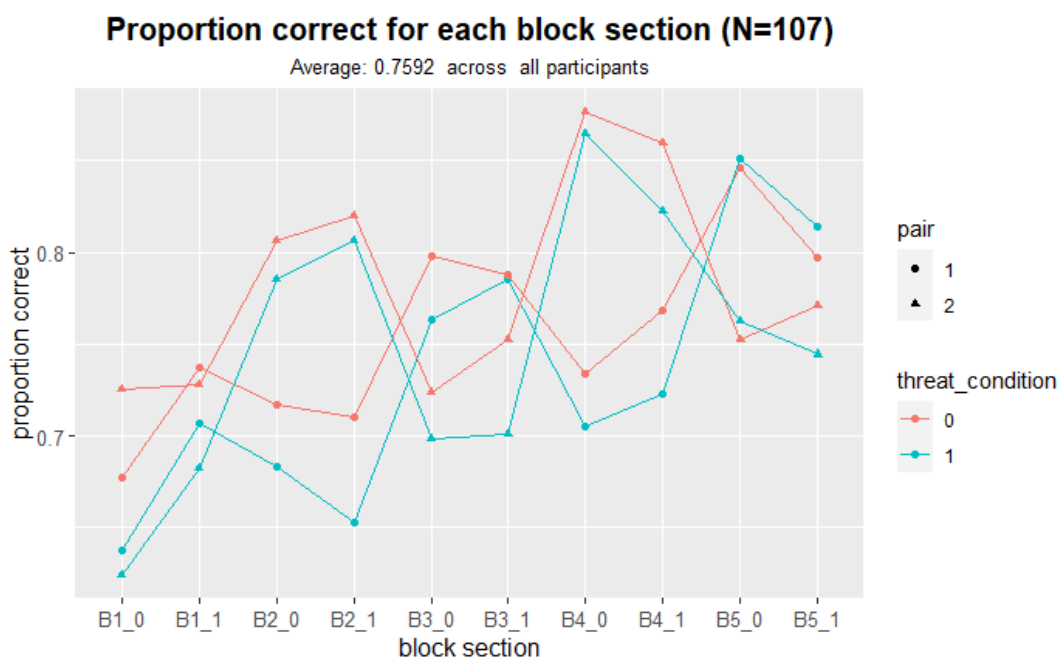
The JSON data collected for each participant was then validated using the Python script check\_results.py, which was created to generate a set of CSV files of data collected from each questionnaire. Each of these CSV files were then copied across to the Excel workbook data\_checks\_V1.xlsx which was created to validate the questionnaire data from participants. The validation method was based on determining consistency between scores of related measures and scores of opposing measures.

Each JSON file generated by read\_csv\_v1.py is then parsed to extract the choices made after playing the Dracoin Doors Game. The process\_game\_data.py script was created to do the extraction and to generate a CSV data set (choice\_scores.csv) that can then be used for data analysis.

For the lab study, the event\_timeseries.py python script is used to read each participant’s JSON file and merge this information with the heart monitoring sensor data collected as described in the above table. The script generates a CSV data set (heart\_rate.csv) that can then be used for data analysis.

### A2.5 Data Plots Script Description

The R Markdown file ‘choices\_plot.Rmd’ was created using RStudio to calculate win-stay and lose-switch, and mean BPM, RMSSD and BR under threat and safe conditions and to generate different data plots to visualise the data sets from the choice\_scores.csv and heart\_rate.csv files. Examples of the plots are shown below.



Note: File path location for files in A2.4 and A2.5: ..\Appendix2\Lab\_Experiment\_Data\_Processing

### Appendix 3

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2020	Aceto, G. et al.	Mice	unspecified	unspecified	unspecified	CUMS for 3 weeks	unspecified	Spike timing-dependent long-term potentiation (LTP) was induced in NAc MSNs, and the level of active glycogen-synthase kinase 3 $\beta$ (GSK3 $\beta$ ) was increased in depressed mice	2020_HB
2018	Zhong, P. et al.	Mice	unspecified	unspecified	unspecified	CUMS for 5 weeks	In vivo recording from the VTA: -2.9 to -3.3 mm AP; 0.6 to 1.1 mm ML; and -3.9 to -4.5 mm DV	Decreased population activity, the frequency of tonic and burst firing in VTA DAergic neurons.	2020_HB
2017	Der-Avakian et al.	Male Wistar Rats	unspecified	unspecified	unspecified	CSDS 3 days	unspecified	↓ Fos mRNA levels ↓ Response bias toward frequently rewarded stimulus (blunted reward learning) N/OFQ peptide and NOPR mRNA levels in VTA inversely related to reward learning; ↑ N/OFQ in striatum	2020_ED
2017	Qu et al.	Male C57BL/6 mice	unspecified	unspecified	unspecified	CSDS 10 days	unspecified	↑ Spine density in "SP" ↑ Social avoidance in "SP"; ↓ Sucrose preference in "SP"	2020_ED
2017	Kaska et al.	Mice	unspecified	unspecified	unspecified	CSDS 10 days	unspecified	↓ Soma size in "SP"; ↓ Active cofilin mTOR signaling dependent	2020_ED
2017	Francis, T. C. et al.	Mice (D1-Cre x RiboTag (D1-Cre-RT))	unspecified	unspecified	unspecified	CSDS for 10 days	unspecified	The expression of the transcription factor early growth response 3 (EGR3) was increased in the D1-MSNs of susceptible mice	2020_HB
2016	Sugama and Kakinuma	Male Wistar rats	unspecified	unspecified	unspecified	CRS 8h/day, 16 weeks	unspecified	↑ Microglial soma size	2020_ED

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2016	Holly et al.	Male Long-Evans rats	unspecified	unspecified	unspecified	ISDS 4 x SDS in 10 days	unspecified	↑ Phasic CRF in pVTA (acute stress); ↑ Phasic CRF in aVTA (chronic stress); ↑ Tonic CRF (aVTA & pVTA) ↑ Cocaine self-administration (CRF-R1 dependent in pVTA; CRF-R2 dependent in aVTA)	2020_ED
2016	Stelly et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	CSDS 10 Days	unspecified	↑ NMDAR-mediated LTP ↑ Cocaine-induced CPP LTP promoted by mGluR/IP3-induced intracellular Ca <sup>2+</sup> -release; GR signaling dependent	2020_ED
2016	Anacker et al.	Male C57BL/6 mice	unspecified	unspecified	unspecified	CSDS 10 days	unspecified	↑ VTA volume in "SP"	2020_ED
2016	Sugama et al.	Male Wistar rats	unspecified	unspecified	unspecified	CRS 8 h/day, 16 weeks	unspecified	↑ VTA-DA neuronal cell loss ↑ Microglial activation; ↑ Oxidative stress	2020_ED
2016	Sugama and Kakinuma	Male Wistar rats	unspecified	unspecified	unspecified	CRS 8 h/day, 16 weeks	unspecified	↑ VTA-DA neuronal cell loss	2020_ED
2016	Khibnik, L. A. et al.	Mice (Drd2-EGFP)	unspecified	unspecified	unspecified	CSDS for 10 days	unspecified	Resilient animals displayed an increase in synaptic strength at large mushroom spines of D1-MSNs and a concomitant decrease in synaptic strength at D2-MSNs	2020_HB
2015	Hernaus	healthy volunteers	4	8	Sensorimotor control task Montreal imaging stress task 1-day fixed order	Psychological stress	[18F]fallypride (D2/D3)	(↑) in medial PFC and temporal cortex, Positive association with subjective stress in ventromedial PFC in whole sample	2015_VT
2014	Dias, C. et al.	Mice (Drd1-EGFP and Drd2-EGFP)	unspecified	unspecified	unspecified	CSDS for 10 days	unspecified	β-catenin expression was upregulated in D2-MSNs in resilient mice but downregulated in susceptible animals	2020_HB
2014	Francis, T. C. et al.	Mice (Drd1-EGFP and Drd2-EGFP)	unspecified	unspecified	unspecified	CSDS for 10 days	unspecified	The frequency of excitatory synaptic inputs was decreased in D1-MSNs and increased in D2-MSNs	2020_HB

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2014	Chang, C. H. & Grace, A. A.	Rat	unspecified	unspecified	unspecified	CUMS for 4 weeks	In vivo recording from the VTA: -5.3 to -5.7 mm AP; -0.6 to -1.0 mm ML; and -6.5 to -9.0 mm DV	Decreased DA neuron population activity but no differences in average firing rate or percentage of spikes in bursts	2020_HB
2014	Friedman, A. K. et al.	Mice	unspecified	unspecified	unspecified	CSDS for 10 days	Slice recording	Increase in VTA DAergic neuron firing frequency in susceptible animals	2020_HB
2014	Watt et al.	SD rat	unspecified	unspecified	unspecified	acute, with three prior social defeats stress for Adolescent social defeat; 20-min exposure to resident sample length of 20 min	unspecified	max of 150 % DA from baseline with Increased during encounter, slowly returned to baseline by 60 min after termination increases at termination? a also	2016_HM
2014	ArriagaAvila et al.	Wistar rat, female	unspecified	unspecified	unspecified	Acute stress for Immobilization 30 min sample length of 15 min	unspecified	max of 200 %, n/a DA from baseline with Increased to 200 % in second half of stress in virgin females, returning to baseline by 45 min after termination. No effect observed in nonvirgins (lactating dams) increases at termination? a also	2016_HM
2014	Pecina	healthy volunteers	22	30	rest hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	(↓) in NAcc, caudate and putamen in whole sample	2015_VT
2014	Pecina	healthy volunteers	21	29	isotonic saline hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	No association with affective state or pain ratings	2015_VT
2014	Pecina	healthy volunteers	21	28	rest hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	Positive association with total, sensory and affective pain ratings in ventral striatum Mediates effect of BDNF on pain ratings in ventral striatum	2015_VT

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2013	Warren et al.	Male C57BL/6 J mice	unspecified	unspecified	unspecified	CSDS 10 Days	unspecified	↓ Gabrd ( $\delta$ -GABAAR) expression; ↑ GABAergic transmission ↑ Social avoidance; ↑ Immobility in forced swim stress; ↓ Exploration in EPM In VTA-GABA interneurons; regulated by neurosteroids	2020_ED
2013	Whitaker et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	SI P21-P42	unspecified	↑ NMDAR-mediated LTP ↑ Amphetamine- and ethanol-induced CPP LTP promoted by mGluR/IP3-induced intracellular Ca <sup>2+</sup> -release	2020_ED
2013	Bessa, J. M. et al.	Rats	unspecified	unspecified	unspecified	CUMS for 6 weeks	unspecified	Medium spiny neurons in the NAc were hypertrophied and showed increased expression of genes encoding brain-derived neurotrophic factor and neural cell adhesion molecule in depressed animals	2020_HB
2013	Lobo, M. K. et al.	Mice (Drd1-EGFP and Drd2-EGFP for the MSN study)	unspecified	unspecified	unspecified	CSDS for 10 days	unspecified	Depressed mice displayed a significant induction of $\Delta$ FosB in D2-MSNs in the NAc core, NAc shell, and dorsal striatum; resilient mice showed significant $\Delta$ FosB induction in D1-MSNs across all striatal regions	2020_HB
2013	Tye, K. M. et al.	Rat	unspecified	unspecified	unspecified	CUMS for 4–6 weeks (for the rest of the experiments the mice were exposed to CUMS for 8–12 weeks were used)	In vivo recording from the VTA: (AP), -5.8; (ML), $\pm$ 0.7; and (DV), -8.2 In vivo recording from the VTA in adult male rats (4–6 weeks of CUMS)	No change in firing rate but a decrease in the proportion of spikes occurring within bursts, the duration of bursts, and the number of spikes in each burst in the VTA neurons of stressed rats	2020_HB
2013	Chaudhury, D. et al.	Mice	unspecified	unspecified	unspecified	CSDS for 10 days	Slice recording	Significant increase in the firing rate in susceptible mice compared to control and resilient mice (VTA slices)	2020_HB



DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2013	Ventura et al. NMRI	outbred female mice	unspecified	unspecified	unspecified	Acute stress for Restraint 180 min sample length of 20 min	unspecified	max of 165 % DA from baseline with Remained elevated for 120 min of restraint increases at termination? Did not measure also	2016_HM
2013	Garrido et al.	Wistar rat	unspecified	unspecified	unspecified	Acute stress for Restraint 20 min sample length of 20 min	unspecified	max of 165 % DA from baseline with Immediate increase in response to stress, remained elevated, back to baseline by 40 min after termination increases at termination? a also	2016_HM
2013	Butts and Phillips	SD rat	unspecified	unspecified	unspecified	Acute stress for 15 min sample length of 15 min	unspecified	max of 225 % DA from baseline with Increase during stress, reduced upon termination and back to baseline by 30 min later increases at termination? a also GR antagonists prevented increase	2016_HM
2013	Naef et al.	SD rat	unspecified	unspecified	unspecified	Repeated 5 days stress for 30 min sample length of 15 min	unspecified	max of 175 %, 240 % DA from baseline with Day 1: immediate increase, slightly decreased after release, back to baseline following sample. day 5: sensitized response, peak during stressor, return to baseline 45 min after termination, but spiked again 90 min later increases at termination? a also	2016_HM

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2013	Mizrahi	healthy volunteers	5	7	Sensorimotor control task Montreal imaging stress task 2-day fixed order	Psychological stress	[11C]-(+)-PHNO (D2/D3)	Positive association with stress-induced cortisol release in associative striatum (AST) in whole sample Positive association with stress-induced cortisol in AST, sensorimotor striatum (SMST) and whole striatum in whole sample (2012 study with same volunteers)	2015_VT
2013	Hernaus	healthy volunteers	15	11	Sensorimotor control task Montreal imaging stress task 1-day fixed order	Psychological stress	[18F]fallypride (D2/D3)	(↑) superior and inferior frontal gyrus in whole sample (COMT Met carriers < non-Met carriers)	2015_VT
2013	Nagano-Saito	Healthy volunteers	11	0	Sensorimotor control task Montreal imaging stress task (2-day counterbalanced)	Psychological stress	[18F]fallypride (D2/D3)	(↑) in medial PFC/anterior cingulate cortex, Positive association with stress-induced increase in heartrate, No association with stress-induced cortisol	2015_VT
2012	Lighthall et al	Unspecified	24	23	BART	CPT	cortisol	-greater reward collection -faster decision speed -activation of the dorsal striatum and anterior insula -less reward collection and slower decision speed in women and decreased brain activation in above regions	2012_SB
2012	Youssef et al	Students	30	35	Moral dilemmas	TSST	cortisol	less utilitarian judgements stress response and utilitarian judgements were correlated	2012_SB
2012	Tanaka et al.	Male c57bl/6 mice	unspecified	unspecified	unspecified	CSDS 10 Days	unspecified	↑ Iba-1 immunoreactivity ↑ Social avoidance; ↓ Exploration in EPM	2020_ED

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2012	Lim, B. K., Huang, K. W., Grueter, B. A., Rothwell, P. E. & Malenka, R. C	Mice (Drd1-tdTomato and Drd2-EGFP)	unspecified	unspecified	unspecified	chronic restraint stress 3–4 h/day for 7–8 days	unspecified	The strength of excitatory synapses on D1-MSNs in the NAc core was decreased	2020_HB
2012	Mickey	healthy volunteers	22	32	rest hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	(↑) in NAcc, caudate and putamen in whole sample Positive association with circulating leptin in ventral striatum and dorsal striatum in entire sample	2015_VT
2012	Love	healthy volunteers	23	32	rest hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	(↑) in the ventromedial caudate in whole sample Negative association with emotional well-being in ventromedial caudate in women Positive association with trait anxiety scores in ventromedial caudate in men	2015_VT
2012	Burghardt	healthy volunteers	22	28	rest hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	(↑) in NAcc, caudate and putamen in whole sample Positive association with circulating leptin in ventral striatum and dorsal striatum in entire sample	2015_VT
2012	Suridjan	healthy volunteers	7	4	Sensorimotor control task Montreal imaging stress task 2-day fixed order	Psychological stress	[11C]-(+)-PHNO (D2/D3)	Negative association with angry-hostile personality trait in AST Negative association with vulnerable personality trait in limbic striatum (LST) Negative association with depressive personality trait in globus pallidus (GP) Negative association with openness to values personality trait in GP and substantia nigra	2015_VT

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2011	Miczek et al.	Male Long-Evans rats	unspecified	unspecified	unspecified	CSDS 36 Days	unspecified	↓ BDNF expression ↔ Cocaine-induced locomotion; ↓ Cocaine selfadministration; ↓ Exploration in OFT; ↓ Sucrose preference	2020_ED
2011	Miczek et al., 2011)	Male Long-Evans rats	unspecified	unspecified	unspecified	ISDS 4 x SDS in 10 days	unspecified	↑ BDNF expression ↑ Cocaine-induced locomotion; ↑ Cocaine selfadministration; ↔ Exploration in OFT; ↔ Sucrose preference	2020_ED
2011	Christoffel, D. J. et al.	Mice	unspecified	unspecified	unspecified	CSDS for 10 days	unspecified	mEPSC frequency was increased, and this increase was associated with significant increases in IκB kinase expression in the NAc in susceptible (depressed) animals	2020_HB
2011	Valenti, O., Lodge, D. J. & Grace, A. A	Rats	unspecified	unspecified	unspecified	chronic restraint stress 1 h/day for 10 days	In vivo recording from the VTA: -5.3 anteroposterior (AP); -0.6 mediolateral (ML); and -6 to -9 mm dorsoventral (DV)	Increase in DA neuron population activity, no change in firing rate	2020_HB
2011	Butts et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 15 min sample length of 15 min	unspecified	max of 300 % DA from baseline with Immediate increase, gradual return to baseline by 90 min after termination of stressor increases at termination? a also GR antagonism in the LV prevented increase	2016_HM
2011	Lataster	healthy volunteers	8	4	Sensorimotor control task Montreal imaging stress task 1-day fixed order	Psychological stress	[18F]fallypride (D2/D3)	(↑) in ventromedial PFC Positive association with subjective stress in ventromedial PFC No association with stress-induced cortisol	2015_VT
2011	Starcke et al	Students	22	18	Moral dilemmas	TSST	- cortisol - alpha-amylase - STAI	cortical reactions and egoistic decisions were correlated	2012_SB

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2010	Tynan et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	CRS 2x 30 min/day, 14 days	unspecified	↔ Iba-1 immunoreactivity ↓ Sucrose preference	2020_ED
2010	Gersner et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	CUS 38 Days	unspecified	↔ BDNF levels ↔ Locomotion; ↓ Exploration; ↔ Immobility in forced swim stress; ↓ Sucrose preference	2020_ED
2010	Fanouos et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	ISDS 4 x SDS in 10 days	unspecified	↑ BDNF expression	2020_ED
2010	Vialou, V. et al.	Mice	unspecified	unspecified	unspecified	CSDS for 10 days	0	Resilient mice showed the greatest induction of ΔFosB in both the core and shell of the NAc	2020_HB
2010	Cao, J. L. et al.	Mice	unspecified	unspecified	unspecified	CSDS for 10 days	In vivo recording from the VTA: -2.92 to -3.88 AP; 0.24 to 0.96 ML; and -3.5 to -4.5 DV.	Increase in spontaneous firing rates and bursting events of VTA DA neurons in vivo in susceptible mice	2020_HB
2010	Putman et al	Students	29	0	Modified CGT	Application of cortisol	- cortisol - STAI	risky choices when rewards were high	2012_SB
2009	van den Bos et al	Students and uni staff	30	34	IGT	TSST	cortisol	cortisol reactions and dysadvantegous decisions were correlated in males and in femals it followed a U- shape relation	2012_SB
2009	Mather et al	Younger and older adults	43	42	Driving task	CPT	cortisol	risk adversion associated with dysfunctional decisions in older adults	2012_SB
2009	Lighthall et al	Unspecified	22	23	BART	CPT	cortisol	increased risk taking in men decreased risk taking in women	2012_SB
2009	Porcelli and Delgado	Students	14	13	Modified CGT	CPT	SCL	conservative choices on gain trials more risky choices in loss trials	2012_SB
2009	Kassam et al	Unspecified	32	71	Anchoring and adjustment	Modified TSST	- heart rate - blood pressure	decrease in adjustment	2012_SB

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2008	Krishnan et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	CSDS 10 Days	unspecified	↓ p-Akt in "SP"; ↓ GABAergic transmission ↑ Social avoidance; ↑ Immobility in forced swim stress; ↓ Sucrose preference Involves PI3K/Akt/mTOR signaling	2020_ED
2008	Covington et al.	Male Long-Evans rats	unspecified	unspecified	unspecified	ISDS 4 x SDS in 10 days	unspecified	↔ NMDAR expression; ↑ GluR1 expression ↑ Cocaine self-administration	2020_ED
2008	Toth et al.	Male & Female Sprague-Dawley rats	unspecified	unspecified	unspecified	CUS 4 Weeks	unspecified	↔ GluR1 levels (aVTA and pVTA); ↔ BDNF levels (aVTA and pVTA) ↓ Sucrose preference; ↓ Exploration	2020_ED
2008	Soliman	healthy volunteers	1	9	Sensorimotor control task Montreal imaging stress task 2-day counter balanced	Psychological stress	[11C]raclopride (D2/D3)	Negative association with maternal care score in whole sample No association with stress-induced cortisol	2015_VT
2008	Starcke et al	Students	18	22	GDT	Anticipated speech	- cortisol - alpha-amylase - STAI	disadvantageous choices correlation between risky decisions and cortisol reactions	2012_SB
2007	Krishnan et al.	Male c57bl/6 mice	unspecified	unspecified	unspecified	CSDS 10 Days	unspecified	↑ BDNF in "SP"; ↑ K <sup>+</sup> -channels in "RP" ↑ Social avoidance in "SP"; ↓ Sucrose preference in "SP"	2020_ED
2007	Jeziarski et al.	juvenile degu	unspecified	unspecified	unspecified	acute stress for 60-min isolation, with or without 3 weeks daily maternal separation sample length of 20 min	unspecified	max of 171 %, 146 % DA from baseline with Larger increase in control compared to early separation group, both groups returned to baseline immediately upon reunion increases at termination? No also Chronic methylphenidate cross-sensitizes	2016_HM

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
2007	Del Arco et al.	Wistar rat	unspecified	unspecified	unspecified	acute stress for 40-min handling sample length of 20 min	unspecified	max of 150 % DA from baseline with Increase during handling, remained elevated at release, return to baseline by 40 min after termination increases at termination? a also No effects of prior environmental enrichment	2016_HM
2007	Mokler et al.	SD rat	unspecified	unspecified	unspecified	Acute, some with prior prenatal malnourishment stress for Restraint 20 min sample length of 20 min	unspecified	max of 150 %, n/a DA from baseline with Controls 150 % during stress, immediately back to baseline on termination. Malnourished did not increase dopamine during stress, but were significantly attenuated 100–160 min after release increases at termination? No also	2016_HM
2007	Scott	healthy volunteers	10	7	isotonic saline hypertonic saline  rest – HTS  ITS – HTS  1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	(↑) in dorsal caudate and putamen Positive association with subjective pain ratings in dorsal caudate and putamen  (↑) in contralateral NAcc  Positive association with negative affect and fear ratings in NAcc	2015_VT
2007	Wood	healthy volunteers	0	11	isotonic saline hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	(↑) in GP, putamen and caudate in HV Positive association with pain rating and whole striatum	2015_VT
2007	Scott	healthy volunteers	4	0	rest hypertonic saline 1-day fixed order	Pain stress	[11C]raclopride (D2/D3)	(↑) in dorsal caudate	2015_VT

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2007	Zellneer et al	Students	36	0	Food selection	Unsolvable anagrams	Questionnaire	preference for healthy food	2012_SB
2007	Preston et al	Students and uni staff	20	20	IGT	Anticipated speech	heart rate	slower learning dysadvantegous decisions in men advantegous decisions in women	2012_SB
2006	Pehek et al.	SD rat	unspecified	unspecified	unspecified	acute stress for 20-min handling sample length of 20 min	unspecified	max of 182 % DA from baseline with Increased during handling, immediate return to baseline increases at termination? No also	2016_HM
2006	Renoldi and Invernizzi	CD-COBS rats, Mongolian Gerbils	unspecified	unspecified	unspecified	Acute stress for Immobilization 40 min sample length of 20 min	unspecified	max of 188 %, 31-6 % DA from baseline with Rats showed immediate increase during immobilization, which remained elevated 40 min after stressor termination. Gerbils showed immediate increase, peaking in second half of stressor presentation, and returning to baseline 40 min after stressor termination increases at termination? a also	2016_HM
2006	Montgomery	healthy volunteers	9	5	Counting backwards Subtraction task 1-day fixed order	Psychological stress	[11C]raclopride (D2/D3)	No association with stress-induced cortisol	2015_VT
2006	Zellneer et al	Students	0	34	Food selection	Unsolvable anagrams	Questionnaire	preference for unhealthy food	2012_SB
2006	Takahashi et al	Students	31	0	Dictator game	Social evaluation	alpha-amyl	more generous decisions in participants with stress reactions	2012_SB
2005	Nikulina et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	CSDS 5 Days	unspecified	↑ MOR mRNA expression; ↓ GABAergic transmission ↑ MOR agonist-induced locomotor activity	2020_ED
2004	Perrotti, L. I. et al.	Rats	unspecified	unspecified	unspecified	chronic restraint stress 1 h/day for 10 days	unspecified	ΔFosB was induced in both dynorphin-positive (D1-MSNs) and enkephalinpositive (D2-MSNs) by stress	2020_HB



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2004	Swanson et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for Immobilization 30 min sample length of 30 min	unspecified	max of 189 % DA from baseline with Increased to 150 % during stress, but peaked at 189 % after termination, with gradual return to baseline by 90 min after initiation of stressor increases at termination? Yes also mglu2/3 agonist blocks increases in both dopamine and noradrenaline	2016_HM
2004	Dazzi et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 0.2 mA for 500 ms every second for 8 min sample length of 20 min	unspecified	max of 190 % DA from baseline with Increased during stress, immediately returning to baseline in next sample increases at termination? Sample included both stress and termination also 2-week olanzapine or clozapine prevented or significantly inhibited, respectively, stressinduced DA increase; haloperidol had no effect	2016_HM
2004	Jackson and Moghaddam	SD rat	unspecified	unspecified	unspecified	Repeated after 3 h stress for Restraint 10 min sample length of 10 min	unspecified	max of 140 % DA from baseline with Immediate increase during first exposure, sustained for one sample after termination, then back to baseline. Second exposure showed habituated da response increases at termination? a also	2016_HM
2004	Jackson and Moghaddam	SD rat	unspecified	unspecified	unspecified	Twice, 3 h apart stress for 10 min sample length of 10 min	unspecified	max of 125 % DA from baseline with Both exposures showed similar increase, peaking at 20 and 30 min, return to baseline by 60 min, increase at termination increases at termination? Yes also	2016_HM

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2003	Wu et al.	SD rat	unspecified	unspecified	unspecified	acute stress for Predator odor (fox) for 20 min sample length of 20 min	unspecified	max of 205 % DA from baseline with Gradual increase in dopamine that was maximal 120 min after beginning of odor presentation increases at termination? a also	2016_HM
2003	Bland et al.	SD rat	unspecified	unspecified	unspecified	Acute, escapable (ES) or inescapable (IS) stress for 1.0 mA, 100 trials, ITI avg 60 s, terminated by escapable shock (ES) rat turning wheel sample length of 20 min	unspecified	max of 150 %, 275 % DA from baseline with ES showed initial immediate increase to 150 %, returning to baseline after the first sample. IS increased to 150 % initially, peaking at 275 % subsequently and gradually returned to baseline by 200 min after initiation of stress increases at termination? No also	2016_HM
2003	Jedema and Grace	SD rat	unspecified	unspecified	unspecified	Acute stress for 20 min sample length of 20 min	unspecified	max of 180 % DA from baseline with Increased during stress, peaked immediately after termination, returned to baseline by 60 min after termination increases at termination? a also AP5 did not blunt response, but CNQX did	2016_HM
2003	Murphy et al.	SD rat	unspecified	unspecified	unspecified	Acute, with prior 14– 20-day chronic cold exposure stress for 1.0-mA constant pulse for 1 s every 10 s for duration of 45 s, repeated every 5 min for 30 min sample length of 15 min	unspecified	max of 183 %, 258 % DA from baseline with Naïve rats immediately increased mPFC DA (183 %), returning to baseline immediately upon shock termination. Prior CCE rats: immediate increase to 258 %, while also immediately returning to baseline on termination increases at termination? No also ICV CRF antagonist did not alter evoked dopamine increase, but attenuated CRF-induced dopamine increase	2016_HM

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2002	Page and Lucki	SD rat	unspecified	unspecified	unspecified	Acute stress for 20 min sample length of 20 min	unspecified	max of n/a DA from baseline with No change increases at termination? n/a also	2016_HM
2002	Pozzi et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for Immobilization 120 min sample length of 20 min	unspecified	max of 250 % DA from baseline with Immediate maximal increase, returned to baseline within 100 min; increase again 20– 60 min after release, although not as high as before increases at termination? Yes also	2016_HM
2002	Matuszewich et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for Immobilization 60 min sample length of 20 min	unspecified	max of 175 % DA from baseline with Immediate maximal increase during first 20 min, then back to baseline for duration increases at termination? No also MDMA pretreatment blocked effect	2016_HM
2002	Marsteller et al.	SD rat	unspecified	unspecified	unspecified	acute stress for 15-min handling sample length of 15 min	unspecified	max of 155 % DA from baseline with Increase during handling, peak after cessation, rapid return to baseline increases at termination? a also	2016_HM
2001	Cuadra et al.	Wistar rat	unspecified	unspecified	unspecified	Acute, 1 week chronic variable stress stress for Restraint 60 min sample length of 30 min	unspecified	max of 139 %, 18-9 % DA from baseline with Without CVS, dopamine increased gradually during restraint, peaking (139 %) and sustained for duration of sampling. CVS group also increased gradually and peaked (189 %) 30 min after termination without returning to baseline increases at termination? a also	2016_HM

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2001	Del Arco et al.	Wistar rat	unspecified	unspecified	unspecified	acute stress for 40-min handling sample length of 20 min	unspecified	max of 189 % DA from baseline with Increased during handling, immediate return to baseline after termination increases at termination? No also	2016_HM
2001	Del Arco and Mora	Wistar rat	unspecified	unspecified	unspecified	acute stress for 40-min handling sample length of 20 min	unspecified	max of 200 % DA from baseline with Increase sustained during handling, decreased slightly at termination and return to baseline by 20 min after release increases at termination? a also No effects on GABA or glutamate in mPFC	2016_HM
2001	Dazzi et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 0.2 mA for 500 ms every second for 8 min sample length of 20 min	unspecified	max of 190 % DA from baseline with Increased during stressor, immediately returned to baseline increases at termination? Sample included both stress and termination also 2-week imipramine or mirtazapine inhibited or prevented (respectively) stress-induced DA increase.	2016_HM
2001	Dazzi et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 0.2 mA for 500 ms, every 2, for 8 min sample length of 20 min	unspecified	max of 190 % DA from baseline with Immediate increase during stress, no longer statistically significant 10 min later increases at termination? Sample included both stress and termination also 2-week (but not single dose) imipramine or mirtazapine reduced and completely antagonized (respectively) increase in DA during footshock	2016_HM

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2001	Feenstra et al.	Wistar rat	unspecified	unspecified	unspecified	Acute stress for Aversive conditioning: 10-s white noise (25 dB) immediately followed by 0.3-mA footshock repeatedly presented 9x (conditioned group), or non-paired presentations (pseudo group) or no conditioning (control group). Later tested just CS sample length of 16 min	unspecified	max of 250 %, 200 %, n/a DA from baseline with Significantly increased immediately in aversive conditioning (250 %) and pseudo conditioning (200 %) groups, gradually returning to baseline, with no changes in control group. Presentation of CS alone resulted in 150 % increase in aversive group only increases at termination? a also	2016_HM
2000	Adler	healthy volunteers	6	0	rest 2-deoxyglucose 1-day fixed order	Metabolic stress	[11C]raclopride (D2/D3)	(1) in whole striatum	2015_VT
2000	Oliver et al	Students and uni staff	27	41	Food selection	Anticipated speech	-heart rate -blood pressure -PANAS	-sweet food eaten by stressed emotional eaters -high-fat food by non-emotional eaters and unstressed	2012_SB
1999	Cuadra et al.	Wistar rat	unspecified	unspecified	unspecified	Acute, with 1 week of chronic variable stress for Restraint 60 min sample length of 30 min	unspecified	max of 146 %, 17-7 % DA from baseline with No CVS group increased dopamine beginning at 60 min, with maximal increase at 120 min, never returning to baseline. CVS group showed maximal (177 %) increase at 120 min, returning to baseline at 300 min. increases at termination? a also Reversed by naloxone	2016_HM
1999	Inglis and Moghaddam	SD rat	unspecified	unspecified	unspecified	acute stress for 20-min handling sample length of 20 min	unspecified	max of 150 % DA from baseline with Immediate increase, sustained 20 min after release increases at termination? a also	2016_HM

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1999	Mendlin et al.	SD rat	unspecified	unspecified	unspecified	Acute, repeated once stress for 20 min sample length of 20 min	unspecified	max of 144 % DA from baseline with Immediate increase, returned to baseline 40 min after sample termination increases at termination? a also Raclopride augmented effect	2016_HM
1999	Kawahara et al.	Wistar rat	unspecified	unspecified	unspecified	acute stress for 10-min handling sample length of 15 min	unspecified	max of 175 % DA from baseline with Increased during handling, slow return to baseline increases at termination? a also Intravenous infusion of sodium nitroprusside (induces hypotension) also potently increases mPFC DA	2016_HM
1999	Venator et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 30 min sample length of 15 min	unspecified	max of 200 % DA from baseline with Immediate increase, remained elevated after cessation, returning to baseline 60 min later increases at termination? a also	2016_HM
1999	Lillrank et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 30 min sample length of 15 min	unspecified	max of 130 % DA from baseline with No changes during restraint, peak only observed 60 min after termination increases at termination? Yes also NAc core, not shell, and probe too long (included more than core)	2016_HM

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1999	Di Chiara et al.	SD rat	unspecified	unspecified	unspecified	Acute, repeated once, one group with prior CMS stress for 10 min sample length of 10 min	unspecified	max of 175 %, 225 % DA from baseline with Controls showed significant increase (175 %) during tail pinch, slowly decreasing back to baseline by 30 min after release, same time course and magnitude with second m pinch. CMS animals showed significantly greater magnitude (225 %) with similar time course increases at termination? a also	2016_HM
1999	Di Chiara et al.	SD rat	unspecified	unspecified	unspecified	Acute, one group with 4wks CMS stress for 10 min, repeated after 120 min sample length of 10 min	unspecified	max of 75 %, 130 % DA from baseline with Non-stressed showed 25 % decrease immediately after tail first tail pinch, no change after second. prior CMS peak DA during first tail pinch, returned to baseline 80 min after release, similar effect during second tail pinch increases at termination? a also	2016_HM
1998	Takahata and Moghaddam	SD rat	unspecified	unspecified	unspecified	acute stress for 20-min handling sample length of 20 min	unspecified	max of 150 % DA from baseline with Increased during handling, immediate return to baseline after termination increases at termination? No also Blockade of AMPA and NMDA receptors in the VTA during handling reduced dopaminergic response	2016_HM
1998	Azzi et al.	Wistar rat	unspecified	unspecified	unspecified	acute stress for 10-min forced swim sample length of 20 min	unspecified	max of n/a DA from baseline with Marginal increase, sustained at least 200 min, but does not report baseline increases at termination? a also Repeated administration of neurotensin antagonist has no effect	2016_HM

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1998	RougePont et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 10 min sample length of 20 min	unspecified	max of 130 % DA from baseline with Immediate rise during stress, gradual decrease back to baseline increases at termination? a also Blocking corticosterone decreased stressinduced DA release	2016_HM
1998	Feenstra et al.	Wistar rat	unspecified	unspecified	unspecified	acute stress for 16-min handling sample length of 15 min	unspecified	max of 300 % DA from baseline with Peaked during handling, gradual return to baseline by 60 min after release increases at termination? a also Local inhibition (reverse dialysis) of ionotropic glutamate receptors did not affect handling induced corticosterone, dopamine, or noradrenaline release, nor did an mGluR antagonist or GABAB agonist	2016_HM
1998	Enrico et al.	Wistar rat	unspecified	unspecified	unspecified	acute stress for 15-min handling sample length of 15 min	unspecified	max of 225 % DA from baseline with 150 % during stress, increased to maximal 225 % after release, gradually decreased back to baseline by 90 min after termination increases at termination? a also Intra-VTA baclofen, CPP, AP5, CNQX suppressed handling induced increases, while intra-VTA muscimol, atropine, mecamylamine, and + -HA-966 did not	2016_HM



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1997	Petty et al.	SD rat	unspecified	unspecified	unspecified	repeated once stress for 8-min forced swim, repeated 24 h later sample length of 30 min	unspecified	max of n/a, 200 % DA from baseline with Day 1: no effect on dopamine. Day 2: increased to 200 % during stress, peaked after termination at approximately 300 %, sustained for 90 min increases at termination? a also Flumazenil increased stress response on day 1; diazepam attenuated stress response on day 2	2016_HM
1997	Merali et al.	SD rat	unspecified	unspecified	unspecified	acute stress for Airpuff and/or cytokine (IL-8) injection sample length of 30 min	unspecified	max of n/a DA from baseline with No effect increases at termination? n/a also	2016_HM
1997	King et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 30 min sample length of 15 min	unspecified	max of 120 % DA from baseline with Peaked during tail pressure, slow return to a baseline after removal increases at termination? also No change in NAc core; DA efflux potentiated with mPFC lesions	2016_HM
1996	Fitzgerald et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	CUS 10 Days	unspecified	↑ NMDAR1 and GluR1 expression	2020_ED
1996	Fitzgerald et al.	Male Sprague-Dawley rats	unspecified	unspecified	unspecified	CRS 45 Min/day for 10 days	unspecified	↑ GluR1 expression	2020_ED

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1996	Wedzony et al.	Wistar rat	unspecified	unspecified	unspecified	Acute stress for 0.5 mA/200 ms for 5 s twice during one 25-min session, then removal, brought back to context 25 min later with no shocks sample length of 25 min	unspecified	max of 150 %, 140 % DA from baseline with Increase to 150 % during footshock, immediately returning to baseline, and increase to 140 % basal levels in response to context increases at termination? no also Diazepam decreased outflow and blunted conditioned stress response. Ipsapirone and buspirone abolished stress-evoked elevation in dopamine	2016_HM
1996	Tidey and Miczek	Long Evans rat	unspecified	unspecified	unspecified	acute, with prior history of 4 social defeats stress for Social threat; 40 min in aggressor homecage without aggressor, 60 min with aggressor behind screen, 40 min again with no aggressor sample length of 20 min	unspecified	max of 160 % DA from baseline with Initial response to cage without aggressor (136 %), with peak in response to introduction of aggressor (162 %), returned to 130 % when aggressor was removed, and increased again (148 %) when returned to homecage increases at termination? Yes, not seen in controls also	2016_HM
1996	Klitenick et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 10 min sample length of 10 min	unspecified	max of 121 % DA from baseline with Increase during and sample after release, a gradual return to baseline increases at termination? also Corticosterone increased DA response by 50 %	2016_HM

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1996	Motzo et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 0.2 mA for 500 ms, every second, for 8 min sample length of 10 min	unspecified	max of 165 % DA from baseline with Immediate rise to 125 % during footshock, peaking at termination, returning to baseline 30 min after termination increases at termination? a also IC Vallopregnalone and midazolam dose dependently reduced basal DA and prevented stress-induced DA increase, midazolam with a greater potency	2016_HM
1995	Finlay et al.	SD rat	unspecified	unspecified	unspecified	Acute, with prior chronic cold exposure stress for 30 min sample length of 30 min	unspecified	max of 154 % DA from baseline with Increased during stressor, remained elevated for 30 min after termination, no difference between controls and CCE Diazepam decreased basal DA and attenuated stress evoked increase in control rats only (no effect of diazepam in CCE group)	2016_HM
1995	Dazzi et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 0.2 mA for 500 ms every second for 8 min sample length of 10 min	unspecified	max of 190 % DA from baseline with Initial increase to 140 % baseline, peaking at termination, returning to baseline 20 min after termination. Repetition one hour later resulted in smaller increase (125 %)	2016_HM
1994	Jordan et al.	SD rat	unspecified	unspecified	unspecified	repeated once stress for 8-min forced swim, repeated 24 h later sample length of 30 min	unspecified	max of n/a, 441 % DA from baseline with No effect on day 1, but second day significant increase, persisting for 60 min after termination increases at termination?	2016_HM

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1994	Gresch et al.	SD rat	unspecified	unspecified	unspecified	acute, with 17–28-day prior chronic cold exposure stress for 1.0-mA pulses for 1 s every 10 s for duration of 45 s, repeated every 5 min for 30 min sample length of 30 min	unspecified	max of 150 %, 271 % DA from baseline with Immediate increases in naïve (150 % max) and CCE (271 %), sustained for 60 min after termination increases at termination?	2016_HM
1993	Sorg and Kalivas	SD rat	unspecified	unspecified	unspecified	Acute stress for 0.55 mA/200 ms/s, 20 min sample length of 20 min	unspecified	max of 200 % DA from baseline with Initial increase to 150 % baseline, 200 % in sample after termination, returning to baseline 40 min after termination increases at termination? also Cocaine pre-treatment abolished stress-induced DA response, and footshock reduced response to subsequent acute cocaine	2016_HM
1993	Hamamura and Fibiger	Wistar rat	unspecified	unspecified	unspecified	Acute, with possible prior injection stress (14 days) stress for 0.4 mA, 10-s duration, 50-s interval, 20 min sample length of 20 min	unspecified	max of 225 % DA from baseline with Immediate increase during footshock, slowly returning to baseline by 40 min after termination increases at termination? a also	2016_HM
1993	Imperato et al.	SD rat	unspecified	unspecified	unspecified	Acute and repeated stress for 120 min, with 5 prior days of 60 min sample length of 10 min	unspecified	max of CTRL 150 %, prev. stress 70 % DA from baseline with Previously non-stressed: immediate increase, peaks at 20 and 30 min, gradual return to baseline by 50 min. Previously stressed: initially stay at baseline, drop below baseline 80–120 min into restraint increases at termination? Not measured also	2016_HM
1992	Cenci et al.	SD rat, female	unspecified	unspecified	unspecified	acute stress for 15-min handling sample length of 15 min	unspecified	max of n/a DA from baseline with No effect increases at termination? n/a also	2016_HM

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
1992	Imperato et al.	SD rat	unspecified	unspecified	unspecified	Repeated stress for 60 min for 6 consecutive days, repeated after 3 days sample length of 10 min	unspecified	max of 150 % DA from baseline with Day 1: immediate rise, peak at 20 min, gradual decrease towards baseline, but increase at release. Days 2, 3, and 4: blunted initial response, no change at termination response. Day 7: same as day 1 increases at termination? Yes also Decrease in dopaminergic tone as well	2016_HM
1991	Imperato et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for Restraint 120 min sample length of 10 min	unspecified	max of 180 % DA from baseline with Immediate increase, peaking 30 min into restraint, and returning to baseline after 90 min. Increase again at termination increases at termination? Yes also Looked at corticosterone—adrenalectomy had no effect, and exogenous corticosterone did not affect dopamine release	2016_HM
1991	Puglisi-Allegra et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 240 min sample length of 10 min	unspecified	max of 140 % DA from baseline with Immediate increase, peaked at 30 min, gradual return to baseline by 80 min, increase at release increases at termination? Yes also	2016_HM
1991	Imperato et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 120 min sample length of 10 min	unspecified	max of 150 % DA from baseline with Immediate increase, peaked at 30 min, gradual decrease to baseline by 80 min, increase at release increases at termination? Yes also Exogenous corticosterone did not increase DA	2016_HM

DATE	AUTHOR	SAMPLE	#MALES	#FEMALES	DM TASK	STRESSORS	MEASURE METHODS	OBSERVATIONS	REVIEW SOURCE
1991	Driskell and Salas	Students	78	0	Team decision-making in ambiguous checkboard task	Announced tear gas drill	Questionnaire	relied most on judgement of others	2012_SB
1990	Imperato et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 120 min sample length of 10 min	unspecified	max of 150 % DA from baseline with Immediate increase, peaked at 30–40 min, returned to baseline by 80 min increases at termination? Not measured also Prevented by 5HT3 antagonist but not diazepam	2016_HM
1989	Abercrombie et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 1.0-mA pulses for 1 s every 10 s for duration of 1 min, repeated every 5 min for 30 min sample length of 20 min	unspecified	max of 195 % DA from baseline with Immediate increase, peaking in 2nd half of stressor immediately returned to baseline after termination increases at termination? No also	2016_HM
1989	Imperato et al.	SD rat	unspecified	unspecified	unspecified	Acute stress for 90 min sample length of 10 min	unspecified	max of 145 % DA from baseline with Immediate increase, returned to baseline by 70 min increases at termination? Not measured also Corticosterone also increased DA	2016_HM
1987	Keinan	Students	42	59	Analogies task	Threat of electric shock	STAI	decreased performance premature closure non-systematic scanning of alternatives	2012_SB

*Notes:*

Review source coding:

[2020\_ED]: Douma and De Kloet, 2020

[2020\_HB]: Baik, 2020

[2016\_HM]: Holly and Miczek, 2016

[2015\_VT]: Vaessen et al., 2015

[2012\_SB]: Starcke and Brand, 2012