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Predictive Processing in Social Decisions - Through the Lens of a Gloomy Perspective

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Maria-Andreea Calin BSc

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Betreut von | Supervisor:	Univ.-Prof. Mag. Dr. Claus Lamm
Mitbetreut von | Co-Supervisor:	Bianca Schuster BSc MSc PhD

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Abstract English:

Increasing prevalence of depressive states in populations, coupled with recent political turbulences around the world should alarm us to investigate how human beings make decisions under conditions of uncertainty. Investigating the topic of depression from a predictive processing perspective is nested on the premise of the brain as a hierarchical, active organ that constantly aligns prior knowledge with current sensory information to make optimal decisions. The problem with depression appears to be two-fold: a high precision to negative priors couple with an impaired belief-updating towards positive information. In this study participants had to make social decision in a poker-like game, where prior and sensory uncertainty was manipulated. However, the claimed assumption of a biased underlying predictive processing inherent to depressive symptoms could not been replicated.

Abstract German:

Eine zunehmende depressive Symptomatik in Verbindung mit globalen politischen Krisensituationen gibt aktuellen Anlass zu fragen: Wie treffen Menschen Entscheidungen unter Unsicherheit?

Ein Ansatz aus der Perspektive des „Predictive Processing“ betrachtet Depression unter der Annahme, dass das menschliche Gehirn ein aktives Organ ist, das sensorische Inputs kontinuierlich mit bereits bestehenden Erwartungen und Kognitionen abgleicht, um optimale Entscheidungen zu treffen. Frühere Forschung deutet darauf hin, dass diese Verarbeitungsstrategie bei Menschen mit depressiven Tendenzen durch zwei Faktoren beeinflusst wird: Einerseits, eine verstärkte Gewichtung negativer Vorinformationen und anderseits ein schnelleres Integrieren von negativen Feedback in deren Entscheidungen. In der vorliegenden Studie trafen die Teilnehmenden Entscheidungen in einem pokerähnlichen Spiel für Protagonist*innen auf Basis dieser Informationsquellen: Bereitgestellt Hintergrundinformationen über die Charaktereigenschaften der Spieler*innen, sowie unmittelbares Feedback nach jeder gesetzten Entscheidung. Die Hypothese einer verzerrten Entscheidungsstrategie aufgrund einer verstärkten Beeinflussung durch negative „priors“ oder durch aufgrund eines höheren Updatens in Richtung negativer „prediction error“ konnte in dieser Studie jedoch nicht bestätigt werden.

Theoretical Background

Prevalence numbers of depressive states have risen by more than 20 % compared to pre-covid times measured in the year 2020. Empirical research (Majumdar, Majumar, Biswas & Sahu, 2020; Fancourt, Steptoe & Bu, 2020; Öztürk Çopur & Karasu, 2022, Dinhof et al., 2024) alongside statistical reports by Statistics Austria (Mühlböck et al., 2023) highlighted growing problem areas in that period revealing aspects such as sleep disruption, social isolation, somatic pain, and any increased screen exposure accompanied by elevated anxiety and depressive symptoms.

Considering last years´ turbulences in the world, such as the COVID pandemic or even more recently, political turbulences in the Middle East and Europe, it is of upmost urgency to reflect upon the way human beings make decisions and act accordingly in times of uncertainty? Are there any depression-related disparities in terms of cognitive and behavioural styles in conditions of uncertainty? And could an underlying mechanism possibly explain the difference between those who do and do not experience depressive symptoms? Several empirical studies have foregrounded the myriad consequences depressive disorders may have on individuals (Maramis et al, 2021; Badcock & et al., 2017; Frohn & Martiny, 2023). Depression should be regarded as a multicomplex biological, cognitive, social and affective disorder (Demeyer et al., 2012). The consequences extend well beyond mere physical and mood-related symptoms. "In particular, cognitive impairments have been identified as a concomitant manifestation of depressive phenomenology and should not be considered merely secondary to it" (Maramis et al., 2021, p.232). Individuals, who feel threatened by changes in the external environment or internal physiological state, may be faced with the question: What decisions and strategy may I select to protect my future physical, mental, and social well-being? In order to answer such questions a predictive processing perspective may shed light on how our brain makes predictions about the world, when it only has subjective limited fragments of it. It helps us understand the mechanism by which human beings adoptively have learned to rely on prior experiences in order to prepare for future challenges. And once this mechanism has been understood, it may subsequently reveal some insights into states when such a learning from a stable environment is impaired, such as in depression.

For the purpose of establishing a nexus between depressive states and predictive processing, first and foremost the basic tenets of the predictive processing theory and its advantages to understanding the human brain in a "normal" healthy state must be understood.

When predictive processing goes right

Under the predictive processing perspective, the brain is seen as an actively coding machine, which is constantly striving to guide organisms towards environmental challenges in the metabolically most adaptive, effective and efficient way (Friston, 2012). According to the free energy principle, this involves managing states of uncertainty to achieve an optimal balance of adaptability and stability. Ideally, this entails pursuing states that are neither overly complex - rendering them unpredictable - nor so simple that they offer no opportunity for new learning (Kiverstein, 2017).

From this perspective, achieving an optimal balance between predictability and volatility allows individuals to leverage their previously acquired knowledge, whereby specific actions are associated with certain outcomes. In predictive processing lingo, this initial belief which is based on past experiences and shapes our perception and decisions, is referred to as a prior. The reliance on past experiences enhances the ability to forecast future outcomes and adapt behaviour accordingly. At the same time, it demands human beings to stay alert to environmental changes to revise their priors by considering new evidence. In doing so agents use their prior knowledge, expectations and predictions about the world and update it based feedback from sensory surroundings, ideally resulting in a well-informed, evidence-based and reliable internal representation about the world – a so called model. ‘‘Models are considered adaptive when they allow one to quickly and efficiently infer the sources of stimulation as well as to predict future states and consequences of action (planning and action)’’ (Van den Bergh et al., 2021, p.229).

The idea of taking the two probability distributions of priors and sensory data into account while forming our posterior beliefs is rooted in Bayes´ theorem - a concept in probability theory that characterizes optimal decisions using both prior knowledge and current sensory information according to their respective uncertainty probability (Vilares & Kording, 2017).

Prediction errors

The concept of “prediction errors” can be traced back to the early computational model of vision by Rao and Ballard in the late 1990s illustrating perception based on a hierarchical neural network which constantly generates perception by the integration of top-down expectation and sensory data (Rao & Ballard, 1997). Rather than seeing the brain as a passive organ perceiving sensory inputs, the computational model of Rao and Ballard (1997) proposes that pyramidal

cells in the higher levels of the cortex actively predict the incoming input they are about to receive. Consequently, cortical feedback connections convey such predictions to lower-level prediction error neurons, whereas cortical feedforward prediction errors neurons create prediction errors, by constantly comparing predictions with sensory inputs. This bidirectional process aims towards refining predictions iteratively, by minimizing prediction errors and, by extension, reducing uncertainty in the perceptual and cognitive system (Koster-Hale & Saxe, 2013).

This leaves one question unanswered: under which circumstances do either top-down predictions or bottom- up signals exert a greater influence on human decision-making?

The role of precision

To answer this question, the term of precision is a key part of the Bayesian Theorem, in so far that it argues that accurately integrating the two-distribution possibility of priors and sensory data is the key to effective decision-making. Bayesian Theorem argues that the one stronger in uncertainty has a greater impact on participants´ decisions. In other words, giving more weight to prior knowledge when current data is less reliable, while precise sensory evidence optimally leads to an overreliance on the latter (Vilares et al. 2012).

Several empirical papers have shown in experiments whilst manipulating either sensory or prior certainty, the one stronger in predictive validity has a greater impact on participants´ decisions (Vilares and Kording, 2017; Chambon et al., 2011; Trimmer et al. 2011). The background assumption here is, the higher the precision of prediction errors, the greater their influence on belief updating. Interestingly, during stress (uncertainty) neuromodulation ideally amplifies the precision of sensory evidence, giving sensory inputs greater weight on decision-making processes. Such selective increases in precision are key for updating beliefs about uncertainty in the world when predictions fail to explain sensory evidence (Kube & Rozenkrantz, 2021).

In summary, it can be said that the Bayesian brain gradually learns through past experiences to infer deeper latent causes, affects, motivations and meanings which shape the environment. Based on this internal representation of the environment, sensory inputs are perceived through creating mismatches between these two streams. Either a change of action or belief-updating can then followingly resolve such discrepancies to regain states of certainty and order (Paulus et al., 2019).

But what happens in the case of depressive states, when such prediction-error minimization actions are constrained by lingering in states of hopelessness, fatigue, incarceration, rumination and behavioural avoidance. Can such phenomenologically different symptoms of depressive states be explained trough a maladaptive predictive processing frame?

What happens when dealing with uncertainty fails?

In a rapidly changing world, uncertainty and volatility are inescapable. Nevertheless, dealing with uncertainty can only be learned from a stable environment experienced over time. Human beings need to have a profound sense of assurance that their certain contingencies will produce expected consequences (Miller & Seligman, 1975).

What are the consequences of disrupted learning experiences in the absence of a controllable, stable environment?

The concept by Seligman about learned helplessness may elucidate such ramifications. In his experiments he showed that a dog that has faced unmanageable shocks before avoidance training soon stops running, howling and lies passively. Whereas dogs that have experienced desired escape of harmful situations, the helpless dog simply fails to learn that barrier-jumping produces shock termination (Seligman & Maier, 1967). Analogously, in depression, people who for example have experienced immense traumatic experiences in their biographical history may have learned to perceive their environment as capricious, pessimistic and uncontrollable. Consequently, they may have learned due to an interplay of biological, psychological, and social factors to adopt a passive state of hopeless lingering, such as the dog that has experienced unmanageable escape (Heim et al., 2008). This may manifest itself as a shrinking of their field of relevant actions leading to energy-conservation behavioural tendencies, such as isolation, avoidance, hypervigilance, and an all-enveloping passivity. “The depressed person literally experiences a world without hope in which no projects call out as worth pursuing” (Ratcliffe, 2015).

The locked- in brain of depression

The ideas of dysfunctional, yet short-term adaptive cognitive beliefs trace back to Beck´s cognitive model of “The Negative Triad” from the year 1967, in which he argued, that depressive people do exhibit specific negative cognitive schemas that consequently bias their emotional, behavioural, and physiological reactions to environmental stressors. He has noted, for instance, that individuals with depressive tendencies often exhibit a propensity to make

long-term, global, and internal attributions for negative outcomes, while positive events are typically perceived as resulting from exceptional circumstances such as luck (Beck, 1967). The idea formed by Beck (1967) of such rigid negative priors in depression aligns well with the predictive processing framework that reclaims a hierarchical in the brain that biases how new information is processed, making negative outcomes more probable in depression-prone perception. Empirical recent papers, aligned with this theory, illustrated indeed strong negative hyper precise priors in depression that dominant participants' view of the world. These papers have claimed that depression is associated with an attentional bias for disorder-relevant stimuli (such as self-referential negative statements), others reported increased amygdala activity response to negative faces among populations at high-risk for depression)Gotlib et al., 2004, Bradley & Mathews, 1988; Hindash et al., 2012, Joormann et al., 2007). All these experimental studies rest on the fundamental assumption that depression is associated with a precise expectation of a negatively valanced environment, which facilitates the resistance to contradictory positive information.

Chronic levels of stress und uncertainty induced by such negative emotional states, however, create allostatic overload - persistent activated stress reactions that may lead to neuroendocrine, cardiovascular, neuroenergetic, and emotional turbulences (Peters et al. 2017). Consequently, from a predictive processing view, this will lead to active interference – the goal is always to reduce surprise and strive towards certainty. Consequently, it appears that individuals with depression become trapped in a vicious cycle characterized by negatively biased cognitive styles and behavioural strategies, such as self-referent thinking, isolation, avoidance behaviour, fatigue, psychomotor retardation and lethargy designed to mitigate short-term uncertainty. This, in turn, contributes to the further dysfunctional rigidification of their hyper-precise predictive priors.

The problem of an impaired belief updating process

It seems that the problem with depressive states from a predictive processing perspective is twofold. First, precise negative priors dominate the lives of those affected by framing their perception of the environment through a lens of negativity. Secondly the process of escaping the negative feedback cycle of psychopathology is further hindered by an impaired updating of such priors towards new positive evidence. Such positive evidence may only be encountered by explorative actions. Exploratory behaviour would demand screening the environment for new evidence to be able to revise old dysfunctional beliefs in the light of opposing evidence.

Abnormalities in the belief updating process of depressed people have been confirmed in prior studies showing their persistence with negative interpretation of situations despite novel positive information. A resistance to positive feedback, such as positive thoughts, emotions, and behaviours has been evidenced, the maintenance of negative interpretations of ambiguous information (Kube et al., 2020; Steele Ramos-Grille). For example, Kube et al. (2020) examined how learning processes differ between healthy individuals and those with depression, particularly when it comes to updating predictions based in the form of unexpected information about desired (positive) vs. unwanted (negative) feedback, whereas for healthy people only a few positive examples were needed to revise their negative expectations, depressed people maintained their negative beliefs longer. This suggests they show impaired belief updating towards positive information. However, when confronted with negative feedback they tended to update their expectations faster and to a greater extent towards such negative prediction errors, than healthy individuals. This has been reflected in a higher activation of the lateral habenula, which is associated with the processing of negative prediction errors (Kube et al., 2020). In contrast, the attenuation of positive stimuli in depression has been linked to reduced activation in brain regions, including the dopaminergic midbrain and medial temporal lobes. The dopaminergic system, which plays a critical role in reward processing and memory formation, is often hypoactive in depressed individuals, suggesting impaired reward processing (Dillon, 2014).

Behavioural studies have illustrated such tendencies with a dampening of positive information in depression via cognitive immunization strategies – the psychological process that corresponds to the attenuation of a better-than-expected prediction error processing, making individuals less responsive to disconfirmatory evidence (Beshkofeh et al., 2020; Kube et al., 2019).

Another example would be counterfactual reinforcement, where people tend to retrospectively interpret positive safe events that contradict their prior learned assumptions, in light of "near-miss disaster", interpreting safe events as rare exceptions, which next time need better "safety" behaviour – in their perspective as heightened attention and avoidance - to mitigate seemingly catastrophic outcomes (Moutoussis et al., 2018).

In sum, it can be stated that the problem within depressive states can be traced back to a vicious cycle– a hyper precise precision of negative-valanced priors coupled with a stagnated updating of these beliefs.

Depression and social cognition

Why is it important to establish the link between depressive states and social cognition?

First and foremost, social support and belonging are key protective factors against depressive disorders (Allen & Badcock, 2003). The social risk hypothesis, however, claims that depression may lead to a risk-averse approach in the context of social situations, concretely displayed by a general decreased tendency to engage in social behaviour that involves uncertainty, such as competitive or conflict-driven settings. Neural correlates of the social risk hypothesis can be found in a hyperactivity of the hypothalamic-pituitary-adrenal axis. The consequence of such would be increased cortisol levels which are linked to heightened sensitivity towards external threats that may render affected individuals especially sensitive to social risk situations, such as in situations of conflict, assessment or unpredictable environments (Allen & Badcock, 2003). In addition to this, there seems to be a tendency of depressive people to exhibit negative self-other relations, reduced empathic abilities resulting from heightened self-focus, and a specific attentional bias towards socially threating stimuli (Schreiter et al., 2013). Consequently, self-efficacy beliefs are undermined by feelings of inadequacies, unworthiness, higher sensitivity towards social rejection and self-damnation (Zemore & Bretell, 1983). In terms of the predictive processing perspective, such perceived shortcomings would restrictively influence individuals' selection and construction of social environments.

The link between depressive states and risk-sensitive behavioural strategies is not a theoretical novelty. "Evidence of a relationship between depression and risk preferences lends support to neuroeconomic and behavioural economic models in which people's decision-making ability and opportunity sets are constrained by their cognitive, emotional, and physiological functioning" (Cobb-Clark et al., 2022, p.1568). The study of Smoski et al. (2008) investigated decision-making processes in depression and found a general tendency of risk avoidance in the

Iowa Gambling tasks, where depressive participants chose fewer risky cards across the entire task. The authors concluded that healthy subjects are expecting success while depressed patients behave as if they are expecting failure. Viewed through a predictive processing lens, risk-averse adaptive priors may reduce short-term uncertainty by promoting self-defeating behaviour such as reassurance seeking, conservative decision-making, and submissiveness (Leahy et al., 2012).

What has not been addressed in prior research – relevant research gaps

Even though a wide swathe of empirical studies tried to link negative cognitive processing styles to depression states, there are still some limitations of such studies. Whereas some studies have found a negativity bias in depression (Kube et al., 2020; Kiverstein et al., 2020; Hindash and Amir, 2012) other have explained such phenomenon in terms of depressive-realism view, where depressed seem to see the world in a more realistic view (Carson et al. 2010; Moore & Fresco, 2007). Findings may be mixed due to a heterogenous nature in selected study tasks, analysed brain regions, sample sizes, but even more crucially a huge variability in confounding aspects of the disorder. Factors such as symptom severity, chronicity, comorbidity, types of depressive disorders and medication status are thought to influence differently cognition, attention and learning (Lam et al., 2014). Therefore, a strength of the current study may be the inclusion of a subclinical sample and medication exclusion for the purpose of gaining larger within-in group homogeneity. In addition to this the investigation of potential cognitive impairments in people that have not yet been diagnosed with depression, may serve as valuable insights into early markers of the disease and therefore enrich our understanding of effective prevention strategies.

In terms of theory of mind and depression the results of empirical studies are rather inconclusive. Mostly, theory of mind tasks have been employed in empirical studies involving patients with other psychiatric disorders, such as autism spectrum disorder, schizophrenia, bipolar disorder, and antisocial personality disorder, whereas depression is still an under researched topic in those terms. Under the assumption of negative self-focused attention, along with cognitive slowing, social avoidance behaviour and hypersensitivity to negative stimuli, it may seem plausible however, that depression also influences reasoning abilities in social situations (Şencan, 2019; Schreiter et al., 2013).

Methods

Participants & recruitment procedure

50 students (27 male, 23 females, mean [M] age = 26,9 years, standard deviation [SD] = 9,9) have been recruited via The Laboratory Administration System for Behavioral Science (Labs) of the University of Vienna. 25 participants of that sample have been used for data analysis in this thesis. All individuals who met the eligibility criteria (see below) participated at the Faculty of Psychology of the University of Vienna in the EEG lab 2, provided written informed consent, and received monetary compensation (25 EUR) for their participation. The study was approved by the University of Vienna Ethics Committee and performed in accordance with the Declaration of Helsinki (World Medical Association, 2013).

Before official study invitations were issued, potential participants underwent an initial eligibility screening using an online questionnaire. The specified inclusion criteria were: age ≥ 18 - 45; fluent German-speakers (native or at least C1 level); not be studying or have studied psychology; no current or history of major neurological or psychiatric disorders; no history of substance abuse; not currently taking prescription medication (except birth control pill); non-smokers or smoke less than 5 cigarettes per day (self-report) and normal or corrected-to-normal eye vision. During the recruitment process, three registered participants were excluded from the study due to medication intake, having studied psychology in the past, and past episodes of psychiatric disorders.

The Inventory of Depressive Symptomatology (German IDS-SR)

To assess depressive tendencies at the sub-clinical level the German version of the Inventory of Depressive Symptomatology ‑ self report (IDS-SR) was used (Rush et al. 2000). As a self-report questionnaire of depression, it has good psychometric properties and has been used in clinical, but also in non-clinical populations. The design is based on key depression criteria of the Diagnostic and Statistical Manual of Mental disorders, Fourth Edition (DSM-IV) and encompasses essential domains of functioning, including sleep, mood, appetite, concentration, energy levels, arousal, and sexual interest−each crucial for capturing the broad spectrum of depressive symptoms (Rush et al. 2000).These areas of functioning are consistent with theoretical frameworks of depression ‑ such as the Biopsychosocial Model (Sarkar et al. 2023) or the Cognitive-Behavioural Model of depression (Driessen et Hollon, 2010) - thereby supporting the construct validity of the questionnaire.

The questionnaire shows strong correlations with other well-established depression inventories, such as the Hamilton Depression Rating Scale (HDRS) (Hamilton, 1960) and the Beck

Depression Inventory (BDI) (Beck et al., 1961) pointing towards high criterion validity (Rush et al., 1996). The depression questionnaire was sent out via e-mail to participants after having received an official study invitation, with a request for completion before study participation. A reminder to complete the questionnaire was sent 24 h prior to the appointment of the study.

Experimental procedure – theory of mind task

In this study, participants performed a computerised social decision-making task in which both prior- and sensory uncertainty were manipulated independently across the experiment. Standardized conditions in the form of a short oral explanation about the experiment, and completion of the written consent form were maintained consistently for all participants. Stimuli were presented using PsychoPy software (Psychology Software Tools, Inc., Pittsburgh, PA, USA) running on a Microsoft Desktop PC.

The experiment started with a comprehensive visual instruction about the upcoming task. Participants were asked to predict players' betting behaviour of four different protagonists with different winning probabilities. For doing that, participants were required to learn the players´ risk proneness. To accomplish this, participants received prior information - in the form of written short sentences which provided the information they needed to infer the protagonist's decisions. Consequently, participants were asked to predict each protagonist´s behaviour across four blocks (1 block per player) and 50 trials per block. Concretely, they had to predict how much money each character would bet on a scale between 1-100 Euros in a card game where winning probabilities varied between trials. To accurately predict protagonists' behaviour, participants could use the prior given information about the players - obtained from the verbal descriptions about them and the current sensory information in the form of the immediate visual feedback they received about the underlying playing behaviour of the characters.

Uncertainty was manipulated across two levels −low prior uncertainty and high prior certainty. Self-ratings such as "*I would describe myself as risk-averse*" represented low prior uncertainty, whereas phrases such as "*my friends would say that I never take risks, but I don't think that is true*" were created to produce high uncertainty surrounding the participants' belief about the risk-proneness of the protagonists. After each choice, visual feedback about the 'true' underlying behaviour of the protagonist was represented as a green bar on the

rating scale, displayed in addition to their given rating, thus representing the prediction error. In addition to prior uncertainty, sensory uncertainty was manipulated, ranging from low to high levels of uncertainty. Whereas a broader bar represented a greater range around the protagonist ´s actual behaviour, and a narrower bar indicated higher precision regarding the protagonist ’s monetary bets. There were no time limits for making the predictions, so participants could decide freely how much time they needed to make a decision.

Hypotheses & experimental design

The study followed a two-by-two factorial design (see Fig. 1).

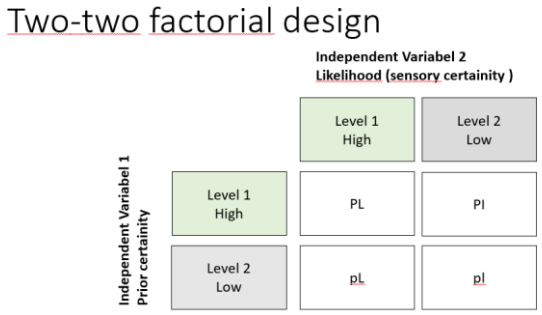


Figure 1 | Experimental design: There are two types of independent variables, each with two levels: The first variable ‑ prior certainty with a high (P) and low (p) prior certainty and the second variable ‑ high (L) and low (l) sensory certainty, resulting in a total of four conditions. The terms precision and certainty are in this context used interchangeably.

To determine whether participants exhibited differential reactions to changes in these two variables and adjusted their behaviour accordingly, the first hypothesis is stated as follows:

H1: A reduced precision in the sensory information coupled with high precision in the prior will lead all participants to update responses less towards the prediction error/sensory evidence. In contrast, decreased prior precision coupled with high sensory evidence will result in enhanced updating in response to the prediction error.

Furthermore - as sketched above in the introduction - prior studies (Kube et al., 2020; Kiverstein et al., 2020; Hindash and Amir, 2012) have shown a higher belief-updating towards negative information of depression-prone individuals. We explored whether there is a difference between individuals without depressive tendencies vs. participants scoring higher on the depression questionnaire.

Hypotheses two and three therefore specify how depressive states affect decision-making under uncertainty:

H2: Participants with depressive tendencies will exhibit increased precision of towards risk-averse information. Such hypersensitivity to risk-averse priors won´t be expected in individuals without depressive tendencies.

In the context of the experiment risk-averse prior refers to statements about the protagonists that would prioritize outcomes that are more predictable and less prone to large variations or risky stakes. Studies have shown that people with depression are more likely to avoid risks, making them more risk-averse especially in uncertain environments. This can be attributed to negative cognitive biases, where depressed individuals may focus on potential losses or negative outcomes, leading to conservative decision-making (Gutierrez et al. 2013, Smoski et al. 2018). Interestingly, in our experiment is the aspect, that participants didn’ t have to make risky choices for themselves but rather infer risk-avoidant behaviour in others. The extent to which depressive people do as well project their own level of risk aversion onto other situations and people remains an underexplored area of research and would thus offer a novel dimension to our study.

H3: Individuals with high depression scores will show increased belief updating towards negative social prediction errors. Such an increased belief-updating towards negative prediction errors wouldn´t be expected in the low depression tendencies group.

A negative prediction error refers to the discrepancy between expected and actual outcomes, where an individual expects a positive or neutral outcome, but actually experiences a negative one. In the context of depression, negative prediction error plays a critical role in how individuals experience and interpret their environment. Concretely, in this experiment, a negative prediction error refers to feedback indicating that the protagonist wagered less money than the amount predicted by the study participants. Prior studies have shown that depression-prone individuals often exhibit heightened feedback sensitivity coupled with amplified learning towards negative prediction errors (Steele et al. 2007; Ramos-Grille et al., 2022; Barrett et al., 2016; Clark et al., 2018; Kube et al., 2020).

Statistical analyses

All data were analysed using non-parametric tests conducted in R studio. Non-parametric tests have been chosen due to the constraints imposed by the small sample size. In this case, the Shapiro-Wilk test for residuals yielded a p-value of 0.04946, suggesting a slight deviation from normality. In addition to this, the Q-Q plot (see fig. 2) shows minor deviations from normality (in the tails). Given that non-parametric tests do not rely on the assumption of normality, they are particularly suitable for small samples (Field, 2018).

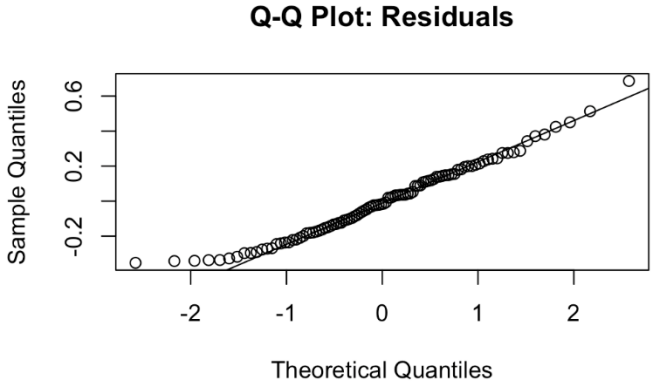


Figure 2 | Q-Q Plot: Residuals: Small departures from the assumption that the residuals (the differences between the observed values and the values predicted by the model) follow a normal distribution.

To answer hypothesis 1, individual beta values were obtained, by calculating a regression for each participant within each cell of the design (beta_Pl = high prior, low sensory precision; beta_PL = high prior, high sensory precision; beta_pL = low prior, high sensory precision, beta_pl = low prior, low sensory precision) of the signed prediction error at trial t-1 predicting the signed update from t-1 to t. A Friedman test was conducted to evaluate the differences in beta coefficients, which represent how much an individual's updating is influenced by the prediction error (PE) across different task conditions (beta_Pl, beta_PL, beta_pL, beta_pl). Under the assumption of the first hypothesis, we may expect to see a difference in beta values between high and low sensory certainty conditions and secondly, higher sensory weights in the beta_pL condition and the beta_PL condition . concretely when the current information (feedback) was more reliable and also when the prior information was more uncertain.

The Friedman test is a non-parametric statistical test used to compare three or more related samples. It assesses whether there are statistically significant differences in the distributions of a continuous variable across multiple conditions or time points while accounting for the

correlation among the samples (i.e., repeated measures), given that the sample was repeatedly exposed to the four conditions throughout trials. (Field, 2018).

For Hypothesis two, the objective was to examine whether response behaviour differed between the group with higher depressive tendencies and with the low-depressed group, concretely we expected higher updating towards risk-averse information in the high depression-prone group. To compare the groups, a median split was done to dichotomizing the depression scores variable in order to divide the dataset in two-equal sized groups (see table 1). Accordingly, values that are equal to or below the median form the first group (low/non-depressive tendencies) whereas values above the median comprises the second group (high depressive tendencies).

DEPRESSION_BINARY		COUNT	PERCENTAGE
1	0	12	48
2	1	13	52

Table 1 | Distribution of the depression variable after median split: The sample is almost evenly split between the two groups based on the median. The first group (low/ non-depressive tendencies) consists of 12 individuals (48%), and the second group (high depressive tendencies) comprises the remaining 13 participants (52%).

A Mann-Whitney U test was conducted to compare the Beta values associated with risk-averse prior information (beta_p_riskAve). The Mann-Whitney U test, also known as the Wilcoxon rank-sum test, is a non-parametric test used to compare the distributions of two independent groups. It assesses whether there is a statistically significant difference in the ranks of the two groups, making it useful when the assumptions of normality and homogeneity of variances are violated (Field, 2018).

For obtaining the variable beta_p_riskAVe a regression was calculated using the signed prediction error in t−1 and the update from t−1t to t, divided into risk-averse (the player's self-report indicated risk aversion ‑ Player A and B) and risk-prone (the player's self-report indicated risk-seeking behavior ‑ Player C and D). The results are individual beta values that reflect the extent to which a person, across all trials, was influenced to a greater extent by the risk-averse self-report, in contrast to the current feedback they got. With other words, the closer this beta value is to 1, the stronger the influence of the risk-averse information on participants´ ratings. Explicitly, risk-averse priors were established via initial self-ratings about the protagonists´ tendency to avoid risks and make more conservative decisions. Meaning that participants had been here more influenced by risk-averse priors.

Finally, for hypothesis three, which states that depressive people may place an increased weight on negative sensory information, the Mann-Whitney U test was applied to see if the group with the higher depressive tendencies adjusted more their updating towards negative prediction errors.

Results

Descriptive results

The gender distribution among the 25 participants consisting of 12 males and 13 females indicates a balanced representation of gender within the sample.

The IDS-SR results are depicted in Table 2, Figure 3 and Figure 4. Each item is scored on a 4-point scale, ranging from 0 to 3. The total score is calculated by adding up the scores for all items, with possible total scores ranging from 0 to 84. Higher scores indicate more severe depressive symptoms.

The score ranges and their interpretations for the IDS-SR are grounded on the initial development and utilization of the questionnaire by Rush et al., based on following cut- off values:

- 0‑13: No depression (minimal symptoms)
- 14‑25: Mild depression
- 26‑38: Moderate depression
- 39‑48: Severe depression
- 49 and above: Very severe depression

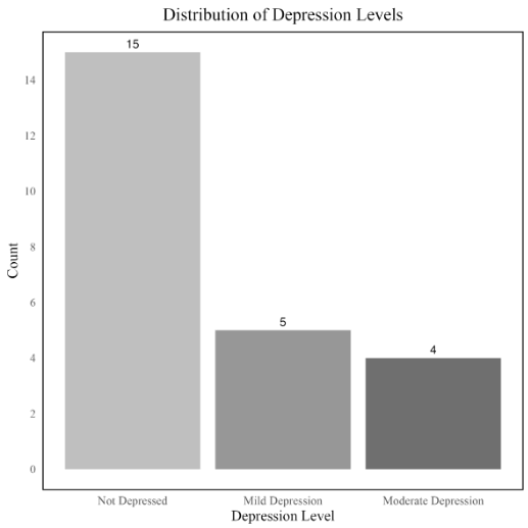
Figure 3 illustrates the distribution of the used data sample for this thesis. From a total of 25 participants, 60 % of the sample have achieved a score below 14 points on the depression questionnaire and are therefore not considered to show any depressive symptoms. However, 24% of the participants achieved a cut off score between 14 and 25 points which is classified as mild depressed according to Rush et al. (1986). Only 16% of the study sample showed signs of a moderate depression score. None of the individuals displayed severe or extremely severe depressive symptomatology (see fig. 3).

Figure 4 illustrates this distribution of depression scores across the study sample. The x-axis represents the sum of depression scores, while the y-axis shows the count of individuals with each score range. Descriptively, it can be concluded that most individuals have lower sum depression scores, with a high concentration in the 5 to 10 range. There is a noticeable drop in frequency as the scores increase, suggesting fewer individuals with higher depression scores. A small number of individuals have scores between 30 and 35, indicating that high depression scores are relatively rare in this sample (see fig. 4).

Overall, the data appears to be right-skewed, meaning most individuals have lower depression scores, and only a few subjects exhibiting higher scores.

Variable	N	Mean	Std. Dev.	Min	Max
sum_depression	25	15	9.9	3	38
depression_level	25				
... Not Depressed	15	60%			
... Mild Depression	6	24%			
... Moderate Depression	4	16%			
... Severe Depression	0	0%			
... Extremely Severe Depression	0	0%			
Age	25	27	6.1	18	41
Gender	25				
... Male	12	48%			
... Female	13	52%			

Table 2 | Descriptive statistics of data: Sum_depression refers to the total score obtained by summing responses to depression-related items, while the depression_level variable categorizes this score into predefined severity categories, e.g., mild, moderate, or severe, based on established cut-off points by Rush et al. 1996.



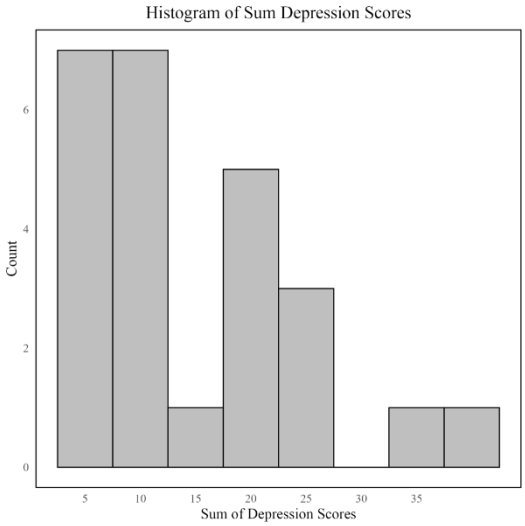


Figure 3 | Distribution of Depression Levels.

Figure 4 | Histogram of Sum Depression Scores.

In terms of Hypothesis 1 a Friedman test was conducted to evaluate the differences in Beta values across the four different conditions. On a descriptive level the box plots already point towards similar distributions of the four conditions (see fig. 5) . The non-parametric Friedman rank sum test yielded no statistically significant differences in beta values among these conditions (χ^2_3=2.95, p=0.3991).

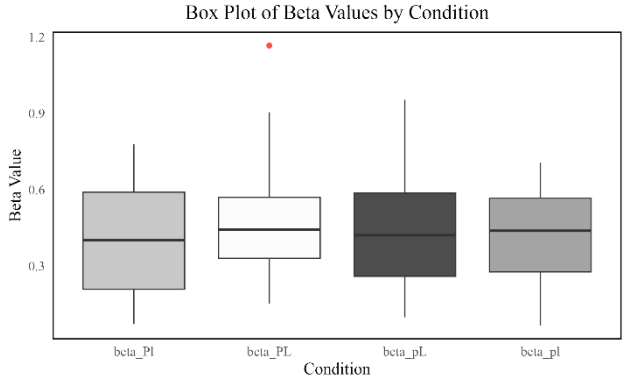


Figure 5 | Beta Values across the four experimental conditions: The distributions of the beta values in the four different conditions of the study: beta_Pl= high prior, low sensory precision; beta_PL= high prior, high sensory precision; beta_pL= low prior, high sensory precision, beta_pl= low prior, low sensory precision.

In light of Hypothesis 2, the difference was tested between the group with higher depression scores vs. individuals with lower or non-depressive tendencies in their updating towards risk-averse priors. In line with the non-parametric no statistically significant differences between these groups can be concluded.

The Wilcoxon rank sum exact test was conducted to compare the Beta values associated with risk-averse prior information (Beta_p_riskAve) between participants with high vs. low(non) depressive tendencies. The results showed no statistically significant differences in Beta values between individuals with higher and those with non-depressive tendencies (W=75, p= 0.8938).

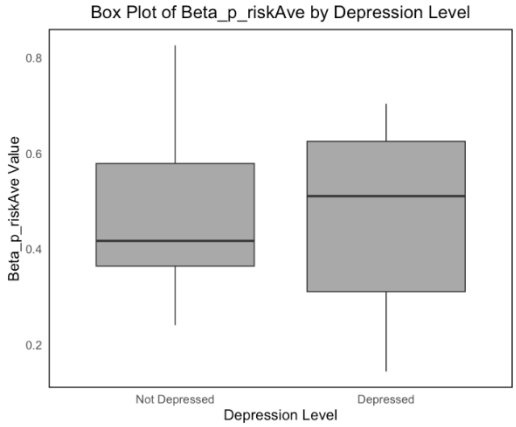


Figure 6 | Box Plot of Beta_p_risk_Ave by Depression Level: The two box plots visualize the distribution of Beta_p_riskAve representing the beta values associated with risk-averse prior information) across depression-prone and non-depression prone individuals. The greater interquartile ranges of the depressed group point towards more diversity in the responses or measurements within that range. There are not any visible outliners that may require a further look.

Finally, Hypothesis 3 posits that individuals with a higher predisposition to depression are more likely to exhibit heightened updating in response to negative prediction errors. In this context, on any given trial, a negative prediction error refers to feedback indicating that the protagonist wagered less money than the amount predicted by the study participants.

For this purpose, the beta values of updates following negative prediction errors from the regression between PE(t-1) and the update from t-1 have been used, namely as variable Beta_P_neg . In light of Hypothesis 3, one may expect that individuals with depression exhibit a greater update in the direction of the negative prediction error. Non-parametric statistical tests did not support this assumption. The non-parametric test yielded again a non-significant result (W= 64, p= 0.4696).

Figure 7 shows the two contrasting groups in their beta values towards negative prediction errors on a descriptive level. The box plot provides a descriptive overview of how each group responds to negative prediction errors, illustrating the range, median, and variability in their updating patterns. While group with higher depressive scores has on a descriptive level a slightly higher median, this difference is statistically not significant.

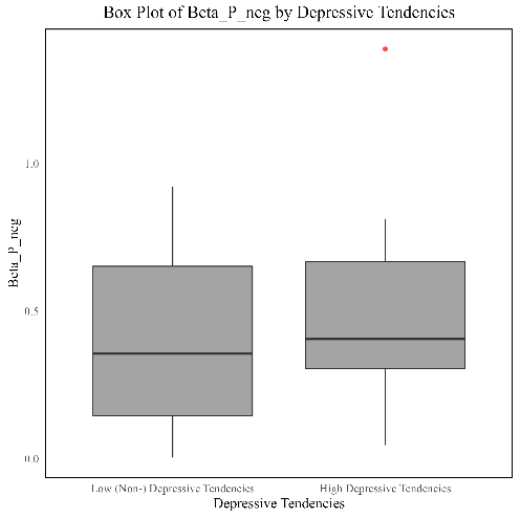


Figure 7 |Box Plot of Beta_p_neg by Depression level: The median Beta_P_neg for the higher-depressive group seems to be a little higher than for the low/non-depressed group it. This suggests a minor difference in the central tendency between the groups. Nevertheless, the statistical tests, however, failed to validate this descriptive tendency.

Discussion of results

The overall aim of the study was to analyse how individuals take decisions under conditions of uncertainty, with particular emphasis on potential disparities related to depression and their impact on such processes.

Hypothesis one was formulated to investigate how potential estimates of prior and sensory uncertainty influenced belief updating as a matter of precision. Prior empirical studies have shown that human beings take two types of information into account when making decisions depending on the degree of certainty to these probability distributions – the prior probability of encountering some inferred causes and the likelihood or sensory evidence that the given sensory data would arise from the inferred causes (Vilares & Kording, 2017; Friston et al., 2006; Friston, 2012).

In line with predictive processing theories when the sensory certainty is high (in our experiment the pL- low prior and high sensory precision and the PL – high prior and high sensory precision conditions), one would expect a tendency of individuals to gain higher beta values in these conditions. Explicitly, this would mean integrating the current sensory feedback more into belief-updating when such feedback is more precise and prior information less clear.

For example, the study of Kording & Wolpert (2004) has shown this Bayesian inference process in the context of a sensorimotor task. Concretely, they have shown that individuals take both – prior knowledge and current sensory evidence into account during learning sensorimotor sequences. The researchers conducted experiments where participants reached for visual targets with their right index fingers, introducing controlled variations in the task and manipulating the uncertainty of sensory feedback. In contrast to our study, Kording & Wolpert (2004) have focused only on manipulating one variable – the sensory evidence. In our study, however, both variables – prior and sensory uncertainty has been altered. This may provide a more realistic perspective of myriad real-world scenarios, in which multiple factors at the same time simultaneously influence our decision-making processes.

The study of Vilares & Kording (2017) has investigated how dopamine medication would influence decisions in people with Parkinson's disease. Their outcomes revealed a higher reliance on current sensory information on dopamine medication potentially to the detriment of making more thoughtful, long-term decisions by taking as well prior knowledge into account.

Similarly to our study uncertainty in both prior and current sensory information was manipulated. Without a healthy control group however, there is lack of baseline for comparison that makes it difficult to determine if behaviours are directly caused by Parkinson's disease or if they arise from other contributing factors, such as medication, cognitive decline or comorbid diseases. In comparison, our study tried to address such questions using population without depressive tendencies compared to a subclinical group.

To review the predictive processing hypothesis, which suggests that both prior knowledge and sensory evidence are integrated based on the level of certainty, we statistically tested the differences in beta weights across the four conditions. No significant differences were found between the conditions. Thus, our results did not confirm prior empirical studies (Vilares & Kording, 2017: Hsu et al. 2005) responding to varying degrees of uncertainty according to rules of Bayes´ theorem. Even on a descriptive level, the boxplots showed similar betas across conditions, independent of prior or sensory precision (see fig. 6).

Linking the initial empirical results about predictive processing and depression (Steele et al. 2007; Ramos-Grille et al., 2022; Barrett et al., 2016; Clark et al., 2018; Kube et al., 2020, Gutierrez et al. 2013, Smoski et al. 2018) with the current empirical results obtained from this study the following can be concluded. Prior empirical studies such as the one from Smoski et al. (2018) proposed a tendency towards risk-averse information in depression-prone people. In their study depressive vs. healthy populations have been contrasted against each other during a gambling task, in which participants had to choose between low-risk, low-reward responses over high-risk, high-reward responses. The authors concluded that depressed participants learned faster in avoiding risky card decks compared to the healthy counterparts, implying a heightened sensitivity to aversive contingencies. To replicate such findings, our study compared people with lower vs. higher depressive scores in their tendency towards taking risk-averse information. Here, no statistical evidence was found that depressive tendencies are in general associated with a higher tendency towards risk-aversive updating. In comparison to the aforementioned paper, we must take a crucial difference into account. In the study by Smoski et al. (2018) participants had to take risky choices for themselves whereas in our study test subjects needed to infer the risk-proneness of learned characters. The other perspective in our study meant, that trial subjects had to observe and predict behaviour of the individuals featured in the game in order to opt for them correctly. However, self-relevant decision-making might evoke stronger emotional reactions and therefore influence belief-updating more drastically. Inferential judgment (like in our study) on the other hand, may engage different cognitive

processes related to empathy and social perception in general, instead of inherent risk-taking tendencies.

As for hypothesis three, the attempt to replicate previous findings on the heightened sensitivity to negative prediction errors in depression-prone individuals was unsuccessful. A wide swath of preceding empirical studies have linked depression to heightened sensitivity to negative feedback alongside increased memory and learning performances from such prediction errors (Steele et al. 2007; Ramos-Grille et al., 2022; Barrett et al., 2016; Clark et al., 2018; Kube et al., 2020). Differing from our study, negative feedback was in most of the studies operationalized as negative life-experiences (such as rejection, punishment, critical or unfavourable feedback in a work context). In our study the operationalization of negative feedback would imply that characters in the game gave less money than the initial amount predicted by the study participant. That being said, in our study feedback was delivered in the form of an abstract monetary reward system. The null findings in our study may as well be explained by lower emotional reactions possibly due to potential less personal and intense emotional reactions elicited by the game-based feedback.

However, the current results prompt several important conclusions that must be taken into account critically.

First, it is important to critically consider that, in the present study, analyses have focused on participants' average behaviour. However, analysing how participants respond to trial-by-trial variations in sensory and prior manipulations would provide a more nuanced understanding of behaviour. For example, individual behavioural responses may change over the course of an experiment reflecting learning effects, adaptation, or changes in mood or cognitive states. By averaging across trials, we may miss out such variability patterns.

Due to the scope and limited resources of the current project, a small sample size of 25 subjects has been used to address the complex symptomatology of depressive tendencies and cognitive styles. The small sample size may have hindered the ability to detected significant differences. Moreover, due to convenience sampling, a sampling bias may have led to the recruitment of a homogeneous study sample with relatively low depressive tendencies. Even though the sample included 40% of participants which could be characterised as showing mild to moderate depressive symptoms on a pre-clinical level, differences to non-depressed individuals may not have been pronounced enough to result in effects. One may expect that any differences are likely to be attenuated relative to those with clinically diagnosed depression or higher subclinical depressive scores.

Another noteworthy consideration is the validity of the chosen questionnaire administered to the subjects. Self-report measures are prone to potential biases, such as anchoring effects and social desirability, which could skew respondents' experiences and affect the accuracy of the data collected. In addition to this, retrospective reports of past episodes may be prone to selective recall bias. Perhaps utilizing a more refined assessment of depressive states accompanied with a more heterogeneous study sample would have yielded different results.

The claimed association between depression and behavioural risk-aversion may as well depend significantly on the contextual setting. For example, Cobb-Clark (2022) et al. has shown that depressive people are less willing to take risks with respect to leisure and trust but are more risk-prone in domains such as driving, financial decisions and occupational matters. Thus, the link between depression and risk-aversion may be moderated by a specific setting and therefore demands domain-specific risk-preference measures. "While our fixed-effects estimates are generally less precise, the overarching conclusion remains−whether depression is associated with a greater or lesser willingness to take risks depends very much on the domain in which decisions are being made" (Cobb-Clark et al., 2022). In respect to this, in our theory of mind experiment subjects had to take the decisions not for themselves but estimate the risk proneness of other people. It is reasonable to assume that their risk-taking behaviour might differ from what it would have been if they were making decisions for themselves vs. for others.

To wrap up this section, it can be said that considering the complexity and versatility of depressive disorder, the quest towards identifying an underlying pathophysiologic process may be challenging and enlightening at the same time. It is worthwhile noting, that given the heterogeneity of depressive symptoms it may be unlikely to identify one single underlying mechanism as the culprit. For a refined understanding of any underlying mechanisms however, the complex intertwining cascade of molecular and cellular interactions requires more investigation. This could be achieved by applying stronger methodological procedures (such as computational models) and as stated previously, a more extensive and representative sample given the following potential benefits of a predictive processing framework in advancing our understanding of depression.

Predictive Processing in Depression: Insights and Benefits

The primary purpose of this study is to assess how individuals draw valid inferences about hidden latent processes of the environment when faced with uncertainty? Of particular interest specific in this project, is how the psychopathology of the complex, multifactorial psychopathology of depression may benefit from a predictive processing perspective?

First and foremost, the theory of predictive processing perceives the brain as an active organ that dynamically constructs a model of the world through the noisy inputs it perceives by making use of prior knowledge. The assumption of the brain as an active organ implies a sense of agency inherent to human beings (Friston, 2012). Seeing depression from this perspective may help us seeing the disorder not only as an unfortunate accumulation of inevitable biographical circumstances, a lack of caring social support systems or a reductionist pathophysiological disfunction, but rather consider it as a transitory, changeable state that can be changed through actively widening again one's perceptions, goals, and actions in the world as captured by Sporns, who wrote:

> *"the architecture of the brain…and the statistics of the environment, [are] not fixed. Rather, brain-connectivity is subject to a broad spectrum of input-, experience-, and activity-dependent processes which shape and structure it is patterning and strengths… These changes, in turn result in altered interactions with the environment, exerting causal influences on what is experienced and sensed in the future" (Sporns, 2007, p.179).*

Secondly, the predictive processing theory combines biological aspects of psychopathology with cognitive aspects explaining how the brain works (Gilbert et al. 2022). This urges towards a perspective of seeing depression as a complex interplay between various influential variables and thus calls for integration of both aspects in treatment of depressive disorders. Important neurotransmitters, implicated in learning - as claimed by the predictive processing perspective - are also connected to depressive disorders, such as serotonin, noradrenalin, glucocorticoids, and dopamine (Nestler & Carlezon, 2006). An exhaustive overview of all these interactions would go far beyond the scope of this paper, but understanding their importance for cognitive processes would analogically explain their disruptive effects on cognition in depression.

At the very least, the hierarchical structure of the generative model, evocated by predictive processing theories, implies that higher-level systems constantly influence lower-level systems.

This structure highlights the bidirectional influence of higher cognitive systems (beliefs, prior knowledge, expectations, implicit learned knowledge) and the sensorium of the environment. This means that what we perceive with our senses is greatly influenced and shaped by higher-level cognition. In the case of depression, a skewed perception towards negativity may produce a rigid vicious cycle in which distorted cognitive biases, subsequent perception and in turn behaviour mutually influence and reinforce each other across time, contributing to the chronic, recurrent, and persistent episodes of depression (Van de Cruys & Van Dessel, 2021).

Clinical implications

As argued within this paper, depression may be characterized by distorted perspectives about the causalities of the environment (Kube et al., 2020). Symptoms of depressive states, such as anhedonia, a loss of appetite, vigour, and motivation, alongside prolonged states of fatigue, lethargy and psychomotor retardation may stem from a prolonged state of dealing with uncertainty. How could therapy intervene in such aspects following predictive processing rules?

As previously discussed in this paper, the predictive processing model is based on two fundamental principles for dealing with uncertainty: altering one's perception or adjusting one's actions (Friston, 2012). Changing predictions would imply changing deeply ingrained hypotheses about the hidden causes of the flux of the sensorium. As elucidated through this whole paper, depressive people do show increased attentional processing of mood-congruent information. Therefore, it is up to therapeutical sessions to confront patients with inconsistent evidence directed toward their own logic, as Van de Cruys and Van Dessel (2021) have stated it concisely “the regularities they have absorbed but that no longer hold (p.109)”. However, when confronted with contrary evidence, depressive individuals, tend to hold up to their beliefs by engaging in immunization techniques or excessive rumination, in which they try to fit their mental model rigidly and blindly to the world, without searching for more efficient models based on new evidence. An example of relearning such rigid beliefs is the use of a scaffolding environment. Here, well-established schemata and self-confirmatory coping mechanisms - such as dysfunctional immunization techniques - should be brought to awareness within the psychotherapeutic process (Moutoussis et al., 2018).

The prominence of "I" experiences - in the form of excessive self-referential repetitive thoughts, negative distorted self-perceptions and social avoidance behaviour should likewise be tackled in therapy. A profound rupture in intersubjectivity may arise from viewing ones experiences as hyper-salient and in contrast to others. Feelings of being misunderstood, prolonged states of rumination and an all-encompassing excessive occupation with one´s concern may lead to further resistance from closed ones. "The experience of this dark and uninviting environment fosters detachment and disengagement" (Fabry, 2019). In response to the experience of social disconnection, therapy should encourage an openness to be affected and entangled by and with others.

The second option to deal with uncertainty would be action. The paper of Kiverstein et al. (2017) argues that positive feelings of mastery, control and hedonic values may arise even in situations of large prediction errors, however with the difference of the rate in uncertainty reduction accomplished by the agent. If circumstances were faced with a great instantaneous error that the individual overcomes in a faster rate as expected, the experience can be interpreted as positive. In achieving that, pleasurable hedonic feelings arise due to an intrinsic felt process of being able to move forward faster than expected. Under such circumstances, subjective mastery and feelings of action readiness are elicited in the "skilled body" (Kiverstein et al., 2017).

Recapitulating this - setbacks and frustrations should attune our attention to explore new paths, reframing prediction errors not as failures, but as opportunities for learning and adaptation. The pleasurable feelings of mastering and adventure arising out of new experiences emerging in the form of better-than-expected feedback should further motivate towards positive engagement. Following this theory, one may understand why in the case of depression this striving forward is disrupted. To be able to look out for new opportunities, individuals must maintain an optimal realistic sensitivity to their current state, both bodily and in terms of sensory changes in the environment. A lack of such sensitivity will lead to suboptimal decision-making thus hinder action readiness. Therefore, sensitivity towards changes in uncertainty is key to stay open, attuned and actively engaged in the rapidly dense changing sensory barrage of life. "Again, we see how possibilities that are integral to the experienced world are inextricable from the capacity for action—a return of the significance of things is also a return of the sense that one can act upon them." (Fulford et al., 2013. p.589)

Sources:

Ahmed, A. T., Frye, M. A., Rush, A. J., Biernacka, J. M., Craighead, W. E., McDonald, W. M., ... & Mood Disorders Precision Medicine Consortium. (2018). Mapping depression rating scale phenotypes onto research domain criteria (RDoC) to inform biological research in mood disorders. *Journal of affective disorders*, 238, 1-7. 10.1016/j.jad.2018.05.005

Allen, N. B., & Badcock, P. B. (2003). The social risk hypothesis of depressed mood: evolutionary, psychosocial, and neurobiological perspectives. *f*, 129(6), 887. 10.1037/0033-2909.129.6.887

Badcock, P. B., Davey, C. G., Whittle, S., Allen, N. B., & Friston, K. J. (2017). The depressed brain: An evolutionary systems theory. *Trends in Cognitive Sciences*, *21*(3), 182-194. 10.1016/j.tics.2017.01.005

Bar, M. (2009). A cognitive neuroscience hypothesis of mood and depression. *Trends in cognitive sciences*, 13(11), 456-463. https://doi.org/10.1016/j.tics.2009.08.009

Barrett, L. F., Quigley, K. S., & Hamilton, P. (2016). An active inference theory of allostasis and interoception in depression. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 371(1708), 20160011. https://doi.org/10.1098/rstb.2016.0011

Beck, A. (1967). Depression: Clinical, Experimental, and Theoretical Aspects. New York, NY: Harper and Row.

Beck, A.T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561 571. https://doi.org/10.1001/archpsyc.1961.01710120031004

Beshkofeh, M., Karami, T., Ahameh, F., Kalvandy, F., & Dasht Bozorgi, Z. (2020). The effectiveness of mental immunization on depression and anxiety in children with diabetes type1. *Quarterly Journal of Child Mental Health*, 7(1), 118-127. http://dx.doi.org/10.29252/jcmh.7.1.11

Bayer, Ya'akov M., Zeev Shtudiner, Oxsana Suhorukov, and Nimrod Grisaru. 2019. "Time and Risk Preferences, and Consumption Decisions of Patients with Clinical Depression."

Journal of Behavioral and Experimental Economics 78:138–45. https://doi.org/10.3368/jhr.58.1.0419-10183R1

Bradley, B. P., & Mathews, A. (1988). Memory bias in recovered clinical depressives. *Cognition and Emotion*, 2, 235−245. https://doi.org/10.1037/bul0000367

Carson, R. C., Hollon, S. D., & Shelton, R. C. (2010). Depressive realism and clinical depression. Behaviour research and therapy, 48(4), 257-265. https://doi.org/10.1016/j.brat.2009.11.011

Chambon, V., Domenech, P., Pacherie, E., Koechlin, E., Baraduc, P., & Farrer, C. (2011). What are they up to? The role of sensory evidence and prior knowledge in action understanding. *PloS one*, 6(2), e17133. https://doi.org/10.1371/journal.pone.0017133

Clark, A. (2013). Whatever next? Predictive brains, situated agents, and the future of cognitive science. *Behavioral and brain sciences*, *36*(3), 181-204. https://doi.org/10.1017/S0140525X12000477

Clark, A. (2020). Beyond desire? Agency, choice, and the predictive mind. *Australasian Journal of Philosophy*, *98*(1), 1-15. https://doi.org/10.1080/00048402.2019.1602661

Chung, Dongil, Kelly Kadlec, Jason A. Aimone, Katherine McCurry, Brooks King-Casas, and Pearl H. Chiu. 2017. "Valuation in Major Depression is Intact and Stable in a Non-Learning Environment." *Scientific Reports* 7:Article 44374. https://doi.org/10.1038/srep44374

Cobb-Clark, D. A., Dahmann, S. C., & Kettlewell, N. (2022). Depression, risk preferences, and risk-taking behavior. *Journal of Human Resources*, *57*(5), 1566-1604. Https://doi.org/10.3368/jhr.58.1.0419-10183R1

Demeyer, I., De Lissnyder, E., Koster, E. H., & De Raedt, R. (2012). Rumination mediates the relationship between impaired cognitive control for emotional information and depressive symptoms: A prospective study in remitted depressed adults. *Behaviour research and therapy*, *50*(5), 292-297. https://doi.org/10.1016/j.brat.2012.02.012

Dillon, D.G., et al. (2014). "Disrupted reward processing in depression: Evidence from neuroimaging and behavior." *Trends in Cognitive Sciences*, 18(3), 136-144. https://doi.org/10.1016/j.copsyc.2014.12.011

Dinhof, C., Humer, E., Haider, K., Rabenstein, R., Jesser, A., Pieh, C., ... & Gächter, A. (2024). Comprehensive examination of support needs and mental well-being: a mixed-method study of the Austrian general population in times of crisis. *Frontiers in Public Health*, *12*, 1345796. https://doi.org/10.3389/fpubh.2024.1345796

Driessen, E., & Hollon, S. D. (2010). Cognitive behavioral therapy for mood disorders: efficacy, moderators and mediators. *Psychiatric Clinics*, 33(3), 537-555. https://doi.org/10.1016/j.psc.2010.04.005

Eshel, N., & Roiser, J. P. (2010). Reward and punishment processing in depression. *Biological psychiatry*, *68*(2), 118-124. https://doi.org/10.1016/j.biopsych.2010.01.027

Fabry, R. E. (2020). Into the dark room: a predictive processing account of major depressive disorder. *Phenomenology and the Cognitive Sciences*, *19*(4), 685-704. https://doi.org/10.1007/s11097-019-09635-4

Fancourt, D., Steptoe, A., & Bu, F. (2020). Trajectories of depression and anxiety during enforced isolation due to COVID-19: longitudinal analyses of 59,318 adults in the UK with and without diagnosed mental illness. 10.1016/S2215-0366(20)30482-X

Field, A. (2018). *Discovering statistics using IBM SPSS Statistics* (5th ed.). SAGE Publications.

Foland-Ross, L. C., & Gotlib, I. H. (2012). Cognitive and neural aspects of information processing in major depressive disorder: an integrative perspective. *Frontiers in psychology*, *3*, 489. 10.3389/fpsyg.2012.00489

Frank, E., Karp, J. F., & Rush, A. J. (1996). Efficacy of treatments for major depression. *Psychopharmacology Bulletin*, *29*(4), 457-475.

Friston, K. (2012). Prediction, perception and agency. *International Journal of Psychophysiology*, 83(2), 248-252. https://doi.org/10.1016/j.ijpsycho.2011.11.014

Friston, K. J., Thornton, C., & Clark, A. (2012b). Free-energy minimization and the dark-room problem. *Frontiers in Psychology*,. https://doi.org/10.3389/fpsyg.2012.00130

Friston, K., Kilner, J., & Harrison, L. (2006). A free energy principle for the brain. *Journal of physiology*-Paris, 100(1-3). 10.1016/j.jphysparis.2006.10.001

Frohn, O. O., & Martiny, K. M. (2023). The phenomenological model of depression: from methodological challenges to clinical advancements. *Frontiers in Psychology*, *14*. https://doi.org/10.3389/fpsyg.2023.1215388

Fulford, K. W. M., Davies, M., Gipps, R., Graham, G., Sadler, J., Stanghellini, G., & Thornton, T. (Eds.). (2013). *The Oxford handbook of philosophy and psychiatry*. OUP Oxford.

Gibson, J. J. (2014). *The ecological approach to visual perception*: Classic edition Original work published 1979. Psychology Press. https://doi.org/10.4324/9781315740218 https://doi.org/10.4324/9781315740218

Gilbert, J. R., Wusinich, C., & Zarate Jr, C. A. (2022). A predictive coding framework for understanding major depression. *Frontiers in human neuroscience*, 16, 787495. https://doi.org/10.3389/fnhum.2022.787495

Gotlib, I. H., Kasch, K. L., Traill, S., Joormann, J., Arnow, B., & Johnson, S. L. (2004). Coherence and specificity of information-processing biases in depression and social phobia. *Journal of Abnormal Psychology*, 113, 386–398. 10.1037/0021-843X.113.3.386

Gutierrez, A. D., Sadek, S. L., & Bos, P. J. (2013). Depression and decision making: A meta-analytic review. *Psychological Bulletin*, 139(5), 789‑821. 10.1037/0033-2909.113.3.472

Hamilton, M. (1960). "A rating scale for depression." *Journal of Neurology, Neurosurgery, and Psychiatry*, 23(1), 56‑62. 10.1136/jnnp.23.1.56

Heim, C., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2008). The link between childhood trauma and depression: insights from HPA axis studies in humans. *Psychoneuroendocrinology*, 33(6), 693-71. https://doi.org/10.1016/j.psyneuen.2008.03.008

Hertel, P. T., & Rude, S. S. (1991). Depressive deficits in memory: focusing attention improves subsequent recall. *Journal of Experimental Psychology: General*, *120*(3), 301. https://doi.org/10.1037/0096-3445.120.3.301

Hindash, A. H. C., & Amir, N. (2012). Negative interpretation bias in individuals with depressive symptoms. Cognitive therapy and research, 36, 502-511. 10.1007/s10608-011-9397-4

Hinton, G. E. (1980). Inferring the meaning of direct perception. *Behavioral and Brain Sciences*, *3*(3), 387-388. https://doi.org/10.1017/S0140525X00005549

Hsu, M., Bhatt, M., Adolphs, R., Tranel, D. & Camerer, C. F. (2005). Neural systems responding to degrees of uncertainty in human decision-making. *Science* 310, 1680‑1683.f. 10.1126/science.1115327

Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional Information in girls at risk for depression. *Journal of Abnormal Psychology*, 116, 135. 10.1037/0021-843X.116.1.135

Karasu, F., Öztürk Çopur, E., & Ayar, D. (2021). The impact of COVID-19 on healthcare workers' anxiety levels. *Journal of Public Health*, 1-11. 10.1007/s10389-020-01466-x

Karp, D. (1996). *Speaking of sadness: Depression, disconnection, and the meanings of illness*. Oxford University Press

Kiverstein, J., Miller, M., & Rietveld, E. (2017). The feeling of grip: novelty, error dynamics, and the predictive brain. *Synthese*, *196*, 2847-2869. https://link.springer.com/article/10.1007/s11229-017-1583-9

Kiverstein, J., Miller, M., & Rietveld, E. (2020). How mood tunes prediction: a neurophenomenological account of mood and its disturbance in major depression. *Neuroscience of Consciousness*, *2020*(1), niaa003. https://doi.org/10.1093/nc/niaa003

Koster-Hale, J., & Saxe, R. (2013). Theory of mind: a neural prediction problem. *Neuron*, *79*(5), 836-848. https://doi.org/10.1016/j.neuron.2013.08.020

Krueger, J. (2023). Affordances and spatial agency in psychopathology. *Philosophical Psychology*, 1-30. https://doi.org/10.1080/09515089.2023.2243975

Kube, T., Glombiewski, J. A., Gall, J., Touissant, L., Gärtner, T., & Rief, W. (2019). How to modify persisting negative expectations in major depression? An experimental study comparing three strategies to inhibit cognitive immunization against novel positive experiences. Journal of Affective Disorders, 250, 231-240. 10.1016/j.jad.2019.03.027

Kube, T., & Rozenkrantz, L. (2021). When beliefs face reality: An integrative review of belief updating in mental health and illness. *Perspectives on Psychological Science*, 16(2), 247-274. 10.1177/1745691620931496

Kube, T., Schwarting, R., Rozenkrantz, L., Glombiewski, J. A., & Rief, W. (2020). Distorted cognitive processes in major depression: A predictive processing perspective. *Biological psychiatry*, *87*(5), 388-398. 10.1016/j.biopsych.2019.07.017

Kording, K. P. & Wolpert, D. M. Bayesian integration in sensorimotor learning. Nature 427, 244‑247 (2004).

Lam, R. W., Kennedy, S. H., McIntyre, R. S., & Khullar, A. (2014). Cognitive dysfunction in major depressive disorder: effects on psychosocial functioning and implications for treatment. *The Canadian Journal of Psychiatry*, 59(12), 649-654. 10.1177/070674371405901206

Leahy, R. L., Tirch, D. D., & Melwani, P. S. (2012). Processes underlying depression: Risk aversion, emotional schemas, and psychological flexibility. *International Journal of Cognitive Therapy*, 5(4), 362-379. 10.1521/ijct.2012.5.4.362

Li, Lu, Andreas Richter, and Petra Steinorth. 2021. "Mental Health Changes and the Willingness to Take Risks." Geneva Risk Insurance Review. https://doi.org/10.1057/s10713-021-00070-7

Maiese, M. (2021). Enactivism, the Field of Affordances, and Mental Disorder. *Journal of Mind & Behavior*, *42*(2). 10.1007/s11229-019-02133-9

Majumdar, P., Biswas, A., & Sahu, S. (2020). COVID-19 pandemic and lockdown: cause of sleep disruption, depression, somatic pain, and increased screen exposure of office workers and students of India. *Chronobiology international*, *37*(8), 1191-1200. 10.1080/07420528.2020.1786107

Maramis, M. M., Mahajudin, M. S., & Khotib, J. (2021). Impaired cognitive flexibility and working memory precedes depression: a rat model to study depression. *Neuropsychobiology*, *80*(3), 225-233. 10.1159/000508682

Miller, M., & Clark, A. (2018). Happily entangled: prediction, emotion, and the embodied mind. *Synthese*, *195*(6), 2559-2575. https://doi.org/10.1007/s11229-017-1399-7

Miller, W. R., & Seligman, M. E. (1975). Depression and learned helplessness in man. *Journal of abnormal psychology*, 84(3), 228. 10.1037/h0076720

Must, A., Horvath, S., Nemeth, V. L., & Janka, Z. (2013). The Iowa Gambling Task in depression−What have we learned about sub-optimal decision-making strategies? *Frontiers in Psychology*, *4*, Article 732. https://doi.org/10.3389/fpsyg.2013.00732

Moore, M. T., & Fresco, D. M. (2007). Depressive realism and attributional style: Implications for individuals at risk for depression. *Behavior Therapy*, 38(2), 144-154. https://doi.org/10.1016/j.beth.2006.06.003

Moutoussis, M., Shahar, N., Hauser, T. U., & Dolan, R. J. (2018). Computation in psychotherapy, or how computational psychiatry can aid learning-based psychological therapies. *Computational Psychiatry* (Cambridge, Mass.), 2, 50.10.1162/CPSY_a_00014

Mühlböck, M., Juen, I., Brunner, S., Till, M., Moser, W., Wittmann, L., & Brüngger, L. (2023). So geht’s uns heute: die sozialen Krisenfolgen im dritten Quartal 2022‐Schwerpunkt Wohlbefinden und Gesundheit. Ergebnisse einer Statistik-Austria-Befragung.Steele, J. D., Kumar, P., & Ebmeier, K. P. (2007). Blunted response to feedback information in depressive illness. *Brain*, 130(9), 2367-2374.

Nestler, E. J., & Carlezon Jr, W. A. (2006). The mesolimbic dopamine reward circuit in depression. *Biological psychiatry*, 59(12), 1151-1159. 10.1016/j.biopsych.2005.09.018

Paulus, M. P., Feinstein, J. S., & Khalsa, S. S. (2019). An active inference approach to interoceptive psychopathology. *Annual review of clinical psychology*, *15*(1), 97-122. 10.1146/annurev-clinpsy-050718-095617

Peters, A., McEwen, B. S., & Friston, K. (2017). Uncertainty and stress: Why it causes diseases and how it is mastered by the brain. *Progress in neurobiology*, *156*, 164-188. https://doi.org/10.1016/j.pneurobio.2017.05.004

Ramos-Grille, I., Weyant, J., Wormwood, J. B., Robles, M., Vallès, V., Camprodon, J. A., & Chanes, L. (2022). Predictive processing in depression: Increased prediction error following negative valence contexts and influence of recent mood-congruent yet irrelevant experiences. Journal of Affective Disorders, 311, 8-16. 10.1016/j.jad.2022.05.030

Ratcliffe, M. (2013). Depression and the phenomenology of free will. https://doi.org/10.1093/oxfordhb/9780199579563.013.0036

Ratcliffe, M. (2015). *Experiences of depression: A study in phenomenology*. International Perspectives in.

Rao, R. P., & Ballard, D. H. (1997). Dynamic model of visual recognition predicts neural response properties in the visual cortex. *Neural computation*, 9(4), 721-763. 10.1162/neco.1997.9.4.721

Rush, A. J., Gullion, C. M., Basco, M. R., Jarrett, R. B., & Trivedi, M. H. (1996). The inventory of depressive symptomatology (IDS): psychometric properties. *Psychological medicine*, 26(3), 477-486. 10.1017/s0033291700035558

Sarkar, B., Ali, A., & Paul, F. A. (2023). A case study of depression: A holistic exploration through the lens of biopsychosocial dynamics. *Odisha Journal of Psychiatry*, 19(2), 59-64. :10.4103/OJP.OJP_3_24

Schreiter, S., Pijnenborg, G. H. M., & Aan Het Rot, M. (2013). Empathy in adults with clinical or subclinical depressive symptoms. *Journal of affective disorders*, 150(1), 1-16. 10.1016/j.jad.2013.03.009

Schwartenbeck, P., Passecker, J., Hauser, T. U., FitzGerald, T. H., Kronbichler, M., & Friston, K. J. (2019). Computational mechanisms of curiosity and goal-directed exploration. *elife*, *8*, e41703. https://doi.org/10.7554/eLife.41703

Şencan, B. (2019). Theory of mind in major depressive disorder. *Psikiyatride Güncel Yaklaşımlar*, 11(1), 42-54. https://doi: 10.18863/pgy.383349

Seligman, M. E., & Maier, S. F. (1967). Failure to escape traumatic shock. *Journal of experimental psychology*, 74(1). https://doi.org/10.1037/h0024514

Smoski, M. J., Lynch, T. R., Rosenthal, M. Z., Cheavens, J. S., Chapman, A. L., & Krishnan, R. R. (2008). Decision-making and risk aversion among depressive adults. *Journal of behavior therapy and experimental psychiatry*, 39(4), 567-576. https://doi.org/10.1016/j.jbtep.2008.01.004

Sporns, O. (2007). What neuro-robotic models can teach us about neural and cognitive development. Neuroconstructivism: *Perspectives and prospects*, 2, 179-204. https://doi.org/10.1093/acprof:oso/9780198529934.003.0008

Sterner, E. Y., & Kalynchuk, L. E. (2010). Behavioral and neurobiological consequences of prolonged glucocorticoid exposure in rats: relevance to depression. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *34*(5), 777-790. Sterner, E. Y., & Kalynchuk, L. E. (2010). https://doi.org/10.1016/j.pnpbp.2010.03.005

Trimmer, P. C., Houston, A. I., Marshall, J. A., Mendl, M. T., Paul, E. S., & McNamara, J. M. (2011). Decision-making under uncertainty: biases and Bayesians. *Animal cognition*, *14*, 465-476.

Van de Cruys, S., & Van Dessel, P. (2021). Mental distress through the prism of predictive processing theory. *Current Opinion in Psychology*, 41, 107-112. https://psycnet.apa.org/doi/10.1016/j.copsyc.2021.07.006

Van den Bergh, O., Brosschot, J., Critchley, H., Thayer, J. F., & Ottaviani, C. (2021). Better safe than sorry: A common signature of general vulnerability for psychopathology. *Perspectives on Psychological Science*, *16*(2), 225-246. https://doi.org/10.1177/1745691620950690

Van den Bergh, O., Witthöft, M., Petersen, S., & Brown, R. J. (2017). Symptoms and the body: Taking the inferential leap. Neuroscience & Biobehavioral Reviews, 74, 185‑203. https://doi.org/10.1016/j.neubiorev.2017.01.015

Vilares, I., Howard, J. D., Fernandes, H. L., Gottfried, J. A. & Kording, K. P. Differential representations of prior and likelihood uncertainty in the human brain. Curr. Biol. 22, 1641‑1648 (2012).

Vilares, I., & Kording, K. P. (2017). Dopaminergic medication increases reliance on current information in Parkinson's disease. *Nature human behaviour*, *1*(8), 0129. https://doi.org/10.1038/s41562-017-0129

Warren, M. B., Pringle, A., & Harmer, C. J. (2015). A neurocognitive model for understanding treatment action in depression. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *370*(1677), 20140213. https://doi.org/10.1098/rstb.2014.0213

Zahavi, A. Y., Sabbagh, M. A., Washburn, D., Mazurka, R., Bagby, R. M., Strauss, J., ... & Harkness, K. L. (2016). Serotonin and dopamine gene variation and theory of mind decoding accuracy in major depression: a preliminary investigation. *PLoS One*, *11*(3), e0150872. https://doi.org/10.1371/journal.pone.0150872

Zemore, R., & Bretell, D. (1983). Depression-proneness, low self-esteem, unhappy outlook, and narcissistic vulnerability. *Psychological Reports*, 52(1), 223-230. https://doi.org/10.2466/pr0.1983.52.1.223

Appendix:

Appendix A: Material for recruitment of participants

Participant Information and Consent Form for Study Participation (German Version)

Die Beurteilung von sozialen Situationen

Sehr geehrte*r Teilnehmer*in,

wir laden Sie ein, an der oben genannten Studie teilzunehmen. Wenn Sie dies lesen, haben Sie bereits die Einschlusskriterien für eine Teilnahme erfüllt. Es folgen nun detaillierte Informationen zur Teilnahme.

Ihre Teilnahme an dieser Studie erfolgt freiwillig. Sie können jederzeit, ohne Angabe von Gründen, Ihre Bereitschaft zur Teilnahme ablehnen oder auch im Verlauf der Studie zurückziehen. Die Ablehnung der Teilnahme oder ein vorzeitiges Ausscheiden aus dieser Studie hat keine nachteiligen Folgen für Sie.

Diese Art von Studien ist notwendig, um verlässliche neue *wissenschaftliche* Forschungsergebnisse zu gewinnen. Unverzichtbare Voraussetzung für die Durchführung von Studien ist jedoch, dass Sie Ihr Einverständnis zur Teilnahme an dieser Studie schriftlich erklären. Bitte lesen Sie den folgenden Text sorgfältig durch und zögern Sie nicht, Fragen zu stellen.

Bitte unterschreiben Sie die Einwilligungserklärung nur

wenn Sie Art und Ablauf der Studie vollständig verstanden haben,
wenn Sie bereit sind, der Teilnahme zuzustimmen und
wenn Sie sich über Ihre Rechte als Teilnehmer*in an dieser Studie im Klaren sind.

Was ist der Zweck der Studie?

Wenn Menschen das Verhalten anderer beurteilen oder vorhersagen, ziehen sie eine Vielzahl an Informationen (z.B. Körpersprache, Wissen über Charaktereigenschaften, etc.) in Betracht und wägen diese gegeneinander ab. Dies passiert oft automatisch und abseits unserer bewussten Kontrolle. Bisher ist allerdings unklar, wie genau dieses unbewusste Abwägen von Informationen von statten geht. Die gegenwärtige Studie untersucht, auf welche Art und Weise Individuen die ihnen zur Verfügung stehenden Informationen verarbeiten, um in sozialen Situationen möglichst präzise Urteile zu fällen.

Anhand einer Reihe von Computeraufgaben werden Teilnehmer*innen verschiedene sozial relevante Informationen präsentiert, bevor sie verschiedene soziale Urteile fällen sollen (z.B. „welche Emotion wurde im vorherigen Video am ehesten präsentiert?“).

Wie läuft die Studie ab?

Die Studie wird von einer Forschungsgruppe der Universität Wien an der Fakultät für Psychologie durchgeführt. Insgesamt werden 50 Personen an dieser Studie teilnehmen.

Ablauf des Hauptexperiments:
Das Hauptexperiment findet an der Fakultät für Psychologie der Universität Wien statt. Im Rahmen dessen werden Sie zwei Aufgaben an einem Computer durchführen, die jeweils aus einer Lern- und einer Testphase bestehen. In beiden Aufgaben werden Ihnen soziale Reize gezeigt (z.B. der sich verändernde Gesichtsausdruck einer Person), die Sie anschließend interpretieren sollen. Daraufhin folgt noch eine weitere computerbasierte Aufgabe, in denen Ihre Merkfähigkeit überprüft wird. Zuletzt werden wir eine kurze (ca. 3-minütige) Videoaufnahme von Ihrem Gesicht erstellen. Dies dient zur non-invasiven Erhebung eines verhaltensbasierten Markers Ihres Dopaminspiegels.
Das Hauptexperiment wird insgesamt nicht länger als zwei Stunden dauern.

Fragebögen:
Anschließend werden Sie gebeten, mehrere Fragebögen zu Ihrem Sozialverhalten auszufüllen. Die Dauer wird hier maximal 15 Minuten betragen.

Im Anschluss an das Experiment werden Sie ausführlich und vollständig über Sinn und Zweck des Experimentes aufgeklärt.

Die Einhaltung der Anweisungen der Studienleiterin und ihrer Mitarbeiter*innen ist von entscheidender Bedeutung für den Erfolg dieser Studie.

Worin liegt der Nutzen einer Teilnahme an der Studie?

Die Teilnahme an dieser Studie hat für Sie persönlich keinen unmittelbaren Nutzen. Die Ergebnisse dieser Studie sollen zur Klärung unseres Verständnisses von sozialen Beurteilungsprozessen beitragen.

Gibt es Risiken bei der Durchführung der Studie und ist mit Beschwerden oder anderen Begleiterscheinungen zu rechnen?

Bei den Testungen im Rahmen dieser Studie sind keinerlei Beschwerden oder negative Begleiterscheinungen zu erwarten. Die Belastung des Hauptexperiments ist mit alltäglicher Arbeit am Computer vergleichbar.

Hat die Teilnahme an der Studie sonstige Auswirkungen auf die Lebensführung und welche Verpflichtungen ergeben sich daraus?

Die Teilnahme an dieser Studie hat keinerlei Auswirkungen auf Ihre Lebensführung.

Was ist zu tun beim Auftreten von Beschwerdesymptomen, unerwünschten Begleiterscheinungen und/oder Verletzungen?

Es sind im Rahmen dieser Studie keine negativen Begleiterscheinungen zu erwarten.

Sollten im Verlauf der Studie dennoch irgendwelche beschwerlichen Symptome, Begleiterscheinungen, Krankheiten oder Verletzungen auftreten, müssen Sie diese der

Studienleiterin und/oder ihren Mitarbeiter*innen mitteilen, bei schwerwiegenden Begleiterscheinungen umgehend.

Wann wird die Studie vorzeitig beendet?

Sie können jederzeit, auch ohne Angabe von Gründen, Ihre Teilnahmebereitschaft widerrufen und aus der Studie ausscheiden, ohne dass Ihnen dadurch irgendwelche Nachteile entstehen.

Zudem ist es möglich, dass Ihre Teilnahme an der Studie von der Studienleitung abgebrochen wird, ohne vorher Ihr Einverständnis einzuholen. Mögliche Gründe hierfür können sein:

Sie können den Erfordernissen der Studie nicht entsprechen
Die Studienleiterin hat den Eindruck, dass eine weitere Teilnahme an der Studie nicht in Ihrem Interesse ist

In welcher Weise werden die im Rahmen dieser Studie gesammelten Daten verwendet?

Im Rahmen dieser Studie werden Daten über Sie erhoben und verarbeitet. Es ist grundsätzlich zu unterscheiden zwischen
jenen personenbezogenen Daten, anhand derer eine Person direkt identifizierbar ist (z.B. Name, Geburtsdatum, Adresse, Sozialversicherungsnummer, Bildaufnahmen...),
pseudonymisierten personenbezogenen Daten, das sind Daten, bei denen alle Informationen, die direkte Rückschlüsse auf die konkrete Person zulassen, entweder entfernt, durch einen Code (z. B. eine Zahl) ersetzt oder (z.B. im Fall von Bildaufnahmen) unkenntlich gemacht werden. Es kann jedoch trotz Einhaltung dieser Maßnahmen nicht vollkommen ausgeschlossen werden, dass es unzulässigerweise zu einer Re-Identifizierung kommt.
anonymisierten Daten, bei denen eine Rückführung auf die konkrete Person ausgeschlossen werden kann.

Zugang zu den Daten, anhand derer Sie direkt identifizierbar sind (siehe Punkt 1), haben lediglich die Studienleiterin und andere an der Studie mitwirkende Mitarbeiter*innen der Universität Wien. Sämtliche Personen, die Zugang zu diesen Daten erhalten, unterliegen im Umgang mit den Daten den jeweils geltenden nationalen Datenschutzbestimmungen und/oder der EU-Datenschutz-Grundverordnung (DSGVO).
Der Code, der eine Zuordnung der pseudonymisierten Daten zu Ihrer Person ermöglicht, wird nur an der Universität Wien aufbewahrt. Eine Weitergabe der Daten erfolgt ausschließlich in pseudonymisierter oder anonymisierter Form. Für etwaige Veröffentlichungen werden nur die pseudonymisierten oder anonymisierten Daten verwendet. Im Rahmen dieser Studie ist keine Weitergabe von Daten in Länder außerhalb der EU (Drittland) vorgesehen.
Ihre Einwilligung bildet die Rechtsgrundlage für die Verarbeitung Ihrer personenbezogenen Daten. Sie können die Einwilligung zur Erhebung und Verarbeitung Ihrer Daten jederzeit ohne Begründung widerrufen. Nach Ihrem Widerruf werden keine weiteren Daten mehr über Sie erhoben. Die bis zum Widerruf erhobenen Daten können allerdings weiter im Rahmen dieser Studie verarbeitet werden.
Nach der DSGVO stehen Ihnen grundsätzlich die Rechte auf Auskunft, Berichtigung, Löschung, Einschränkung der Verarbeitung, Datenübertragbarkeit und Widerspruch zu, soweit dies die Ziele der Studie nicht unmöglich macht oder ernsthaft beeinträchtigt und soweit dem nicht andere gesetzliche Vorschriften widersprechen.
Die voraussichtliche Dauer der Studie ist ein Jahr (bis September 2024). Die Dauer der Speicherung Ihrer Daten über das Ende oder den Abbruch der Studie hinaus ist durch Rechtsvorschriften geregelt.
Falls Sie Fragen zum Umgang mit Ihren Daten in dieser Studie haben, wenden Sie sich zunächst an die Studienleiterin. Diese kann Ihr Anliegen ggf. an die Personen, die für den Datenschutz

verantwortlich sind, weiterleiten.
Kontaktdaten der Datenschutzbeauftragten der an dieser Studie beteiligten Institutionen:

Datenschutzbeauftragte/r der Universität Wien: dsba@univie.ac.at

Sie haben das Recht, bei der österreichischen Datenschutzbehörde eine Beschwerde über den Umgang mit Ihren Daten einzubringen (www.dsb.gv.at; E-Mail: dsb@dsb.gv.at).

Entstehen für die Teilnehmer*innen Kosten? Gibt es einen Kostenersatz oder eine Vergütung?

Durch Ihre Teilnahme an dieser Studie entstehen für Sie keine Kosten. Sie erhalten nach erfolgter Teilnahme an der Hauptuntersuchung entweder **€ 25 (€ 10 / Stunde) oder course credits als Aufwandsentschädigung**.

Bei einer vorzeitigen Beendigung der Hauptuntersuchung erhalten Sie einen Betrag, der aliquot zu ihrem Zeitaufwand bis dahin ist.

Möglichkeit zur Diskussion weiterer Fragen

Für weitere Fragen im Zusammenhang mit dieser Studie stehen Ihnen Ihre Studienleiterin und ihre Mitarbeiter*innen gern zur Verfügung. Auch Fragen, die Ihre Rechte als Proband*in in dieser Studie betreffen, werden Ihnen gerne beantwortet.
Namen der Kontaktperson bzw. der Kontaktpersonen:

Studienleiterin	Name: Dr. Bianca Schuster E-Mail: bianca.schuster@univie.ac.at Tel.: (0043) 1 4277 47131
Arbeitsgruppenleiter	Name: Univ.-Prof. Dr. Claus Lamm E-Mail: claus.lamm@univie.ac.at Tel.: (0043) 1 4277 47130

Appendix B: Questionnaire values per individual

SERIAL	sum_depression	depression_level	ppt_ID	depression_binary
TOM01	9	Not Depressed	1	0
TOM02	5	Not Depressed	2	0
TOM03	26	Moderate Depression	3	1
TOM04	27	Moderate Depression	4	1
TOM05	10	Not Depressed	5	0
TOM06	13	Not Depressed	6	1
TOM07	38	Moderate Depression	7	1
TOM08	21	Mild Depression	8	1
TOM09	11	Not Depressed	9	1
TOM10	4	Not Depressed	10	0
TOM11	4	Not Depressed	11	0
TOM12	10	Not Depressed	12	0
TOM13	22	Mild Depression	13	1
TOM14	23	Mild Depression	14	1
TOM15	18	Mild Depression	15	1
TOM16	7	Not Depressed	16	0
TOM17	9	Not Depressed	17	0
TOM18	36	Moderate Depression	18	1
TOM19	7	Not Depressed	19	0
TOM20	11	Not Depressed	20	1
TOM21	7	Not Depressed	21	0
TOM22	20	Mild Depression	22	1
TOM23	8	Not Depressed	23	0
TOM24	3	Not Depressed	24	0
TOM25	22	Mild Depression	25	1

Appendix C: Questionnaire

1. Einschlafen:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft

Ich brauchte nie länger als 3O Minuten um einzuschlafen.

☐

Ich brauchte bei weniger als der Hälfte der Nächte mindestens 3O Minuten um einzuschlafen.

Ich brauchte bei mehr als der Hälfte aller Nächte mindestens 3O Minuten um einzuschlafen.

Ich brauchte bei mehr als der Hälfte der Nächte mehr als 6O Minuten um einzuschlafen

2. Nächtlicher Schlaf:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich wachte nachts nicht auf.

Ich hatte einen unruhigen, leichten Schlaf und wachte jede Nacht ein paar Mal kurz auf.

Ich wachte mindestens einmal pro Nacht auf; schlief jedoch leicht wieder ein.

Ich wachte in mehr als der Hälfte der Nächte mehr als einmal pro Nacht auf und blieb mindestens 2O min wach.

3. Frühes Aufwachen:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Meistens wachte ich nicht früher als 3O Minuten, bevor ich aufstehen musste, auf.

In mehr als der Hälfte der Tage der letzen Woche wachte ich mindestens 30 bis 60 Minuten bevor ich aufstehen musste, auf.

In mehr als der Hälfte der Tage der letzen Woche wachte ich mindestens eine Stunde zu früh auf, konnte jedoch wieder einschlafen.

In mehr als der Hälfte der Zeit wachte ich mindestens eine Stunde zu früh auf ohne wieder einschlafen zu können.

4. Erhötes Schlafbedürfnis:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich schlief pro Nacht nicht mehr als 7-8 Stunden und brauchte keine Nickerchen.

Ich schlief innerhalb von 24 Stunden nicht mehr als 1O Stunden einschliefllich Nickerchen.

Ich schlief innerhalb von 24 Stunden nicht mehr als 12 Stunden einschliefllich Nickerchen.

Ich schlief innerhalb von 24 Stunden mehr als 12 Stunden einschliefllich Nickerchen.

5. Gefühl von Traurigkeit:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft

Ich fühlte mich nicht traurig.

Ich fühlte mich weniger als die Hälfte der Zeit traurig.

Ich fühlte mich mehr als die Häfte der Zeit traurig.

Ich fühlte mich während der gesamten Zeit traurig

6. Gereizte Stimmung:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich fühlte mich nicht gereizt.

Ich fühlte mich weniger als der Hälfte der Zeit gereizt.

Ich fühlte mich mehr als die Hälfte der Zeit gereizt.

Ich fühlte mich die gesamte Zeit gereizt.

7. Gefühl von Angst und Anspannung:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich fühlte mich nicht ängstlich oder angespannt.

Ich fühlte mich weniger als die Hälfte der letzen Woche ängstlich (angespannt).

Ich fühlte mich mehr als die Hälfte der letzten Woche ängstlich (angespannt).

Ich fühlte mich die ganze letzte Woche ängstlich (angespannt).

8. Stimmungsreaktion auf positive Ereignisse:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Nach positiven Ereignissen verbesserte sich meine Stimmung für mehrere Stunden bis zum Normalniveau.

Nach positiven Ereignissen verbesserte sich meine Stimmung, jedoch nicht bis zum Normalniveau.

Meine Stimmung verbesserte sich nur nach wenigen und bestimmten positiven Ereignissen.

Meine Stimmung verbesserte sich überhaupt nicht, auch nicht nach sehr positiven oder erwünschten Ereignissen.

9. Stimmungsschwankungen im Laufe des Tages:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft

Zwischen meiner Stimmung und der Tageszeit gab es keinen klaren Zusammenhang.

Meine Stimmung hing oft von Ereignissen und Umständen zu bestimmten Tageszeiten ab (z.B. Alleinsein, Arbeit)

Im allgemeinen war meine Stimmung mehr von der Tageszeit als von Ereignissen abhängig.

Meine Stimmung war zu bestimmten Tageszeiten eindeutig und vorhersagbar besser oder schlechter.

9a. Wann war Ihre Stimmung normalerweise schlechter?

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft

- morgens.
- nachmittags.
- abends.

9b. War Ihre Stimmungsschwankung von der Umgebung abhängig?

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

- ja.
- nein.

10. Qualität Ihrer Stimmung:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft

- Die Stimmung (innere Gefühle), die ich empfand, entsprach weitgehend meiner normalen Stimmung.
- Meine Stimmung war traurig im Sinne von Traurigkeit, die ich nach dem Tod oder Verlust einer nahestehenden Person empfinden würde.
- Meine Stimmung war traurig. Diese Traurigkeit hatte eine andere Qualität als die Traurigkeit nach dem Tod oder dem Verlust einer nahestehenden Person.
- Meine Stimmung war traurig und unterschied sich von der, die man mit Verlust oder Trauer in Verbindung setzt.

11. Verminderter Appetit:

Bitte entweder Frage 11 oder 12 beantworten (nicht beide!)

- Mein Appetit war normal und unverändert.
- Ich habe weniger oft oder geringere Mengen als gewöhnlich gegessen.
- Ich habe deutlich weniger als gewöhnlich gegessen und musste mich dazu auch noch überwinden.
- Ich habe im Laufe von 24 Stunden kaum etwas gegessen und nur mit äußerster Anstrengung oder wenn mich andere überredet haben, zu essen.

12. Gesteigerter Appetit:

Bitte entweder Frage 11 oder 12 beantworten (nicht beide!)

Mein Appetit war normal und unverändert.

Ich verspürte ein Bedürfnis häufiger als üblich zu essen.

Ich habe regelmäflig häufiger und größere Mengen als üblich gegessen.

Ich verspürte einen Drang mehr zu essen, sowohl bei den Mahlzeiten als auch dazwischen.

13. Während der letzten 2 Wochen:

Bitte entweder Frage 13 oder 14 beantworten (nicht beide!)

- hat sich mein Gewicht nicht geändert.
- habe ich wahrscheinlich etwas abgenommen.
- habe ich 1-2,5 kg abgenommen.
- habe ich mehr als 2,5 kg abgenommen.

14. Während der letzten 2 Wochen:

Bitte entweder Frage 13 oder 14 beantworten (nicht beide!)

- hat sich mein Gewicht nicht geändert.
- habe ich wahrscheinlich etwas zugenommen.
- habe ich 1- 2,5 kg zugenommen.
- habe ich mehr als 2,5 kg zugenommen.

15. Konzentration und Entscheidungsvermögen

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

- Mein Konzentrations- und Entscheidungsvermögen war unverändert.
- Ich fühlte mich gelegentlich unentschlossen und unaufmerksam.
- Meistens hatte ich sehr große Schwierigkeiten, mich zu konzentrieren oder mich zu entscheiden.
- Ich konnte mich auf das Lesen nicht ausreichend konzentrieren oder selbst weniger wichtige Entscheidungen nicht treffen.

16. Selbstbild:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

- Ich betrachtete mich als genauso wertvoll und verdienstvoll wie andere Menschen.
- Ich klagte mich öfter als gewöhnlich selbst an.
- Ich glaubte meistens, dass ich für andere nur Probleme verursachte.
- Ich grübelte fast die gesamte Zeit über größere und kleinere Fehler meiner Person.

17. Sicht meiner Zukunft:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Bezüglich meiner Zukunft war ich optimistisch.

Ich war gelegentlich pessimistisch bezüglich meiner Zukunft; aber meistens glaubte ich, dass sich die Dinge zum Besseren entwickeln.

Ich war mir ziemlich sicher, dass meine nächste Zukunft (1-2 Monate) nicht viel Gutes bringen wird.

Ich sah keine Hoffnung, dass mir in der Zukunft jemals etwas Positives passieren wird

18. Gedanken an Tod oder Selbstmord:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich hatte keinerlei Gedanken an Selbstmord oder Tod.

Ich empfand das Leben als leer bzw. fragte mich, ob es lebenswert ist.

Ich dachte mehrfach in der Woche während mehrerer Minuten an Selbstmord oder den Tod.

Ich dachte jeden Tag wiederholt und im Detail an Selbstmord oder Tod bzw. machte ernsthaft Selbstmordpläne oder versuchte tatsächlich, mich umzubringen.

19. Allgemeine Interessen:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Meine Interessen an anderen Menschen oder Aktivitäten waren unverändert.

Ich bemerkte eine Verminderung meines Interesses an Leuten oder Aktivitäten.

Ich merkte, dass ich mich nur noch für eine oder zwei meiner früheren Aktivitäten interessierte.

Ich hatte überhaupt kein Interesse mehr an irgendwelchen früheren Aktivitäten.

20. Energieniveau:

Meine Energie war unverändert.

Ich ermüdete leichter als gewöhnlich

Ich musste mich sehr anstrengen, um mit meinen alltäglichen Aktivitäten (z.B.: Einkaufen, Haushaltsarbeit, zur Arbeit gehen, Kochen usw.) zu beginnen und sie abzuschließen.

Ich war aufgrund meiner Energielosigkeit nicht in der Lage, alltägliche Dinge zu erledigen.

21. Fähigkeit zu Freude und Vergnügen (außer sexuelle Aktivität):

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich genoss in gewohnter Weise angenehme Aktivitäten.

Ich empfand nicht wie sonst Vergnügen an angenehmen Aktivitäten.

Ich empfand selten Vergnügen an irgendwelchen Aktivitäten.

Ich war wirklich unfähig, Freude oder Vergnügen an irgendeiner Aktivität zu empfinden

22. Sexuelles Interesse (bitte Interesse und nicht Aktivität angeben)

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich hatte ein unverändertes Interesse an Sex.

Mein Interesse an Sex war etwas geringer als üblich bzw. ich hatte nicht das gleiche Vergnügen wie früher.

Ich hatte wenig Interesse an Sex bzw. hatte dabei selten Freude.

Ich hatte gar kein Interesse an Sex bzw. hatte keine Freude dabei.

23. Verlangsamung:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft

Die Geschwindigkeit meines Denkens, Sprechens und meiner Gesten war unverändert.

Ich empfand mein Denken als langsamer bzw. meine Stimme als stumpf und matt.

Auf die meisten Fragen konnte ich erst nach einigen Sekunden antworten, und ich bin sicher, dass mein Denken verlangsamt war.

Oft fiel es mir ausgesprochen schwer, auf Fragen zu antworten.

24. Ruhelosigkeit:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich fühlte mich nicht ruhelos.

Ich war oft zappelig, knetete meine Hände und rutschte beim Sitzen hin und her.

Ich fühlte mich gezwungen herumzugehen und war ziemlich rastlos.

Manchmal konnte ich nicht stillsitzen und musste ständig herumlaufen.

25. Schmerzen:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich hatte keinerlei Schweregefühl in Armen oder Beinen und keine Schmerzen.

Manchmal bekam ich Kopf-, Bauch-, Rücken- oder Gelenkschmerzen, die mich jedoch nicht von meinen üblichen Aktivitäten abhielten.

Ich hatte die oben genannten Schmerzen die meiste Zeit.

Die genannten Schmerzen waren so stark, dass ich gezwungen war, meine jeweilige Aktivität abzubrechen.

26. Weitere körperliche Beschwerden:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich hatte keines der folgenden Symptome: Herzrasen, verschwommenes Sehen, Schwitzen, Hitzewallungen oder Kältegefühl, Ohrensausen, Brustschmerzen oder Zittern.

Ich hatte einige dieser Symptome; sie waren jedoch nur leicht ausgeprägt und nur zeitweilig vorhanden.

Ich hatte mehrere dieser Symptome, die mich ziemlich störten.

Ich hatte mehrere dieser Symptome, und wenn sie auftraten, musste ich jegliche Aktivität abbrechen.

27. Panik und Angst:

Ich hatte keine zeitweise auftretende panische Angst oder Angst vor etwas Bestimmtem (Phobien), wie z.B. vor Tieren oder Höhe.

Ich hatte Zustände von nicht sehr ausgeprägter panischer Angst oder Phobien, die normalerweise mein Verhalten nicht änderten bzw. mich nicht vom normalen Leben abhielten.

Ich hatte Zustände von ausgeprägter panischer Angst oder Phobien, die mich dazu zwangen mein Verhalten zu ändern, mich aber nicht von meinem normalen Leben abhielten.

Ich hatte mindestens einmal pro Woche Panikanfälle oder ausgeprägte Phobien, die mich an der Ausführung meiner täglichen Aktivitäten hinderten.

28. Verdauungsbeschwerden:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Mein Stuhlgang war unverändert.

Ich hatte gelegentlich Verstopfung oder leichten Durchfall.

Ich litt die meiste Zeit unter Verstopfung oder Durchfall, was jedoch meine tägliche Funktionsfähigkeit nicht beeinträchtigte.

Ich litt an Verstopfung oder Durchfall. Dies erforderte eine medizinische Behandlung bzw. beeinträchtigte meine täglichen Aktivitäten.

29. Zwischenmenschliche Empfindsamkeit:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ich habe mich durch andere weder abgewiesen, noch beleidigt, kritisiert oder gekränkt gefühlt.

Ich habe mich durch andere gelegentlich abgewiesen, beleidigt, kritisiert oder gekränkt gefühlt.

Ich habe mich durch andere häufig abgewiesen, beleidigt, kritisiert oder gekränkt gefühlt, was aber nur leichte Auswirkungen auf meine Beziehungen zu anderen Personen oder Arbeit hatte.

Ich habe mich durch andere häufig abgewiesen, beleidigt, kritisiert oder gekränkt gefühlt, was zu einer spürbaren Beeinträchtigung meiner Beziehungen zu anderen Personen und zu meiner Arbeit führte.

30. Schweregefühl/körperliche Energie:

Bitte kreuzen Sie zu jeder der folgenden Behauptungen jeweils nur eine Antwort an und zwar die, die auf Sie für die vergangenen 7 Tage am ehesten zutrifft.

Ein Gefühl von bleierner Körperschwere oder mangelnder Energie habe ich in der letzten Woche nicht erlebt.

Ich hatte gelegentlich ein Gefühl von bleierner Körperschwere und mangelnder Energie, jedoch ohne Beeinträchtigung von Arbeit, Schule oder Aktivitätsniveau.

Ich hatte mehr als die Hälfte der letzen Woche ein Gefühl von bleierner Körperschwere (Mangel an körperlicher Energie).

Ich hatte die meiste Zeit ein Gefühl von bleierner Schwere (Mangel an körperlicher Energie), d.h.mehrere Stunden am Tag und mehrere Tage pro Woche.