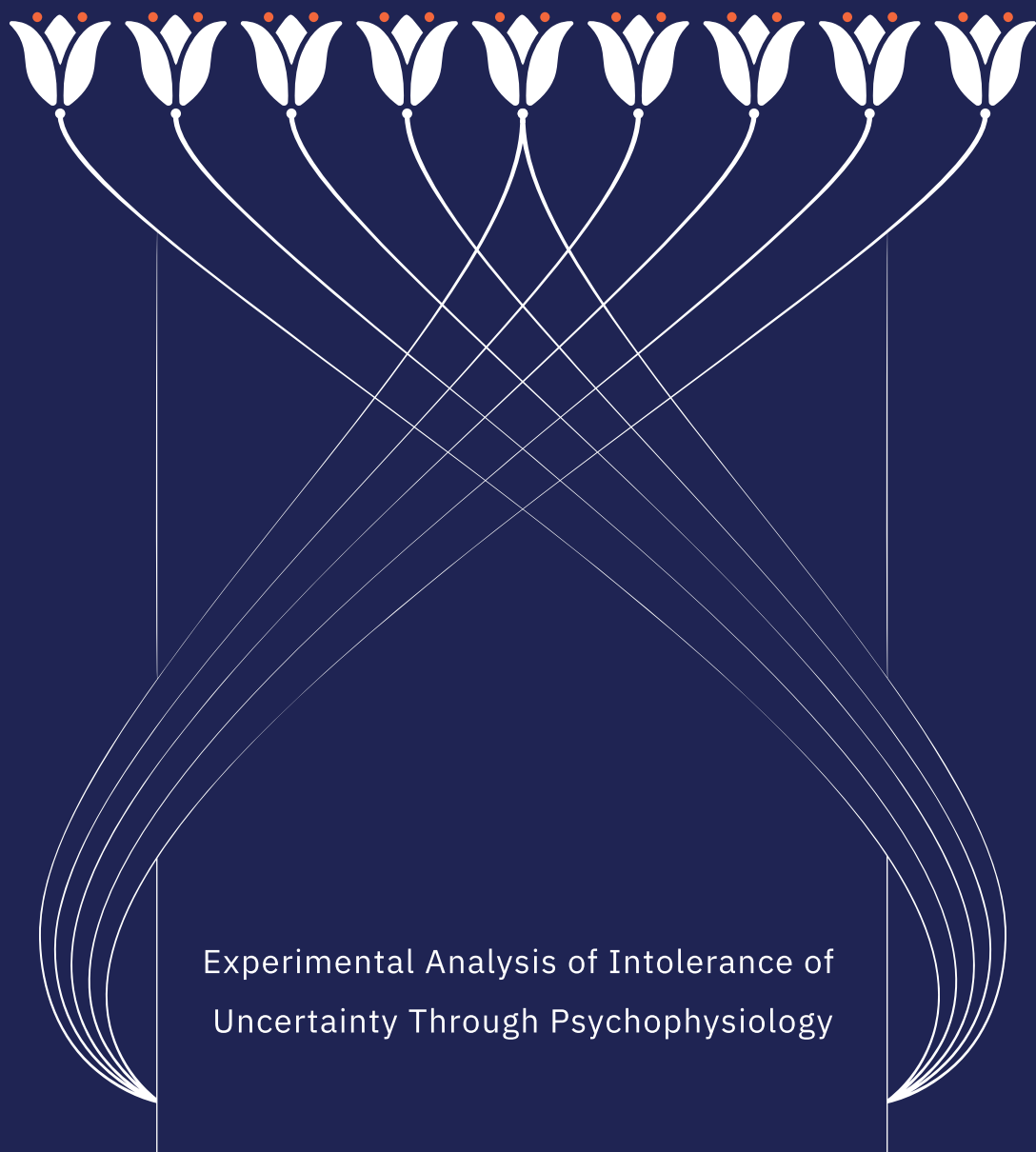


WHAT THE FUTURE HOLDS



Experimental Analysis of Intolerance of
Uncertainty Through Psychophysiology

M A R C E L O M A L B E C

**What the Future Holds:
Experimental Analysis of Intolerance of
Uncertainty Through Psychophysiology**

Marcelo Malbec

1. Intolerance of uncertainty is better conceptualized as a context-sensitive disposition rather than a general mechanism of vulnerability (this thesis).
2. Support for strong and general effects of intolerance of uncertainty across performance monitoring, fear learning and extinction, and reward processing is limited (this thesis).
3. Self-report, behavioral, and neurophysiological measures often capture distinct levels of analysis that are only weakly interrelated (this thesis).
4. A well-powered null finding supported by open science can be as compelling as the asterisk of statistical significance (this thesis).
5. The underreporting of null findings obscures the true strength of associations between individual differences and neurocognitive measures (this thesis).
6. To understand what happens in the mind, it is often informative to ask the body as well.
7. Doing science is an act of connection that links past knowledge, present needs, and the communities it serves.
8. The world we bring forth with others combines solidity with shifting sands (based on Humberto Maturana and Francisco Varela).
9. Although the future does not yet exist, imagined futures actively shape decisions and actions in the present (based on Stefan Bergheim).
10. Open science demands courage, but ultimately protects: transparency strengthens both the researcher and the research.
11. We scare because we care (Monsters INC's motto).

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ISBN 978-94-6534-496-6

Cover Anastassia Wilhelm | www.alunawork.com

Layout Anastassia Wilhelm | www.alunawork.com

Printing ProefschriftMaken | www.proefschriftmaken.nl

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What the Future Holds: Experimental Analysis of Intolerance of Uncertainty
Through Psychophysiology

Wat de toekomst in petto heeft: Experimentele analyse van intolerantie voor
onzekerheid met behulp van psychofysiologie

Thesis

to obtain the degree of Doctor from

Erasmus University Rotterdam

by command of the

rector magnificus

Prof.dr.ir. A.J. Schuit

and in accordance with the decision of the Doctorate Board.

The public defence shall be held on

Thursday 9 July 2026 at 15:30 hrs

by

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Chapter 1



General Introduction

Introduction

Numerous situations in human experience evoke uncertainty, bringing individuals into closer contact with the unknown. Consider, for instance, the experience of migration. Relocating to a different country inherently involves confronting numerous unfamiliar elements. Individuals facing this situation will experience varied emotions. While some of these may be excited, the presence of the unknown can also give rise to feelings of unease, apprehension, or anxiety. Importantly, the type and intensity of this response can vary considerably from one person to another, suggesting that individual differences play a key role in how uncertainty is experienced.

Intolerance of Uncertainty

Uncertainty is an inherent and pervasive aspect of human life. Despite our constant efforts to predict and control the future, the unpredictable nature of reality often challenges these endeavors. The psychological construct that reflects the tendency to perceive uncertainty as threatening and undesirable is called intolerance of uncertainty (IU). IU is a dispositional characteristic where individuals are unable to endure ambiguous or uncertain situations, perceiving them as aversive and threatening (Carleton, 2012, 2016; Freeston et al., 1994; Dugas et al., 2005). This tendency significantly influences various aspects of cognition, emotion, and behavior (Mahoney & McEvoy, 2012b). IU is typically measured using self-report questionnaires. The questions aim to explore individuals' reactions and beliefs surrounding uncertainty and uncertain scenarios. IU is understood to manifest in two primary ways: prospective IU and inhibitory IU. Prospective IU is characterized by a proactive desire for predictability and an active seeking of certainty. In contrast, inhibitory IU involves reactive avoidance, behavioral inhibition, and paralysis when confronted with uncertainty (Berenbaum et al., 2008; Birrell et al., 2011; Carleton, 2012).

As a transdiagnostic factor for internalizing disorders, IU is positioned in a key place that can help us to better understand the experience of people struggling with internalizing symptoms and how these symptoms interact with each other. One approach that can help us understand IU's position in this complex diagnostic landscape is the Research Domain Criteria (RDoC) framework. RDoC proposes a dimensional approach, focusing on core domains to address diagnostic heterogeneity (Insel et al., 2010; Cuthbert & Insel, 2013). Briefly, the cur-

rent RDoC domains are: Negative Valence Systems, Positive Valence Systems, Cognitive Systems, Systems for Social Processes, Arousal/Regulatory Systems, and Sensorimotor Systems (National Institute of Mental Health, n.d). IU has been associated with multiple RDoC domains, for example: IU aligns with Negative Valence Systems through heightened sensitivity to potential threat (Grupe & Nitschke, 2013); with Cognitive Systems through alterations in attention, cognitive control, and decision-making under uncertainty (Tanovic et al., 2018; Luhmann et al., 2011); and with Arousal/Regulatory Systems through dysregulated emotional and physiological responses (Carleton, 2016; Morriss, 2021). This cross-domain relevance positions IU as a unifying mechanism for understanding shared vulnerabilities between anxiety and depression, offering new avenues for research and intervention. Given IU's importance as a transdiagnostic factor, it is now crucial to investigate its specific role in cognitive processes relevant to internalizing disorders. This dissertation therefore aims to thoroughly study IU's influence on various cognitive processes, seeking to define the scope of its impact and its limitations. A deeper understanding of IU's transdiagnostic function can clarify its widespread effects and help develop better interventions, especially for individuals not adequately served by current categorical diagnostic approaches.

IU is both a risk and a maintaining factor for several internalizing disorders, including generalized anxiety disorder (GAD), social anxiety disorder (SAD), obsessive-compulsive disorder (OCD), panic disorder, and depression. As a risk factor, IU heightens vulnerability for internalizing disorders by amplifying threat perceptions and impairing adaptive coping strategies (Carleton, 2016; McEvoy & Mahoney, 2012). Individuals with elevated IU interpret ambiguous situations as threatening, which leads to chronic worry, rumination, and avoidance, all core features of internalizing disorders (Dugas et al., 2005; Mahoney & McEvoy, 2012b). IU also exacerbates maladaptive cognitive processes, such as distress intolerance and overestimation of negative outcomes, increasing the likelihood of internalizing symptoms (McEvoy et al., 2019; Shihata et al., 2016). As a maintaining factor, IU perpetuates internalizing disorders by reinforcing maladaptive coping mechanisms, such as excessive reassurance-seeking, repetitive checking, and avoidance (Tolin et al., 2003; Flores et al., 2018). These behaviors prevent individuals from learning that uncertain situations are manageable, thereby sustaining symptoms over time (Dugas et al., 2005). Additionally, individuals with high IU exhibit resistance to learn safety, as evidenced by persistent physiological responses to formerly threatening stimulus (e.g., heightened skin conductance reactivity) and difficulties in updating safety-related information (Morriss et al.,

2016; Morriss, Wake, et al., 2021).

Importantly, IU is also a modifiable target for therapeutic intervention for internalizing disorders. Research has shown that reductions in IU during therapy are associated with significant improvements in symptoms, particularly for anxiety disorders. For example, Dugas and Ladouceur (2000) developed a cognitive-behavioral therapy (CBT) protocol specifically targeting IU in individuals with GAD, demonstrating that decreases in IU were correlated with symptom reduction. Similarly, Boswell et al. (2013) found that greater reductions in IU during transdiagnostic therapy were associated with lower posttreatment anxiety and depression symptoms across individuals with GAD, panic disorder, social anxiety, OCD, and comorbid depression. These findings highlight the central role of IU in shaping maladaptive responses to uncertainty and its relevance for both understanding and treating internalizing disorders.

Exploring the Neurocognitive Correlates of IU

Psychopathology is intrinsically linked to altered cognitive functioning, including disruptions in performance monitoring, safety learning, and reward processing (e.g., Duits et al., 2015; Proudfit, 2015; Weinberg et al., 2010). Disruptions of these processes are increasingly considered central to the emergence and persistence of mental health symptoms. In this dissertation, I will focus on three cognitive processes:

- **Performance monitoring:** refers to the ability to continuously evaluate and adjust one's actions in response to mistakes or feedback. It helps in maintaining goal-directed behaviors by enabling the detection and correction of errors (Ullsperger et al., 2014; Dehaene et al., 1994). Deficits in performance monitoring are commonly observed in anxiety and obsessive-compulsive disorders, resulting in difficulty regulating behavior and heightened sensitivity to mistakes or perceived threats (Endrass & Ullsperger, 2014; Weinberg et al., 2010).
- **Fear acquisition and extinction:** refers to the process by which a neutral stimulus acquires threat value through association with an aversive outcome (Lonsdorf et al., 2017), which is an important mechanism for survival. Extinction, in turn, occurs when this stimulus stops predicting threat, resulting in a new association that inhibits the original one (Bouton, 1993; Bouton & King, 1983; Lonsdorf et al., 2017). This allows researchers to look into the expected reduction of defensive responses when threats are no longer pres-

ent. Extinction is compromised in anxiety-related conditions, often leading to an overgeneralization of threat and persistent fear responses even in safe contexts (Lissek et al., 2005; Duits et al., 2015).

- **Reward processing:** refers to how the brain evaluates and responds to positive outcomes, which is essential for adaptive motivated behavior and learning (Berridge & Kringelbach, 2015). Dysfunctions in reward processing, including reduced sensitivity to rewards, diminished capacity to experience pleasure, and impaired learning from positive feedback, are established as a marker of depression (e.g., Proudfit, 2015) and, to a lesser extent, anxiety (Nelson & Jarcho, 2021; Silk et al., 2012).

These interconnected cognitive processes are not merely disrupted in internalizing disorders; rather, they are central to the expression and maintenance of these conditions (e.g., Cuthbert & Insel, 2013; Harvey et al., 2004). Given the proposed role of IU as a transdiagnostic factor influencing these processes, expanding our understanding of their relationship with IU is essential for improving the conceptualization, diagnosis, and treatment of internalizing disorders.

These cognitive processes are often examined using behavioral tasks. Moreover, experimental studies consistently combine these behavioral tasks with physiological recordings (e.g., brain activity or skin conductance) to link biological signals to cognitive processes. The study of such physiological changes and their connection with psychological constructs is referred to as *psychophysiology*. These methods provide real-time, objective indices of neural and autonomic function, which complement self-report and observable behavior. This is particularly important for capturing mechanisms that may not be accessible through introspection or verbal report. Over decades, robust methodologies have been developed to map psychological experiences onto biological markers (Ax, 1964; Patrick & Hajcak, 2016; Ritz & Dien, 2023). In this dissertation, I employ a multi-modal psychophysiological approach combining EEG, SCR, and behavioral measures (see Box 1) to examine how IU is expressed in performance monitoring, threat and safety learning, and reward processing at the biological level.

Box 1.

Key methodological definitions.

Electroencephalography (EEG): A non-invasive neurophysiological method used to record the brain's continuous electrical activity. By placing electrodes on the scalp, EEG captures the electrical activity generated by large populations of neurons. Its primary strength lies in its excellent temporal resolution, allowing for the precise measurement of neural dynamics as they unfold in real-time (e.g., Schomer & Lopes da Silva, 2018).

Event-Related Potentials (ERPs): Derived from EEG recordings, they represent specific neural responses that are time-locked to the presentation of a particular event (e.g., a stimulus, feedback, or a motor response). By averaging segments of the EEG signal aligned to several repetitions of a specific event, consistent and clear neural responses of interest are distinguishable. Different components of the ERP waveform are thought to reflect distinct stages of cognitive processing, such as those involved in attention, error detection, or perception (e.g., Luck, 2014).

Steady-State Visually Evoked Potentials (ssVEP): A neural response generated in the visual cortex that is elicited by a visual stimulus flickering at a specific frequency. The brain's electrical activity (measured via EEG), synchronizes with this flicker, producing a steady-state oscillatory signal at the same frequency as the stimulus. The amplitude of this neural signal serves as an index of the attentional allocation to that stimulus, making it a powerful tool to track shifts in attention between different stimuli, such as threatening versus safe ones (Norcia et al., 2015; Vialatte et al., 2010).

Skin Conductance Response (SCR): A measure that reflects changes in the skin's electrical conductance. These changes are driven by the activity of sweat glands, which are innervated by the sympathetic nervous system. Because of this direct link, SCR serves as a sensitive index of sympathetic arousal, which is commonly used to measure emotional responses and cognitive engagement during relevant experimental events (Dawson et al., 2007).

Performance monitoring

Performance monitoring is particularly relevant to understanding how IU influences cognitive processing, as it predominantly involves coping with unpredictability. Errors, a central component of performance monitoring, have been conceptualized as forms of unpredictable and endogenous threat because their consequences can impact wellbeing (Weinberg et al., 2016). The body's defensive physiological reactions to mistakes are similar to its response to external threats (Hajcak and Foti, 2008; Hajcak et al., 2003; Pourtois et al., 2010). However, in contrast, errors turn our focus inward on our own behavior (Weinberg et al., 2016). Moreover, because errors are often unpredictable (Proudfit et al., 2013), they can be particularly distressing for people who cannot tolerate uncertainty.

The process of the detection of commission of errors is done rapidly by our brain and it can be measured using the EEG. The error-related negativity (ERN) is an electrophysiological index of error detection, reflected as a negative deflection in the ERP signal that occurs approximately 50–100 milliseconds after a person makes a mistake in a reaction-time task (Gehring et al., 1993). It represents the early registration of errors (Hoffmann and Falkenstein, 2012) and a signal of increased cognitive control and required behavioral adjustments (Weinberg et al., 2016). Research on IU's impact on performance monitoring remains equivocal. Some studies found no direct association between the total score of IU and ERN amplitude (Carsten et al., 2025; Jackson et al., 2016; Ruchensky et al., 2020). However, some studies reported distinct effects depending on IU subfactors: prospective IU has sometimes been associated with an enhanced ERN (Jackson et al., 2016, but see Grabowska et al., 2024; Carsten et al., 2025; Sandre & Weinberg, 2019), whereas inhibitory IU has been linked to attenuated ERN amplitudes (Jackson et al., 2016; Grabowska et al., 2024, though also see Carsten et al., 2025; Sandre & Weinberg, 2019). Thus, the mixed pattern of findings regarding the association between IU and performance monitoring underscores the need for further research using designs that can more precisely characterize these relationships, including larger samples and careful consideration of IU subfactors.

Fear acquisition and extinction

Another important cognitive process fundamentally linked to IU is fear acquisition and extinction learning. Both processes are tested using an experimental paradigm broadly referred to as fear conditioning, which typically uses a fear acquisition training phase to assess fear learning, followed by an extinction training phase to examine extinction learning (for a comprehensive review on this

experimental design, its elements, and variations see Lonsdorf et al., 2017). Traditionally, responses to conditioned stimuli have been measured using subjective ratings of fear, valence, and arousal, along with physiological indices such as SCR (Beckers et al., 2013). More recently, these methods have been complemented by EEG-derived measures such as the ssVEP (Miskovic & Keil, 2013; Wieser et al., 2014) and the late positive potential (LPP) of the ERP (e.g., Bacigalupo & Luck, 2018; Bauer et al., 2020).

Fear acquisition is typically studied using a differential conditioning procedure, where one neutral stimulus (CS+) is paired with an aversive unconditioned stimulus (US), while another neutral stimulus (CS-) is never paired with the US. This setup, referred to as fear acquisition training, leads to the development of conditioned fear responses selectively to the CS+. The resulting changes in physiological or behavioral responses reflect fear learning, which serves as an observable indicator of the underlying process of fear acquisition (Lonsdorf et al., 2017). Previous findings have shown an unclear picture regarding the impact of IU on the responses to threat (CS+) and safe (CS-) cues during fear acquisition. Some studies report that higher IU is linked with either impaired or enhanced discrimination between these cues, depending on experimental conditions such as reinforcement rate (Kanen et al., 2021; Chin et al., 2016). Johnson et al. (2023) did not find an overall IU effect on fear learning, but did report specific effects depending on IU subfactors: inhibitory IU was associated with reduced SCR discrimination between CS+ and CS-, whereas prospective IU was associated with enhanced SCR discrimination. Other studies show that the link between IU and fear ratings of the threat stimulus (CS+) is also unclear. Recent findings show this association can either persist (Klingelhöfer-Jens et al., 2022) or disappear (Mertens et al., 2022) when controlling for trait anxiety, though both studies found no IU effects on SCR, startle responses, or expectancy ratings during the fear acquisition training. In addition, several studies consistently failed to detect any reliable effect of IU on fear learning (Morriss, 2019; Morriss & van Reekum, 2019; Morriss, Christakou, & van Reekum, 2016; Morriss, Macdonald, & van Reekum, 2016; Morriss et al., 2015; Morriss, Saldarini, & van Reekum, 2019; Morriss, Saldarini, Chapman, et al., 2019; Morriss et al., 2020; Klingelhöfer-Jens et al., 2022; Sjouwerman et al., 2020; Wake et al., 2020, 2021; Wroblewski et al., 2022; see also Morriss, Zuj, & Mertens, 2021). Despite this apparent consensus that IU may not impact fear acquisition, examining this process remains critical. Methodologically, since studying extinction requires successful fear acquisition, it is important to also examine and

report acquisition results. Systematic reporting across diverse designs allows researchers to determine whether null effects reflect a true absence of association or whether IU plays a role under specific acquisition conditions.

While IU may not consistently impact fear acquisition, its influence has become more apparent during extinction. Extinction is examined by presenting the previously reinforced CS+ without the aversive US, leading to a reduction in conditioned responses over time. This change, referred to as extinction learning, reflects a new learning process rather than the erasure of the original association (LeDoux, 2014). A growing body of research shows that individuals high in IU tend to maintain threat-related responses even when the threat is no longer present (Bauer et al., 2020; Morriss et al., 2015; Morriss, Christakou, & van Reekum, 2016; Morriss, Macdonald, & van Reekum, 2016; Morriss, 2019; Morriss, Saldarini, & van Reekum, 2019; Morriss & van Reekum, 2019; Sjouwerman et al., 2020; Wake et al., 2020, 2021; Wroblewski et al., 2022). In other words, individuals with high IU show difficulties in learning that a previously threatening stimulus is now safe. High IU individuals often show a pattern of generalized responding early in extinction and a persistence in differentiating between CS+ and CS- over time, which contrasts with the typical reduction in this differentiation observed in low IU individuals (Morriss et al., 2015; Morriss, Macdonald, & van Reekum, 2016; Morriss, Saldarini, & van Reekum, 2019; Morriss, Saldarini, Chapman, et al., 2019; Wake et al., 2020, 2021). This pattern was also observed for both sub-factors (i.e., prospective IU and inhibitory IU) in a recent meta-analysis, however the effect sizes were weaker than for total IU (Morriss, Wake, et al., 2021). This difficulty in extinction learning is clinically relevant, as it mirrors the persistent heightened threat-related responses characteristic of anxiety disorders (Duits et al., 2015), and their often-observed resistance to extinction. In addition, extinction learning is theorized to reflect the mechanisms involved in cognitive-behavioral exposure therapy for pathological fears (Craske et al., 2014; Rachman, 1989; Vervliet et al., 2013). In this context, research on the role of IU in extinction can inform the development of tailored interventions aimed at helping individuals respond more adaptively to threat and safety cues.

Reward processing

A third cognitive process that will broaden the scope of our investigation of the impact of IU on cognition is reward processing. This process helps illustrate how uncertainty affects the way individuals form and update expectations about outcomes. Specifically, these expectations are shaped by the nature of the avail-

able information, whether the probabilities of outcomes are known (risk) or unknown (ambiguity). An important aspect of this process is the concept of reward prediction error, which is experienced when an actual outcome deviates from our expectations (Schultz, 2016; Watabe-Uchida et al., 2017). It is central for learning, as it helps us adjust our behavior based on whether a result was better or worse than anticipated. This reward prediction error can be identified through EEG, as it is captured by the brain activity evoked after feedback about the performance. The amplitude of this feedback-related ERP is modulated by outcome expectancy, with enhanced responses typically observed for outcomes that are unexpected or less probable (Holroyd & Coles, 2002; Sambrook & Goslin, 2015). In addition, this ERP tends to show larger amplitudes for positive compared to negative outcomes (Holroyd et al., 2008; Proudfit, 2015). In this dissertation, I will analyze the brain responses to rewards and no-rewards separately and referring to them as *feedback-related ERPs*. This decision to analyze the brain activity separately is motivated by previous research: first, evidence suggests that the brain activity for reward and no-reward originates from slightly different neural circuits (Fouragnan et al., 2015; Gheza et al., 2018); and second, separate analyses offer a clearer picture of individual differences compared to examining only the difference between feedback conditions (Clayson et al., 2021; Meyer et al., 2017). Previous studies usually use the terms reward positivity (RewP) or feedback-related negativity (FRN) for the brain potential after feedback presentation, and those ERPs are usually based on the differences between feedback conditions (Proudfit, 2015).

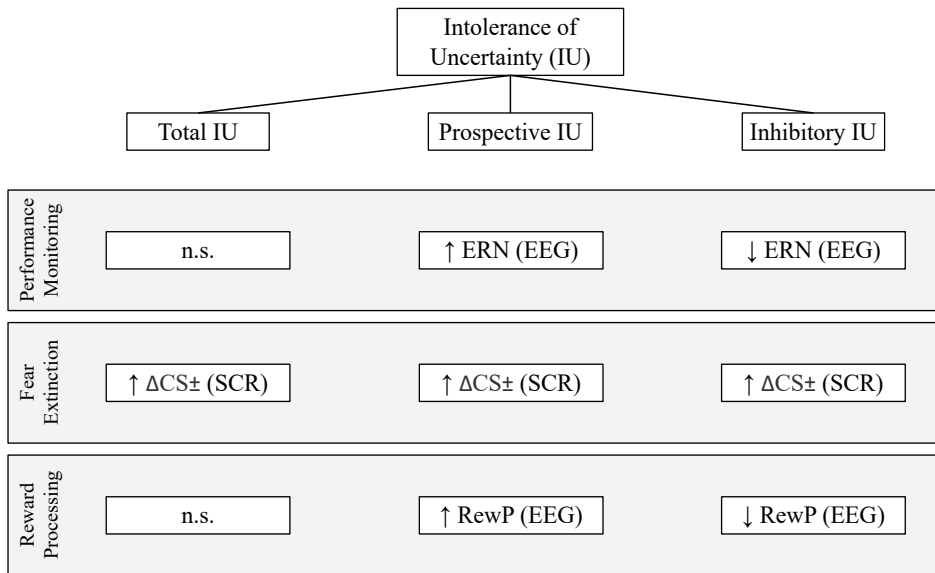
Previous results using the RewP or FRN offer valuable insights into how reward processing and IU impact internalizing disorders. In this line, previous research has reported a link between RewP amplitude to individual differences in reward sensitivity, both in behavior and self-report (Bress & Hajcak, 2013). Although the literature is still developing, initial evidence suggests that IU, particularly its subfactors, may shape responses to reward. Inhibitory IU has been associated with blunted neural responses to gains, while prospective IU has been linked to increased sensitivity to reward outcomes (using EEG: Nelson et al., 2016; using fMRI: Gorka et al., 2016). However, Muir et al. (2021) could not find a link between the level of IU and the RewP. Additional research is needed to clarify the associations between IU or its subfactors and the way that our brain processes rewarding outcomes.

Taken together, these three cognitive processes (i.e., performance monitoring, fear and extinction learning, and reward processing) provide a comprehensive framework for examining how IU influences human cognition. Figure 1 shows a

summary of the main previous findings on the impact of IU over these cognitive processes. By combining behavioral and psychophysiological methodologies, this dissertation aims to identify specific cognitive processes through which IU exerts its influence on internalizing symptoms. Following an RDoC-informed approach, this work will culminate in an integrative analysis to examine how these cognitive processes together explain individual differences in IU (*Chapters 2, 3, and 4*). Specifically, I will explore data-driven neuro-cognitive profiles derived from several variables collected in this project, both behavioral and physiological. Then, I will test how the distinct profiles relate to different levels of IU (in *Chapter 5*). Overall, a better understanding of these cognitive processes will help to specify how IU influences cognition and behavior. This, in turn, is essential for developing targeted interventions for individuals who struggle with uncertainty.

Figure 1.

Graphical summary of main previous findings regarding IU's impact on three cognitive processes (Performance monitoring, Fear extinction, and Reward processing).



Note. ↑ = larger response; ↓ = smaller response; n.s. = no statistically significant effect ($p \geq .05$) detected between IU facet and cognitive process main variables; ERN = error-related negativity; ΔCS± = threat-safety discrimination; RewP = reward positivity; EEG = electroencephalogram; SCR = skin conductance response

Outline of this Dissertation

A key methodological strength of this dissertation lies in the relatively large sample ($n = 214$) employed across experiments, particularly in the context of EEG research, where smaller samples have historically limited statistical power and reproducibility (Button et al., 2013; Clayson et al., 2019). Additionally, participants completed all three experimental tasks which allows for within-subject comparisons across cognitive processes. By increasing the number of participants beyond conventional EEG standards, this work enhances the reliability and precision of neural estimates, such as ERP components (e.g., ERN), and contributes to the growing call for more robust psychophysiological science (Keil et al., 2014; Boudewyn et al., 2018). In addition to statistical power, this dissertation also aligns with ongoing efforts to improve methodological transparency and generalizability in the field. We have employed *open science* practices in all studies, including pre-registration, code-sharing, and open data, to improve the transparency of our findings and conclusions, and facilitate reuse by other researchers (e.g., Carsten et al., 2025). Furthermore, the experimental designs presented here build upon previously reported associations between IU and cognitive functioning, forming hypotheses that are grounded in established empirical findings. This effort follows the logic of experimental replication (Hudson, 2021), which involves reproducing core theoretical effects while introducing systematic modifications to test the boundary conditions of existing claims. For instance, the studies examining performance monitoring were designed to test IU-related ERN effects observed by Jackson et al. (2016) and Ruchensky et al. (2020), while adapting task parameters to explore their generalizability. Such replications are essential for theory refinement, particularly when investigating constructs like IU that may be sensitive to task context, measurement modality, or subfactor composition (Morris, Zuj, & Mertens, 2021). By leveraging both increased statistical power, open science, and the logic of experimental replication, the present work aims to provide more reliable and theoretically informative evidence regarding IU's role in cognitive-affective functioning.

Chapter 2 describes an experiment about how IU (including its prospective and inhibitory subfactors) may influence performance monitoring. The central goal of this study is to determine whether IU modulates neural responses to errors, specifically the ERN and correct-response negativity (CRN). To address this question, I analyze EEG and behavioral data from a final sample of 188 participants who completed the Eriksen flanker task, a well-established paradigm for

eliciting response conflicts that often result in errors. Additionally, this study investigates whether IU moderates the relationship between depression or anxiety and error-related brain activity.

Chapter 3 investigates the role of IU in fear acquisition and extinction. The two central goals of this study are: 1) exploring the impact of IU on aversive learning (fear acquisition); 2) investigating the way that IU influences the subsequent learning of safety when the threat is no longer present (extinction). To provide a comprehensive answer, the study examines these processes from a multi-modal perspective. Using a differential fear conditioning paradigm, I measured various aspects of defensive responding in a final sample of 176 participants (self-report ratings, SCR, and electrocortical indicators such as ssVEP and LPP).

Chapter 4 examines the impact of IU on reward processing. The study investigates how IU influences responses to rewards and no-rewards in a modified Doors Task that includes varying levels of uncertainty. EEG data are recorded to capture electrocortical responses to reward and non-reward feedback, with results based on a final sample of 203 participants. By systematically manipulating uncertainty (e.g., low, moderate, high risk), the study tests whether IU or its subfactors (i.e., prospective IU and inhibitory IU) differentially relate to the processing of rewarding versus non-rewarding outcomes. It also explores whether worry, anxiety, and depression contribute to this relationship, providing a comprehensive overview of cognitive responses to uncertain reward contingencies.

Chapter 5 brings together data from the preceding experiments to develop a more comprehensive understanding of how IU and related constructs (worry, anxiety, and depression) are related to variations in behavioral and psychophysiological responses. Employing a latent profile analysis, this study aims to identify distinct subgroups or “profiles” that capture meaningful differences in how participants respond across cognitive processes (i.e., performance monitoring, fear and extinction learning, and reward processing). In doing so, this chapter’s overarching research question is whether these emergent profiles align with specific IU dimensions, offering a more nuanced view of the ways individuals navigate uncertainty in cognition.

Chapter 6 synthesizes the work presented in Chapters 2 through 5, offering an integrative discussion of the main findings and overarching conclusions. It outlines the strengths and limitations of the research, considers theoretical and applied implications, and proposes avenues for future investigation and policy development.

Chapter 2



No intolerance of errors: The effect of intolerance of uncertainty on performance monitoring revisited

This chapter has been published as:

Malbec, M., Hindmarsh, J. N., Jongerling, J., Franken, I. H., & Wieser, M. J. (2022). No intolerance of errors: The effect of intolerance of uncertainty on performance monitoring revisited. *International Journal of Psychophysiology*, 179, 77–88. <https://doi.org/10.1016/j.ijpsycho.2022.07.001>

A Corrigendum was added to fix minor errors in the original publication. In this dissertation the revised version is presented:

Malbec, M., Hindmarsh, J. N., Jongerling, J., Franken, I. H., & Wieser, M. J. (2024). Corrigendum to “No intolerance of errors: The effect of intolerance of uncertainty on performance monitoring revisited” [*International Journal of Psychophysiology*, 179, 77–88 (2022)]. *International Journal of Psychophysiology*, 196, 112285. <https://doi.org/10.1016/j.ijpsycho.2023.112285>

Abstract

Errors have been conceptualized as internal forms of threat that can cause harm in unpredictable ways. An index of error processing is the error-related negativity (ERN), an event-related potential reflecting variability in the sensitivity to errors. Prior work has shown the relationship between psychopathology symptoms and the ERN is unclear, and may be moderated by intolerance of uncertainty (IU), a trait that captures how people react to unpredictability. IU includes two subfactors of prospective IU (active seeking of predictability) and inhibitory IU (behavioral paralysis). In the present study, 188 undergraduates performed an Eriksen flanker task designed to elicit the ERN, while brain activity was recorded using electroencephalography (EEG). Participants completed the Intolerance of Uncertainty Scale, Short Form (IUS-12), and other measures of anxiety, depression and worry. Total IU explained 5% of the variance in correct-response negativity (CRN), but was not associated with the ERN in our sample. In contrast to previous findings, the IU subfactors did not predict the ERN or post-error slowing (PES), nor did total IU and depression interact to predict the ERN. Exploratory analyses also showed that total IU did not moderate the relationship between trait anxiety and the ERN. Small samples may have previously exaggerated the links between self-reported IU and the ERN. As such, further high-powered replications are required to confirm if, and how, they are related.

Keywords: Intolerance of uncertainty; Error monitoring; Error-related negativity; Flanker task; EEG.

Introduction

Error processing and psychopathology

Errors are a vital source of information. Learning from our mistakes allows us to guide adaptive behaviors in response to a constantly changing environment. Error processing is part of a higher cognitive function called performance monitoring, which is the ability to scrutinize ongoing performance in order to detect, and therefore correct, mistakes (Dehaene et al., 1994). Despite the adaptive function of error processing, errors themselves have been conceptualized as forms of endogenous threat because their consequences can jeopardize wellbeing (Weinberg et al., 2016). Indeed, several defensive physiological responses observed following errors (e.g., increased startle reflex and pupil dilation, changes in the skin conductance response, increased amygdala activity (Hajcak and Foti, 2008; Hajcak et al., 2003; Pourtois et al., 2010) are similar to other exogenous types of threat, such as predators. However, unlike external threats, errors promote a uniquely interoceptive focus on one's behavior (Weinberg et al., 2016) and may be appraised more negatively by individuals at risk for internalizing psychopathology (Pasion and Barbosa, 2019). Moreover, errors are inherently unpredictable (Proudfit et al., 2013) which means they may be particularly aversive to people who cannot tolerate the unexpected or unknown, regardless of psychopathology status.

An electrophysiological index of error detection is the error-related negativity (ERN), a negative deflection in the event-related potential (ERP) that occurs 50–100 ms after a person makes a mistake in a reaction-time task (Gehring et al., 1993). The ERN represents early and automatic registration of errors (Hoffmann and Falkenstein, 2012) and functions as a signal for increased cognitive control and necessary behavioral adjustments (Weinberg et al., 2016). By contrast, more conscious and explicit processing of errors is indexed by the error positivity (Pe) which occurs 200–400 ms after error commission (Hewig et al., 2011). Enhanced ERNs have been observed in patients with obsessive-compulsive disorder (OCD; Endrass et al., 2008), generalized anxiety disorder (GAD; Weinberg et al., 2010) and social anxiety disorder (SAD; Endrass et al., 2008). While the ERN has been found to correlate with trait anxiety in non-clinical samples (Moser et al., 2013; Weinberg et al., 2015a), more recent meta-analytic work has found that the association between trait anxiety (as measured by the State-Trait Anxiety Inventory; Spielberger, 1983) and the ERN was smaller

than previously assumed after correcting for publication bias (Saunders and Inzlicht, 2020), with no overall correlation found between anxiety and the ERN across 29 non-clinical volunteer samples.

The relation between depression and the ERN is unclear, with studies finding both enhanced (Weinberg et al., 2012) and diminished ERNs (Holmes and Pizzagalli, 2008). One recent meta-analysis found no overall relationship between depression and ERN amplitude (Moran et al., 2017). Another meta-analysis found a negligible unique contribution of depression to the association between internalizing spectrum disorders and increased ERN (Pasion and Barbosa, 2019). Indeed, depression has been found to obscure the relationship between GAD and the ERN, with the enhanced ERN found in those with a single GAD diagnosis not visible in comorbid subjects (i.e., GAD + depression; Weinberg et al., 2015b). In short, the direct relationship between depression and the ERN appears weak at best. Findings suggesting weak associations between the ERN and symptoms are apparent even when considering symptoms at the transdiagnostic level (i.e., anxious-depression; Seow et al., 2020). These relationships might be more adequately explained by considering a potential underlying mechanism: variability in how people react to the unpredictability of threats.

Intolerance of uncertainty

Intolerance of uncertainty (IU) is a dispositional tendency that reflects an individual's inability to endure ambiguous or uncertain events (Carleton, 2012, Carleton, 2016; Freeston et al., 1994). Individuals with higher levels of IU then perceive and interpret these events as aversive and threatening (Dugas et al., 2005). IU is present in unselected samples, with higher levels associated with internalizing psychopathology, including GAD, SAD, OCD, panic disorder and depression (Mahoney and McEvoy, 2012). Moreover, IU is also associated with various factors that underlie such internalizing symptoms, such as greater rumination (Liao and Wei, 2011), elevated anxiety sensitivity (Carleton et al., 2007), increased checking behavior (Tolin et al., 2003), difficulties in safety learning (Morris, Wake et al., 2021) and avoidance (Flores et al., 2018). As such, IU is considered a transdiagnostic risk and maintaining factor for internalizing disorders (McEvoy et al., 2019). Further, IU and worry are also highly related, independent of shared variance with anxiety or depression symptoms (Dugas et al., 1997; Freeston et al., 1994). Two subfactors of IU are thought to capture different ways people interpret and respond to uncertainty. Prospective IU (P-IU) reflects a desire for predictability and active seeking of certainty, while inhibitory IU (I-

IU) points towards avoidance, behavioral inhibition, and paralysis in the face of uncertainty (Carleton, 2012).

Relationships between ERN, IU and psychopathology

Studies examining the relationship between IU and the ERN are limited and pose some contradictions. Two studies did not find a direct association between the total score of IU and the ERN (Jackson et al., 2016; Ruchensky et al., 2020). Concerning the IU subfactors, Jackson et al. (2016) observed that both subfactors had opposing associations with the ERN during a flanker task, which requires participants to rapidly discriminate a single centered element (e.g., letter) in a string (Eriksen and Eriksen, 1974). ERN amplitude was enhanced for prospective IU but attenuated for inhibitory IU. The enhanced ERN in P-IU might have indicated an increase in performance monitoring in response to an error, however, this did not translate into better overall task accuracy for this group (with P-IU and I-IU performing comparably). Relatedly, Jackson et al. (2016) also found behavioral associations with IU subfactors; P-IU was associated with slower post-correct reaction time (RT) and higher I-IU with faster RT for post-corrects and post-errors. Post-error slowing (PES) is the behavioral adaptation characterized by increased RTs on trials that follow error commission compared to correct ones (Schroder et al., 2020), and an indication that processes responsible for cognitive control have responded to the ERN (Weinberg et al., 2016).

These findings of differential ERN amplitude for IU subfactors in Jackson et al. (2016) were not replicated by Sandre and Weinberg (2019) who found no association between the ERN and either subscale during a flanker task. However, Sandre and Weinberg (2019) employed a modified version of the flanker task that also included ambiguous trials (i.e., where they explicitly instructed participants there would be no right or wrong responses). Inhibitory IU was associated with a smaller negative deflection in response to these trials. If this response to ambiguous trials still represents a response to the possibility of having made an error, Sandre and Weinberg's evidence of blunted performance monitoring for I-IU under conditions of uncertainty is not completely at odds with the I-IU-ERN attenuation found by Jackson et al. (2016). Still, the exact relationship between the IU subtypes and the ERN remains inconclusive. In the only study examining IU, ERN and psychopathology symptoms, Ruchensky et al. (2020) found that IU moderated the relationship between depression and the ERN. Specifically, depression symptoms were associated with a larger ERN, but only when IU was low (with no depression-ERN relationship at high levels of IU).

Of note, Ruchensky et al. (2020) found no significant results when examining the subscales of IU. The authors contend that one reason previous research may have failed to find an association between depression and the ERN (e.g., Moran et al., 2017) was by not considering other underlying confounding variables, such as IU.

The present study

This study aimed to investigate various relationships between intolerance of uncertainty, self-reported symptoms of anxiety/depression, and brain activity associated with error processing. We sought to extend the small body of literature focused on ERN and IU, where evidence has thus far been inconclusive (Jackson et al., 2016; Ruchensky et al., 2020; Sandre and Weinberg, 2019). Importantly, our replication used a large sample of undergraduates ($N = 188$) to ensure high statistical power in order to avoid the presence of chance findings that can occur with smaller samples. High-powered replications are especially needed considering the context of the replication crisis in ERP research (Clayson et al., 2019), whereby low samples can lead to an overestimation of the associations between electrophysiological and self-report variables (“false-positives”; Bernoster et al., 2019; Larson and Carbine, 2017). Our well-powered sample aims to disentangle discrepancies in the literature surrounding our constructs of interest. Specifically, mixed findings regarding the effect of IU subfactors on ERN amplitudes (Jackson et al., 2016; Sandre and Weinberg, 2019), mixed findings for direct relationships between depression error monitoring (Moran et al., 2017), and prior potential overestimation of the relationship between ERN and trait anxiety (Saunders and Inzlicht, 2020). We have attempted an experimental replication of Jackson et al. (2016) and Ruchensky et al. (2020), which seeks to verify previous findings using particular variables while adjusting the experimental procedure (Hudson, 2021). By these adjustments it differs from a direct or exact replication, which aims to repeat the identical procedure of its reference. An experimental replication, on the other hand, seeks increased generalizability of findings that were obtained by a specific design. We employed a version of the flanker task that has been previously used with other populations (Andreu et al., 2017; Bernoster et al., 2019; Franken et al., 2007, Franken et al., 2010, Franken et al., 2018; Zijlmans et al., 2021). While it differs from our replication references (Jackson et al., 2016; Ruchensky et al., 2020) in several features (i.e., the duration of stimulus presentation, type of stimulus, out-of-time feedback inclusion, number of trials and blocks),

this modified version does evoke the equivalent behavioral and neural effects related to performance monitoring (Franken et al., 2007, Franken et al., 2010).

The first objective of this study was to examine the relationship between the IU subfactors, ERN amplitudes and post-error slowing. Contradictions exist between Jackson et al. (2016) who found an enhanced ERN for P-IU compared to I-IU, and Sandre and Weinberg (2019), who found no effect. It should be noted that Sandre and Weinberg (2019) did find an attenuated ERN for ambiguous (feedback not given) trials, which we have argued could overlap with error trials. Therefore, we expected to replicate the results of Jackson et al. (2016) whereby P-IU would be associated with an enhanced ERN while I-IU would be associated with an attenuated ERN (Hypothesis 1). However, both Ruchensky et al. (2020) and Jackson et al. (2016) found no relationship between ERN and total IU, as such, we also tested if this possible relationship exists in a larger sample (Hypothesis 2). Similar to Jackson et al. (2016) we expected to find an effect of IU subfactor on post-error RT: we expected slower RT in higher P-IU after error trials and faster RT for increased I-IU after error trials. Additionally, we calculated post-error slowing (PES) as difference scores by subtracting average reaction time after correct responses from average reaction time after error responses (Schroder et al., 2020) of which we tested a possible association with IU scores (Ruchensky et al., 2020; Hypothesis 3). The second aim of this study was to perform a replication of the moderation analysis of Ruchensky et al. (2020). As such, we hypothesized that depression and IU would interact to predict the Δ ERN (i.e., difference in amplitudes between error trials and correct trials), such that higher levels of depression would be associated with larger Δ ERNs at low (but not medium or high) levels of IU.

Materials and methods

The hypotheses and methods of this study were pre-registered (<https://osf.io/c5yr3>). The final sample data and analyses (R script) are publicly available (<https://osf.io/4mtn3/>).

Participants

In total, 212 psychology undergraduate students from Erasmus University Rotterdam were invited to the lab session via an online recruitment system. After the data-related (EEG and behavioral responses) exclusion criteria (see below) were applied, a final sample of 188 participants was used for all anal-

yses (age range from 17 to 46 years, $M = 21.23$, $SD = 3.31$, 81 % female, 55 % Dutch). A breakdown of their ethnic self-identification was 74 % for White/Caucasian, 9 % for Asian, 4 % for Middle Eastern, 4 % for Black, 3 % for mixed, 2 % for Latin-American, and around 5 % for Other and non-declared. Participants received course credit and monetary compensation (€5) related to a different task in the same session (see Procedure). A priori exclusion criteria were family history of photic epilepsy, related to another task that used flickering images. Informed consent was obtained before participation, and the research protocol was approved by the ethical board of the Erasmus School of Behavioural and Social Sciences.

Questionnaires

After finishing the experimental protocol, participants completed the following self-report questionnaires (either in Dutch or English, for Dutch or international students, respectively):

The Intolerance of Uncertainty, Short Form (IUS-12; Carleton et al., 2007) is a shortened version of the original 27-item Intolerance of Uncertainty Scale (Freeston et al., 1994; Dutch translation: de Bruin et al., 2006). It assesses the degree to which individuals find uncertain scenarios distressing and undesirable, as well as their reactions to them. The items are rated on a five-point Likert scale ranging from 1 (not at all characteristic of me) to 5 (entirely characteristic of me). The scale ranges from 12 to 60, with higher scores indicating higher intolerance of uncertainty. In addition to the total score, two subfactors can be extracted, Prospective IU (7 items) and Inhibitory IU (5 items; McEvoy and Mahoney, 2011). Internal consistency was acceptable – good for the total scale IU ($\alpha = 0.87$) and both subfactors P-IU ($\alpha = 0.81$) and I-IU ($\alpha = 0.77$). Total IU scores in the current sample ranged from 12 to 51 ($M = 29.36$; $SD = 8.05$). Prospective IU ranged from 7 to 31 ($M = 18.21$, $SD = 5.21$) while Inhibitory IU ranged from 5 to 20 ($M = 5.81$, $SD = 7.68$). The distribution of IU total scores was found to be approximately normal (see Supplemental material for density plot). The response patterns/densities in this sample are comparable to community and undergraduate samples reported by Carleton et al. (2012) while being lower than clinical samples, as expected.

The Beck Depression Inventory, Second Edition (BDI-II; Beck et al., 1996; Dutch translation: Does, 2002) is a widely used self-report measure of depressive symptoms, both cognitive and somatic. It consists of 21 items where participants rate items on a four-point Likert scale ranging from 0 to 3 based on

perceived severity in the last two weeks. The score ranges from 0 to 63, with higher scores indicating more severe depression symptoms. Internal consistency of this scale was good ($\alpha = 0.94$). Scores in the current sample ranged from 0 to 34 ($M = 5.81$, $SD = 7.68$). The current sample showed less severity of depression scoring on the BDI-II than that of Ruchensky et al. (2020; $M = 11.68$; $SD = 8.51$) who reported 67 % of the sample in the “no depression” range (0–13; compared to our 85 %), 17 % in the “mild depression” range (14–19; compared to our 7 %) and 16 % in the moderate to severe depression range (20–63; compared to our 8 %). Normative data for the BDI-II in undergraduates from the USA ($M = 9.14$, $SD = 8.45$; Whisman and Richardson, 2015) and community samples from The Netherlands ($M = 10.6$, $SD = 10.9$; Roelofs et al., 2013) further illustrates that our sample’s mean and standard deviation are lower than that of comparable samples.

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990; Dutch translation: Van Rijsoort et al., 1999) is a 16-item self-report questionnaire that measures the trait of worry across the dimensions of excessiveness, generality and uncontrollability. Each item is rated on a five-point Likert scale from 1 (not at all typical of me) to 5 (very typical of me). The scale ranges from 16 to 80 with higher scores indicative of higher levels of trait worry. Internal consistency of this scale was good ($\alpha = 0.83$). Scores in the current sample ranged from 25 to 72 ($M = 44.97$; $SD = 10.82$).

The trait subscale of the State Trait Anxiety Inventory, Form Y (STAI-T; Spielberger, 1983; Dutch translation: Ploeg, 2000) is a self-report questionnaire consisting of 20 items that measure someone’s general propensity for anxiety. Respondents rate items based on how they generally feel, using a four-point Likert scale from 1 (almost never) to 4 (almost always), with higher scores indicating greater anxiety (range: 20 to 80). Internal consistency of this scale was acceptable ($\alpha = 0.79$). Scores in the current sample ranged from 24 to 66 ($M = 45.33$; $SD = 8.00$).

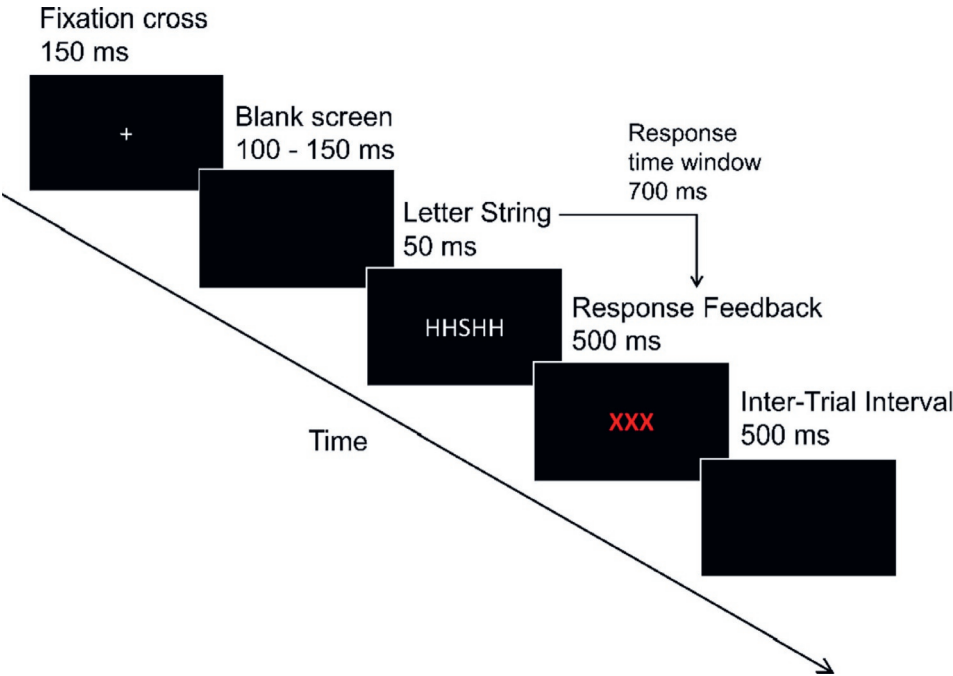
Task and materials

Participants completed an adapted version of the Eriksen flanker task (Franken et al., 2010). The task was programmed in E-prime 3 software (Psychology Software Tools, Pittsburgh, PA). There were four types of a letter string: two congruent (HHHHH and SSSSS) and two incongruent (SSHSS and HSHHH). The target letters array was displayed in white against a black background on a 20-inch PC monitor with a resolution of 1024×768 pixels which was placed at

a distance of 120 cm from the seated participant. Participants were instructed to answer by pressing either ‘1’ or ‘5’ on a QWERTY keyboard, as fast and accurately as possible, if the middle letter was either H or S. Those number buttons were associated with H and S in the instructions and the association was counterbalanced among participants.

A schematic depicting the progression of each trial can be seen in Figure 1. Each trial started with a fixation cross (+) visible for 150 ms, followed by a blank screen of random duration between 100 and 150 ms. The letters were displayed for 50 ms. Participants had 700 ms to respond using both hands. After each trial, participants received feedback for 500 ms in red font (OOO for correct; XXX for incorrect; ! for no answer). The inter-trial interval (ITI) had a duration of 500 ms. Participants completed a practice block of eight trials and the actual task consisted of five blocks of 80 trials with self-administered breaks in between (400 total trials). Participant response type (correct or incorrect) and reaction time (in ms) on every trial was recorded for later analysis.

Figure 1.
Schematic of a flanker task trial.



Note. A single trial lasted between 1600 and 1650 ms (accounting for the variable duration of the blank screen). A fixation cross was presented for 150 ms, followed by a blank screen

for 100–150 ms. The letter string was presented for 50 ms, then the screen remained blank for a further 650 ms (giving participants a 700 ms time frame to indicate their response). Feedback was then shown for 150 ms (XXX for incorrect, OOO for correct, ! for “too late”) and the trial ended with a blank screen of 500 ms.

Procedure

After arrival at the lab, participants received a verbal description of the experimental procedure from the researcher in charge and provided written informed consent. After this, they were sat in an isolated dimly-lit room where the EEG was attached. The estimated duration of the whole experimental procedure was as follows: first, they performed a Doors task (40 min) followed by a long break (10 min), secondly, there was a Fear Conditioning task (25 min), both tasks not reported here. After a last break (10 min), they performed the Flanker task (15 min). Duration lengths are estimated and vary according to self-administered breaks during the tasks. During the long breaks the researcher checked the overall status of the participant.

EEG recording and processing

Continuous EEG was recorded using a 32 + 2 electrodes cap arranged according to the 10/20 layout. Electro-oculogram (EOG) was recorded using 4 external electrodes: two above and below the left eye, and another two at the outer canthus of each eye. All electrodes were sintered Ag/AgCl electrodes. Data were recorded using the Active-Two BioSemi system (BioSemi, Amsterdam, Netherlands). This system includes two additional electrodes, CMS (Common Mode Sense) and DRL (Driven Right Leg), that serve as online reference and ground. The EEG data were digitized at a 512 Hz sampling rate and 24-bit A/D conversion. All electrode impedances were kept below 20 k Ω .

Offline EEG data were analyzed with Brain Vision Analyzer 2 (Brain Products, Gilching, Germany). Data were re-referenced to the average voltage at all scalp electrodes (i.e., average reference), band-pass filtered from 0.1 to 35 Hz (Butterworth zero phase filter with a 24 dB/octave roll-off), notch-filtered at 50 Hz, and corrected for eye movement artifacts (Gratton et al., 1983). Data were segmented in epochs of 1200 ms (400 ms before and 800 ms after response). Artifact rejection was applied to all epochs using: (a) an amplitude criterion of ± 75 μ V, (b) a gradient criterion of ± 50 μ V/ms, (c) a difference criterion of 200 μ V and (d) a low activity criterion of 2 μ V (interval length: 200 ms). Overall, the percentage of removed epochs by component was for the ERN 4.76 %, for the

CRN 4.04 %, for the Pe 4.56 %, and for the Pc, 4.25 %. Baseline correction was applied using the interval from 400 to 200 ms pre-response (Weinberg et al., 2010). From the 212 participants, two participant's questionnaire data was not saved and three participants were removed due to poor EEG data quality. Further, exclusion criteria based on the behavioral performance were: (a) participants who missed >20 % of trials of the flanker task ($n = 3$), or (b) participants who scored <55 % accuracy ($n = 10$; Seow et al., 2020). In addition, (c) participants with <10 error commissions were also excluded ($n = 6$; Olvet and Hajcak, 2009). Finally, (d) trials with reaction times below 200 ms were not further considered in the analysis (Sandre et al., 2020; Weinberg et al., 2015b). Altogether, 188 full datasets remained for further analysis after applying these criteria. The average number of error and correct trials in our sample was 50.01 ($SD = 25.29$), and 328.45 ($SD = 37.25$), respectively. Internal consistency of the ERPs and behavioral measures was calculated using odd-even split-half reliability with Spearman-Brown Correction (using SPSS v. 28). The reliability analysis of the ERPs showed good to excellent reliability (ERN: 0.92, CRN: 0.98, Pe: 0.85, Pc: 0.97), and the reliability analysis of the behavioral measures showed excellent reliability for accuracy (0.95) and for reaction times (0.98).

Power calculations

Power calculations for linear multiple regression were performed using G*Power3 (Faul et al., 2007). For analyses predicting the ERN amplitude from IU subfactors, we entered a minimum effect size of $f^2 = 0.11$ (corresponding to the R^2 of 0.10 found by Jackson et al., 2016), an alpha level of 0.05 and two predictors. A-priori tests showed that a sample size of $N = 90$ was required to achieve a power of 0.80. For our moderation analysis, we entered a minimum effect size of $f^2 = 0.09$ (corresponding to the R^2 of 0.08 found by Ruchensky et al., 2020), an alpha level of 0.05 and two predictors. A-priori tests showed that a sample size of $N = 114$ was required to achieve a power of 0.80. After applying the exclusion criteria, the sample size of this study ($N = 188$) had a power of 0.96 to detect an effect size of $f^2 = 0.09$, and a power of 0.99 to detect an effect size of $f^2 = 0.11$.

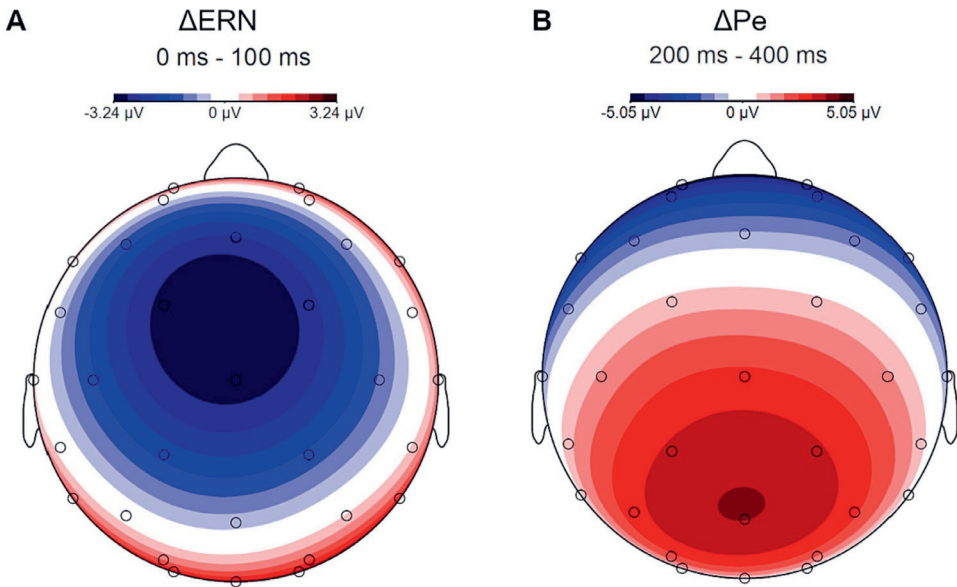
Data analysis

Average ERP waves were calculated for artifact-free trials at each electrode of interest in two conditions (correct and incorrect responses). The ERN and CRN were defined as the mean activity on error and correct trials, respectively, at 0 to 100 ms after the participant's response at Cz electrode, where the signal was

maximal (Sandre et al., 2020), see Figure 2a. The Pe and Pc were defined as the mean activity on error and correct trials, respectively, at 200 to 400 ms after response at both Cz and Pz electrodes (Ruchensky et al., 2020), see Figure 2b. We also calculated difference potentials to account for individual variation between correct and error trials. This is referred to as Δ ERN and Δ Pe, calculated as the difference between the respective error potential amplitude and correct potential amplitude (e.g., ERN - CRN).

Figure 2.

Topographic map depicting the average difference (μ V) between error and correct responses.



Note. Scalp maps display the grand average voltage distribution of (A) Δ ERN from 0 to 100 ms post-response, and (B) Δ Pe from 200 to 400 ms post-response. The voltage distributions are plotted on a minimum-maximum scale going from blue (negative polarity) to red (positive polarity). Compared to other electrodes, the most pronounced negative polarities were seen at Cz for the Δ ERN and Pz/Cz for the Δ Pe, during their respective time windows.

The behavioral measures extracted from the flanker task were: (1) number of correct trials (accuracy); (2) RT of the correct and incorrect trials; (3) average RTs of the next trial after error and correct feedback (i.e., post-correct and post-error RTs); (4) post-error slowing (PES), as the difference between average RT after error trials and the average RT after correct trials.

To examine the effects of the IU subfactors on performance monitoring, we ran a multiple regression using both IU subfactors (P-IU and I-IU) as simultaneous predictors and the ERN as the dependent variable. Next, we ran a simple linear regression with total IU as predictor as the ERN as the dependent variable. Further exploratory regression analyses were run for several dependent variables (ERN, Pe and RTs) using both total IU (single predictor) and the IU subfactors (simultaneous predictors). To assess the effect that depression and trait anxiety has on the relationship between IU and performance monitoring, we conducted a moderation analysis. Depression and trait anxiety levels were based on the participants' scores in the BDI-II and the trait scale of the STAI, respectively. For this, we conducted a multiple regression with total IU and the interaction term (depression $\times$ total IU) entered simultaneously to predict the Δ ERN, based on Ruchensky et al. (2020). Predictors were mean centered. Similar exploratory analyses were performed substituting (a) trait anxiety scores in the interaction term and (b) Δ Pe as the outcome variable.

All analyses were performed using R Statistical Software (v4.1.2; R Core Team, 2021). The assumptions for multiple linear regression were tested prior to running each confirmatory analysis and can be viewed in the Supplemental material. Our outlier treatment was consistent with the replication references: for the regression analyses we removed univariate outliers from the ERN data (Jackson et al., 2016), while for the moderation analysis neither univariate nor multivariate outliers were removed (Ruchensky et al., 2020). Correlations, t-tests, and regressions were performed using the 'stats' package (R Core Team, 2021). The figures were generated using the 'jtools' (Long, 2020) and 'ggplot2' (Wickham, 2016) packages, and a tutorial by Craddock (2016).

Results

Table 1 shows means, standard deviations, and correlations for variables of interest which ranged from small (Age and CRN, $r = 0.14$, $p < .05$) to large (P-IU and I-IU, $r = 0.63$, $p < .01$), with the IU subfactors having very large correlations with the total score IU (P-IU and IUS-12, $r = 0.93$, $p < .01$; I-IU and IUS-12, $r = 0.86$, $p < .01$). Both measures of the ERN were uncorrelated with any questionnaire variables. In contrast, the CRN amplitude correlated with total IU and both subfactors (all p values $< .01$), as well as with STAI-T ($p < .05$). Regarding the behavioral measures, both correlated with Pe amplitude (i.e., Correct %: $r = 0.30$, $p < .01$; PES; $r = 0.17$, $p = .021$), however, PES did not correlate with Δ Pe ($r = 0.11$, p

= .128). The accuracy rate was also correlated with the amplitudes of Δ ERN ($r = -0.17, p = .019$) and CRN ($r = 0.18, p = .014$). Further, both behavioral measures correlated negatively with trait anxiety scores (Correct %: $r = -0.24, p < .01$; PES; $r = -0.17, p = .019$), while PES was in addition negatively associated with I-IU scores ($r = -0.15, p = .038$). Interestingly, accuracy and PES were correlated with each other such that a higher accuracy rate was associated with longer PES ($r = 0.34, p \leq 0.01$).¹

¹ Because total IU, I-IU and STAI-T intercorrelated with several of our later outcome variables (CRN and PES), we assessed the specificity of IU-related measures above the STAI-T with several partial correlations. The correlation between total IU and CRN remained significant after controlling for STAI-T ($r = -.19, p = .011$). This speaks to the specificity of IU in predicting the CRN, above-and-beyond trait anxiety. However, the significant observed correlation of PES and I-IU did not persist after controlling for STAI-T ($r = -.09, p = .227$).

Table 1

Means, standard deviations, and correlations of study variables

Column Label	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. ΔERN (μV)	-3.27	3.13	1													
2. ΔPe (μV)	3.23	2.60	.23**	1												
3. ERN (μV)	0.28	3.69	.73**	.27**	1											
4. CRN (μV)	3.55	2.56	-.17*	.11	.55**	1										
5. Pe (μV)	4.00	2.57	.03	.74**	.33**	.44**	1									
6. Pc (μV)	0.77	1.87	-.28**	-.38**	.07	.44**	.34**	1								
7. IUS-12	29.36	8.05	.10	-.08	-.07	-.23**	-.11	-.03	1							
8. P-IU	18.21	5.21	.05	-.09	-.11	-.22**	-.09	-.00	.93**	1						
9. I-IU	11.15	3.68	.14	-.06	-.01	-.19**	-.10	-.06	.86**	.63**	1					
10. STAI-T	45.33	8.00	.09	-.11	-.03	-.16*	-.19*	-.10	.36**	.26**	.42**	1				
11. BDI-II	5.81	7.68	.08	-.04	-.03	-.14	-.10	-.08	.28**	.19*	.34**	.40**	1			
12. PSWQ	44.97	10.82	-.04	-.06	-.12	-.13	-.13	-.09	.49**	.39**	.52**	.54**	.61**	1		
13. Correct %	82.11	9.31	-.17*	.29**	-.02	.18*	.30**	.01	-.10	-.08	-.11	-.24**	-.06	-.06	1	
14. PES (ms)	20.64	30.02	.01	.11	-.02	-.05	.17*	.08	-.10	-.05	-.15*	-.17*	-.07	-.10	.34**	1
15. Age	21.23	3.31	-.19**	-.24**	-.06	.14*	-.12	.17*	-.12	-.08	-.16*	-.08	-.19*	-.12	.18*	-.04

Note. *N* = 188. ERN = Error related negativity; Pe = Error positivity; CRN = Correct response negativity; Pc = Correct positivity; IUS-12 = Intolerance of Uncertainty, Short Form; P-IU = Prospective IU; I-IU = Inhibitory IU; STAI-T = Trait subscale of the State-Trait Anxiety Inventory; BDI-II = Beck Depression Inventory, Second Edition; PSWQ = Penn State Worry Questionnaire; Correct % = percentage of correct responses; PES = PES traditional [Mean Post-Error RT – Mean Post-Correct RT]. Pearson product-moment correlation coefficients are given.

*indicates *p* < .05. ** indicates *p* < .01.

Behavioral results

Table 2 displays selected behavioral data for the flanker task. Paired-sample *t*-tests revealed behavioral patterns typical for the task. Mean error counts were larger for incongruent trials ($M = 35.32$, $SD = 16.19$), relative to congruent trials ($M = 14.69$, $SD = 10.83$; $t_{187} = -25.86$, 95% confidence interval (CI) $[-22.21, -19.01]$, $p < .001$, $d = 1.88$). Participants were faster on congruent trials ($M = 445.78$ ms, $SD = 26.90$ ms) versus incongruent trials ($M = 478.73$ ms, $SD = 29.70$ ms; $t_{187} = -31.63$, 95% CI $[-35.00, -30.89]$, $p < .001$, $d = 2.31$). Participants were also faster on error ($M = 418.52$ ms, $SD = 38.73$ ms) compared to correct trials ($M = 468.95$ ms, $SD = 26.22$ ms; $t_{187} = -25.48$, 95% CI $[-54.33, -46.52]$, $p < .001$, $d = 1.86$). Finally, post-error mean RTs ($M = 480.47$ ms, $SD = 40.32$ ms) were slower than post-correct mean RTs ($M = 459.83$ ms, $SD = 27.56$ ms; $t_{187} = -9.43$, 95% CI $[-24.96, -16.32]$, $p < .001$, $d = 0.70$).

Table 2

Descriptive Statistics of Flanker Task Behavioral Data

	<i>M</i>	<i>SD</i>	Minimum	Maximum
Accuracy (<i>n</i>)				
Correct	328.45	37.25	229	382
Error	50.01	25.29	12	128
Missed	19.10	14.78	2	79
Average RT (ms)	462.08	27.32	387.19	531.92
Correct	468.95	26.22	396.26	541.89
Error	418.52	38.73	304.70	517.18
Post-correct	459.83	27.56	389.89	538.46
Post-error	480.47	40.32	335.81	599.08

Note. $N = 188$. Participants completed 400 trials. The average accuracy rate was 82.11%, the average inaccuracy rate was 12.50% and the average rate of missed responses was 4.77%.

ERPs

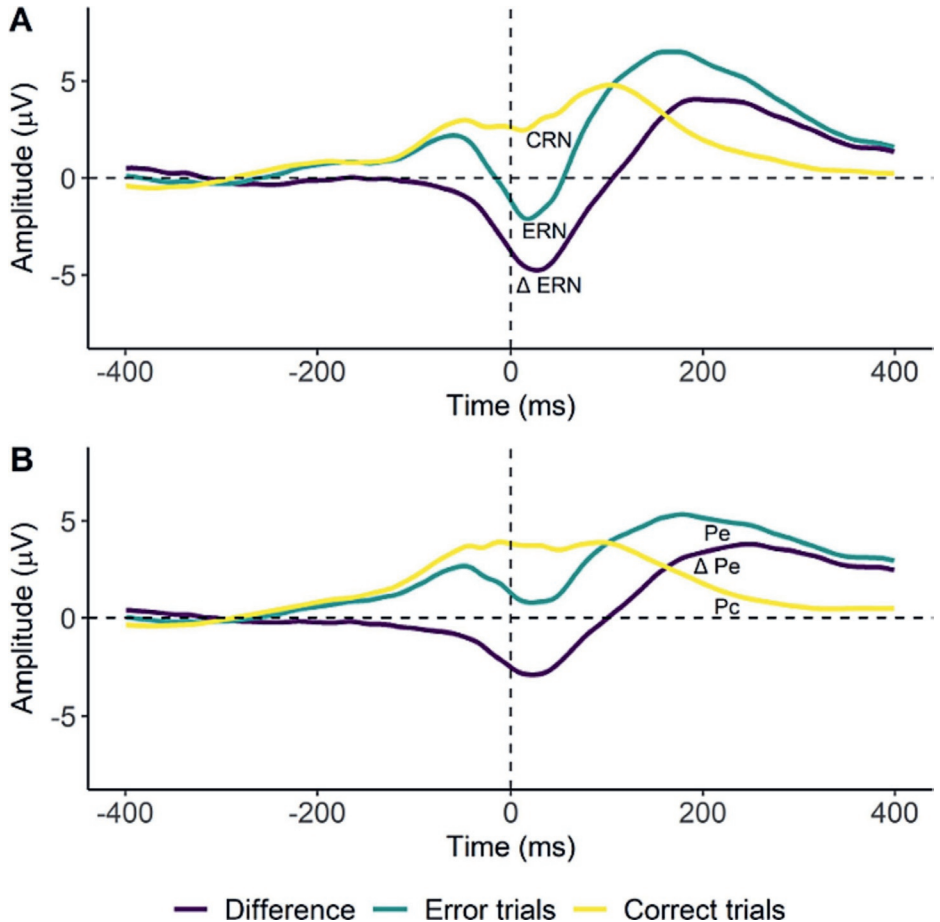
Grand average ERP waveforms at electrode Cz are presented in Figure 3a for the ERN and CRN. Across the sample, the ERN exhibited a mean amplitude of $0.28 \mu\text{V}$ ($SD = 3.69 \mu\text{V}$) while the CRN had a mean amplitude of $3.55 \mu\text{V}$ ($SD = 2.56 \mu\text{V}$). A paired sample *t*-test revealed that the ERN was larger (i.e., more negative) than the CRN within the 0–100 ms window ($t_{187} = 14.33$, 95 % CI $[2.82,$

3.72], $p < .001$, $d = 1.04$).

Grand average ERP waveforms at electrode Cz are presented in Figure 3b for the Pe and Pc. Across the sample, the Pe exhibited a mean amplitude of $4.00\mu\text{v}$ ($SD = 2.57\mu\text{v}$) while the Pc had a mean amplitude of $0.77\mu\text{v}$ ($SD = 1.87\mu\text{v}$). A paired sample t -test revealed that the Pc was larger (i.e., more negative) than the Pe within the 200-400 ms window ($t_{187} = -17.01$, 95% CI [-3.60, -2.85], $p < .001$, $d = 1.24$).

Figure 3.

Response-locked grand average waveforms showing the ERN and Pe.



Note. Figure 3A shows grand average activity for error and correct trials at Cz. Figure 3B shows grand average activity for error and correct trials averaged across Cz and Pz. 0 ms represents response commission. Event-related potentials are labelled, indicating the peak of each wave between 0-100 ms post-onset (Figure 3A) and 200-400 ms post-onset (Fig-

ure 3B). ERN = error-related negativity; CRN = correct-related negativity; Pe = error-related positivity; Pc = correct-related positivity; Difference = grand average amplitude of error trials minus average amplitude correct trials.

Regression Analyses

In line with Jackson et al. (2016), the ERN data was screened for univariate outliers using $\pm 1.5 \times$ interquartile range, leading to the removal of eight cases ($n = 180$). A multiple linear regression was performed to predict ERN amplitude via total IU. The results of the regression indicated that total IU was unable to explain any variance in the ERN, ($F_{1,178} = 3.02, p = .084, R^2 = 0.02$), nor could total IU predict ERN amplitude ($\beta = -0.05, SE = 0.03, t = -1.73, p = .084$). A linear regression was then performed to predict ERN amplitude using P-IU and I-IU as simultaneous predictors. The two predictors were unable to explain any variance in the ERN, $F_{2,177} = 1.85, p = .160, R^2 = 0.02$. Both P-IU ($\beta = -0.08, SE = 0.05, t = -1.59, p = .115$) and I-IU ($\beta = 0.01, SE = 0.08, t = 0.17, p = .867$) were unable to predict ERN amplitude.

A multiple linear regression was performed to predict post-error RT using P-IU and I-IU as simultaneous predictors. The results of the regression indicated that two predictors were unable to explain any variance in post-error RT, $F_{2,177} = 0.32, p = .728, R^2 < 0.01$. Both P-IU ($\beta = -0.29, SE = 0.75, t = -0.39, p = .699$) and I-IU ($\beta = -0.31, SE = 1.06, t = -0.29, p = .772$) were unable to predict post-error RT. Using PES (i.e., mean RT after error minus mean RT after correct) as the dependent variable in the same regression model, the subfactors could not predict any variance in post-error slowing, $F_{2,177} = 2.22, p = .112, R^2 = 0.02$. I-IU was able to predict post-error slowing ($\beta = -1.56, SE = 0.79, t = -1.98, p = .049$) while P-IU was not ($\beta = 0.39, SE = 0.56, t = 0.70, p = .483$). However, this significant effect of I-IU on post-error RT should be interpreted with caution due to the non-significance of the overall model.

Moderation Analyses

Table 3 presents results for regression analyses predicting the Δ ERN. There were no significant results when entering depression and IU into separate regressions to predict the Δ ERN. Likewise, there were also no significant results when depression and IU were entered simultaneously to predict the Δ ERN. Finally, a model including depression, IU and the interaction term (depression $\times$ IU) did not explain any variance in the Δ ERN ($F_{3,184} = 1.33, p = .265, R^2 = 0.02$); the predictors and their interaction were also not statistically significant (see

Table 3 and Figure 4).

There were no significant results when entering trait anxiety and IU into separate regressions to predict the Δ ERN (see Table 3). Likewise, there were also no significant results when trait anxiety and IU were entered simultaneously to predict the Δ ERN. Finally, a model including trait anxiety, IU and the interaction term (trait anxiety $\times$ IU) did not explain any variance in the Δ ERN ($F_{3,184} = 1.03$, $p = .380$, $R^2 = .02$); the predictors and their interaction were also not statistically significant (see Table 3 and Figure 5).

There were no significant results when entering worry and IU into separate regressions to predict the Δ ERN (see Table 3). Likewise, there were also no significant results when worry and IU were entered simultaneously to predict the Δ ERN. Finally, a model including worry, IU and the interaction term (worry $\times$ IU) did not explain any variance in the Δ ERN ($F_{3,184} = 1.40$, $p = .244$, $R^2 = .02$); the predictors and their interaction were also not statistically significant (see Table 3).

Table 3

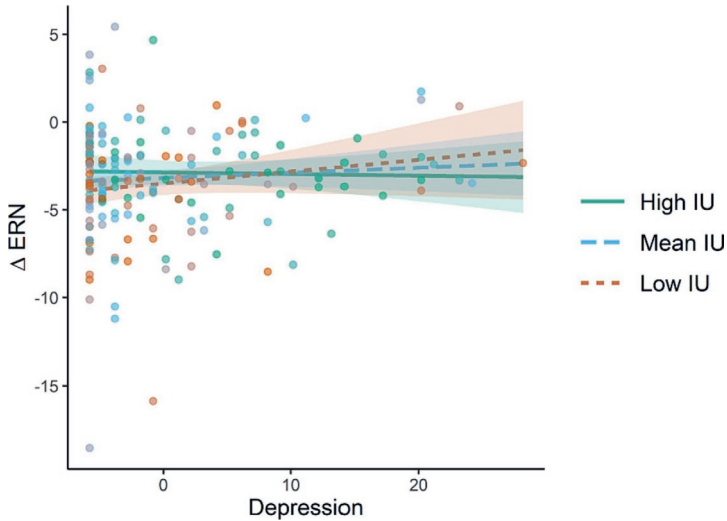
Standardized beta coefficients (β) for regression analyses predicting the Δ ERN

	Model 1	Model 2	Model 3
Predictors (Depression)			
Depression	0.03	0.02	0.03
Total IU	0.04	0.03	0.04
Depression $\times$ IU	-	-	< -0.01
Predictors (Trait Anxiety)			
Trait Anxiety	0.04	0.03	0.02
Total IU	0.04	0.03	0.03
Trait Anxiety $\times$ IU	-	-	< -0.01
Predictors (Worry)			
Worry	-0.01	-0.03	-0.03
Total IU	0.04	0.06	0.06
Worry $\times$ IU	-	-	< -0.01

Note. $N = 188$. Model 1 = separate predictors; model 2 = simultaneous predictors; model 3 = simultaneous predictors plus interaction term. No coefficients were significant at $p < .05$. These null results still held when using the absolute ERN amplitude as an outcome variable (see Supplemental Material).

Figure 4.

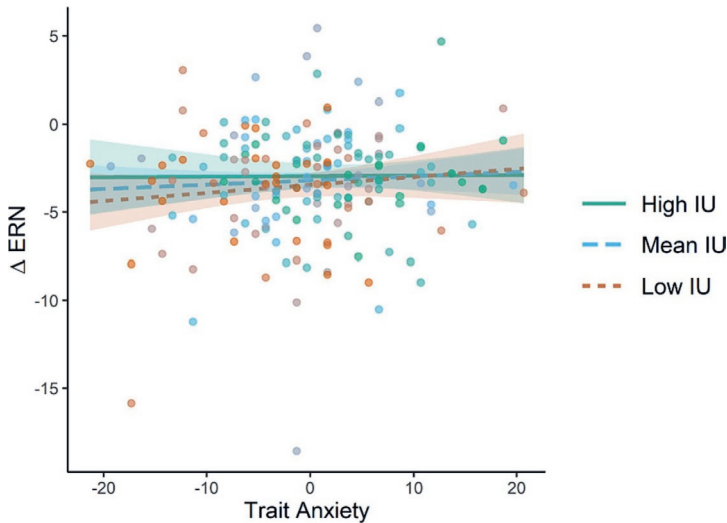
Relationship between depression and the Δ ERN at different levels of IU.



Note. The relationship between depression and Δ ERN at low (red line; -1 SD), medium (blue line; mean) and high (green line; +1 SD) levels of IU. There were no main effects nor an interaction between any variables in predicting the outcome.

Figure 5.

Relationship between trait anxiety and the Δ ERN at different levels of IU.



Note. The relationship between trait anxiety and Δ ERN at low (red line; -1 SD), medium (blue line; mean) and high (green line; +1 SD) levels of IU. There were no main effects nor an interaction between any variables in predicting the outcome.

Exploratory regression analyses

Several simple and multiple regressions were run using total IU and the IU subfactors as predictors (see Table 4). Total IU explained 5% of the variance in correct-response negativity, $F_{1,186} = -10.25, p = .002, R^2 = 0.05$. Neither total IU or the IU subfactors could predict any variance in the Δ ERN, Pe, Pc or Δ Pe.

Table 4

Standardized beta coefficients (β) for exploratory regression analyses

Predictors	Outcome				
	CRN	Δ ERN	Pe	Pc	Δ Pe
Total IU	-0.07**	0.04	-0.03	-0.01	-0.03
Simultaneous predictors					
P-IU	-0.08	-0.03	-0.03	0.02	-0.04
I-IU	-0.06	0.15	-0.05	-0.05	< -0.01

Note. $N = 188$. P-IU = Prospective intolerance of uncertainty; I-IU = Inhibitory intolerance of uncertainty.

** indicates $p < .01$

Discussion

Previous research has been inconclusive regarding the relationships between neural indices of performance monitoring, intolerance of uncertainty (including IU subfactors) and self-reported symptoms of anxiety and depression in non-clinical samples (Jackson et al., 2016; Ruchensky et al., 2020; Sandre and Weinberg, 2019; Saunders and Inzlicht, 2020). As such, we used a large sample with high statistical power to further clarify these relationships. As expected, total IU was not associated with the error-related negativity in our sample. However, contrary to our expectations, the IU subfactors did not predict the ERN or post-error slowing (PES), nor did IU and depression interact to predict the ERN. Exploratory analyses also showed that IU did not moderate the relationship between trait anxiety and the ERN; further, IU was not associated with the error positivity (Pe). Overall, our sample shows no evidence for the relationship between intolerance of uncertainty and the registration of errors at the implicit or conscious level (i.e., ERN and Pe, respectively; Hewig et al., 2011). Nevertheless, our findings should not be misinterpreted as conclusive proof of “no effect”, but rather considered as a contribution to a small existing literature. Further high-powered replications

as well as alternative statistical approaches (e.g., equivalence testing, Bayesian approaches; Harms and Lakens, 2018) are needed to determine whether the effect of IU on the ERN is meaningful. Moreover, the precise relationship between IU and general performance monitoring may be more nuanced than we have accounted for presently.

Our observation that total IU and the ERN were not associated is in line with Jackson et al. (2016) and Ruchensky et al. (2020). Regarding the additional explanatory value of the IU subfactors compared to the total measure, Jackson et al. (2016) had previously found that the IU subfactors were able to predict the ERN in different directions (i.e., enhanced ERN for P-IU; attenuated ERN for I-IU). However, we were not able to replicate this; our null finding instead aligns with two other studies showing no association between the IU subfactors and the error-related negativity (Ruchensky et al., 2020; Sandre and Weinberg, 2019). A priori power calculations recommended a sample of $N = 90$, meaning both Ruchensky et al. (2020; $N = 100$) and the present study ($N = 188$) were adequately powered to detect this association, should it exist. Previous studies with comparatively smaller samples may have run the risk of having spurious findings due to insufficient statistical power (i.e., “false positive”; Button et al., 2013). While further replications are required, the lack of effect of IU subfactors on ERN amplitude suggests that studies previously reporting these associations should be interpreted with caution.

Regarding post-error slowing (PES), we did not replicate the observations of an effect of IU subfactors on post-error RT (Jackson et al., 2016). In their original paper this analysis is referred to as PES, however, this approach does not accurately index PES because it considers post-accuracy RTs (i.e., error and correct responses) separately, rather than a comparison of both in one index (i.e., how much is the next correct trial delayed by a previous mistake compared to the response after a correct trial). Accordingly, two approaches have been defined for calculating PES: (a) PES defined as the difference between average RT after error trials and the average RT after correct trials, and, (b) PES as the mean of the differences from the RT of the correct trial after an error minus the RT of the correct trial before that error (Dutilh et al., 2012; Schroder et al., 2020). In line with Ruchensky et al. (2020), using the former so-called traditional PES, we also obtained a null result, whereby total IU could not predict PES in our sample (Ruchensky et al., 2020). In sum, we did not find evidence for an association of IU and behavioral measures. It is important that studies investigating behavioral adjustments after error commission consider improved estimates of PES to ensure

reliability and internal consistency as recommended by Schroder et al. (2020) and Sandre et al. (2020).

Our study also found no relationship between error processing and symptoms of depression or anxiety. We found no main effect of depression on the ERN which is in line with other research showing no relation (Moran et al., 2017; Ruchensky et al., 2020). We also failed to find an association between trait anxiety and the ERN. Previous studies have advanced the ERN as a potential biomarker for anxiety (Meyer, 2016), but the meta-analysis of Saunders and Inzlicht (2020) found that publication bias may have exaggerated the relationship between trait anxiety and the ERN, with no overall correlation for volunteer non-clinical samples. Our null results from a sample of undergraduates support this notion and relate to other work that has shown that transdiagnostic anxiety-depression symptom dimensions are not related to ERN magnitude (Seow et al., 2020). While meta-analytic work has shown a small effect for increased ERN amplitude in internalizing disorders (Pasion and Barbosa, 2019), the effects of attenuated ERNs in externalizing disorders are larger overall (Lutz et al., 2021; Pasion and Barbosa, 2019). As such, investigations into error processing in externalizing disorders and its relationship to intolerance of uncertainty deserve attention, particularly given findings showing that the desire to remove uncertainty is implicated in impulsive risky behaviors, such as substance abuse (Sadeh and Bredemeier, 2021).

Based on our lack of direct effects, it was conceivable that the broad measure of total IU might moderate the relationship between depression and the ERN, such that depression and the ERN are only related at low levels of IU (Ruchensky et al., 2020). However, this was not the case for our sample. Moreover, our exploratory analysis showed no interaction between trait anxiety and total IU in explaining the ERN. The fact that total IU or IU subfactors were not associated with symptoms or the ERN/Pe in our models was surprising given that errors have been conceptualized as inherently uncertain threats (Weinberg et al., 2016). Despite our null results, approaching errors as endogenous forms of threat does remain valid, particularly considering the neural and behavioral commonalities shared by errors and exogenous threats (e.g., heightened skin conductance response and pupil dilation, increased amygdala activity; Hajcak and Foti, 2008; Hajcak et al., 2003; Pourtois et al., 2010). The idea that the IU subfactors should differentially predict ERN amplitudes is elegant because these subfactors capture different ways people respond to unpredictability, either by engaging cognitive control (P-IU) or by inhibition of action (I-IU). However, these constructs overlapped somewhat in our sample ($r = 0.61$) which raises questions about their

usefulness relative to the total IUS-12 score.

Questions about the validity of the IUS-12 subfactors appear in the extant literature, with support being found for bifactor models with a high loading on a general IU factor (Bottesi et al., 2019; Hale et al., 2016; Lauriola et al., 2016; Shihata et al., 2018; Yao et al., 2021). However other groups have shown discriminant validity for P-IU and I-IU relating to psychopathology (Boelen et al., 2015; McEvoy and Mahoney, 2011; Penney et al., 2020; Saulnier et al., 2019). While considering the IU subfactors as separate constructs may be useful for distinguishing psychopathology (Penney et al., 2020; Yao et al., 2021), research designs employing additional neural, behavioral and psychophysiological approaches become complex. In particular, associations are harder to find when measurement levels exist at different levels of abstraction (Brandt and Mueller, 2022), such that trait personality variables (e.g., IU) can exist at highly abstracted levels of feeling and behavior and therefore may struggle to predict specific contextualized neurobiological variables (e.g., ERN). Moreover, adjacent research from our lab involving impulsivity has shown that self-report and EEG measurement levels can have within- but not between-level correlations (Bernoster et al., 2019). Consequently, more research is needed before linking IU to ERP measures of neurocognitive processes. Studies that validate the assessment of trait IU via self-report measures as well as other units of analysis should be prioritized, especially with regard to accumulating evidence that the IU subfactors do not link well with the ERN.

Any critique of the IU subfactors is tempered by the fact that total IU was also not a good predictor in the current study: Total IU could not predict the ERN, Pe or Pc. The only significant regression result in this study was the finding that total IU (but not subfactors) predicted a larger (i.e., more negative) CRN. This effect was independent of the relationship of total IU and trait anxiety, showing the unique contribution of IU to elevated CRN. This finding is somewhat consistent with a previous study showing that prospective and inhibitory IU were associated with a larger CRN (Sandre and Weinberg, 2019), although they did not report on total IU. High IU constitutes a risk and maintaining factor for OCD (Tolin et al., 2003), and IU has been shown to predict unique variance in OCD symptoms above other cognitive processes such as perfectionism (Fergus and Wu, 2010). It has been posited that a desire to reduce uncertainty may underlie the compulsions in the disorder (particularly repetitive checking behavior; Tolin et al., 2003). Our finding supports the notion of Sandre and Weinberg (2019) that enhanced CRNs may reflect the salience of correctness, and may explain

why hypervigilant checking behavior persists despite a registration of having performed correctly. Such associations between IU and the CRN reflect the need for more research and replications in clinical populations, perhaps integrating disorder-specific forms of IU that have been distinguished from dispositional IU (Shihata et al., 2017). Task-elicited uncertainty may also prove relevant given that some conceptualizations of the CRN regard it as sensitive to uncertainty of responses in general, rather than merely indexing commission of correct ones (Pailing and Segalowitz, 2004). This suggests the CRN could be relevant to performance monitoring under conditions of uncertainty. Along this line Sandre and Weinberg (2019) found relationships between I-IU and flanker trials where correctness was ambiguous. Such adaptations to the flanker task and other paradigms may help understand if the relationship between performance monitoring and IU depends on the task, with the possibility for divergent relations across different forms of psychopathology. As noted by Sandre and Weinberg (2019), it is possible that IU is more related to the recruitment of cognitive control under uncertain conditions, rather than performance monitoring per se.

While adaptations to the flanker task may prove fruitful in clarifying relationships, we do rule out the role of paradigm considerations in explaining our null results. In line with others, we assume that errors can be considered as endogenous and unpredictable forms of threat (Proudfit et al., 2013; Weinberg et al., 2016). However, errors committed during a flanker task may only elicit a relatively mild level of threat compared to other paradigms employing external threats. Individual differences in threat responding (e.g., fear-potentiated startles, skin conductance responses) have been observed in fear-conditioning tasks employing various stressors (e.g., electrical shocks, bursts of white noise/air; see Lonsdorf et al., 2017). An attenuated level of threat/uncertainty generated by errors compared to external threats may explain our null results; however, there are also good reasons for assuming that even the mild uncertainty evoked by errors can moderate responses in high dispositional IU. This has been argued by and shown in the earlier studies using the flanker paradigm (Jackson et al., 2016; Ruchensky et al., 2020), underlying that IU/uncertainty distress need not necessarily be evoked or manipulated directly at the task-level.

Our null results were against expectations meaning several general explanations must be considered. Our sample was adequately powered to find a small-to-medium effect if one existed, indicating that sample size was not an issue; indeed, this study had almost triple the sample size of Jackson et al. (2016) and Sandre and Weinberg (2019) whom we sought to replicate. Nevertheless, our

power was based on effect sizes from published results which may have been exaggerated. The self-report measures employed were reliable, with internal consistency estimates ranging from good to excellent for all measures; the distribution of IUS-12 scores reflected other undergraduate and community samples (Carleton et al., 2012; see Supplemental material). Moreover, our data reflected the typical behavioral and electrophysiological patterns expected for a flanker task. The use of the ΔERN (i.e., ERN minus CRN) has been criticized because subtraction-based difference scores remain highly correlated with their constituent signals, meaning the ΔERN is not independent of the CRN (Meyer et al., 2017). As such, the use of a difference score may not isolate neurophysiological activity specific to error processing and can also lower between-subject variance. Based on this, our analyses were additionally performed using the absolute ERN/Pe (instead of $\Delta\text{ERN}/\Delta\text{Pe}$) as a predictor; all effects were all non-significant, suggesting that the use of a difference score did not bear on the null results observed in this study. This approach of comparing different methods of calculating the results is aligned with the objective of the experimental replication (Hudson, 2021), by testing the variables in different but equivalent ways we can ensure that the effects found are there (or not, as in our case), and do not depend on the specific experimenter choice either in design or analysis steps. Finally, our data fulfilled the assumptions for the various statistical tests which points to the robustness of our null results.

Limitations

A couple of limitations of our study need to be mentioned. First, the whole data collection took place during the COVID-19 pandemic, which in itself constitutes a situation of permanent uncertainty. Especially when investigating anxiety-related constructs, we need to acknowledge that the lab situation before and during the pandemic cannot be compared. This may be a general issue for clinical research. Second, we used a slightly different experimental design (e.g., letters instead of arrows and faster stimuli presentation) and EEG analysis approach (e.g., EEG signal re-referenced to average instead of mastoids and ERN calculated with single electrode instead of pool of electrodes) compared to the original studies, which however is supported both by previous research using the same experimental design of a flanker task to assess performance monitoring (e.g., Franken et al., 2010) and methodological research testing different analytical pipelines of the flanker task (Sandre et al., 2020). Third, due to the diversity in our sample, we had participants answering the questionnaires in their native

language (Dutch, $n = 103$), while international participants received the questionnaires in English, which for most of them was their second language ($n = 85$). However, internal consistency in our sample was good for both versions (see Supplemental material). Fourth, to keep our variables comparable with our reference papers, we did not use a more robust index of the PES. Finally, the BDI-II scores were skewed towards the lower end of the scale (i.e., less severe symptoms) compared to our replication reference (Ruchensky et al., 2020; see Questionnaires). Contrary to our sample, the means and variance of Ruchensky et al. (2020) are comparable to the norms of undergraduates in both the USA (Whisman and Richardson, 2015) and the Netherlands (Roelofs et al., 2013). Thus, our relative lack of depression symptoms could have obscured the relationships between depression and other study variables.

Implications and conclusions

Altogether, our study shows the need for high-powered replication studies in clinical and individual differences (EEG) research, which is currently underway in different EEG Many Labs replication attempts (Pavlov et al., 2021). Our high-powered replication failure of linking intolerance of uncertainty to an electrophysiological index of error-monitoring underlines the need for further replicability and reproducibility within psychophysiology. Attempts should be made to replicate studies with small samples. Recommendations for transparent and open-science practices in psychophysiology have been discussed extensively elsewhere (Garrett-Ruffin et al., 2021) and will contribute to the viability of replication attempts while making sure findings are disseminated regardless of statistical significance. In a field that is particularly time- and resource- intensive, efforts are better directed at theory-testing related to uncertainty as opposed to discovery-oriented approaches which run the risk of Type 1 errors and non-replicability (Brandt and Mueller, 2022). While the ERN and IU have transdiagnostic potential to cut across multiple forms of psychopathology, further rigorous and incremental work is needed to see if, and how, they are related.

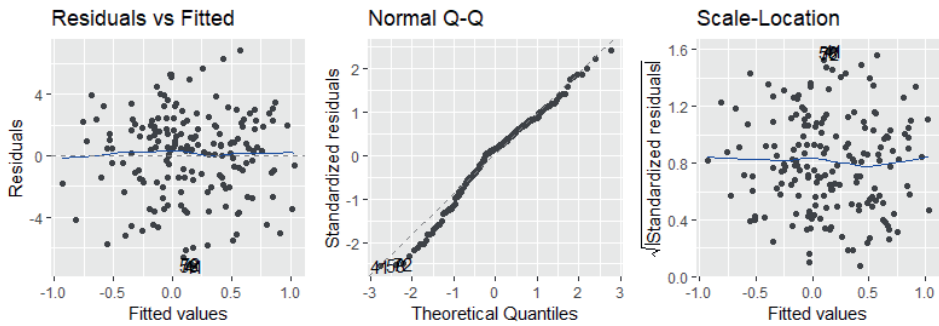
Supplementary Materials

Supplement A: Results of assumption testing plots

As per Field et al. (2013), the assumptions for multiple linear regression were tested, including linearity (Residuals vs Fitted in Figure A1-A4), normality (Normal Q-Q in Figure A1-A4) and homoscedasticity (Scale-Location in Figure A1-A4). No violations were detected for any regression analyses (Hypothesis 1, 2, 3) or the moderation analysis (Hypothesis 4). Multicollinearity was tested based on the presence of correlations above $r > .80$ (see Results Table 3), and was also not an issue.

Figure A1.

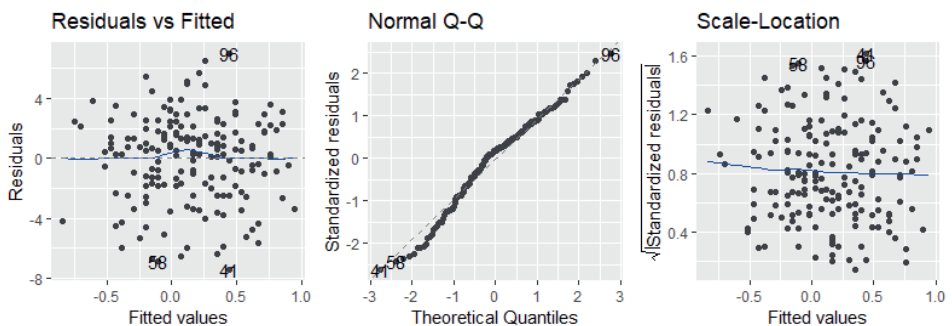
Assumption testing plots for relationship between IU subtypes and ERN amplitude (H1).



Note. No assumptions violated.

Figure A2.

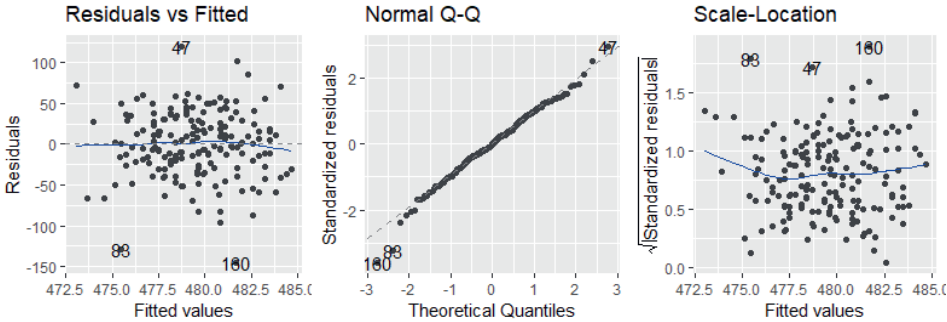
Assumption testing plots for relationship between total IU and ERN amplitude (H2).



Note. No violations.

Figure A3.

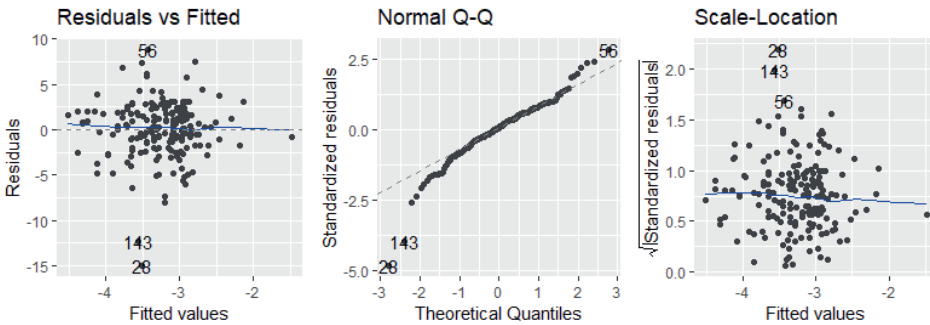
Assumption testing plots for relationship between Total IU and Post-Error slowing (H3).



Note. No violations.

Figure A4.

Assumption testing plots for depression predicting Δ ERN as moderated by IU (H4).



Note. No violations observed.

Supplement B: Internal consistency results for questionnaires by language

Table B1

Internal consistency of questionnaires by language

Questionnaire	Dutch α	English α	Total α
IUS-12	.79	.91	.87
P-IU	.73	.87	.81
I-IU	.69	.83	.77
BDI-II	.94	.94	.79
STAI-T	.74	.84	.79
PSWQ	.84	.81	.83

Note. Total $N = 188$. Dutch $n = 103$. English $n = 85$. α = Cronbach's alpha. IUS-12 = Intolerance of Uncertainty, Short Form; P-IU = Prospective intolerance of uncertainty; I-IU = Inhibitory intolerance of uncertainty; STAI-T = trait subscale of the State Trait Anxiety Inventory; BDI-II = Beck Depression Inventory, Second Edition; PSWQ = Penn State Worry Questionnaire

Supplement C: Moderation analyses performed with ERN

Table C1

Standardized beta coefficients (β) for regression analyses predicting the ERN

	Model 1	Model 2	Model 3
Predictors (Depression)			
Depression	0.03	0.02	0.03
Total IU	0.04	0.03	0.04
Depression $\times$ IU	-	-	< -0.01
Predictors (Trait Anxiety)			
Trait Anxiety	0.04	0.03	0.02
Total IU	0.04	0.03	0.03
Trait Anxiety $\times$ IU	-	-	< -0.01
Predictors (Worry)			
Worry	-0.04	-0.04	-0.04
Total IU	-0.03	-0.01	-0.01
Worry $\times$ IU	-	-	< -0.01

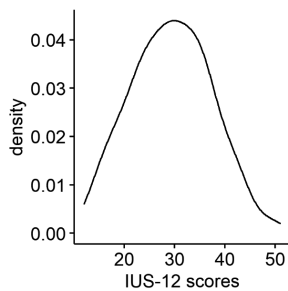
Note. $N = 188$. Model 1 = separate predictors; model 2 = simultaneous predictors; model 3 = simultaneous predictors plus interaction term. No coefficients were significant at $p < .05$.

Supplement D: Normality testing for the Intolerance of Uncertainty Scales

Figure D1 shows that the scores for the IUS-12 in this sample have an approximately normal distribution. This was also verified by conducting a Shapiro-Wilk test for normality ($W = 0.99$, $p = 0.336$); as p is > 0.05 the data is assumed to not significantly differ from the normal distribution.

Figure D1.

Density plot of IUS-12 scores.



Note. The bell-shaped curve shows an approximately normal distribution.

The study by Carleton and colleagues (2012) examined the distributions of the IUS-12 across undergraduate, community and clinical samples (see their Fig. 1 below). Visual inspection reveals that our density plot above resembles distribution of Carleton and colleague's undergraduate and community samples (i.e., roughly in the ranges of 15 to 50). Diagnostic samples naturally score higher on the measure. Given our overlap with these community and clinical samples we therefore argue that the levels/distribution of IU in our sample were sufficient to detect any effects, and thus do not explain our null findings.

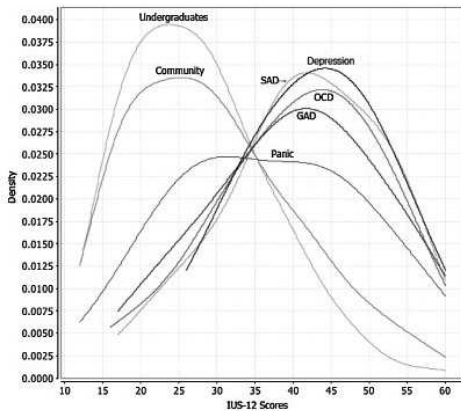


Fig. 1. Kernel density estimation of IUS-12 scores across groups, with density reflecting frequency of cases along IUS-12 scores.

Supplemental Material References

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Chapter 3



Multimodal assessment of the role of intolerance of uncertainty in fear acquisition and extinction

This chapter has been published as:

Malbec, M., Andreatta, M., & Wieser, M. J. (2024). Multimodal assessment of the role of intolerance of uncertainty in fear acquisition and extinction. *Biological Psychology*, 192, 108860. <https://doi.org/10.1016/j.biopsycho.2024.108860>

Abstract

Personality traits linked to internalizing disorders influence the way we develop fears, but also how we regain a sense of safety. In the present study, we investigated the effect of intolerance of uncertainty (IU) on defensive responses using a differential fear conditioning protocol with an extinction phase. The conditioned stimulus was associated with an aversive sound (90 dB) in 75 % of the presentations during acquisition. A final sample of 176 participants completed the experiment. We measured self-reports of associative (expectancy of the unconditioned stimulus in acquisition) and evaluative learning (arousal and valence), and both physiological (skin conductance response) and electrocortical responses (steady-state visually evoked potentials, ssVEPs; late positive potentials, LPP) to the conditioned stimuli. Our results show that IU's impact is limited, with no effect in both acquisition and extinction. These findings emphasize the necessity of large samples in research on inter-individual differences and contribute to our understanding of how IU may or may not be involved in fear and safety learning processes considering multiple aspects of fear responding.

Keywords: Intolerance of uncertainty; EEG; Skin conductance response; Fear acquisition; Fear extinction; Internalizing disorders.

Introduction

Learning to associate sensory cues with threats (aversive learning) is critical for enabling the analysis of the environment for potential threats. The ability to learn these associations and to keep them updated plays a significant role in our survival and general well-being. Alterations in aversive learning are characterized by displaying fear responses to an element that has no threatening features (i.e., overgeneralization) or by consistently showing fear responses towards an element that is no longer associated with threat anymore. This alteration has been linked to the etiology and maintenance of anxiety disorders (Beckers et al., 2023; Carpenter et al., 2019; Craske et al., 2018; Pittig et al., 2018).

Aversive learning has been studied mainly via experimental fear conditioning paradigms (Lonsdorf et al., 2017), in which a threat association is established by presenting a neutral stimulus (CS+; threat stimulus) along with an aversive unconditioned stimulus (US; e.g., an electric shock or a loud noise). In contrast, a safety association is established by presenting another stimulus (CS-; safe stimulus) without an accompanying aversive stimulus. This phase is called fear acquisition. In addition, fear conditioning paradigms can contain an extinction phase, where a former threat stimulus is re-introduced without the accompanying US (Hermans et al., 2006; Sangha et al., 2020). These two phases of Pavlovian conditioning allow researchers to answer two different questions: during the acquisition phase, they investigate ‘why do fears develop?’, while during extinction they explore ‘why do fears persist when no longer relevant?’ (Pittig et al., 2018).

In human fear conditioning studies, conditioned fear responses (CR) are quantified using a range of self-report and physiological measures (Lonsdorf et al., 2017). These measures provide valuable insights into the various facets of fear discrimination and its underlying systems. Self-report measures allow for the assessment of conscious experiences associated with the conditioned stimuli. These measures include ratings of fear, valence, arousal, and US-expectancy. Physiological measures offer an additional level of how fear affects us, and an objective means to bypass potential self-report biases by assessing biological changes in the central and peripheral nervous systems. Some measures include steady-state visually evoked potentials (ssVEPs), which capture attention allocation to a stimulus through the oscillatory response of the visual cortex to an oscillating stimulus (Norcia et al., 2015; Vialatte et al., 2010). This signal has shown a discriminative response towards the CS+ and CS- in fear conditioning experiments (Miskovic & Keil, 2013; Wieser et al., 2014), although

in some studies this discrimination and expected generalization effects are absent (e.g., Aslanidou et al., 2024; Stegmann et al., 2020). The late positive potential (LPP), an event-related potential (ERP), reflects arousal and is larger for pleasant and unpleasant stimuli compared to neutral ones (e.g., Cuthbert et al., 2000). Recently, it has been measured in differential conditioning paradigms and found enlarged LPP amplitudes for CS+ compared to CS- (Bacigalupo & Luck, 2018; Bauer et al., 2020; Bruchmann et al., 2021; Klein et al., 2023; Panitz et al., 2015, 2018). The skin conductance response (SCR) is associated with activity in the autonomic nervous system (Boucsein et al., 2012) and has been used to differentiate threat and safety responses (Dawson & Biferno, 1973; Mordkoff et al., 1967) and it is still commonly employed (for reviews using SCR see Beckers et al., 2023; Lonsdorf et al., 2017; Ojala & Bach, 2020). Combining these diverse measures in fear conditioning studies yields a comprehensive understanding of the different processes involved in fear discrimination. It is worth noting that these measures are often not aligned (Lonsdorf et al., 2017), and studies employing multiple measures provide a more holistic view of the fear system and its responses.

Analysis of the fear responses during the extinction phase can serve as a model for exposure therapy (Craske et al., 2018). A maladaptive reaction in this phase (e.g., persistent fear responses in the absence of threat) is thought to reflect resistance to exposure and has been linked to pathological anxiety (Duits et al., 2015). One possible important factor for this resistance to extinction learning may be uncertainty (Morris, Zuj, & Mertens, 2021; Van den Bergh et al., 2021), since in the extinction process the uncertainty related to threat and safe contingencies may maintain the conditioned response (Bouton, 2002). The degree to which the perception and emotional reaction to uncertainty impacts a person's life has been conceptualized as intolerance of uncertainty (IU). IU refers to the individual inability to endure uncertain or ambiguous situations and events (Carleton, 2012, 2016; Freeston et al., 1994). It is connected to fear processes through the concept of "fear of the unknown" (Carleton, 2016; McEvoy et al., 2019) and threat anticipation (Grupe & Nitschke, 2013). People high in IU interpret uncertain events as aversive and threatening (Dugas et al., 2005). IU is closely related to a wide range of anxiety disorders and depression (Carleton, 2012), forming a transdiagnostic construct (Gentes & Ruscio, 2011; McEvoy & Mahoney, 2011; Shapiro et al., 2020). In the same vein, IU is considered a risk and a maintaining factor for internalizing disorders (McEvoy et al., 2019). Two sub-factors of IU have been proposed: Prospective IU (P-IU) pertains to the in-

clination for predictability and actively seeking certainty, whereas inhibitory IU (I-IU) refers to avoidance, behavioral inhibition, and feeling paralyzed in uncertain situations (Carleton, 2012). Thus, intolerance of uncertainty seems to be a candidate trait for a potential risk factor in maladaptive extinction learning and consequently, vulnerability for the maintenance of anxiety (Morriss, Zuj, & Mertens, 2021; Morriss et al., 2023).

The acquisition phase is thought to model the development of anxiety disorders (Mineka & Zinbarg, 2006; Pittig et al., 2018). Indeed, some evidence has shown that individuals with heightened and even clinical anxiety show stronger fear acquisition, however this evidence is inconsistent and limited to single cue acquisition paradigms (Duits et al., 2015; Lissek et al., 2005; Pittig et al., 2018). However, it has been argued that individual differences in fear acquisition may depend on the US's intensity or aversiveness. This intensity on one hand is required to be strong for a successful fear acquisition, while on the other hand a too strong intensity is thought to be responsible of blurring inter-individual differences in the participants' responses (Beckers et al., 2013, 2023; Lissek et al., 2006). Therefore, when researchers are interested in how different people develop fear, they must find ways of adjusting from a strong to a weakened intensity (Lissek et al., 2006). One way of weakening this situation is by reducing the reinforcement rate of the CS-US association, which then will increase uncertainty. This amplifies the possible impact that an individual's level of IU can have in this phase, as previous research suggests that IU is more prominently associated with compromised fear responses in situations characterized by heightened uncertainty (Lonsdorf & Merz, 2017; Morriss, Zuj, & Mertens, 2021). For instance, using a 37.5% reinforcement rate, Kanen et al. (2021) found a worse SCR threat-safety discrimination for individuals with higher IU. In contrast, Chin et al. (2016) found that the IU level was positively related with the differential (CS+ vs. CS-) FPS response when the reinforcement rate was 50% and no longer when it was 75%. Surprisingly, with a similar approach Johnson et al. (2023) used a continuous (100%) and partial (50%) reinforcement rate and could not find an association of IU with SCR threat discrimination. However, when looking into IU's subfactors they found that, regardless of the reinforcement rate, higher I-IU was associated with diminished SCR discrimination and the opposite was the case for higher P-IU (i.e., heightened threat discrimination). In addition, a recent study by Mertens et al. (2022), analyzing 120 participants found that IU correlated positively with the fear ratings of the CS+, however this effect was no longer present after controlling for trait-anxiety

and neither when it was tested on the differential responses (CS+ vs. CS-). In addition, this association was not observed in physiological responses (SCR and startle response) nor in expectancy ratings (Mertens et al., 2022). Furthermore, Klingelhöfer-Jens et al. (2022) found a positive correlation of IU (controlled for trait-anxiety) with fear ratings of CS+ (but not with SCR and startle responses). It is important to note that several studies have not found an effect of IU on fear responses during acquisition (Johnson et al., 2023; Mertens & Morriss, 2021; Morriss et al., 2015; Morriss, Christakou, et al., 2016; Morriss, 2019; Morriss, Saldarini, Chapman, et al., 2019; Morriss, Saldarini, & van Reekum, 2019; Morriss et al., 2020; Morriss & van Reekum, 2019; Sjouwerman et al., 2020; Wake et al., 2020, 2021; Wroblewski et al., 2022). Thus, which role IU plays in fear acquisition is not clear. This ambiguity arises from the inconsistent influence of reinforcement rates and the varying presence of effects on ratings or physiological measures (Chin et al., 2016; Kanen et al., 2021). Considering the substantial body of research reporting a lack of a link between IU and these responses in fear acquisition, the effect of IU might be very small and dependent on certain aspects in the experimental design or IU does not exert a significant impact in this phase. This observation aligns with the concept of acquisition as a ‘strong situation’ (Lissek et al., 2006). Interestingly, even after adjustments are made to weaken this situation, the reviewed studies also fail to reveal a substantial role of IU in this phase. In our study we aim to contribute to the clarification of this relationship by analyzing the responses in the acquisition phase and exploring their link to the individual’s level of IU.

The extinction phase of fear conditioning helps us to understand the persistence of fears, by testing the defensive responses that appear when the feared stimulus is presented without the aversive consequences. In a way, it reflects the individual’s difficulties in trusting that the situation is safer than expected. The absence of US presentations triggers the establishment of a new safe association that competes with the former threatening one (Bouton, 1993; Bouton & King, 1983), progressively updating the responses until they are equivalent for both CS+ and CS-. This can be referred to as a successful extinction. A meta-analysis by Duits et al. (2015) found that anxiety patients in this phase show stronger fear responses towards the CS+ (small to medium effect size), and persistent discrimination of CS+ and CS- (small effect size), while there were no differences between groups regarding the responses to the CS-. It is relevant to mention that a more recent study (Pöhlchen et al., 2020) could not find these differences in the fear responses in extinction, while they further differentiated in their clinical sample

between post-traumatic stress disorder and other anxiety disorders.

During extinction, the absence of US can be either instructed or uninstructed, the latter meaning that without being explicitly mentioned, the CS+ is no longer followed by the US and hence no longer threatening (Lonsdorf et al., 2017). Thus, in uninstructed extinction a period where the uncertainty about the CS-noUS link increases is created. This increase in uncertainty would affect individuals with higher IU particularly at the beginning of the extinction phase (Morriss, Zuj, & Mertens, 2021). Morriss, Christakou, et al. (2016) analyzed the time course of fear responses during extinction learning and found that lower IU showed a differentiation of CS+ and CS- in the SCR during early extinction which in late extinction disappeared (signaling successful extinction). Conversely, higher IU manifested heightened SCR values for both stimuli in early extinction, suggesting a generalization effect from the threat to the safe stimulus in this uncertain stage. Moreover, in late extinction, higher IU participants exhibited larger SCR for CS+ than CS-, signifying a resistance to extinction. Similarly, another study found also an unsuccessful extinction for participants with high IU and trait anxiety (Wroblewski et al., 2022). It is relevant to note that Wroblewski et al. (2022) used a delayed extinction design, conducting the extinction phase 24 hours after the initial acquisition period. In their study, all participants exhibited higher fear responses (expectancy, arousal, and valence ratings) to the CS- at early extinction compared to the end of acquisition. This pattern resembles the effect found by Morriss, Christakou, et al. (2016) on SCR in early extinction, but there it was limited to high IU individuals. Further, after the initial rise in responses to CS- during early extinction, the low IU/trait anxiety group decreased responses to both stimuli, achieving successful extinction at the end of the extinction phase. In contrast, the high IU/trait anxiety group continued showing a robust differentiation for CS+ vs CS- expressed in the FPS and SCR, throughout extinction. Despite some differences in the previous studies, an effect that has been consistently found is the resistance to extinction in individuals with higher IU (Bauer et al., 2020; Morriss, 2019; Morriss, Christakou, et al., 2016, 2016; Morriss et al., 2015; Morriss, Macdonald, et al., 2016; Morriss, Saldarini, & van Reekum, 2019; Morriss & van Reekum, 2019; Wake et al., 2020, 2021; Wroblewski et al., 2022).

Altogether, it seems that the role of IU on the individuals' responses during acquisition and extinction phases of fear conditioning is nuanced. Regarding the acquisition phase, the results are inconsistent, with studies finding an influence of IU on CS discrimination in opposite directions in FPS and SCR when using relatively low reinforcement rates (Chin et al., 2016; Kanen et al., 2021, respectively),

and another study not finding this effect with IU but with its subfactors (Johnson et al., 2023). In addition, two other studies (Klingelhöfer-Jens et al., 2022; Mertens et al., 2022) found that higher IU was linked to enhanced fear ratings to CS+ and not with its SCR or startle responses, but for one of them this effect disappeared when controlling for trait-anxiety (Mertens et al., 2022). Importantly, most studies have not found effects of IU in this phase (Mertens & Morriss, 2021; Morriss, 2019; Morriss, Christakou, et al., 2016; Morriss et al., 2015, 2020; Morriss, Saldarini, Chapman, et al., 2019; Morriss, Saldarini, & van Reekum, 2019; Morriss & van Reekum, 2019; Sjouwerman et al., 2020; Wake et al., 2020, 2021; Wroblewski et al., 2022). Regarding the extinction phase, individuals with higher IU consistently exhibit resistance to successful extinction, mainly in SCR and fear ratings (Morriss, 2019; Morriss, Christakou, et al., 2016; Morriss et al., 2015; Morriss, Macdonald, et al., 2016; Morriss, Saldarini, & van Reekum, 2019; Morriss & van Reekum, 2019; Wake et al., 2020, 2021; Wroblewski et al., 2022), but a few studies also report non findings (Morriss et al., 2020; Morriss, Rodriguez-Sobstel, et al., 2024; Wendt & Morriss, 2022) .

The current study seeks to further unveil the nuanced interplay between IU and fear responses during both fear acquisition and extinction learning in a relatively large sample, aligning with recent calls for larger sample sizes in the field (Mertens et al., 2022). Fear responses were measured on multiple levels (ratings, SCR, LPP, and ssVEPs), providing a detailed understanding of how IU impacts physiological and subjective fear responses. Recognizing the dynamic nature of fear learning, each phase is divided into early and late sections, allowing a detailed exploration of the evolution of fear responses, and shedding light on critical moments characterized by heightened uncertainty (i.e., early uninstructed extinction). We expected a successful fear acquisition, characterized by higher ratings (valence, arousal, and US-expectancy) and greater amplitudes of SCR, LPP, and ssVEPs in response to the threat compared to the safety cue. Successful fear extinction should be manifested through a decrease in the discrimination of CS+ and CS- in respective variables.

We investigated the impact that IU has on fear responses and the specificity of this influence (Morriss, 2023) while controlling for the influence of other traits such as trait anxiety, worry, and depression. We expected to replicate previous results which found elevated intolerance of uncertainty to hinder extinction learning (e.g., Morriss, 2019; Morriss, Christakou, et al., 2016; Morriss, Saldarini, & van Reekum, 2019), evident through heightened fear responses to both safe and threat stimuli during early extinction (especially in SCR, Morriss,

Wake, et al., 2021; and in LPP, Bauer et al., 2020; and exploratorily in the ssVEP and ratings). Moreover, we expected that threat-safety discrimination persists in all measured variables during the late extinction phase for individuals with high intolerance of uncertainty. Given aforementioned inconsistent findings about the influence of IU on fear acquisition, we had no directed hypothesis a-priori here, but rather exploratorily tested possible effects in all dependent variables as well.

Methods

Participants

In total, 213 psychology undergraduate students from Erasmus University Rotterdam were invited via an online recruitment system to participate in the lab session. Thirty-seven participants had to be removed (13 had missing data in at least one dependent variable; and twenty-four stopped the experiment), resulting in 176 participants for the analyses. The age range was 17-46 years ($M = 21.3$, $SD = 3.30$), 81% were females, and 53% were Dutch. A breakdown of the ethnic self-identification was 71% for White/Caucasian, 10% for Asian, 5% for Middle Eastern, 4% for Black, 4% for mixed, 2% for Latin-American, and 3% for other. Participants received course credit and a monetary compensation (€5) related to a decision-making task performed in the same session, but not reported in this article (see Procedure). A-priori exclusion criteria were family history of photic epilepsy, related to the flickering images in this experiment. Informed consent was obtained before participation, and the research protocol was approved by the ethical board of the Erasmus School of Behavioural and Social Sciences in accordance with the declaration of Helsinki. The lab sessions took place between September 2020 and July 2021.

Questionnaires

After finishing the experimental task, participants completed the following questionnaires (either in Dutch or English, for Dutch or international students, respectively):

The *Intolerance of Uncertainty Scale* (Buhr & Dugas, 2002; Freeston et al., 1994; Dutch translation: de Bruin et al., 2006). It is a 27-item questionnaire that assesses the degree to which individuals find uncertain scenarios distressing and undesirable, as well as their reactions to them. The items are rated on a five-point Likert scale ranging from 1 (“not at all characteristic of me”) to 5 (“entirely char-

acteristic of me”). Internal consistency was excellent for the total scale IU ($\alpha = 0.94$). Total IU scores in the current sample ranged from 28 to 110 ($M = 65.47$; $SD = 17.97$). In addition to the total score, two subfactors can be extracted (McEvoy & Mahoney, 2011), Prospective IU (7 items; $\alpha = 0.83$; $M = 18.03$; $SD = 5.35$) and Inhibitory IU (5 items; $\alpha = 0.78$; $M = 11.06$; $SD = 3.69$).

The *Beck Depression Inventory, Second Edition* (BDI-II; Beck et al., 1996; Dutch translation: Van der Does, 2002) is a widely used self-report measure of depressive symptoms, both cognitive and somatic. It consists of 21 items where participants rate items on a four-point Likert scale ranging from 0 to 3 based on perceived severity in the last two weeks. Internal consistency of this scale was excellent ($\alpha = 0.94$). Scores in the current sample ranged from 0 to 34 ($M = 5.64$; $SD = 7.48$).

The *Penn State Worry Questionnaire* (PSWQ; Meyer et al., 1990; Dutch translation: van Rijsoort et al., 1999) is a 16-item self-report questionnaire that measures the trait of worry across the dimensions of excessiveness, generality, and uncontrollability. Each item is rated on a five-point Likert scale from 1 (“not at all typical of me”) to 5 (“very typical of me”). Internal consistency of this scale was good ($\alpha = 0.83$). Scores in the current sample ranged from 25 to 72 ($M = 44.80$; $SD = 10.63$).

The trait subscale of the *State Trait Anxiety Inventory, Form Y* (STAIT; Spielberger, 1983; Dutch translation: Van der Ploeg, 2000) is a self-report questionnaire consisting of 20 items that measure someone’s general propensity for anxiety. Respondents’ rate items based on how they generally feel, using a four-point Likert scale from 1 (“almost never”) to 4 (“almost always”), with higher scores indicating greater anxiety (range: 20 to 80). Internal consistency of this scale was good ($\alpha = 0.80$). Scores in the current sample ranged from 24 to 66 ($M = 44.78$; $SD = 8.20$).

Materials and stimuli

The conditioned stimuli (CS) were two pictures of female faces with a neutral expression from the NimStim Set of Facial Expressions (03F NE C, 10F NE C; Tottenham et al., 2009). The CSs were presented in the center of a 22-inch iiyama HM204DT-A computer screen and their size was 8.6 x 11.6 cm, the participants were seated at 1.60 m from the screen resulting in 3.08 x 4.15° visual angle size. The images were converted to grey scale and luminance and brightness were kept constant. The background was constant throughout the experiment and was grey (R: 133, G: 133, B: 133). Each CSs was presented flickering at a driving fre-

quency of 15 Hz². The US consisted of a female scream taken from the International Digitized Affective Sounds database (Bradley & Lang, 2007) combined with white noise played through the room speakers for 1 s at 90 dB upon termination of the face stimulus (CS+).

Procedure

Upon the arrival at the lab, participants received a verbal description of the experimental procedure and signed the written informed consent. After this, they were seated in an isolated dimly lit room where the EEG and EDA electrodes were attached. Afterwards, they completed the Doors task (not reported here) with a duration of approximately 40 min. Then they had a break (10 min) and after this the Fear conditioning experiment took place (with an approximate duration of 25 min). Lastly, they completed a Flanker task (duration of around 10 min). This task is not part of this manuscript and is reported in Malbec et al. (2022) and *Chapter 2* of this dissertation.

The fear conditioning protocol consisted of three phases: habituation, acquisition, and extinction. During habituation, both CSs were presented 10 times each, while no US was presented. Participants were then instructed to pay attention to the faces presented and that one of them will be followed sometimes by a loud noise. During the acquisition phase, the CS+ was followed by the US in 75% of the times, starting at CS+ offset, while the CS- was never associated with the US. Both CS- and CS+ were presented 20 times each. Finally, an uninstructed extinction phase followed, where CS+ and CS- were presented 20 times each and the US was never delivered. In all phases, the duration of the CSs was 5 s, while the inter-trial-interval (ITI), defined as the time between the offset of one stimulus and the onset of a following stimulus, had a random duration between 4.5 – 5 s, during which a white fixation cross was presented. The order of presentation was fully randomized in habituation and extinction phases, whereas in acquisition phase it was pseudo-randomized meaning that the same stimulus was not

² Due to a misconfiguration in the monitor's refresh rate, 25 participants in the final sample experienced CS flickering at a stable frequency of 12.5 Hz. Consequently, we calculated their ssVEPs using this frequency and compared the results with the remaining sample, which experienced flickering at 15 Hz. However, there were no significant differences observed between the two groups. To verify, we conducted similar ANOVAs as in our main analysis, including flickering frequency as a between-subjects factor. In acquisition, the flickering frequency did not yield any significant interactions with withinsubject factors (all $p > .078$) or as a between-subject factor ($p = .825$). Similarly, in extinction, interactions with within-subject factors were nonsignificant (all $p > .485$), as was the between-subjects factor ($p = .774$). Therefore, we averaged ssVEP amplitudes across frequencies and report statistics for the entire sample.

presented more than twice in a row, and the first trial of the phase was always a CS+ that was linked with the US. The associations of each picture as CS+ or CS- was counterbalanced across participants.

After each phase, participants' arousal and valence for each CS was assessed by using the Self-Assessment Manikin scale (SAM; Bradley and Lang, 1994). The scales ranged from 1 as "calm" or "happy" to 9 "excited" or "unhappy", for arousal and valence, respectively. The scales were presented after the respective CS for 1 s. To explore contingency learning, we measured the US-expectancy using an analogous slider during the acquisition phase (after ten trials) and at its end (after 40 trials). It consisted of the question "How likely is to hear a noise after this image?" and 0% and 100% were displayed in both ends of the slider, the corresponding CS image was presented on top of the slider until response.

Psychophysiological signals recording and processing

Continuous EEG was recorded using Ag/AgCl 32 electrodes arranged according to the 10/20 configuration. The electro-oculogram (EOG) was recorded using 4 electrodes, two above and below the left eye, and two at the outer canthus of each eye. Data were recorded using the Active-Two BioSemi system (BioSemi, Amsterdam, Netherlands). This system includes two additional electrodes, common mode sense (CMS) and driven right leg (DRL), which serve as online reference and ground. The signal was digitized at a 512 Hz sampling rate and 24-bit A/D conversion. All electrode impedances were kept below 20 k Ω . Additionally, EDA was recorded with two auxiliary channels, two 8 mm flat electrodes with a 0.05M NaCl electrolyte gel. These electrodes were attached to the second phalanx of the middle and ring finger of the non-dominant hand after lightly cleaned with water.

EEG and EDA were analyzed offline with the software BrainVision Analyzer 2.0 (BrainProducts Inc., Gilching, Germany). First, EEG data were re-referenced to the average voltage at all scalp electrodes (i.e., average reference), band-pass filtered from 0.1 to 40 Hz (Butterworth zero phase filter order 8), notch-filtered at 50 Hz, and corrected for eye-movement artifacts (Gratton et al., 1983). Data were segmented in epochs of 6000 ms (500 ms before and 5500 ms after CS onset). Artifacts were rejected according to the following criteria: (a) maximal absolute amplitude allowed was 200 μ V, (b) a gradient criterion of ± 50 μ V/ms, (c) a difference criterion of 100 μ V and (d) a low activity criterion of 0.5 μ V (interval length: 100 ms). The remaining trials were averaged according to the experimental conditions. The average amount of kept trials after artifact rejection

for the ssVEP signal (electrodes O1, Oz, and O2) was for habituation: $CS+ = 9.8$ and $CS- = 9.8$; for early acquisition: $CS+ = 9.6$ and $CS- = 9.6$; for late acquisition: $CS+ = 9.7$ and $CS- = 9.6$; and for early extinction, $CS+ = 9.8$ and $CS- = 9.8$; for late extinction, $CS+ = 9.7$ and $CS- = 9.7$. The amount of trials did not significantly differ between conditions (habituation: $t = .00$, $p = 1.00$; acquisition: $F(3, 525) = 2.19$, $p = .089$; extinction: $F(3, 525) = 0.08$, $p = .972$). For the LPP signal (electrode Pz) the mean number of kept trials was for habituation, $CS+ = 10.0$ and $CS- = 9.9$; for early acquisition, $CS+ = 9.8$ and $CS- = 9.8$; for late acquisition, $CS+ = 9.9$ and $CS- = 9.9$; and for early extinction, $CS+ = 9.9$ and $CS- = 9.9$; for late extinction, $CS+ = 9.9$ and $CS- = 9.9$. Again, with no differences between conditions per phase (habituation: $t = 1.19$, $p = .238$; acquisition: $F(3, 525) = 1.35$, $p = .256$; extinction: $F(3, 525) = 0.56$, $p = .641$).

For the ssVEP analysis, the signal was then transformed into the frequency domain using a Fast-Fourier-Transformation for the last 2000 ms of stimulus presentation to eliminate initial non-stationary ssVEP components and to highlight ssVEP power which has shown to be more sensitive to conditioning effects in the second half of CS presentation (Miskovic & Keil, 2013; Moratti et al., 2006; Moratti & Keil, 2005). The ssVEP power was averaged across O1, Oz, and O2 electrodes, where ssVEP amplitudes were maximal (see Figure 3) and in line with previous studies (Aslanidou et al., 2024; Stegmann et al., 2020; Wieser et al., 2014).

The LPP was measured as the mean amplitude at the Pz electrode in the time window between 400 ms to 1000 ms after CS onset (based on Bacigalupo & Luck, 2018; Bauer et al., 2020; Klein et al., 2023). For the LPP analysis we applied a band-rejection filter to diminish the effect of the flickering images at 15 Hz (12.5 Hz for the participants that were exposed to that frequency) with 0.1 Hz bandwidth (4th order). Then, a baseline correction was applied using the 200 ms before CS onset.

Second, to calculate SCR, the EDA was filtered offline with a high cut-off filter of 1 Hz and a notch filter of 50 Hz. The signal was segmented in epochs of -1000 ms to 9000 ms after CS onset. The signal was quantified using a manual Foot-to-Peak method by identifying the foot in a time-window of 800 ms – 4000 ms after stimulus onset and the Peak as the first following response peak (Boucsein et al., 2012). Responses below 0.02 μS were scored as zero.³

For both EEG signals and the SCR, we calculated averages based on the

³ Additionally, we applied a square root transformation and z-transformation to the SCR signal before rerunning the same analyses. Notably, the results closely mirror those obtained using the raw SCR scores; further details can be found in the Supplementary materials.

phases and stimulus type, resulting in one for CS+ and one for CS- in the habituation phase. To investigate the temporality of fear acquisition and extinction, we divided these phases into *early* (first 10 trials per condition) and *late* (last 10 trials per condition) sections (Morris, Christakou, et al., 2016). This results in four averages in acquisition (CS+ early, CS- early, CS+ late, CS- late) and four for extinction (CS+ early, CS- early, CS+ late, CS- late).

Data analysis

The analyses were conducted using IBM SPSS (Version 29.0, IBM, Armonk, NY). For all the analyses, we considered statistical significance at the alpha level of $p = .05$. We report the Cohen's d or partial eta squared as estimations of the effect sizes.

In habituation, we compared the average responses to CS+ and CS- through paired t -tests (two-tailed). This was applied to both ratings and physiological responses.

In the acquisition and extinction phases, we compared the affective ratings of CS+ and CS- after each phase using paired t -tests (two-tailed). To analyze the US-expectancy ratings, we used an ANOVA with stimulus (CS+, CS-) and time (early [after 10 first trials] and late [after 40 trials, end of phase]) as within-subject factors. We ran ANOVAs with stimulus (CS+, CS-) and time (early, late) as within-subject factors for SCR, ssVEPs, and LPP, separately. Significant effects and interactions were followed up with post-hoc paired t -tests (Bonferroni corrected). Further, to investigate the effect of IU on these responses we used ANCOVAs with the same factors and including total IU as a covariate.⁴

To assess the specificity of the influence of IU on conditioned fear responses during fear acquisition and fear extinction, we performed a bi-variate correlational analysis between the IUS and its subscales with the outcome measures of conditioned fear responding (i.e., valence and arousal ratings, expectancy ratings in acquisition, SCR, ssVEPs, and LPPs) for each phase. To control for the influence of the other trait measures (STAI, BDI, and PSWQ), a partial correlational analysis was performed between the IUS and the respective outcome measures. These correlations were tested for acquisition and extinction phases and divided by time (i.e., early and late sections). For each outcome measure, we examined

⁴ Following a suggestion from an anonymous reviewer, we explored the impact of IU scores on SCR by grouping participants, either by extreme high or low IU scores ($M \pm 1 SD$) or by dividing the sample into quartiles. However, neither grouping method revealed an effect of IU on SCR. The full results and additional details are provided in the Supplementary materials.

the association with IUS by looking at average responses to the CS+ only, the CS- only, and the difference score (i.e., CS discrimination), which was calculated by subtracting the CS- response from the CS+ response. The Holm-Bonferroni multiple testing correction method was used to control for the fact that several different outcome measures were included (Holm, 1979). To this end, the p -values from the tests were ordered from lowest to highest. We began by comparing the lowest p -value to a Holm-Bonferroni-adjusted alpha, which is the original alpha level divided by the total number of tests. The next smallest p -value was then compared with an adjusted alpha calculated for one fewer test than the previous step (Holm, 1979). Once a p -value exceeded the adjusted significance level, the procedure was stopped, and all remaining hypotheses were considered non-significant. As reported in Tables 2 and 4, the alpha level was divided by 4 (early acquisition), 6 (late acquisition), 3 (early extinction), and 5 (late extinction), based on the number of measures at each time.

Results

The dataset of the final sample is publicly available at <https://osf.io/vupj5/>

Habituation

The analysis of the ratings and physiological responses to CS+ and CS- after and during the habituation phase showed no significant differences (all p values > .205).

Acquisition

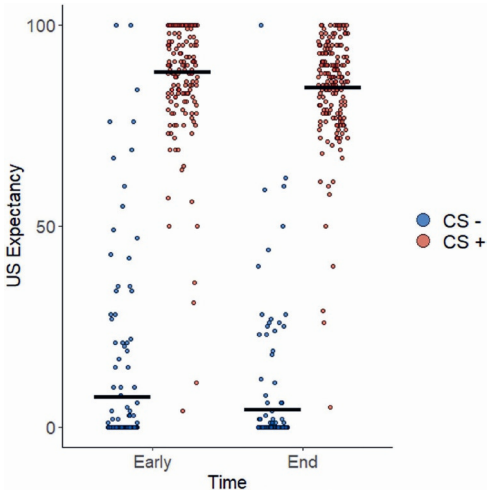
Ratings

In the acquisition phase, there were significant differences in the affective ratings between the two stimuli. For arousal, the t -test revealed higher arousal ratings for CS+ ($M = 6.72$, $SD = 1.70$) than for CS- ($M = 4.38$, $SD = 2.03$), $t(175) = 15.63$, $p < .001$, $d = 1.18$. For valence, the t -test also revealed more negative valence ratings for CS+ ($M = 6.24$, $SD = 1.80$) than for CS- ($M = 4.59$, $SD = 1.64$), $t(175) = 10.30$, $p < .001$, $d = 0.78$.

Regarding US-expectancy ratings, we found a significant main effect of stimulus, $F(1,175) = 2579.07$, $p < .001$, $\eta_p^2 = .936$, and a significant main effect of time, $F(1,175) = 22.50$, $p < .001$, $\eta_p^2 = .114$. However, the interaction between Stimulus and Time was not significant, $F(1,175) = 0.52$, $p = .820$, $\eta_p^2 < .001$ (Figure 1).

Figure 1.

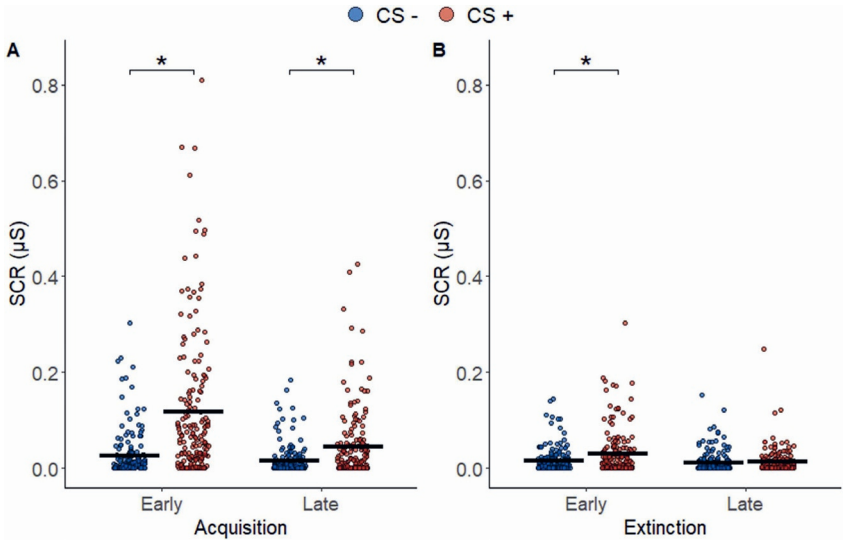
US-Expectancy ratings for CS+ (red dots) and CS- (blue dots) during early (after 10 trials) and end (after 40 trials) of acquisition.



Note. The dots depict individual participants, while the black horizontal lines indicate the group means.

Figure 2.

SCR responses to CS+ (red dots) and CS- (blue dots) during early and late acquisition (A) and during extinction (B) phases.



Note. The dots depict individual participants, while the black horizontal lines indicate the group means. Asterisks represent *post-hoc* significant differences at $p < .025$ threshold.

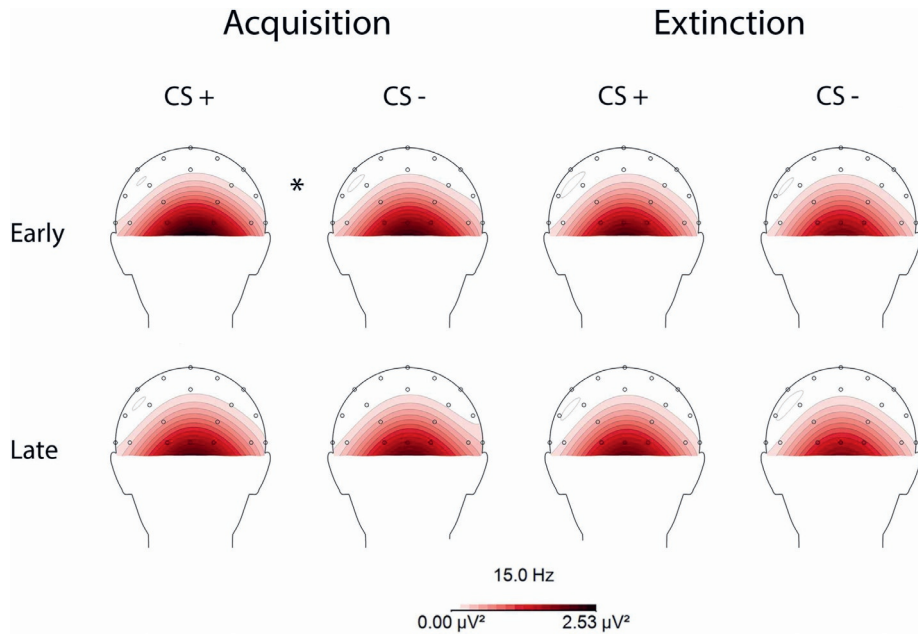
Psychophysiology and EEG

For SCR, the main effect of stimulus type was significant, $F(1,175) = 83.65, p < .001, \eta_p^2 = .323$. There was also a significant main effect of time, $F(1,175) = 74.40, p < .001, \eta_p^2 = .298$. Furthermore, the interaction between Stimulus and Time was significant, $F(1,175) = 49.13, p < .001, \eta_p^2 = .219$. *Post-hoc* pairwise *t*-tests revealed that for early acquisition, the amplitude of CS+ ($M = 0.12 \mu S, SD = 0.15$) was larger than CS- ($M = 0.03 \mu S, SD = 0.05$), $t(175) = 9.10, p < .001, d = 0.13$. During late acquisition, also CS+ ($M = 0.05 \mu S, SD = 0.07$), showed larger SCR than CS- ($M = 0.02 \mu S, SD = 0.03$), $t(175) = 5.82, p < .001, d = 0.07$ (Figure 2 A).

Exploring the ssVEPs, we identified a significant main effect of stimulus, $F(1,175) = 7.32, p = .008, \eta_p^2 = .040$. The effect of time was also significant, $F(1,175) = 8.70, p = .004, \eta_p^2 = .047$. Additionally, the interaction between Time and Stimulus was significant, $F(1,175) = 4.55, p = .034, \eta_p^2 = .025$. *Post-hoc* *t*-tests showed that in early acquisition CS+ evoked a larger amplitude than CS-, $t(175) = 3.31, p = .001, d = 0.25$, while in late acquisition the amplitudes for CS+ and CS- were not different, $t(175) = 1.01, p = .31, d = 0.08$ (Figure 3; Table 1).

Figure 3.

Grand-averaged topographic maps of the ssVEPs (steady-state visually evoked potentials), depicting the power at 15 Hz.



Note. Asterisks represent *post-hoc* significant differences at $p < .025$ threshold, only calculated for acquisition where the interaction Time x Stimulus was significant.

For the LPP, the main effect of stimulus was not significant, $F(1,175) = 0.13, p = .719, \eta_p^2 = .001$. The effect of time was significant, $F(1,175) = 13.17, p < .001, \eta_p^2 = .070$. Additionally, the interaction between Time and Stimulus was significant, $F(1,175) = 14.49, p < .001, \eta_p^2 = .076$. *Post-hoc* t-tests showed that in early acquisition CS+ evoked a lower LPP amplitude than CS-, $t(175) = 2.49, p = .014, d = 0.19$. In late acquisition the LPP amplitude was larger for CS+ than CS-, $t(175) = 2.17, p = .031, d = 0.1$, but this difference does not survive the Bonferroni correction ($p < .025$) (Figure 4 A and B; Table 1).

Table 1.

Mean and standard deviations of LPP and ssVEP amplitudes for CSs during Acquisition.

	Early		Late	
	CS +	CS -	CS +	CS -
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
LPP (μV)	4.02 (5.04)	5.33 (7.96)	3.83 (4.20)	2.81 (5.90)
ssVEP ($\mu V^2/Hz$)	0.94 (1.09)	0.80 (0.93)	0.80 (0.90)	0.76 (0.95)

Note. $N = 176$. LPP = Late positive potential; ssVEP = steady-state visually evoked potential.

Influence of intolerance of uncertainty

To analyze the effect of IU on the ratings we ran ANCOVAs. For valence, the main effect stimulus had a significant role still after accounting for IU, $F(1,174) = 5.80, p = .017, \eta_p^2 = .032$. However, the interaction of Stimulus with IU revealed no influence of IU on the valence ratings after acquisition, $F(1,174) = 0.10, p = .754, \eta_p^2 = .001$. For arousal, stimulus also had a significant role still after accounting for IU, $F(1,174) = 12.75, p < .001, \eta_p^2 = .068$. The role of IU as a covariate also was not significant, $F(1,174) = 0.32, p = .570, \eta_p^2 = .002$. For the expectancy ratings, stimulus had a significant role still after accounting for IU, $F(1,174) = 164.398, p < .001, \eta_p^2 = .486$, while the interaction of Stimulus with IU was not significant, $F(1,174) = 0.34, p = .563, \eta_p^2 = .002$.

The results of the ANCOVAs for SCR revealed no significant effect of IU. The interactions of IU with the factors (stimulus, time, and Stimulus X Time) were all not significant (all p values $> .122$). The only factor that remains significant after accounting for IU is *stimulus* ($F(1,174) = 6.40, p = .012, \eta_p^2 = .035$). For ssVEPs, also the results show no significant role of IU in acquisition. The interaction of IU with the factors as well as the single factors are not significant (all p values $> .126$). The same pattern was found for LPP, neither the interactions with IU nor the individual factors showed significant effects (all p values $> .339$).

We in addition ran bi-variate correlations and partial correlations (BDI, PSWQ, STAI-T) of IU and the subscales (I-IU and P-IU) separately with all dependent variables for CS+ and CS- and the differences scores (CS+ - CS-) for early and late acquisition. The results are presented in Tables 2a -2c. For CS+, there was an association found between total IU and arousal ratings after the acquisition phase ($r = .19, p = .014$). For CS-, total IU and inhibitory IU, when controlled for other traits, were correlated with expectancy ratings in late acquisition ($r = -.18, p = .018$, and $r = -.15, p = .049$, respectively). Additionally, during the same period, there was a link between arousal ratings and prospective IU ($r = .17, p = .024$). When exploring the difference score in this phase, there was an association between total IU (when controlled) and expectancy ratings ($r = .16, p = .034$). However, none of the correlations remain significant after Holm-Bonferroni corrections for multiple testing.

Table 2a.

Correlation coefficients and p-values reported for the relation between intolerance of uncertainty and the threat cue (CS+) during early and late acquisition.

Phase	Early Acquisition					Late Acquisition				
	IU (r)	IU (partial r)	I-IU (r)	I-IU (partial r)	P-IU (r)	P-IU (partial r)	IU (r)	IU (partial r)	I-IU (r)	I-IU (partial r)
Outcome Measures										
Expectancy										
Correlation	.02	.03	.02	.03	.02	.04	.09	.08	.03	.01
p-Value	.744	.682	.763	.742	.772	.644	.249	.297	.685	.892
Arousal ^a										
Correlation	-	-	-	-	-	-	.19	.07	.08	-.06
p-Value	-	-	-	-	-	-	.014	.388	.262	.469
Valence ^a										
Correlation	-	-	-	-	-	-	.11	.07	.02	-.05
p-Value	-	-	-	-	-	-	.153	.389	.801	.491
SCR										
Correlation	.07	.04	.05	.02	.09	.05	-.04	-.03	-.08	-.08
p-Value	.369	.641	.520	.799	.236	.491	.607	.688	.276	.299
ssVEP										
Correlation	-.04	-.06	-.01	-.02	-.07	-.09	-.08	-.09	-.04	-.04
p-Value	.596	.416	.934	.824	.367	.264	.293	.224	.610	.610
LPP										
Correlation	.06	.04	-.02	-.06	.08	.07	.05	-.04	.00	-.10
p-Value	.457	.593	.770	.466	.286	.337	.516	.585	.959	.176

Note. P-values reported are uncorrected. None of the p-values are significant after Bonferroni-Holm corrections ($p = .013$ for early acquisition and $p = .008$ for late acquisition). IU = Total IU; P-IU = Prospective IU; I-IU = Inhibitory IU; r = Pearson correlations; Partial r = Correlations were controlled for STAI-T, BDI-II, and PSWQ using partial correlations.

^aValence and arousal ratings were only obtained one time after the acquisition phase.

Table 2b.

Correlation coefficients and *p*-values reported for the relation between intolerance of uncertainty and the safety cue (CS-) during early and late acquisition.

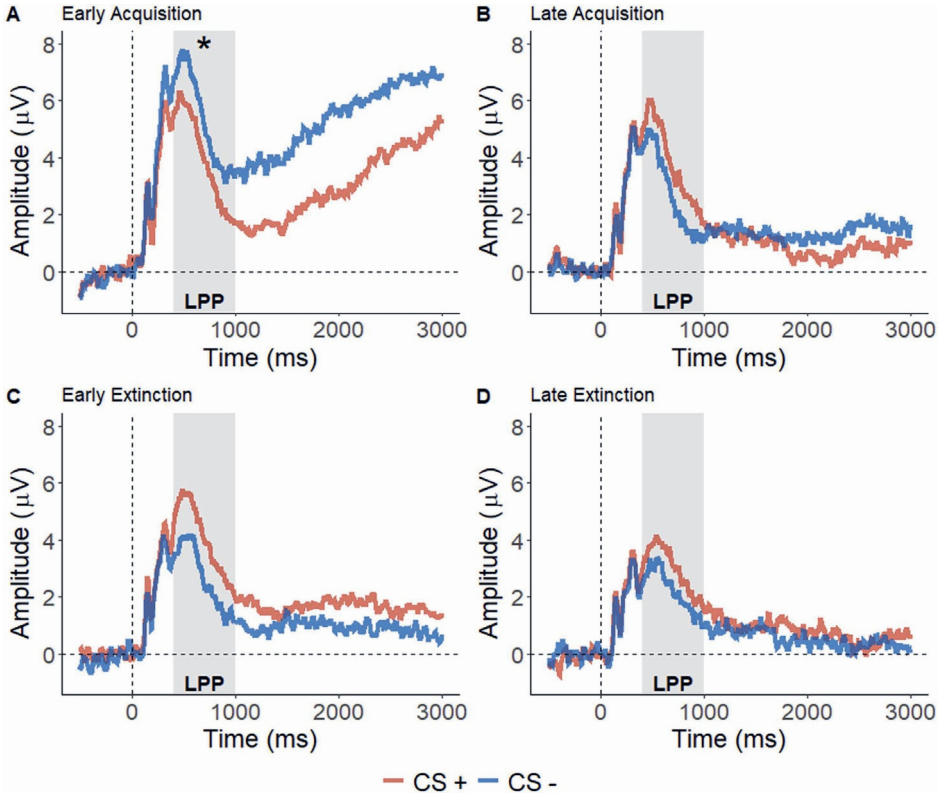
Phase	Early Acquisition				Late Acquisition			
	IU (<i>r</i>)	IU (partial <i>r</i>)	I-IU (<i>r</i>)	I-IU (partial <i>r</i>)	P-IU (<i>r</i>)	P-IU (partial <i>r</i>)	I-IU (<i>r</i>)	P-IU (partial <i>r</i>)
Expectancy								
Correlation	.05	.04	.03	.02	.05	.04	-.09	-.13
<i>p</i> -Value	.545	.633	.669	.780	.491	.627	.246	.091
Arousal ^a								
Correlation	-	-	-	-	-	-	.11	.12
<i>p</i> -Value	-	-	-	-	-	-	.138	.108
Valence ^a								
Correlation	-	-	-	-	-	-	.09	.12
<i>p</i> -Value	-	-	-	-	-	-	.246	.106
SCR								
Correlation	.11	.06	.08	.02	.07	.02	.11	.08
<i>p</i> -Value	.136	.425	.294	.748	.340	.787	.138	.291
ssVEP								
Correlation	-.02	-.05	-.03	-.05	-.05	-.07	-.10	-.14
<i>p</i> -Value	.755	.533	.738	.541	.532	.389	.206	.075
LPP								
Correlation	.10	.07	.00	-.05	.12	.09	.08	.12
<i>p</i> -Value	.182	.367	.999	.486	.107	.219	.320	.103

Note. *P*-values reported are uncorrected. None of the *p*-values are significant after Bonferroni-Holm corrections ($p = .013$ for early acquisition and $p = .008$ for late acquisition. IU = Total IU; P-IU = Prospective IU; I-IU = Inhibitory IU; *r* = Pearson correlations; Partial *r* = Correlations were controlled for STAI-T, BDI-II, and PSWQ using partial correlations.

^a Valence and arousal ratings were only obtained one time after the acquisition phase.

Figure 4.

Grand-averaged LPP waveforms at Pz electrode, time-locked to the onset of the CS.



Note. The asterisks show significant post-hoc differences between stimuli responses (CS + vs. CS -) at $p < .025$, which were only carried out for acquisition since the interaction of CS type and Time was significant. The grey rectangle shows the time-window (400 – 1000 ms) used for calculating the LPP as mean amplitude.

Extinction

Ratings

After the extinction phase, the differences between CS+ and CS- were still present in the affective ratings. A t -test revealed higher arousal ratings for CS+ ($M = 4.53$, $SD = 2.01$) than CS- ($M = 3.72$, $SD = 1.78$), $t(175) = 7.47$, $p < .001$, $d = 0.56$. For valence there were higher (i.e., more negative) ratings for CS+ ($M = 5.15$, $SD = 1.50$) than for CS- ($M = 4.55$, $SD = 1.38$), $t(175) = 5.72$, $p < .001$, $d = 0.43$.

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Regarding the SCR, a main effect of stimulus was significant $F(1,175) = 16.69$, $p < .001$, $\eta_p^2 = .087$. In addition, the main effect of time was significant, $F(1,175)$

= 30.88, $p < .001$, $\eta_p^2 = .150$. The interaction of Time and Stimulus also was significant, $F(1,175) = 12.79$, $p < .001$, $\eta_p^2 = .068$. Post-hoc paired t -tests showed that SCR were larger for the CS+ ($M = 0.03 \mu S$, $SD = 0.05$) than CS- ($M = 0.02 \mu S$, $SD = 0.03$) during early extinction ($t(175) = 4.59$, $p < .001$, $d = .35$), while in late extinction the SCR did not differ between stimuli anymore, $t(175) = 0.65$, $p = .518$, $d = .05$ (CS+: $M = 0.02 \mu S$, $SD = 0.03$; CS-: $M = 0.01 \mu S$, $SD = 0.02$) (Figure 2 B).

Exploring the ssVEPs, the effect stimulus was significant, $F(1,175) = 9.36$, $p = .003$, $\eta_p^2 = .051$, with a larger ssVEP amplitude for CS+ vs CS- in early and late extinction. The effect of time was also significant, $F(1,175) = 6.48$, $p = .012$, $\eta_p^2 = .036$, showing a decrease in the ssVEP from early to late extinction, whereas the interaction between Stimulus and Time was not significant ($p = .843$). (Figure 3; Table 3).

For the LPP, the analysis revealed a significant effect of stimulus, $F(1, 175) = 13.30$, $p < .001$, $\eta_p^2 = .071$, with a larger LPP for CS+ than for CS-. Additionally, there was a significant effect of time, $F(1, 175) = 4.46$, $p = .036$, $\eta_p^2 = .025$, reflecting a decrease in the amplitude of this signal from early to late extinction. However, the interaction between Stimulus and Time was not significant ($p = .274$, see figure 4 C and D; Table 3).

Table 3.

Mean and standard deviations of LPP and ssVEP for CSs during early and late extinction.

	Early		Late	
	CS +	CS -	CS +	CS -
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
LPP (μV)	4.04 (4.60)	2.68 (3.56)	2.98 (4.02)	2.21 (5.03)
ssVEP ($\mu V^2/Hz$)	0.79 (0.91)	0.71 (0.78)	0.74 (0.80)	0.66 (0.81)

Note. $N = 176$. LPP = Late positive potential; ssVEP = steady-state visually evoked potential.

Influence of intolerance of uncertainty

To analyze the effect of IU we used ANCOVAs. There was no influence of IU neither on valence ($F(1,174) = 0.02$, $p = .899$, $\eta_p^2 < .001$) nor on arousal ratings after the extinction phase, $F(1,174) = 0.31$, $p = .581$, $\eta_p^2 = .002$. None of the effects and their interactions (all $ps > .225$) or the interactions with IU scores (all $ps > .556$) were significant for SCR. The same was found for ssVEPs, with all the main effects $ps > .132$ and their interactions with IU $ps > .106$. Also, for the LPP, no main factor (all $ps > .070$) nor interactions with IU (all $ps > .191$) were significant. Overall, this analysis shows that IU did not influence the physiological responses

to CS+ and CS- during extinction.

Like for acquisition, we also ran bi-variate correlations of total IU and subscales I-IU and P-IU with all dependent variables separately for early and late extinction. The results are presented in Tables 4a-4c. Regarding CS+, there was an association found between prospective IU (when controlled) and ssVEP during late extinction ($r = -.17, p = .030$). For CS-, during early extinction there was an association found between prospective IU (both controlled and alone) with the ssVEP ($r = -.16, p = .035$, and $r = -.15, p = .049$, respectively). In late extinction, valence ratings were also associated with prospective IU ($r = .15, p = .050$). No associations were found for the difference score during both sections of extinction. Resembling the results from acquisition, no significant correlations survived the Holm-Bonferroni corrections.

Table 4a.

Correlation coefficients and *p*-values reported for the relation between intolerance of uncertainty and the threat cue (CS+) during early and late Extinction.

Phase	Early Extinction				Late Extinction			
	IU (<i>r</i>)	IU (partial <i>r</i>)	I-IU (<i>r</i>)	I-IU (partial <i>r</i>)	P-IU (<i>r</i>)	P-IU (partial <i>r</i>)	IU (<i>r</i>)	P-IU (partial <i>r</i>)
Arousal^a								
Correlation	-	-	-	-	-	-	.06	-.05
<i>p</i> -Value	-	-	-	-	-	-	.438	.491
Valence^a								
Correlation	-	-	-	-	-	-	.09	-.03
<i>p</i> -Value	-	-	-	-	-	-	.245	.729
SCR								
Correlation	.06	-.03	.05	-.04	.06	-.02	.05	.07
<i>p</i> -Value	.416	.659	.539	.567	.417	.833	.496	.327
ssVEP								
Correlation	-.04	-.08	-.01	-.05	-.11	-.13	-.10	-.13
<i>p</i> -Value	.578	.273	.857	.556	.164	.079	.188	.082
LPP								
Correlation	-.10	-.07	-.11	-.10	-.03	.01	.09	.07
<i>p</i> -Value	.197	.341	.133	.201	.678	.924	.213	.330

Note. *P*-values reported are uncorrected. None of the *p*-values are significant after Bonferroni-Holm corrections ($p = .017$ for early extinction and $p = .010$ for late extinction). IU = Total IU; P-IU = Prospective IU; I-IU = Inhibitory IU; *r* = Pearson correlations; Partial *r* = Correlations were controlled for STAI-T, BDI-II, and PSWQ using partial correlations.

^a Valence and arousal ratings were only obtained one time after the extinction phase.

Table 4c.

Correlation coefficients and *p*-values reported for the relation between intolerance of uncertainty and the difference score (CS+ minus CS-) during early and late extinction.

Phase	Early Extinction				Late Extinction			
	IU (<i>r</i>)	IU (partial <i>r</i>)	I-IU (<i>r</i>)	I-IU (partial <i>r</i>)	P-IU (<i>r</i>)	P-IU (partial <i>r</i>)	I-IU (<i>r</i>)	I-IU (partial <i>r</i>)
Outcome Measures								
Arousal ^a								
Correlation	-	-	-	-	-	-	-	-
<i>p</i> -Value								
Valence ^a								
Correlation	-	-	-	-	-	-	-	-
<i>p</i> -Value								
SCR								
Correlation	.04	-.01	.08	-.01	.07	.03	-.09	-.07
<i>p</i> -Value	.643	.901	.280	.852	.353	.671	.228	.338
ssVEP								
Correlation	.08	.02	.04	.06	.04	.03	-.05	-.06
<i>p</i> -Value	.280	.769	.563	.436	.563	.671	.351	.435
LPP								
Correlation	-.06	-.06	.11	-.04	-.03	.09	.12	.08
<i>p</i> -Value	.443	.458	.138	.582	.674	.241	.114	.309

Note. *P*-values reported are uncorrected. None of the *p*-values are significant after Bonferroni-Holm corrections ($p = .017$ for early extinction and $p = .010$ for late extinction). IU = Total IU; P-IU = Prospective IU; I-IU = Inhibitory IU; *r* = Pearson correlations; Partial *r* = Correlations were controlled for STAI-T, BDI-II, and PSWQ using correlations.

^aValence and arousal ratings were only obtained one time after the extinction phase.

Discussion

This study aimed to investigate the role of intolerance of uncertainty (IU) during both fear and safety learning in a differential conditioning paradigm. In contrast to our expectations, we could not replicate earlier findings and we did not find an association between IU and conditioned defensive responses during both fear acquisition and extinction. Overall, we observed the anticipated indicators of fear acquisition, characterized by heightened defensive responses to the CS+ compared to the CS-. Extinction learning was not evident in the affective ratings as verbal fear responses towards the CS+ remained elevated compared to the CS-. This pattern corroborates with the results of the LPP and ssVEP signals during both the early and late phases of extinction. In contrast, the SCR signal gradually extinguished meaning that discriminative responses were visible during the early trials but not during later trials.

For fear acquisition, our findings align with the majority of studies that have not observed an effect of IU on fear learning (Johnson et al., 2023; Mertens & Morriss, 2021; Morriss, 2019; Morriss, Christakou, et al., 2016; Morriss et al., 2015, 2020; Morriss, Saldarini, Chapman, et al., 2019; Morriss, Saldarini, & van Reekum, 2019; Morriss & van Reekum, 2019; Sjouwerman et al., 2020; Wake et al., 2020, 2021; Wroblewski et al., 2022). However, some studies manipulating the reinforcement rate between the CS+ and US to increase uncertainty during learning have shown contradictory results. For example related with high IU levels, a 37.5% reinforcement rate was associated with worse threat-safety discrimination (Kanen et al., 2021), while a 50% rate showed enhanced discrimination compared to 75% (Chin et al., 2016). In contrast, Mertens et al. (2022) using a 75% reinforcement rate and Klingelhöfer-Jens et al. (2022) using 100% reinforcement rate demonstrated more pronounced discrimination in the fear ratings for individuals with higher IU. In our study, we did not collect fear ratings, which might be the reason why we did not observe effects for the IU even when using a 75% contingency rate. The US in our study was probably strongly aversive because of its loudness (90 dB) and its nature (white noise mixed with a female scream). It is therefore conceivable that these experimental settings created a so-called ‘strong situation’ (Lissek et al., 2006), which in turn may have overridden most of the inter-individual differences in acquisition phase.

In fear extinction, contrary to our hypothesis, we found no significant effect of IU, which aligns with some (e.g., Morriss et al., 2020, 2024; Wendt & Morriss, 2022) but not with other studies (Morriss, 2019; Morriss, Christakou, et al., 2016;

Morriss et al., 2015; Morriss, Macdonald, et al., 2016; Morriss, Saldarini, & van Reekum, 2019; Morriss & van Reekum, 2019; Wake et al., 2020, 2021; Wroblewski et al., 2022). Our findings also contrast Morriss and colleagues' (2021) meta-analysis, which analyzed 18 experiments and found that IU and both subfactors influenced SCR threat-safety discrimination in extinction. Interestingly, our results are in line with recent findings from clinical patients where no significant effects of self-reported IU were observed for differential SCR during acquisition and extinction (Morriss, Rodriguez-Sobstel, et al., 2024). Our findings are well in line with the only higher-powered study as of now. Morriss et al. (2020) using a well powered sample size ($N = 144$) investigated the effect of extended extinction, both directly after acquisition (i.e., immediate extinction) and next day (i.e., delayed extinction). They did not find an effect of the total IU score on both types of extinction. However, higher I-IU was associated with poorer SCR threat-safety discrimination (i.e. better extinction) in delayed extinction compared to immediate extinction. Considering that our and another well-powered study (Morriss et al., 2020) reported zero-findings, one might cautiously conclude that the influence of IU on extinction learning is not so robust as previously thought. However, this may also be dependent on the type of quantification of the individual differences of interest, as a recent multiverse meta-analysis showed (Morriss, Biagi, et al., 2024). This underscores the importance of well-powered replications to establish the conceptual basis of IU's role in fear extinction.

Our zero-findings may also be explained partly by the relative low uncertainty in our fear conditioning paradigm. With an initial reinforcement rate of 75% and an immediate extinction the situation is likely not uncertain or ambiguous enough. Indeed, an influence of IU is more often observed in situations where uncertainty is maximal. For example, Chen & Lovibond (2016) found that for high IU individuals, ambiguity, rather than uncertainty per se, leads to biased threat appraisal and negative affect. Other studies employing NPU threat paradigms and affective cuing also showed that high IU seems especially prone to different processing of these ambiguous cues, where uncertainty is maximized such that, the chance is 50/50 that an aversive stimulus can follow (Del Popolo Cristaldi et al., 2022; Gole et al., 2012; Nelson & Shankman, 2011).

Our results also point to a more general issue: The assessment of interindividual differences in psychophysiology faces some challenges, particularly in bridging different types of responses (e.g., questionnaire-based trait variables and neurobiological variables) (Bernoster et al., 2019; Brandt & Mueller, 2022). Personality traits like IU can represent higher abstracted levels, of variables that

represent experience and behavior, thus posing challenges in their association with specific neurobiological variables such as LPP or ssVEP (Brandt & Mueller, 2022). Similarly, associations between self-report measures and EEG tend to show correlations within the same type of variable rather than between types (Bernoster et al., 2019). Within this broader context, there is also a lack of consensus on how to measure and interpret IU. While some researchers advocate for the use of total IU scores (Bottesi et al., 2019; Hale et al., 2016; Lauriola et al., 2016; Morriss, Wake, et al., 2021; Shihata et al., 2018; Yao et al., 2021), others have found differential associations between IU subfactors and measured variables (e.g., Boelen et al., 2016; Jackson et al., 2016; Johnson et al., 2023; McEvoy & Mahoney, 2011; Penney et al., 2020; Saulnier et al., 2019). Further, the two IU subfactors may underline opposite mechanisms, which is challenging to merge into one construct such as the total IU. The prospective component (P-IU) is in fact thought to reflect cognitive engagement, while the inhibitory component (I-IU) represents the inhibition of action in the face of uncertainty (Birrell et al., 2011; McEvoy & Mahoney, 2011). A better clarification of IU measurement practices is essential to establish a robust conceptual and psychometric framework.

Altogether, fear conditioning was observed as predicted and we successfully induced fear acquisition. This was evidenced by significant changes in ratings (arousal, valence, and US-expectancy) and the physiological responses (SCR and ssVEP), albeit less clear for the LPP. This signal showed an enhanced amplitude for CS- compared to CS+ in early acquisition, which is contrary to our expectations and previous research (Bacigalupo & Luck, 2018). However, this effect was reversed and in line with aforementioned studies in the second half of the acquisition phase in our experiment. Other studies have also found no discrimination during acquisition (Bauer et al., 2020; Kastner et al., 2015), which distinguishes this signal from other measures of fear learning (as SCR or FPS) mainly by showing sometimes a delayed discrimination. Future research should consider these results when further validating the LPP as a reliable readout measure for both fear acquisition and extinction.

We found less consistent extinction of the conditioned responses. The arousal and valence ratings as well as the ssVEPs and LPPs still showed a difference between CS+ and CS-, while in the SCR successful extinction was observed. Previous studies have shown that ratings may be more resistant to extinction than physiological responses (Díaz et al., 2005; Hermans et al., 2002; Luck & Lipp, 2015). The resistance to extinction observed in the ssVEP and LPP might be linked to top-down attentional processes (Antov et al., 2020), which might also be involved

in verbal reports, in contrast with measures that closely reflect associative learning, such as the SCR (Constantinou et al., 2021), meaning that this signal reflected more accurately the change in CS-US contingencies characteristic of the extinction phase. The discrepancy between these measures (i.e., affective ratings, LPP and ssVEP vs. SCR) may indicate that these measures index different aspects of fear as proposed by the dual-process model of fear learning, where the subjective experience of fear can be separated from other physiological and behavioral responses (LeDoux & Brown, 2017; LeDoux & Pine, 2016; Öhman & Mineka, 2001), but can also be due to differences in habituation rates, measurements errors, or the noisiness of physiological measures (Sjouwerman et al., 2020).

Some limitations of our study should be considered. First, the data collection was carried out during the COVID-19 pandemic, this context could have influenced the reactions to threat and safety as well as beliefs around uncertainty (e.g., Korte et al., 2022; Segovia et al., 2024; Tanir et al., 2020). Second, the lack of fear ratings in our design limited the direct comparison of findings with other studies. Although our affective ratings are an indicator of evaluative learning, future research can benefit from also using fear ratings as they broaden the scope of the self-report. Third, the results of the LPP analysis must be interpreted cautiously, since the design of our experiment with flickering stimuli, relatively few trials, and the lack of mastoid references are suboptimal for LPP scoring and therefore these results cannot easily be compared to other findings (Bacigalupo & Luck, 2018; Bauer et al., 2020; Bruchmann et al., 2021; Klein et al., 2023; Nelson et al., 2015). Fourth, our sample mostly consist of female participants (81%), who have previously been shown to demonstrate slower extinction, seemingly caused by the hormonal fluctuations in their menstrual cycle (for a review see Merz et al., 2018). Even though women more often suffer from anxiety disorders, a more gender-balanced sample would allow for stronger conclusions in the general population.

In conclusion, our study utilized a fear acquisition and extinction paradigm with a large sample size to investigate the role of IU in conditioned fear responses. Our findings suggest that IU does not significantly impact fear acquisition and extinction. Altogether, these findings shed further light on a delimited role of this trait in fear acquisition and extinction in differential conditioning paradigms. Future studies with adequately powered samples are warranted to replicate our findings, alongside investigations into IU's influence on related paradigms such as fear generalization, renewal, and avoidance.

Supplementary materials

Results using square root and z-transformed SCR scores

We performed a square root transformation on our SCR data at trial level, then we z-standardized per participant their responses (across habituation, acquisition, and extinction), and averaged these for each stimulus (CS+ and CS-) at each phase and time (Acquisition and Extinction; for early and late). This procedure removed 10 participants from the SCR analysis (non-responders), resulting in a sample size of 166. These new means are available in our publicly shared dataset.

In Table 1 and 2, we present the results of the ANOVAs and ANCOVAs using the z-transformed SCR. The influence of total IU remains insignificant compared to the raw scores of the SCR. In Table 3 and 4, we present the bivariate and partial correlations of IU (and its subfactors) with the z-transformed SCR. Considering the same multiple test correction (Holm, 1979) used in the main correlational analysis, there are still no significant associations between IU and SCR. However, inhibitory IU is correlated with the responses to CS+ and the difference between CS+ and CS- during late acquisition, the latter remained significant after controlling for related questionnaires.

Table 1.

Analysis of the z-SCR in acquisition.

ANOVA	<i>F</i>	<i>p</i>	η_p^2
Stimulus	209.93	< .001*	.560
Time	169.66	< .001*	.507
Stimulus x Time	106.74	< .001*	.393
ANCOVA	<i>F</i>	<i>p</i>	η_p^2
Stimulus	29.70	< .001*	.153
Stimulus x IU	2.94	.088	.018
Time	5.73	.018*	.034
Time x IU	1.07	.302	.006
Stimulus x Time	2.60	.109	.016
Stimulus x Time x IU	1.25	.265	.008

Note. *P* values smaller than .05 are indicated by an asterisk (*).

Table 2.

Analysis of the z-SCR in extinction.

ANOVA	<i>F</i>	<i>p</i>	η_p^2
Stimulus	21.52	< .001*	.115
Time	33.51	< .001*	.169
Stimulus x Time	4.72	.031*	.028
ANCOVA	<i>F</i>	<i>p</i>	η_p^2
Stimulus	3.56	.061	.021
Stimulus x IU	0.50	.481	.003
Time	4.51	.035*	.027
Time x IU	0.41	.523	.002
Stimulus x Time	0.32	.573	.002
Stimulus x Time x IU	0.00	1.000	.000

Note. *P* values smaller than .05 are indicated by an asterisk (*).

Table 3.

Correlation coefficients and *p*-values reported for the relation between intolerance of uncertainty and the stimulus z transformed SCR during early and late acquisition.

Phase	Early Acquisition				Late Acquisition			
	IU (<i>r</i>)	IU (partial <i>r</i>)	I-IU (<i>r</i>)	I-IU (partial <i>r</i>)	P-IU (<i>r</i>)	P-IU (partial <i>r</i>)	I-IU (<i>r</i>)	P-IU (partial <i>r</i>)
CS+								
z-SCR								
Correlation	.02	.02	-.03	-.05	.07	.07	-.21	-.18
<i>p</i> -Value	.775	.849	.682	.531	.363	.408	.007*	.019
CS-								
z-SCR								
Correlation	.16	.18	.10	.11	.12	.12	.20	.18
<i>p</i> -Value	.040	.020	.194	.162	.129	.137	.011	.024
CS diff								
z-SCR								
Correlation	-.05	-.07	-.07	-.09	.01	.00	-.25	-.22
<i>p</i> -Value	.499	.383	.352	.248	.933	.976	.001*	.004*

Note. *P*-values reported are uncorrected. All *p*-values that remained significant after Holm-Bonferroni correction are indicated by an asterisk

(*). IU = Total IU; P-IU = Prospective IU; I-IU = Inhibitory IU; *r* = Pearson correlations; Partial *r* = Correlations were controlled for STAI-T, BDI-II, and PSWQ using partial correlations.

Table 4.

Correlation coefficients and *p*-values reported for the relation between intolerance of uncertainty and the stimulus z transformed SCR during early and late extinction.

Phase	Early Extinction				Late Extinction					
	IU (<i>r</i>)	IU (partial <i>r</i>)	I-IU (<i>r</i>)	I-IU (partial <i>r</i>)	P-IU (<i>r</i>)	P-IU (partial <i>r</i>)	IU (<i>r</i>)	IU (partial <i>r</i>)	I-IU (<i>r</i>)	I-IU (partial <i>r</i>)
CS+										
z-SCR										
Correlation	-.02	-.09	.01	-.06	-.01	-.07	.03	.10	.02	.08
<i>p</i> -Value	.843	.245	.954	.429	.859	.407	.698	.200	.816	.298
CS-										
z-SCR										
Correlation	.04	-.05	.04	-.04	.00	-.06	.09	.07	.15	.13
<i>p</i> -Value	.627	.562	.591	.625	.965	.458	.232	.393	.061	.095
CS diff										
z-SCR										
Correlation	-.04	-.04	-.02	-.02	-.01	-.01	-.05	.05	-.10	-.03
<i>p</i> -Value	.656	.628	.771	.786	.919	.886	.546	.569	.186	.724

Note. *P*-values reported are uncorrected. All *p*-values that remained significant after Holm-Bonferroni correction are indicated by an asterisk (*). IU = Total IU; P-IU =Prospective IU; I-IU = Inhibitory IU; *r*= Pearson correlations; Partial *r* = Correlations were controlled for STAI-T, BDI-II, and PSWQ using partial correlations.

Results using grouping of IU

As suggested by an anonymous reviewer we divided our sample in groups based on their IU scores. We included these groups as a between-subjects factor in our ANOVA models, exploring possible effects of IU on the z-transformed SCR.

We created the IU groups using two different approaches. First (Table 5 and 6), we divided our sample in four groups based on the quartiles of the IUS distribution. Here no effects of IU group were significant. Second (Table 7 and 8), we created two groups using the higher and lower scores in our sample. Both groups were based either in the cases scoring above or below one standard deviation from the sample mean. Again, this grouping approach did not result in any significant interaction of IU with the main effects. Importantly, this approach reduced significantly the sample size, resulting 27 participants for the low IU group and 29 for high IU.

Table 5.

Acquisition using IU quartiles

	<i>F</i>	<i>p</i>	η_p^2
Stimulus	209.23	< .001*	.564
Stimulus x IU quartile	0.85	.470	.015
Time	167.83	< .001*	.509
Time x IU quartile	0.50	.683	.009
Stimulus x Time	111.37	< .001*	.407
Stimulus x Time x IU quartile	2.45	.065	.043

Note. *P* values smaller than .05 are indicated by an asterisk (*).

Table 6.

Extinction using IU quartiles

	<i>F</i>	<i>p</i>	η_p^2
Stimulus	21.10	< .001*	.115
Stimulus x IU quartile	1.04	.377	.019
Time	33.46	< .001*	.171
Time x IU quartile	0.37	.775	.007
Stimulus x Time	5.11	.025*	.031
Stimulus x Time x IU quartile	1.60	.191	.029

Note. *P* values smaller than .05 are indicated by an asterisk (*).

Table 7.Acquisition using IU extreme groups ($\pm 1 SD$)

	<i>F</i>	<i>p</i>	η_p^2
Stimulus	56.27	< .001*	.510
Stimulus x IU extremes	1.85	.179	.033
Time	48.68	< .001*	.474
Time x IU extremes	2.14	.150	.038
Stimulus x Time	87.48	< .001*	.618
Stimulus x Time x IU extremes	1.66	.203	.030

Note. *P* values smaller than .05 are indicated by an asterisk (*).**Table 8.**Extinction using IU extremes groups ($\pm 1 SD$)

	<i>F</i>	<i>p</i>	η_p^2
Stimulus	5.58	.022*	.094
Stimulus x IU extremes	0.84	.364	.015
Time	25.31	< .001*	.319
Time x IU extremes	0.98	.326	.018
Stimulus x Time	7.89	.007*	.127
Stimulus x Time x IU extremes	0.18	.670	.003

Note. *P* values smaller than .05 are indicated by an asterisk (*).

Chapter 4



The uncertain path to reward:
Neural mechanisms of intolerance
of uncertainty in reward processing

This chapter has been published as:

Malbec, M., Harrewijn, A., Franken, I. H. A., & Wieser, M. J. (2025). The uncertain path to reward: Neural mechanisms of intolerance of uncertainty in reward processing. *Cortex*, 192, 227–241. <https://doi.org/10.1016/j.cortex.2025.09.010>

Abstract

Reward processing is a neurocognitive process involving the evaluation of and response to rewarding stimuli, which is critical for learning and motivated behavior. This cognitive mechanism is also influenced by mental health. Intolerance of uncertainty (IU) is a dispositional tendency to perceive uncertainty as distressing and respond negatively to uncertain situations. Proposed as a transdiagnostic factor for internalizing disorders, IU may help explain shared features across these conditions. This study investigated the influence of IU on electrocortical responses to reward (feedback-related ERP) using the Doors Task, which incorporated varying levels of uncertainty (i.e., risk) regarding rewards. In 203 participants, we found that risk levels significantly influenced ERPs, particularly following reward feedback, with high-risk trials eliciting larger (i.e. more positive) ERPs compared to ambiguous or low-risk trials. While total IU did not correlate with feedback-related brain activity, its subfactors showed distinct effects after reward and no-reward feedback: higher prospective IU scores were associated with increased brain activity, whereas higher inhibitory IU scores were linked to decreased activity. These effects persisted after accounting for related internalizing traits, including worry, depression, and trait anxiety. Additionally, depressive symptoms were associated with blunted feedback-related ERPs, particularly following no-reward feedback. Taken together, the findings suggest a more nuanced and complex role of IU and its subfactors in reward processing and demonstrate the impact of risk on electrocortical responses to reward outcomes.

Keywords: Reward processing, Intolerance of uncertainty, Event related potentials, Risk, Reward positivity.

Introduction

The ability to react differently to rewards or to adverse outcomes is key for adaptive goal-directed behavior. Reward processing refers to how the brain evaluates and responds to positive outcomes (Berridge & Kringelbach, 2015). Further, when the actual outcome deviates from our expectations, a reward prediction error is experienced (Schultz, 2016; Watabe-Uchida et al., 2017), which is processed in brain regions such as the anterior cingulate cortex, prefrontal cortex, and basal ganglia (Krigolson, 2018; Schultz, 2016; Watabe-Uchida et al., 2017). An experimental task used to study reward processing is the so called ‘Doors task’ (Hajcak et al., 2007). In this task, participants are required to make a decision by picking one door from an array of doors (typically 2-5). Based on their selection, feedback is presented as reward or negative outcome (either loss or no-reward). This allows for an isolated and detailed observation of the individuals’ responses to both types of feedback. Some researchers have implemented probability cues presented before the doors appear, signaling the chances of a reward in that trial (i.e., how many doors contain a prize). After the participants’ decision is recorded, feedback is presented (i.e., reward, no-reward, or loss). Typically, this task allows researchers to differentiate the electrocortical responses associated with the different types of feedback, making it ideal for the investigation of reward processing (Levinson et al., 2017).

Reward processing and the reward prediction error can be identified through the electroencephalogram (EEG); the majority of studies have used the reward positivity (RewP), a frontocentral component detected 250–350 ms after feedback indicating gains over no-reward or loss conditions (Holroyd et al., 2008; Proudfit, 2015). The RewP is usually calculated as a difference potential, based on the amplitude after reward feedback minus the amplitude of loss or no-reward feedback. However, the RewP is sometimes analyzed separately as the response to gains (RewP to gain), losses (RewP to loss or feedback negativity, FN), or both. This brain potential has demonstrated convergent validity with measures of reward sensitivity, as behavioral and self-report ones (Bress & Hajcak, 2013). While the RewP captures how the brain evaluates outcomes, the degree of uncertainty—whether stemming from risk or ambiguity—further modulates these electrocortical responses.

The expectations individuals hold during decision-making, shaped by the available information, play a crucial role in how they evaluate and process the outcomes of their actions. Two key factors that illustrate this aspect are risk and

ambiguity. Unexpected outcomes typically elicit stronger ERP responses, such as the RewP (Holroyd & Coles, 2002; Sambrook & Goslin, 2015). These responses are often modulated by the probability of an outcome (Holroyd et al., 2003; Nieuwenhuis et al., 2005) and also by the subjective expectations (Hajcak et al., 2007). Further, Holroyd et al. (2009) found that modulation of the RewP by winning probability was stronger in tasks involving learning, highlighting the role of expectations (see also San Martín, 2012). In addition to expectedness, uncertainty—whether due to ambiguous or uncertain cues—also plays a crucial role in modulating feedback-related brain activity. For example, Xu et al. (2011) found that presenting cues indicating uncertainty in a passive gambling task heightened neural responses across different ERP components. This suggests that uncertainty intensifies sensitivity to outcomes, enhancing the brain's attention to feedback when future results are less predictable. So, uncertainty intensifies neural sensitivity to feedback and disruptions in this process have been implicated in internalizing psychopathology.

Impairments in reward processing have been associated with various forms of psychopathology, such as depression (Forbes & Dahl, 2005; Proudfit, 2015) and substance abuse disorders (e.g., Crane et al., 2021; Parvaz et al., 2016). Although anxiety disorders have been rarely investigated with regard to reward processing, dysfunctions in the neural reward circuitry have been linked with the onset and progression of anxiety disorders (Silk et al., 2012). Nelson and Jarcho (2021) using both a social and a monetary task found an opposing relationship of the RewP after 'reward feedback' for depression and social anxiety in both contexts, namely higher depression was associated with a blunted RewP while elevated social anxiety symptoms were linked with an enhanced RewP after positive feedback. The relationship between depression and reward processing is better established. A reduced RewP amplitude has been consistently associated with various measures of depression including current depressive disorders and depressive symptoms across youth and adults (Bress et al., 2012; Burani et al., 2019; Foti & Hajcak, 2009; Liu et al., 2014). Moreover, a reduced RewP amplitude has been linked to familial history of depression in youth (Foti et al., 2011), as well as the prediction of future depressive symptoms and disorders (Bress et al., 2013). A reduced RewP is even proposed as a biomarker for depression (Proudfit, 2015). Taken together, these findings underscore the critical role of disrupted reward processing in internalizing disorders, emphasizing the importance of exploring how distinct but interconnected symptoms influence this cognitive mechanism. Among factors common to these disorders,

intolerance of uncertainty (IU) emerges as a transdiagnostic trait that may influence altered reward processing.

IU is considered a dispositional trait that reflects an individual's aversiveness or incapacity to accept uncertain scenarios (Carleton, 2016). People with high IU find uncertainty distressing and even threatening (Carleton, 2016; Dugas et al., 2005) regardless of the actual chances of a bad outcome (Buhr & Dugas, 2002). Two dimensions of IU have been identified using the 'intolerance of uncertainty scale' (Buhr & Dugas, 2002; Freeston et al., 1994): Prospective IU (P-IU), which involves a desire for predictability and actively seeking out certainty, and Inhibitory IU (I-IU), which relates to avoidance behaviors, behavioral inhibition, and feelings of paralysis in the face of uncertainty (Carleton, 2012). IU has been mainly associated with anxiety disorders (Carleton, 2012) and increasingly with a wider array of other internalizing disorders including depression, suggesting IU as a transdiagnostic construct for internalizing disorders (Boswell et al., 2013; Carleton et al., 2012; Gentes & Ruscio, 2011; Mahoney & McEvoy, 2012a, 2012b; McEvoy et al., 2019; McEvoy & Erceg-Hurn, 2016). However, some studies have not found a direct link between IU and depression (Jensen et al., 2016; Yook et al., 2010). Furthermore, IU has been associated with key neurocognitive processes like decision making under uncertainty and threat anticipation (as detailed in the Uncertainty and Anticipation Model of Anxiety; Grupe & Nitschke, 2013; Tanovic et al., 2018), as well as reward processing (Gorka et al., 2016; Nelson et al., 2014, 2016). Research has also demonstrated that IU is linked to dampened positive emotional states (Morris, Goh et al., 2023). Moreover, IU's impact extends to other cognitive processes crucial for navigating uncertain environments, including safety learning and the anticipation of uncertain threat, with these associations increasingly being investigated through neural and psychophysiological markers (Morris, Abend et al., 2023; Tanovic et al., 2018).

Although IU is broadly linked to anxiety and depression, its specific impact on feedback-related brain activity remains underexplored and has yielded mixed results. To our knowledge, only a few studies have explored the impact of IU on feedback-related brain activity using the Doors task. Nelson and colleagues (2016) found no relationship between total IU and the RewP, however there was an opposing relationship of both IU subfactors with the RewP. Inhibitory IU was associated with an attenuated RewP, while prospective IU was linked with a larger RewP. The other study that explored the impact of IU on feedback-related brain activity employed two adaptations of the Doors Task (Muir et al., 2021). One version manipulated the probability of reward outcomes

(reward uncertainty), while the other altered the probability of delays in feedback (timing uncertainty). The researchers found that uncertain rewards enhanced the RewP amplitude, whereas this neural response was less sensitive to timing uncertainty. However, no relationship between IU and the RewP amplitude was observed (Muir et al., 2021). Notably, the total level of IU has also not shown an influence on reward-related responses in other paradigms, such as reward conditioning (Morris, Biagi, et al., 2021) and tasks assessing how individuals choose between seeking new rewards or keeping known rewards (Krypotos et al., 2022). Furthermore, using fMRI, Gorka and colleagues (2016) found different effects for IU's subfactors on reward processing. They investigated insula activation while participants received feedback in a slot machine task. Specifically, they found that higher P-IU was associated with more anterior insula activation to uncertain rewards, while no effect was found for I-IU (Gorka et al., 2016). To clarify these inconsistencies, the present study examines how total IU and its subfactors influence electrocortical responses to reward under varying levels of uncertainty (risk and ambiguity) in the Doors task.

The present study examined the association between intolerance of uncertainty (IU) and electrocortical responses related to reward processing (e.g., post-feedback brain activity) during a monetary gambling task. Additionally, we explored the potential effects of reward uncertainty on this electrocortical response. Both the overall IU score and its subfactors (i.e., prospective and inhibitory IU) were tested to evaluate their unique contributions to reward processing in this context. Based on previous findings (Nelson et al., 2016), we hypothesized that both IU subfactors would show an opposing relationship with post-feedback brain activity depending on feedback type. Specifically, higher I-IU will be linked to diminished, while P-IU will be linked to enhanced brain responses after feedback. This aligns with Nelson and colleagues' (2016) findings, where the subfactors showed opposing associations with the difference between reward and negative feedback (i.e., RewP). The hypothesis about the role of total IU on reward processing remains explorative, because in previous studies (Nelson et al., 2016; Muir et al., 2021) no role of total IU was detected. However, considering that uncertain rewards have evoked elevated brain potentials compared to certain outcomes (Hajcak et al., 2007; Pfabigan et al., 2011; Walentowska et al., 2019) and individuals high on IU should manifest an equivalent sensitivity to uncertainty; this sensitivity could be observable in an enhanced reward-related ERP after uncertain rewards. Further, since IU is a transdiagnostic factor, we will also investigate associations between reward

processing and symptoms of anxiety, depression, and worry. We do this by including the scores of these trait questionnaires whenever we find a significant association with either IU or its subfactors. This will allow us to further clarify IU's role on reward processing, as well as exploratively shed light on the influence that these other internalizing traits might have on reward processing within our study.

Methods

Participants

In total, 214 psychology undergraduate students from Erasmus University Rotterdam took part in the study, recruited via an online recruitment system. Data of two participants had to be removed from the final sample because they reported wrong comprehension of the instructions after completion of the task. Due to inadequate behavioral performance, six participants were removed from the final sample (see Data analysis). One participant had corrupted EEG data, and two had missing questionnaire data. As a result, the final sample used in all the analyses consisted of 203 participants. The age range was 17-46 years ($M = 21.07$, $SD = 3.02$), 80.8% were female, and 51.7% were Dutch. A breakdown of their ethnic self-identification was: 72.9% for White/Caucasian, 8.9% for Asian, 4.4% for Middle Eastern, 3.9% for Black, 3.5% for mixed, 2.0% for Latin-American, and 3.9% for other. A-priori exclusion criteria were family history of photic epilepsy, related to the flickering images in another task in the same lab session (Malbec et al., 2024 and *Chapter 3*). Informed consent was obtained before participation, and the research protocol was approved by the ethical board of the Erasmus School of Behavioural and Social Sciences in accordance with the declaration of Helsinki. The lab sessions took place between September 2020 and July 2021.

Questionnaires

After the experimental task, participants filled out the following questionnaires (either in Dutch or English, depending on whether they were Dutch or international students; see Table 1):

The Intolerance of Uncertainty Scale-12 (IUS-12; Carleton et al., 2007), a short version of the Intolerance of Uncertainty Scale (Buhr & Dugas, 2002; Freeston et al., 1994; Dutch translation: de Bruin et al., 2006), was used to measure intolerance of uncertainty. The IUS evaluates individuals' discomfort with uncertain situations and their reactions to them. Responses are recorded on a five-

point Likert scale ranging from 1 (“not at all characteristic of me”) to 5 (“entirely characteristic of me”).

The Beck Depression Inventory, Second Edition (BDI-II; Beck et al., 1996; Dutch translation: Van der Does, 2002) was utilized to assess depressive symptoms, both cognitive and somatic. Participants rated all 21 items on a four-point Likert scale from 0 to 3, reflecting the perceived severity over the past two weeks.

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990; Dutch translation: van Rijsoort et al., 1999) was used to measure the trait of worry across dimensions of excessiveness, generality, and uncontrollability. Participants rated each of the 16 items on a five-point Likert scale from 1 (“not at all typical of me”) to 5 (“very typical of me”).

The trait subscale of the State Trait Anxiety Inventory, Form Y (STAI-T; Spielberger, 1983; Dutch translation: Van der Ploeg, 2000) was used to measure an individual’s general propensity for anxiety. Participants rated the 20 items based on their typical feelings, using a four-point Likert scale from 1 (“almost never”) to 4 (“almost always”).

Table 1

Overview of Questionnaire Data from Final Sample

Questionnaire	Range	<i>M</i>	<i>SD</i>	Internal Consistency (α)
Intolerance of Uncertainty	12 - 51	29.46	8.25	0.88
Prospective IU	7 - 31	18.29	5.34	0.83
Inhibitory IU	5 - 20	11.17	3.74	0.78
Depression (BDI)	0 - 34	6.00	7.69	0.94
Worry (PSWQ)	25 - 72	45.18	10.61	0.83
Trait Anxiety (STAI-T)	24 - 66	45.08	8.33	0.81

Material and Stimuli

Study design and Procedure

After arriving in the lab, participants received verbal instructions of the experimental procedure and signed written informed consent. Then, they were

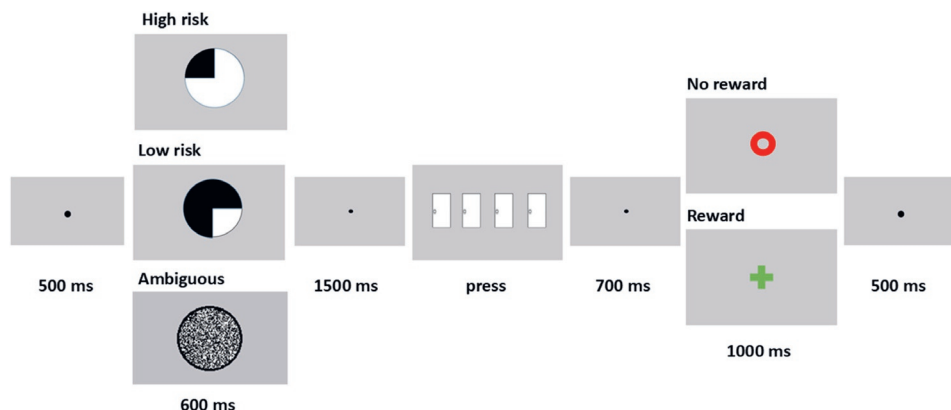
seated in an isolated dimly lit room and the EEG and EDA electrodes were attached. Afterwards, they completed the Doors task with an approximate duration of 40 min. Secondly, participants completed a fear conditioning task and thirdly a flanker task experiment; results of these experiments are reported elsewhere (Carsten et al., 2025; Malbec et al., 2022, 2024). After completion of the experimental task, the EEG and EDA electrodes were removed and participants completed the self-report questionnaires on a PC.

Doors Task

We employed a gambling task, almost identical in design to the Doors task used by Paul and Pourtois (2017) (itself adapted from Hajcak et al., 2007; see Figure 1). The images used as stimuli are available at <https://osf.io/gkymz/>. Each trial began with a fixation dot for 700 ms, followed by a cue (600 ms) indicating the level of risk (i.e., chances of winning) for that specific trial. This cue was represented by a small circle indicating the probabilities of winning: either one-quarter filled (high risk, 25% chance of winning), three-quarters (low risk, 75% chance of winning), or filled with a static pattern of white and black pixels (ambiguous risk, unknown chances of winning). The informative color of high and low risk cues was counterbalanced (i.e., for half of the participants the chances were represented by black and the other half, white). After this cue, a 1500 ms fixation dot preceded the selection of doors. Participants were presented with an array of four identical doors and instructed to select one using numbers 1 to 4 on a QWERTY computer keyboard, corresponding to each door. After 700 ms, feedback on their choice was provided for 1000 ms, with a green cross indicating a reward of 2.5 cents or a red circle indicating no reward. To ensure that participants paid attention to both the risk cues and feedback, they were asked in 24 trials about the risk of the current trial (“How many doors contain a prize?”), we will refer to them as cue-catch trials. In other 24 trials they were asked about the expectedness of the feedback (rated on a visual analog scale from “very unexpected” to “very expected”).

Figure 1.

Schematic of the trial design of the Doors Task.



Note. The images presented in the figure are enlarged for the purpose of illustration.

The experiment commenced with instructions, then a training phase of twelve trials, followed by four blocks of 92 trials each. Participants were instructed about the meaning of the risk cues, the rewards for successful trials, and about the questions in between trials. A practice round was used for verifying the correct understanding of these instructions. The three risk cues were presented randomly; there were 80 trials for ambiguous risk, and 144 for each of the high- and low-risk trial types. After each block participants had a self-administered break and were reminded of the total amount of money accumulated so far. Unbeknownst to the participants, feedback was based solely on these three objective reward probabilities. Specifically, the likelihood of receiving reward feedback was three times higher for trials where three doors contained a prize compared to trials where only one door had a prize. Thus, for low-risk trials, participants received reward feedback in 108 trials and no-reward feedback in 36 trials. Conversely, for high-risk trials, participants received no-reward feedback in 108 trials and reward feedback in 36 trials. Ambiguous trials had an equal distribution of feedback, with 40 trials each for reward and no-reward feedback. In this way, all participants received €5.

EEG

EEG data were recorded using 32 Ag/AgCl electrodes arranged according to the 10/20 configuration. The electro-oculogram (EOG) was captured using 4 external electrodes: two placed above and below the left eye, and two at the outer canthus of each eye. Data were collected using the Active-Two BioSemi system (BioSemi, Amsterdam, The Netherlands), which includes two additional elec-

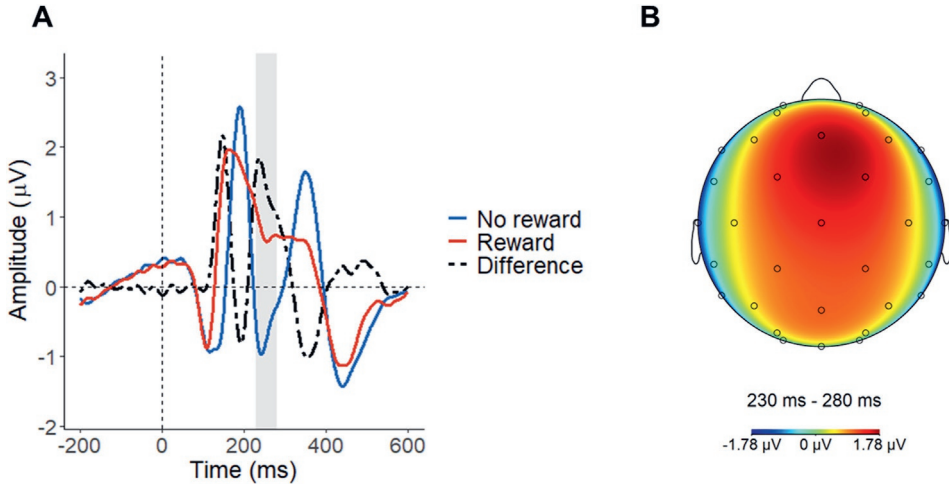
trodes, common mode sense (CMS, as reference during the recording) and driven right leg (DRL, as ground). The signal was digitized at a 512 Hz sampling rate with 24-bit A/D conversion.

EEG was analyzed offline using BrainVision Analyzer 2.3 (BrainProducts Inc., Gilching, Germany). First, EEG data were re-referenced to the average voltage at all (32) scalp electrodes, band-pass filtered from 0.1 to 30 Hz (Butterworth zero phase filter order 8), and notch-filtered at 50 Hz. Then, data were segmented in 2500 ms epochs, using the feedback onset as reference (900 ms before and 1600 ms after). Eye-movement artifacts were corrected using the Gratton et al. (1983) approach. Artifacts were removed based on the following criteria: (a) maximal amplitude allowed of $\pm 75 \mu\text{V}$, (b) a gradient criterion of $\pm 50 \mu\text{V/ms}$, (c) a difference criterion of $200 \mu\text{V}$ in 200 ms segments, and (d) a low activity criterion of $2 \mu\text{V}$ (interval length: 200 ms). We applied a baseline correction using the 250 ms before feedback onset. Lastly, the data was averaged per feedback type (reward and no-reward) and risk condition (high, low, and ambiguous), resulting in six averages per participant. The post-feedback ERP was defined as the mean amplitude between 230 and 280 ms after feedback onset averaged across channels Fz and Cz (based on Paul et al., 2020; Paul & Pourtois, 2017; see Figure 2). We computed the amplitudes for each feedback type separately rather than using a difference score ERP, before including both in the same statistical model. This decision facilitates a more detailed investigation of how IU and related traits uniquely affect electrocortical responses to distinct feedback types. Furthermore, this feedback-specific approach avoids potential psychometric challenges and the masking of individual difference relationships that can arise with the use of difference scores, particularly in reward processing research⁵ (Clayson et al., 2021; Levinson et al., 2017). The percentage of removed epochs for the post feedback ERP signal was 1.12% for no-reward and 1.04% for reward feedback trials.

⁵ We calculated the RewP as a difference score and included it in a similar linear mixed model as our main analysis. The corresponding methods and results are available on our Supplementary Materials.

Figure 2.

Brain activity after feedback presentation.



Note. A) ERPs by feedback type and their difference, averaged across Cz and Fz channels. The difference waveform corresponds to the grand averaged difference of reward minus no-reward trials. B) Scalp topography of the difference waveform (i.e., reward positivity or RewP) between reward and no-reward trials at 230-280 ms after feedback onset.

Data analysis

All analyses were carried out using R version 4.4.0 (R Core Team, 2024) and RStudio version 2024.04.1 (Posit team, 2024). A public version of the R code is available at <https://osf.io/gkymz/>. To assess the behavioral performance, we calculated the accuracy of the cue-catch trials (see Procedure) and explored the participants' selection pattern. Based on the behavioral performance in the task, we decided to keep participants with an accuracy level above chance (.25), therefore removing two participants. In a similar vein, we excluded four additional participants who selected the same door for most of the experiment trials (>75%).

Firstly, to investigate the impact of IU on the brain activity after feedback presentation, linear mixed models were conducted with the post-feedback ERP as outcome variable. These models were fitted using the R-packages *lme4* (Bates et al., 2015) and *lmerTest* (Kuznetsova et al., 2017), and significance is reported with the Kenward-Rogers approximation for the degrees of freedom (Kenward & Roger, 1997). Fixed effects included risk (3 factor levels: low, high, and ambiguous), feedback (2 factor levels: reward and no-reward), and IU (continuous and mean centered). A unique intercept was estimated per participant (i.e.,

random intercept) to model variance in intercepts due to unexplained differences between participants. Bonferroni-corrected pairwise comparisons of the estimated marginal means were used as post-hoc test for comparing predictor levels and estimated trends for comparing interactions (using the *emmeans* or *emtrends* functions respectively; Lenth, 2024). Effect sizes were estimated using semi-partial R^2 (unique variance explained) and confidence intervals were computed for each fixed effect using the “r2beta” function (r2glmm R package, v0.1.2; Jaeger, 2017; see also: Nakagawa & Schielzeth, 2013; Rights & Sterba, 2019), with the Kenward-Roger method for CIs.

Secondly, for investigating the role of IU’s subfactors on reward processing, a separate model was fitted for the ERP using IU subfactors (i.e., prospective, and inhibitory IU) as continuous, mean-centered, fixed effects instead of total IU.

Thirdly, exploratory analysis included the addition of depression, trait anxiety, and worry besides IU in the same model predicting both ERPs’ amplitude. This final model would allow us to control for the influence of IU on reward processing by accounting for these internalizing traits, and simultaneously explore their unique contributions to the same cognitive process. All these variables were mean centered in all models.

Results

The processed dataset of the final sample, raw EEG data, and raw questionnaire data are publicly available at <https://osf.io/gkymz/>. The details and results from the supplementary analysis using the difference score RewP, along with histograms and correlations for all our continuous variables are available as Supplementary Materials (also available at <https://osf.io/gkymz/>).

Behavioral results

The overall accuracy of the cue-catch was good and well above chance level: $M = 85.40\%$, $SD = 11.81$. The percentage of selection of each door was 21.8 %, 29.5 %, 29.2 %, and 19.4 % respectively for each door from left to right in the array.

ERP results

The first model investigated the impact of IU on reward processing (see Table 2). First, as a check of the principal effects of the task, we found that reward trials show a more positive amplitude ($M = 0.76 \mu V$, $SD = 2.39$), than no-reward trials ($M = -0.67 \mu V$, $SD = 2.25$), visible in the significant main effect of feedback ($F = 465.74$, $p < .001$) (Figure 4). This was influenced by the level of risk,

as shown by the significant interaction of Risk x Feedback ($F = 5.91, p = .003$). Subsequent post-hoc pairwise comparisons for reward and non-reward feedback separately, showed that the effect of risk level on the post feedback ERP amplitude varied depending on the type of feedback (Bonferroni-corrected). For reward feedback, more pronounced differences between the different risk levels were found, which was not the case for no-reward feedback (Figure 3 and 4). Specifically, high risk trials had a higher amplitude than ambiguous risk trials ($b = -0.35, p = .007$) and low risk trials ($b = 0.43, p < .001$), while ambiguous trials did not differ significantly from low risk trials ($p > .999$). In contrast, for no-reward feedback, no significant differences in post feedback amplitudes were found between the different risk levels (all $ps > .05$). Then, we observed that the interactions involving IU were all non-significant (all $ps > .110$), suggesting that IU did not significantly alter the effects of risk or feedback on the feedback-related brain activity.

Table 2

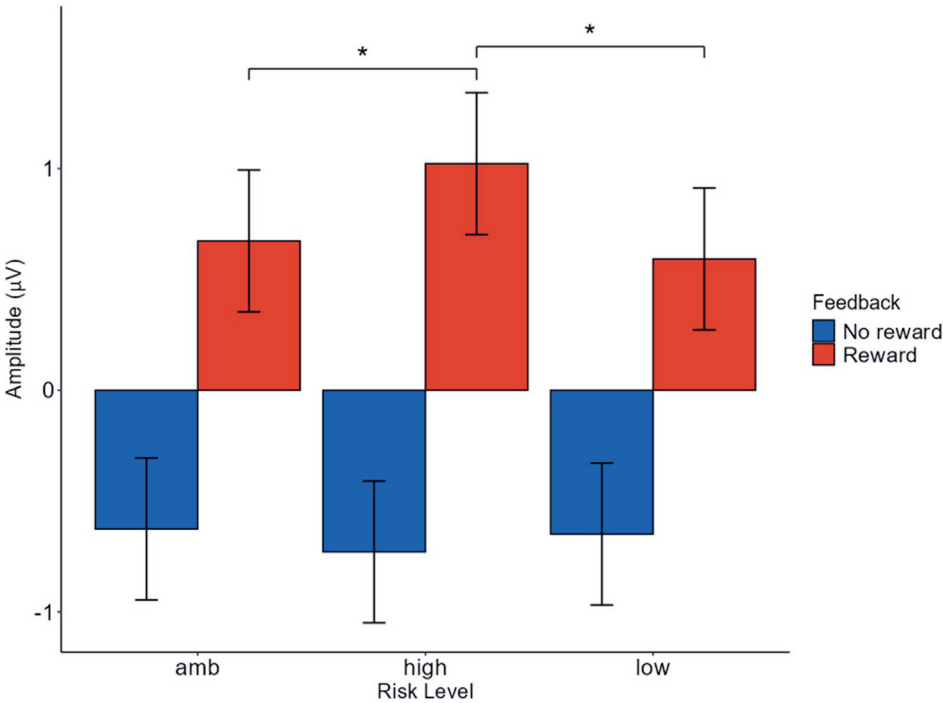
Linear mixed model analyses for the feedback-related brain activity using total IU.

Factor(s)	<i>F</i> -value	<i>df</i>	<i>p</i> -value	<i>R</i> ²	95% CI
Risk	2.45	(2, 1005)	.087	.005	[.000, .018]
Feedback	465.74	(1, 1005)	< .001	.317	[.273, .361]
IU	0.87	(1, 201)	.352	.004	[.000, .041]
Risk x Feedback	5.91	(2, 1005)	.003	.012	[.003, .030]
Risk x IU	0.37	(2, 1005)	.689	.001	[.000, .010]
Feedback x IU	2.55	(2, 1005)	.110	.003	[.000, .012]
Feedback x Risk x IU	0.35	(2, 1005)	.704	.001	[.000, .010]

Note. IU = Intolerance of uncertainty. The *R*² values presented in this table represent semi-partial *R*² effect sizes (see Methods).

Figure 3.

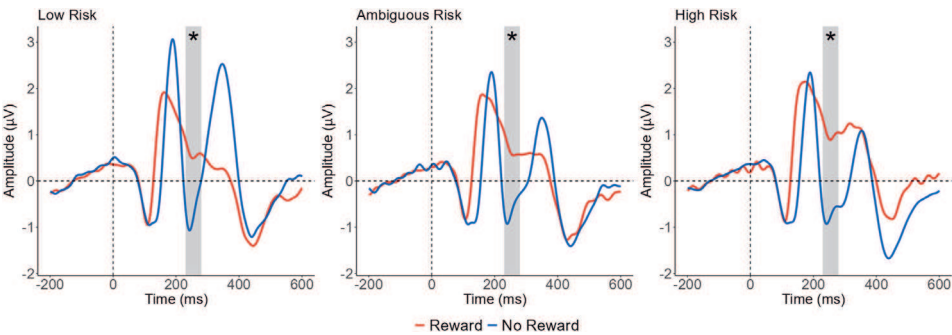
Comparisons of the amplitudes of the feedback-related brain potentials between risk levels divided by feedback type.



Note. Amb = Ambiguous trials; high = High risk trials; low = Low risk trials.

Figure 4.

Feedback related brain activity in the three risk conditions.



Note. The gray rectangle highlights the time-window (230 to 280 ms after feedback onset) used to calculate the ERPs.

We ran a separate model using both IU subfactors instead of the total IU score, to test their individual effects on post-feedback brain activity. Here,

we obtained similar results as in the previous model (see Table 3). The feedback-related brain activity was larger for reward than no-reward trials (feedback: $F = 464.01$, $p < .001$), and this effect changed depending on the level of risk, as shown by the significant interaction of Risk x Feedback ($F = 5.89$, $p = .003$). Notably, both prospective and inhibitory IU subfactors had inverse main effects on the amplitudes of the brain activity regardless of feedback type. Higher prospective IU scores were related with increased amplitude ($b = 0.10$, $p = .008$), while higher scores of the inhibitory IU scale predicted a decrease in this brain activity ($b = -0.14$, $p = .010$). The interactions of IU subfactors with other factors were all non-significant (all $ps > .236$).

Table 3

Linear mixed model analyses for the feedback-related brain activity using prospective and inhibitory IU.

Factor(s)	<i>F</i> -value	<i>df</i>	<i>p</i> -value	<i>R</i> ²	95% CI
Risk	2.44	(2, 1000)	.088	.005	[.000, .018]
Feedback	464.01	(1, 1000)	< . .001	.317	[.274, .361]
P-IU	9.89	(1, 200)	.002	.047	[.007, .118]
I-IU	6.52	(1, 200)	.011	.032	[.002, .095]
Risk x Feedback	5.89	(2, 1000)	.003	.012	[.003, .030]
Risk x P-IU	0.10	(2, 1000)	.909	.000	[.000, .008]
Feedback x P-IU	1.41	(1, 1000)	.236	.001	[.000, .010]
Risk x I-IU	0.73	(2, 1000)	.485	.001	[.000, .012]
Feedback x I-IU	0.02	(1, 1000)	.893	.000	[.000, .005]
Risk x Feedback x P-IU	0.01	(2, 1000)	.991	.000	[.000, .007]
Risk x Feedback x I-IU	0.19	(2, 1000)	.828	.000	[.000, .009]

Note. P-IU = Prospective IU; I-IU = Inhibitory IU. The R^2 values presented in this table represent semi-partial R^2 effect sizes (see Methods).

To broaden the understanding of how a transdiagnostic factor like IU influences reward processing, we also explored the role of related traits, including depression, anxiety, and worry. These traits were included in a model alongside both IU subfactors, since both showed a significant association with the feedback-related brain activity in the previous model. As in the previous models, the brain potential amplitude was higher for reward than no-reward trials (feedback: $F = 466.56$, $p < .001$). Both IU subfactors, again had a significant main effect. Higher scores of prospective IU ($b = 0.10$, $p = .015$) were linked with more positive

post feedback ERP, while higher inhibitory IU ($b = -0.17, p = .007$) was related to lower amplitudes after either feedback type.

Additionally, there was a significant interaction between Feedback and Depressive symptoms (see Table 4). Specifically, for no-reward feedback, more depressive symptoms were associated with more negative amplitudes ($b = -0.053, SE = 0.024, CI = [-0.100, -0.006]$). For the reward feedback condition, although the point estimate suggested a similar trend ($b = -0.026, SE = 0.024$), the confidence interval ($CI = [-0.073, 0.021]$) included zero, indicating uncertainty about the presence or direction of this effect. A direct comparison of the slopes revealed that the relationship between depression and amplitude differed significantly between feedback types ($\Delta b = -0.027, SE = 0.011, p = .014$).

Similarly, the level of trait anxiety impacted the relationship between feedback type and amplitude of the post feedback ERP (see Table 4). For no-reward feedback, higher anxiety symptoms showed a weak positive association with amplitude ($b = 0.020, SE = 0.021, CI = [-0.021, 0.062]$). For reward feedback, the association was slightly negative ($b = -0.003, SE = 0.021, CI = [-0.045, 0.038]$). However, in both cases, the confidence intervals included zero, indicating no clear evidence for a consistent directional effect. Pairwise comparisons of the slopes showed that the trend for no-reward feedback differed significantly from that for reward feedback ($\Delta b = 0.023, SE = 0.010, p = .015$). Overall, this highlights that the impact of trait anxiety is small and should be interpreted carefully.

Table 4

Linear mixed model analyses for the feedback-related brain activity using IU subfactors, trait anxiety, depression, and worry.

Factor(s)	<i>F</i> -value	<i>df</i>	<i>p</i> -value	<i>R</i> ²	95% CI
Risk	2.45	(2, 985)	.087	.005	[.001, .019]
Feedback	466.56	(1, 985)	< .001	.321	[.278, .366]
P-IU	8.32	(1, 197)	.004	.041	[.004, .109]
I-IU	6.26	(1, 197)	.013	.031	[.002, .094]
Anxiety	0.18	(1, 197)	.675	.001	[.000, .029]
Depression	2.89	(1, 197)	.091	.014	[.000, .065]
Worry	2.06	(1, 197)	.153	.010	[.000, .056]
Risk x Feedback	5.92	(2, 985)	.003	.012	[.003, .030]
Risk x P-IU	0.13	(2, 985)	.875	.000	[.000, .008]
Risk x I-IU	1.71	(2, 985)	.182	.003	[.000, .016]

Feedback x P-IU	1.37	(1, 985)	.242	.001	[.000, .010]
Feedback x I-IU	0.03	(1, 985)	.870	.000	[.000, .005]
Risk x Anxiety	0.87	(2, 985)	.420	.002	[.000, .012]
Feedback x Anxiety	5.92	(1, 985)	.015	.006	[.000, .019]
Risk x Depression	0.86	(2, 985)	.423	.002	[.000, .012]
Feedback x Depression	6.12	(1, 985)	.014	.006	[.000, .020]
Risk x Worry	1.12	(2, 985)	.326	.002	[.000, .013]
Feedback x Worry	0.37	(1, 985)	.545	.000	[.000, .007]
Risk x Feedback x P-IU	0.01	(2, 985)	.993	.000	[.000, .008]
Risk x Feedback x I-IU	0.20	(2, 985)	.820	.000	[.000, .009]
Risk x Feedback x Anxiety	0.13	(2, 985)	.881	.000	[.000, .008]
Risk x Feedback x Depression	0.38	(2, 985)	.686	.001	[.000, .010]
Risk x Feedback x Worry	0.01	(2, 985)	.989	.000	[.000, .008]

Note. P-IU = Prospective IU; I-IU = Inhibitory IU. The R^2 values presented in this table represent semi-partial R^2 effect sizes (see Methods).

Discussion

Using the Doors Task, we examined the influence of intolerance of uncertainty (IU) on the electrocortical responses during reward processing under different levels of risk. Our results from a relatively large sample ($n = 203$) provide insights into the relationship between IU and reward processing. We found the expected effects of the task on feedback-related brain activity: more positive amplitudes after reward feedback compared to no-reward feedback (i.e., a reward positivity, RewP). Risk levels modulated these effects after reward feedback, where high risk trials were associated with enhanced positivity compared to ambiguous or low risk trials, suggesting a successful impact of our risk manipulation on the electrocortical level. Importantly, we found no relationship of total IU with the feedback-related brain activity, in line with Nelson et al. (2016) and Muir et al. (2021). Interestingly, scores of both IU subfactors were related to the amplitude of the brain activity after either feedback presentation. Specifically, higher prospective IU scores were related with an overall increased amplitude, while higher scores of the inhibitory IU scale predicted an overall decrease in feedback-related brain activity. This effect was also present when we included related internalizing traits (i.e., worry, depression, trait anxiety) into the model. In addition, higher

depressive symptoms were associated with an overall blunted electrocortical response in the no-reward feedback condition, but not in the reward condition, as after gains this effect lacked a clear direction. Similarly, trait anxiety showed a small effect on the electrocortical responses to each feedback type, however no clear direction can be distinguished for either feedback. In summary, we found no effect of IU on reward processing specifically, while its subfactors did modulate the overall brain response to feedback in general.

The lack of an impact of IU total score on the electrocortical responses to reward aligns with previous conclusions from our lab (Malbec et al., 2022, 2024 and *Chapters 2 and 3*) and collaborators (Carsten et al., 2025) regarding IU's influence on other cognitive processes. This highlights broader challenges in psychophysiology research exploring inter-individual differences: specifically, the difficulty in bridging different types of variables (e.g., questionnaire-based trait variables and neurobiological variables). Variables of the same type tend to correlate more reliably, whereas cross-type correlations are rarer (Bernoster et al., 2019). This difficulty is compounded by the fact that traits like IU often represent higher-order constructs derived from experience and behavior, making their direct association with specific neurobiological variables such as the RewP more complex (Brandt & Mueller, 2022; DeYoung & Gray, 2009). While this does not negate the potential for meaningful connections between such variables, it underscores the importance of cautious hypothesis formulation and interpretation. Researchers should mind the theoretical and methodological gaps that might hinder such associations (Fried & Nesse, 2015; Ioannidis, 2005), avoid overgeneralizations, and prioritize multimodal and cross-validated approaches to strengthen findings (e.g., Carmichael & Driver, 2021).

Previous research with the Doors Task (Muir et al., 2021; Nelson et al., 2016) also did not find a link between total IU and reward processing. Interestingly, this consistent lack of association has been observed even across distinct versions of the task employed in different studies. On the one hand, Nelson et al. (2016) used a version with an array of two doors, without presenting a probability cue for the trial, and using a sound tone sequence to emulate uncertain context. On the other hand, Muir et al. (2021) used also a two-door array, but presented cues that either signaled the certainty of the coming reward, or possible delays regarding the timing of the feedback presentation. Although these differences highlight the need for more direct replications to strengthen the reliability of the conclusion regarding the lack of IU's impact on feedback-related brain activity, their diversity also supports the generalizability of the observed non-effect. This aligns with the con-

cept of experimental replication emphasized by Hudson (2021). This framework suggests that replication efforts, focusing on the same primary research question as the original study, while making minor adjustments to certain elements of the experimental design or analysis, enhance the ability to generalize conclusions. In the same vein, Umemoto and Holroyd (2017) also did not find IU to be associated with the RewP using a probabilistic learning task, further demonstrating the lack of impact of IU's total score when the feedback is related with the content of a single trial (i.e., gambling tasks) and when the feedback has learning related implications within the task (i.e., probabilistic learning task).

In our study, the two IU subfactors showed opposite effects on overall amplitudes following feedback presentation. Higher P-IU scores were associated with increased amplitudes, whereas higher I-IU scores predicted decreased brain activity in response to both reward and non-reward feedback. These findings partially align with those of Nelson et al. (2016), who observed that P-IU was linked to a larger RewP, while I-IU was associated with an attenuated RewP. However, our study differs in that the IU subfactors did not significantly impact the amplitude differences between reward and no-reward feedback types, as reflected in the non-significant interactions between feedback type and either IU subfactor (when using the RewP as difference score, we do not replicate the subfactors associations found by Nelson et al., 2016, neither the subfactors main effects described in this study. See Supplementary Materials).

Several key differences between the studies may explain these results. Nelson et al. (2016) calculated the RewP as a difference potential between gain and loss trials, incorporating feedback type into the metric. In contrast, we used raw amplitude values to directly examine electrocortical responses to individual feedback conditions. Both studies included uncertainty, but the methods differed: Nelson et al. (2016) used unpredictable sound sequences, while we employed a visual probability cue and increased uncertainty by using four door options instead of two. Additionally, their negative feedback involved monetary loss, whereas ours indicated a lack of gain. Furthermore, Nelson et al. (2016) studied a smaller sample of 55 university students, while our sample included 203 participants. These differences may account for a shift in the observed effects of IU subfactors across studies, from feedback-specific (e.g., reward-related) responses to broader patterns of increased or decreased brain responses to feedback in general. Prospective IU seems to be associated with a general tendency to respond stronger to feedback, regardless of reward or non-reward. This can be interpreted as heightened sensitivity to feedback salience, in line with this

subfactor definition as a tendency to increase the cognitive engagement in the face of uncertainty (Berenbaum et al., 2008; Birrell et al., 2011). In contrast, Inhibitory IU was associated with attenuated neural responses to feedback, potentially reflecting motivational withdrawal or reduced engagement when faced with uncertainty. This profile is also consistent with the conceptualization of I-IU as a tendency toward functional inhibition in uncertain contexts (Berenbaum et al., 2008; Birrell et al., 2011). Diminished feedback responsivity in this context may reflect an avoidant processing style, in which external signals are downregulated or deprioritized as a coping strategy.

It is also worth noting that the effects of IU subfactors may extend beyond reward processing to other cognitive domains. For instance, an impact on threat learning has been reported where higher I-IU was associated with worse threat discrimination in skin conductance during acquisition, while the opposite was found for P-IU (Johnson et al., 2023); however, consistent effects are not always observed (e.g., Morriss, Zuj, & Mertens, 2021; Malbec et al., 2024 and *Chapter 3*). For responses to uncertain threats, diminished startle responses have been observed for individuals higher on I-IU, though this was not modulated by P-IU (Nelson & Shankman, 2011). Regarding uncertain rewards, higher levels of P-IU are linked to an over-activation of the anterior insula, which in turn is not influenced by I-IU (Gorka et al., 2016). Furthermore, opposite effects have been described for error processing: higher levels of P-IU are associated with larger error-related negativity (ERN), while higher I-IU is linked with a diminished amplitude of this ERP (Jackson et al., 2016); however, a study from our lab failed to observe the same effect (Malbec et al., 2022 and *Chapter 2*). Importantly, the limited number of studies and the presence of these mixed results highlight the need for further investigation into the precise impact of IU subfactors on cognition.

The distinct impact of IU subfactors on cognitive processes, such as reward processing observed in our study, warrants careful consideration in the context of how IU is measured and reported. Currently, the field demonstrates varied approaches to the analysis and presentation of IU scores. Some researchers have primarily reported the IU total score (e.g., Aslanidou et al., 2024; Morriss, Biagi, et al., 2021; Morriss, Goh, et al., 2023; Shapiro et al., 2020). Other studies have focused on the associations of the IU subfactors with variables of interest (e.g., Gorka et al., 2016; Jacoby et al., 2013; Saulnier et al., 2019). A third group of studies has presented analyses for both the total IU score and its subfactors (e.g., Cobos et al., 2024; Morriss, Wake, et al., 2021; Thibodeau et al., 2013; Yao et al., 2021). This latter approach aligns with recommendations from a review of Ta-

novic et al. (2018), which emphasize the utility of analyzing and reporting both the total score and subfactors to enhance clarity regarding IU's conceptualization and its empirical correlates. Consistent with these recommendations and the aim of providing a comprehensive picture, our analysis included both IU's total score and its subfactors.

Considering their current definitions (Birrell et al., 2011; McEvoy & Mahoney, 2011), both IU subfactors seem to reflect opposing aspects of the general IU construct: the prospective component (P-IU) is associated with active cognitive engagement in situations of uncertainty, while the inhibitory factor (I-IU) is linked to the suppression of actions when faced with uncertainty. This duality, however, can create interpretive challenges. For instance, individuals scoring high on both subscales—an expected outcome given their shared inclusion in the IU construct—may exhibit behaviors or electrocortical responses influenced by both subscales in opposing directions, complicating the interpretation of results. Therefore, we urge caution when presented with opposing effects of the subfactors, both in interpreting such results and in generalizing their conclusions. Future studies interested in subfactor-specific effects should carefully differentiate individuals based on their scores for each subscale, such as subgroup analyses, to further clarify these dynamics.

When we included related internalizing traits in our exploratory analysis, several notable findings emerged. First, the former opposing effects of the IU subfactors persisted. This means that the influence of IU subfactors on the amplitudes to both feedback types is independent of the levels of depression, trait anxiety, and worry. Second, both trait anxiety and depression levels influenced the different responses to reward or no-reward outcomes. Higher presence of depressive symptoms was linked with more negative amplitudes after no-reward feedback, but not after reward feedback, aligning with prior evidence of blunted electrocortical responses in depression (Forbes & Dahl, 2005; Proudfit, 2015). Trait anxiety showed a modest but distinct trend; however, no direction can be clearly interpreted since for both types of feedback the confidence intervals included zero. These nuanced effects underscore the complexity of internalizing traits in shaping electrocortical feedback responses.

The differences in brain activity associated with both types of feedback in our study were influenced by the level of risk involved. This effect was particularly evident following reward feedback, where amplitudes were larger for high-risk trials compared to both ambiguous and low-risk trials (see Fig. 3). Previous research found that less-likely outcomes evoke larger brain responses (Hajcak et

al., 2007; Holroyd et al., 2003; Holroyd & Coles, 2002; Sambrook & Goslin, 2015). Our results show that this effect occurs solely in reward compared to no-rewards outcomes. Secondly, we were able to disentangle this effect by showing that enhanced amplitudes are particularly associated with high-risk rather than ambiguous cues. Both risk and ambiguity play a role in the subjective expectations of an outcome, however they differ by the role of the unknown on each of them. In risk, even if the information is incomplete, the individual has a defined idea of the chances of occurrence of a determined future outcome (this is referred to as *first order uncertainty*). However, in the case of ambiguity, the imprecision about the chances of a certain outcome brings another level of how the unknown is perceived, this is referred to as *second order uncertainty* (Bach et al., 2011). Importantly, we have to consider that based on our design reward feedback under the high-risk condition was presented fewer times compared to ambiguous and low-risk rewards, and the less frequent feedback is related with larger amplitudes (Holroyd et al., 2003).

Our results should be interpreted considering the study limitations and strengths. First, as a strength, our study's sample size exceeds those used in previous research of this type, enhancing the reliability of our results and reducing the likelihood of Type I errors in our conclusions. Second, the data collection was carried out during the COVID-19 pandemic, this context could have influenced the reactions to uncertain or risky rewards, as well as beliefs around uncertainty (e.g., Korte et al., 2022; Segovia et al., 2024; Tanir et al., 2020). Third, while the differences between our design and those of previous studies (Muir et al., 2021; Nelson et al., 2016) offer a broader perspective for validating some of the effects previously reported, they also highlight the need for more direct replication efforts. Such replications are crucial to further clarify the role of IU and its subfactors in reward processing. Fourth, the length of the task was perceived by some participants as excessive. While the extended task provided a large number of trials, thereby increasing the reliability of the calculated ERPs (Luck et al., 2021), it may have also introduced fatigue which may influence the valuation of rewards and engagement in the task (Boksem & Tops, 2008). Future studies should account for this by carefully balancing the number of trials needed for reliable ERP estimation with considerations of participant fatigue and calculate power requirements accordingly. Fifth, the monetary reward provided may have elicited varied responses among participants. While some verbally reported indifference toward receiving it, others expressed urgency for prompt payment, suggesting diverse motivational and emotional reactions to the reward. This can

be related with inter-individual differences in reward sensitivity (Rosell-Negre et al., 2017), as well as with each participant's financial situation. Sixth, we acknowledge that our participant sample is diverse in terms of country of origin, language, and culture and that this may be different in other studies. Differences in sample characteristics have the potential to influence the results and their comparison with other studies (e.g., Pavlenko, 2012; Lewis et al., 2008; Na & Kitayama, 2011). However, we do not expect the diversity of our sample to influence the comparison with the study by Nelson et al. (2016), because they also describe their sample as diverse. Sample composition has not been clearly described in all studies, so future studies should clearly report the demographic information of their sample to allow for comparisons.

In conclusion, our study further corroborates findings of a lack of influence of total IU on reward processing while highlighting the distinct and opposing effects of its subfactors on overall brain responses to feedback. Specifically, prospective IU was associated with increased feedback-related brain activity, whereas inhibitory IU showed the opposite trend. This duality underscores the importance of understanding and reporting IU related results both as a unitary construct (i.e., total IU score) and as a multidimensional construct (i.e., P-IU and I-IU subfactors). Additionally, depression and trait anxiety modestly influenced differential responses to reward and no-reward feedback, reflecting the nuanced interplay between mental health factors and neural mechanisms of reward processing.

These findings contribute to a deeper understanding of the role of IU and related traits in reward processing, with implications for both theoretical models and clinical approaches. The impact of IU on reward processing should therefore not be considered solely as the impact of uncertainty as a whole on this cognitive process, but include the specific manner that uncertainty impacts each individual (i.e., through heightened engagement or avoidance when faced with uncertain situations).

Supplementary Material

This and other Supplementary materials are available at <https://osf.io/gkymz/>. Other documents include:

1. Histograms of the continuous variables (for visual inspection of distributions).
2. Correlation analyses among the continuous variables.
3. Publicly available datasets (behavioral, EEG-derived, and questionnaires).
4. A data dictionary describing each variable in the dataset, including coding and definitions.
5. Session information files detailing the R version and package versions used in the analyses.
6. Code used for data wrangling and statistical analysis.

Raw EEG repository address: <https://doi.org/10.5281/zenodo.17099892>

Analysis of the RewP as a difference score

To calculate the RewP, we used the same preprocessing steps as for the post-feedback ERP in our main analysis (see Methods). However, for this analysis, we: 1) extracted the average activity from a 230–330 ms time window; 2) focused on the same fronto-central pooled electrodes (Cz and Fz); and 3) calculated a difference wave by subtracting the ERP signal in no-reward trials from that in reward trials.

We used the RewP in equivalent statistical models to our main analysis (linear mixed models), now removing the factor feedback from them. This analysis replicates our primary finding that risk level modulates the RewP (see Supplementary Table 1-3). Specifically, we observed that high-risk trials ($M = 1.56 \mu\text{V}$, $SD = 1.59$) elicited a larger RewP than both ambiguous ($M = 0.91 \mu\text{V}$, $SD = 1.60$; $t(404) = -4.14$, $p < .001$) and low-risk trials ($M = 0.28 \mu\text{V}$, $SD = 1.60$; $t(404) = 8.06$, $p < .001$). Furthermore, the RewP amplitude was greater for ambiguous trials than for low-risk trials ($t(404) = 3.92$, $p < .001$).

Supplementary Table 1.

Linear mixed model analyses for the RewP using total IU.

Factor(s)	<i>F</i> -value	<i>df</i>	<i>p</i> -value	<i>R</i> ²	95% CI
Risk	39.49	(2, 402)	< .001	0.164	[.107, .233]
IU	2.24	(1, 201)	.136	0.011	[.000, .057]
Risk x IU	0.59	(2, 402)	.552	0.003	[.000, .027]

Note: IU = Intolerance of uncertainty. The *R*² values presented in this table represent semi-partial *R*² effect sizes (see Methods).

Supplementary Table 2.

Linear mixed model analyses for the RewP using prospective and inhibitory IU instead of total IU.

Factor(s)	<i>F</i> -value	<i>df</i>	<i>p</i> -value	<i>R</i> ²	95% CI
Risk	39.32	(2, 400)	< .001	.164	[.107, .233]
P-IU	2.53	(1, 200)	.113	.012	[.000, .060]
I-IU	0.15	(1, 200)	.697	.001	[.000, .028]
Risk x P-IU	0.06	(2, 400)	.941	.000	[.000, .019]
Risk x I-IU	0.34	(2, 400)	.713	.002	[.000, .024]

Note. P-IU = Prospective IU; I-IU = Inhibitory IU. The *R*² values presented in this table represent semi-partial *R*² effect sizes (see Methods).

Supplementary Table 3.

Linear mixed model analyses for the RewP using IU subfactors, trait anxiety, depression, and worry.

Factor(s)	<i>F</i> -value	<i>df</i>	<i>p</i> -value	<i>R</i> ²	95% CI
Risk	39.20	(2, 394)	< .001	.166	[.108, .236]
P-IU	2.17	(1, 197)	.142	.011	[.000, .058]
I-IU	0.53	(1, 197)	.466	.003	[.000, .036]
Anxiety	0.90	(1, 197)	.344	.005	[.000, .042]
Depression	0.72	(1, 197)	.397	.004	[.000, .039]
Worry	0.87	(1, 197)	.353	.004	[.000, .041]
Risk x P-IU	0.04	(2, 394)	.963	.000	[.000, .019]
Risk x I-IU	0.67	(2, 394)	.513	.003	[.000, .028]
Risk x Anxiety	0.23	(2, 394)	.797	.001	[.000, .022]
Risk x Depression	1.07	(2, 394)	.345	.005	[.000, .033]
Risk x Worry	0.38	(2, 394)	.687	.002	[.000, .024]

Note. P-IU = Prospective IU; I-IU = Inhibitory IU. The *R*² values presented in this table represent semi-partial *R*² effect sizes (see Methods).

Chapter 5



Profiles across error, fear, and reward processing:
Associations of neurocognitive profiles
with intolerance of uncertainty

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Abstract

Intolerance of uncertainty (IU) has been linked to aberrant responding across threat, performance monitoring, and reward processing. We used a person-centered approach to identify joint neurocognitive response profiles across three laboratory tasks and examine their associations with IU and related symptoms. Participants ($N = 214$) completed a Flanker task, a Pavlovian fear-conditioning and extinction task, and a Doors reward task. Indicators spanned behavioral performance, electrophysiology, and peripheral physiology. Latent profile analysis compared profile solutions using standard fit criteria, yielding a best-fitting three-profile solution. It reflected broad differences in threat and performance reactivity versus feedback reactivity, alongside an average-reactivity group. Exploratory analysis tested differences on IU scores (total, prospective, inhibitory), worry, trait and state anxiety, and depressive symptoms. Profiles showed minimal associations between neurocognitive profile membership and internalizing measures. An omnibus effect for state anxiety emerged, with exploratory pairwise differences indicating higher state anxiety in the high threat and performance reactivity, low feedback reactivity profile relative to the average group; these contrasts did not remain after Holm correction. No omnibus effects were observed for IU or the other questionnaire scores. Findings demonstrate that multimodal indicators from performance monitoring, threat and safety learning, and reward processing can be combined into interpretable person-level profiles. Although associations with internalizing traits were limited, the profiles offer a data-driven framework for hypothesis generation and future work on clinical prediction and treatment personalization.

Keywords: latent profile analysis; intolerance of uncertainty; performance monitoring; fear conditioning; reward processing; psychophysiology; EEG; error.

Introduction

Intolerance of uncertainty (IU) is a dispositional tendency to find uncertainty aversive and difficult to tolerate (Carleton, 2016). IU has been proposed as a transdiagnostic feature of internalizing psychopathology (Carleton et al., 2012; Mahoney & McEvoy, 2012b). Additionally, IU has been divided into two subfactors that may influence the different ways individuals react to uncertain elements: Prospective IU (P-IU), which reflects a preference for predictability and active certainty-seeking, and Inhibitory IU (I-IU), which is characterized by avoidance, behavioral inhibition, and paralysis in the face of uncertainty (Berenbaum et al., 2008; Carleton, 2012). IU is also linked to cognition under uncertainty, including decision making and the anticipation of possible threat (Grupe & Nitschke, 2013), and to altered responding to reward outcomes (Gorka et al., 2016; Nelson et al., 2016). Building on these links, the present study focuses on three neurocognitive domains that are relevant to IU: performance monitoring, fear and extinction learning, and reward processing.

In performance monitoring, errors can be perceived as internal threats that elicit defensive responses. This process is indexed by the error-related negativity (ERN), an event-related potential (ERP) occurring 50-100 ms after an erroneous response (Gehring et al., 1993). Although studies have found an association between heightened ERN amplitudes and anxiety-related traits (Weinberg et al., 2015), the role of IU in modulating this relationship remains uncertain. Some studies suggest that individuals high in P-IU exhibit an enhanced ERN, indicating increased sensitivity to errors, while those high in I-IU may show attenuated ERN responses (Jackson et al., 2016). However, other studies have failed to replicate associations of ERN and IU (Carsten et al., 2025; Malbec et al., 2022 and *Chapter 2*; Ruchensky et al., 2020; Sandre & Weinberg, 2019), underscoring the value of integrating performance monitoring with threat and reward domains in a person-centered analysis.

Similarly, IU may influence fear acquisition and extinction processes. Fear conditioning tasks typically include habituation, acquisition, and extinction phases. During habituation, all cues are presented without the aversive outcome to establish baseline responding and reduce novelty effects. During acquisition, a threat-signaling cue (CS+) is repeatedly paired with the aversive outcome, whereas a safe cue (CS-) is never paired, producing greater responding to the CS+ than the CS-. During extinction, both cues are presented without the aversive outcome to reduce conditioned responding to the CS+ (Lonsdorf et al., 2017). Experimental fear conditioning studies have demonstrated that individuals with high IU exhibit exag-

gerated fear responses to uncertain threats and show resistance to fear extinction (Morriss, Wake, et al., 2021). The persistence of fear responses, particularly during extinction, suggests that individuals with high IU may struggle to update their threat expectations in changing environments. This effect has particularly been observed in heightened skin conductance responses (SCR) toward safe stimulus throughout extinction (Morriss, Wake, et al., 2021). SCR reflects activity in the autonomous nervous system, and it has been used to differentiate the emotional responses to stimuli (Boucsein et al., 2012). While evidence for IU effects during fear acquisition is mixed, higher IU may be linked to stronger fear responding during acquisition when threat–safety contingencies are uncertain (Johnson et al., 2023; Morriss, Zuj, & Mertens, 2021). In contrast, the impact of IU on the extinction phase seems clearer, specifically higher IU scores are often linked with a pattern of generalized responses to all stimuli early in extinction. Then later in this phase, they show a continued persistence in differentiating between safety and threat cues, which contrasts with the typical reduction in this discrimination observed in low IU individuals (for a review: Morriss, Zuj, & Mertens, 2021; for a meta-analysis on extinction: Morriss, Wake, et al., 2021). However, our own experiment, from which data is also analyzed in the current paper, did not reveal any association between IU and neither fear acquisition nor extinction (Malbec et al., 2024 and *Chapter 3*).

Reward processing involves evaluating outcomes relative to expectations, a computation shaped by the reward prediction error, defined as the difference between expected and received results (Schultz, 2016). A common electro-cortical index for this process is the reward positivity (RewP), an ERP observed approximately 250 to 350 ms after feedback; it is often computed as a gain minus loss or no-gain difference (Holroyd et al., 2008; Proudfit, 2015). A blunted RewP has been associated with depression (Bress et al., 2012; Liu et al., 2014), whereas larger RewP amplitudes have been reported in social anxiety (Nelson & Jarcho, 2021). Evidence for IU is mixed: some work suggests opposite associations for its subfactors, with I-IU relating to attenuated RewP and P-IU to enhanced RewP (Nelson et al., 2016), while other studies report no reliable associations (Muir et al., 2021). Reviews also highlight limited evidence on how IU manifests in reward-guided choice behavior and decision-making, underscoring the need for integrative approaches (Shihata et al., 2016). Beyond variable-centered effects, person-centered work has begun to parse reward-responsiveness profiles. Park and colleagues (2023) identified three latent profiles in EEG and self-report measures spanning monetary, food, social, and erotic reward: a blunted neural/self-report profile linked to anhedonic depression and social dysfunction, a monetary-sensitive neural profile, and

a high self-report/variable neural profile associated with risk-related tendencies. These findings show that qualitatively distinct reward-processing patterns map onto different clinical correlates, motivating a person-centered approach for reward systems (Park et al., 2023). Methodologically, some authors recommend modeling neural responses to positive and negative feedback separately, not only as difference scores (Clayson et al., 2021; Fouragnan et al., 2015; Gheza et al., 2018). In our own study, which data is also included in the current paper, we observed that IU subfactors related to overall feedback-locked activity irrespective of valence, with higher P-IU showing larger overall responses and higher I-IU showing smaller overall responses (Malbec et al., 2025 and *Chapter 4*).

Evidence across performance monitoring, fear learning, and reward processing shows both convergences and inconsistencies in how IU relates to neural and behavioral responses. In light of IU's transdiagnostic features, its role across these related cognitive processes remains insufficiently understood. Moreover, all of these processes and their link to IU have been studied mostly in isolation and in relatively small samples (e.g., Jackson et al., 2016; Morriss et al., 2016; Nelson et al., 2016). Clarifying transdiagnostic mechanisms requires a joint, multimodal evaluation of these processes and their links with IU. Person-centered, data-driven approaches such as Latent Profile Analysis (LPA) can integrate central, peripheral, behavioral, and rating indicators on a common scale to recover clinically meaningful subgroups and test their correlates, revealing patterns that variable-centered averages may miss (Ferguson et al., 2020; Park et al., 2023; Park & Naragon-Gainey, 2024; Verdi et al., 2021).

LPA is a clustering approach that groups individuals by similarity across continuous indicators and estimates posterior probabilities of class membership (Ferguson et al., 2020). This makes it well suited to multimodal data because indicators can come from heterogeneous sources and scales, for example central electrophysiology, peripheral physiology, behavior, and ratings, after standardization places them on a common metric. Profiles capture concordant as well as discordant patterns across modalities, and membership can be related to external variables while accounting for classification uncertainty. In practice, class enumeration balances fit indices (e.g., BIC, SSABIC), likelihood-ratio tests (e.g., LMR, BLRT), entropy, and interpretability with attention to avoiding over extraction and ensuring adequate class sizes (Ferguson et al., 2020). Recent work also shows how profile solutions can map meaningfully onto theoretically relevant constructs, for instance, as emotion regulation ability profiles that differ in strategy use, goals, and internalizing symptoms (Park & Naragon-Gainey, 2024).

Applications in clinical and cognitive-affective research illustrate this flexibility. Studies have used LPA to combine EEG features with behavior to differentiate attentional or performance monitoring subtypes (e.g., Arnett & Flaherty, 2022), to derive profiles of learning responses across phases and modalities in conditioning paradigms (e.g., Kirlic et al., 2022), and to integrate behavioral, physiological, and self-report indicators when characterizing risk or symptom patterns, including self-injury and broader mental health stratification (e.g., Chen et al., 2025). Across these contexts, person-centered profiling recovered subgroups that variable-centered averages obscured, and those subgroups showed distinct associations with clinically relevant correlates, paralleling our aim to profile cross-domain responding and examine its links with IU (Ferguson et al., 2020; Park et al., 2023; Park & Naragon-Gainey, 2024).

The present study examines how IU relates to performance monitoring, fear and extinction learning, and reward processing by combining behavioral, central electrophysiological, and peripheral physiological data across these paradigms in a single multimodal analysis. The experimental paradigms used here (described below) were not primarily designed to maximize participants' experience of uncertainty; however, each contains uncertainty-relevant features (see Discussion). Using LPA, we estimated person-level profiles defined by similarity across indicators within and across tasks, allowing profiles to reflect both concordant patterns (for example, broadly heightened responding) and discordant patterns (for example, stronger neural signals with muted behavior). We then evaluated whether IU total, prospective IU and inhibitory IU, as well as worry, anxiety, and depression symptoms, differentiate these profiles. This approach clarifies whether IU helps organize cross-domain neurocognitive responding into a limited set of neurocognitive profiles.

Methods

Participants

This study involved secondary data analysis, results of the three separate experimental tasks were presented in previous publications (Malbec et al., 2022, 2024, 2025) and included in this dissertation in *Chapters 2-4*.

A total of 214 undergraduate psychology students (80.2% female, age: $M = 21.16$ years, $SD = 3.18$, range = 17–46 years,) from Erasmus University Rotterdam participated in the study, recruited via an internal online system. Most par-

ticipants identified as White (72.4 %), followed by Asian (8.9 %), Black (3.7 %), Middle-Eastern (4.2 %), Mixed (3.3 %), Latin-American (1.9 %), and Other (3.7 %). Roughly half (49 %) completed the Dutch-language survey, the remainder the English version. Participants received course credit and monetary compensation of €5 as part of the Doors Task. The study was approved by the Erasmus School of Behavioural and Social Sciences Ethics Board and conducted in accordance with the Declaration of Helsinki. A-priori exclusion criteria included a family history of photic epilepsy due to the flickering stimuli used in one of the tasks.

In the original studies (Malbec et al., 2022, 2024, 2025), if data of one variable type (e.g. behavior, EEG, or SCR) was missing or corrupted, that participant was removed from the final sample (i.e., list-wise deletion). Here, to retain as much data as possible, missingness was handled using full-information maximum likelihood (FIML), which uses all available observations without imputation (Hartley & Hocking, 1971). No participant had missing data in all experiments and variable types. Therefore, data from all 214 participants were considered for the main analysis.

Self-report Questionnaires

Following the experimental tasks, participants completed the following self-report questionnaires in Dutch or English, depending on their language preference.

IU was assessed with the 12-item Intolerance of Uncertainty Scale (IUS-12; Carleton et al., 2007), the short form of the original IUS (Buhr & Dugas, 2002; Freeston et al., 1994; Dutch translation: de Bruin et al., 2006). Two subfactors are extracted: Prospective IU (P-IU; desire for predictability and active certainty-seeking) and Inhibitory IU (I-IU; avoidance and behavioral freezing under uncertainty). Items are rated from 1 (“not at all characteristic of me”) to 5 (“entirely characteristic of me”).

Depressive symptoms were measured with the Beck Depression Inventory–II (BDI-II; Beck et al., 1996; Dutch translation: Van der Does, 2002). The 21 items are rated 0–3 to reflect symptom severity over the past two weeks.

Worry was measured with the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990; Dutch translation: van Rijsoort et al., 1999). The 16 items are rated from 1 (“not at all typical of me”) to 5 (“very typical of me”).

Trait and state anxiety were assessed with the State–Trait Anxiety Inventory, Form Y (STAI; Spielberger, 1983; Dutch translation: Van der Ploeg, 2000). Trait anxiety (STAI-T) indexes general propensity for anxiety across 20 items rated

from 1 (“almost never”) to 4 (“almost always”). State anxiety (STAI-S) captures momentary anxiety “right now” with 20 items rated from 1 (“not at all”) to 4 (“very much so”).

Internal consistency values ranged from $\alpha = 0.78$ to $\alpha = 0.94$, demonstrating good reliability, see Table 1.

Table 1

Internal consistency of the questionnaires by language

Measure	Items	α (Overall)	α (English)	α (Dutch)
IUS-12	12	0.87	0.91	0.79
P-IU	7	0.82	0.88	0.73
I-IU	5	0.78	0.84	0.69
BDI-II	21	0.94	0.94	0.94
STAI-S	20	0.84	0.84	0.84
STAI-T	20	0.80	0.85	0.75
PSWQ	16	0.83	0.81	0.84

Note. α = Cronbach’s alpha (raw). IUS-12 = Intolerance of Uncertainty Scale (12-item version); P-IU = Prospective IU; I-IU = Inhibitory IU; BDI-II = Beck Depression Inventory–II; STAI-S = State Anxiety Inventory; STAI-T = Trait Anxiety Inventory; PSWQ = Penn State Worry Questionnaire. Overall $N = 212$, English $N = 103$, Dutch $N = 109$.

Table 2

Descriptive Statistics for Self-Report Measures of Internalizing Symptoms

Measure	M	SD	Sample Range	Possible Range
IU (IUS-12)	29.40	8.32	12 – 51	12 – 60
Prospective IU	18.24	5.35	7 – 31	7 – 35
Inhibitory IU	11.16	3.78	5 – 20	5 – 25
Depressive Symptoms (BDI-II)	5.92	7.67	0 – 34	0 – 63
State Anxiety (STAI-State)	41.43	8.27	20 – 67	20 – 80
Trait Anxiety (STAI-Trait)	45.09	8.24	24 – 66	20 – 80
Worry (PSWQ)	44.96	10.70	25 – 72	16 – 80

Note. IUS-12 = Intolerance of Uncertainty Scale (12-item version); BDI-II = Beck Depression Inventory–II; STAI = State-Trait Anxiety Inventory; PSWQ = Penn State Worry Questionnaire.

Experimental Tasks and Materials

A full account of the task procedures and EEG protocols is available in Malbec et al. (2022, 2024, 2025) and *Chapters 2-4* of this dissertation. Briefly, participants completed three EEG tasks during a single lab session, always presented in the same order: Doors Task, Fear Conditioning, and Flanker Task.

Flanker Task (Performance Monitoring).

The Eriksen Flanker Task assessed neural responses to response conflict and errors (Eriksen & Eriksen, 1974). On each trial, participants viewed a string of five letters (e.g., HHHHH or SSHSS) for 50 ms and responded to the central letter (“H” or “S”) using designated keys. Each trial began with a fixation cross (150 ms), followed by a blank screen (100-150 ms), then the letter array (50 ms). Feedback on accuracy appeared for 500 ms, followed by a 500 ms inter-trial interval. The task consisted of 400 trials in total, with congruent and incongruent strings randomly intermixed (Malbec et al., 2022).

Fear Conditioning Task (Fear Learning and Extinction).

This task investigated the acquisition and extinction of conditioned fear using a differential fear conditioning design. It included three phases: Habituation, CS+ and CS- each presented 10 times without reinforcement; Acquisition: CS+ paired with a 90 dB female scream (US) on 75% of trials; CS- was never reinforced; Extinction: CS+ and CS- presented without the US to assess reduction in defensive responses.

The conditioned stimuli were grayscale images of neutral female faces (NimStim Set), displayed for 5 seconds at a flickering rate of 15 Hz to evoke steady-state visual evoked potentials (ssVEPs). Inter-trial intervals varied between 4.5 and 5 seconds. Stimulus-US pairing was counterbalanced across participants.

Doors Task (Reward Processing).

This gambling paradigm assessed neural responses to feedback under different levels of reward uncertainty (Hajcak et al., 2007; Paul & Pourtois, 2017). Each trial began with a fixation dot (700 ms), followed by a 600 ms cue indicating the probability of winning: High risk (25% chance; one of four doors contained a reward); Low risk (75% chance; three of four doors contained a reward); Ambiguous risk (unknown probability; static black-and-white pattern).

After a 1500 ms delay, four identical doors were displayed, and participants selected one by pressing keys 1-4. Feedback appeared for 1000 ms as either a green cross (reward, +€0.025) or a red circle (no reward). The task included four blocks of 92 trials (368 in total). Risk probabilities were fixed across participants,

and all earned €5. EEG was continuously recorded, and electrocortical responses to feedback were extracted. The primary measure was a frontocentral ERP in the 230-280 ms window, reflecting reward processing (Malbec et al., 2025).

Procedures

Upon arrival at the laboratory, participants were provided with a verbal overview of the study and signed a written informed consent form. They completed self-report questionnaires via Qualtrics survey (details provided below) before completing three electroencephalogram (EEG) tasks. They were then seated in an isolated, dimly lit room, where EEG and electrodermal activity (EDA) electrodes were attached. The experimental session consisted of three main tasks presented in the following fixed order: a reward-processing task (Doors task; ~40 minutes), a differential fear-conditioning and extinction task (~25 minutes), and a performance-monitoring task (Flanker task; ~15 minutes). Self-administered breaks were provided between tasks. After completing the experimental tasks, EEG and psychophysiological sensors were removed, and participants were directed to a computer to complete the self-report questionnaires. The entire session lasted approximately 90 minutes. Upon completion, participants were debriefed, compensated, and thanked.

EEG and Psychophysiology Recording and Data Processing

General Acquisition Procedures

Continuous EEG data were collected across all three tasks using a BioSemi ActiveTwo amplifier (Amsterdam, Netherlands) with a 32-channel Ag/AgCl electrode cap arranged according to the international 10/20 electrode placement system. Four external electrodes recorded the electro-oculogram (EOG; vertical: above/below left eye; horizontal: outer canthi). EEG was digitized continuously at a sampling rate of 512 Hz (24-bit A/D conversion) with online referencing to Common Mode Sense (CMS) and grounding via Driven Right Leg (DRL). Electrodermal activity (EDA) was simultaneously recorded from the middle and ring fingers at the intermediate phalanges of the non-dominant hand using flat electrodes and a 0.05M NaCl electrolyte gel.

General EEG Preprocessing Steps

Preprocessing of EEG data was conducted offline using BrainVision Analyzer (BrainProducts, Gilching, Germany). The following steps were applied consistently across tasks: continuous data were re-referenced to the average of all scalp electrodes, and a 50 Hz notch filter removed line noise. Data were then band-pass

filtered with task-specific frequency ranges (specified below). Ocular activity was corrected using the Gratton and Coles (1983) procedure, after which data were segmented into task-specific epochs. Trials were rejected based on task-specific thresholds for amplitude, gradient/step, signal difference, and low activity, as detailed in the task sections. Baseline correction was applied where appropriate according to the analytic approach of each paradigm.

Flanker Task (Performance Monitoring)

EEG data were segmented into epochs spanning -400 to +800 ms relative to each participant's response. Baseline correction was applied using the -400 to -200 ms window prior to the response. Artifact rejection criteria included an amplitude threshold of $\pm 75 \mu\text{V}$, gradient of $\pm 50 \mu\text{V/ms}$, a signal difference of $200 \mu\text{V}$ in 200 ms, and a low activity criterion of $2 \mu\text{V}$ over 200 ms. Data were band-pass filtered from 0.1 to 35 Hz. The primary ERP components analyzed were the ERN and the CRN, calculated at electrode Cz within 0-100 ms post-response. Additionally, the error positivity (Pe) and the correct positivity (Pc) were extracted from 200-400 ms at both Cz and Pz (Malbec et al., 2022).

Fear Conditioning Task (Fear Acquisition and Extinction)

EEG data were segmented from -500 to +5500 ms relative to stimulus onset. Artifact rejection followed used the following criteria: $\pm 200 \mu\text{V}$ amplitude, $\pm 50 \mu\text{V/ms}$ gradient, $100 \mu\text{V}$ signal difference, and $0.5 \mu\text{V}$ low activity over 100 ms. The data were band-pass filtered from 0.1 to 40 Hz. For ssVEP analysis, Fast-Fourier-Transformation (FFT) was performed on the final 2000 ms of each trial, and amplitudes were averaged across electrodes O1, Oz, and O2. For late positive potential (LPP) analysis, data were additionally band-stop filtered around the flicker frequency (i.e., 15 Hz), and baseline correction was applied using the 200 ms preceding stimulus onset. The LPP was quantified at electrode Pz within 400-1000 ms post-response (Malbec et al., 2024). SCR was calculated from the recorded EDA data by segmenting from -1000 to +9000 ms relative to stimulus onset and scored using a manual foot-to-peak method. Identifying the foot within the 800-4000 ms window and a valid peak in the rest of the trial segment (Malbec et al., 2024).

Doors Task (Reward Processing)

EEG data were segmented from -900 to +1600 ms relative to feedback onset. Baseline correction was performed using the 250 ms preceding feedback presentation. Artifact rejection criteria matched those of the Flanker task: amplitude threshold of $\pm 75 \mu\text{V}$, gradient of $\pm 50 \mu\text{V/ms}$, difference of $200 \mu\text{V}$, and low activity threshold of $2 \mu\text{V}$ over 200 ms. The data were filtered from 0.1 to 30 Hz. The pri-

mary neural measure was the feedback-locked ERP in the 230-280 ms window, averaged across Fz and Cz electrodes. ERP amplitudes were computed separately for reward and no-reward outcomes and for the three levels of risk (high, low, and ambiguous). To retain condition-specific variance, ERP amplitudes were modeled per condition rather than through difference scores (Malbec et al., 2025).

Statistical Analyses

To distinguish different latent classes, we conducted latent profile analysis (LPA) in Mplus version 8.11 using robust maximum likelihood (MLR); missing data were handled using FIML within this ML-based framework. This person-centered analysis method identifies latent subgroups based on similarity in participants' patterns across continuous indicators (Nylund-Gibson & Choi, 2018).

Model selection was based on a holistic evaluation of the profile solutions (from one to four in this study; see Table 3), integrating statistical fit, classification quality, and substantive interpretability. Consistent with methodological recommendations, we emphasized reductions in the Bayesian Information Criterion (BIC) and sample-size-adjusted BIC (SABIC) while treating improvements as relative rather than relying on fixed cutoffs (Ferguson et al., 2020). Entropy values were used as supporting evidence of classification precision, with values $\geq .80$ indicating low classification uncertainty (Park & Naragon-Gainey, 2024). Likelihood-based tests, including the Lo-Mendell-Rubin adjusted likelihood-ratio test (LMR) and the parametric bootstrap likelihood-ratio test (BLRT), were evaluated in conjunction with these indices, acknowledging that LMR may underdetect meaningful class separations in complex, multidimensional data (Nylund-Gibson & Choi, 2018) and that fit statistics often continue to improve with the addition of redundant classes (Ferguson et al., 2020).

The variables entered in the LPA comprised behavioral, electrophysiological, and psychophysiological indices derived from the three experimental paradigms. Indicators were not standardized or otherwise transformed prior to LPA; however, some indicators were computed as difference scores to index condition-specific effects. However, for visualization purposes, profile means are presented as z-scores in Figure 1. From the Flanker task, ten behavioral and electrophysiological measures were used, including accuracy, reaction times, and ERP components reflecting error monitoring (ERN, CRN, Pe, Pc, and their difference scores). The Fear Conditioning task contributed subjective ratings (expectancy, valence, arousal), physiological indices (SCRs), and EEG measures (ssVEPs, LPPs), each computed for conditioned threat (CS+) and safety (CS-) cues during acquisition

and extinction, as well as their differential scores. From the Doors task, ERP amplitudes were extracted for reward and no-reward feedback under high, ambiguous, and low risk, together with corresponding difference scores. Collectively a total of 49 indicators were used, these variables captured behavioral, neural, and physiological reactivity to errors, threats, and rewards across tasks.

After selecting the best model, we tested if the different profiles indeed varied on their uncertainty tolerance, cognitive and somatic depressive symptoms, worrying behavior, and anxiety tendencies. For this, we exported most-likely class membership and used it as a between-groups factor (three profiles) in R version 4.4.0 (R Core Team, 2024). For each questionnaire outcome (STAI-S, STAI-T, BDI-II, PSWQ, IUS-12 total, Prospective IU, Inhibitory IU), we ran one-way ANOVAs on raw scores. When the omnibus test indicated a profile effect, we conducted pairwise *t*-tests with Holm correction; uncorrected pairwise results are described as exploratory. Assumptions were checked with Levene's test for homogeneity of variances; normality was evaluated from residuals. All tests were two-tailed with $\alpha = .05$.

Results

Model Selection and Fit Evaluation

From the one-profile to the two-profile solution, information criteria improved and entropy was high (.957), but the two-profile model offered limited differentiation because one large class (82%) closely tracked the grand mean (Supplementary Figure 1). From the two-profile to the three-profile solution, AIC, BIC, and SABIC decreased substantially (−474.11, −305.81, −464.25), and entropy remained excellent (.949). The three-profile model yielded three coherent and well-sized classes (about 16%, 25%, 59%). Visual inspection of the model showed that each profile captured unique response patterns that were substantively informative (Figure 1). Moving from three to four profiles produced smaller incremental gains (AIC −303.01; BIC −134.71; SABIC −293.15), introduced a small additional class (11%) with limited distinctiveness (Supplementary Figure 2), and showed reduced estimation stability. For the four-profile model, the Mplus output (TECH14) indicated local maxima. In 4 of 5 bootstrap draws the best log-likelihood for the 4-class solution was not replicated, so the BLRT *p*-value is unreliable. Although LMR tests were non-significant for the two- versus three-profile ($p = .77$) and three- versus four-profile ($p = .50$) comparisons, prior work recommends

weighing these alongside information criteria, entropy, class sizes, and interpretability (Ferguson et al., 2020; Park et al., 2023; Park & Naragon-Gainey, 2024). Given diminishing returns beyond three profiles and superior interpretability and stability, the three-profile solution was retained.

Table 3
Model Fit Information and Profile Proportions

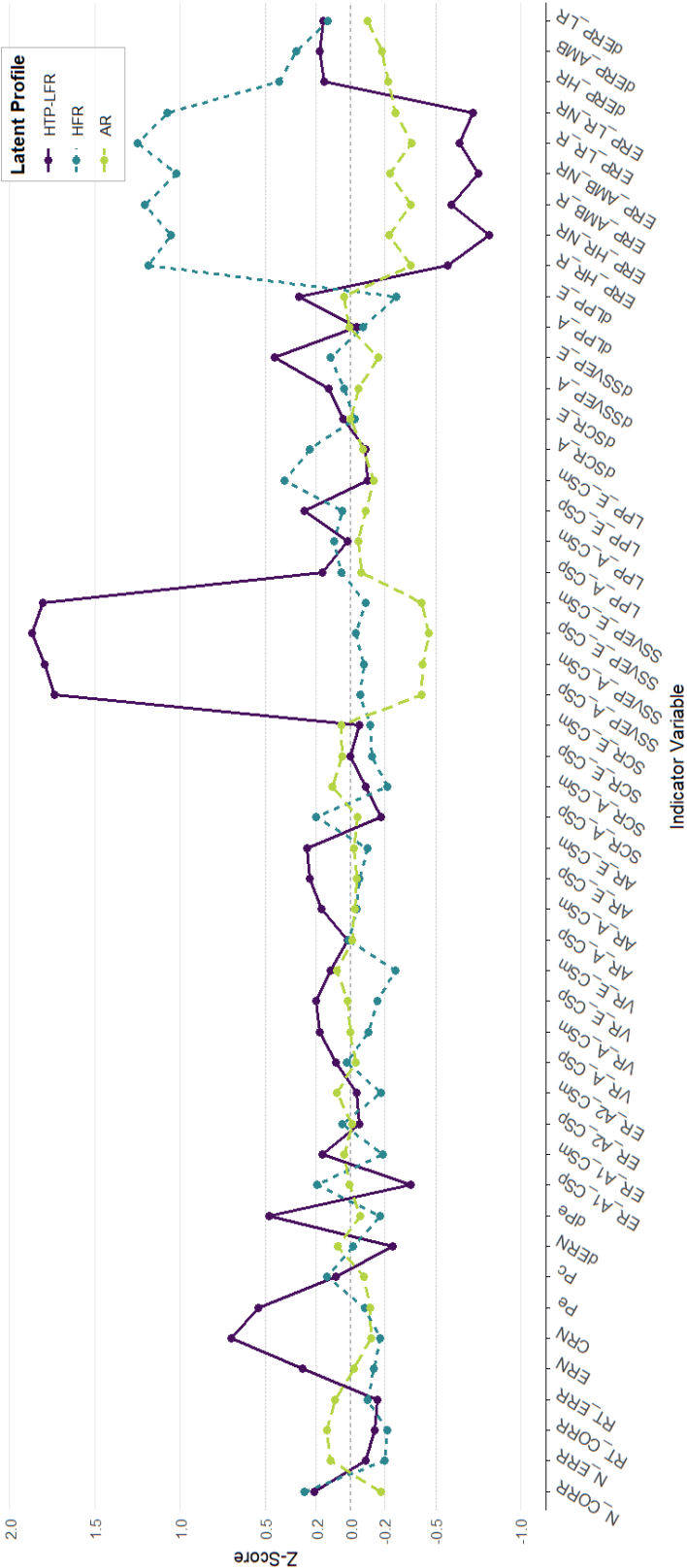
Model	npar	LL	AIC	BIC	SABIC	LMR (p)	BLRT (p)	Entropy	Smallest profile (n)	Profile proportions
1 profile	98	-21176.55	42549.10	42878.96	42568.42	-	-	-	214	1.00
2 profiles	148	-20827.135	41950.27	42448.43	41979.46	0.2377	<0.001	0.957	39	0.82, 0.18
3 profiles	198	-20540.08	41476.16	42142.62	41515.21	0.7695	<0.001	0.949	35	0.16, 0.25, 0.59
4 profiles	248	-20338.575	41173.15	42007.91	41222.06	0.5008	<0.001	0.952	24	0.18, 0.46, 0.24, 0.11

Note. The information of the best fitting model is shown in boldface. npar = number of parameters estimated; LL = log-likelihood; BIC = Bayes information criterion; SABIC = sample-size adjusted BIC; AIC = Akaike information criterion; LMR = Lo-Mendell-Rubin adjusted likelihood ratio test; BLRT = bootstrap likelihood-ratio test. BLRT *p*-value for the four-profile comparison is unreliable due to local maxima indicated by TECH14 (non-replication of best log-likelihood in 4 of 5 bootstrap draws).

Figure 1
Integrated Multimodal Model (3 profiles).

Integrated Multimodal Model (3 Profiles)

Standardized Profile Means (Z-scores)



Note. Lines depict standardized profile means (z scores). Z-scores were categorized based on standard criteria (Cohen, 1988; Sawilowsky, 2009): average ($|z| < 0.2$), above/below average ($0.2 < |z| < 0.5$), moderately high/low ($0.5 \leq |z| < 0.8$), and high/low ($|z| \geq 0.8$).

Profiles: HTP-LFR = High Threat/Performance Reactivity, Low Feedback Reactivity; HFR = High Feedback Reactivity; AR = Average Reactivity. **Flanker:** N_CORR = number of correct trials; N_ERR = number of error trials; RT_CORR and RT_ERR = mean reaction times; ERN, CRN, Pe, Pc = ERP components; dERN and dPe = error minus correct. **Fear conditioning:** ER = expectancy ratings; VR = valence ratings (Self-Assessment Manikin); AR = arousal ratings (Self-Assessment Manikin); SCR = skin conductance responses; SSVEP = steady-state visual evoked potentials; LPP = late positive potential; A/E = Acquisition/Extinction; CSp/CSm = CS+ / CS-; dSCR_A/E, dSSVEP_A/E, dLPP_A/E = difference of CSp minus CSm in the given phase and variable. **Doors:** ERP_HR/AMB/LR = ERPs for high-reward, ambiguous, and low-reward cues; R/NR = reward, no-reward; dERP_HR/AMB/LR = reward minus no-reward within each cue.

Profiles Description

After selecting the three-profile solution, we labeled the profiles based on their characteristic patterns across the three experiments. The descriptions below describe standardized means (z -scores) relative to the sample and indicate the proportion of participants assigned to each profile. Profile names reflect the dominant response features observed within each domain, providing a compact guide for interpreting the subsequent descriptions.

Profile 1 · High Threat and Performance - Low Feedback Reactivity (16% of sample)

This profile was primarily distinguished by marked physiological and cognitive sensitivity to threat-related stimuli and heightened performance monitoring, alongside reduced responsiveness to reward feedback. Individuals in this profile exhibited elevated ERN ($z = 0.28$), moderately elevated CRN ($z = 0.70$), and increased Pe to errors ($z = 0.54$) during performance monitoring. These neural indices collectively indicate enhanced sensitivity and heightened attention to performance monitoring. In the Fear Conditioning Task, this group showed very high ssVEP amplitudes to both conditioned threat (CS+) and safety (CS-) cues (z scores ranging from 1.74 to 1.87), suggesting sustained and generalized attentional orienting towards fear-related stimuli. Notably, this profile also demonstrated consistently blunted ERP responses to both reward and non-reward feedback across risk levels in the Doors Task ($z = -0.57$ to -0.82), highlighting diminished feedback sensitivity.

Profile 2 · High Feedback Reactivity (25% of sample)

Individuals in this profile were characterized predominantly by robust neural responsiveness to reward feedback, with average responding across other paradigms. In the Doors Task, they exhibited elevated ERP amplitudes across all feedback conditions ($z = 1.02$ to 1.25), these were slightly greater for rewarding conditions, indicating high neural sensitivity to reward. In contrast, their performance monitoring responses (Flanker Task) and physiological and behavioral responses during threat processing (Fear Conditioning Task) were average.

Profile 3 · Average Reactivity (59% of sample)

The largest group demonstrated generally average responding on performance-monitoring and fear-conditioning measures (most indicators within ± 0.2 SD of the sample mean). In contrast, feedback-related ERP responses were below the sample mean and displayed an inverted pattern relative to the other two profiles: responses to no-reward were slightly larger than to reward in the

Doors task. Additionally, modest reductions in ssVEP responses to fear-related and safe stimuli were observed ($z = -0.42$ to -0.46), suggesting somewhat lower attentional engagement with these cues. Overall, the profile reflects near-average functioning across non-feedback domains, alongside selective attenuation and inversion in feedback evaluation.

Associations with Intolerance of Uncertainty and Internalizing Traits

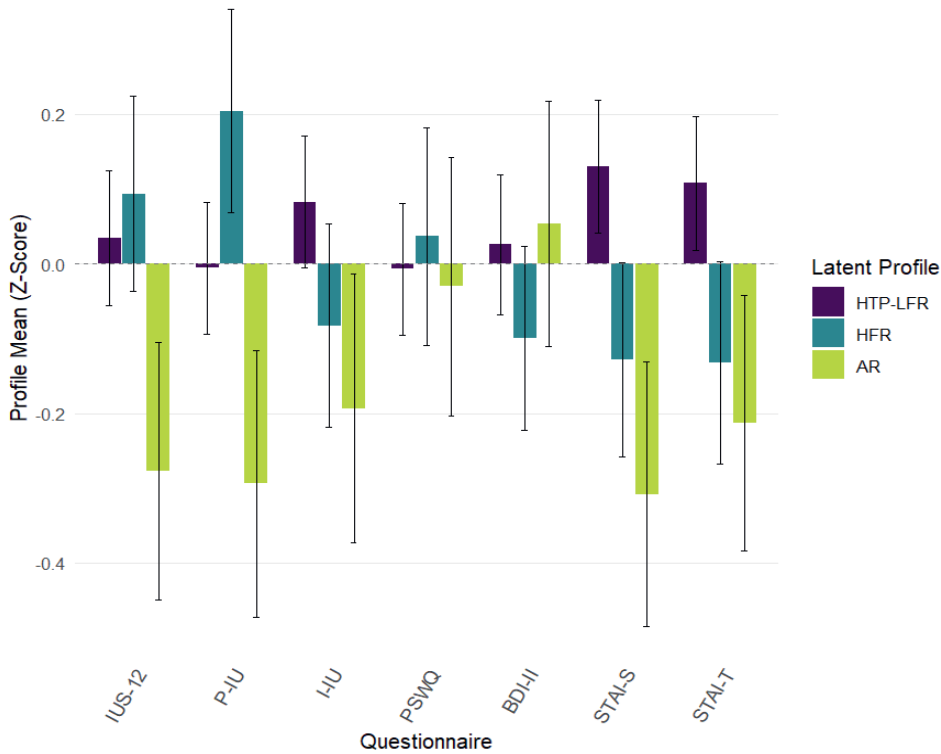
All questionnaire analyses were conducted on raw score units. An ANOVA indicated a profile effect for state anxiety (STAI-S). Exploratory pairwise comparisons showed that the High Threat and Performance Reactivity, Low Feedback Reactivity profile reported higher state anxiety than the Average Reactivity group, whereas differences with the High Feedback Reactivity group and between High Feedback Reactivity and Average Reactivity were not significant; see Table 4 and Figure 2 for detailed results. Importantly, these pairwise differences did not remain after Holm correction. Assumption checks indicated homogeneity of variances across all outcomes (all Levene’s $ps \geq .585$). No omnibus effects emerged for the other measured questionnaires trait anxiety, $F(2, 209) = 1.95, p = .144$; depressive symptoms (BDI-II), $F(2, 209) = 0.34, p = .712$; inhibitory IU, $F(2, 209) = 1.23, p = .293$; total IU (IUS-12), $F(2, 209) = 1.57, p = .210$; prospective IU, $F(2, 209) = 2.53, p = .082$; and worry (PSWQ), $F(2, 209) = 0.05, p = .949$.

Table 4
Group Differences in State Anxiety (STAI-S) Across Latent Profiles

Test	$F(2, 209)$	p
Omnibus ANOVA	$F = 3.14$	.046
Pairwise t -tests (uncorrected)		
Contrast	$t(209)$	p
High Threat & Performance – Low Feedback Reactivity vs. High Feedback Reactivity	$t = 1.58$	.117
High Threat & Performance – Low Feedback Reactivity vs. Average Reactivity	$t = 2.27$	.024
High Feedback Reactivity vs. Average Reactivity	$t = 0.82$	.415

Note. The omnibus ANOVA tests whether mean state-anxiety scores differ across the three latent profiles. Subsequent pairwise t -tests identify which profile comparisons account for any overall effect. Means (SE): High Threat & Performance – Low Feedback Reactivity = 42.5 (0.72), High Feedback Reactivity = 40.4 (1.15), Average Reactivity = 38.9 (1.43).

Figure 2
Standardized questionnaire scores by profile.



Note. Bars show profile-wise standardized means (z-scores) for questionnaire measures: IUS-12 (total), P-IU, I-IU, PSWQ, BDI-II, STAI-S, and STAI-T. Error bars depict ± 1 SEM. Profile abbreviations: HTP-LFR = High Threat and Performance Reactivity, Low Feedback Reactivity; HFR = High Feedback Reactivity; AR = Average Reactivity. Positive values indicate scores above the sample mean; negative values indicate scores below the sample mean. For omnibus and pairwise tests of state anxiety, see Table 4.

Discussion

We aimed to identify latent profiles in transdiagnostic processes spanning performance monitoring, fear and extinction learning, and reward processing. We then tested whether these profiles show meaningful differences in IU and related symptoms. We identified three latent neurocognitive profiles based on responses across three cognitive domains: performance monitoring, fear conditioning, and reward processing. These profiles included High Threat and Performance Reactivity, Low Feedback Reactivity profile, High Feedback Reactivity profile, and Average Reactivity profile. Participants assigned to the High Threat and Performance Reactivity, Low Feedback Reactivity profile showed slightly higher state anxiety compared to participants in the Average Reactivity profile; however, this difference did not survive correction for multiple comparisons. Overall, associations between profile and IU or internalizing symptoms were small and often not statistically supported, indicating that larger and prospective studies are needed to clarify these nuanced links.

The High Threat and Performance Reactivity, Low Feedback Reactivity profile combined elevated performance monitoring brain activity (i.e., larger ERN/CRN and Pe) with stronger threat and safety learning indicators of visual cortex responsiveness (i.e., ssVEPs to CS+ and CS- during acquisition and extinction phases), alongside comparatively reduced reactions to both reward and non-reward feedback (i.e., ERPs after feedback presentation). Heightened performance-monitoring potentials have been observed in anxiety (Moser et al., 2013; Riesel et al., 2017), and stronger fear learning has also been linked to anxiety (Duits et al., 2015; Lissek et al., 2005). Conversely, blunted responsiveness to feedback and rewards has been associated with depression and anhedonia (Keren et al., 2018; Proudfit, 2015; Whitton et al., 2015; Zhang et al., 2013). However, in the present data this profile was not related to depression scores, and although state anxiety was slightly higher than in the Average Reactivity group, this difference did not survive correction for multiple comparisons. Taken together, this pattern suggests a response style characterized by heightened vigilance and internal performance monitoring, alongside comparatively reduced sensitivity to external feedback signals, including potential rewards. These features may have clinical relevance for understanding vulnerability to anxious states and diminished reward responsiveness, but the present cross-sectional data do not permit causal or diagnostic inferences. Longitudinal work should test whether this pattern reflects brief, situation-bound increases in anxiety under uncertainty or a

stable response style that predicts later symptoms.

Conversely, the High Feedback Reactivity profile showed pronounced sensitivity to task feedback, with elevated feedback-locked ERPs to both reward and no-reward feedback, and a larger response to reward, alongside modestly reduced reactivity to threat and to errors. This configuration is consistent with a reward-focused orientation under uncertainty, in which feedback cues receive prioritized processing and may support approach-motivated responding (Koenig et al., 2017; Rothkirch et al., 2014; Shen et al., 2015). The relatively greater response to reward than no-reward suggests some valence specificity in motivational drive under uncertainty (Gorka et al., 2016), although alternative explanations remain plausible, such as higher feedback expectancy or sharper discrimination of feedback valence rather than a broad motivational strategy.

The Average Reactivity profile, encompassing the majority of the sample (59%), showed mostly responses close to the sample mean across the three different tasks. After correction for multiple comparisons, group differences on IU (total, Prospective, and Inhibitory), trait anxiety, and depressive symptoms were not statistically significant, and state anxiety likewise did not differ between profiles. Consistent with guidance to avoid over-interpreting mid-level classes and to use them primarily as comparative anchors, we treat the Average Reactivity group as a reference for interpreting the more extreme profiles rather than as a normative baseline (Ferguson et al., 2020). In related person-centered studies, the largest profile was not the symptom-elevated group in either sample. Additionally, differences between the reference and an above-average profiles on dysphoria and panic were nonsignificant, underscoring that prevalent classes can show limited symptom separation relative to each other (Park & Naragon-Gainey, 2024). Overall, most participants displayed balanced reactivity across domains, with limited questionnaire distinctions at the group level in this sample.

Our High Feedback Reactivity (HFR) profile, marked by larger feedback-locked responses to reward, resembles a profile in Park et al. (2023) characterized by stronger neural responsiveness to monetary reward, whereas our High Threat and Performance Reactivity with Low Feedback Reactivity (HTP-LFR) profile shares features with a profile in Park et al. (2023) characterized by blunted neural and self-reported reward responsiveness. In their study, they also reported a profile marked by high self-reported reward responsiveness with more variable neural responses, and these distinctions illustrate that profile-specific clinical signatures can emerge within the reward domain. Extending beyond reward, person-centered work on emotion-regulation ability similarly shows sep-

arable ability profiles that relate to strategies, goals, and internalizing symptoms (Park & Naragon-Gainey, 2024).

Integrating multiple cognitive-affective domains through LPA addresses gaps noted in prior work, where different types of variables (e.g., behavioral, physiological, and trait measures) show persistent separation between them, while associations within types are more commonly present individuals (Bernoster et al., 2019; Brandt & Mueller, 2022). LPA estimates subgroups defined by cross-domain patterns in a single model, enabling us to describe within-person configurations across physiological, behavioral, and self-report indices rather than treating each in isolation (Ferguson et al., 2020). For instance, in our case the High Threat and Performance Reactivity, Low Feedback Reactivity profile summarized how visual-cortex responsiveness during fear and safety learning (ssVEP) co-occurs with performance-monitoring activity (ERN, CRN, Pe), feedback-locked ERPs to reward and no-reward, behavioral measures, and expectancy ratings. Although LPA does not test statistical interactions among indicators, it reveals co-occurring patterns that can then be related to external variables (e.g., IU, symptoms), an approach mirrored in recent person-centered applications linking profile membership to goals and psychopathology outcomes (Park & Naragon-Gainey, 2024). Approaches like LPA complement single-domain analyses by offering an integrative perspective on cross-domain response patterns. Work, that aggregates indicators across paradigms, has shown that person-centered, multimodal profiles capture meaningful heterogeneity in mental health, yielding subgroups with distinct cognitive signatures and links to symptoms and functioning (e.g., Lichenstein et al., 2022; van Hulst et al., 2015). Future studies using similar integrative designs can clarify cross-domain relations among measurement types and their implications.

Although performance monitoring, threat learning, and reward processing are typically studied in isolation, in everyday settings they unfold together and shape one another, which motivates an integrated analysis across paradigms. Standard experimental tasks are designed to disentangle specific processes and optimize internal validity; complementing them with integrative approaches helps address ecological validity when the goal is to understand behavior in natural contexts (Nastase et al., 2020). Converging evidence shows that affective and motivational states modulate executive and performance-monitoring operations, from broad emotion–cognition coupling to concrete trade-offs between threat and reward during task performance (Choi et al., 2015; Pessoa, 2009).

The profiles identified here inform theory by illustrating distinct neurocog-

nitive response patterns in contexts that contain uncertain elements. Although these experiments were selected to assess how IU relates to cognitive functioning rather than to maximize uncertainty per se, each paradigm nonetheless embeds uncertainty in a specific way. In the Flanker task, incongruity of the array and time pressure increase error likelihood, and errors are considered endogenous and uncertain source of threat (Weinberg et al., 2016). In the Fear Conditioning task, partial reinforcement during acquisition (75% CS–US pairing) and an uninstructed extinction phase produce outcome uncertainty as contingencies change. In the Doors task, the presence of risk cues (especially the ambiguous cue) creates uncertainty about reward receipt on each trial. While direct associations between IU and the identified profiles were not found in this sample, the profiles highlight meaningful individual variability that traditional group-mean approaches (for example, ANOVA or standard regressions) may miss. In contrast, LPA characterizes within-person configurations across threat learning, performance monitoring, and reward processing, revealing cross-domain patterns that can be overlooked by single-domain analyses. Future research utilizing similar integrative approaches can further elucidate how individual differences in neurocognitive responses relate to specific traits such as IU, thereby enriching our understanding of the mechanisms underlying variability in vulnerability to internalizing disorders.

Detailed limitations for each single-domain study are provided in their respective publications (Malbec et al., 2022, 2024, 2025) and in *Chapters 2-4*. Here, we summarize the principal limitations highlighted in those reports together with the key constraints of the present integrative analysis. First, the sample size ($N = 214$) is modest for latent profile analysis, which can reduce the stability of class enumeration and the power to detect subtle between-profile differences (Ferguson et al., 2020). Second, data were collected during the COVID-19 pandemic, a period of heightened uncertainty and stress that may limit generalizability beyond pandemic conditions. Third, the sample primarily comprised undergraduate students and included a high proportion of female participants (80.2%), which restricts demographic diversity and external validity. Fourth, the sample was largely psychologically healthy with a restricted range on symptom measures. This likely increased the proportion of an average-responding profile and may have attenuated profile differences in IU and psychopathology. Future studies should recruit more diverse participants with respect to symptom severity, including community and clinical cohorts, or use stratified sampling to ensure adequate variability. Fifth, the single-session, cross-sectional design precludes

causal inference and does not address the temporal stability of the profiles. Finally, heterogeneity in indicator sensitivity and reliability across paradigms may have influenced the obtained profile structure, underscoring the need for replication and external validation in independent cohorts.

In conclusion, employing an integrative LPA approach provided valuable insights into distinct neurocognitive profiles relevant to internalizing psychopathology. By modeling responses across multiple experimental paradigms, this study showed the value of identifying naturally occurring subgroups that differ not only in magnitude but also in configuration across contexts that include minimal uncertainty features. Person-centered models such as LPA can recover subgroup patterns that population-average, variable-centered analyses often obscure (Ferguson et al., 2020). This is particularly relevant in internalizing research, where extensive overlap and comorbidity indicate shared liabilities and heterogeneous presentations (Caspi et al., 2014). Our findings underscore that individuals vary meaningfully in how they process performance feedback, threatening stimuli, and reward outcomes. By highlighting nuanced cross-domain patterns that are often studied in isolation, this work advances a more comprehensive account of neurocognitive variability that better reflects how these processes interact in real-world settings.

Although associations with internalizing traits were modest in this sample, reproducible cross-domain profiles can still inform prognosis and personalization. A concrete next step is to test predictive validity: does profile membership at the initial assessment predict later symptom trajectories, stress reactivity, or functional impairment, and does it add prediction beyond single-task indicators and trait scales such as IU measured at the same initial session? Equally important is treatment utility: do profiles moderate response to specific intervention components that target error processing and performance monitoring (i.e., A. Meyer et al., 2020), safety learning under uncertainty (i.e., Craske et al., 2014), and reward engagement (i.e., Dimidjian et al., 2006)? Rigorous evaluation will require larger and more diverse samples, preregistered longitudinal designs, external validation, and tests of temporal stability and transition probabilities. With further validation, profiles may contribute as a complementary input to both hypothesis generation and treatment planning.

Chapter 6



Summary and Discussion

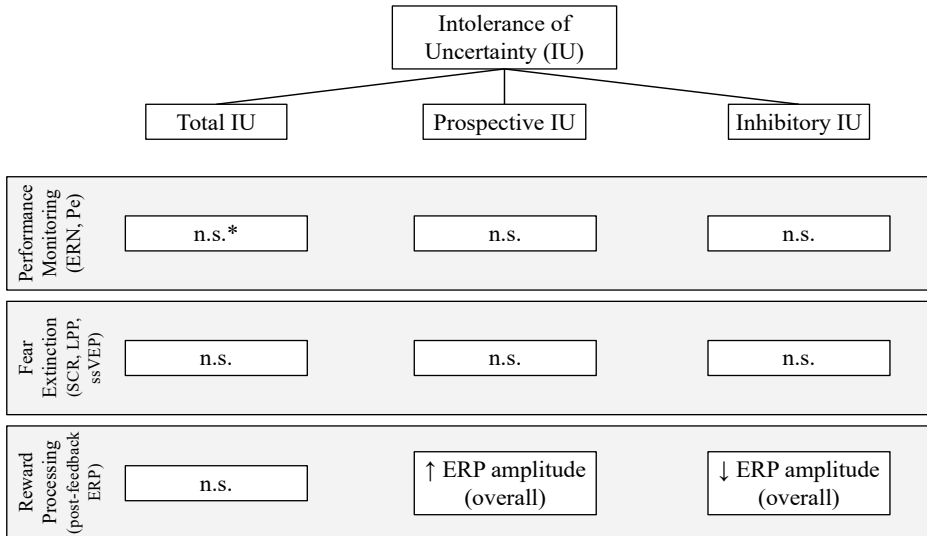
The aim of this dissertation was to investigate the influence of intolerance of uncertainty (IU) in shaping cognitive and affective responses across different experimental contexts. IU is considered a transdiagnostic construct for internalizing disorders, thus understanding its influence can improve our understanding of its impact on anxiety and depression. In *Chapters 2, 3, and 4*, I present results from investigations on three cognitive processes: performance monitoring, fear conditioning, and reward processing. Each study used a combination of behavioral and physiological measures to assess how individual differences in IU and its subfactors (i.e., prospective and inhibitory IU), relate to affective and cognitive processes relevant for internalizing psychopathology. In *Chapter 5*, we adopted a data-driven approach to identify distinct neurocognitive profiles underlying individual differences in the cognitive processes previously described. Leveraging the comprehensive assessment made possible by the use of the same sample of participants across all tasks, this approach allowed us to uncover latent patterns that could explain variability in intolerance of uncertainty and related mental disorders.

Summary

Figure 1 presents a graphical summary of the main findings described above and serves as a counterpart to the figure in the introduction, which depicted prior evidence on IU's impact on human cognition. Together with the chapter summaries, this visual representation sets the stage for the following sections, where the findings of this dissertation are contrasted in more detail with the existing IU literature.

Figure 1

Observed Effects of IU Across Three Cognitive Processes.



Note. ↑ = larger response; ↓ = smaller response; n.s. = no statistically significant effect ($p \geq .05$) detected between IU facet and cognitive process main variables; ERN = error-related negativity; Pe = error positivity; SCR = skin conductance response; LPP = late positive potential ERP; ssVEP = steady-state visually evoked potential. * = Higher total IU correlated with larger correct-response negativity (CRN). Measures in parentheses denote main variables analyzed.

Performance monitoring and IU: *Chapter 2* focused on performance monitoring using a modified Flanker task. Neural and behavioral indices of performance monitoring showed the expected patterns with more negative amplitudes for errors than correct trials for the ERN and the opposite pattern for the Pe, as well as a significant increase in reaction times after errors compared to correct responses (i.e., post-error slowing). These findings confirmed the sensitivity of the task to detect performance monitoring responses. Regarding IU and its subfactors, they were not associated with the ERN or Pe, neither predict post-error adjustments. However, a small but significant association was observed between total IU and the correct-response negativity (CRN), with higher IU predicting more negative CRN amplitudes. This effect remained after controlling for trait anxiety, suggesting a degree of specificity to IU. Exploratory moderation analyses also found no significant interactions between IU and depression, trait anxiety, or worry in predicting ERN amplitude. Together, the findings suggest a limited role for IU in modulating performance monitoring, with one exception emerging for cor-

rect-response processing. These findings contrast with previous studies reporting an opposing effect of IU subfactors on the ERN and IU playing a moderation role in the relationship between ERN and depression.

Fear conditioning and IU: *Chapter 3* focused on both fear acquisition and extinction using a differential conditioning paradigm with immediate extinction. During fear acquisition training, successful fear conditioning was indicated by increased SCRs and ssVEP amplitudes as well as increased subjective ratings (valence, arousal, and threat expectancy) towards the threat cue (CS+). During acquisition, LPP amplitudes were larger for the safe cue (CS-) than for the threat cue (CS+) in the first half of acquisition, a pattern that then disappeared, showing no differences between cues during the second half. No significant associations were found between IU (total or subfactors) and fear learning at this stage, across any of the outcome measures. These findings are consistent with prior studies reporting no effects of IU during acquisition and may reflect the limited influence of IU under high-threat, high-contingency conditions. During extinction, physiological and self-report responses showed partial extinction of conditioned responses. SCRs no longer differentiated between CS+ and CS- at late extinction, indicating successful extinction learning. In contrast, LPPs, ssVEPs, and valence/arousal ratings continued to show larger responses to the CS+, suggesting persistence in subjective and cortical indices of threat discrimination. However, IU did not significantly modulate extinction dynamics in this sample, contrasting with earlier studies that found some evidence of delayed or impaired extinction in high IU individuals. Overall, these findings suggest that IU plays a limited role in differential fear learning and extinction in the context of this study, which used a relatively high reinforcement rate and immediate, uninstructed extinction.

Reward Processing and IU: *Chapter 4* focused on reward processing under uncertainty using a modified version of the Doors Task. Participants received monetary feedback (reward or no-reward) in this task after choosing among options with varying levels of reward outcome probability (low, high, and ambiguous risk). Feedback-related ERPs showed robust modulation by feedback type and risk level, with more positive amplitudes following reward compared to no-reward outcomes, and higher amplitudes under high-risk conditions. IU total scores were not significantly associated with electrocortical responses. However, the IU subfactors showed independent and opposing effects: prospective IU was associated with increased ERP amplitudes following both reward and no-reward feedback, while inhibitory IU predicted reduced amplitudes across both conditions. These effects remained after controlling for depressive symptoms, trait

anxiety, and worry. Previous studies have also reported opposing effects of IU subfactors on reward processing markers. However, those effects were typically observed in difference scores between feedback types, whereas our findings reflect an impact of IU subfactors on overall neural responses across feedback conditions. Additionally, depressive symptoms were associated with blunted amplitudes specifically following no-reward outcomes. The findings highlight distinct associations of IU subfactors with feedback-related neural responses, independent of overall internalizing symptomatology.

Neurocognitive Profiles Across Tasks: *Chapter 5* used latent profile analysis (LPA) to combine the data across the three cognitive processes to identify subgroups of participants with similar patterns of responding. The analysis yielded three distinct profiles based on behavioral and neurophysiological indicators of performance monitoring, fear and extinction learning, and reward processing. Profile 1, High Threat and Performance Reactivity with Low Outcome Reactivity, showed larger ERN/CRN and Pe, higher ssVEPs to threat and safety cues, and reduced feedback-locked ERPs to outcomes. Profile 2, High Outcome Reactivity, showed strong feedback-locked ERPs with otherwise typical responses in threat and cognitive control. Profile 3, Average Reactivity, was close to the sample mean across most measures, with a slight reduction in ssVEPs through fear conditioning. These profiles, however, were not different on measures of IU, including both subfactors, nor depressive symptoms or worry. State anxiety was modestly higher in the more reactive profile, but this difference should be interpreted with caution. Overall, the profiles capture meaningful variation in neurocognitive responding, while links with trait questionnaires were limited.

Interpretation and Theoretical Integration

In direct response to the central research question of this dissertation, IU did not consistently predict behavioral or neurophysiological responses across the cognitive processes examined. Associations with total IU and its subfactors were largely absent, with only limited and domain-specific effects emerging. To contextualize these findings, we first outline areas of convergence with previous research, followed by a discussion of points of divergence.

Our findings contribute important clarification to the existing literature as they align with prior studies that report minimal or no IU effects across various cognitive processes. For performance monitoring, our findings showed no significant relationships between IU and the ERN or Pe amplitudes. This null re-

sult aligns with those from studies conducted by Sandre and Weinberg (2019), Ruchensky et al. (2020), and Carsten et al. (2025), all of which did not observe a direct effect of IU or its subfactors on error-related brain activity. In the domain of fear learning and extinction, our results mirror those of previous studies reporting null or weak IU effects. For fear learning, this lack of influence by IU has been observed in multiple publications (for a review see Morriss, Zuj, and Mertens, 2021). However, for fear extinction the studies that also reported no significant effect from IU are rarer (Morriss et al., 2020, 2024; Wendt & Morriss, 2022). Regarding reward processing, our observation that IU subfactors were differentially associated with neural responses to feedback is to some extent in line with previous research showing similar patterns (Nelson et al., 2016), albeit not specific for positive or negative feedback. These convergences suggest that IU may not have broad or consistent effects across these cognitive systems, and that its measurable influence may emerge primarily under specific conditions.

At the same time, our findings diverge in meaningful ways from parts of the existing literature. The lack of significant associations between IU and extinction-related responses contrasts with studies reporting persistent threat signals in high-IU individuals during extinction phases (for a review: Morriss, Zuj, and Mertens, 2021; for a meta-analysis: Morris, Wake, et al., 2021). Methodological factors such as reinforcement schedules and extinction timing may contribute to these inconsistencies. In performance monitoring, although we did not replicate prior findings linking IU to ERN amplitude (Jackson et al., 2016; Grabowska et al., 2024), we found a small but reliable association between total IU and CRN amplitude. This finding, not widely discussed in previous literature, suggests a potential IU-related sensitivity during evaluation of correct responses, distinct from error detection. Moreover, while our findings in reward processing aligned with studies reporting IU subfactor effects, they contrast with null results from other work (e.g., Muir et al., 2021). These discrepancies emphasize the need to further delineate task characteristics and sample features that may constrain the detectability of IU-related effects.

In the same vein, the findings from *Chapter 5*, which identified three distinct neurocognitive profiles based on patterns of behavioral and physiological responses across the three experimental tasks, showed no association with IU levels. Because this analysis approach is novel, direct comparisons with prior studies are limited. Nevertheless, the profile-based analysis introduces a promising framework for characterizing individual variability across multiple cognitive-affective systems. By integrating information across tasks, *Chapter 5* offers a

more comprehensive view of how response tendencies may shape cognitive and emotional functioning. While IU did not differ between profiles, the approach demonstrates how multidimensional data can be used to identify meaningful patterns of individual differences beyond single-domain analyses.

Altogether, these findings carry important theoretical implications. Rather than supporting a general model in which IU exerts widespread influence across cognitive-affective processes, the evidence from this dissertation favors a more nuanced and context-sensitive view. Perhaps, instead of functioning as a global mechanism of vulnerability, IU may better be described as a cognitive-affective style or disposition that becomes particularly relevant in specific contexts. IU may, for example, be more clearly expressed during subjective appraisals of uncertainty in real-life situations or in contexts characterized by sustained ambiguity. This interpretation is in line with Morriss, Zuj, and Mertens (2021) claims that IU's effects might be especially pronounced in paradigms or environments where anticipatory threat and sustained uncertainty are explicitly evoked. This has potential clinical implications, suggesting that the influence of IU may be most relevant in situations where individuals are consciously aware of uncertainty within a given context. This stands in contrast to the initial assumptions of this project, which were based on the hypothesis that IU would exert a broad and pervasive influence on cognitive functioning across domains.

More broadly, this refined conceptualization aligns with recent critiques about measurement and conceptual specificity in transdiagnostic constructs. Brandt and Mueller (2022), for example, emphasize that constructs measured via self-report, behavioral paradigms, and neurophysiological indicators tend to represent distinct levels of analysis that are only weakly interrelated. This limited convergence between self-report, behavioral, and neurophysiological variables was also reflected in our data. As described in the supplementary analyses reported in *Chapter 4*, correlations tended to cluster within measurement modality: questionnaire-based variables were interrelated, as were ERP components, but associations between these types of measures were generally weak or absent. In *Chapter 3*, divergence across modalities was also evident. While some physiological and self-report indices aligned during certain phases, contrast between them even within variable types suggests that different modalities may capture distinct features of fear learning and extinction. For example, successful extinction was evident in SCR but not clear in affective ratings nor in ssVEP and LPP. Rather than reflecting inconsistency, such divergence reinforces the utility of a multimodal approach, as each type of measure may be sensitive to different lay-

ers of affective and cognitive processing. Supporting this view, the profile-based analysis in *Chapter 5* revealed that latent classes were distinguished primarily by task-specific patterns of responding, and not by measurement type. This indicates that physiological and behavioral indices varied meaningfully between tasks, despite being collected in the same testing session, and did not stick together into cross-modal subtypes. This perspective urges caution in expecting robust cross-domain correlations and suggests the need for theoretical precision regarding the extent to which psychological constructs are able to produce consistent physiological or behavioral markers. Future models should integrate these insights to refine hypotheses regarding where, when, and how IU-related effects are most likely to emerge. Based on our findings, this should occur most likely in responses to reward processing at the IU subfactors level, while the definitive role of IU on fear extinction responses should be further explored. For performance monitoring, unexpectedly, IU appears to be more closely linked to neural responses following correct trials rather than errors. Finally, we also consider that the use of several measures can facilitate a comprehensive outlook of the specific impact of IU on cognitive-affective processes.

It is important to emphasize that the findings of this dissertation call for a careful re-evaluation of IU's influence on the cognitive processes examined, rather than a dismissal of its clinical relevance. Specifically, the results invite a more precise understanding of the limits of IU's impact on biological and behavioral markers of cognitive-affective functioning. These findings suggest that IU may not exert broad or uniform effects across tasks but instead operates in a more context-dependent and process-specific manner. Importantly, this does not undermine IU's established theoretical and clinical importance. A substantial body of evidence continues to support its association with anxiety, depression, maladaptive coping, and vulnerability to internalizing psychopathology (Carleton, 2016; Mahoney & McEvoy, 2012b; McEvoy & Mahoney, 2011). Then, future research and clinical applications of our findings should consider the more delimited figure of IU presented here.

General Discussion

This dissertation employed a broad array of measurement approaches to investigate the influence of IU and internalizing traits on cognitive-affective processing. These methods included behavioral indices (e.g., reaction times), subjective self-reports (e.g., arousal ratings), autonomic responses (e.g., skin con-

ductance responses), and neurophysiological measures (e.g., ERPs such as ERN). The use of this multimodal strategy aimed to capture the potential influence of IU across different levels of analysis, reflecting the idea that cognitive-affective traits may manifest in diverse response systems.

However, in contrast to the variety of outcome measures, IU was assessed exclusively through a self-report instrument, the Intolerance of Uncertainty Scale (IUS; Buhr & Dugas, 2002; Freeston et al., 1994). Although validated and psychometrically robust (e.g., Birrell et al., 2011; McEvoy & Mahoney, 2011), the IUS primarily assesses the subjective difficulty tolerating uncertainty. Thus, it may not adequately capture the dynamic cognitive or emotional processes occurring during real-time experimental tasks. This constraint reflects a broader methodological issue inherent to self-report measures. Bernoster et al. (2019) demonstrated that measurements within the same modality (e.g., self-report scales) often correlate strongly with one another but show weaker and less consistent relationships with measures from other modalities (e.g., behavioral or physiological indices). According to Brandt and Mueller (2022), these discrepancies arise because different measurement types capture distinct levels of abstraction. Specifically, self-reports typically manifest reflective, consciously accessible evaluations, whereas physiological and behavioral measures tap into automatic, moment-to-moment processes. Such conceptual misalignment between modalities may limit cross-modal correlations and interpretability. We consider that perhaps future studies can complement the measurement of IU by finding assessment tools in modalities other than self-report.

One prominent candidate for this is the Beads Task, a probabilistic reasoning paradigm in which participants decide how much information to gather before making decisions under uncertainty (Jacoby et al., 2014; Jacoby et al., 2016). This experimental design has been proposed by these authors as a valid way of measuring IU at the behavioral level. They showed that high IU is expressed through a tendency toward earlier decisions and greater subjective distress, even when objective performance differences are minimal. Another related but different strategy was employed in a recent collaboration study (Carsten et al., 2025), in which the data collected for this dissertation (specifically *Chapter 2*) was combined with data collected at Hamburg University. They applied to their participants an uncertainty inducing intervention based on a difficult Frank task (Frank et al., 2004). Briefly, participants were told they needed to learn correct choices based on feedback. The feedback was deliberately unpredictable. To increase uncertainty, participants were told their learning would be monitored and compared to others, and the ex-

perimeter periodically entered to comment on task difficulty. Interestingly, their results show the effectiveness of this induction procedure only on self-reported uncertainty ratings, but its effect was not visible on ERN changes. Finally, integrating assessments of IU into ecologically valid contexts represents a promising avenue for future research. Real-life uncertainty scenarios, such as awaiting results from academic tests, medical diagnoses, or immigration decisions, are emotionally salient and inherently uncertain, potentially providing more direct and meaningful assessments of IU. A recent study by Appel et al. (2024) successfully utilized Experience Sampling Methodology (ESM) to capture IU in naturalistic daily decision-making contexts, finding that high IU predicted increased indecisiveness and safety behaviors. More broadly, ESM has demonstrated potential in capturing uncertainty-related emotional responses in specific contexts such as academic settings (e.g., awaiting test results) and other daily life scenarios (Pejović et al., 2016). Despite this emerging evidence, further research is needed to specifically explore the role of IU within these ecologically valid assessment methods. Incorporating these ecological and momentary assessment methods could significantly enhance the ecological validity of IU measurement, thus reducing interpretive ambiguity regarding its manifestation and impact.

Across the studies in this dissertation, the sample was internationally diverse. Detailed descriptions of participant demographics are presented in *Chapters 2–5*, but briefly, about half of the participants were international students, representing a broad range of cultural backgrounds. Such demographic variability might influence findings and their comparability with prior research (e.g., Pavlenko, 2012; Lewis et al., 2008; Na & Kitayama, 2011). Nonetheless, diverse samples are increasingly encouraged in psychological and psychophysiological research, as they enhance the generalizability and applicability of findings (Henrich et al., 2010). In the same vein, it is important to note that our sample was drawn exclusively from undergraduate students at a Dutch university, which limits generalizability to populations that differ in socioeconomic circumstances, educational attainment, broader cultural contexts, in addition to clinical populations. Finally, both language versions of the IUS employed in this dissertation have previously demonstrated good psychometric properties (de Bruin et al., 2006; Buhr & Dugas, 2002) and exhibited high internal consistency in our own. For an analysis of the internal consistency of the questionnaires used in this dissertation, see *Chapter 5*.

An additional consideration when interpreting IU-related findings concerns the conceptual structure of the IU subfactors. As discussed in *Chapter 4*, the prospective and inhibitory components of IU represent distinct but correlated fac-

ets of the construct (Birrell et al., 2011; McEvoy & Mahoney, 2011). Prospective IU reflects a tendency toward cognitive engagement and worries in anticipation of uncertain outcomes, while inhibitory IU refers to behavioral suppression or freezing in the face of uncertainty. Although this distinction is conceptually valuable, it might include interpretative challenges when both subscales are contrasted simultaneously. Individuals often score high on both subfactors, given their shared inclusion in the broader IU construct (Birrell et al., 2011). In such cases, their behavioral or neurophysiological responses may reflect competing influences from both components. This can obscure meaningful patterns or lead to misleading interpretations, particularly when effects appear in opposite directions. For this reason, findings involving IU subfactors should be interpreted cautiously, and future studies aiming to isolate their effects may benefit from grouping participants based on subscale profiles or using analytical strategies that account for their overlap.

Implications for scientific practice

A key implication of these findings is the need for a more refined theoretical framework that specifies the exact cognitive processes or conditions under which IU is most likely to exert its influence. The rigorous methodological approach of the present studies, including preregistration, open data sharing, relatively large sample sizes, and the use of multiple physiological measures, provides a strong interpretive context for the findings reported here. This methodological rigor reduces the likelihood that the observed absence of effects can be attributed to insufficient power or to flaws in the experimental procedures. Consequently, these null findings suggest that some previously reported associations between IU and cognitive or affective processes may be more context-dependent or susceptible to analytic variability than previously assumed.

Moreover, these results contribute to broader discussions concerning the reporting and interpretation of null findings in psychological science. Positive findings are disproportionately published, while null results often remain unpublished, exacerbating the so-called “file drawer problem” (Rosenthal, 1979). This selective reporting can inflate effect sizes and distort theoretical conclusions (Fanelli, 2012). In the rapidly expanding IU literature, null findings remain relatively underreported. The present dissertation offers a meaningful counterpoint to this trend by explicitly documenting null effects obtained under rigorous conditions. Future meta-analyses and theoretical reviews of IU should incorporate such well-documented null results to provide a more balanced empirical

understanding. Broadly, this aligns with ongoing reform efforts in psychological science, especially regarding replicability (Open Science Collaboration, 2015; Tackett et al., 2019). Increasingly, researchers emphasize larger samples, pre-registered hypotheses, and transparent reporting practices as foundational for cumulative scientific progress (Nosek et al., 2015). Rather than undermining IU as a construct, the current findings support a more context-sensitive, empirically grounded understanding of its psychological correlates.

Conclusions

In summary, this thesis provides converging evidence that IU does not exert a broad or robust influence on core neurocognitive processes involved in performance monitoring, fear learning and extinction, or reward processing. Across a series of well-powered, multimodal experimental tasks, IU showed limited and context-dependent effects, with most associations failing to reach statistical significance. These findings challenge assumptions that IU is a pervasive transdiagnostic risk factor manifesting uniformly across cognitive-affective domains. Instead, the results suggest that the impact of IU is more nuanced, potentially emerging only under specific contextual or methodological conditions. By clarifying the boundaries of IU's influence, this work contributes to a more precise understanding of its role in individual differences in adaptive and maladaptive behavior.

Clinically, these results can be translated as an indication to find the more specific presence of IU in patients' cognition. In individuals with internalizing symptoms, IU may be expressed as conscious distress or worry, even if its impact on underlying cognitive-affective functioning is weaker than previously assumed. This perspective aligns with transdiagnostic frameworks such as the RDoC, which emphasize the importance of contextualizing risk factors within particular domains of functioning. Ultimately, these findings highlight the value of personalized treatment approaches, where targeting IU may be most effective when uncertainty is central to the presenting problem. Future research should further specify the conditions under which IU meaningfully affects neurocognitive functioning to inform clinical interventions.

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Samenvatting



Summary in Dutch

Intolerantie voor onzekerheid en neurocognitieve responsen

Onzekerheid is een vast onderdeel van het dagelijks leven. Toch verschillen mensen sterk in hoe belastend zij onzekerheid vinden. Intolerantie voor onzekerheid (Engels: *intolerance of uncertainty*, IU) verwijst naar de neiging om onzekere situaties als stressvol of bedreigend te ervaren. IU wordt vaak gezien als een transdiagnostisch kenmerk dat samenhangt met internaliserende problematiek, zoals angst- en depressieve klachten. Verschillende cognitieve en affectieve processen dragen bij aan de ontwikkeling en het in stand houden van zulke internaliserende problematiek, maar het is nog onduidelijk hoe deze processen samenhangen met IU. Het doel van dit proefschrift was om te onderzoeken hoe IU samenhangt met cognitieve en affectieve responsen in verschillende experimentele contexten. In Hoofdstuk 2, 3 en 4 worden drie neurocognitieve processen onderzocht: prestatiemonitoring (*performance monitoring*), angstconditionering en extinctie (*fear acquisition and extinction*) en beloningsverwerking (reward processing). In deze studies is gebruikgemaakt van gedragsmatige, subjectieve en neurofysiologische maten, waaronder EEG (electro-encefalogram), gebeurtenisgerelateerde potentialen (event-related potentials; ERPs), huidgeleiding (skin conductance responses; SCR) en visueel opgeleverde potentialen (steady-state visually evoked potentials; ssVEP). Daarnaast is IU niet alleen als totaalscore bestudeerd, maar ook uitgesplitst in twee componenten: prospectieve IU (de neiging om onzekerheid actief te willen verminderen, bijvoorbeeld door informatie te zoeken) en remmende IU (de neiging om te blokkeren, vermijden of vast te lopen bij onzekerheid). In Hoofdstuk 5 is vervolgens een data-gedreven benadering gebruikt om te onderzoeken of er in de hele dataset neurocognitieve profielen bestaan die variabiliteit in IU en gerelateerde internaliserende problematiek kunnen verklaren.

Prestatiemonitoring en IU (Hoofdstuk 2)

In Hoofdstuk 2 werd prestatiemonitoring onderzocht met een aangepaste Flanker-taak. Deze taak meet hoe mensen conflicterende informatie verwerken en hoe zij reageren op fouten. De taak riep de verwachte patronen op: fouten gingen gepaard met een vroege ERP-component (*error-related negativity*; ERN) en een latere ERP-component (*error positivity*; Pe). Gedragmatig zagen we dat deelnemers langzamer reageerden na een fout dan na correcte responsen (*post-error slowing*). Deze resultaten bevestigen dat de taak inderdaad prestatie-monitoring meet.

De centrale vraag was vervolgens of IU samenhangt met deze neurale en ge-

dragsmatige maten. Over het geheel genomen waren IU en de IU-componenten niet significant gerelateerd aan de ERN of Pe en zij voorspelden ook geen langzamere reacties na fouten. Eén specifieke bevinding viel op: een kleine maar consistente associatie tussen hogere totale IU en een meer negatieve ERP-component na correcte responsen (*correct-response negativity*; CRN). Dit verband bleef aanwezig na controle voor angst, wat wijst op een zekere mate van specificiteit. Samengevat suggereren de bevindingen dat IU in deze context een beperkte rol speelt bij foutverwerking, maar mogelijk een specifiek verband heeft met de verwerking van correcte responsen.

Angstconditionering, angstextinctie en IU (Hoofdstuk 3)

Hoofdstuk 3 focuste op differentiële angstconditionering en extinctie, met een paradigma waarin de extinctiefase direct volgde op de acquisitiefase. Tijdens de acquisitiefase werd succesvolle conditionering aangetoond op meerdere niveaus: zowel SCR, ssVEP-amplitudes als subjectieve beoordelingen (valentie, arousal en verwachting van dreiging) lieten duidelijk grotere reacties zien op de bedreigende stimulus (CS+) dan op de veilige stimulus (CS-). In de eerste helft van de acquisitiefase waren LPP-amplitudes groter voor de veilige stimulus (CS-) dan voor de bedreigende stimulus (CS+). Dit patroon verdween, in de tweede helft van de acquisitiefase waren er geen verschillen meer tussen de stimuli. Belangrijk is dat IU, zowel de totaalscore als uitgesplitst in de twee componenten, niet samenhang met het aanleren van angst tijdens de acquisitiefase, ongeacht de gebruikte uitkomstmaat. Dit sluit aan bij eerdere bevindingen dat IU tijdens de acquisitiefase niet altijd effecten laat zien, mogelijk omdat situaties met hoge dreiging en duidelijke contingenties weinig ruimte bieden voor verschillen in intolerantie voor onzekerheid.

Tijdens de extinctiefase was er sprake van gedeeltelijke extinctieleren. Huidgeleiding (SCR) was aan het einde van de extinctiefase niet langer verschillend tussen de CS+ en de CS-, wat wijst op succesvol extinctieleren op autonoom niveau. Tegelijkertijd bleven ssVEPs, LPP en subjectieve beoordelingen groter voor de CS+ dan voor de CS-, wat suggereert dat deze neurale en subjectieve verschillen langer blijven bestaan. Er was geen effect van IU op deze bevindingen in de extinctiefase, in tegenstelling tot andere studies die vonden dat IU gerelateerd was aan verlaat of verminderd extinctieleren. De rol van IU in angstconditionering en angstextinctie lijkt dus beperkt in dit specifieke onderzoeksdesign waarin relatief veel CS+ stimuli werden gepaard met een schok tijdens de acquisitiefase en er geen instructies waren in de extinctiefase die daar meteen op volgde.

Beloningsverwerking en IU (Hoofdstuk 4)

In Hoofdstuk 4 werd beloningsverwerking onderzocht met een aangepaste Doors-taak, waarin deelnemers financiële feedback ontvingen (geldbeloning versus geen beloning) nadat ze gekozen hadden tussen opties met uiteenlopende kansen op beloning (laag, hoog en ambigu). Feedback-gerelateerde ERPs werden beïnvloed door feedbacktype en risico: beloningen gingen gepaard met positievere amplitudes en alle reacties waren groter als het risico hoog was.

Voor IU totaalscore werd er geen consistent verband gevonden met electrocorticale responsen. Daarentegen vertoonden de IU-componenten tegengestelde effecten: prospectieve IU hing samen met grotere ERP-amplitudes na zowel beloning als geen beloning, terwijl remmende IU juist samenhang met kleinere ERP-amplitudes na zowel beloning als geen beloning. Deze effecten bleven bestaan nadat er werd gecontroleerd voor depressieve symptomen, angst en piekeren. Daarmee laten de resultaten zien dat het onderscheiden van IU-componenten cruciaal kan zijn om te begrijpen hoe intolerantie van onzekerheid gerelateerd is aan electrocorticale responsiviteit tijdens feedbackverwerking. Daarnaast werd gevonden dat depressieve symptomen samenhangen met verminderde ERP-amplitudes als er geen beloning kwam, wat aansluit bij de literatuur over afgevlakte responsiviteit voor negatieve of teleurstellende uitkomsten bij mensen met depressieve klachten.

Neurocognitieve profielen gebaseerd op alle taken (Hoofdstuk 5)

Hoofdstuk 5 integreerde alle maten uit de drie experimentele taken met een latente profielanalyse (*latent profile analysis*; LPA). Het doel was om te onderzoeken of er subgroepen mensen bestaan met een vergelijkbaar patroon van neurocognitieve responsen in één of meerdere processen. De analyse leverde drie profielen op: profiel 1 werd gekenmerkt door hoge reacties op dreiging en prestatie (grotere ERN/CRN en Pe; hogere ssVEP-responsen op stimuli) in combinatie met lage reacties op uitkomst (lagere feedback-gerelateerde ERPs); profiel 2 liet juist sterkere feedback-gerelateerde ERPs zien, terwijl de overige processen grotendeels gemiddeld waren; profiel 3 scoorde rond het steekproefgemiddelde op de meeste maten, met lagere ssVEP-responsen tijdens angst-conditionering.

Opvallend was dat deze profielen niet betekenisvol verschilden in IU en ook niet in depressieve symptomen of piekeren. Angst was enigszins hoger in profiel 1, maar dit moet voorzichtig geïnterpreteerd worden. Deze resultaten laten zien dat er duidelijke en interpreteerbare variatie bestaat in neurocognitieve

responsiviteit, maar dat de relatie met karaktereigenschappen in deze steekproef beperkt was.

Overkoepelende conclusie

De bevindingen van dit proefschrift schetsen een genuanceerd beeld over de rol van intolerantie voor onzekerheid in neurocognitieve processen. IU bleek niet samen te hangen met de meeste onderzochte neurocognitieve processen. Er werd wel een matig verband gevonden tussen totale IU en neurale verwerking van correcte responsen tijdens prestatie-monitoring en tegengestelde verbanden voor prospectieve en remmende IU met neurale responsiviteit op feedback tijdens beloningsverwerking. De latente profielanalyse toonde daarnaast dat er taak-overstijgende neurocognitieve profielen bestaan, maar dat de mensen met deze profielen niet verschilden in IU.

Samen suggereren de resultaten van dit proefschrift dat de invloed van IU contextafhankelijk is en dat het onderscheiden van IU-componenten belangrijk is om de relatie tussen intolerantie voor onzekerheid en neurocognitieve processen te begrijpen. Deze bevindingen dragen bij aan het verfijnen van theorieën over IU en leveren aanknopingspunten voor toekomstig onderzoek naar wanneer, hoe en bij wie intolerantie voor onzekerheid het sterkst doorwerkt in neurocognitieve processen die relevant zijn voor internaliserende problematiek.

Curriculum Vitae



Marcelo Malbec was born in Santiago, Chile, on January 6, 1991. He completed secondary education at Colegio Campanario in 2008 and then studied Psychology at Pontificia Universidad Católica de Chile (PUC). He specialized in Clinical Psychology and obtained the professional degree of Psychologist in 2014.

After graduation, he worked on several research projects, most notably as coordinator of a nationally funded study on the social and affective neuroscience of Attention Deficit and Hyperactivity Disorder (Fondecyt #1150241, awarded to Dr. Vladimir López). Through this and related projects, he developed experience with electroencephalography, skin conductance responses, and eye tracking. During this period he also met Prof. Ingmar Franken, who contributed to the project as an international collaborator.

Marcelo later received a doctoral scholarship from the Chilean government (Becas Chile) and began his PhD at Erasmus University Rotterdam in September 2018. His work was supervised by Prof. Matthias Wieser, together with Prof. Ingmar Franken, Dr. Anita Harrewijn, and Dr. Marta Andreatta. Alongside his doctoral research, he collaborated with colleagues within the Department of Clinical Psychology and with international partners, contributed to teaching in the International Bachelor Programme in Psychology, and mentored and coordinated a team of master students linked to his PhD project.

After returning to Chile in 2024, he joined Universidad San Sebastián (Patagonia Campus) as a lecturer, where he has taught courses in Neuroscience and Systems Theory.

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Submitted articles

- Wong, A. H. K., Harrewijn, A., Aslanidou, A., Malbec, M., Franzen, M., Andreatta, M., & Wieser, M. J. (*under review*). Persistence of safety behaviors or the acquisition of new ones? The effect of cross-US reinstatement in safety behaviors. *Journal of Behavior Therapy and Experimental Psychiatry* (Registered Report, Stage 2).

Conference participation

- Graduate Research Day, Erasmus University Rotterdam (EUR) — Rotterdam, the Netherlands (2024; 2023; 2022) 2022 presentation: “No Intolerance of Errors”
- European Meeting on Human Fear Conditioning — Salzburg, Austria (2023); Online (2021)
- Society for Psychophysiological Research (SPR) Annual Meeting — Vancouver, Canada (2022)
- European Society for Cognitive and Affective Neuroscience (ESCAN) Meeting — Vienna, Austria (2022)

Courses

- EGSB - Professionalism and integrity in research (2021)
- Leiden Univ., TU Delft, and EUR - Data Carpentry for Social Sciences and Humanities workshop (2021)
- EGSB - English academic writing for PhD candidates (2019)
- EGSB - How to finish your PhD in time (2019)

Acknowledgements



Acknowledgements

Proving oneself as an autonomous researcher is a path that carries its share of solitude. Gladly, and proudly, I can say I found the finest companion along the way. I want to express you my gratitude.

Matthias, for your patience. For adapting just enough to my style and teaching me the ropes of high-quality science. The journey was longer than expected, pandemics and political crises included, yet you never wavered in your commitment to rigour, even when the statistical star failed to appear.

Ingmar, for letting me into your scientific world. Without your belief in me, none of this would have happened. Your confidence and practical wisdom helped me get to the finish line.

Anita, for your constant support in the second half of this journey, and for giving me the space to express how I was feeling.

Marta, for teaching me to strive for the highest level of scientific work.

EBL: Christiaan, Michel, Sharon, and Marcel (rest in peace). You offered human connection where one usually expects only refresh rates and E-Prime talk. DPECS: Alex, Minita, Ruth, Reshmi, and all those who made the ROCKS such a lively space to talk science.

Master students involved in this, especially Karla and Josh. Your assistance with data collection was essential to this thesis. Karla, your professionalism at the dawn of the pandemic was remarkable. Josh, you began as a student, became a coordinator, then a co-first author, and are now a dear academic companion and friend.

My Chilean mentor Dr. Vladimir López, you showed me that science and care are not opposites, and your friendship has been a gift. To Nerea, Diego Cosmelli, the PhD students in your labs, for welcoming me. Pablo, for being always available to help with stats or beer tastings.

Erasmus University colleagues, it is the accumulation of small moments and shared smiles that made you great. To the ping pong family: Wei, Mike, Roger, Sergio (both seasons), Mehmet DAY, Idil, Louise, and Sashank; and to Ibrahim and Sabrina. Hannes, for your openness to work and laugh together, and for looking after my kittens. To those who welcomed this Latino searching for friendship in the workplace: Job, the surfer boy; Nouran, my sister from another culture (a big hug to you); Miranda, for sharing your Latin warmth with me and my family.

Rob, for your extraordinary friendship, your laugh, and for opening the doors to your family; André, mi hermano, for always being there, for the chillings, and for curing the saudade with good music, even in your country phase. Fleur, our friendship began with Sergio's season 1 at EUR and I am glad it reached far be-

Acknowledgements

yond. All your support has meant the world. Simy, I was so lucky to have you by my side throughout this PhD. No one else could understand my journey quite like you do, having lived it alongside me. We grew together through all of it, and I admire you deeply, both as a person and in your work. Say hi to Niko!

To our friends from the hood: Eva, Anthony, and Klio; Familia Reinders, for being a family away from home. To all the Chileans who made the most of their time in the gezellig swamp: Negra, Vale, Pitu, Javier, Nicole, Ale, Luzma, Lucas, Lore, Pato, Enzo y Coni, and especially those who joined our young family and made our home warmer: Diego y Vale, Juan Chiqui y Vale Terra.

Ine, you have been my accomplice in this adventure since it was still just a dream. You are an integral part of everything built in this PhD, a partner in every sense. We turned this dream into our shared history, and most importantly, into our family. My admiration for you was a key driver to get here. Te amo mucho washita.

Gracias a ti también, Elo. Naciste de la locura que fue esta linda aventura. Gracias por tu paciencia y comprensión cuando no estaba disponible. Ahora podemos tener muchas más aventuras, ¡aunque sea en pijama!

A mi familia Malbec de Vidts, por su distante pero constante compañía. A mis padres, por enseñarme la constancia de seguir adelante y por abrirme las alas para explorar el mundo. A mis hermanos, por ser la compañía primordial, estar juntos siempre se siente como casa, sin importar bajo qué techo. A mis abuelas, por su infinito cariño. A mi otra familia, los Labarca Hoyl, gracias por su cariño y por acompañarnos en esta locura. A los Johnson.

A mis amigos del colegio: gracias por acompañar esas largas divagaciones, esta tesis es producto de esos juegos. Diego, Pablo, Tomás, Mono, Ricardo, JM y Franco, los extrañé muchísimo.

A mis nuevos colegas en la USS, por su apoyo en esta nueva etapa.

And finally, to Music, faithful friend, mood-lifter, and focus booster throughout it all.

