Learning Different Types of Associations with Immediate and Delayed Feedback: Neural Mechanisms and Clinical Perspectives

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Contents

List of Abbreviations

ACC	Anterior Cingulate Cortex
BDI-II	Beck Depression Inventory II
CS	Conditioned Stimulus
EEG	Electroencephalography
ERP	Event-Related Potential
fMRI	Functional Magnetic Resonance Imaging
$FRN_{(diff)}$	Feedback-Related Negativity (Difference Wave)
GLME	Generalized Linear Mixed Effects
ICD	International Classification of Diseases
LME	Linear Mixed Effects
DIPS	Diagnostic Interview for Psychological Disorders
MTL	Medial Temporal Lobe
PHQ-9	Patient Health Questionnaire (Mood Module)
RewP	Reward Positivity
US	Unconditioned Stimulus
VTA	Ventral Tegmental Area

Summary

Midbrain dopamine neurons encode whether feedback is better or worse than expected, sending this reinforcement learning signal to striatal and fronto-cortical regions to provide a neural basis for learning and adaptive behavior. When feedback is temporally delayed, processing appears to shift from the striatum to regions within the medial temporal lobe (MTL). This dissertation set out to shed light on the question of how the brain links feedback to specific events despite temporal gaps between them, and whether the underlying mechanisms are altered in depression. In three consecutive studies using electroencephalography, it was explored how feedback timing, different association types, and depression influence two event-related potential components—namely, the feedback-related negativity (FRN) and N170—which have been associated with feedback processing in the striatum and MTL, respectively. Across all studies, both components scaled with feedback that was better or worse than expected, interestingly reflecting the full range of prediction errors. Contrary to previous views of a mutual inhibition between striatum and MTL during feedback learning, results from Study 1 suggest that various brain regions giving rise to the FRN and N170 receive the same reinforcement learning signal and work together to support immediate and delayed feedback processing, as well as the formation of various types of associations. Study 2 further demonstrated that the N170 was especially pronounced when visual stimuli were linked to delayed feedback. This suggests that, in addition to MTL activity, the N170 may also be driven by reactivations of visual areas, providing a mechanism for linking feedback to previous events. Adding to earlier findings of an altered FRN in depression, Study 3 indicates that the N170 may serve as an additional biomarker in future clinical research. To conclude, this dissertation provides insights into fundamental mechanisms of learning and their disruption in the context of mental disorders.

1 INTRODUCTION

1.1 SUPERPOWER FEEDBACK LEARNING

As humans, we develop enormously throughout our lifetime. Born as small creatures, completely dependent on other people, we learn who our primary caregivers are and form interpersonal bonds in interaction with them. We learn that we can move things with purpose and that our actions have consequences. We even learn to use tools when our body alone is insufficient to reach a goal. At the same time, we learn that there are words for nearly everything and how to use language to speak and write. Throughout human history, we evolved to become a dominant species on Earth, unfortunately with devastating consequences for non-human animals and their environment. But what drives the progression of our abilities? A profoundly foundational observation about our behavior is that its ultimate goal is to earn reward and avoid punishment (Cohen & Blum, 2002). In other words, this implies that the outcome of an action can be good or bad. We can use these good or bad outcomes, i.e., rewards and punishments, as feedback to learn from and guide future decisions. In fact, we do not even need a real physical reward for this, but the verbal information whether we have done something right or wrong can suffice as feedback. You could even go so far as to claim that reward is enough to develop all kinds of abilities, including knowledge formation and learning, social intelligence, and language—in other words, learning from and maximizing rewards empowers intelligent behavior (Silver et al., 2021). But what is the neuronal basis that enables us to learn from reward?

1.1.1 The Reward System of the Brain

At the core of the brain's reward system are dopamine neurons with widespread projections to much larger neuron populations in the brain (for a review see Schultz & Dickinson, 2000) like for example the striatum (Oldehinkel et al., 2022), motor cortex

(Hosp et al., 2011; Luft & Schwarz, 2009) or hippocampus (Gasbarri et al., 1997). Most dopamine neurons are located in the substantia nigra and the ventral tegmental area (VTA), therefore often referred to as the dopaminergic midbrain. Early fluorescence studies suggested that their axons project to several brain regions, forming three distinct dopaminergic pathways constituting the brain‘s reward system (for a review see Björklund & Dunnett, 2007): The nigrostriatal pathway mainly projects to the dorsal striatum (caudate nucleus and putamen), while the mesolimbic and mesocortical pathways primarily project to the ventral striatum (nucleus accumbens) and cerebral cortex, especially to fronto-cortical regions (for a review see Ayano, 2016 and Glimcher, 2011). Although they are not the only dopaminergic pathways in the brain, they form the basis of the brain’s reward system (Björklund & Dunnett, 2007; Haber & Knutson, 2010).

As the central neurotransmitter of the reward system, dopamine is not only involved in reward processing and approach behavior. Since the three pathways reach various brain regions, dopamine is involved in diverse aspects of human experience and behavior, like movements, cognition, and motivation (for a review see Schultz, 2002). The importance of dopamine in the nigrostriatal pathway for smooth behavior is evident in people who have Parkinson's disease, caused by the degeneration of dopamine neurons in the substantia nigra (Damier et al., 1999; for a historical review see Parent & Parent, 2010). Most prominent are motoric symptoms like bradykinesia, which is the slowness of the initiation of voluntary movements and a progressive decline in both the speed and amplitude of repetitive motor actions. Further symptoms are tremors, muscular rigidity, and postural instability (Sveinbjornsdottir, 2016). However, the degeneration of the nigrostriatal pathway also leads to alterations in feedback learning, i.e., using outcome-based information, such as rewards or punishments, to optimize

behavior over time: Shohamy et al. (2004) found that Parkinson's patients had problems in a learning task involving feedback, but not in a task that did not contain feedback. Another study by Frank et al. (2004) found that healthy seniors learn equally well from positive vs. negative feedback, while Parkinson's patients off medication learn better from negative than positive feedback. At the same time, this bias is reversed for Parkinson's patients on medication that elevates their dopamine levels, indicating that the influence of dopamine on the striatum determines the preferred type of feedback learning. To conclude, the nigrostriatal pathway is relevant for motor functions, but also motivation, learning and choice (Collins & Frank, 2014; Hamid et al., 2016).

The mesolimbic pathway oversees the experience of pleasurable situations: The nucleus accumbens is activated by primary reinforces like food (for a meta-analysis see Tang et al., 2012), attractive faces (Cloutier et al., 2008), and sexual content (Hamann et al., 2004), as well as secondary reinforcers such as money (Knutson et al., 2001; Rademacher, 2014). In a clinical context, increased reward sensitivity in the nucleus accumbens to food and sexual stimuli is associated with overeating, impulsive sexual activity, and drug addiction (Demos et al., 2012; Di Chiara, 2002; for a review see Pierce & Kumaresan, 2006).

The mesocortical pathway, targeting (prefrontal) cortical structures, mediates cognitive and executive functions like working memory, behavioral flexibility, and decision-making (for a review see Floresco & Magyar, 2006). Given the wide-ranging and diverse functions of dopaminergic signals, the question arises which specific information dopaminergic neurons in the midbrain encode in feedback learning. Although the beforementioned studies suggest an encoding of outcome valence, i.e., good or bad outcomes, midbrain dopaminergic signals are way more refined (Schultz et al., 1997).

1.1.2 This Is Not What We Expected: The Prediction Error

In 1997, Schultz et al. summarized a series of findings from single-cell recordings in monkeys while they performed behavioral acts and received rewards. The studies revealed that midbrain dopaminergic neurons do not encode reward per se but instead indicate whether an outcome is better or worse than expected, in other words, they encode a prediction error. More precisely, they increase their action potential frequency after unexpected rewards and decrease it when an expected reward does not occur. This decrease happens exactly when the reward should have occurred—thus, dopamine neurons carry information related to both, reward predictability and timing (Schultz et al., 1997). For expected rewards, action potential frequency increases only in response to a conditioned stimulus (CS) that announces the reward, not in response to the reward itself. This phenomenon was already illustrated about a century ago through Pavlov's (1927) popular experiments on classical (pavlovian) conditioning: When Pavlov's dog first heard a bell ring (CS) and then was surprised with a snack, his dopaminergic neurons probably fired in response to the food, a rewarding unconditioned stimulus (US). However, after the dog frequently experienced that the bell predicts food, his dopaminergic neurons probably already fired when hearing the bell (accompanied by salivation). In contrast, if no food was provided although the predictive bell rang, his dopaminergic neurons most likely decreased their activity. Until the early 2000s, these dopaminergic firing patterns had only been observed in animal studies, but in 2009, Zaghloul et al. provided evidence that dopaminergic neurons in the human substantia nigra also encode a prediction error. They recorded single-cell activity during deep brain stimulation surgery while patients with Parkinson's disease engaged in a feedback-based learning task motivated by monetary rewards. The firing rate of neurons in the substantia nigra was higher for gains than losses, but this difference was

only seen for unexpected outcomes.

To conclude, reward-based learning is driven by predictions, or rather prediction errors, i.e., reward-based learning occurs only when the actual outcome differs from the prediction and the resulting prediction errors can be used to induce synaptic changes (Bao et al., 2001; Glimcher, 2011; Reynolds et al., 2001; Schultz, 2000; Schultz & Dickinson, 2000). Thus, synapses are formed or modified until an outcome can be reliably predicted. If everything occurs as predicted, there is nothing left to be learned and no need for further neuronal changes. The so-called blocking effect (Kamin, 1967) in classical conditioning illustrates this: Once Pavlolv's dog has learned that the bell announces food, pairing the bell with an additional light stimulus repeatedly will not cause the dog to salivate in response to the light when it is presented without the bell. An association between the light and the food is blocked because the bell already predicts the food. In conclusion, rewards that are better than predicted stimulate learning, fully predicted rewards do not promote further learning, and rewards that are worse than predicted result in the extinction of learned behavior (Tobler et al., 2006; for a review see Schultz, 2002 and Iordanova et al., 2021).

1.1.3 Reinforcement Learning

There are situations in which performing a certain action frequently, but not always leads to a reward, i.e., the feedback is probabilistic, so that learning can only be achieved through an accumulation of experiences (Fu & Anderson, 2008). Predictions can be helpful for choosing between alternative stimuli or actions that lead to positive or negative outcomes (Schultz et al., 1997) and reward strengthens the action that caused it, known as the Law of Effect (Thorndike, 1927; Thorndike, 1933). Using positive and negative prediction errors to make more accurate predictions about future reward and also using predictions to choose actions that maximize reward form the core of

reinforcement learning (Dayan & Balleine, 2002). Reinforcement learning theories are used to explore how humans and non-human animals choose their actions. Besides allowing to model animal behavior, they form abstract computational frameworks in computer science, machine learning, artificial intelligence and deep neural networks (Hougen & Shah, 2019; Sutton & Barto, 2018). Many of the basic reinforcement learning algorithms were inspired by psychological theories, and vice versa, these algorithms have helped to develop new animal learning models (Sutton & Barto, 2018). One example is the Rescorla-Wagner Model (Rescorla & Wagner, 1972), which describes an error-driven acquisition of associations between arbitrary stimuli, like the ring of a bell and food in classical conditioning (for a review see Miller et al., 1995 and Zhang et al., 2020). In the model, the experience with a stimulus (e.g., the ring of a bell) and its related outcomes (e.g., food vs. no food) is represented by an expected value Q, which is also an estimate of future rewards expected from the stimulus (for a review, see Curtis and Lee, 2010). Q is updated based on the difference between the actual outcome and the expected outcome, i.e., the prediction error, formulated as follows (Weber & Bellebaum, 2024):

$$\delta_t = r_t - Q_t$$

where r_t is the outcome (negative or positive) in a given trial t, and Q_t is the expected stimulus value or outcome. Q is continuously adjusted according to experience: It increases when the outcome for a given stimulus is better than expected and decreases when the outcome is worse than expected (for a review, see Curtis & Lee, 2010).

$$Q_{t+1} = Q_t + \alpha * \delta_t$$

In more detail, the prediction error is used to update the expected outcome for the next trial, Q_{t+1}, weighted with an estimated learning rate α ($0 < \alpha < 1$) which reflects the extent to which the prediction error is used to update the stimulus value (for similar

approaches and practical applications see Burnside et al., 2019; Lefebvre et al., 2017; Weber & Bellebaum, 2024). Thus, the Rescorla-Wagner Model describes that a human or non-human animal only learns when events violate its predictions (Sutton & Barto, 2018). Just like the prediction error δ in the Rescorla-Wagner Model, phasic dopamine responses in the brain indicate whether an outcome is better or worse than expected. This resemblance was the starting point leading to the idea that dopaminergic neurons encode a prediction error that can be used for synaptic changes and that underlies learning (Glimcher, 2011; Montague et al., 1996; Schultz, 2002; Schultz et al., 1997).

However, as outlined above, predictions of reward are only one side of the coin of reinforcement learning. Predicting rewards is especially useful when we can maximize them through our actions. While the Rescorla-Wagner Model focuses on using prediction errors to strengthen associations between stimuli and outcomes in classical conditioning, other reinforcement learning models focus on using prediction errors to optimize choice behavior in instrumental learning contexts. Classical and instrumental conditioning both have in common that animals learn to predict and respond to rewarding and punishing events in their environment. However, in classical conditioning, Pavlov's dog cannot influence the occurrence of food through his behavior, whereas in instrumental conditioning whether a reward or punishment is received depends on the action the animal performs (Dayan & Balleine, 2002; Sutton & Barto 2018). The Actor-Critic Model (Barto, 1995; for a review see Joel et al., 2002; O'Doherty et al., 2004; Sutton & Barto, 2018) can explain such outcome-oriented behavior in instrumental learning by distinguishing two learning processes: One process, the *critic*, is about learning which situations, events or stimuli promise rewards. The other process, the *actor*, is about learning to adjust actions to maximize rewards. The critic can criticize the actor's action choices via sending prediction errors, δ, to the

actor (Sutton & Barto, 2018). A positive δ means that the action was good because it led to an outcome better than expected; a negative δ means that the action was bad because it led to an outcome worse than expected (Sutton & Barto, 2018). These critiques enable the actor to constantly update its policy (Sutton & Barto, 2018). Learning by the critic is conceptionally similar to classical conditioning, i.e., using prediction errors to improve predictive accuracy—learning by the actor shares aspects with instrumental conditioning, trying to take actions that keep prediction errors as positive as possible (Sutton & Barto, 2018).

By differentiating between stimulus-reward (classical) and stimulus-response-reward (instrumental) learning tasks, O'Doherty et al. (2004) demonstrated that actor and critic are distributed across different parts of the basal ganglia. While the ventral striatum reflected prediction errors for stimulus-reward and stimulus-response-reward tasks, the dorsal striatum encoded prediction errors only for stimulus-response-reward tasks, in other words, tasks in which an action was required for obtaining a reward. Therefore, the ventral striatum was linked to the critic and the dorsal striatum to the actor component. In summary, the dopamine system is not only involved in the prediction of reward but also in using this information to promote behaviors that will make them more likely to occur in the future (for a review see Cohen & Blum, 2002 and Schultz, 2002).

1.1.4 The FRN: An Icon Among Feedback-Driven ERPS

Event-related potentials (ERPs) reflect brain-generated electrical signals that are triggered by specific internal or external events (Luck, 2023). These ERPs, recorded through electroencephalography (EEG) using electrodes placed on the scalp, appear as a series of positive- and negative-going waveforms (Luck, 2023). Each of these waveforms or peaks represents distinct neural processes occurring in the brain (Luck,

2023). The feedback-related negativity (FRN) is an ERP component specific to feedback processing, peaking between 200 and 300 ms after feedback onset over frontocentral electrode sites (Miltner et al., 1997). Its amplitude is pronounced in the negative direction for negative feedback, while it appears to be reduced (i.e., more positive) for positive feedback. However, recent studies suggest that the ERP response to losses and breaking even (neither winning nor losing) can be understood as the baseline response, while rewards evoke a relative positivity (Holroyd et al., 2006; Kujawa et al., 2013). Accordingly, the ERP in the FRN time window is increasingly understood as a signal that is determined by a reward positivity ([RewP]; for a review see Proudfit, 2015). In the remainder of this work, both terms (FRN and RewP) refer to the amplitude of the ERP in the time window about 200 to 300 ms after feedback. As the amplitude of this component is sensitive to the valence of feedback, it is often represented as the difference wave FRN_{diff} for negative minus positive feedback (for example see Peterburs et al., 2016; Weismüller & Bellebaum, 2016). With respect to its origin, several studies point to an error processing and action adaptation system encompassing posterior medial frontal cortex, striatum and anterior cingulate cortex ([ACC]; Becker et al., 2014; Bellebaum & Daum, 2008; Foti et al., 2011; Gehring & Willoughby, 2002; Holroyd et al., 2004; Holroyd & Coles, 2002; Oerlemans et al., 2025; for a review see Nieuwenhuis et al., 2004).

In 2002, Holroyd and Coles introduced the reinforcement learning theory of the FRN, stating that this component reflects the influence of dopaminergic neurons on neurons in the ACC: Increased activity of dopaminergic neurons, triggered by an unexpected reward, leads to the inhibition of neurons in the ACC, whereby the amplitude of the FRN becomes more positive. Conversely, reduced activity of dopaminergic neurons, triggered by the absence of an expected reward, leads to the

disinhibition of neurons in the ACC, which makes the amplitude of the FRN more negative. To conclude, Holroyd and Coles proposed that the FRN is modulated by prediction errors, the core of reinforcement learning, which has subsequently been supported by several studies: To begin with, reward magnitude can impact prediction errors because with higher rewards bigger deviations from the prediction can arise. Accordingly, the FRN has been found to be sensitive to reward magnitude (Holroyd et al., 2004). In addition, Hajcak et al. (2007) reported that the difference between negative and positive feedback was particularly evident in the FRN when feedback was unexpected. Moreover, like the prediction error, the amplitude difference between negative and positive feedback (FRN_{diff}) becomes smaller during learning (Bellebaum & Colosio, 2014; Eppinger et al., 2008). Finally, more recent studies provided evidence that the FRN is modulated by estimates of prediction errors that have been modeled computationally (Burnside et al., 2019; Fischer & Ullsperger, 2013; Weber & Bellebaum, 2024).

1.2 THE CREDIT ASSIGNMENT PROBLEM

The FRN is less sensitive to feedback valence when feedback is delayed by just a few seconds in various task types, including declarative, probabilistic, and gambling paradigms (Arbel et al., 2017; Peterburs et al., 2016; Weinberg et al., 2012; Weismüller & Bellebaum, 2016). Results of a functional magnetic resonance imaging (fMRI) study by Foerde and Shohamy (2011) might explain why differences between negative and positive feedback are less evident in the FRN when feedback is delayed: the (dorsal) striatum, target of the nigrostriatal pathway and likely part of the FRN-generating network (see above), shows stronger prediction error related activity for immediate than delayed feedback. Furthermore, Foerde and Shohamy (2011) and Foerde et al. (2013) found that the neurodegeneration of the nigrostriatal pathway in Parkinson's disease

patients leads to impaired learning from immediate, but not from delayed feedback. The striatum thus seems to be particularly important for learning from immediate feedback.

Jocham et al. (2016) found that while the lateral orbitofrontal cortex reflected causal relationships between outcomes and the choices that caused them, activity in sensorimotor corticostriatal circuitry was related to learning based on temporal proximity. Dopamine-dependent neuronal plasticity in the striatum seems to be limited to a narrow time window of up to 2 seconds (Yagishita et al., 2014) which is supported by the findings by Foerde and Shohamy (2011). If an action or stimulus is temporally close to feedback, the two can be linked by the help of the dopamine reward system. This idea is not exactly new: Aristotle declared temporal proximity as a fundamental principle of association in his work *De Memoria et Reminiscentia* (for a translation see Barnes, 2014). More than 2000 years later, Hebb (1949) suggested that synapses in the brain are formed based on the same principle that is nowadays often simplified by the popular phrase: Cells that fire together wire together. In this line, Schultz (2002) suggests that cortical areas are activated by a reward-predicting event, while reward itself broadcasts a signal from the dopaminergic midbrain to the cortex allowing synapses to form between overlappingly activated cells. Accordingly, Bao et al. (2001) were able to show that stimulating the VTA together with an auditory stimulus changes the neural responses to that sound stimulus in the primary auditory cortex.

But if only cells that fire together wire together, how can humans still learn from outcomes occurring seconds, minutes, days, or even years after a respective event? In real-life situations, feedback is often temporally separated from our choices, creating a temporal gap in neural processing. Additionally, we often perform a sequence of actions but only receive feedback at the end. Imagine cooking a meal that turns out to taste amazing. There are a lot of actions that you performed and ingredients that you used to

obtain this rewarding outcome—but which of the actions or ingredients is responsible for your success? Real-life situations therefore pose a challenge to the brain referred to as the problem of delayed reinforcement (see Sutton & Barto, 2018) or the temporal/distal credit-assignment problem (Curtis & Lee, 2010; for a review, see Stolyarova, 2018). These synonymous concepts revolve around the following question: If events and feedback are not temporally proximal, how can they still be associated with each other. In the case of neuroscience, the question is how the brain knows which event likely caused an outcome?

In general, credit for a reward can be assigned by establishing eligibility traces that can be understood as memory of previous actions and experienced stimuli (Sutton & Barto, 2018; for a review see Curtis & Lee, 2010 and Stolyarova, 2018). According to Curtis and Lee (2010), persistent neural activity might enable the short-term storage of information that is relevant for behavior, thereby supporting the computation of expected values (Q). Furthermore, they point out that persistent activity in the prefrontal cortex, posterior parietal cortex, and basal ganglia can be influenced by previous actions over repeated experiences and may represent eligibility traces. However, it seems implausible that persistent neural activity is the only way to form associations, given temporal delays of several seconds, minutes, or days for feedback in the real world.

When a prediction error occurs, only eligible stimuli or actions can be assigned credit or blame for it. Thereby, eligibility traces could help to bridge the gap between events and feedback. They are a basic mechanism for temporal credit assignment and as Sutton and Barto (2018, p. 317) formulated metaphorically "the first line of defense against long-delayed rewards." But how does a stimulus or action become eligible? How are eligibility traces for long-delayed rewards implemented in the brain? When it comes to delayed feedback and remembering which stimulus or action caused it, we

need support from structures other than the striatum.

1.2.1 The Medial Temporal Lobe

The medial temporal lobe (MTL) includes the hippocampus, adjacent perirhinal, entorhinal, and parahippocampal cortex (Raslau et al., 2015). Probably the most prominent and dramatic example of MTL and particularly hippocampal (dys-) functioning is patient H.M. In 1957, Scoville and Milner published an article in which they described a case study of a patient who suffered from epileptic seizures. At the time, a bilateral MTL resection was considered a justifiable treatment for his condition. Unfortunately, the operation caused strong anterograde amnesia, i.e., a complete loss of memory for events following the resection, combined with partial retrograde amnesia for the three years before his operation, while early memories, as well as his personality and general intelligence, were seemingly intact. In the following decades, the hippocampus emerged as a central structure for declarative and episodic long-term memory and learning (Eichenbaum et al., 1992; Knowlton et al., 1996; Squire et al., 1989; Tulving & Markowitsch, 1998). Within the MTL, different types of information are brought together to form cohesive memory episodes (Sugar & Moser, 2019), contextual associations (Aminoff et al., 2013), and associations of events non-overlapping in time (Qin et al., 2007). The MTL could therefore form a bridge between events and feedback when they are separated in time (Qin et al., 2007; Staresina & Davachi, 2009).

Supporting this, amnestic patients with hippocampal lesions can only learn via classical conditioning when CS and US overlap, but not when there is a temporal gap between them (Clark & Squire, 1998). Moreover, amnestic patients with lesions in the hippocampus were able to learn from immediate but not from delayed feedback, thus showing opposite deficits compared to Parkinson's disease patients (Foerde et al.,

2013). In accordance, Foerde and Shohamy (2011) found stronger prediction error related activity in the hippocampus for delayed compared to immediate feedback. Thus, while learning from immediate feedback involves the striatum, learning from delayed feedback recruits the hippocampus. The hippocampus is innervated by midbrain dopamine neurons (for a review, see Edelmann & Lessmann, 2018; Gasbarri et al., 1994; Tsetsenis et al., 2021) and correlates of prediction error activity have been found in regions receiving input from dopaminergic projections (Garrison et al., 2013). Accordingly, Dickerson et al. (2011) reported prediction error related activity not only within the striatum but also within the hippocampus during probabilistic feedback learning. Therefore, it was suggested that prediction error signals in the hippocampus might mirror phasic dopaminergic inputs, similar to the striatum (Foerde & Shohamy, 2011).

1.2.2 The N170: A Newcomer Among Feedback-Driven ERPs

Strengthening the picture of a shift away from fronto-striatal circuits toward MTL involvement including the hippocampus with increasing feedback delay painted by patient and imaging studies, EEG studies also suggest that a few seconds delay is enough for feedback to be processed differently (Arbel et al., 2017; Kim & Arbel, 2019; Peterburs et al., 2016). Offering the benefit of high temporal resolution, i.e., the precision of milliseconds, EEG is a suitable method to provide insights into immediate and delayed feedback processing (Kim & Arbel, 2019). The N170 is an ERP component measured over occipitotemporal sites that is more pronounced following delayed than immediate feedback (Arbel et al., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019). Consequently, it was hypothesized that while the FRN reflects striatal activity evoked by immediate feedback, the N170 could reflect the involvement of the MTL in delayed feedback processing (Arbel et al., 2017; Kim & Arbel, 2019). However,

historically the N170 became popular in the context of higher-order visual processes; it is for example particularly pronounced for faces (Bentin et al., 1996; Itier & Taylor, 2004; for a review see Yovel, 2016) and words (for a review see Carreiras et al., 2014), but also other stimulus categories like for example cars (Kloth et al., 2013).

For the N170 peaking over the occipitotemporal cortex about 170 ms after face presentation (for a review see Sigurdardottir et al., 2021), an origin in the fusiform gyrus was found (Deffke et al., 2007; Gao et al., 2019; Iidaka et al., 2006). As part of the ventral stream of visual information processing (Goodale & Milner, 1992), the fusiform gyrus contains specialized regions for numerous visual stimuli (Hoffman & Haxby, 2000; Kanwisher et al., 1997; Puce et al., 1995; for a review see Weiner & Zilles, 2016). For example, the fusiform face area is specialized for face processing (for a review see Kanwisher & Yovel, 2006). For the N170 following words, sources were found near the left fusiform gyrus (Brem et al., 2006), which contains the visual word form area (Cohen et al., 2002).

These source localization studies on the N170 in the context of visual processing suggest that it represents activity in higher-order visual areas. However, studies investigating the N170 in the context of navigational feedback learning found that it is sensitive to the spatial location of reward stimuli in a maze and linked it to activity within the right MTL, or more precisely, the right parahippocampal cortex (Baker & Holroyd, 2009, 2013; Baker et al., 2015). As no source localization studies on the N170 in the context of feedback processing exist yet, there are only speculations regarding its neural generator. It is, however, conceivable that the enhanced N170 following delayed feedback also results from activity in higher-order visual areas. While it has previously been hypothesized that the N170 results from a delayed reward signal transmitted to the MTL to reinforce a memory representation of a stimulus stored there (Arbel et al., 2017;

Kim & Arbel, 2019), visual areas might as well contribute to the memory representation of a stimulus. Kim and Arbel (2019) found that pronounced N170 amplitudes following delayed feedback are not specific to visual feedback stimuli, but also evident for auditory feedback. If the N170 reflects activity in higher-order visual areas, it might be generated by reactivating the visual stimuli that preceded the feedback. Reactivating representations of previously chosen stimuli in higher-order visual areas when feedback is delayed could be a way to solve the temporal credit assignment problem. Imagine a man who enjoys eating bananas. One day, he peels a banana, discards the skin, eats the fruit, and finds it exceptionally delicious. The image of the banana's slightly green skin lingers in his mind, and from that moment on, he only buys green bananas. While savoring the fruit, his brain associates the delightful taste with the visual characteristics of the banana's skin—although it was no longer visible because it was in the trash already. In this example, the image of the green skin was reactivated in the man's brain, linking it to the pleasure of the taste.

1.2.3 Sensory Reactivation as a Solution for the Credit Assignment Problem

Singer and Frank (2009) emphasize the importance of remembering experiences that lead to reward for survival, yet the mechanisms linking specific experiences to rewarding outcomes are poorly understood. The authors suggest that some kind of reactivation triggered by reward might serve as a mechanism for linking rewarding outcomes to preceding experiences. A form of neuronal reactivation has already been discovered in the field of memory research: Sensory brain regions active during the encoding of somatosensory, acoustic or visual stimuli become partially active again when participants recall the stimuli (Wheeler et al., 2000; for a review see Danker & Anderson, 2010). In addition, fMRI data revealed overlapping activation during melody perception and imagery, involving secondary auditory areas (Herholz et al., 2012).

Another fMRI study by Schiffer et al. (2014) suggests that a similar mechanism applies to feedback processing. Their participants had to discriminate visual stimuli according to their category (face vs. house). Later reward reactivated stimulus-specific representations in visual association cortices, particularly in the fusiform face area. They concluded that reactivating representations of rewarded stimuli in sensory cortices upon reward presentation could represent eligibility traces for credit assignment. Participants in an fMRI study by Pleger et al. (2008) had to discriminate somatosensory stimuli regarding their frequency (high vs. low) and were rewarded for correct judgments. Interestingly, the primary somatosensory cortex was reactivated when reward was presented, without somatosensory input at that moment. Results from a follow-up study indicate that the reactivation is mediated by dopamine (Pleger et al., 2009): While levodopa (dopamine agonist) increased the somatosensory reactivation, it was decreased under haloperidol (dopamine antagonist). Qin et al. (2007) identified brain regions engaged in storing associations of events that do not overlap in time into long-term memory using fMRI. They found that the prefrontal cortex and MTL, especially the hippocampus, engaged in associative memory formation. Based on their results, they suggest that signals from the MTL may initiate the reactivation of an internal representation of an event, thus allowing it to be associated with a later event. Taken together, findings on the involvement of MTL structures and sensory cortices in (delayed) feedback processing suggest that the MTL might mediate a reactivation of sensory areas to specifically link stimuli to delayed feedback.

1.2.4 The Underrated Role of Association Types

So far, this introduction has focused primarily on the influence of feedback timing on learning from feedback, while another possibly important determinant has been neglected: the type of association we learn. We can associate all kinds of events

with feedback. Remember the example of cooking a meal, where both ingredients and actions can lead to a delicious result. At this point it is important to emphasize that both actions and stimuli can be linked to feedback. McDougle et al. (2016) illustrate this with another vivid example: When a person reaches across the table and knocks over her coffee, the omission of the anticipated rewarding taste of coffee should be attributed to her action rather than reduce her love for coffee. But how is the action and not the coffee be blamed for the mishap at the neuronal level? Could different neuronal structures be involved in forming associations between actions or stimuli and feedback? If we look at the structures involved in processing immediate and delayed feedback more precisely, we might notice the following: The striatum has properties that predestine it for linking actions with feedback, while the hippocampus could be helpful for linking stimuli with feedback.

To begin, let us review empirical evidence for the anatomical and functional proximity of the striatum to motor function, movement, and action execution during feedback-based learning: In a study by Haruno and Kawato (2006), correlates of stimulus-action-reward associations were located in parts of the striatum. Hiebert et al. (2014) found that during stimulus-response learning, activity in the dorsal striatum correlated with response selection, while ventral striatum activation correlated with feedback. Reynolds et al. (2001) studied intracranial self-stimulation in rats, where each rat learned to press a lever that delivered rewarding electrical stimulation to its own substantia nigra. The stimulation of the substantia nigra caused a potentiation of synapses between the cortex and the striatum and the extent of the potentiation was linked to the time it took the rats to learn to press the lever. fMRI has shown that the striatum gates sensory information transfer to the premotor cortex, potentially shaping motor selection (den Ouden et al., 2010). Shohamy (2011) proposed that the striatum is

part of a broader network that guides action selection based on predicted values, thereby optimizing behavior. Furthermore, reward feedback has been found to enhance motor learning (Nikooyan & Ahmed, 2015), with non-invasive striatal stimulation being able to facilitate (Wessel et al., 2023) or impair it (Vassiliadis et al., 2024). In an ERP study, valence-sensitive activity following feedback was found over the motor cortex (Cohen & Ranganath, 2007), which might suggest a reactivation of a previous action during striatum-based feedback learning. Moreover, a study by Yeung et al. (2005) found that the FRN_{diff} was larger when a reward followed an own active choice instead of an (automatic) random choice. However, it must be put to the test whether the FRN as a potential reflection of striatal activity is indeed more pronounced when feedback refers to an action as opposed to a stimulus.

After discussing a potentially distinct role of the striatum in action–feedback associations, let us now briefly review why the MTL or hippocampus might be critical for linking feedback to stimuli: As outlined earlier, the hippocampus may serve as a bridge between temporally separated stimuli and feedback (Qin et al., 2007; Staresina & Davachi, 2009) by reactivating internal representations of reward predicting events and enabling their association with later outcomes (Qin et al., 2007). In other words, when feedback is presented, the hippocampus may reactivate the representation of a preceding stimulus. However, it is unclear whether the hippocampus itself contains representations of stimuli across different sensory modalities or whether they are stored in modality-specific sensory areas and reactivated by the MTL. The reactivation of higher-order visual areas could play a role in generating the feedback-locked N170 to assign delayed feedback to a previously selected stimulus. In a first more general step, it must be put to the test whether the N170 is indeed more pronounced when feedback refers to a stimulus as opposed to an action. Subsequently, the influence of the modality of a

feedback-preceding stimulus on feedback processing needs to be investigated. The question that arises is whether the N170 is particularly pronounced for feedback that refers to visual stimuli.

1.3 CLINICAL ALTERATIONS IN FEEDBACK LEARNING

1.3.1 Modernity's Burden? A Society Struggling with Depression

On behalf of the World Health Organization, the Global Burden of Disease study investigated mortality and disability caused by diseases and injuries all over the world (Murray & Lopez, 1996). The study concluded that in developing regions of the world, noninfectious diseases such as heart disease, but also mental disorders like depression are expected to slowly replace infectious diseases and malnutrition as primary causes of disability and premature death (Murray & Lopez, 1996). Today, mental disorders like depression are one of the leading causes of years lived with disability, a measure used to quantify the burden of disease (Vos et al., 2012). In Germany, for example, around one in five to six adults is affected by depression at some point in their life (Jacobi et al., 2004). With a prevalence between 8% and 9% (Busch et al., 2013; Hapke et al., 2019), we can infer that right now, over six million adults are affected by depressive symptoms in Germany alone. This poses a serious challenge for society: Depression is associated with high healthcare utilization and spending and enormous economic costs, for example due to sick leave and reduced productivity at work (for a review see Donohue & Pincus, 2007 and Wang et al., 2003). But most importantly, depression is associated with huge personal costs in terms of pain and suffering for those affected, their families and friends.

According to the eleventh version of the International Classification of Diseases ([ICD-11], World Health Organization, 2019), depressive disorders “are characterized by depressive mood (e.g., sad, irritable, empty) or loss of pleasure accompanied by

other cognitive, behavioral, or neurovegetative symptoms that significantly affect the individual's ability to function." In more detail, a depressive episode "is characterized by a period of depressed mood or diminished interest in activities occurring most of the day, nearly every day during a period lasting at least two weeks", and is "accompanied by other symptoms such as difficulty concentrating, feelings of worthlessness or excessive or inappropriate guilt, hopelessness, recurrent thoughts of death or suicide, changes in appetite or sleep, psychomotor agitation or retardation, and reduced energy or fatigue." Depressive symptoms have probably existed for as long as humans have been around. Under the term melancholia, depressive moods were studied in ancient medicine by Hippocrates (see Orfanos, 2007) and already considered an epidemic disease in the 19th century (Burton, 1857). In a review from 2012, Hidaka concludes that there is epidemiologic evidence that the prevalence of depression has increased over the past century, and that modernization is linked to higher rates of depression. He explains the problem in clear terms (p. 205): "Modern populations are increasingly overfed, malnourished, sedentary, sunlight-deficient, sleep-deprived, and socially isolated." Therefore, modern lifestyle is blamed for fueling poor physical and mental health and the increasing incidence of depression.

1.3.2 Anhedonia and Altered Feedback Learning in Depression

The term anhedonia comes from the Greek and means "without joy/pleasure". It was first introduced by Ribot (1896) as a loss of pleasure and linked to mental conditions like depression. Today, diminished interest or diminished pleasure in all, or almost all, activities is defined as a cardinal symptom of depression (American Psychiatric Association, 2022). Anhedonia affects the experience of rewards, including the formation of stimulus-reward associations, reward anticipation, hedonic responses (i.e., enjoyment of reward), and the updating of reward values (Rizvi et al., 2016). Thus,

anhedonia may account for learning deficits seen in depression in situations where reward processing and utilization are essential, such as in reinforcement learning (Bakic et al., 2017). Accordingly, studies by Kumar et al. (2018) and Admon et al. (2017) indicated reduced reward learning in depressed participants. Huys et al. (2013) performed a meta-analysis on behavioral data from probabilistic reward tasks and used reinforcement learning models to isolate learning rate and reward sensitivity, i.e., the subjective value of an external reward. They concluded that depression and anhedonia did not strongly affect the learning rate per se, but specifically reduced reward sensitivity. In this line, Kunisato et al. (2012) found that depressed participants seem to have no learning deficits per se, but rather a diminished tendency to base their decisions on the likelihood of receiving rewards. Besides hyposensitive responses to reward, depressed individuals also show maladaptive responses to punishment (for a review see Eshel & Roiser, 2010): As soon as depressed participants commit a mistake in a task, their performance drops sharply, i.e., further mistakes become more likely (Elliott et al., 1996, 1997). In their review, Eshel and Roiser (2010) provide two explanations for this phenomenom: A hypersensitivity to negative feedback could trigger thoughts about future failure and interfere with performance. Alternatively, depressed individuals might be unable to use negative feedback to learn from it and adapt their behavior. Furthermore, not only clinically depressed individuals (Pizzagalli et al., 2008), but also non-clinical individuals with elevated depressive symptoms (Pizzagalli et al., 2005) and remitted individuals (Pechtel et al., 2013) were impaired at modulating their behavior as a function of previously received reinforcements in probabilistic tasks. Reviewing computational research, Chen et al. (2015) concluded that depression is related to altered brain signals of reward prediction errors and expected values and/or learning, while the causality is unclear.

1.3.3 Depression-Related Physiological Changes in Feedback Processing

The range of symptoms in depression implies that multiple synaptic circuits and brain regions are affected because a single circuit cannot explain the entirety of the depressed phenotype (for a review see Nestler et al., 2002 and Thompson, 2023). To decompose complex neuropsychiatric diseases, Gottesman and Gould (2003) introduced the concept of endophenotypes to psychiatry: Endophenotypes lie along the pathway between a heterogenous disease and a distal, complex genotype, providing simpler indications of the genetic basis than the disease syndrome itself. They are neurobiological disease correlates that are stable over time, genetically influenced, and inheritable (Zobel & Maier, 2004). Altered behavioral responses to rewards and punishments in depression correspond to deviant functioning in fronto-striatal systems (Eshel & Roiser, 2010), which might represent an insightful endophenotype for depression (Luking et al., 2016).

In more detail, hypo-function of striatal regions in depression is consistently observed across various reward-related processes, highlighting striatal hypo-function as a key neural mediator underlying altered reward processing in depression (for a review see Admon & Pizzagalli, 2015). While striatal activity in healthy individuals has been found to increase in proportion to the size of monetary reward during reward anticipation, depressed individuals showed less adjustment of neural responses for variable amounts of reward (Takamura et al., 2017). Furthermore, in an fMRI study by Kumar et al. (2018) depressed individuals showed deficits in reward learning, blunted reward prediction error signaling in the striatum, and reduced VTA-striatal connectivity to feedback relative to controls. In the same study, striatal reward prediction error signals became more blunted with an increasing number of depressive episodes. In a study by Pizzagalli et al. (2009) participants with major depression showed significantly

reduced activation to reward in the left nucleus accumbens and the caudate nucleus bilaterally compared to healthy comparison subjects. While under healthy conditions, the nucleus accumbens and its dopaminergic inputs from the VTA are important for mood regulation, they might also mediate symptoms like low mood in depression (Nestler et al., 2002). Accordingly, the nucleus accumbens and VTA play an important role in the pathophysiology and symptomatology of depression (Nestler & Carlezon, 2006), especially in the context of dysfunctional reward processing (Admon & Pizzagalli, 2015) and reward learning (Admon et al., 2017) and may even be involved in its etiology. The latter is supported by studies that investigated children of depressed mothers, who are at increased risk for developing depression (for a review see Luking et al., 2016): Without being depressed themselves, these children also show blunted responses to reward within the dorsal and ventral striatum compared to less vulnerable peers. This reduced striatal activation was found to precede the onset of depression in adolescents (for a meta-analytic review see Keren et al., 2018). In addition, some studies report that high-risk groups also showed enhanced responses to negative feedback within similar regions (for a review see Luking et al., 2016). Luking et al. (2016) conclude that alterations in feedback processing occurring prior to the onset of depressive symptoms suggest that these changes may contribute to the etiology of depression, rather than result from it.

In line with significantly reduced striatal activation in depressed compared to healthy individuals during reward feedback in fMRI studies, EEG studies revealed that the differentiation between gains and losses in the FRN amplitude was also significantly reduced in depression, and that the blunting was stronger in individuals under age 18 compared to those 18 and older (Foti et al., 2014; for a meta-analytic review see Keren et al., 2018). In several studies, the association between depression and altered FRN

amplitudes was mainly driven by blunted neural response to reward (Bress et al., 2012, 2013, 2015; Brush et al., 2018; Foti et al., 2014). Bress et al. (2012) found that in children, an increase in both self-rated and parent-rated depressive symptom scores was linked to a reduction in the FRN following rewards. Furthermore, a blunted FRN_{diff} in healthy adolescent girls was able to predict the onset of new major depressive episodes and depressive symptoms (Bress et al., 2013). Another longitudinal study of this group found that the FRN for losses and the FRN for gains were stable over a timespan of approximately two years, and moreover the relationship between a blunted FRN_{diff} and more severe depression was reproduced after two years, and smaller FRN_{diff} amplitudes at the first observation predicted more severe depressive symptomatology after two years (Bress et al., 2015). To conclude, blunted FRN amplitudes following rewards may be useful as a biomarker for depression (Proudfit, 2015; but see Hager et al., 2022).

Because depression is multifaceted, there is neither a single brain region that is correlated with all facets, nor can one region be identified as their sole cause (Greenberg, 2010). The heterogeneity of the clinical picture implies that other structures apart from the dopaminergic midbrain and striatum also contribute to the various symptoms seen in depression (for a review see Nestler et al., 2002): For example, impaired hippocampal functioning might contribute to some of the cognitive deficits seen in depression. Overlapping symptoms between Alzheimer's disease and major depressive episodes (e.g., difficulty thinking and concentrating) led to the birth of the term depressive pseudodementia in the past (Caine, 1981; Raskind, 1998). Nowadays, hippocampal atrophy accompanying depression is held responsible for the observed memory deficits (Fairhall et al., 2010; Shah et al., 1998; for a review see Thompson, 2023). The atrophy could be caused by stress either from traumatic life events or internal stressors like excessive rumination (for a review, see Thompson, 2023). The

brain reacts to acute and chronic stress by activating the hypothalamic-pituitary-adrenal axis, which under conditions of prolonged and severe stress may damage hippocampal neurons (for a review see Nestler et al., 2002). Depressed individuals typically show altered recollection with impaired memory for positive events, but enhanced memory for negative events (for a review see Dillon & Pizzagalli, 2018). The hippocampus sends information to and receives inputs from numerous brain regions in the reward circuit, therefore, its role in feedback processing might also be affected by depression (for a review see Heshmati & Russo, 2015). So far, research on depression has focused on striatal changes and reduced $FRN_{(diff)}$ amplitudes during feedback processing. However, there is a lack of knowledge when it comes to delayed feedback processing that involves the hippocampus (Foerde et al., 2013; Foerde & Shohamy, 2011) and evokes pronounced N170 amplitudes (Arbel et al., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019). Considering the reports of hippocampal atrophy, changes in the processing of delayed feedback seem just as possible as those found for the processing of immediate feedback. To complete the picture, it needs to be investigated whether parallel to changes in the FRN following immediate feedback, there are also changes in the N170 following delayed feedback in the context of depression.

1.4 RESEARCH QUESTIONS AND OBJECTIVES

Based on previous findings regarding effects of feedback timing on feedback processing as measured by EEG, this dissertation aims to further investigate how temporal aspects of feedback presentation influence neural responses in interaction with other factors. In particular, it seeks to examine the role of different association types in shaping feedback-related brain activity. Specifically, it will explore whether the FRN is more closely linked to action-feedback associations, while the N170 component might be more responsive to stimulus-feedback associations. A central focus will also be

placed on further clarifying the functional significance of the N170 in the context of feedback processing. One key question is whether the feedback-locked N170 could reflect activity in visual areas as a mechanism to link visual stimuli to (delayed) feedback. Finally, this dissertation addresses the broader relevance of feedback learning and processing in the clinical context of depression. By aiming to replicate previously reported alterations in FRN amplitudes following immediate feedback in individuals experiencing depressive symptoms and exploring potential similar changes in the N170 following delayed feedback, this dissertation sets out to contribute to a more differentiated understanding of the neural mechanisms underlying impaired feedback learning and processing in depression.

2 OVERVIEW OF STUDIES

This dissertation aims to deepen our understanding of the neural mechanisms involved in processing immediate and delayed feedback and to explore their relevance to the clinical condition of depression. The foundation for this is three experimental studies using EEG to investigate the neural processing of immediate and delayed feedback as reflected in the FRN and N170 in probabilistic learning tasks. Studies 1 and 2 were designed to gain insights into how the human brain deals with (and solves) the credit assignment problem by investigating different types of associations. The first study aimed to shed more light on differences in the neural processing of feedback linked to actions compared to feedback linked to stimuli. In the second study, particular attention was given to the hypothesis that the reactivation of higher-order visual areas could play a role in generating the feedback-locked N170 to assign delayed feedback to a previously selected (visual) stimulus. Therefore, the second study aimed to investigate the extent to which the sensory modality of a stimulus (i.e., visual vs. auditory) that needs to be associated with feedback influences the processing of feedback and particularly whether the N170 is increased for feedback that refers to visual stimuli. Study 3 focused on the potential clinical relevance of neural feedback processing in depression and intended to extend findings of altered feedback processing reflected in the FRN to the processing of delayed feedback and the N170. The following section offers a concise overview of each of the three studies. A detailed description of the studies is provided in the original research articles in Appendix C.

2.1 STUDY 1

2.1.1 Research Question and Hypotheses

Learning from immediate feedback is associated with striatal activity, presumably leading to pronounced differences between negative and positive feedback

in the FRN—in contrast, the hippocampus becomes active when delayed feedback is processed, and a more pronounced N170 can be observed (Arbel et al., 2017; Foerde et al., 2013; Foerde & Shohamy, 2011; Höltje & Mecklinger, 2020; Kim & Arbel, 2019; Peterburs et al., 2016). However, other factors besides feedback timing might also modulate the involvement of striatum and hippocampus in feedback processing. Being able to associate feedback with a specific stimulus we have sensed or an action we have performed is crucial to predict rewards and enable adaptive behavior. Still, the underlying neural processing might vary depending on the type of learned association. The purpose of this study was to compare the processing of feedback related to a stimulus with the processing of feedback related to an action. Ultimately, we wanted to get closer to an answer to the question of how the human brain solves the credit assignment problem. Therefore, we had participants learn different types of associations (action-feedback and stimulus-feedback) with different feedback timings (immediate feedback and delayed feedback) while recording EEG. Due to the central role of the striatum for reward-based motor learning (Nikooyan & Ahmed, 2015; Vassiliadis et al., 2024; Wessel et al., 2023) and immediate feedback processing (Foerde & Shohamy, 2011; Foerde et al., 2013) we expected that the assignment of feedback to actions rather involves the striatal system, especially in the case of immediate feedback. On the other hand, we expected the hippocampal system to be especially involved in linking feedback to stimuli. More precisely, the MTL may reactivate an internal representation of an earlier event, allowing it to be associated with a later event (Qin et al., 2007). Thus, when a visual stimulus is followed by feedback, the MTL may help to reactivate the representation of the stimulus when feedback is presented to bridge the temporal gap caused by the delay. Combining the (potential) influences of feedback timing and association type, we hypothesized the largest difference between negative and positive

feedback and the strongest reflection of the prediction error in the FRN (assumed to reflect striatal activity) for immediate feedback that refers to actions. In contrast, we hypothesized to see the largest N170 (assumed to reflect hippocampal and/or visual activity) and the strongest prediction error reflection in the N170 for delayed feedback that relates to stimuli.

2.1.2 Methods

78 healthy adults underwent a computer-based probabilistic feedback learning task including monetary rewards (+ 4 cents) and punishments (- 2 cents). Due to technical problems, poor learning performance and low EEG data quality, only 62 participants (40 women, 21 men, and 1 non-binary person, mean age = 23.66 years, *SD* = 3.87 years) were included in the analyses. Participants were randomly assigned to one of three experimental conditions, between which the type of association to be learned was varied: 18 participants learned action-feedback associations, 22 learned stimulus-feedback associations actively (by choosing between two stimuli themselves), and 22 learned stimulus-feedback associations passively (by observing how one stimulus was automatically chosen to rule out motoric actions).

In more detail, participants in the action-feedback condition were asked to choose between two specific actions in every trial to press either the left or right button of a response box (e.g., pressing the right button with their right thumb vs. the left button with their left thumb). The two actions were represented by two identical rectangles on the screen's right and left side. After their choice, participants received monetary feedback for their action. By forming action-feedback associations, their task was to learn which action led to more frequent rewards and maximize their earnings.

In the active stimulus-feedback condition, participants were asked to choose between two visual stimuli (hiragana-like characters) in each trial by pressing either the

left or right button of a response box. After their choice, monetary feedback was presented, and participants were instructed to learn which stimulus was rewarded more frequently to maximize their earnings. Thus, this condition was similar to the action-feedback condition, because it also contained actions, but the important difference was that the feedback referred to the stimuli, not the actions. Therefore, participants had to form stimulus-feedback associations.

The task for participants of the passive stimulus-feedback condition was similar to the task for the active stimulus-feedback condition, with the only difference that participants could not choose between the stimuli themselves. Instead, one of the stimuli was automatically highlighted by a red circle to indicate a choice. Subsequently, positive or negative monetary feedback was presented. Again, the participant's task was to form stimulus-feedback associations and to learn which stimulus was rewarded more often. However, this task contained no actions and participants could only maximize their earnings in subsequent test trials, in which they could choose between the two stimuli themselves without receiving feedback. These test trials allowed us to assess wether participants learned to choose the more rewarding stimulus more frequently over time. For comparability reasons, test trials were also included in the action-feedback condition and in the active stimulus-feedback condition.

To conclude, either motor aspects of the task (action-feedback condition) or stimulus identity (active and passive stimulus-feedback condition) predicted reward. Unbeknown to the participants, one action or stimulus of every pair presented was slightly better than the other in the sense that it led to a reward in 65% of the trials and a loss in 35%; for the other action or stimulus, the probabilities were reversed. Within each association type condition, feedback timing was also manipulated: In one condition, feedback was given immediately (immediate feedback; 1 s after the event),

while in a second condition it was given delayed (delayed feedback; 7 s after the event). To control for effects of reduced temporal predictability of delayed feedback, a third condition contained a feedback delay of 7 seconds with six regular tones presented during the delay, one per second (Kimura & Kimura, 2016). The two conditions with delayed feedback will be referred to as delayed feedback without tone and delayed feedback with tone.

In each of the three association type conditions, participants learned under the three feedback timing conditions (immediate feedback, delayed feedback without tone, and delayed feedback with tone). The order of these feedback timing conditions was counterbalanced across participants to minimize sequence effects. Each participant learned with a total of six pairs of actions or stimuli, with two pairs assigned to each feedback timing condition. For each pair, participants completed four learning blocks, with each block consisting of 20 learning trials, followed by 20 test trials (in which no feedback was presented). Overall, participants completed 960 trials: 480 learning trials (20 trials × 4 blocks × 6 pairs) and 480 test trials (20 trials × 4 blocks × 6 pairs). Feedback timing remained constant within each pair but changed only when a new pair of actions or stimuli was introduced. During the task, EEG was recorded from 60 scalp electrodes evenly distributed across the head based on the extended 10-20 system.

We first analyzed the behavioral single trial data to see if participants successfully learned to form action-feedback and stimulus-feedback associations. We checked whether they learned to choose the more rewarding action or stimulus more frequently by comparing their choices to the 50% chance level with a single-sample *t*-test. Only in the passive stimulus-feedback condition, accuracy levels were not significantly above chance level. Therefore, we focused all following analyses on the comparison of the action-feedback and active stimulus-feedback condition and added an

exploratory analysis comparing the action-feedback and passive-stimulus feedback condition to the appendix. To explore differences in learning performance depending on certain conditions, we applied a generalized linear mixed effects (GLME; Bates et al., 2015) model suitable for binomial distributions in R (The R Foundation, 2021). Specifically, we explored effects of feedback timing (immediate, delayed without tone, delayed with tone), association type (action-feedback vs. active stimulus-feedback), and learning block (1-4) on response accuracy, i.e., how often participants chose the more rewarding stimulus. Furthermore, we used the behavioral data to model the prediction error for each trial by computationally fitting a reinforcement learning model (similar to the one described in the Introduction section 1.1.3) to the participants' choices and their received feedback.

To allow a meaningful analysis of the EEG data, we first ran a standard preprocessing procedure in BrainVision Analyzer 2.2 (Brain Products GmbH, 2018) that cleaned the data from artifacts. Afterwards, we extracted single trial amplitude values for the FRN from electrodes Fz, FCz, Cz, FC1 and FC2 at the latency of the maximum negative peak in the FRN_{diff} in a time-window ranging from 200 to 400 ms post feedback and for the N170 from electrodes P7 and P8 between 140 and 250 ms post feedback using MATLAB (R2021a, The MathWorks, Inc., 2021). Consequently, FRN and N170 single trial amplitude values were separately analyzed as dependent variables by applying linear mixed effects (LME) models in R (Bates et al., 2015; The R Foundation, 2021). Specifically, we investigated effects of association type (action-feedback vs. active stimulus-feedback), feedback timing (immediate, delayed with tone, delayed without tone), feedback valence (negative vs. positive) and the unsigned prediction error, which represents general surprise but in combination with feedback valence enabled us to explore representations of the whole range of the prediction error,

i.e., the signed prediction error (Weber & Bellebaum, 2024).

2.1.3 Results and Discussion

The GLME analysis of the behavioral data revealed that overall, participants improved their response accuracy across the four learning blocks, i.e., they learned to choose the more rewarding stimulus (active stimulus-feedback condition) or perform the more rewarding action (action-feedback condition) more frequently through the course of the experimental task. However, participants in the active stimulus-feedback condition showed significantly enhanced response accuracies compared to participants in the action-feedback condition.

For the FRN, the LME analysis replicated findings of more negative amplitudes for negative than positive feedback, with this difference being larger for immediate than delayed feedback (both with and without tone; for similar results, see Albrecht et al., 2023; Arbel et al., 2017; Höltje & Mecklinger, 2020; Peterburs et al., 2016; Weinberg et al., 2012; Weismüller & Bellebaum, 2016). However, against our hypothesis, this interaction between feedback valence and timing was not stronger for action-feedback associations than stimulus-feedback associations. For the N170, we were able to replicate more pronounced amplitudes following delayed compared to immediate feedback (Arbel et a., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019). In addition, we observed more pronounced amplitudes for negative than positive feedback (see Kim & Arbel, 2019), but in contrast to the FRN, this difference was only observable for delayed feedback. However, neither the effect of feedback timing, nor the interaction between feedback valence and timing was affected by the association type—in other words, we could not find evidence for our hypothesis that effects of feedback timing should be more pronounced in the N170 for stimulus-feedback associations.

The hypothesized effects of the association type on feedback processing only emerged in interaction with the prediction error. For both, FRN and N170, we expected that the reflection of the prediction error (interaction between unsigned prediction error and feedback valence) would depend on feedback timing and association type. For the FRN following positive feedback, amplitudes became more positive with increasing prediction errors, i.e., for more unexpected feedback. For negative feedback, the FRN amplitudes became more negative the higher the prediction error was (for similar effects see Burnside et al., 2019; Fischer & Ullsperger, 2013). Hence, the FRN reflected the whole range of prediction error coding as suggested by the Reinforcement Learning Theory (Holroyd & Coles, 2002). In contrast, previous studies found prediction error reflections in the FRN especially for positive feedback (Kirsch et al., 2022; Weber & Bellebaum, 2024). Interestingly, prediction error coding in our study was more pronounced for immediate than delayed feedback (both with and without tone). Again, this differed from findings by Weber and Bellebaum (2024) indicating similar prediction error coding in the FRN following immediate and delayed feedback. Finally, the interaction between feedback valence and prediction error was also more pronounced for active stimulus-feedback associations compared to action-feedback associations. More precisely, the FRN in the active stimulus-feedback condition reflected the whole range of the prediction error (as described above), while in the action-feedback condition it only scaled with positive prediction errors, similar to the pattern reported by Kirsch et al. (2022) and Weber and Bellebaum (2024). Thus, for action-feedback associations prediction error coding was restricted to positive feedback, indicating that for negative feedback, it did not make a difference (at least in the FRN) whether it was unexpected or not. Since prediction errors pose a bear necessity for learning (Schultz & Dickinson, 2000), these findings in the action-feedback condition

align with the typical observation that participants tend to learn better from positive than negative feedback (see Weber & Bellebaum, 2024). Our results can be interpreted as demonstrating that for reinforcement learning of actions, positive reinforcement is particularly important. To conclude, reflections of the prediction error in the FRN were indeed modulated by feedback timing and association type, but not as hypothesized by an interaction of the two factors.

Regarding the N170, we also observed reflections of the prediction error in its amplitude, but with a reversed pattern compared to the FRN. Here, higher prediction error values, i.e., more unexpected feedback, led to more negative N170 (more pronounced) amplitudes for positive feedback, while for negative feedback, higher prediction error values led to more positive (reduced) amplitudes. This stresses the relevance of the N170 as a feedback-locked signal to a new degree, as this ERP component has not been linked to prediction errors in the context of feedback learning so far. Enhanced N170 amplitudes for unexpected rewards may indicate that the MTL is particularly active when it comes to reinforcing memories for events that led to positive feedback. Likewise, enhanced amplitudes for expected negative feedback could reflect an additional effort to reactivate events for which expectations about negative feedback have been confirmed. As hypothesized, the MTL could initiate the reactivation of a stimulus representation in visual areas to help remember which stimulus led to reward and bind the two events together. Accordingly, we found that reflections of the prediction error in the N170 were more pronounced for active stimulus-feedback associations than for action-feedback associations (like for the FRN). Previous studies further corroborate our hypothesis by finding post-reward reactivation in the hippocampus (Singer & Frank, 2009), higher order visual areas (Schiffer et al., 2014) and somatosensory regions (Pleger et al., 2008, 2009). Importantly, prediction error

coding in the N170 was additionally modulated by feedback timing: For action-feedback associations, prediction error coding emerged only following immediate feedback, while it was present in all feedback timing conditions for stimulus-feedback associations. Finding similarly pronounced prediction error reflections in the N170 for both association types following immediate feedback seems contrary to our hypothesis that we should find them especially following delayed feedback that refers to stimuli. However, consistent with our expectations, for delayed feedback we found a stronger prediction error effect on the N170 for stimulus-feedback compared to action-feedback associations. Based on these results, we suppose that the N170 reflects a combination of MTL activity (as suggested by Arbel et al., 2017) and visual reactivation (as we hypothesized): For both association types, prediction error coding following immediate feedback in the MTL might appear in parallel to prediction error coding in the striatum and ACC. However, the need for reactivations in visual areas (potentially initiated by the MTL) as a key to solving the temporal credit assignment problem should be stronger for delayed feedback and would only happen for stimulus-feedback associations, not action-feedback associations.

Combining results for the FRN and N170, the study provides evidence that the involvement of diverse neural systems in feedback learning depends not only on feedback timing, but also on the type of association learned. Superior learning of active stimulus-feedback associations together with prediction error reflections for FRN and N170 regardless of feedback timing led us to assume a better cooperation of the striatal and the MTL/hippocampal system for stimulus-feedback associations. On the contrary, for action-feedback associations the cooperation may have worked only for immediate feedback. White and McDonald (2002) suggested that both the striatal and hippocampal system receive the same information during learning—and indeed, there is evidence for

dopaminergic projections to the striatum (Chuhma et al., 2023; Oldehinkel et al., 2022) and shared information between midbrain dopamine neurons and the MTL (Lisman & Grace, 2005; Schott et al., 2004, 2008). Additionally, both striatum and MTL reflect prediction error signals during feedback-based learning (Dickerson et al., 2011). However, they might specialize in encoding different types of associations (White & McDonald, 2002) and interact by reinforcing memory for rewarding episodes to guide future behaviour (Shohamy & Adcock, 2010).

In contrast to Kimura and Kimura (2016), who used a task where participants could not learn, we overall found similar feedback timing effects for delayed feedback with and without tone, indicating that feedback delay effects are not a function of reduced temporal predictability, at least when feedback can be used for learning.

What limits our study is that participants struggled to learn passive stimulus-feedback associations, which were intended to assess stimulus-feedback learning without actions. However, despite inconsistent learning, participants in this condition seemed to have formed (false) expectations while trying to choose the more rewarding stimulus in test trials without feedback, allowing meaningful prediction error analyses, and including the prediction error in the LME model partly accounts for differences in learning performance between conditions. Therefore, findings from the analysis comparing the action-feedback condition with the passive stimulus-feedback condition reported in the appendix also contributed to the understanding of the role of the N170 in feedback learning: For passive stimulus-feedback associations, we indeed found larger N170 amplitudes for delayed than immediate feedback, as hypothesized. Nevertheless, we primarily based our interpretations on the comparison of action-feedback associations with the actively learned stimulus-feedback associations, which also contained actions (i.e., button presses). However, feedback was only related to the

chosen stimuli and not to the actions. Likewise, the action-feedback condition also contained stimuli (i.e., rectangles on the screen), but the feedback referred only to the actions and not to the stimuli. Therefore, the two conditions were similar but differed in one crucial point: While in the active stimulus-feedback condition stimulus identity predicted reward, motor aspects of the task predicted reward in the action-feedback condition. As described above, superior learning of active stimulus-feedback associations might be caused by the fact that this condition contained both actions and stimuli, allowing the striatal and MTL systems to cooperate in the most effective way.

2.1.4 Conclusion

From this study investigating effects of the type of learned association on the FRN and N170, two ERP components thought to represent feedback processing in the striatal and MTL system, we derive two key findings: Remarkably, a reflection of the whole range of the prediction error in the N170 provides new evidence that, alongside the FRN, this is an important component reflecting feedback processing, especially for delayed feedback. Furthermore, we drop our hypothesis that either the striatal or MTL/hippocampal learning system takes over sole control under certain learning conditions. Instead, the association type, together with feedback timing, seems to modulate how well the systems can cooperate during learning.

2.2 STUDY 2

2.2.1 Research Question and Hypotheses

The study was based on the idea that delayed feedback could lead to the reactivation of a selected stimulus in higher-order sensory areas, serving as a mechanism to bridge the temporal gap between feedback and the stimulus that caused it. Particularly, we assumed that the reactivation of higher-order visual areas could play a role in generating the feedback-locked N170 to assign delayed feedback to a previously

selected visual stimulus. Therefore, the study set out to investigate effects of the chosen stimulus' modality and feedback timing on feedback processing. We manipulated the modality of the stimuli to choose from (visual and auditory) and the feedback timing (immediate and delayed) while recording EEG. Based on studies on visual processing where origins in higher-order visual areas were found (Deffke et al., 2007; Gao et al., 2019; Iidaka et al., 2006), we assumed that the N170 specifically represents visual activity and therefore hypothesized that its amplitude would be larger when feedback is associated with visual compared to auditory stimuli. In addition, we expected this effect to be stronger for delayed than immediate feedback, as the reactivation of a stimulus in visual areas should especially be necessary in situations where a temporal gap needs to be bridged between the stimulus and feedback. In addition, we explored prediction error representations in the N170. Regarding the FRN, we aimed to replicate previous effects found for the prediction error, the valence, and the timing of feedback. As the FRN is understood as a modality unspecific reflection of striatal activity, we additionally aimed to explore whether the modality of the stimulus associated with the feedback has an effect.

2.2.2 Methods

40 healthy adults participated in the experiment, but after checking for exclusion criteria (for example neurological diseases), data quality and technical problems, the final sample included in the analyses consisted of 35 participants, 30 women and 5 men, between 19 and 35 years with a mean age of 23.2 years (*SD* = 4.5 years). Participants underwent a computer-based probabilistic feedback learning task, in which they could learn associations between stimuli and positive or negative monetary feedback (+4 ct vs. -2 ct). Each participant could choose between two visual stimuli (two hiragana characters on the left and right side of the computer screen) in half of the learning

blocks or two auditory stimuli (two melodies played by different instruments simultaneously on the left and right ear via headphones) in the other half. Furthermore, feedback appeared 1 s (immediate feedback) or 7 s (delayed feedback) after the participants' choice and was always presented visually on the screen. The experiment was divided into two halves: one with visual stimuli and one with auditory stimuli. Participants completed four learning phases in one modality (either visual or auditory) before switching to the other modality, with the order of modalities counterbalanced across participants. In each learning phase, a new stimulus pair was presented, and the feedback timing (immediate or delayed) remained consistent throughout the phase. Feedback timing changed when a new learning phase began, coinciding with the presentation of a new stimulus pair. Each learning phase was further divided into 4 blocks of 20 trials, yielding 640 trials in total. One stimulus of each visual or auditory pair was associated with reward in 65% of the trials and punishment in 35%, while probabilities were reversed for the other stimulus. The participant's task was to learn which stimulus was more likely to be rewarded and maximize reward through their choices. During the task, we recorded EEG from 60 scalp electrodes, evenly distributed on the head according to the extended 10-20 system.

We analyzed behavioral single-trial data by applying a GLME model in R (Bates et al., 2015; The R Foundation, 2021). We investigated effects of stimulus modality (visual vs. auditory), feedback timing (immediate vs. delayed) and learning block (1-4) on the response accuracy (i.e., how often a participant chose the more rewarding stimulus). In addition, we used the behavioral data to computationally model the prediction error for every single trial in MATLAB (R2021a, The MathWorks, Inc., 2021; for similar approaches see Burnside et al., 2019; Lefebvre et al., 2017; Weber & Bellebaum, 2024). For the analysis of the EEG data, we first preprocessed the EEG data

using BrainVision Analyzer 2.2 (Brain Products GmbH, 2018) to clean it from artifacts and prepare it for the analyses. Then, using MATLAB (R2021a, The MathWorks, Inc., 2021), we extracted single trial amplitude values for the N170 from electrodes P7 and P8 between 130 and 230 ms post feedback and operationalized the N170 as a peak-to-peak amplitude by subtracting the preceding positive peak (P1). For the FRN, we extracted single trial amplitude values from electrodes Fz, FCz, Cz, FC1 and FC2 at the latency of the maximum negative peak in the FRN_{diff} in a time-window ranging from 230 to 360 ms post feedback. Both ERP components were separately analyzed by applying an LME analysis in R (Bates et al., 2015; The R Foundation, 2021). Precisely, we investigated effects of stimulus modality (visual vs. auditory), feedback timing (immediate vs. delayed), feedback valence (negative vs. positive) and the unsigned prediction error (indicating general expectation violations or surprise) on N170 and FRN. For the N170, we additionally investigated hemispheric differences between the electrode sites (P7 vs. P8).

2.2.3 Results and Discussion

The behavioral data analysis revealed an increasing number of correct responses across the four learning blocks, indicating that participants did learn to choose the more rewarding stimulus more frequently throughout the experimental task. In addition, we did not find differences in response accuracy between immediate and delayed feedback or visual and auditory stimuli, suggesting that learning was comparable between these conditions.

For the N170, we found that over the right hemisphere (P8), stimulus modality affected the amplitude following delayed feedback, with larger N170 amplitudes for feedback referring to visual compared to auditory stimuli. This is in line with our hypothesis that the N170 reflects stimulus reactivations in higher-order visual areas,

which may represent a mechanism to link visual stimuli to delayed feedback. Evidence for this mechanism also comes from fMRI studies that revealed post-reward reactivations in visual (Schiffer et al., 2014) and somatosensory brain regions (Pleger et al., 2008, 2009) as a way to assign credit for a reward to a stimulus of a certain sensory modality. The functional specialization of the right hemisphere for visuo-spatial processing (Thiebaut de Schotten et al., 2011) combined with the visuo-spatial stimuli used in our study (hiragana characters) could explain differences between the hemispheres. Surprisingly, we found larger feedback-locked N170 amplitudes over the right hemisphere for choices between auditory compared to visual stimuli following immediate feedback. We explain this by suggesting that the N170 reflects both activity in the MTL and extrastriate visual areas during feedback processing. Although the hippocampus seems to be particularly important to process delayed feedback (Foerde et al., 2013; Foerde & Shohamy, 2011), it is also involved in processing feedback with relatively short delays (Dickerson et al., 2011). While for delayed feedback, the visual cortex contribution to the N170 may have been particularly high, hippocampal contributions following immediate feedback for auditory stimuli may have been high as well: The auditory condition required cross-modal associations, which activates the hippocampus more than unimodal associations (Butler & James, 2011).

Another surprising finding were effects of stimulus modality on the FRN: We found larger FRN amplitudes following immediate feedback for the choice between visual compared to auditory stimuli. However, effects of feedback valence and the reflection of the prediction error in the FRN were not affected by stimulus modality, therefore it did not seem to influence processes underlying the FRN generation.

Apart from effects of stimulus modality on N170 and FRN, the analyses made the N170 appear as a kind of counterpart to the FRN: The N170 was sensitive to

feedback valence only following delayed and not immediate feedback, while we could replicate that the FRN is more sensitive to feedback valence when feedback is presented immediately (Arbel et al., 2017; Höltje & Mecklinger, 2020; Peterburs et al., 2016; Weinberg et al., 2012; Weismüller & Bellebaum, 2016). This strengthens the notion that sources underlying the FRN like the striatum are rather involved in processing immediate feedback, while potential generators of the N170 like the MTL are more important for delayed feedback processing (Foerde et al., 2013; Foerde & Shohamy, 2011).

Another finding that positioned the N170 as a counterpart to the FRN was the interaction between prediction error and feedback valence that indicated a reflection of the whole range of the prediction error in both ERP components: While the FRN, in line with the reinforcement learning theory (Holroyd & Coles, 2002), became more negative when negative feedback was unexpected and more positive when positive feedback was unexpected, the N170 (especially over the right hemisphere) was enhanced (more negative) for unexpected positive feedback and reduced (more positive) for unexpected negative feedback. While previous studies reported more pronounced N170 amplitudes for unpredictable compared to predictable stimuli during the perceptual processing of visual stimuli, linking the N170 to surprise in general (Baker et al., 2021, 2023), this is the first study to report reflections of reward prediction errors on the N170. Enhanced amplitudes following unexpected positive feedback might indicate that it is especially important to remember which stimulus led to a reward and strengthen that relationship through reactivation mechanisms (Singer & Frank, 2009). Since the midbrain dopamine system projects to various regions within the brain, it could send reinforcement learning signals not only to the striatum and frontal cortex (Schultz, 2002), but also to the hippocampus (Schott et al., 2004). Accordingly, prediction error related activity has

been found within the hippocampus (Dickerson et al., 2011; Foerde & Shohamy, 2011), which might be reflected in the N170. Nevertheless, the prediction error coding in the N170 over the right hemisphere was particularly pronounced for the prior choice between visual stimuli and especially for delayed feedback. Finally, this aligns with our hypothesis that the N170 could specifically represent a reactivation of visual areas during feedback processing.

2.2.4 Conclusion

The combination of finding a more pronounced N170 following delayed feedback related to the choice of visual compared to auditory stimuli over the right hemisphere, and a particularly strong representation of the prediction error under the same conditions, adds weight to our hypothesis that this ERP component could reflect activity within higher-order visual areas of the brain. However, signals from the MTL may initiate the reactivation of an internal representation of a stimulus, allowing it to be linked to temporally delayed feedback (Qin et al., 2007). Therefore, we propose that the N170 reflects overlapping activity of MTL and reactivations in higher order visual areas. A reactivation reflected in the N170 might be the bridge that is needed to connect stimuli with delayed feedback in complex real-world situations and a key mechanism for solving the credit assignment problem.

2.3 STUDY 3

2.3.1 Research Question and Hypotheses

While several studies have reported altered FRN amplitudes as predictors, concomitants, or consequences of depression (Bress et al., 2012, 2013, 2015; Foti et al., 2014; Klawohn et al., 2021; for a meta-analytic review see Keren et al., 2018), other studies have failed to replicate these findings (Hager et al., 2022; Clayson et al., 2020; Moran et al., 2017). With Study 3, we aimed to examine the connection between

depression and blunted effects of feedback valence on the FRN amplitude. While the FRN seemingly reflects influences of the dopaminergic midbrain on striatum and ACC (Becker et al., 2014; Carlson et al., 2011; Foti et al., 2011; Hauser et al., 2014; Holroyd & Coles, 2002; Nieuwenhuis et al., 2004; Oerlemans et al., 2025), other ERP components could reflect slightly different processes that are also relevant during feedback processing: For example, we suggest that the N170 reflects a reactivation of visual brain regions potentially initiated by structures within the MTL—a mechanism to link delayed feedback to the prior choice of a visual stimulus. In line with this, previous studies found that the N170 is not only associated with visual activity (Deffke et al., 2007; Gao et al., 2019; Iidaka et al., 2006) but also with activity in the right MTL including the hippocampus (Baker & Holroyd, 2009, 2013; Baker et al., 2015). As the latter can be reduced in size and impaired or altered in function in depression (Fairhall et al., 2010; Shah et al., 1998; for a review see Thompson, 2023), we aimed to explore depression-related alterations in (delayed) feedback processing in the N170. In contrast to earlier studies, we assessed and conceptualized currently experienced depressive symptoms on a continuum rather than as a binary state (healthy vs. depressed) to avoid a loss of information and increase statistical power (see Clayson et al., 2020; for a similar approach see Hager et al., 2022). Our sample was recruited in both a non-clinical and clinical setting in order to reach an adequate range of different symptom severities. Since blunted reward processing has not only been found in currently depressed individuals, but also in remitted individuals (McCabe et al., 2009) and those at high risk for depression due to a familial vulnerability (Weinberg et al., 2015; for a review see Luking et al., 2016), we additionally assessed past depressive episodes and the familial history of depression to investigate their influence on feedback learning and processing alongside currently experienced depressive symptoms. Participants

performed a probabilistic feedback learning task with immediate and delayed feedback while we recorded EEG. Based on previous studies, we anticipated that depression (current symptom severity, past episodes and familial vulnerability) would be associated with a diminished sensitivity of the FRN to feedback valence, particularly due to a reduced neural response to rewards. Furthermore, we expected the association between depression and the FRN to be most pronounced following immediate feedback. Regarding the N170, we hypothesized to find blunted (less negative) N170 amplitudes associated with depression, especially following delayed feedback, because hippocampal atrophy in depression might cause alterations in delayed feedback processing.

2.3.2 Methods

Instead of conducting a group comparison between individuals with depression and healthy controls, we aimed to use the severity of depressive symptoms as a continuous variable within a sample recruited in a non-clinical and clinical setting. To achieve this, we advertised the study on the campus of Heinrich Heine University Düsseldorf and at the outpatient psychotherapy unit of the LVR Clinic for Psychosomatic Medicine and Psychotherapy in Düsseldorf, inviting participation from both individuals experiencing depressive symptoms and those without. 50 adults participated in the study, due to exclusion criteria (see below), data quality and technical issues, only 45 of them were included in the statistical analysis (37 women and eight men, 41 right-handed, three left-handed and one ambidextrous). The mean age was 24.87 years (*SD* = 5.54 years, *Min* = 18 years, *Max* = 39 years). All participants reported no current or former neurological disorders, no acute psychotic conditions, and no regular or acute consumption of substances affecting the central nervous system. Depression is often accompanied by comorbid disorders like anxiety disorders,

substance addiction disorders, eating disorders, obsessive-compulsive disorders, other affective disorders, and suicidality (Jacobi et al., 2004; Lamers et al., 2011; Zimmerman et al., 2002). We decided against defining these psychiatric disorders as exclusion criteria and instead assessed them by conducting the short version of the Diagnostic Interview for Psychological Disorders ([Mini-DIPS]; Margraf & Cwik, 2017; Margraf et al., 2017; for a similar approach see Bress et al., 2013 and Foti et al., 2014). Since not only depressed individuals but also remitted individuals (McCabe et al., 2009) and the first-degree relatives of persons with major depression (Weinberg et al., 2015; for a review see Luking et al., 2016) show blunted (more negative) FRN amplitudes or striatal responses following rewards, we assessed all three aspects to study their influence on feedback learning and processing separately: First, we asked participants whether a first-degree relative, i.e., parent or sibling (excluding half-siblings), has ever been diagnosed with depression. Afterward, we used a modified version of the mood module of the Patient Health Questionnaire ([PHQ-9]; Kroenke et al., 2001; German version: Gräfe et al., 2004) to evaluate past depressive episodes, based on the approach by Bress et al. (2013). Finally, we used the Beck Depression Inventory ([BDI-II]; Beck et al., 1996; German version: Hautzinger et al., 2006) as a measure for acute depression severity (for a similar account, see Bress et al., 2013).

Participants underwent a computer-based probabilistic feedback learning task, where in every trial they could choose between two visually presented stimuli and learn associations between these stimuli and positive (+4 ct) or negative (-2 ct) monetary feedback. Feedback appeared either 1 s (immediate feedback) or 7 s (delayed feedback) after the participants' choice. Unbeknown to the participants, one stimulus of each presented pair was associated with reward in 65 % of the trials and with punishment in 35 %, while the probabilities were reversed for the other stimulus. Participants were

instructed that wins and losses contribute to the compensation paid out at the end. Their task was to maximize their earnings by learning which stimulus is more likely to be rewarded. Participants completed four learning phases (two with immediate and two with delayed feedback). In each learning phase, a new stimulus pair was presented, and the feedback timing remained consistent throughout the phase. The feedback timing changed only at the beginning of a new learning phase, coinciding with the presentation of a new stimulus pair. Each learning phase was further divided into 4 blocks of 20 trials, yielding 320 trials in total. During the task, EEG was continuously acquired from 60 scalp electrodes, evenly distributed across the head based on the extended 10-20 system.

We analyzed the behavioral data by performing GLME analyses in R (version 1.1.34; Bates et al., 2015; The R Foundation, 2021). We aimed to investigate effects of depression (BDI-II, modified PHQ-9 and familial vulnerability), feedback timing (immediate vs. delayed) and block (1-4) on response accuracy (how often the more rewarding stimulus was chosen) in the probabilistic feedback learning task. Precisely, we calculated three separate models, one for each measure of depression as a predictor. The first model contained the BDI (a measure for current severity of depressive symptoms) as a predictor alongside the other factors mentioned above (feedback timing and block), while the second model contained the PHQ (a measure of past depressive episodes) and the third model familial vulnerability for depression (whether a first degree relative has ever been diagnosed with depression) as a predictor. Additionally, we utilized the behavioral data to computationally model the prediction error for every single trial in MATLAB (R2021a, The MathWorks, Inc., 2021; for similar approaches see Burnside et al., 2019; Lefebvre et al., 2017; Weber & Bellebaum, 2024). For EEG data analyses, we performed a standard preprocessing procedure using BrainVision

Analyzer 2.2 (Brain Products GmbH, 2018) to clean the signal from artifacts and prepare it for the analyses. Then in MATLAB (R2021a, The MathWorks, Inc., 2021), we extracted single trial amplitude values for the N170 from electrodes P7 and P8 between 130 and 230 ms post feedback and operationalized the N170 as a peak-to-peak amplitude by subtracting the preceding positive peak (P1). For the FRN, we extracted single trial amplitude data from electrodes Fz, FCz, Cz, FC1 and FC2 at the latency of the maximum negative peak in the FRN_{diff} in a time-window ranging from 230 to 360 ms post feedback. Finally, we analyzed both ERP components separately with LME analyses in R (Bates et al., 2015; The R Foundation, 2021). For the N170, we were interested in effects of depression (BDI, PHQ and familial vulnerability), feedback timing (immediate vs. delayed), feedback valence (negative vs. positive), unsigned prediction error (general expectation violations or surprise) and electrode (left hemisphere/P7 vs. right hemisphere/P8). Like for the behavioral data analysis, we built three separate models, one for each of the three different depression variables as a predictor. The first model contained the BDI as a measure of severity of current depressive symptoms, alongside feedback timing, feedback valence, unsigned prediction error and electrode as predictors. Similarly, we built the other two models, with the only difference being the depression variable used as predictor. Thus, the second model contained the PHQ as a measure of past depressive episodes and the third model included familial vulnerability for depression (whether a first degree relative has ever been diagnosed with depression) as a predictor. For the analyses of the FRN, we followed the same approach as in the N170 analysis by building three models varying only regarding the depression variable used as predictor (BDI, PHQ or familial vulnerability) alongside feedback timing (immediate vs. delayed), feedback valence (negative vs. positive), and the unsigned prediction error (indicating general expectation

violations or surprise).

2.3.3 Results and Discussion

The behavioral data analysis revealed that participants currently experiencing more severe depressive symptoms showed worse learning performance in the probabilistic feedback learning task. Thus, as expected, learning from immediate and delayed feedback was affected by depression, possibly due to altered striatal and hippocampal functioning (Admon & Pizzagalli, 2015; Fairhall et al., 2010; Luking et al., 2016; Nestler et al., 2002; Pizzagalli et al., 2009; Takamura et al., 2017; Thompson, 2023)—structures that are both relevant for feedback processing (Foerde et al., 2013; Foerde & Shohamy, 2011).

For the FRN, we could not replicate the expected reduced feedback valence sensitivity in participants currently experiencing depressive symptoms (Bress et al., 2012, 2015; Foti et al., 2014; Klawohn et al., 2021), corroborating studies that reported no or only weak, task-dependent relationships between the FRN and depression (Hager et al., 2022; Clayson et al., 2020; Moran et al., 2017). However, it needs to be considered that participants in our study had an average BDI score of 9.89 (*SD* = 9.34), which is below the cut-off score for depression (von Glischinski et al., 2019). Moreover, our sample lacked participants with more severe depressive symptoms: The highest observed BDI score was 39, considerably below the theoretical maximum of 63. Prior studies that found relationships between the FRN amplitude and self-rated depressive symptoms in a non-clinical sample investigated 8- to 13-year-old children/adolescents (Bress et al., 2012, 2015), a group in which depression-related changes in the FRN seem to be particularly pronounced (Keren et al., 2018).

Although we could also not find the hypothesized pattern of reduced N170 amplitudes in participants currently experiencing more severe depressive symptoms—

especially following delayed feedback—these symptoms still influenced prediction error processing as reflected in the N170: More unexpected positive feedback was associated with more pronounced N170 amplitudes, but only in participants showing low levels of depressive symptoms and especially following (delayed) positive feedback. Although we did not consider this pattern in our hypothesis, it aligns with our assumption that structures and processes underlying the N170 are affected by depression. In healthy individuals, interactions between midbrain dopamine regions and the MTL may reinforce memory representations of rewarded stimuli to improve behavior (Shohamy & Adcock, 2010). However, this process could be impaired in individuals experiencing depressive symptoms, for example due to hippocampal changes and altered memory functions (Dillon & Pizzagalli, 2018; Fairhall et al., 2010; Nestler et al., 2002; Thompson, 2023). As altered feedback learning in depression is typically marked by anhedonia and reduced reward responsiveness (Admon et al., 2017; Bakic et al., 2017; Huys et al., 2013; Kumar et al., 2018; Kunisato et al., 2012; Rizvi et al., 2016), this might explain reduced prediction error coding in the N170 particularly following positive feedback in the present study.

In contrast to effects of currently experienced depressive symptoms, we did not find any behavioral or neurophysiological effects of past depressive episodes as measured via a modified version of the PHQ-9. Although this questionnaire was intended to be a measure of lifetime depression (Cannon et al., 2007), we argue that a high score is not necessarily reflective of past depressive episodes. For example, symptoms assessed by the modified PHQ-9 like low mood, problems sleeping, changes in appetite and difficulties concentrating also commonly appear in non-pathological forms of grief (Shear et al., 2011; Zisook & Shear, 2009), making it hard to reliably and validly assess clinically relevant past depressive episodes.

Regarding the role of familial vulnerability, we did not find reduced behavioral performance in the probabilistic feedback learning task with an increased familial risk for depression. Neurophysiological responses to gains and losses in the FRN were also not affected by a familial history of depression, but it affected the N170: The N170 (over the right hemisphere) was more pronounced following negative than positive feedback (for similar results see Kim & Arbel, 2019), but this valence sensitivity was not present in participants with a familial history of depression. Blunted hippocampal activity could have caused the reduced differentiation between responses to gains and losses in the N170 in our study.

An aspect that complicates the interpretation of our results is the prevalence of other mental disorders apart from depression, which were identified during the Mini-DIPS. For example, a notable proportion of 26% of our participants met the criteria for a social anxiety disorder (currently or in the past) and anxiety has been found to also affect the FRN (Aarts & Pourtois, 2012; Gu et al., 2010; Jiang et al., 2018; Takács et al., 2015; Tobias & Ito, 2021). Thus, inspired by Grabowska et al. (2024), we think that because ERP components like the FRN and N170 are modulated by various interindividual differences, investigating only a small set of them might be too simple: Mental disorders like anxiety and depression are intertwined, influencing an individual's way of processing feedback in complex ways.

2.3.4 Conclusion

In this study, we found that performance in a feedback learning task was reduced in participants experiencing more severe depressive symptoms, irrespective of feedback timing, but we found no depression-related effects on the FRN. However, currently experienced depressive symptoms were associated with poorer encoding of prediction errors in the N170 and a familial vulnerability for depression was related to a reduced

differentiation between responses to rewards and punishments in the N170. We conclude that the N170 has the potential to be considered a biomarker alongside the FRN in clinical research on depression and feedback-based learning processes.

3 GENERAL DISCUSSION

3.1 REVISITING RESEARCH QUESTIONS AND KEY FINDINGS

3.1.1 A Potential Solution for the Credit Assignment Problem

This dissertation set out to enhance our understanding of a core feature of human behavior—namely, the pursuit of rewards and the avoidance of punishment—a process fundamentally guided by our ability to employ feedback as a teacher that enables all kinds of intelligent behavior (Cohen & Blum, 2002; Silver et al., 2021). Starting point was the well-established insight that the dopaminergic reward system, originating in the midbrain and projecting to subcortical regions, for example within the striatum, and to fronto-cortical structures like the ACC, forms the central neuronal basis for feedback processing (Björklund & Dunnett, 2007; Glimcher, 2011; Haber & Knutson, 2010; Oldehinkel et al., 2022; Schultz & Dickinson, 2000). However, it was pointed out that characteristics of the learning context like the temporal delay of feedback can influence the neural sources involved in feedback processing in terms of a shift from striatal to hippocampal/MTL regions (Foerde et al., 2013; Foerde & Shohamy, 2011). The following questions arose: How can we associate feedback with events that occurred several moments ago, and how do we know which particular event in a chain of performed actions or seen stimuli deserves credit? From a cognitive neuroscientific perspective, we faced the problem of credit assignment (Curtis & Lee, 2010; Stolyarova, 2018; Sutton & Barto, 2018) and how it might be solved by the human brain.

To deepen our understanding towards solving this problem and finding out which factors might mediate the involvement of striatal and hippocampal learning systems during immediate and delayed feedback processing, Study 1 examined both the effects of feedback timing and the so far insufficiently explored impact of the

association type on the electrophysiological processing of feedback. We expected that the striatal system is not only important for immediate feedback processing, but also particularly involved in linking feedback to actions, while the MTL system might be especially helpful for linking delayed feedback to stimuli. We investigated modulations of the FRN and N170, two ERP components thought to reflect feedback processing in the striatal (Becker et al., 2014; Bellebaum & Daum, 2008; Foti et al., 2011; Gehring & Willoughby, 2002; Holroyd et al., 2004; Holroyd & Coles, 2002; Oerlemans et al., 2025; for a review see Nieuwenhuis et al., 2004) and MTL system, respectively (Arbel et al., 2017; Baker & Holroyd, 2009, 2013; Kim & Arbel, 2019). The results suggested that both feedback timing and association type modulated the involvement of striatal and MTL systems during feedback learning, but we could not find evidence for our hypothesis that each system operates independently and specifically under certain feedback timing and association type conditions. A study by Weismüller et al. (2018) also speaks against a strict dissociation of neural correlates underlying immediate and delayed feedback processing as dopamine depletion in unmedicated Parkinson's disease patients led to enhanced learning from both immediate and delayed negative feedback. The combination of different association types and feedback timings in Study 1 appeared to modulate how well striatal and MTL learning systems were able to cooperate during learning: Better learning combined with stronger prediction error reflections in the FRN and N170 (following both immediate and delayed feedback) for stimulus-feedback associations led us to assume a better cooperation for this association type. Here, both actions and stimuli were present during the task and thus striatal and MTL regions were assumedly both able to contribute to the formation of associations. Regarding the problem of credit assignment, results from Study 1 suggest that the striatal and hippocampal systems receive the same information (White & McDonald,

2002), namely dopaminergic prediction error signals (Dickerson et al., 2011), and use it to contribute to the formation of various types of associations together.

In Study 2 we further investigated different association types, this time focusing on the sensory modality of the stimuli that need to be associated with feedback. Again, the aim was to unpack the cognitive and neural mechanisms involved in credit assignment during feedback learning. One way to especially assign delayed feedback to earlier encountered stimuli could be the reactivation of their representations in higher-order sensory areas of the brain when feedback is presented (Pleger et al., 2008, 2009; Schiffer et al., 2014). Particularly, we assumed that the reactivation of higher-order visual areas could play a role in generating the N170 (Deffke et al., 2007; Gao et al., 2019; Iidaka et al., 2006) in situations where delayed feedback needs to be assigned to a previously chosen visual stimulus. Thus, we focused on determining the nature of the signal reflected by the feedback-evoked N170, which in this context has previously only been hypothesized to reflect MTL activity (Arbel et a., 2017; Kim & Arbel 2019). Therefore, Study 2 investigated effects of the chosen stimulus' modality and feedback timing on feedback processing. As hypothesized, we found a more pronounced N170 following delayed feedback related to the choice of visual compared to auditory stimuli over the right hemisphere. In addition, the prediction error was most reflected in the N170 under the same conditions. We concluded that the N170 might indeed reflect reactivations within higher-order visual areas of the brain, possibly initiated by the MTL (Qin et al., 2007), revealing a mechanism to assign credit for delayed feedback to a previously chosen visual stimulus.

3.1.2 A Potential New Biomarker of Depression

While it is insightful to utilize ERP components such as the FRN and N170 for investigating basic processes of learning and feedback processing under healthy

conditions, prior research has also demonstrated that the FRN can serve as a valuable biomarker or, more precisely, an endophenotype for a highly prevalent and debilitating mental disorder, namely depression (Bress et al., 2012, 2013, 2015; Brush et al., 2018; Foti et al., 2014, for a meta-analytic review see Keren et al., 2018). Building on the findings from Studies 1 and 2, which showed that alongside the FRN the N170 also reflects key processes involved in feedback processing, Study 3 aimed to examine whether the N170 might also be altered in depression, especially in situations where feedback is temporally delayed. We found that learning from immediate and delayed feedback was impaired in participants experiencing more severe depressive symptoms. While we could not replicate earlier findings of depression-related reduced responses to gains and losses in the FRN (Bress et al., 2012, 2013, 2015; Brush et al., 2018; Foti et al., 2014), a familial vulnerability for depression was linked to a reduced differentiation between responses to gains and losses in the N170. Additionally, currently experienced depressive symptoms came along with poorer encoding of prediction errors in the N170. Therefore, we suggested to further explore the N170 as a potential biomarker alongside the FRN in future clinical research on depression and feedback-based learning processes.

As this was the first study to examine the N170 in the context of feedback learning in depression, it remains to be tested whether the effects found can be replicated in future studies. Since changes in the FRN were not found consistently in the context of depression, replications may also prove difficult for the N170. However, all three studies reported in this dissertation clearly showed that both ERP components, FRN and N170, reflect feedback processing. In addition, results from Study 1 suggest that the brain structures underlying these components may interact with each other depending on the learning context. Thus, changes in feedback processing in depression

might manifest in one of the two components depending on the learning context. It could therefore be considered whether the N170 poses a potential new biomarker or rather a different manifestation of the same underlying pathological reward processing sometimes reflected in the FRN. For future research it could be useful to investigate alterations in both components to uncover connections between complex phenomena such as feedback processing in various contexts and depression.

3.2 INTEGRATION OF FINDINGS: THREE STUDIES ONE PICTURE

This dissertation benefits from the highly similar design of its three underlying empirical studies. All studies employed a comparable paradigm—specifically, a probabilistic feedback learning task. In each study, effects of feedback valence, feedback timing, and the prediction error on feedback-based learning and the neural processing of feedback, reflected in the FRN and N170, were investigated using identical EEG setups. Each study then placed particular focus on an additional factor: Studies 1 and 2 focused on different association types, and Study 3 on depression. Moreover, all three studies used the same statistical approach, namely (G)LME models. These models are particularly well-suited for single-trial data analyses in research involving human participants, as their data often have a hierarchical structure (Meteyard & Davies, 2020): For example, responses in a feedback learning task are likely correlated within each participant and ERP signals for certain levels of a factor such as immediate and delayed feedback are also likely correlated across participants. These ways in which data can be grouped results in a hierarchical error structure, which can be explicitly modeled in (G)LME models by including random intercepts and slopes (Meteyard & Davies, 2020). Including random intercepts for participants accounts for differences in general learning performance or ERP amplitudes across individuals. Including random slopes allows the effect of an experimental factor to vary between

participants, for example feedback valence may influence one participant's electrophysiological feedback processing more strongly than another's. The methodological consistencies across the three studies led to an accumulation of converging evidence for specific effects:

In all conducted studies, we were able to replicate a well-established finding that the FRN is more negative following losses than gains, and that this effect is stronger when feedback is presented immediately after a choice than delayed (for similar results see Arbel et al., 2017; Höltje & Mecklinger, 2020; Peterburs et al., 2016; Weinberg et al., 2012; Weismüller & Bellebaum, 2016). In addition, across all three studies, we found that the FRN reflected the whole range of the prediction error by becoming more negative for unexpected negative feedback and more positive for unexpected positive feedback. In contrast, previous studies found that the prediction error was reflected in FRN amplitudes following positive, but not negative feedback (Weber & Bellebaum, 2024; Kirsch et al., 2022), supporting the idea that the signal in this time window is specifically shaped by positive feedback and thus reflects a RewP. However, our findings are in line with the reinforcement learning theory of the FRN (Holroyd & Coles, 2002), suggesting that feedback better than expected activates dopaminergic neurons, which leads to the inhibition of the ACC and more positive FRN amplitudes, while feedback worse than expected reduces activity of dopaminergic neurons, disinhibiting the ACC and making the FRN more negative (Holroyd & Coles, 2002). While much attention has traditionally been paid to the response of dopaminergic neurons to reward and its omission, it is important to note that some dopamine neurons (in monkeys) specifically show phasic activations in response to (conditioned) aversive stimuli (Mirenowicz & Schultz, 1996). In addition, the human habenula has been found to react to punishment prediction errors, not only when expected reward is omitted, but

also when unexpected punishment is perceived (Lawson et al., 2014; Salas et al., 2010). Thus, the habenula is referred to as the brain's anti-reward center with connections to the dopaminergic reward system (for reviews see Hu et al., 2020 and Metzger et al., 2021). Our findings suggest that the frontocentral ERP component in the time window about 200 to 400 ms post feedback is shaped by both, neural responses to (un)expected rewards and punishments.

Regarding the N170, we were also able to demonstrate a sensitivity to the valence of feedback (for similar results, see Kim & Arbel, 2019). However, the distinction between positive and negative feedback in Studies 1 and 2 was more/only pronounced for delayed feedback compared to immediate feedback—an opposite pattern to that observed for the FRN. Moreover, Study 3 revealed that differences in the N170 between positive and negative feedback were not detectable in individuals with a family history—and thus an increased risk (Halligan et al., 2007; Raposa et al., 2014)—of depression.

Importantly, we were primarily able to show consistently across all three studies that the feedback-locked N170 also reflects the full range of prediction errors, with increasingly negative amplitudes the more unexpected positive feedback was, and increasingly positive amplitudes the more unexpected negative feedback was. This finding provided new evidence that, alongside the FRN, the N170 is an important component reflecting feedback processing. We suggested that pronounced (negative) amplitudes in response to unexpected positive feedback might indicate that it is especially important to remember—or reactivate—what led to an unexpected reward (Singer & Frank, 2009). However, from an evolutionary perspective, one could argue that it should also be important to remember what led to an unexpected loss, as subjectively, losses weigh about twice as much as gains (Tversky & Kahneman, 1992).

Nevertheless, previous findings of a so-called confirmation bias indicate that humans particularly rely on positive feedback by showing higher learning rates for positive than negative feedback (for example see Weber & Bellebaum, 2024), a bias we also observed in our experiments. Thus, positive reinforcement may be more effective than negative feedback and there seems to be some truth to the old saying that *"*you catch more flies with honey than with vinegar".

Overall, incorporating the prediction error as a predictor in our analyses proved to be highly informative. Specifically, we used computational modeling of reinforcement learning parameters to explore how dopaminergic prediction error coding shapes brain activity during feedback learning (Glimcher, 2011). This allowed us to account for the underlying processes involved in the generation of both the FRN and N170. Interestingly, effects of association type (including stimulus modality) and depression were mostly observed in interaction with the prediction error. When it comes to learning, using computational modeling approaches to understand human behavior and brain activity thus appears to be a promising approach for future research.

Another modulator of feedback processing investigated across all three studies—enabling an integrated and comprehensive overview—was feedback timing. Previous research has suggested a shift from striatal to MTL engagement with increasing feedback delays (Foerde et al., 2013; Foerde & Shohamy, 2011). In line with this, our findings of reduced feedback valence sensitivity in the FRN following delayed feedback support the notion that the striatum is particularly involved in processing immediate feedback (for similar results see Arbel et al., 2017; Höltje & Mecklinger, 2020; Peterburs et al., 2016; Weinberg et al., 2012; Weismüller & Bellebaum, 2016). While we could replicate previously reported effects of generally larger N170 amplitudes following delayed versus immediate feedback only in Study 1 (see Arbel et al., 2017;

Kim & Arbel, 2019; Höltje & Mecklinger, 2020), the enhanced sensitivity to feedback valence in delayed feedback conditions observed in Studies 1 and 2 aligns with the idea that the MTL plays a prominent role in processing delayed feedback.

However, looking at prediction error signals in both ERP components, a more nuanced picture emerges: For the FRN, more pronounced effects for immediate feedback were only observed in Study 1, whereas prediction error coding was independent of feedback timing in the other two studies. Similarly, the N170 did not show a consistent pattern across studies: Depending on association types, stimulus modalities or depressive symptom severity, prediction error signals were more prominently reflected following either immediate or delayed feedback. This aligns with previous findings that the hippocampus can be engaged in feedback processing even for short feedback delays (Dickerson et al., 2011). Given the functional connections and flow of information between striatal and MTL regions (Davidow et al., 2016; Dickerson et al., 2011; Kahn & Shohamy, 2013), it seems plausible that there is no clear-cut division or exclusive responsibility for either immediate or delayed feedback processing where one system inhibits the other (but see Poldrack et al., 2001). Instead, we propose that under healthy conditions, striatal and MTL-based learning systems interact cooperatively for achieving best results (Shohamy & Adcock, 2010; Dickerson et al., 2011; White & McDonald, 2002). Inspired by the framework of White and McDonald (2002), we assume that both systems use dopaminergic prediction error signals to contribute their respective strengths to the formation of different association types depending on the nature of the learning context. Accordingly, it seems appropriate to soften the dichotomy that associates the FRN and striatal processing with immediate feedback, and the N170 and MTL-related activity with delayed feedback.

Much like the fluid functional boundaries in the brain, real life offers no clear-

cut distinction between where immediate feedback ends and delayed feedback begins. Across all three studies, participants were able to flexibly use both immediate and delayed rewarding and punishing outcomes as learning signals to guide their behavior, which is ecologically valid: In everyday life, we adapt and shape our (future) behavior based on both immediate consequences (like instantly feeling pain after getting your finger caught folding up a drying rack) and delayed outcomes (like seeing joy slowly appear on someone's face a few seconds after you have given them a gift and they have unwrapped it).

3.3 CHALLENGES AND FUTURE RESEARCH AVENUES

One major advantage of EEG is its ability to measure changes in brain activity in response to specific events with high temporal resolution (Luck, 2014). This makes EEG particularly useful for investigating neural reactions and signals that occur within a few hundred milliseconds after an event—such as dopaminergic prediction error coding (Schultz et al., 1997; Zaghloul et al., 2009). In contrast, EEG has a poor spatial resolution, making it unsuitable to assign specific neural signals to distinct anatomical sources or measuring the operation of specific neural systems (Luck, 2014). Thus, assumptions we drew from Studies 1, 2 and 3 about the involvement of striatal, MTL or visual structures during feedback learning are only interpretations that cautiously need to be tested in future studies. The EEG signal reflects multiplexed, temporally and spatially overlapping neural signals, and it is difficult to trace these signals back to their sources precisely (Ullsperger, 2024). For example, prediction error signals were found in both striatal and hippocampal structures (Bellebaum et al., 2012; Davidow et al., 2016; Dickerson et al., 2011; Foerde & Shohamy, 2011; O'Doherty et al., 2004). Therefore, a methodologically sound approach is needed to draw conclusions about the neural mechanisms underlying specific ERP components. Precisely, combining EEG

with fMRI can provide insights about structures giving rise to the FRN and N170. For the FRN, studies combining these two methods already exist, linking it to activity within ventral striatum, midcingulate, and midfrontal cortices (for example see Becker et al., 2014). In addition, a recent intracranial ERP study provided compelling evidence that the signal in the time window of the FRN is generated by the ACC (Oerlemans et al., 2025). For the N170, however, a combination of EEG and fMRI approaches have so far only been applied in the context of face perception, identifying higher-order visual areas as likely generators (Gao et al., 2019; Iidaka et al., 2006). For the feedback-locked N170, its topography has led to the assumption that it reflects activity within the MTL (Arbel et al., 2017; Baker & Holroyd, 2009, 2013; Baker et al., 2015; Kim & Arbel, 2019). Future studies should employ fMRI to investigate whether the feedback-locked N170, as proposed here, may in fact reflect reactivations of visual areas, potentially initiated by the MTL. This would be particularly relevant, as the N170 represents a promising ERP component that reflects feedback learning processes and may even hold potential for identifying pathological alterations in feedback processing in disorders such as depression.

3.4 FROM LAB TO LIFE: LEARNING FROM FEEDBACK IN THE WILD

In laboratory settings, researchers try to reduce complex phenomena to their essential components and investigate them under highly standardized conditions, minimizing the influence of potential confounding variables. While this reductionist approach is valuable in terms of isolating specific mechanisms, it can sometimes lead us to lose sight of the broader context—of what our research is ultimately good for, and what value it may hold beyond the scientific bubble of our specific field. So, which role do different feedback timings play in real-world scenarios? And why is it important to investigate different association types in the context of feedback-based learning? Why

does it matter whether the N170 reflects MTL activity or visual reactivation? And what is the added value in knowing that certain EEG signals are altered in depression? Let us take a step back and reflect on each of these questions in light of the broader relevance of feedback-based learning, beginning with some applications of immediate and delayed feedback in everyday life.

3.4.1 Immediate and Delayed Feedback

First of all, it should be noted that, unlike in our studies, real life examples of feedback-based learning often contain situations in which feedback is delayed by more than a few seconds. The findings we obtained from the three studies conducted as part of this dissertation are limited to feedback that was delayed by a maximum of seven seconds. It is questionable whether the neural mechanisms of feedback processing remain consistent for longer delays. Thus, studying neural processes that underly learning from feedback that is delayed by several minutes, days or even years remains a challenge for future research.

An example for feedback-based learning that fits our studies, at least in terms of temporal dimensions, relates to the world of sports: Hitting a tennis ball over the net with the right technique and watching it land on a perfect spot in the opponent's court about a second later poses a form of immediate feedback that can be used to improve motor skills. In contrast, when playing golf, the ball usually flies through the air in a high arc for several seconds until it lands in the targeted hole, providing delayed feedback to reinforce motor skills. Another prominent source of immediate and delayed feedback in everyday life are parents: They can immediately praise their child after observing prosocial behavior—such as helping a younger sibling—thereby positively reinforcing the child's social behavior. At the same time, delayed praise at home for a good grade can reinforce earlier efforts in studying. When it comes to learning new

linguistic structures in a foreign language, both immediate and delayed feedback have been shown to support learning, although immediate feedback appears slightly more effective—likely due to its progressive use during the production of new sentences (Li et al., 2016). However, a study by Butler et al. (2007) suggests that delayed feedback following a multiple-choice test may be particularly beneficial, due to the temporally distributed presentation of information. In the context of addiction, behavior such as consuming a glass of champagne does not produce an immediate effect within seconds, yet the delayed onset of a euphoric state can increase the likelihood of future engagement in drinking. Early evidence that individuals are capable of forming associations between aversive outcomes and prior events even after long delays of 24 hours comes from studies on conditioned taste aversion in rats (Smith & Roll, 1967) and humans (Arwas et al., 1989). This phenomenon also has implications for understanding chemotherapy-induced food aversions (Arwas et al., 1989). In addition, there are studies investigating the impact of one-week delayed feedback in the form of website-delivered information about one's driving style (Dijksterhuis et al., 2015). Such feedback was found to reduce undesirable driving behaviors, suggesting that pay-as-you-drive insurance models may reinforce not only safer but also more eco-friendly driving (Dijksterhuis et al., 2015). Finally, the growing availability of devices capable of tracking hundreds of organic aspects and functions of the human body reflects the demand for feedback in the pursuit of optimizing physical health. In these cases, effects of behavioral changes like a particular diet or exercise routine do not manifest immediately. Nonetheless, delayed feedback allows individuals to evaluate whether their recent behavior has had positive or negative effects on their health and to maintain or adjust it accordingly. Taken together, these examples underscore the fundamental importance of both immediate and delayed feedback in shaping behavior across a wide

range of everyday contexts. They highlight the areas of application that research on feedback timing can offer outside the laboratory.

3.4.2 Clinical Implications

In general, studying basic learning principles in animals and humans like classical or instrumental conditioning has the potential to explain psychopathology, for example illustrated by the model of learned helplessness from Seligman (1974; Barnes-Holmes et al., 2004). However, according to relational frame theory, animals learn associations only in one direction, while humans form bidirectional relations—a capacity thought to underlie the development of language, complex cognition, and thus human dominance on the planet (Hayes, 2005). Barnes-Holmes et al. (2004) illustrate the principle of bidirectional relations with a simple example: On the one hand, when you hear the words “orange juice” you might mentally re-experience sensory qualities like its smell or taste, even though no juice is actually present. On the other hand, when you perceive the smell of oranges, the words “hmm, orange juice” might spontaneously come to mind. In the first scenario, the words “orange juice” can reactivate its pleasurable, rewarding taste. For the second scenario, the pleasurable smell of oranges can reactivate the words “orange juice”. Our findings that especially rewarding stimuli might reactivate preceding situations could be a mechanism underlying the human ability to form bidirectional relations during feedback learning: In short, certain events can make us anticipate feedback, but feedback can also make us remember certain events. According to Hayes (2005), this capacity to relate anything to anything else is also the reason why humans sometimes suffer: For instance, relaxing on vacation may evoke memories of the stress experienced just prior—such as the pressure to complete tasks at work before leaving—which may help explain why relaxation can, paradoxically, trigger feelings of stress.

Better understanding the neural mechanisms underlying feedback-based learning may help develop interventions that support individuals who struggle with these learning processes, like individuals experiencing depressive symptoms (see Study 3). If, as our findings from Study 2 suggest, the reactivation of previously encountered stimuli facilitates learning from delayed feedback, this insight could be applied in counseling or therapeutic contexts. Humans can use affective experience in everyday life to initiate motivated action (Wichers et al., 2015). Thus, it may be beneficial to encourage individuals experiencing mental health issues like depression to actively recall, or mentally visualize, the situations that preceded a given emotional state. This could for example help to link low mood to earlier activities like lying alone on the couch, eating junk food, and binge-watching a TV series. Instead, individuals could learn that they may feel better after being physically active, engaging in social activities, eating healthily, and getting enough sleep—and thus increase the likelihood of choosing these behaviors more often. This is by no means to suggest that depression can simply be overcome by, say, eating a salad or going for a walk. But raising awareness for the link between these small steps and their effect on personal well-being might support the process of building healthier habits over time.

In a clinical or counseling context, the concept of different association types poses another relevant aspect: In clinical psychology and psychotherapy, it is crucial to distinguish between different types of associations—such as those linking actions to outcomes versus those linking stimuli to outcomes—when designing effective therapeutic interventions. The key differentiation may be whether the goal is to influence the likelihood of health-promoting or harmful behavior, or to modify the evaluation of a particular stimulus or situation occurring in a patient's environment.

When it comes to evaluating the effectiveness of therapeutic interventions—such

as treatments for depression—physiological biomarkers like ERPs can serve as objective measures. For example, increases in the RewP have been associated with reductions in depressive symptoms following both cognitive-behavioral therapy and pharmacological treatment (Burkhouse et al., 2018). However, the link between the FRN and depression has been inconsistent across studies (see Study 3; Hager et al., 2022; Clayson et al., 2020; Moran et al., 2017). Our findings suggest that the N170 may represent a promising additional outcome measure for assessing the effect of therapeutic interventions on feedback processing in depression. Moreover, ERPs are also well suited as objective indicators to highlight the importance of preventive efforts regarding mental health. On the one hand, findings of altered feedback processing before the onset of depression (Bress et al., 2013) as well as evidence linking a familial history of depression to impaired feedback processing and an increased risk for developing the disorder (see Study 3; Halligan et al., 2007; Raposa et al., 2014; Weinberg et al., 2015) may imply the presence of a latent pathology that will eventually manifest. On the other hand, they can also serve to empower individuals to take early action and care for their mental well-being proactively. Using insights from EEG studies on feedback-based learning for psychoeducation and raising awareness is therefore essential.

4 BIBLIOGRAPHY

Aarts, K., & Pourtois, G. (2012). Anxiety disrupts the evaluative component of performance monitoring: An ERP study. *Neuropsychologia*, *50*(7), 1286-1296. https://doi.org/10.1016/j.neuropsychologia.2012.02.012

Admon, R., Kaiser, R. H., Dillon, D. G., Beltzer, M., Goer, F., Olson, D. P., Vitaliano, G., & Pizzagalli, D. A. (2017). Dopaminergic Enhancement of Striatal Response to Reward in Major Depression. *The American Journal of Psychiatry*, *174*(4), 378–386. https://doi.org/10.1176/appi.ajp.2016.16010111

Admon, R., & Pizzagalli, D. A. (2015). Dysfunctional Reward Processing in Depression. *Current Opinion in Psychology*, *4*, 114–118. https://doi.org/10.1016/j.copsyc.2014.12.011

American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.). American Psychiatric Publishing.

Aminoff, E. M., Kveraga, K., & Bar, M. (2013). The role of the parahippocampal cortex in cognition. *Trends in cognitive sciences*, *17*(8), 379-390. https://doi.org/10.1016/j.tics.2013.06.009

Arbel, Y., Hong, L., Baker, T. E., & Holroyd, C. B. (2017). It's all about timing: An electrophysiological examination of feedback-based learning with immediate and delayed feedback. *Neuropsychologia*, *99*, 179–186. https://doi.org/10.1016/j.neuropsychologia.2017.03.003

Arwas, S., Rolnick, A., & Lubow, R. E. (1989). Conditioned taste aversion in humans using motion-induced sickness as the US. *Behaviour research and therapy*, *27*(3), 295-301. https://doi.org/10.1016/0005-7967(89)90049-1

Ayano G. (2016). Dopamine: Receptors, Functions, Synthesis, Pathways, Locations and

Mental Disorders: Review of Literatures. *Journal of Mental Disorders and Treatment*, *2*(2), 1-4. https://doi.org/10.4172/2471-271X.1000120

Baker, T. E., & Holroyd, C. B. (2009). Which way do I go? Neural activation in response to feedback and spatial processing in a virtual T-maze. *Cerebral cortex*, *19*(8), 1708-1722. https://doi.org/10.1093/cercor/bhn223

Baker, T. E., & Holroyd, C. B. (2013). The topographical N170: electrophysiological evidence of a neural mechanism for human spatial navigation. *Biological psychology*, *94*(1), 90-105. https://doi.org/10.1016/j.biopsycho.2013.05.004

Baker, K. S., Johnston, P., Yamamoto, N., & Pegna, A. J. (2023). Event-related potentials index prediction error signalling during perceptual processing of emotional facial expressions. *Brain Topography*, *36*(3), 419-432. https://doi.org/10.1007/s10548-023-00951-2

Baker, K. S., Pegna, A. J., Yamamoto, N., & Johnston, P. (2021). Attention and prediction modulations in expected and unexpected visuospatial trajectories. *PLoS One*, *16*(10), e0242753. https://doi.org/10.1371/journal.pone.0242753

Baker, T. E., Umemoto, A., Krawitz, A., & Holroyd, C. B. (2015). Rightward-biased hemodynamic response of the parahippocampal system during virtual navigation. *Scientific reports*, *5*(1), 9063. https://doi.org/10.1038/srep09063

Bakic, J., Pourtois, G., Jepma, M., Duprat, R., Raedt, R. de, & Baeken, C. (2017). Spared internal but impaired external reward prediction error signals in major depressive disorder during reinforcement learning. *Depression and Anxiety*, *34*(1), 89–96. https://doi.org/10.1002/da.22576

Bao, S., Chan, V. T., & Merzenich, M. M. (2001). Cortical remodelling induced by activity of ventral tegmental dopamine neurons. *Nature*, *412*(6842), 79-83. https://doi.org/10.1038/35083586

Barnes, J. (Ed.). (2014). *The Complete Works of Aristotle, Volume One: The Revised Oxford Translation*. Princeton University Press.

Barnes-Holmes, Y., Barnes-Holmes, D., McHugh, L., & Hayes, S. C. (2004). Relational frame theory: Some implications for understanding and treating human psychopathology. *International journal of psychology and psychological therapy*, *4*, 355-375. https://mural.maynoothuniversity.ie/id/eprint/402

Barto, A. G. (1995). Adaptive critics and the basal ganglia. In J. C. Houk, J. L. Davis, and D. G. Beiser (Eds.), *Models of Information Processing in the Basal Ganglia*, pp. 215–232. MIT Press, Cambridge, MA. https://doi.org/10.7551/mitpress/4708.003.0018

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of statistical software*, *67*, 1-48. https://doi.org/10.18637/jss.v067.i01

Beck, A. T., Steer, R. A., & Brown, G. (1996). Beck depression inventory–II. *APA PsycTests*. https://psycnet.apa.org/doi/10.1037/t00742-000

Becker, M. P., Nitsch, A. M., Miltner, W. H., & Straube, T. (2014). A single-trial estimation of the feedback-related negativity and its relation to BOLD responses in a time-estimation task. *Journal of Neuroscience*, *34*(8), 3005-3012. https://doi.org/10.1523/JNEUROSCI.3684-13.2014

Bellebaum, C., & Colosio, M. (2014). From feedback-to response-based performance monitoring in active and observational learning. *Journal of cognitive neuroscience*, *26*(9), 2111-2127. https://doi.org/10.1162/jocn_a_00612

Bellebaum, C., & Daum, I. (2008). Learning-related changes in reward expectancy are reflected in the feedback-related negativity. *European Journal of Neuroscience*, *27*(7), 1823-1835. https://doi.org/10.1111/j.1460-9568.2008.06138.x

Bellebaum, C., Jokisch, D., Gizewski, E. R., Forsting, M., & Daum, I. (2012). The neural coding of expected and unexpected monetary performance outcomes: Dissociations between active and observational learning. *Behavioural Brain Research*, *227*(1), 241–251. https://doi.org/10.1016/j.bbr.2011.10.042

Bentin, S., Allison, T., Puce, A., Perez, E., & McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *Journal of cognitive neuroscience*, *8*(6), 551-565. https://doi.org/10.1162/jocn.1996.8.6.551

Björklund, A., & Dunnett, S. B. (2007). Dopamine neuron systems in the brain: an update. *Trends in neurosciences*, *30*(5), 194-202. https://doi.org/10.1016/j.tins.2007.03.006

Brain Products GmbH. (2018). BrainVision Analyzer 2.2 [Computer software]. Gilching, Germany: Brain Products GmbH.

Brem, S., Bucher, K., Halder, P., Summers, P., Dietrich, T., Martin, E., & Brandeis, D. (2006). Evidence for developmental changes in the visual word processing network beyond adolescence. *Neuroimage*, *29*(3), 822-837. https://doi.org/10.1016/j.neuroimage.2005.09.023

Bress, J. N., Foti, D., Kotov, R., Klein, D. N., & Hajcak, G. (2013). Blunted neural response to rewards prospectively predicts depression in adolescent girls. *Psychophysiology*, *50*(1), 74-81. https://doi.org/10.1111/j.1469-8986.2012.01485.x

Bress, J. N., Meyer, A., & Proudfit, G. H. (2015). The stability of the feedback negativity and its relationship with depression during childhood and adolescence. *Development and Psychopathology*, *27*(4 Pt 1), 1285–1294. https://doi.org/10.1017/S0954579414001400

Bress, J. N., Smith, E., Foti, D., Klein, D. N., & Hajcak, G. (2012). Neural response to

reward and depressive symptoms in late childhood to early adolescence. *Biological Psychology*, *89*(1), 156–162. https://doi.org/10.1016/j.biopsycho.2011.10.004

Brush, C. J., Ehmann, P. J., Hajcak, G., Selby, E. A., & Alderman, B. L. (2018). Using multilevel modeling to examine blunted neural responses to reward in major depression. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *3*(12), 1032-1039. https://doi.org/10.1016/j.bpsc.2018.04.003

Burkhouse, K. L., Gorka, S. M., Klumpp, H., Kennedy, A. E., Karich, S., Francis, J., ... & Phan, K. L. (2018). Neural responsiveness to reward as an index of depressive symptom change following cognitive-behavioral therapy and SSRI treatment. *The Journal of clinical psychiatry*, *79*(4), 12587. https://doi.org/10.4088/JCP.17m11836

Burnside, R., Fischer, A. G., & Ullsperger, M. (2019). The feedback-related negativity indexes PE in active but not observational learning. *Psychophysiology*, *56*(9), e13389. https://doi.org/10.1111/psyp.13389

Burton, R. (1857). *The anatomy of melancholy*. JW Moore.

Busch, M., Maske, U., Ryl, L., Schlack, R., & Hapke, U. (2013). Prävalenz von depressiver Symptomatik und diagnostizierter Depression bei Erwachsenen in Deutschland. *Bundesgesundheitsbl. 56*, 733–739. https://doi.org/10.1007/s00103-013-1688-3

Butler, A. J., & James, K. H. (2011). Cross-modal versus within-modal recall: Differences in behavioral and brain responses. *Behavioural brain research*, *224*(2), 387-396. https://doi.org/10.1016/j.bbr.2011.06.017

Butler, A. C., Karpicke, J. D., & Roediger III, H. L. (2007). The effect of type and timing of feedback on learning from multiple-choice tests. *Journal of*

Experimental Psychology: Applied, *13*(4), 273-281. https://doi.org/10.1037/1076-898X.13.4.273

Caine, E. D. (1981). Mental status changes with aging. In *Seminars in Neurology* (Vol. 1, No. 01, pp. 36-42). Thieme Medical Publishers, Inc..

Cannon, D. S., Tiffany, S. T., Coon, H., Scholand, M. B., McMahon, W. M., & Leppert, M. F. (2007). The PHQ-9 as a brief assessment of lifetime major depression. *Psychological assessment*, *19*(2), 247-251. https://psycnet.apa.org/doi/10.1037/1040-3590.19.2.247

Carlson, J. M., Foti, D., Mujica-Parodi, L. R., Harmon-Jones, E., & Hajcak, G. (2011). Ventral striatal and medial prefrontal BOLD activation is correlated with reward-related electrocortical activity: a combined ERP and fMRI study. *Neuroimage*, *57*(4), 1608-1616. https://doi.org/10.1016/j.neuroimage.2011.05.037

Carreiras, M., Armstrong, B. C., Perea, M., & Frost, R. (2014). The what, when, where, and how of visual word recognition. *Trends in cognitive sciences*, *18*(2), 90-98. https://doi.org/10.1016/j.tics.2013.11.005

Chen, C., Takahashi, T., Nakagawa, S., Inoue, T., & Kusumi, I. (2015). Reinforcement learning in depression: A review of computational research. *Neuroscience and Biobehavioral Reviews*, *55*, 247–267. https://doi.org/10.1016/j.neubiorev.2015.05.005

Chuhma, N., Oh, S. J., & Rayport, S. (2023). The dopamine neuron synaptic map in the striatum. *Cell reports*, *42*(3). https://doi.org/10.1016/j.celrep.2023.112204

Clark, R. E., & Squire, L. R. (1998). Classical conditioning and brain systems: the role of awareness. *Science*, *280*(5360), 77-81. https://doi.org/10.1126/science.280.5360.77

Clayson, P. E., Carbine, K. A., & Larson, M. J. (2020). A registered report of error-related negativity and reward positivity as biomarkers of depression: P-Curving the evidence. *International Journal of Psychophysiology*, *150*, 50-72. https://doi.org/10.1016/j.ijpsycho.2020.01.005

Cloutier, J., Heatherton, T. F., Whalen, P. J., & Kelley, W. M. (2008). Are attractive people rewarding? Sex differences in the neural substrates of facial attractiveness. *Journal of cognitive neuroscience*, *20*(6), 941-951. https://doi.org/10.1162/jocn.2008.20062

Cohen, J. D., & Blum, K. I. (2002). Reward and decision. *Neuron*, *36*(2), 193-198. https://doi.org/10.1016/S0896-6273(02)00973-X

Cohen, L., Lehéricy, S., Chochon, F., Lemer, C., Rivaud, S., & Dehaene, S. (2002). Language-specific tuning of visual cortex? Functional properties of the Visual Word Form Area. *Brain*, *125*(5), 1054-1069. https://doi.org/10.1093/brain/awf094

Cohen, M. X., & Ranganath, C. (2007). Reinforcement learning signals predict future decisions. *Journal of Neuroscience*, *27*(2), 371-378. https://doi.org/10.1523/JNEUROSCI.4421-06.2007

Collins, A. G., & Frank, M. J. (2014). Opponent actor learning (OpAL): modeling interactive effects of striatal dopamine on reinforcement learning and choice incentive. *Psychological review*, *121*(3), 337-366. https://psycnet.apa.org/doi/10.1037/a0037015

Curtis, C. E., & Lee, D. (2010). Beyond working memory: the role of persistent activity in decision making. *Trends in cognitive sciences*, *14*(5), 216-222. https://doi.org/10.1016/j.tics.2010.03.006

Damier, P., Hirsch, E. C., Agid, Y., & Graybiel, A. (1999). The substantia nigra of the

human brain: II. Patterns of loss of dopamine-containing neurons in Parkinson's disease. *Brain*, *122*(8), 1437-1448. https://doi.org/10.1093/brain/122.8.1437

Danker, J. F., & Anderson, J. R. (2010). The ghosts of brain states past: Remembering reactivates the brain regions engaged during encoding. *Psychological Bulletin*, *136*(1), 87–102. https://doi.org/10.1037/a0017937

Davidow, J. Y., Foerde, K., Galván, A., & Shohamy, D. (2016). An upside to reward sensitivity: the hippocampus supports enhanced reinforcement learning in adolescence. *Neuron*, *92*(1), 93-99. https://doi.org/10.1016/j.neuron.2016.08.031

Dayan, P., & Balleine, B. W. (2002). Reward, motivation, and reinforcement learning. *Neuron*, *36*(2), 285-298. https://doi.org/10.1016/S0896-6273(02)00963-7

Deffke, I., Sander, T., Heidenreich, J., Sommer, W., Curio, G., Trahms, L., & Lueschow, A. (2007). MEG/EEG sources of the 170-ms response to faces are co-localized in the fusiform gyrus. *Neuroimage*, *35*(4), 1495-1501. https://doi.org/10.1016/j.neuroimage.2007.01.034

Demos, K. E., Heatherton, T. F., & Kelley, W. M. (2012). Individual differences in nucleus accumbens activity to food and sexual images predict weight gain and sexual behavior. *Journal of Neuroscience*, *32*(16), 5549-5552. https://doi.org/10.1523/JNEUROSCI.5958-11.2012

den Ouden, H. E., Daunizeau, J., Roiser, J., Friston, K. J., & Stephan, K. E. (2010). Striatal prediction error modulates cortical coupling. *Journal of Neuroscience*, *30*(9), 3210-3219. https://doi.org/10.1523/JNEUROSCI.4458-09.2010

Di Chiara, G. (2002). Nucleus accumbens shell and core dopamine: differential role in behavior and addiction. *Behavioural brain research*, *137*(1-2), 75-114. https://doi.org/10.1016/S0166-4328(02)00286-3

Dickerson, K. C., Li, J., & Delgado, M. R. (2011). Parallel contributions of distinct

human memory systems during probabilistic learning. *Neuroimage*, *55*(1), 266-276. https://doi.org/10.1016/j.neuroimage.2010.10.080

Dijksterhuis, C., Lewis-Evans, B., Jelijs, B., de Waard, D., Brookhuis, K., & Tucha, O. (2015). The impact of immediate or delayed feedback on driving behaviour in a simulated Pay-As-You-Drive system. *Accident Analysis & Prevention*, *75*, 93-104. https://doi.org/10.1016/j.aap.2014.11.017

Dillon, D. G., & Pizzagalli, D. A. (2018). Mechanisms of Memory Disruption in Depression. *Trends in Neurosciences*, *41*(3), 137–149. https://doi.org/10.1016/j.tins.2017.12.006

Donohue, J. M., & Pincus, H. A. (2007). Reducing the societal burden of depression: a review of economic costs, quality of care and effects of treatment. *Pharmacoeconomics*, *25*, 7-24. https://doi.org/10.2165/00019053-200725010-00003

Edelmann, E., & Lessmann, V. (2018). Dopaminergic innervation and modulation of hippocampal networks. *Cell and tissue research*, *373*, 711-727. https://doi.org/10.1007/s00441-018-2800-7

Eichenbaum, H., Otto, T., & Cohen, N. J. (1992). The hippocampus—what does it do? *Behavioral and Neural Biology*, *57*(1), 2–36. https://doi.org/10.1016/0163-1047(92)90724-I

Elliott, R., Sahakian, B. J., Herrod, J. J., Robbins, T. W., & Paykel, E. S. (1997). Abnormal response to negative feedback in unipolar depression: evidence for a diagnosis specific impairment. *Journal of Neurology, Neurosurgery & Psychiatry*, *63*(1), 74-82. https://doi.org/10.1136/jnnp.63.1.74

Elliott, R., Sahakian, B. J., McKay, A. P., Herrod, J. J., Robbins, T. W., & Paykel, E. S. (1996). Neuropsychological impairments in unipolar depression: the influence of

perceived failure on subsequent performance. *Psychological medicine*, *26*(5), 975-989. https://doi.org/10.1017/S0033291700035303

Eppinger, B., Kray, J., Mock, B., & Mecklinger, A. (2008). Better or worse than expected? Aging, learning, and the ERN. *Neuropsychologia*, *46*(2), 521-539. https://doi.org/10.1016/j.neuropsychologia.2007.09.001

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

Fairhall, S. L., Sharma, S., Magnusson, J., & Murphy, B. (2010). Memory related dysregulation of hippocampal function in major depressive disorder. *Biological Psychology*, *85*(3), 499–503. https://doi.org/10.1016/j.biopsycho.2010.09.002

Fischer, A. G., & Ullsperger, M. (2013). Real and fictive outcomes are processed differently but converge on a common adaptive mechanism. *Neuron*, *79*(6), 1243-1255. https://doi.org/10.1016/j.neuron.2013.07.006

Floresco, S. B., & Magyar, O. (2006). Mesocortical dopamine modulation of executive functions: beyond working memory. *Psychopharmacology*, *188*, 567-585. https://doi.org/10.1007/s00213-006-0404-5

Foerde, K., Race, E., Verfaellie, M., & Shohamy, D. (2013). A role for the medial temporal lobe in feedback-driven learning: Evidence from amnesia. *Journal of Neuroscience*, *33*(13), 5698–5704. https://doi.org/10.1523/JNEUROSCI.5217-12.2013

Foerde, K., & Shohamy, D. (2011). Feedback timing modulates brain systems for learning in humans. *Journal of Neuroscience*, *31*(37), 13157–13167. https://doi.org/10.1523/JNEUROSCI.2701-11.2011

Foti, D., Carlson, J. M., Sauder, C. L., & Proudfit, G. H. (2014). Reward dysfunction in

major depression: Multimodal neuroimaging evidence for refining the melancholic phenotype. *NeuroImage*, *101*, 50–58. https://doi.org/10.1016/j.neuroimage.2014.06.058

Foti, D., Weinberg, A., Dien, J., & Hajcak, G. (2011). Event-related potential activity in the basal ganglia differentiates rewards from nonrewards: Temporospatial principal components analysis and source localization of the feedback negativity. *Human brain mapping*, *32*(12), 2207-2216. https://doi.org/10.1002/hbm.21182

Frank, M. J., Seeberger, L. C., & O'reilly, R. C. (2004). By carrot or by stick: cognitive reinforcement learning in parkinsonism. *Science*, *306*(5703), 1940-1943. https://doi.org/10.1126/science.1102941

Fu, W. T., & Anderson, J. R. (2008). Solving the credit assignment problem: explicit and implicit learning of action sequences with probabilistic outcomes. *Psychological research*, *72*, 321-330. https://doi.org/10.1007/s00426-007-0113-7

Gao, C., Conte, S., Richards, J. E., Xie, W., & Hanayik, T. (2019). The neural sources of N170: Understanding timing of activation in face-selective areas. *Psychophysiology*, *56*(6), e13336. https://doi.org/10.1111/psyp.13336

Garrison, J., Erdeniz, B., & Done, J. (2013). Prediction error in reinforcement learning: a meta-analysis of neuroimaging studies. *Neuroscience & Biobehavioral Reviews*, *37*(7), 1297-1310. https://doi.org/10.1016/j.neubiorev.2013.03.023

Gasbarri, A., Sulli, A., & Packard, M. G. (1997). The dopaminergic mesencephalic projections to the hippocampal formation in the rat. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *21*(1), 1-22. https://doi.org/10.1016/S0278-5846(96)00157-1

Gasbarri, A., Verney, C., Innocenzi, R., Campana, E., & Pacitti, C. (1994). Mesolimbic dopaminergic neurons innervating the hippocampal formation in the rat: a combined retrograde tracing and immunohistochemical study. *Brain research*, *668*(1-2), 71-79. https://doi.org/10.1016/0006-8993(94)90512-6

Gehring, W. J., & Willoughby, A. R. (2002). The medial frontal cortex and the rapid processing of monetary gains and losses. *Science*, *295*(5563), 2279-2282. https://doi.org/10.1126/science.1066893

Glimcher, P. W. (2011). Understanding dopamine and reinforcement learning: the dopamine reward prediction error hypothesis. *Proceedings of the National Academy of Sciences*, *108*(supplement_3), 15647-15654. https://doi.org/10.1073/pnas.1014269108

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in neurosciences*, *15*(1), 20-25. https://doi.org/10.1016/0166-2236(92)90344-8

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: current status and future directions. *Annual review of clinical psychology*, *6*(1), 285-312. https://doi.org/10.1146/annurev.clinpsy.121208.131305

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: etymology and strategic intentions. *American journal of psychiatry*, *160*(4), 636-645. https://doi.org/10.1176/appi.ajp.160.4.636

Grabowska, A., Sondej, F., & Senderecka, M. (2024). A network analysis of affective and motivational individual differences and error monitoring in a non-clinical sample. *Cerebral Cortex*, *34*(10), bhae397. https://doi.org/10.1093/cercor/bhae397

Gräfe, K., Zipfel, S., Herzog, W., & Löwe, B. (2004). Screening psychischer Störungen

mit dem “Gesundheitsfragebogen für Patienten (PHQ-D)“. *Diagnostica*, *50*(4), 171-181. https://doi.org/10.1026/0012-1924.50.4.171

Greenberg, G. (2010). *Manufacturing depression: The secret history of a modern disease*. Simon and Schuster.

Gu, R., Ge, Y., Jiang, Y., & Luo, Y. J. (2010). Anxiety and outcome evaluation: the good, the bad and the ambiguous. *Biological psychology*, *85*(2), 200-206. https://doi.org/10.1016/j.biopsycho.2010.07.001

Haber, S. N., & Knutson, B. (2010). The reward circuit: linking primate anatomy and human imaging. *Neuropsychopharmacology*, *35*(1), 4-26. https://doi.org/10.1038/npp.2009.129

Hager, N. M., Judah, M. R., & Rawls, E. (2022). Win, lose, or draw: Examining salience, reward memory, and depression with the reward positivity. *Psychophysiology*, *59*(1), e13953. https://doi.org/10.1111/psyp.13953

Hajcak, G., Moser, J. S., Holroyd, C. B., & Simons, R. F. (2007). It's worse than you thought: The feedback negativity and violations of reward prediction in gambling tasks. *Psychophysiology*, *44*(6), 905-912. https://doi.org/10.1111/j.1469-8986.2007.00567.x

Halligan, S. L., Murray, L., Martins, C., & Cooper, P. J. (2007). Maternal depression and psychiatric outcomes in adolescent offspring: A 13-year longitudinal study. *Journal of Affective Disorders*, *97*(1-3), 145–154. https://doi.org/10.1016/j.jad.2006.06.010

Hamann, S., Herman, R. A., Nolan, C. L., & Wallen, K. (2004). Men and women differ in amygdala response to visual sexual stimuli. *Nature neuroscience*, *7*(4), 411-416. https://doi.org/10.1038/nn1208

Hamid, A. A., Pettibone, J. R., Mabrouk, O. S., Hetrick, V. L., Schmidt, R., Vander

Weele, C. M., ... & Berke, J. D. (2016). Mesolimbic dopamine signals the value of work. *Nature neuroscience*, *19*(1), 117-126. https://doi.org/10.1038/nn.4173

Hapke, U., Cohrdes, C., & Nübel, J. (2019). Depressive Symptomatik im europäischen Vergleich–Ergebnisse des European Health Interview Survey (EHIS) 2. *Journal of Health Monitoring*, *4*(4), 62-70. https://doi.org/10.25646/6221

Haruno, M., & Kawato, M. (2006). Different neural correlates of reward expectation and reward expectation error in the putamen and caudate nucleus during stimulus-action-reward association learning. *Journal of neurophysiology*, *95*(2), 948-959. https://doi.org/10.1152/jn.00382.2005

Hauser, T. U., Iannaccone, R., Stämpfli, P., Drechsler, R., Brandeis, D., Walitza, S., & Brem, S. (2014). The feedback-related negativity (FRN) revisited: new insights into the localization, meaning and network organization. *Neuroimage*, *84*, 159-168. https://doi.org/10.1016/j.neuroimage.2013.08.028

Hautzinger, M., Keller, F., & Kühner, C. (2006). *BDI II*. Frankfurt a.M.: Harcourt Test Services.

Hayes, S. C. (2005). *Get out of your mind and into your life: The new acceptance and commitment therapy.* New Harbinger Publications.

Hebb, D. O. (1949). *The organization of behavior: A neuropsychological theory*. Psychology Press. https://doi.org/10.4324/9781410612403

Herholz, S. C., Halpern, A. R., & Zatorre, R. J. (2012). Neuronal correlates of perception, imagery, and memory for familiar tunes. *Journal of cognitive neuroscience*, *24*(6), 1382-1397. https://doi.org/10.1162/jocn_a_00216

Heshmati, M., & Russo, S. J. (2015). Anhedonia and the brain reward circuitry in depression. *Current Behavioral Neuroscience Reports*, *2*(3), 146–153. https://doi.org/10.1007/s40473-015-0044-3

Hidaka, B. H. (2012). Depression as a disease of modernity: explanations for increasing prevalence. *Journal of affective disorders*, *140*(3), 205-214. https://doi.org/10.1016/j.jad.2011.12.036

Hiebert, N. M., Vo, A., Hampshire, A., Owen, A. M., Seergobin, K. N., & MacDonald, P. A. (2014). Striatum in stimulus–response learning via feedback and in decision making. *Neuroimage*, *101*, 448-457. https://doi.org/10.1016/j.neuroimage.2014.07.013

Hoffman, E. A., & Haxby, J. V. (2000). Distinct representations of eye gaze and identity in the distributed human neural system for face perception. *Nature neuroscience*, *3*(1), 80-84. https://doi.org/10.1038/71152

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*(4), 679–709. https://doi.org/10.1037/0033-295X.109.4.679

Holroyd, C. B., Hajcak, G., & Larsen, J. T. (2006). The good, the bad and the neutral: Electrophysiological responses to feedback stimuli. *Brain Research*, *1105*, 93–101. https://doi.org/10.1016/j.brainres.2005.12.015

Holroyd, C. B., Larsen, J. T., & Cohen, J. D. (2004). Context dependence of the event-related brain potential associated with reward and punishment. *Psychophysiology*, *41*(2), 245-253. https://doi.org/10.1111/j.1469-8986.2004.00152.x

Höltje, G., & Mecklinger, A. (2020). Feedback timing modulates interactions between feedback processing and memory encoding: Evidence from event-related potentials. *Cognitive, Affective, & Behavioral Neuroscience*, *20*(2), 250-264. https://doi.org/10.3758/s13415-019-00765-5

Hosp, J. A., Pekanovic, A., Rioult-Pedotti, M. S., & Luft, A. R. (2011). Dopaminergic projections from midbrain to primary motor cortex mediate motor skill learning. *Journal of Neuroscience*, *31*(7), 2481-2487. https://doi.org/10.1523/JNEUROSCI.5411-10.2011

Hougen, D. F., & Shah, S. N. H. (2019). The evolution of reinforcement learning. In *2019 IEEE Symposium Series on Computational Intelligence* (SSCI) (pp. 1457-1464). IEEE. https://doi.org/10.1109/SSCI44817.2019.9003146

Hu, H., Cui, Y., & Yang, Y. (2020). Circuits and functions of the lateral habenula in health and in disease. *Nature Reviews Neuroscience*, *21*(5), 277-295. https://doi.org/10.1038/s41583-020-0292-4

Huys, Q. J., Pizzagalli, D. A., Bogdan, R., & Dayan, P. (2013). Mapping anhedonia onto reinforcement learning: a behavioural meta-analysis. *Biology of mood & anxiety disorders*, *3*, 1-16. https://doi.org/10.1186/2045-5380-3-12

Iidaka, T., Matsumoto, A., Haneda, K., Okada, T., & Sadato, N. (2006). Hemodynamic and electrophysiological relationship involved in human face processing: Evidence from a combined fMRI–ERP study. *Brain and Cognition*, *60*(2), 176-186. https://doi.org/10.1016/j.bandc.2005.11.004

Iordanova, M. D., Yau, J. O. Y., McDannald, M. A., & Corbit, L. H. (2021). Neural substrates of appetitive and aversive prediction error. *Neuroscience & Biobehavioral Reviews*, *123*, 337-351. https://doi.org/10.1016/j.neubiorev.2020.10.029

Itier, R. J., & Taylor, M. J. (2004). N170 or N1? Spatiotemporal differences between object and face processing using ERPs. *Cerebral cortex*, *14*(2), 132-142. https://doi.org/10.1093/cercor/bhg111

Jacobi, F., Wittchen, H. U., Hölting, C., Höfler, M., Pfister, H., Müller, N., & Lieb, R.

(2004). Prevalence, co-morbidity and correlates of mental disorders in the general population: results from the German Health Interview and Examination Survey (GHS). *Psychological medicine*, *34*(4), 597-611. https://doi.org/10.1017/S0033291703001399

Jiang, D., Zhang, D., Chen, Y., He, Z., Gao, Q., Gu, R., & Xu, P. (2018). Trait anxiety and probabilistic learning: Behavioral and electrophysiological findings. *Biological Psychology*, *132*, 17-26. https://doi.org/10.1016/j.biopsycho.2017.10.010

Jocham, G., Brodersen, K. H., Constantinescu, A. O., Kahn, M. C., Ianni, A. M., Walton, M. E., Rushworth, M. F. S., & Behrens, T. E. J. (2016). Reward-Guided Learning with and without Causal Attribution. *Neuron*, *90*(1), 177–190. https://doi.org/10.1016/j.neuron.2016.02.018

Joel, D., Niv, Y., & Ruppin, E. (2002). Actor–critic models of the basal ganglia: New anatomical and computational perspectives. *Neural networks*, *15*(4-6), 535-547. https://doi.org/10.1016/S0893-6080(02)00047-3

Kahn, I., & Shohamy, D. (2013). Intrinsic connectivity between the hippocampus, nucleus accumbens, and ventral tegmental area in humans. *Hippocampus*, *23*(3), 187-192. https://doi.org/10.1002/hipo.22077

Kamin, L. J. (1967). Predictability, surprise, attention, and conditioning. In *Symp. on punishment* (No. TR-13).

Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: a module in human extrastriate cortex specialized for face perception. *Journal of neuroscience*, *17*(11), 4302-4311. https://doi.org/10.1523/JNEUROSCI.17-11-04302.1997

Kanwisher, N., & Yovel, G. (2006). The fusiform face area: a cortical region

specialized for the perception of faces. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *361*(1476), 2109-2128. https://doi.org/10.1098/rstb.2006.1934

Keren, H., O'Callaghan, G., Vidal-Ribas, P., Buzzell, G. A., Brotman, M. A., Leibenluft, E., Pan, P. M., Meffert, L., Kaiser, A., Wolke, S., Pine, D. S., & Stringaris, A. (2018). Reward Processing in Depression: A Conceptual and Meta-Analytic Review Across fMRI and EEG Studies. *The American Journal of Psychiatry*, *175*(11), 1111–1120. https://doi.org/10.1176/appi.ajp.2018.17101124

Kim, S., & Arbel, Y. (2019). Immediate and delayed auditory feedback in declarative learning: An examination of the feedback related event related potentials. *Neuropsychologia*, *129*, 255-262. https://doi.org/10.1016/j.neuropsychologia.2019.04.001

Kimura, K., & Kimura, M. (2016). Temporal prediction restores the evaluative processing of delayed action feedback: an electrophysiological study. *NeuroReport*, *27*(14), 1061-1067. https://doi.org/10.1097/WNR.0000000000000657

Kirsch, F., Kirschner, H., Fischer, A. G., Klein, T. A., & Ullsperger, M. (2022). Disentangling performance-monitoring signals encoded in feedback-related EEG dynamics. *NeuroImage*, *257*, 119322. https://doi.org/10.1016/j.neuroimage.2022.119322

Klawohn, J., Burani, K., Bruchnak, A., Santopetro, N., & Hajcak, G. (2021). Reduced neural response to reward and pleasant pictures independently relate to depression. *Psychological Medicine*, *51*(5), 741-749. https://doi.org/10.1017/S0033291719003659

Kloth, N., Itier, R. J., & Schweinberger, S. R. (2013). Combined effects of inversion and feature removal on N170 responses elicited by faces and car fronts. *Brain and cognition*, *81*(3), 321-328. https://doi.org/10.1016/j.bandc.2013.01.002

Knowlton, B. J., Mangels, J. A., & Squire, L. R. (1996). A neostriatal habit learning system in humans. *Science*, *273*(5280), 1399–1402. https://doi.org/10.1126/science.273.5280.1399

Knutson, B., Adams, C. M., Fong, G. W., & Hommer, D. (2001). Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *The Journal of neuroscience*, *21*(16), RC159.

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9: validity of a brief depression severity measure. *Journal of general internal medicine*, *16*(9), 606-613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x

Kujawa, A., Smith, E., Luhmann, C., & Hajcak, G. (2013). The feedback negativity reflects favorable compared to nonfavorable outcomes based on global, not local, alternatives. *Psychophysiology*, *50*, 134–138. https://doi.org/10.1111/psyp.12002

Kumar, P., Goer, F., Murray, L., Dillon, D. G., Beltzer, M. L., Cohen, A. L., Brooks, N. H., & Pizzagalli, D. A. (2018). Impaired reward prediction error encoding and striatal-midbrain connectivity in depression. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *43*(7), 1581–1588. https://doi.org/10.1038/s41386-018-0032-x

Kunisato, Y., Okamoto, Y., Ueda, K., Onoda, K., Okada, G., Yoshimura, S., Suzuki, S., Samejima, K., & Yamawaki, S. (2012). Effects of depression on reward-based decision making and variability of action in probabilistic learning. *Journal of*

Behavior Therapy and Experimental Psychiatry, *43*(4), 1088–1094. https://doi.org/10.1016/j.jbtep.2012.05.007

Lamers, F., van Oppen, P., Comijs, H. C., Smit, J. H., Spinhoven, P., van Balkom, A. J., ... & Penninx, B. W. (2011). Comorbidity patterns of anxiety and depressive disorders in a large cohort study: the Netherlands Study of Depression and Anxiety (NESDA). *The Journal of clinical psychiatry*, *72*(3), 341-348. https://doi.org/10.4088/JCP.10m06176blu

Lawson, R. P., Seymour, B., Loh, E., Lutti, A., Dolan, R. J., Dayan, P., ... & Roiser, J. P. (2014). The habenula encodes negative motivational value associated with primary punishment in humans. *Proceedings of the National Academy of Sciences*, *111*(32), 11858-11863. https://doi.org/10.1073/pnas.1323586111

Lefebvre, G., Lebreton, M., Meyniel, F., Bourgeois-Gironde, S., & Palminteri, S. (2017). Behavioural and neural characterization of optimistic reinforcement learning. *Nature Human Behaviour*, *1*(4), 0067. https://doi.org/10.1038/s41562-017-0067

Li, S., Zhu, Y., & Ellis, R. (2016). The effects of the timing of corrective feedback on the acquisition of a new linguistic structure. *The Modern Language Journal*, *100*(1), 276-295. https://doi.org/10.1111/modl.12315

Lisman, J. E., & Grace, A. A. (2005). The hippocampal-VTA loop: controlling the entry of information into long-term memory. *Neuron*, *46*(5), 703-713. https://doi.org/10.1016/j.neuron.2005.05.002

Luck, S. J. (2014). *An introduction to the event-related potential technique* (2nd ed.). MIT press.

Luck, S. J. (2023). Event-related potentials. In H. Cooper, M. N. Coutanche, L. M. McMullen, A. T. Panter, D. Rindskopf, & K. J. Sher (Eds.), *APA handbook of*

research methods in psychology: Foundations, planning, measures, and psychometrics (2nd ed., pp. 605–630). American Psychological Association. https://doi.org/10.1037/0000318-028

Luft, A. R., & Schwarz, S. (2009). Dopaminergic signals in primary motor cortex. *International Journal of Developmental Neuroscience*, *27*(5), 415-421. https://doi.org/10.1016/j.ijdevneu.2009.05.004

Luking, K. R., Pagliaccio, D., Luby, J. L., & Barch, D. M. (2016). Reward Processing and Risk for Depression Across Development. *Trends in Cognitive Sciences*, *20*(6), 456–468. https://doi.org/10.1016/j.tics.2016.04.002

Margraf, J. & Cwik, J. C. (2017). Mini-DIPS Open Access: Diagnostisches Kurzinterview bei psychischen Störungen. Bochum: Forschungs- und Behandlungszentrum für psychische Gesundheit, Ruhr-Universität Bochum. https://doi.org/10.13154/rub.102.91

Margraf, J., Cwik, J. C., Pflug, V., & Schneider, S. (2017). Strukturierte klinische Interviews zur Erfassung psychischer Störungen über die Lebensspanne. *Zeitschrift für Klinische Psychologie und Psychotherapie*, *46*(3), 176-186. https://doi.org/10.1026/1616-3443/a000430

McCabe, C., Cowen, P. J., & Harmer, C. J. (2009). Neural representation of reward in recovered depressed patients. *Psychopharmacology*, *205*(4), 667–677. https://doi.org/10.1007/s00213-009-1573-9

McDougle, S. D., Boggess, M. J., Crossley, M. J., Parvin, D., Ivry, R. B., & Taylor, J. A. (2016). Credit assignment in movement-dependent reinforcement learning. *Proceedings of the National Academy of Sciences*, *113*(24), 6797-6802. https://doi.org/10.1073/pnas.1523669113

Meteyard, L., & Davies, R. A. (2020). Best practice guidance for linear mixed-effects

models in psychological science. *Journal of Memory and Language*, *112*, 104092. https://doi.org/10.1016/j.jml.2020.104092

Metzger, M., Souza, R., Lima, L. B., Bueno, D., Gonçalves, L., Sego, C., ... & Shammah-Lagnado, S. J. (2021). Habenular connections with the dopaminergic and serotonergic system and their role in stress-related psychiatric disorders. *European Journal of Neuroscience*, *53*(1), 65-88. https://doi.org/10.1111/ejn.14647

Miller, R. R., Barnet, R. C., & Grahame, N. J. (1995). Assessment of the Rescorla-Wagner model. *Psychological bulletin*, *117*(3), 363-386. https://psycnet.apa.org/doi/10.1037/0033-2909.117.3.363

Miltner, W. H., Braun, C. H., & Coles, M. G. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience*, *9*(6), 788–798. https://doi.org/10.1162/jocn.1997.9.6.788

Mirenowicz, J., & Schultz, W. (1996). Preferential activation of midbrain dopamine neurons by appetitive rather than aversive stimuli. *Nature*, *379*(6564), 449-451. https://doi.org/10.1038/379449a0

Montague, P. R., Dayan, P., & Sejnowski, T. J. (1996). A framework for mesencephalic dopamine systems based on predictive Hebbian learning. *Journal of neuroscience*, *16*(5), 1936-1947. https://doi.org/10.1523/JNEUROSCI.16-05-01936.1996

Moran, T. P., Schroder, H. S., Kneip, C., & Moser, J. S. (2017). Meta-analysis and psychophysiology: A tutorial using depression and action-monitoring event-related potentials. *International Journal of Psychophysiology*, *111*, 17-32. https://doi.org/10.1016/j.ijpsycho.2016.07.001

Murray, C. J. L., Lopez, A. D., World Health Organization, World Bank, & Harvard School of Public Health. (1996). *The global burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020: Summary.* World Health Organization. https://apps.who.int/iris/bitstream/handle/10665/41864/0965546608_eng.pdf

Nestler, E. J., Barrot, M., DiLeone, R. J., Eisch, A. J., Gold, S. J., & Monteggia, L. M. (2002). Neurobiology of depression. *Neuron*, *34*(1), 13-25. https://doi.org/10.1016/S0896-6273(02)00653-0

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

Nieuwenhuis, S., Holroyd, C. B., Mol, N., & Coles, M. G. (2004). Reinforcement-related brain potentials from medial frontal cortex: origins and functional significance. *Neuroscience & Biobehavioral Reviews*, *28*(4), 441-448. https://doi.org/10.1016/j.neubiorev.2004.05.003

Nikooyan, A. A., & Ahmed, A. A. (2015). Reward feedback accelerates motor learning. *Journal of neurophysiology*, *113*(2), 633-646. https://doi.org/10.1152/jn.00032.2014

O'Doherty, J., Dayan, P., Schultz, J., Deichmann, R., Friston, K., & Dolan, R. J. (2004). Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science*, *304*(5669), 452-454. https://doi.org/10.1126/science.1094285

Oerlemans, J., Alejandro, R. J., Van Roost, D., Boon, P., De Herdt, V., Meurs, A., & Holroyd, C. B. (2025). Unravelling the origin of reward positivity: a human intracranial event-related brain potential study. *Brain*, *148*(1), 199-211. https://doi.org/10.1093/brain/awae259

Oldehinkel, M., Llera, A., Faber, M., Huertas, I., Buitelaar, J. K., Bloem, B. R., ... & Beckmann, C. F. (2022). Mapping dopaminergic projections in the human brain with resting-state fMRI. *Elife*, *11*, e71846. https://doi.org/10.7554/eLife.71846

Orfanos, C. E. (2007). From Hippocrates to modern medicine. *Journal of the European Academy of Dermatology and Venereology*, *21*(6), 852-858. https://doi.org/10.1111/j.1468-3083.2007.02273.x

Parent, M., & Parent, A. (2010). Substantia nigra and Parkinson's disease: a brief history of their long and intimate relationship. *Canadian journal of neurological sciences*, *37*(3), 313-319. https://doi.org/10.1017/S0317167100010209

Pavlov, I. P. (1927). *Conditioned reflexes: An investigation of the physiological activity of the cerebral cortex*. Oxford University Press. https://archive.org/details/conditioned-reflexes-an-investigation-of-the-physiological-activity-of-the-cerebral-cortex

Pechtel, P., Dutra, S. J., Goetz, E. L., & Pizzagalli, D. A. (2013). Blunted reward responsiveness in remitted depression. *Journal of Psychiatric Research*, *47*(12), 1864–1869. https://doi.org/10.1016/j.jpsychires.2013.08.011

Peterburs, J., Kobza, S., & Bellebaum, C. (2016). Feedback delay gradually affects amplitude and valence specificity of the feedback-related negativity (FRN). *Psychophysiology*, *53*(2), 209–215. https://doi.org/10.1111/psyp.12560

Pierce, R. C., & Kumaresan, V. (2006). The mesolimbic dopamine system: the final common pathway for the reinforcing effect of drugs of abuse? *Neuroscience & biobehavioral reviews*, *30*(2), 215-238. https://doi.org/10.1016/j.neubiorev.2005.04.016

Pizzagalli, D. A., Holmes, A. J., Dillon, D. G., Goetz, E. L., Birk, J. L., Bogdan, R., Dougherty, D. D., Iosifescu, D. V., Rauch, S. L., & Fava, M. (2009). Reduced

caudate and nucleus accumbens response to rewards in unmedicated individuals with major depressive disorder. *The American Journal of Psychiatry*, *166*(6), 702–710. https://doi.org/10.1176/appi.ajp.2008.08081201

Pizzagalli, D. A., Iosifescu, D., Hallett, L. A., Ratner, K. G., & Fava, M. (2008). Reduced hedonic capacity in major depressive disorder: Evidence from a probabilistic reward task. *Journal of Psychiatric Research*, *43*(1), 76–87. https://doi.org/10.1016/j.jpsychires.2008.03.001

Pizzagalli, D. A., Jahn, A. L., & O'Shea, J. P. (2005). Toward an objective characterization of an anhedonic phenotype: A signal-detection approach. *Biological Psychiatry*, *57*(4), 319–327. https://doi.org/10.1016/j.biopsych.2004.11.026

Pleger, B., Blankenburg, F., Ruff, C. C., Driver, J., & Dolan, R. J. (2008). Reward facilitates tactile judgments and modulates hemodynamic responses in human primary somatosensory cortex. *Journal of Neuroscience*, *28*(33), 8161-8168. https://doi.org/10.1523/JNEUROSCI.1093-08.2008

Pleger, B., Ruff, C. C., Blankenburg, F., Klöppel, S., Driver, J., & Dolan, R. J. (2009). Influence of dopaminergically mediated reward on somatosensory decision-making. *PLoS biology*, *7*(7), e1000164. https://doi.org/10.1371/journal.pbio.1000164

Poldrack, R. A., Clark, J., Paré-Blagoev, E. J., Shohamy, D., Creso Moyano, J., Myers, C., & Gluck, M. A. (2001). Interactive memory systems in the human brain. *Nature*, *414*(6863), 546-550. https://doi.org/10.1038/35107080

Proudfit, G. H. (2015). The reward positivity: From basic research on reward to a biomarker for depression. *Psychophysiology*, *52*(4), 449-459. https://doi.org/10.1111/psyp.12370

Puce, A., Allison, T., Gore, J. C., & McCarthy, G. (1995). Face-sensitive regions in human extrastriate cortex studied by functional MRI. *Journal of neurophysiology*, *74*(3), 1192-1199. https://doi.org/10.1152/jn.1995.74.3.1192

Qin, S., Piekema, C., Petersson, K. M., Han, B., Luo, J., & Fernández, G. (2007). Probing the transformation of discontinuous associations into episodic memory: An event-related fMRI study. *NeuroImage*, *38*(1), 212–222. https://doi.org/10.1016/j.neuroimage.2007.07.020

Rademacher, L., Salama, A., Gründer, G., & Spreckelmeyer, K. N. (2014). Differential patterns of nucleus accumbens activation during anticipation of monetary and social reward in young and older adults. *Social Cognitive and Affective Neuroscience*, *9*(6), 825-831. https://doi.org/10.1093/scan/nst047

Raposa, E., Hammen, C., Brennan, P., & Najman, J. (2014). The long-term effects of maternal depression: Early childhood physical health as a pathway to offspring depression. *The Journal of Adolescent Health: Official Publication of the Society for Adolescent Medicine*, *54*(1), 88–93. https://doi.org/10.1016/j.jadohealth.2013.07.038

Raskind, M. A. (1998). The clinical interface of depression and dementia. *Journal of Clinical Psychiatry*, *59*(10), 9-12.

Raslau, F. D., Mark, I. T., Klein, A. P., Ulmer, J. L., Mathews, V., & Mark, L. P. (2015). Memory part 2: the role of the medial temporal lobe. *American Journal of Neuroradiology*, *36*(5), 846-849. https://doi.org/10.3174/ajnr.A4169

Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and non-reinforcement. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 64–99). Appleton-Century-Crofts.

Reynolds, J. N., Hyland, B. I., & Wickens, J. R. (2001). A cellular mechanism of reward-related learning. *Nature*, *413*(6851), 67-70. https://doi.org/10.1038/35092560

Ribot, T. (1896*). La psychologie des sentiments.* Paris: Félix Alcan. https://gallica.bnf.fr/ark:/12148/bpt6k654204.texteImage

Rizvi, S. J., Pizzagalli, D. A., Sproule, B. A., & Kennedy, S. H. (2016). Assessing anhedonia in depression: Potentials and pitfalls. *Neuroscience & Biobehavioral Reviews*, *65*, 21-35. https://doi.org/10.1016/j.neubiorev.2016.03.004

Salas, R., Baldwin, P., De Biasi, M., & Montague, R. (2010). BOLD responses to negative reward prediction errors in human habenula. *Frontiers in human neuroscience*, *4*, 1596. https://doi.org/10.3389/fnhum.2010.00036

Schiffer, A. M., Muller, T., Yeung, N., & Waszak, F. (2014). Reward activates stimulus-specific and task-dependent representations in visual association cortices. *Journal of Neuroscience*, *34*(47), 15610-15620. https://doi.org/10.1523/JNEUROSCI.1640-14.2014

Schott, B. H., Minuzzi, L., Krebs, R. M., Elmenhorst, D., Lang, M., Winz, O. H., ... & Bauer, A. (2008). Mesolimbic functional magnetic resonance imaging activations during reward anticipation correlate with reward-related ventral striatal dopamine release. *Journal of Neuroscience*, *28*(52), 14311-14319. https://doi.org/10.1523/JNEUROSCI.2058-08.2008

Schott, B. H., Sellner, D. B., Lauer, C. J., Habib, R., Frey, J. U., Guderian, S., ... & Düzel, E. (2004). Activation of midbrain structures by associative novelty and the formation of explicit memory in humans. *Learning & memory*, *11*(4), 383-387. https://doi.org/10.1101/lm.75004

Schultz, W. (2000). Multiple reward signals in the brain. *Nature reviews neuroscience*,

1(3), 199-207. https://doi.org/10.1038/35044563

Schultz, W. (2002). Getting formal with dopamine and reward. *Neuron*, *36*(2), 241-263. https://doi.org/10.1016/S0896-6273(02)00967-4

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, *275*(5306), 1593–1599. https://doi.org/10.1126/science.275.5306.1593

Schultz, W., & Dickinson, A. (2000). Neuronal coding of prediction errors. *Annual review of neuroscience*, *23*(1), 473-500. https://doi.org/10.1146/annurev.neuro.23.1.473

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of neurology, neurosurgery, and psychiatry*, *20*(1), 11-21. https://doi.org/10.1136/jnnp.20.1.11

Seligman, M. E. (1974). Depression and learned helplessness. In R. J. Friedman & M. M. Katz (Eds.), *The psychology of depression: Contemporary theory and research*. John Wiley & Sons.

Shah, P. J., Ebmeier, K. P., Glabus, M. F., & Goodwin, G. M. (1998). Cortical grey matter reductions associated with treatment-resistant chronic unipolar depression: controlled magnetic resonance imaging study. *The British journal of psychiatry*, *172*(6), 527-532. https://doi.org/10.1192/bjp.172.6.527

Shear, M. K., Simon, N., Wall, M., Zisook, S., Neimeyer, R., Duan, N., ... & Keshaviah, A. (2011). Complicated grief and related bereavement issues for DSM-5. *Depression and anxiety*, *28*(2), 103-117. https://doi.org/10.1002/da.20780

Shohamy, D. (2011). Learning and motivation in the human striatum. *Current opinion in neurobiology*, *21*(3), 408-414. https://doi.org/10.1016/j.conb.2011.05.009

Shohamy, D., & Adcock, R. A. (2010). Dopamine and adaptive memory. *Trends in*

Cognitive Sciences, *14*(10), 464–472. https://doi.org/10.1016/j.tics.2010.08.002

Shohamy, D., Myers, C. E., Grossman, S., Sage, J., Gluck, M. A., & Poldrack, R. A. (2004). Cortico-striatal contributions to feedback-based learning: Converging data from neuroimaging and neuropsychology. *Brain*, *127*(4), 851-859. https://doi.org/10.1093/brain/awh100

Sigurdardottir, H. M., Ólafsdóttir, I. M., & Devillez, H. (2021). Words as visual objects: Neural and behavioral evidence for high-level visual impairments in dyslexia. *Brain Sciences*, *11*(11), 1427. https://doi.org/10.3390/brainsci11111427

Silver, D., Singh, S., Precup, D., & Sutton, R. S. (2021). Reward is enough. *Artificial Intelligence*, *299*, 103535. https://doi.org/10.1016/j.artint.2021.103535

Singer, A. C., & Frank, L. M. (2009). Rewarded outcomes enhance reactivation of experience in the hippocampus. *Neuron*, *64*(6), 910-921. https://doi.org/10.1016/j.neuron.2009.11.016

Smith, J. C., & Roll, D. L. (1967). Trace conditioning with X-rays as an aversive stimulus. *Psychonomic Science*, *9*, 11-12. https://doi.org/10.3758/BF03330734

Squire, L. R., Shimamura, A. P., & Amaral, D. G. (1989). Memory and the hippocampus. In *Neural models of plasticity* (pp. 208-239). Academic Press. https://doi.org/10.1016/B978-0-12-148955-7.50016-3

Staresina, B. P., & Davachi, L. (2009). Mind the gap: binding experiences across space and time in the human hippocampus. *Neuron*, *63*(2), 267-276. https://doi.org/10.1016/j.neuron.2009.06.024

Stolyarova, A. (2018). Solving the credit assignment problem with the prefrontal cortex. *Frontiers in neuroscience*, *12*, 182. https://doi.org/10.3389/fnins.2018.00182

Sugar, J., & Moser, M.B. (2019). Episodic memory: Neuronal codes for what, where, and when. *Hippocampus*, *29*(12), 1190–1205.

https://doi.org/10.1002/hipo.23132

Sutton, R. S., & Barto, A. G. (2018). *Reinforcement learning: An introduction* (2nd ed.). MIT Press.

Sveinbjornsdottir, S. (2016). The clinical symptoms of Parkinson's disease. *Journal of neurochemistry*, *139*, 318-324. https://doi.org/10.1111/jnc.13691

Takács, Á., Kóbor, A., Janacsek, K., Honbolygó, F., Csépe, V., & Németh, D. (2015). High trait anxiety is associated with attenuated feedback-related negativity in risky decision making. *Neuroscience letters*, *600*, 188-192. https://doi.org/10.1016/j.neulet.2015.06.022

Takamura, M., Okamoto, Y., Okada, G., Toki, S., Yamamoto, T., Ichikawa, N., Mori, A., Minagawa, H., Takaishi, Y., Fujii, Y., Kaichi, Y., Akiyama, Y., Awai, K., & Yamawaki, S. (2017). Patients with major depressive disorder exhibit reduced reward size coding in the striatum. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *79*(Pt B), 317–323. https://doi.org/10.1016/j.pnpbp.2017.07.006

Tang, D. W., Fellows, L. K., Small, D. M., & Dagher, A. (2012). Food and drug cues activate similar brain regions: a meta-analysis of functional MRI studies. *Physiology & behavior*, *106*(3), 317-324. https://doi.org/10.1016/j.physbeh.2012.03.009

The MathWorks, Inc. (2021). MATLAB R2021a [Computer software]. Natick, MA: The MathWorks, Inc.

The R Foundation. (2021). R: A language and environment for statistical computing (Version 4.2.2) [Computer software]. Vienna, Austria: R Foundation for Statistical Computing. https://www.R-project.org/

Thiebaut de Schotten, M., Dell'Acqua, F., Forkel, S., Simmons, A., Vergani, F.,

Murphy, D. G., & Catani, M. (2011). A lateralized brain network for visuo-spatial attention. *Nature Precedings*. https://doi.org/10.1038/npre.2011.5549.1

Thompson, S. M. (2023). Plasticity of synapses and reward circuit function in the genesis and treatment of depression. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *48*(1), 90–103. https://doi.org/10.1038/s41386-022-01422-1

Thorndike, E. L. (1927). The law of effect. *The American journal of psychology*, *39*(1/4), 212-222. https://doi.org/10.2307/1415413

Thorndike, E. L. (1933). A proof of the law of effect. *Science*, *77*(1989), 173-175. https://doi.org/10.1126/science.77.1989.173.b

Tobias, M. R., & Ito, T. A. (2021). Anxiety increases sensitivity to errors and negative feedback over time. *Biological psychology*, *162*, 108092. https://doi.org/10.1016/j.biopsycho.2021.108092

Tobler, P. N., O'Doherty, J. P., Dolan, R. J., & Schultz, W. (2006). Human neural learning depends on reward prediction errors in the blocking paradigm. *Journal of Neurophysiology*, *95*(1), 301-310. https://doi.org/10.1152/jn.00762.2005

Tsetsenis, T., Badyna, J. K., Wilson, J. A., Zhang, X., Krizman, E. N., Subramaniyan, M., ... & Dani, J. A. (2021). Midbrain dopaminergic innervation of the hippocampus is sufficient to modulate formation of aversive memories. *Proceedings of the National Academy of Sciences*, *118*(40), e2111069118. https://doi.org/10.1073/pnas.2111069118

Tulving, E., & Markowitsch, H. J. (1998). Episodic and declarative memory: role of the hippocampus. *Hippocampus*, *8*(3), 198-204. https://doi.org/10.1002/(SICI)1098-1063(1998)8:3%3C198::AID-HIPO2%3E3.0.CO;2-G

Tversky, A., & Kahneman, D. (1992). Advances in prospect theory: Cumulative

representation of uncertainty. *Journal of Risk and uncertainty*, *5*, 297-323. https://doi.org/10.1007/BF00122574

Ullsperger, M. (2024). Beyond peaks and troughs: Multiplexed performance monitoring signals in the EEG. *Psychophysiology*, *61*(7), e14553. https://doi.org/10.1111/psyp.14553

Vassiliadis, P., Beanato, E., Popa, T., Windel, F., Morishita, T., Neufeld, E., ... & Hummel, F. C. (2024). Non-invasive stimulation of the human striatum disrupts reinforcement learning of motor skills. *Nature Human Behaviour*, *8*(8), 1581-1598. https://doi.org/10.1038/s41562-024-01901-z

von Glischinski, M., von Brachel, R., & Hirschfeld, G. (2019). How depressed is "depressed"? A systematic review and diagnostic meta-analysis of optimal cut points for the Beck Depression Inventory revised (BDI-II). *Quality of Life Research*, *28*, 1111-1118. https://doi.org/10.1007/s11136-018-2050-x

Vos, T., Flaxman, A. D., Naghavi, M., Lozano, R., Michaud, C., Ezzati, M., ... & Harrison, J. E. (2012). Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *The lancet*, *380*(9859), 2163-2196. https://doi.org/10.1016/S0140-6736(12)61729-2

Wang, P. S., Simon, G., & Kessler, R. C. (2003). The economic burden of depression and the cost-effectiveness of treatment. *International journal of methods in psychiatric research*, *12*(1), 22-33. https://doi.org/10.1002/mpr.139

Weber, C., & Bellebaum, C. (2024). Prediction-error-dependent processing of immediate and delayed positive feedback. *Scientific Reports*, *14*(1), 9674. https://doi.org/10.1038/s41598-024-60328-8

Weinberg, A., Liu, H., Hajcak, G., & Shankman, S. A. (2015). Blunted neural response

to rewards as a vulnerability factor for depression: Results from a family study. *Journal of Abnormal Psychology*, *124*(4), 878–889. https://doi.org/10.1037/abn0000081

Weinberg, A., Luhmann, C. C., Bress, J. N., & Hajcak, G. (2012). Better late than never? The effect of feedback delay on ERP indices of reward processing. *Cognitive, Affective, & Behavioral Neuroscience*, *12*(4), 671–677. https://doi.org/10.3758/s13415-012-0104-z

Weiner, K. S., & Zilles, K. (2016). The anatomical and functional specialization of the fusiform gyrus. *Neuropsychologia*, *83*, 48-62. https://doi.org/10.1016/j.neuropsychologia.2015.06.033

Weismüller, B., & Bellebaum, C. (2016). Expectancy affects the feedback-related negativity (FRN) for delayed feedback in probabilistic learning. *Psychophysiology*, *53*(11), 1739–1750. https://doi.org/10.1111/psyp.12738

Weismueller, B., Ghio, M., Logmin, K., Hartmann, C., Schnitzler, A., Pollok, B., ... & Bellebaum, C. (2018). Effects of feedback delay on learning from positive and negative feedback in patients with Parkinson's disease off medication. *Neuropsychologia*, *117*, 46-54. https://doi.org/10.1016/j.neuropsychologia.2018.05.010

Wessel, M. J., Beanato, E., Popa, T., Windel, F., Vassiliadis, P., Menoud, P., ... & Hummel, F. C. (2023). Noninvasive theta-burst stimulation of the human striatum enhances striatal activity and motor skill learning. *Nature neuroscience*, *26*(11), 2005-2016. https://doi.org/10.1038/s41593-023-01457-7

Wheeler, M. E., Petersen, S. E., & Buckner, R. L. (2000). Memory's echo: Vivid remembering reactivates sensory-specific cortex. *Proceedings of the National Academy of Sciences of the United States of America*, *97*(20), 11125–11129.

https://doi.org/10.1073/pnas.97.20.11125

White, N. M., & McDonald, R. J. (2002). Multiple parallel memory systems in the brain of the rat. *Neurobiology of learning and memory*, *77*(2), 125-184. https://doi.org/10.1006/nlme.2001.4008

Wichers, M., Kasanova, Z., Bakker, J., Thiery, E., Derom, C., Jacobs, N., & van Os, J. (2015). From affective experience to motivated action: Tracking reward-seeking and punishment-avoidant behaviour in real-life. *PloS one*, *10*(6), e0129722. https://doi.org/10.1371/journal.pone.0129722

World Health Organization. (2019). *International classification of diseases 11th revision (ICD-11)*. World Health Organization. https://icd.who.int/

Yagishita, S., Hayashi-Takagi, A., Ellis-Davies, G. C., Urakubo, H., Ishii, S., & Kasai, H. (2014). A critical time window for dopamine actions on the structural plasticity of dendritic spines. *Science*, *345*(6204), 1616-1620. https://doi.org/10.1126/science.1255514

Yeung, N., Holroyd, C. B., & Cohen, J. D. (2005). ERP correlates of feedback and reward processing in the presence and absence of response choice. *Cerebral cortex*, *15*(5), 535-544. https://doi.org/10.1093/cercor/bhh153

Yovel, G. (2016). Neural and cognitive face-selective markers: An integrative review. *Neuropsychologia*, *83*, 5-13. https://doi.org/10.1016/j.neuropsychologia.2015.09.026

Zaghloul, K. A., Blanco, J. A., Weidemann, C. T., McGill, K., Jaggi, J. L., Baltuch, G. H., & Kahana, M. J. (2009). Human substantia nigra neurons encode unexpected financial rewards. *Science*, *323*(5920), 1496-1499. https://doi.org/10.1126/science.1167342

Zhang, L., Lengersdorff, L., Mikus, N., Gläscher, J., & Lamm, C. (2020). Using

reinforcement learning models in social neuroscience: frameworks, pitfalls and suggestions of best practices. *Social Cognitive and Affective Neuroscience*, *15*(6), 695-707. https://doi.org/10.1093/scan/nsaa089

Zimmerman, M., Chelminski, I., & McDermut, W. (2002). Major depressive disorder and axis I diagnostic comorbidity. *Journal of Clinical Psychiatry*, *63*(3), 187-193.

Zisook, S., & Shear, K. (2009). Grief and bereavement: what psychiatrists need to know. *World psychiatry*, *8*(2), 67-74. https://doi.org/10.1002/j.2051-5545.2009.tb00217.x

Zobel, A., & Maier, W. (2004). Endophänotypen—ein neues Konzept zur biologischen Charakterisierung psychischer Störungen. *Der Nervenarzt*, *75*, 205-214. https://doi.org/10.1007/s00115-003-1642-y

5 APPENDIX A: AFFIDAVIT

Eidesstattliche Erklärung gemäß § 5 der Promotionsordnung vom 15.06.2018 der Mathematisch-Naturwissenschaftlichen Fakultät der Heinrich-Heine-Universität Düsseldorf:

Ich versichere an Eides Statt, dass die Dissertation von mir selbständig und ohne unzulässige fremde Hilfe unter Beachtung der „Grundsätze zur Sicherung guter wissenschaftlicher Praxis an der Heinrich-Heine-Universität Düsseldorf“ erstellt worden ist. Die Dissertation wurde in der vorliegenden oder ähnlichen Form noch bei keiner anderen Institution eingereicht. Ich habe bisher keine erfolglosen Promotionsversuche unternommen.

Düsseldorf, den ____01.07.2025____ _____

Datum Madita Röhlinger

6 APPENDIX B: DECLARATION OF USED AI TECHNOLOGIES

During the preparation of this work, I used generative AI and AI-assisted technologies like OpenAI's GPT and DeepL Translator to translate and edit text and to improve readability. After using these tools, I reviewed and edited the content as needed and I take full responsibility for the content of this dissertation.

Düsseldorf, den ____01.07.2025____ _____

Datum Madita Röhlinger

7 APPENDIX C: ORIGINAL RESEARCH ARTICLES

Original Article of Study 1

Röhlinger, M., Albrecht, C., Ghio, M., & Bellebaum, C. (2025). Neural Processing of Immediate versus Delayed Feedback in Action–Feedback and Stimulus–Feedback Associations [Uncorrected proof; advance online publication]. *Journal of Cognitive Neuroscience*. https://doi.org/10.1162/jocn.a.49

Dr. Christine Albrecht and I were the main authors of this article. I was involved in the development of the paradigm. I contributed to the conduction and the supervision of the data acquisition. I assisted with the preprocessing and interpretation of the data.

Original Article of Study 2

Röhlinger, M., Albrecht, C., & Bellebaum, C. (2025). The Role of the N170 in Linking Stimuli to Feedback—Effects of Stimulus Modality and Feedback Delay. *Psychophysiology*, *62*(4), e70050. https://doi.org/10.1111/psyp.70050

I was the main author of this article. I contributed to the conceptualization and methodology and the extension of the paradigm, including the creation of the study material. I planned, conducted and supervised the data acquisition. I preprocessed, analyzed and interpreted the data.

Original Article of Study 3

Röhlinger, M., & Bellebaum, C. (2025). Links between Altered Feedback Learning and Symptoms of Depression: Insights from an EEG Study on FRN and N170. *Manuscript submitted for publication.*

I was the main author of this article. I contributed to the conceptualization and methodology and modified the paradigm. I conducted and supervised the data acquisition. I preprocessed, analyzed and interpreted the data.

Neural Processing of Immediate versus Delayed Feedback in Action–Feedback and Stimulus–Feedback Associations

Madita Röhlinger*, Christine Albrecht*, Marta Ghio, and Christian Bellebaum

Abstract

■ The feedback-related negativity (FRN) or reward positivity (RewP), reflecting striatal reward system activity, is reduced with delayed feedback, whereas the N170, associated with medial temporal lobe (MTL) activity, is increased. The type of the learned association could also affect which system is involved: We expected the striatal reward system to be adept at learning action–feedback associations and the MTL to be primarily involved in learning stimulus–feedback associations, which should be reflected in stronger prediction error (PE) representations in the FRN/RewP and N170, respectively. The relative contributions of the striatum and MTL to feedback learning and processing, however, also seem to be determined by the feedback's timing (immediate vs. delayed). We recorded EEG while 40 participants learned in an action–feedback condition or a stimulus–feedback condition with immediate and delayed feedback. Replicating previous studies, the FRN/RewP was most negative for unexpected negative feedback and most positive for unexpected positive feedback. Surprisingly, this PE × Feedback Valence interaction was more pronounced for the stimulus–feedback condition than the action–feedback condition. Interestingly, we found a PE × Feedback Valence interaction also in the N170, but with most negative amplitudes for unexpected positive and expected negative feedback. This interaction appeared across feedback timings for the stimulus–feedback condition, but only for immediate feedback for the action–feedback condition. The results suggest that striatal and MTL systems cooperate across feedback timings for stimulus–feedback associations, but not for action–feedback associations learned with delayed feedback. ■

INTRODUCTION

We can learn how to use a coffee machine either by reading the manual or by trial-and-error learning. Reading the manual would involve declarative learning, associated with hippocampal activity within the medial temporal lobe (MTL), resulting in flexible, factual knowledge (Myers et al., 2003; Knowlton, Mangels, & Squire, 1996; Scoville & Milner, 1957; for a review see Eichenbaum, Otto, & Cohen, 1992). Learning by trial and error, or, in other words, feedback learning, has been considered to be a type of procedural learning resulting in habit-like, automatically retrieved and inflexible knowledge associated with striatal activity (Knowlton & Patterson, 2016; Knowlton et al., 1996).

The striatum receives input from midbrain dopaminergic neurons in the substantia nigra and ventral tegmental area (Chuhma, Oh, & Rayport, 2023; Oldehinkel et al., 2022; Steiner & Tseng, 2016; Zhang et al., 2015). These neurons encode a reward prediction error (PE), indicating whether an outcome is better or worse than expected (Zaghloul et al., 2009; Schultz, 2000; Schultz & Dickinson, 2000; Schultz, Dayan, & Montague, 1997), thereby building a core component of the cortical–striatal circuit described as the reward system (for a review, see Haber & Knutson, 2010). Although the reward system has been shown to underlie feedback learning (Vassiliadis et al., 2024; Cooper, Dunne, Furey, & O'Doherty, 2012; for a review, see Daniel & Pollmann, 2014), this type of learning can also be supported by the MTL, or, more specifically, the hippocampus, and thus presumably be declarative. Plachti and colleagues (2019) linked the hippocampus to associative memory, learning, and reinforcement, underlining its potential role for feedback processing. Along similar lines, Dickerson, Li, and Delgado (2011) found correlates of the PE in the striatum as well as the hippocampus in a feedback learning task.

One factor determining the relative contributions of the striatum and MTL to feedback learning and processing seems to be the timing of the feedback: Learning with immediate feedback has been shown to be dependent on the striatum, whereas learning from feedback with a delay of only a few seconds was dependent on the hippocampus (Foerde, Race, Verfaellie, & Shohamy, 2013; Foerde & Shohamy, 2011). In line with this, dopamine-driven synaptic connections in the striatum, that is, reinforcement plasticity, was found to be limited to a narrow time window of up to 2 sec (Yagishita et al., 2014), suggesting that the striatum is involved in linking feedback to directly preceding events (Jocham et al., 2016). The hippocampus, on the other hand, can bridge representational gaps in our experience (Staresina & Davachi, 2009), which

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is a prerequisite for linking feedback to preceding events that are more distant in time.

Several studies investigating feedback processing by means of electroencephalography (EEG) corroborate the idea that feedback timing is important. They found that the amplitudes of two ERP components, the feedback-related negativity (FRN) and the N170, vary with feedback delay (Höltje & Mecklinger, 2020; Kim & Arbel, 2019; Arbel, Hong, Baker, & Holroyd, 2017; Peterburs, Kobza, & Bellebaum, 2016). The FRN peaks between 230 and 330 msec after feedback onset (Miltner, Braun, & Coles, 1997) and is more negative for negative than for positive feedback. However, it is likely that this difference is due to a relative positivity after rewards, which has been labeled reward positivity (RewP; Proudfit, 2015; Lukie, Montazer-Hojat, & Holroyd, 2014; Holroyd, Krigolson, & Lee, 2011). The FRN/RewP has been associated with the influence of the mesencephalic dopamine system on the ACC (Foti, Weinberg, Dien, & Hajcak, 2011; Bellebaum & Daum, 2008; Holroyd, Larsen, & Cohen, 2004; Holroyd & Coles, 2002), and striatum (Becker, Nitsch, Miltner, & Straube, 2014; Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Foti et al., 2011), and its amplitude has been shown to reflect the PE (Weber & Bellebaum, 2024; Burnside, Fischer, & Ullsperger, 2019; Sambrook & Goslin, 2015; Fischer & Ullsperger, 2013). In line with the reduced involvement of the striatum for delayed feedback (Foerde et al., 2013; Foerde & Shohamy, 2011), the FRN/RewP amplitude difference between positive and negative feedback is diminished when feedback is delayed (Höltje & Mecklinger, 2020; Arbel et al., 2017; Peterburs et al., 2016; Weismüller & Bellebaum, 2016; Weinberg, Luhmann, Bress, & Hajcak, 2012).

In several more recent studies, the N170, a component occurring over occipito-temporal cortex 100–200 msec after stimulus onset, was found to be more pronounced following delayed compared with immediate feedback (Höltje & Mecklinger, 2020; Kim & Arbel, 2019; Arbel et al., 2017; but see Albrecht, van de Vijver, & Bellebaum, 2023, for the opposite pattern), suggesting that it reflects MTL activity. However, the N170 has originally been linked to visual processing of faces (Yovel, 2016; Rossion, 2014; Rossion & Jacques, 2012; Bentin, Allison, Puce, Perez, & McCarthy, 1996), and also other stimulus categories (Kloth, Itier, & Schweinberger, 2013), with a source in the fusiform gyrus (Gao, Conte, Richards, Xie, & Hanayik, 2019; Deffke et al., 2007). Therefore, the N170 following delayed feedback may indicate the feedback-locked activation of higher-order visual areas. This may be interpreted as a reactivation of those areas that processed the stimulus associated with the feedback (Schiffer, Muller, Yeung, & Waszak, 2014; Pleger et al., 2009; Pleger, Blankenburg, Ruff, Driver, & Dolan, 2008), a process possibly mediated by the hippocampus. Thus, the N170 might not directly reflect hippocampal activity, but rather a mechanism to link stimuli to feedback via reactivation of higher visual areas, especially when feedback is delayed.

An important aspect possibly affecting the involvement of the respective memory systems has so far been neglected: We propose that the type of association that is learned via feedback plays a significant part in feedback processing. When a rewarding or punishing stimulus is experienced, the problem of credit assignment arises, which deals with the question how individuals know which event the received feedback refers to and how this is implemented in the brain (Fu & Anderson, 2008; for a review, see Stolyarova, 2018). Feedback might be linked to previous actions (e.g., pushing a respective button leads to a cup of coffee) or to a previous stimulus (e.g., the display lighting up means that the machine is producing a cup of coffee). On the one hand, there is evidence suggesting that action–feedback associations are likely to be associated with striatal activity: The striatum is connected to habit learning based on feedback (Shohamy et al., 2004; Poldrack et al., 2001; Knowlton et al., 1996). As part of the basal ganglia's direct and indirect pathways, the striatum is connected to action selection and action inhibition, respectively (Aubert, Ghorayeb, Normand, & Bloch, 2000; Hernández-López et al., 2000; Calabresi, Picconi, Tozzi, Ghiglieri, & Di Filippo, 2014; Hernández-López, Bargas, Surmeier, Reyes, & Galarraga, 1997; Gerfen, 1992). Along similar lines, Hiebert and colleagues (2014) found that during stimulus–response learning, activity in the dorsal striatum correlated with response selection, whereas ventral striatum activation correlated with feedback. Shohamy (2011) suggested that the striatum is part of a distributed network that, aiming to optimize behavior, learns to select actions based on their predicted values. Valence-sensitive activity after feedback, descriptively similar to an FRN/RewP response, was found over motor cortex in an ERP study (Cohen & Ranganath, 2007), which suggests an activation of the previous action for striatum-based feedback learning. In healthy participants, reward feedback can accelerate motor learning (Nikooyan & Ahmed, 2015), with noninvasive stimulation of the human striatum either improving motor skill learning (Wessel et al., 2023) or disrupting reinforcement learning of motor skills (Vassiliadis et al., 2024). In a study by Haruno and Kawato (2006), neuronal correlates of stimulus–action–reward associations were located in parts of the striatum. Finally, Jocham and colleagues (2016) showed that cortico-striatal motor circuits are involved in linking choices to outcomes based on temporal proximity. This suggests that the neural mechanisms underlying the learning of action–feedback associations may also interact with feedback timing. We thus hypothesize that the striatum links motor activity to feedback, especially if the two events are temporally close. Given that the FRN/RewP has been linked to striatal processing (Becker et al., 2014; Carlson et al., 2011; Foti et al., 2011), the ERP signal in the respective time window may be particularly pronounced when participants learn associations between actions and immediate feedback.

On the other hand, stimulus–feedback associations might rather depend on the hippocampus: The hippocampus' role in episodic memory is to bind different types of sensory information together to form a memory episode (Sugar & Moser, 2019; Squire, Shimamura, & Amaral, 1989). From a study with rats, Singer and Frank (2009) concluded that hippocampal reactivation at reward presentation could be a mechanism to bind rewarding outcomes to the prior experiences. Several studies suggest that the reactivation of sensory cortices may also play a role for feedback processing in humans. For example, primary somatosensory cortex was reactivated at the point of reward delivery in a somatosensory discrimination task (Pleger et al., 2008, 2009). In a classification task with visual stimuli, Schiffer and colleagues (2014) found that reward activated stimulus-specific representations in visual association cortices, possibly providing a solution for the credit assignment problem. Qin and colleagues (2007) suggested that top–down signals from the MTL may trigger an internal representation of a previous event, thus allowing it to be linked to a present event. We hypothesize that the MTL might mediate the reactivation of sensory areas to particularly link stimuli (rather than actions) to feedback, especially when there is a delay between stimulus and feedback.

To sum up, with the present work, we aimed to investigate the influence of feedback timing and association type on feedback processing as reflected in the FRN/RewP and N170, which can be considered measures of striatal activity and MTL-initiated reactivations of visual brain regions, respectively. We manipulated feedback timing within participants by having each participant complete a feedback learning task involving both immediate and delayed feedback. We furthermore manipulated the association type between participants by having one group learn action–feedback and two other groups learn stimulus–feedback associations (actively vs. passively; see below). We hypothesized that the FRN/RewP, or rather the differentiation between positive and negative feedback in the FRN/RewP (Arbel et al., 2017), would be largest for immediate feedback in the action–feedback condition (AFC). In contrast, we hypothesized that the N170 is largest for delayed feedback when stimulus–feedback associations are learned. Regarding PE effects, we expected the strongest effect on FRN/RewP amplitude for immediate feedback when action–feedback associations are learned. Furthermore, we hypothesized an effect of the PE on N170 amplitude especially for delayed feedback when stimulus–feedback associations are learned.

METHODS

Participants

Our analysis strategy was based on the separate comparison of two (out of three) experimental groups linked to the between-subject factor association type (i.e., the type of association that had to be learned, see below). We aimed to acquire at least 25 participants per group, assuming a 20% exclusion rate, as preregistered on the Open Science Framework (https://doi.org/10.17605/OSF.IO/GVMWP). The reasons for this were twofold: Previous studies have shown that this sample size suffices to find differences in the neural pattern of feedback processing between two groups (Bellebaum & Colosio, 2014; Kobza et al., 2012) and to determine ERP correlates of PE processing (Weber & Bellebaum, 2024; Burnside et al., 2019). Taking potential dropouts into account, overall, 78 healthy young adults (between 18 and 40 years old) took part in the experiment, randomly assigned to one of three experimental groups, between which the factor association type was varied (see below for details). All participants declared no history of neurological or psychiatric disorders, no regular or acute consumption of substances affecting the central nervous system, no knowledge about Hiragana characters (as Hiragana-like characters were used in the experiment, see below), and normal or corrected-to-normal vision and normal hearing. Three participants had to be excluded from the analyses due to technical problems during the data acquisition and two participants due to not paying enough attention in the learning task (see below). Another participant was excluded because the behavioral data contained a lot of button presses that did not align with the task. Furthermore, 10 participants were excluded because of poor EEG data quality. Water damage in the EEG system led to noise in the EEG signal at the beginning of the recruitment process, which was probably the reason for the bad data quality of four out of the 10 excluded participants, who were excluded immediately after initial visual inspection of the raw data. Another 4 out of the 10 were excluded after the artifact rejection had removed more than 20% of their data, indicating bad data quality. Two out of the 10 participants were excluded because the visual inspection revealed considerable alpha activity. This left 62 participants (40 women, 21 men and 1 nonbinary person, mean age = 23.66 years, SD = 3.87) for the analyses. From these remaining participants, 18 (13 women, 5 men) were assigned to the AFC, 22 (14 women, 8 men) to the active stimulus–feedback condition (ASFC) and 22 (13 women, 8 men, 1 nonbinary person) to the passive stimulus–feedback condition (PSFC; see below). The study was approved by the ethics committee of the Faculty of Mathematics and Natural Sciences at Heinrich-Heine-University, Düsseldorf, and complied with the Declaration of Helsinki.

Experimental Task and Conditions

Figure 1 shows the structure of the probabilistic feedback learning tasks. Participants were randomly assigned to one of three between-subject conditions, in which different types of associations could be learned (association

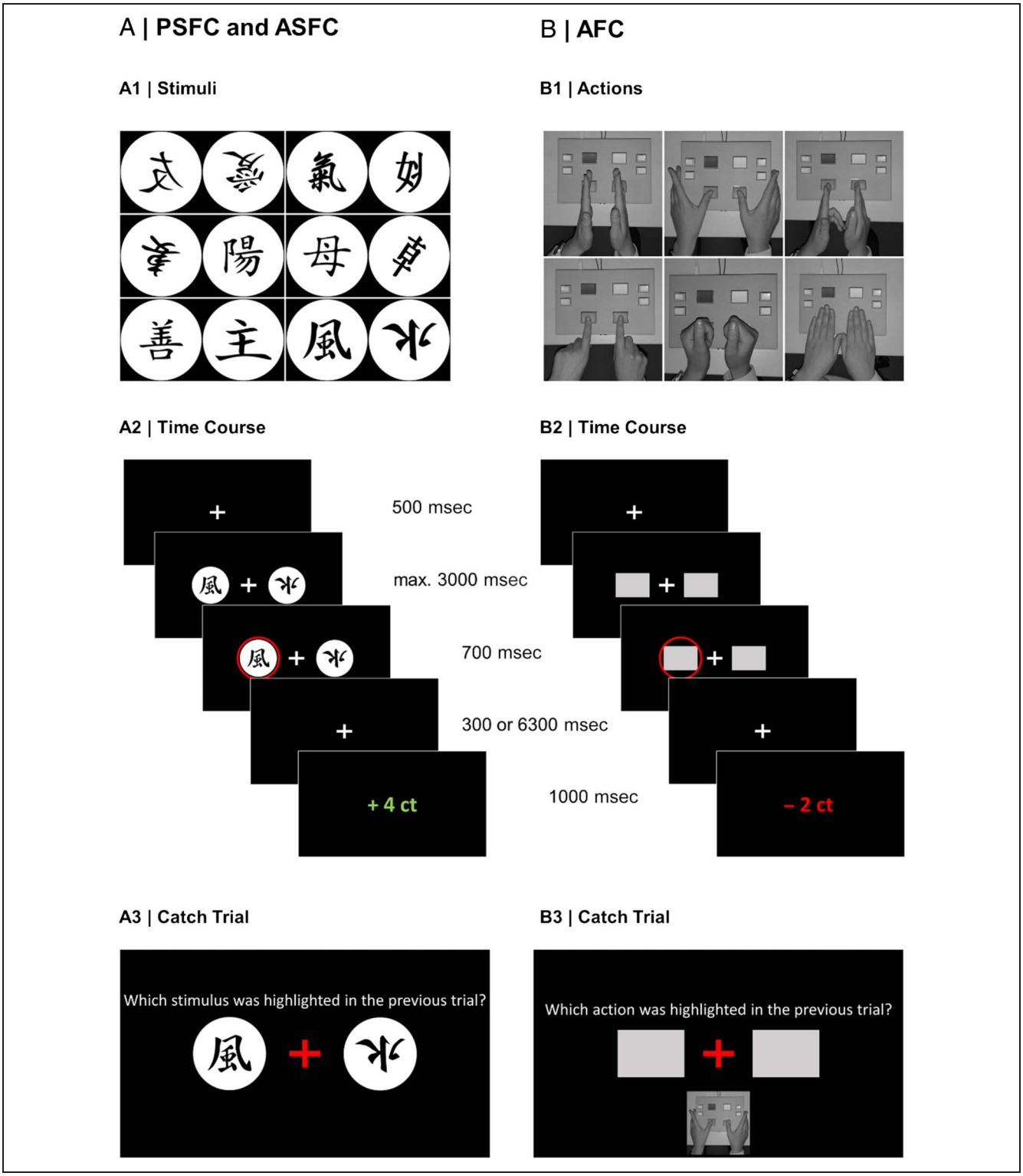


Figure 1. Experimental feedback-learning task. (A) PSFC and ASFC: Task in the PSFC and ASFC. (A1) Stimuli: Stimuli in the PSFC and ASFC. (A2) Time course: Time course of events in a single trial in the PSFC and ASFC. Whereas in the ASFC, participants could choose between the two hiragana characters themselves, in the PSFC, one stimulus was automatically highlighted in each learning trial. (A3) Catch trial: Catch trial in the PSFC and ASFC. (B) AFC: Task in the AFC. (B1) Actions: Actions in the AFC. (B2) Time course: Time course of events in a single trial of the AFC. (B3) Catch trial: Catch trial in the AFC.

type conditions). One group learned action–feedback associations (AFC; see below), whereas the other two groups learned stimulus–feedback associations either actively or passively (ASFC and PSFC; see below). All participants could learn associations between events and positive or negative monetary feedback (feedback valence: +4 ct vs. −2 ct—because subjectively, losses weigh about twice as much as gains; Tversky & Kahneman, 1992). For each of the three between-subject association type conditions, the task comprised three within-subject feedback timing conditions, in which the delay between the event (stimulus or action) and the feedback was varied. In one condition, the feedback was given immediately (immediate feedback; 1 sec after the event), whereas in a second condition, it was given delayed (delayed feedback; 7 sec after the event). To control for effects of the reduced temporal predictability of delayed feedback, a third condition entailed a feedback delay of 7 sec with six regular tones presented during the delay, one per second (Kimura & Kimura, 2016). Specifically, the first tone (700 msec long, 800 Hz) was presented exactly 1000 msec after the choice, and the following tones (all with the same acoustic characteristics as the first one) were presented each 1 sec after the previous tone onset. We will thus refer to the two conditions with delayed feedback as delayed feedback without tone and delayed feedback with tone. The order of the different feedback timing conditions was counterbalanced across participants. In all association type conditions, participants completed six sessions, two per feedback timing condition. Each session consisted of four blocks, and each block contained 20 learning trials, followed by 20 test trials, in which no feedback was presented. Participants were instructed that during the test trials, monetary wins and losses would still be counted as in the learning trials, but not shown on the screen. With every new session, new associations had to be learned (see below), and participants were instructed that learning would start anew.

PSFC

On every learning trial of the PSFC, a pair of visual stimuli was presented on the computer screen, one on the left and one on the right of a fixation cross. We used Hiragana-like characters (see Figure 1A1) that cannot easily be verbalized (see Frank, Seeberger, & O'reilly, 2004). Figure 1A2 shows the sequence of events in one learning trial of the PSFC. Participants saw how one of the stimuli was automatically highlighted by a red circle for 700 msec (see O'Doherty et al., 2004). Then, a fixation cross was presented for 300 msec (in the immediate feedback condition) or 6300 msec (in the delayed feedback conditions), and, subsequently, positive or negative monetary feedback followed for 1000 msec. In each of the six different sessions, new Hiragana-like characters were introduced, and new stimulus–feedback associations had to be learned. Thus, six different pairs of stimuli (randomly paired for each participant) were used, with the screen side on which each stimulus could appear being counterbalanced. Unbeknown to the participants, in each session, one stimulus was associated with reward in 65% of the trials and with punishment in 35%, whereas probabilities were reversed for the other stimulus. The participants' task was to learn associations between stimuli and feedback. To make sure that participants paid attention to the stimuli on the screen and to the highlighting, three catch trials (see Figure 1A3) were included randomly along the 20 learning trials of each block in each session. In the catch trials, participants were asked which of the two stimuli was highlighted in the last trial. For the analyses, we excluded all participants ($n = 2$) that seemed to pay not enough attention, as suggested by their fulfillment of the following two criteria: First, they answered wrong in more than 20% of catch trials. Second, they chose the stimulus associated with the higher reward probability in less than 55% of the test trials.

In test trials, participants actively chose between the two stimuli by pressing the corresponding (left vs. right) button on a response box without receiving feedback. These trials, in which participants were asked to choose the "more rewarding" stimulus in each trial, were included to measure if participants actually learned the stimulus–feedback associations, which could not be measured in the learning trials. However, wins and losses in both learning and test trials contributed to the overall earnings of the participants.

To ensure that the stimulus highlighting in the learning trials of the PSFC reflected a realistic choice pattern, we conducted a behavioral pilot experiment with 25 participants fulfilling the criteria described above in the "Participants" section. The pilot experiment contained the same Hiragana characters, but participants selected one of the two presented stimuli in each learning trial themselves to learn from feedback. The recorded choices of one pilot participant determined which stimulus was highlighted on a particular learning trial for one participant of the PSFC. Because RTs differed between the feedback timing conditions within the pilot experiment, the mean RT of all participants for one trial in the pilot experiment determined when a Hiragana character was highlighted in one trial of the PSFC.

AFC

In every learning trial of the AFC, participants were asked to choose between two specific actions to press either the left or right button of a response box (e.g., pressing the right button with their right thumb vs. the left button with their left thumb). Figure 1B1 shows the six different pairs of actions that were used in the different sessions. After the choice, participants received feedback for their

action, with the delay varying according to the feedback timing condition assigned to the corresponding session, and the participants' task was to learn an association between the chosen action and the feedback. In each of the six different sessions, new actions were introduced and participants had to learn new action–feedback associations. The experimenter checked regularly whether participants performed the actions as requested. Figure 1B2 shows the sequence of events in one learning trial of the AFC. On the screen, two rectangles to the left and right of a fixation cross were shown. After participants performed one of the actions, the rectangle on the side of the chosen action was highlighted for 700 msec indicating that the choice was recorded. Afterward, a fixation cross and then feedback was presented with the same durations as in the PSFC (see above). Participants had maximally 3000 msec for their choice. If they did not respond within this time window, the trial was considered a "miss" and not included in any further analysis, while an instruction to react faster was presented on the screen. Unbeknown to the participants, in each session, the choice of one action led to a reward in 65% of the trials and to a loss in 35%; for the other action, the probabilities were reversed. Participants were instructed to maximize reward. For comparability between the conditions, test trials without feedback and catch trials (see Figure 1B3) were included in an analogous way to the PSFC.

ASFC

The ASFC was identical to the PSFC, with the only difference that stimuli were actively chosen by the participants in the learning trials via button press. The ASFC was added for two reasons. First, pilot testing had shown that learning in the PSFC was difficult. Second, the trial-by-trial choices in the ASFC allowed the application of a reinforcement learning model to the choice data and thus modeling PEs in the same way as for the AFC (see below). Like in the AFC, participants had maximally 3000 msec for their choice. Although this condition thus also entailed actions, the instruction emphasized that feedback was only related to the stimuli, not to the actions. This was secured by counterbalancing the side on which the stimuli appeared. To make the conditions as similar as possible, test trials without feedback and catch trials were also included in the ASFC.

To conclude, in the AFC, the motor response predicted reward, whereas in both the PSFC and ASFC, stimulus identity predicted reward. More in detail, in the AFC, visual stimuli (rectangles to indicate the choice) were nonrelevant for learning, whereas actions were relevant; in the ASFC, actions (choosing left or right) were nonrelevant for learning (because stimuli sides were counterbalanced), whereas stimuli were relevant. The PSFC omitted the nonrelevant actions altogether.

Procedure and Data Acquisition

Upon arrival in the laboratory, participants were informed about the experimental procedure and gave written informed consent to participate in the study. They were then asked to fill in a demographic questionnaire. Afterward, we attached the EEG electrodes (preparation time = about 60 min), and then participants were placed in front of a 27-in., 1920 × 1080 px W-LED monitor (BENQ EW2740L) with a refresh rate of 50 Hz–76 Hz, and the experimental task began. Auditory stimuli were presented via dynamic stereo headphones (Sennheiser HD 201). The software Presentation (Version 22; Neurobehavioral Systems Inc., 2020) controlled the timing of stimulation and the recording of responses. Responses were performed on a Cedrus RB-844 response pad (www.cedrus.com).

The experiment lasted about 75 min. Participants were informed that the amount of money they would earn during the feedback-learning task would be paid out at the end. They received a starting amount of 7 €. However, the sums earned by each participant were rounded up at the end of the experiment, and all received 25 €.

EEG Data

EEG was acquired from 60 scalp electrodes, fixed with an actiCap (BrainProducts) textile softcap and evenly distributed on the scalp based on the extended 10–20 system. Electrodes were attached to the scalp sites AF3, AF4, AF7, AF8, C1, C2, C3, C4, C5, C6, CP1, CP2, CP3, CP4, CP5, CP6, CPz, Cz, F1, F2, F3, F4, F5, F6, F7, F8, FC1, FC2, FC3, FC4, FC5, FC6, FT10, FT7, FT8, FT9, Fz, O1, O2, Oz, P1, P2, P3, P4, P5, P6, P7, P8, PO10, PO3, PO4, PO7, PO8, PO9, POz, Pz, T7, T8, TP7, TP8. In addition, the ground electrode was attached to the AFz position, the online reference to the FCz position. Moreover, we attached two electrodes on the mastoids behind the left and right ear to cover as much of the scalp as possible for the calculation of the average reference (see below). For the monitoring of vertical eye movements and blinks, two further electrodes (VEOG) were attached, one above and one below the left eye, respectively. For data recording, a BrainAmp DC amplifier and the Brain Vision Recorder software (BrainProducts) were used, with a sampling rate of 1000 Hz and an online lowpass filter of 100 Hz. Impedances were kept below 15 kΩ.

Data Analysis

Behavioral Data Analysis

Learning check. Before the main behavioral data analysis, we checked if participants learned on average in all conditions. Participants conducted four test blocks in each of the six learning sessions, one after each learning block. The fourth test block of each learning session should

indicate the maximum learning success of each participant. Therefore, we calculated both the mean accuracy of all participants across all test trials as well as the mean accuracy of participants only in Block 4 in percent, and compared the accuracy rates of each condition to chance level (50%) using single-sample *t* tests. Because we performed six tests, we used a Bonferroni-corrected level of $\alpha = .008$ for statistical interpretation.

Comparison between conditions. For behavioral data statistical analysis, the dependent variable was response accuracy in learning and/or test trials, defined as 1 for correct responses and 0 for incorrect responses. Correct responses were defined as the choice of the action/stimulus associated with the higher reward probability. Trials in which participants failed to answer ($M = 0.25\%$, $SD = 0.61\%$) were excluded from all further (behavioral and EEG) analyses. We applied generalized linear mixed-effects (GLME) model analyses suitable for binomial distributions and single-trial data by means of the lme4 package (Bates, Mächler, Bolker, & Walker, 2015) in R (The R Foundation, 2021). Because learning in the PSFC was impaired (see below), we based our analysis primarily on the comparison between the AFC and ASFC (see the Appendix, Section A2, for analyses including data from the PSFC). The model comprised as fixed-effect predictors association type (action–feedback [−0.5] vs. active stimulus–feedback [0.5]), feedback timing (simple coding contrast matrix with immediate feedback set as baseline that was compared with delayed feedback with tone and delayed without tone), and Block (1 [−0.5], 2 [−0.167], 3 [0.167], 4 [0.5]), together with all two-way and three-way interactions. Random intercepts were allowed per participant. For random slopes per participant, we adhered to best practice (Meteyard & Davies, 2020): All within-subject main and interaction effects were included as random factors per participant, as long as their inclusion did not lead to model over- or underfit, using the *buildmer* function (Version 2.11; Voeten, 2020). This procedure for random effects was used for all mixed model analyses described in this article. To sum up, the model for the GLME analysis of behavioral data was:

$$\begin{aligned} &\textit{Accuracy} \sim 1 + \textit{Block} \times \textit{Association Type} \\ &\quad \times \textit{Feedback Timing} + (1 + \textit{Block} + \textit{Feedback Timing} \\ &\quad + \textit{Block} : \textit{Feedback Timing} \mid \textit{Subject}) \end{aligned}$$

PE modeling. As outlined above, we decided to primarily focus our analysis on the comparison of the AFC and ASFC. Therefore, modeling of the PE in each learning trial of each participant was based on the participants' responses in the learning trials. The Appendix, Section A2, contains additional analyses involving the PSFC as well as a description of the procedure for the PE calculation based on the test trials for all the three conditions (Bellebaum, Jokisch, Gizewski, Forsting, & Daum, 2012).

On the basis of the approach by Weber and Bellebaum (2024), we derived single-trial values of the PE for each participant by fitting a reinforcement learning model to the behavioral data using MATLAB Version R2021a (The MathWorks, Inc., 2021). The starting point was each participants' sequence of choices and the received feedback. The PE $\delta_{c,t}$ was calculated as follows:

$$\delta_{c,t} = r_t - Q_{c,t}$$

where in a given trial t the reward r_t is 1 for positive feedback and 0 for negative feedback, and $Q_{c,t}$ is the value of the chosen action or stimulus in a given trial t. $Q_{c,t}$ was updated using a reinforcement learning model (see Rescorla & Wagner, 1972):

$$Q_{c,t+1} = Q_{c,t} + \alpha \times \delta_{c,t}$$

where α is the participants' individual learning rate. Because the underlying model offers some degrees of freedom (for an example, see Weber & Bellebaum, 2024; Burnside et al., 2019), we tested four different models to update action and stimulus values.

In the first model (M_1), separately for each of the six pairs of actions or stimuli (and therefore for each of the three feedback timing conditions), both actions or stimuli of a pair were initially assigned a value of 0.5, that was iteratively updated in every trial t in which the respective action or stimulus was associated with feedback. The value of the chosen action or stimulus, Q_c, was updated based on the deviation between the prior value and the received outcome, that is, the PE δ, and a learning rate α, reflecting the degree to which the PE was used to update the action or stimulus value. The value of the unchosen stimulus or action equaled 1-Q_c and was therefore complementary to Q_c and updated accordingly. As both stimuli or actions were always presented together, receiving feedback for the chosen stimulus or action would automatically convey some information about the unchosen stimulus.

For each trial, $t_{1, \ldots, n_{trials}}$, the probability p that the model would choose the action or stimulus that was indeed chosen by the participant was calculated using the *softmax* function. This calculation was based on prior values of both stimuli or actions that participants could choose from, that is, values of the chosen action or stimulus, $Q_{c,t}$, and the unchosen action or stimulus in trial t, $Q_{u,t}$ (equaling 1-$Q_{c,t}$). The calculation also included an exploration parameter β:

$$p_{c,t} = \frac{e^{Qc,t \times \beta}}{e^{Qc,t \times \beta} + e^{Qu,t \times \beta}}$$

with β indicating the impact of prior action or stimulus values on a participant's choices. A larger β indicates that

a participant utilized prior values (i.e., a larger impact of prior values), whereas a smaller β indicates rather explorative choice behavior (i.e., a smaller impact of prior values).

In a next step, the probabilities p were used to calculate the negative summed log-likelihood ($-LL$) as measure for the model's goodness of fit:

$$-\sum \log\left(p_{c,t_1,\ldots,n_{trials}}\right)$$

We used the optimization function *fmincon* from the Optimization Toolbox of MATLAB to minimize the $-LL$ value by estimating values for the free parameters (α, β) that result in the least deviation between the model's predicted choices and the participants' behavior. To reduce the risk of local minima, the model was fitted to the participants' behavior repeatedly (50 iterations). As start values for the free parameters, we allowed random numbers within the interval [0; 1]. Value constraints for the free parameters were set to [0; 1] for the learning rate α and to [0; 100] for the exploration parameter β.

For the second model (M_2), we allowed different learning rates for learning from positive and negative feedback. Thus, the action or stimulus value was updated with the learning rate α_{con} for trials with positive feedback that confirmed the choice as follows:

$$Q_{c,t+1} = Q_{c,t} + \alpha_{con} \times \delta_{c,t}$$

Analogously, for trials with negative feedback that disconfirmed the choice, the value was updated with the learning rate α_{dis}:

$$Q_{c,t+1} = Q_{c,t} + \alpha_{dis} \times \delta_{c,t}$$

For both learning rates, boundary constraints were set to [0; 1] as for the one learning rate in M1. Everything else remained unchanged.

For the third model (M_3), we allowed two learning rates just as in M_2. However, we allowed each action or stimulus an independent Q_t value: This means that the values for the two actions or stimuli of a pair would no longer necessarily add to 1. Because positive feedback for the chosen stimulus can be interpreted as confirmation of both the choice of the chosen stimulus and the nonchoice of the unchosen stimulus, the update of the stimulus value for the unchosen stimulus, $Q_{u,t}$, was calculated for trials with positive feedback (i.e., reward for the chosen stimulus) as follows:

$$Q_{u,t+1} = Q_{u,t} + \alpha_{con} \times \delta_{u,t}$$

The update in trials with negative feedback for the chosen stimulus was done analogously with α_{dis}. The PE for the unchosen stimulus was computed as follows:

$$\delta_{u,t} = 1 - r_t - Q_{u,t}$$

Everything else remained unchanged compared with M_2.

For the last model (M_4), we allowed one Q_t value for each pair as for M_1 and M_2. Regarding learning rates, we allowed only one learning rate for learning from positive and negative feedback as in M1, but it was allowed to differ between the six pairs of actions or stimuli.

For a detailed comparison of the likelihoods of all four models, see the Appendix, Section A3). Model fit was best for M_2, so this model was used for the PE calculation subsequently used for the analyses.

EEG Data Analysis

Preprocessing. BrainVision Analyzer 2.2 software (Brain Products GmbH, 2018) and MATLAB R2021a (The MathWorks, Inc., 2021) were used for EEG data preprocessing. After rereferencing to the average of all scalp electrodes and the mastoids (and calculating the signal at the online reference site FCz), data were 30 Hz low-pass and 0.1 Hz high-pass filtered. To correct for blink artifacts, an independent component analysis was performed on single-subject EEG data. A component representing blinks (determined via its topography and correspondence with the VEOG signal) was then removed, and the EEG signal was reconstructed from the remaining components. We created segments from 200 msec before to 800 msec after feedback onset and performed a baseline correction relative to the first 200 msec. Then, segments with artifacts were removed (all segments containing voltage steps > 50 μV/msec, differences between values > 80 μV or < 0.1 μV within an interval of 100 msec or amplitudes > 80 μV or < −80 μV). This removed 1.69% of segments, on average (SD = 3.88%, maximum per participant = 19.17%). Although the analysis was based on single-trial data (see below), averages were also created. The remaining segments were thus averaged for each of the within-subject conditions (positive and negative immediate feedback, positive and negative delayed feedback without tone, and positive and negative delayed feedback with tone), yielding six averages per participant. Subsequently, all single-trial segment data as well as all averages per condition and participant were exported for later analysis. All further processing steps were performed in MATLAB.

In previous studies, visual inspection of the frontocentral electrodes showed that the FRN was maximal at FCz (Kim & Arbel, 2019; Arbel et al., 2017). To account for individual differences, we preregistered to measure FRN amplitudes at a group of five frontocentral electrode sites (for a similar approach, see Zottoli & Grose-Fifer, 2012), including Fz, FC1, FCz, FC2, and Cz (see Weber & Bellebaum, 2024). As the FRN/RewP amplitude difference between negative and positive feedback has been shown to reflect feedback expectancy (Sambrook & Goslin, 2015; Hajcak, Moser, Holroyd, & Simons, 2007), and single-trial amplitudes based on the difference wave reflect a PE (Weber

& Bellebaum, 2024) and have been linked to striatal processing (see above; Becker et al., 2014; Carlson et al., 2011), the time window for the single-trial analysis was determined based on the peak of the difference wave. Thus, for all three feedback timing conditions (immediate, delayed with tone, and delayed without tone), the average negative feedback − positive feedback difference wave was created for each participant. Then, the maximum negative peak in the difference wave between 200 and 400 msec after feedback onset was determined as specified in the preregistration (for a similar approach, see Paul, Vassena, Severo, & Pourtois, 2020). Through visual inspection, we ensured that the condition-specific difference wave peaks for each participant lay in fact within this time window. The latency of this peak in the respective feedback timing condition of each participant was then used to extract single-trial amplitude data. More specifically, the mean amplitude of a time window from 10 msec before to 10 msec after the condition-specific difference wave peak of each participant was calculated for each trial, representing the FRN/RewP.

For the N170, the signal at electrodes P7 and P8 (see Höltje & Mecklinger, 2020; Kim & Arbel, 2019; Arbel et al., 2017) was considered and single-trial amplitudes were derived with the help of the average ERPs at these electrodes as specified in the preregistration. We did not consider the hemisphere in the main analysis because we did not expect any hemisphere effects and aimed to keep model complexity low. We still determined the peaks separately for P7 and P8 because the electrodes, unlike the electrode sites used for the FRN/RewP, were so far apart that their signal was more independent. First, the latency of the peak amplitude between 140 and 250 msec postfeedback was determined in each participants' average, at both electrode sites and for all the six within-subject conditions (see above). Originally, we intended to use a time window of 140–200 msec like Höltje and Mecklinger (2020), as stated in the preregistration. However, the visual inspection of the condition-specific averages per participant led us to extend the time window to 250 msec. Then, for each single trial, the mean amplitude around (i.e., 10 msec before to 10 msec after) the condition-specific peak was calculated.

Statistical analysis of the ERP data. The single-trial amplitudes of the two ERP components were separately analyzed as dependent variables by applying linear mixed effect (LME; Bates et al., 2015) analyses in R (The R Foundation, 2021). The model for FRN/RewP included as fixed-effect predictors association type (between-subject: action–feedback [−0.5] vs. active stimulus–feedback [0.5]), feedback timing (within-subject with a simple coding contrast matrix; see also above), feedback valence (within-subject: negative [−0.5] vs. positive [0.5]), and PE (scaled and mean-centered). Importantly, PE in this analysis and in the analysis of the N170 (see below) refers to the unsigned or absolute PE, which represents general surprise. This was done because the signed PE is confounded by feedback valence, which is used as separate predictor in the model. A representation of the full (signed) PE would be reflected in an interaction between feedback valence and unsigned PE (see Weber & Bellebaum, 2024). The unsigned PE values were mean centered, yielding negative values for rather small PEs that lie below the mean versus positive values for rather high PE values above the mean. In addition, all possible interactions between the fixed-effect predictors were included. Random effects were determined as described above. To sum up, the model for the FRN/RewP was as follows:

$$\begin{aligned} &\textit{FRN Amplitude} \sim 1 + \textit{Association Type} \\ &\quad \times \textit{Feedback Timing} \times \textit{Feedback Valence} \times \textit{PE} \\ &\quad + (1 + \textit{Feedback Timing} + \textit{Feedback Valence} \mid \textit{Subject}) \end{aligned}$$

The model for the N170 comprised the same fixed-effect predictors as the one for the FRN/RewP described above (Feedback Association, Feedback Timing, Feedback Valence, PE, as well as all possible interactions). In addition, we added the Electrode as a random intercept to account for differences in amplitude. Random slopes by subject were determined as for the FRN/RewP model. To sum up, the model for the N170 was as follows:

$$\begin{aligned} &N170\ \textit{Amplitude} \sim 1 + \textit{Association Type} \\ &\quad \times \textit{Feedback Timing} \times \textit{Feedback Valence} \times \textit{PE} \\ &\quad + (1 + \textit{Feedback Timing} + \textit{Feedback Valence} \mid \textit{Subject}) \\ &\quad + (1 \mid \textit{Electrode}) \end{aligned}$$

For both the FRN/RewP and the N170, we hypothesized that PE effects (as interaction between unsigned PE and Feedback Valence) were modulated by Feedback Timing and Association Type. Our main interest was thus in higher-order interactions of the different predictors involving the factors Feedback Timing and/or Association Type.

RESULTS

Behavioral Results

Learning Check

t Tests comparing the mean accuracy rates in the test trials across all blocks of the AFC, ASFC, and PSFC to chance level (50%) revealed that accuracy rates were significantly above chance level (on a Bonferroni-corrected alpha level of .008) in the AFC, $t(17) = 6.95$, $p < .001$ ($M = 70.18\%$, $SD = 12.31\%$), and ASFC, $t(21) = 13.13$, $p < .001$ ($M = 82.36\%$, $SD = 11.56\%$), respectively. However, accuracy rates in the PSFC were not above chance level, $t(21) = 2.13$, $p = .045$ ($M = 52.60\%$, $SD = 5.72\%$). Interestingly, this pattern was even more pronounced in the fourth test block: Again, accuracy rates were

significantly above chance level in the AFC, $t(17) = 6.65$, $p < .001$ ($M = 71.74\%$, $SD = 13.86\%$), and ASFC, $t(21) = 12.46$, $p < .001$ ($M = 82.77\%$, $SD = 12.33\%$), but not in the PSFC, $t(21) = 0.84$, $p = .41$ ($M = 52.59\%$, $SD = 14.43\%$. A detailed illustration of the single-subject accuracy in the test trials (Figure A1 in the Appendix) as well as a statistical analysis of accuracy variation between blocks (Section A1 in the Appendix) revealed that accuracy varied more strongly from block to block in the PSFC than in the other conditions: This means that some participants' accuracies fluctuated between 0% and 100% from block to block due to choosing always the same stimulus in one block of test trials and correspondingly only the other stimulus in the following block of test trials. Although average accuracy rates were at chance level, the variations between the test blocks suggest that participants did form predictions during the learning blocks, which led then also to PEs that were modeled in our analyses. Nevertheless, the predictions and PEs in the PSFC, which were calculated based on the test trials without feedback, seem less reliable (see Section A1 in the Appendix), because the accuracy in the test trials in the PSFC is more variable than in the other learning conditions (see Section A1 in the Appendix) and affects also the PEs (see the Appendix, Section 4). We thus decided to focus mainly on the comparison of AFC and ASFC, whereas the analyses comparing the AFC and PSFC are reported in the Appendix, Section A2.

Comparison between Conditions

We compared learning performance between AFC and ASFC. Figure 2 displays the descriptive data underlying the GLME analysis on the accuracy in the learning trials for the AFC and ASFC. Table A4.1 in the Appendix lists b-estimates and effect-specific z tests. GLME analysis revealed a significant main effect of association type, $z = 3.04$, $p = .002$, $b = 0.71$. Participants of the ASFC showed enhanced performance compared with participants of the AFC. We also found a significant main effect of block, $z = 3.46$, $p = .001$, $b = 0.50$. Participants improved their performance across the four blocks in a learning session. All other effects were not significant (all $p \geq .059$). An additional post hoc analysis including only the first and last block aimed to focus more clearly on learning effects. This analysis also revealed a main effect of association type, $z = 2.71$, $p = .007$, $b = 1.06$ (see above), whereas there was a trend effect of block, $z = 1.96$, $p = .050$, $b = 0.26$. An interaction between block, association type, and the contrast between immediate feedback and delayed feedback without tones, $z = -1.99$, $p = .047$, revealed that the association type effect was present for both feedback timings in the first and for immediate feedback in the last block (all $p \leq .05$), but not for delayed feedback without tone in the last block ($p = .683$). Analyzing learning (i.e., Block) effects separately for each condition, no significant block effect emerged for any condition, only a trend for the ASFC and delayed feedback without tone ($p = .097$, $b = 0.81$).

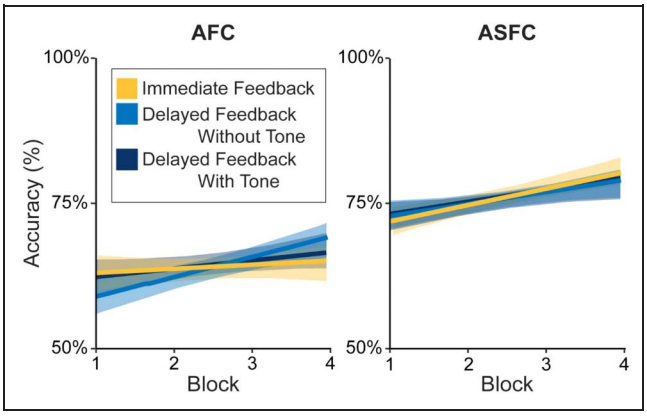


Figure 2. Accuracy in the learning parts. Error margins indicate 95% confidence intervals.

ERP Results

FRN/RewP

Grand averages and topographical maps of the ERPs according to association type, feedback timing, and feedback valence can be found in Figure 3.

Descriptive data underlying the FRN/RewP analysis are depicted in Figure 4. For F and p values of all main and interaction effects, refer to Table 1. In the text, statistical indices will only be reported for resolutions of interactions. For b-estimates and effect-specific t tests concerning the analysis of the FRN/RewP amplitude, see Table A4.2 of the Appendix. In the main text, we will focus on those results that are relevant regarding our hypotheses, that is, effects of the factors association type and feedback timing in interaction with the other factors.

In accordance with known findings from the literature, we found more negative FRN/RewP amplitudes for negative than positive feedback and this amplitude difference was larger for immediate than delayed feedback, immediate: $F(1, 205.56) = 102.25$, $p < .001$, $b = 1.85$, delayed without tone: $F(1, 210.11) = 41.17$, $p < .001$, $b = 1.22$ delayed with tone: $F(1, 232.53) = 71.96$, $p < .001$, $b = 1.63$ (for the two-way-interaction between feedback timing and feedback valence, see Tables 1 and A4.2).

Importantly, we also found a two-way interaction between feedback valence and PE (see Tables 1 and A4.2). For positive feedback, FRN/RewP amplitudes were significantly more positive the higher the PE was, $F(1, 11339.36) = 69.59$, $p < .001$, $b = 1.85$. For negative feedback, FRN/RewP amplitudes were more negative the

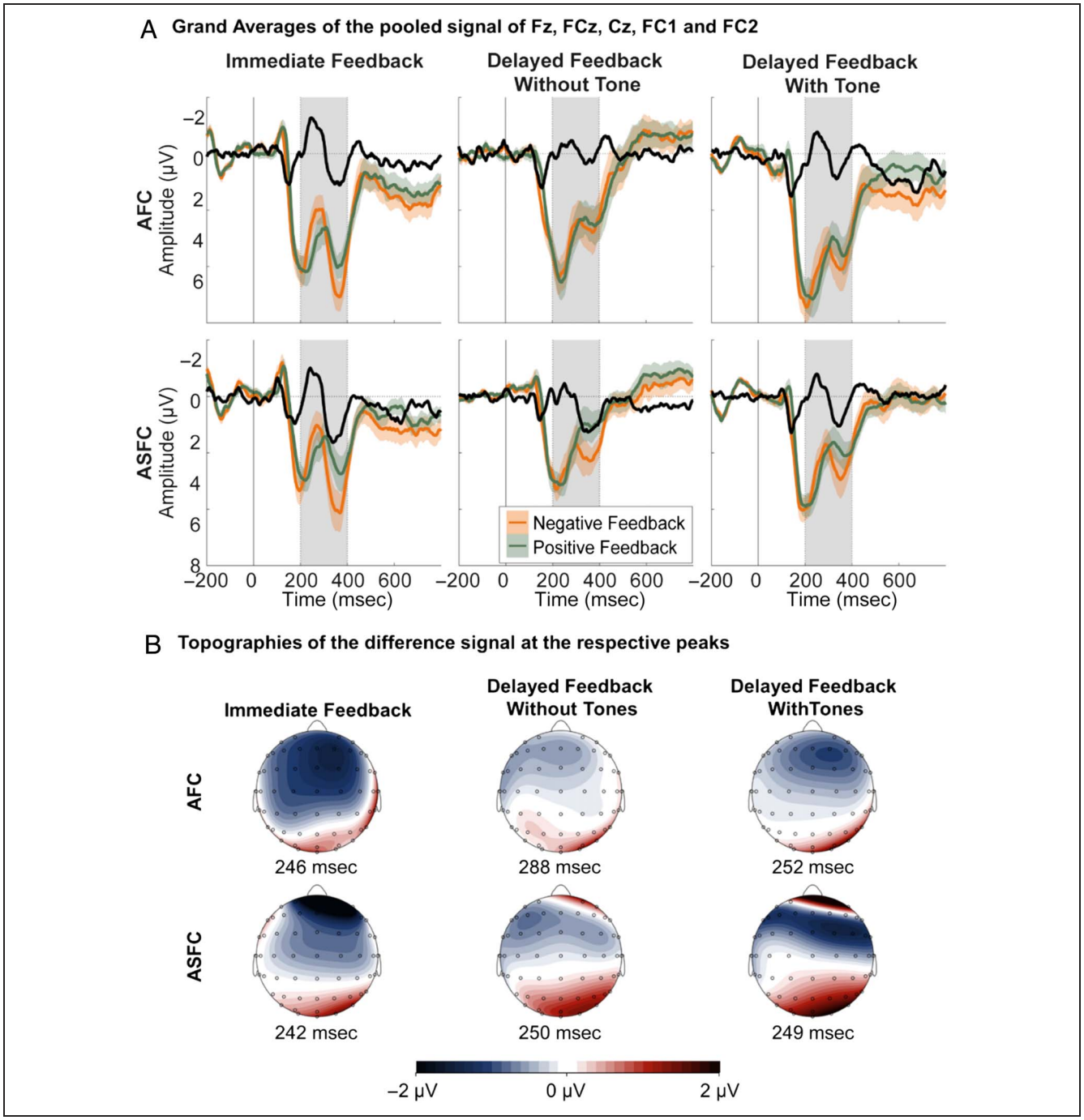


Figure 3. Grand averages and topographical maps of the FRN/RewP. (A) Grand averages of the pooled signal of Fz, FCz, Cz, FC1, and FC2: The dotted vertical lines represent the search window for the FRN/RewP peak of the difference wave. Error margins represent standard errors. (B) Topographies of the difference signal at the respective peaks: distribution of activation within the FRN/RewP time window.

higher the PE was, $F(1, 13631.45) = 8.99$, $p = .003$, $b = -0.67$. For a display of this interaction, see Figure 5.

This interaction was further modulated by the factors of main interest in our study. First, a significant three-way interaction of feedback valence, PE, and feedback timing emerged (see Tables 1 and A4.2), indicating that the Feedback Valence × PE interaction with the above-described pattern was stronger for immediate, $F(1, 17546.57) = 40.88$, $p < .001$; $F(1, 15985.23) = 48.44$, $p < .001$, $b = 2.58$ for positive feedback; $F(1, 16879.95) = 4.07$, $p = .024$, $b = -0.83$ for negative feedback; than delayed feedback without tones, $F(1, 16919.16) = 22.22$, $p < .001$; positive feedback: $F(1, 13411.40) = 18.27$, $p < .001$, $b = 1.67$; negative feedback: $F(1, 17197.83) = 6.61$,

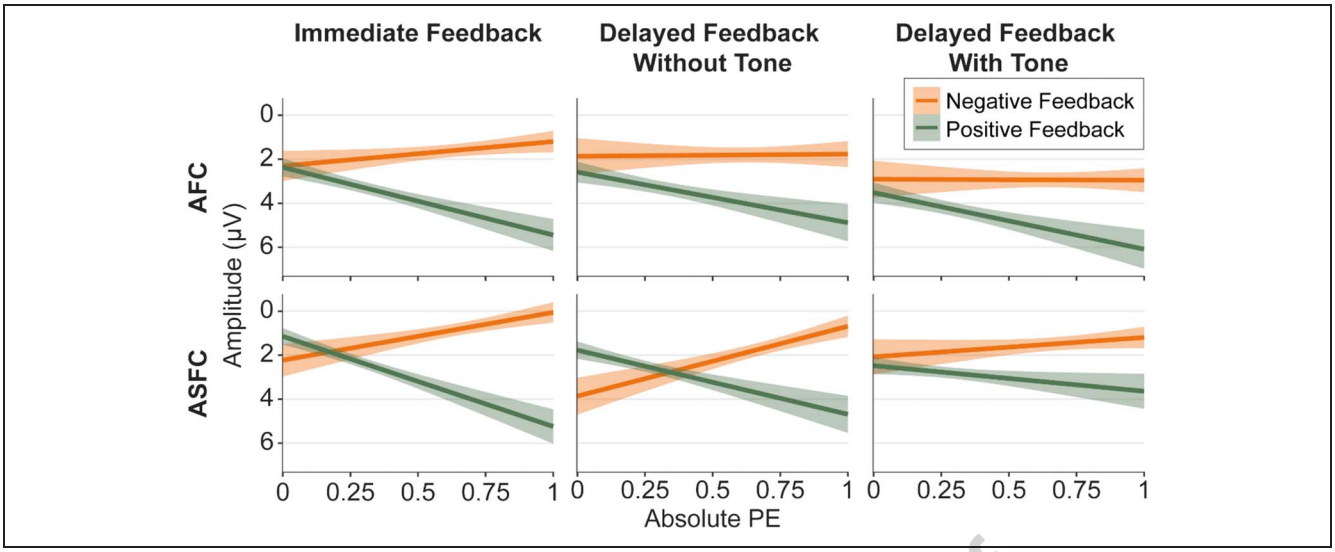


Figure 4. Descriptive statistics for the FRN/RewP model. Error margins indicate 95% confidence intervals. PE = absolute (unsigned) PE.

$p = .010$, $b = -1.01$; and delayed feedback with tones, $F(1,16732.41) = 7.03$, $p = .008$; positive feedback: $F(1, 15355.51) = 11.85$, $p < .001$, $b = 1.31$; negative feedback: $p = .64$.

Finally, the interaction between feedback valence and PE was modulated by association type, as revealed by another three-way interaction of the mentioned factors (Tables 1 and A4.2). In follow-up analyses, a

Table 1. *F* and *p* Values for the LME Analysis on the FRN/RewP Amplitude in the AFC and ASFC

Effects	*Num DF*	*Den DF*	*F*	*p*
Association type	1.00	38.56	1.65	.207
Feedback timing	2.00	41.66	2.31	.112
Feedback valence	1.00	53.27	137.14	**<.001**
PE	1.00	6695.48	14.48	**<.001**
Association Type × Feedback Timing	2.00	41.66	2.20	.123
Association Type × Feedback Valence	1.00	53.27	3.18	.080
Feedback Timing × Feedback Valence	2.00	17801.32	4.50	**.011**
Association Type × PE	1.00	6695.48	3.77	.052
Feedback Timing × PE	2.00	17767.70	1.12	.325
Feedback Valence × PE	1.00	17851.58	61.73	**<.001**
Association Type × Feedback Timing × Feedback Valence	2.00	17801.32	0.67	.510
Association Type × Feedback Timing × PE	2.00	17767.70	1.48	.229
Association Type × Feedback Valence × PE	1.00	17851.58	4.85	**.028**
Feedback Timing × Feedback Valence × PE	2.00	16503.34	3.12	**.044**
Association Type × Feedback Timing × Feedback Valence x PE	2.00	16503.34	0.52	.594

df = degrees of freedom; *Num DF* = numerator degrees of freedom; *Den DF* = denominator degrees of freedom; association type = AFC versus ASFC; feedback timing = immediate feedback, delayed feedback without tone, and delayed feedback with tone; feedback valence = negative versus positive; PE = unsigned.

Bolded *p* values indicate statistical significance at $p < .05$.

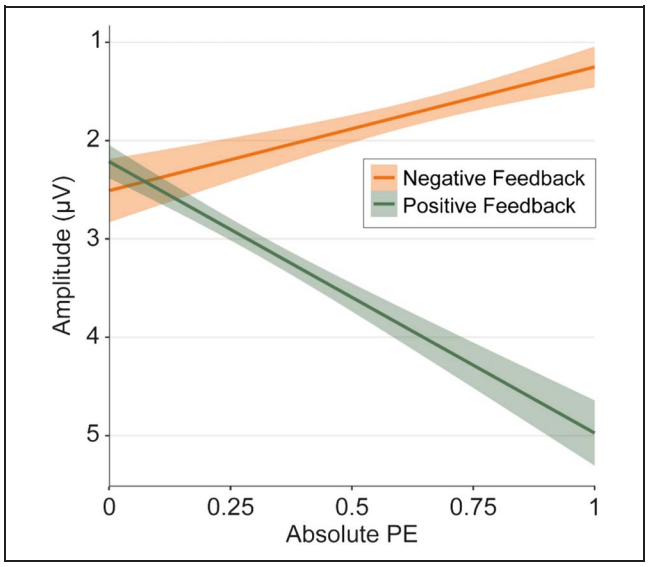


Figure 5. Descriptive statistics for the PE × Feedback Valence interaction of the FRN/RewP. Error margins indicate 95% confidence intervals. Absolute PE = absolute (unsigned) PE.

significant Feedback Valence × PE interaction emerged for action–feedback associations, $F(1, 17861.99) = 15.43$, $p < .001$; for positive feedback, FRN/RewP amplitudes were more positive the larger the PE was, $F(1, 10918.30) = 31.95$, $p < .001$, $b = 1.80$; and for negative feedback, no effect of PE was found, $F(1, 12702.16) < 0.01$, $p = .96$, $b = -0.02$. For stimulus–feedback associations, the two-way interaction was more pronounced, $F(1, 17823.99) = 52.48$, $p < .001$; for positive feedback, higher PEs meant more positive FRN/RewP amplitudes, $F(1, 11774.27) = 37.86$, $p < .001$, $b = 1.90$, and for negative feedback, higher PEs meant more negative FRN/RewP amplitudes, $F(1, 14601.96) = 18.05$, $p < .001$, $b = -1.33$. Although thus effects of feedback timing and association type did emerge in interaction with feedback valence and PE, neither the three-way interaction between feedback valence, feedback timing, and association type, nor the four-way interaction including the mentioned factors and the additional factor PE reached significance, which was against our hypotheses (see Table 1).

N170

Grand averages and topographical maps of the ERPs according to association type, feedback timing, and feedback valence can be found in Figure 6. Descriptive data underlying the N170 analysis are depicted in Figure 7. In Table 2, we report the F and p values of all main and interaction effects. For b-estimates and effect-specific t tests concerning the analysis of the N170 amplitude, see Table A4.3 of the Appendix. Again, we include only the effects that are most relevant for our research question and report statistical indices in the text only for resolutions of interactions.

Among other effects, we found larger N170 amplitudes for delayed than immediate and for negative than positive feedback (main effects of feedback timing [immediate vs. delayed feedback with tone: $p < .001$, $b = -1.22$; immediate vs. delayed feedback without tone: $p = .38$, $b = -0.28$] and feedback valence [$b = 0.62$]; see Tables 2 and A4.3).

Interestingly, also for the N170, there was a significant two-way interaction between feedback valence and PE (see Tables 2 and A4.3). Significant effects of PE were found for both positive feedback, $F(1, 28404.85) = 27.75$, $p < .001$, $b = -1.00$, and negative feedback, $F(1, 31236.99) = 8.91$, $p = .003$, $b = 0.57$; for positive feedback, higher PE values led to more negative N170 amplitudes, and for negative feedback, higher PE values led to more positive N170 amplitudes. See Figure 8 for a display of this interaction.

As for the FRN, this interaction was modulated by association type, as shown by the three-way interaction between feedback valence, PE, and association type (see Tables 2 and A4.3). The resolution of this interaction revealed that the two-way interaction effect between feedback valence and PE was more pronounced for stimulus–feedback associations, $F(1, 35660.91) = 37.31$, $p < .001$, than for action–feedback associations, $F(1, 35104.91) = 4.51$, $p = .034$. In both groups, higher PE values for positive feedback led to more negative N170 amplitudes, $F(1, 28328.20) = 28.66$, $p < .001$, $b = -1.41$, and $F(1, 28387.10) = 4.60$, $p = .032$, $b = -0.58$, respectively, but the effect was more pronounced for stimulus–feedback associations. An effect of PE for negative feedback emerged for stimulus–feedback associations, $F(1, 32484.84) = 11.28$, $p = .001$, $b = 0.89$, with more positive amplitudes the higher the PE, but not for action–feedback associations ($p = .37$).

Finally, we also found a significant four-way interaction between feedback valence, PE, association type, and feedback timing (Tables 2 and A4.3), indicating that the interaction between feedback valence and PE described above was modulated by both, feedback timing and association type. Resolving the four-way interaction by association type, there was no significant interaction between feedback timing, feedback valence, and PE for stimulus–feedback associations, $F(2, 23509.87) = 1.16$, $p = .32$. Irrespective of feedback timing, the pattern of PE coding for positive and negative feedback described above for stimulus–feedback associations emerged. However, there was an interaction effect for the action–feedback associations, $F(2, 18378.69) = 3.08$, $p = .046$. Further resolving by feedback timing, there was no significant interaction between feedback valence and PE for action–feedback associations for delayed feedback without tones, $F(1, 21047.58) = 0.93$, $p = .33$, or for delayed feedback with tones, $F(1, 18349.99) = 0.10$, $p = .76$,

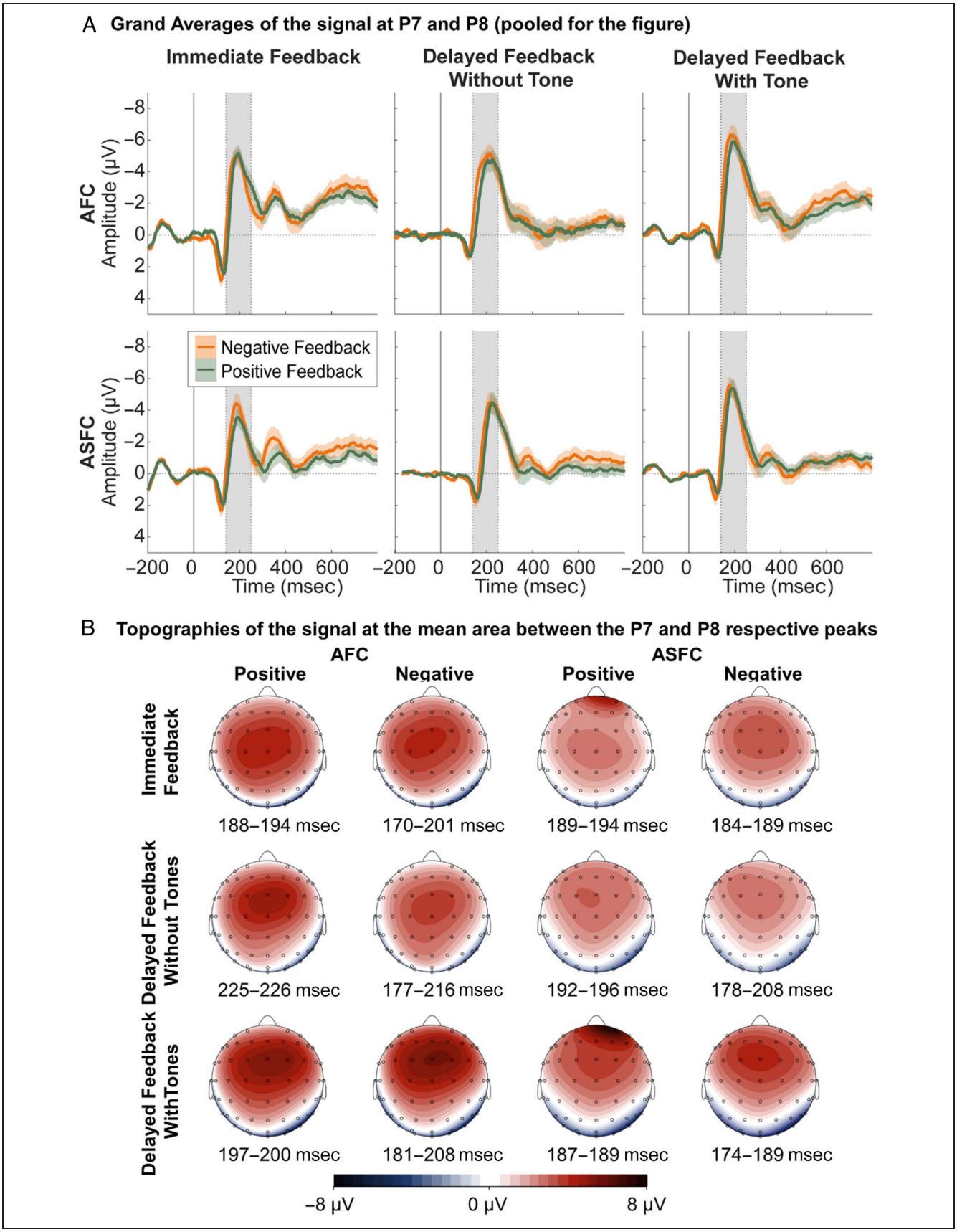


Figure 6. Grand averages and topographical maps of the N170. (A) Grand averages of the signal at P7 and P8: The dotted vertical lines represent the search window for the N170 negative peak. Error margins represent standard errors. (B) Topographies of the signal at the respective peaks: distribution of activation within the N170 time window.

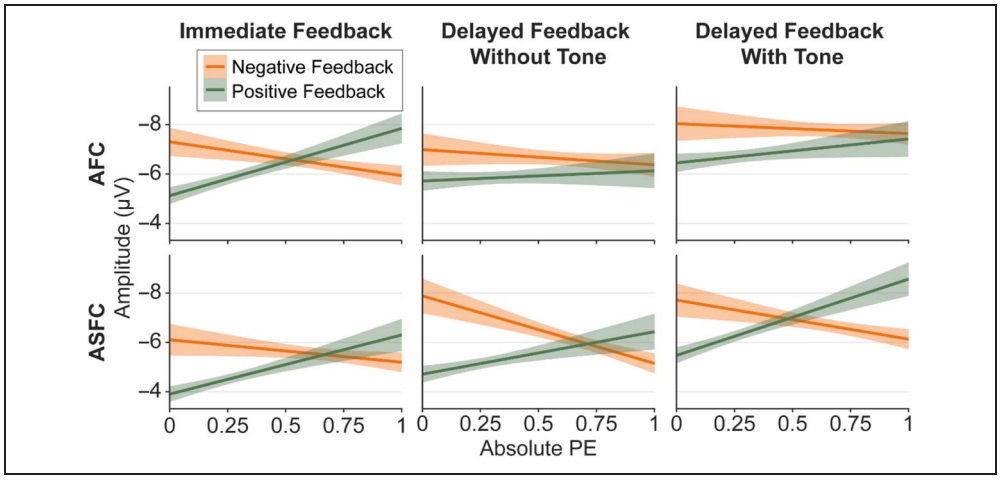


Figure 7. Descriptive statistics of the N170 model. Error margins indicate 95% confidence intervals. PE = absolute (unsigned) PE.

indicating that the PE did not modulate the N170 amplitude in these conditions. However, for immediate feedback, an interaction between PE and feedback valence was found, $F(1, 33400.93) = 10.48$, $p = .001$. For immediate negative feedback in action–feedback associations, there was no significant effect of PE, $F(1, 35059.21) = 2.14$, $p = .14$, $b = 0.65$, but there was a PE effect for immediate positive feedback in action–feedback associations, $F(1, 29227.14) = 10.35$, $p = .001$, $b = -1.42$. The higher the PE, the more negative the N170 amplitude.

To sum up, we found the PE effect on the N170 (as interaction between the absolute PE and feedback valence) to be modulated by both feedback timing and association type, reflected in the significant four-way interaction, as hypothesized. However, the pattern underlying this interaction differed from our hypothesis. Moreover, we did not find the hypothesized two-way interaction between feedback timing and association type with most pronounced N170 amplitudes for delayed feedback for stimulus feedback associations.

Table 2. Statistical Results for the LME Analysis on the N170 Amplitude in the AFC and ASFC

Effects	*Num DF*	*Den DF*	*F*	*p*
Association type	1.00	38.30	1.16	.289
Feedback timing	2.00	44.75	19.40	**<.001**
Feedback valence	1.00	44.32	13.06	**.001**
PE	1.00	21005.49	2.59	.107
Association Type × Feedback Timing	2.00	44.75	0.72	.493
Association Type × Feedback Valence	1.00	44.32	0.48	.492
Feedback Timing × Feedback Valence	2.00	35710.36	4.43	**.012**
Association Type × PE	1.00	21005.60	0.12	.732
Feedback Timing × PE	2.00	35673.87	3.90	**.020**
Feedback Valence × PE	1.00	35460.14	33.33	**<.001**
Association Type × Feedback Timing × Feedback Valence	2.00	35710.36	1.68	.186
Association Type × Feedback Timing × PE	2.00	35673.87	0.34	.715
Association Type × Feedback Valence × PE	1.00	35460.08	7.40	**.007**
Feedback Timing × Feedback Valence × PE	2.00	20798.09	0.27	.762
Association Type × Feedback Timing × Feedback Valence × PE	2.00	20797.69	4.03	**.018**

Bolded p values indicate statistical significance at $p < .05$.

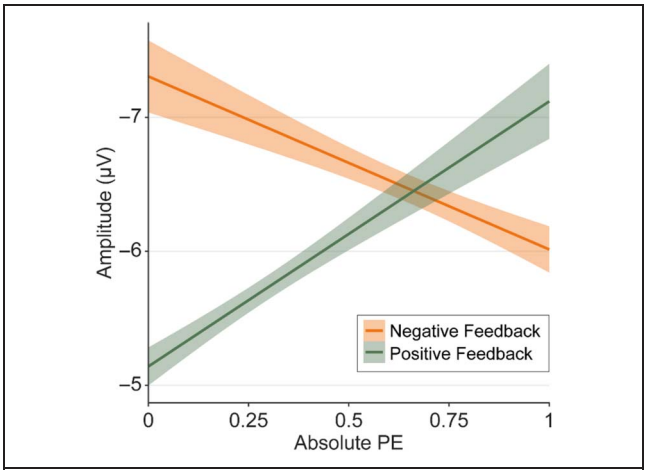


Figure 8. Descriptive statistics for the PE × Feedback Valence interaction of the N170. Error margins indicate 95% confidence intervals. Absolute PE = absolute (unsigned) PE.

DISCUSSION

Previous research indicates that delaying feedback leads to a shift in the neural correlates of feedback processing: Whereas the FRN/RewP difference between negative and positive feedback, assumedly reflecting striatal reward system activity (Becker et al., 2014; Foti et al., 2011), was reduced for delayed feedback, the N170, possibly associated with MTL and/or extrastriate cortex activity, was increased (Höltje & Mecklinger, 2020; Kim & Arbel, 2019; Arbel et al., 2017; Peterburs et al., 2016). We hypothesized that also the type of the learned association (action–feedback vs. stimulus–feedback associations) could affect which system is involved in feedback processing, potentially in interaction with feedback timing effects. Consequently, we expected a stronger role of the FRN/RewP in feedback processing for action–feedback associations for immediate feedback and of the N170 in learning stimulus–feedback associations for delayed feedback. To test this, participants learned either action–feedback or stimulus–feedback associations with immediate and delayed feedback while we recorded EEG. Stimulus–feedback associations were learned in two different ways, either actively, involving active stimulus choices, or passively, without active choices. Significant learning was found only for action–feedback associations and for the stimulus–feedback associations involving active choices. Due to this, we focus the discussion on results from these conditions, especially concerning effects of the PE, which might be more meaningful when learning has taken place.

Influences of Valence, Feedback Timing, and Association Type on Feedback Processing

Regarding the FRN/RewP, we expected the largest valence effect for immediate feedback for action–feedback associations. Although we replicated previous findings of a more pronounced feedback valence effect for immediate than delayed feedback (for similar results, see Albrecht et al., 2023; Höltje & Mecklinger, 2020; Arbel et al., 2017; Peterburs et al., 2016; Weismüller & Bellebaum, 2016; Weinberg et al., 2012), the interaction between feedback valence and timing did not differ between type of learned association, which was against our hypotheses. In contrast, in the analysis involving passive stimulus–feedback associations reported in the Appendix, there was evidence for a feedback valence effect on the FRN/RewP modulated by both feedback timing and type of learned association. The valence effect differed between passive stimulus–feedback associations and action–feedback associations only for immediate feedback, with smaller differences for stimulus–feedback associations. This was partly in line with our hypothesis, suggesting a slight preference for action–feedback associations in immediate feedback (although no difference between groups was found for the delayed feedback conditions).

For the N170, we expected the largest amplitude for delayed feedback for stimulus–feedback associations. Similar as for the FRN/RewP, we found a feedback timing by valence interaction, but in the opposite direction, such that a feedback valence effect (as Kim & Arbel, 2019) with more negative amplitudes following negative compared with positive feedback was only found for delayed feedback. In contrast to our hypothesis, neither the effect of feedback timing nor the reported interaction between feedback valence and timing was affected by the association type in our main analysis. However, in the analysis involving passive stimulus–feedback associations, such an effect did emerge. As hypothesized, we found larger N170 amplitudes for delayed feedback, irrespective of temporal predictability (i.e., with or without tone) only for (passive) stimulus–feedback associations.

Reflections of the PE in the FRN/RewP

For our main analysis, effects of the type of the learned association on feedback processing were only seen in interaction with the PE. For both, the FRN/RewP and the N170, we expected that the effect of the PE (as interaction between unsigned or absolute PE and valence) would be modulated by feedback timing and association type. For the FRN/RewP, we found an interaction between feedback valence and absolute PE, which is in line with PE coding by the FRN/RewP as suggested by the Reinforcement Learning Theory (Holroyd & Coles, 2002) and with many other studies (see Burnside et al., 2019; Fischer & Ullsperger, 2013): This two-way interaction reflecting PE processing was further modulated by feedback timing, as it was strongest for immediate feedback. This appears to contradict the finding of a recent study by Weber and Bellebaum (2024), in which the PE was similarly represented in the FRN/RewP for immediate and delayed positive feedback, and no PE effect was found for negative feedback. Finally,

PE processing, again as reflected in the two-way interaction between valence and unsigned PE, was also modulated by association type. The interaction was more pronounced for stimulus–feedback associations, for which the above-described pattern of PE effects for both negative and positive feedback emerged. For action–feedback associations, a PE effect was found only for positive feedback, mirroring a pattern also described by Weber and Bellebaum (2024). Similarly, Kirsch, Kirschner, Fischer, Klein, and Ullsperger (2022) found stronger expectancy coding in the FRN/RewP for positive compared with negative feedback. Thus, the PE coding in the FRN/RewP was indeed modulated by feedback timing and association type, but not as hypothesized in interaction of the two factors.

Of particular interest is the finding that for action–feedback associations, PE coding is restricted to positive feedback. This may indicate that for nonrewarded actions, it does not matter how large the negative PE is. Because PEs are a prerequisite for learning (Schultz & Dickinson, 2000), this aligns with findings of a confirmation bias with higher learning rates from positive compared with negative feedback (Weber & Bellebaum, 2024). Furthermore, people tend to be better at learning to achieve rewards through active choice actions and avoid punishments by remaining passive—for which the term *pavlovian bias* was coined (Peterburs, Albrecht, & Bellebaum, 2022; Cavanagh, Eisenberg, Guitart-Masip, Huys, & Frank, 2013; Guitart-Masip et al., 2012). Studies on this bias challenge the existing view that neural representations in the striatum are centered on valence, suggesting that the striatum might rather encode a tendency toward action (Guitart-Masip et al., 2011; for a review, see Guitart-Masip, Duzel, Dolan, & Dayan, 2014). On this background, our results might suggest that positive reinforcement is especially important for action learning.

Reflections of the PE in the N170

For the N170, we also observed PE coding, with the reversed pattern compared with the FRN/RewP. This is a novel finding, as the N170 has not been linked to PE processing in the context of feedback learning before. The finding supports not only the relevance of the N170 as a feedback-locked signal, but it further sheds light on the relationship between striatal and MTL learning systems. Enhanced amplitudes following unexpected rewards may indicate that the MTL is particularly involved in reinforcing memories for affirming feedback. The MTL's function could be to remember which stimulus resulted in a reward by reactivating its representation in visual areas, thereby strengthening the association. This would mean increasing activity in the fusiform gyrus, which would lead to enhanced N170 amplitudes (Gao et al., 2019; Deffke et al., 2007). Previous studies have identified not only reward representations in the hippocampus (Gauthier & Tank, 2018; Zeithamova, Gelman, Frank, & Preston, 2018) but also postreward reactivation as a mechanism to link rewarding outcomes to the preceding experiences (Singer & Frank, 2009). In addition, fMRI studies revealed the reactivation of stimulus-specific visual (Schiffer et al., 2014) and somatosensory regions (Pleger et al., 2008, 2009) following reward. We found that the N170 was more pronounced when negative feedback was expected versus unexpected. As participants could choose between two options in the current task, expected negative feedback could be processed as confirming feedback for the not-chosen stimulus or action, leading to additional effort to reactivate the not-chosen stimulus or action representation.

Importantly, the PE coding in the N170 was modulated by both association type and feedback timing, as revealed by a four-way interaction. The above-described PE coding pattern in the N170 (as Feedback Valence × PE interaction) emerged only for immediate feedback in action–feedback associations, whereas it was present in all feedback timing conditions for stimulus–feedback associations. Although this pattern does not exactly match our hypothesis, it is still consistent with our expectations that, for delayed feedback, there is a stronger PE effect for stimulus–feedback than action–feedback associations. This is in line with a stronger role of hippocampally mediated reactivations of visual stimuli associated with feedback in the stimulus–feedback condition during delayed feedback. The enhanced N170 for delayed feedback found for stimulus–feedback, but not action–feedback associations in the analysis involving the PSFC (see above), further supports this notion. Unexpectedly, we observed a (similarly pronounced) PE effect on the N170 for both types of associations for immediate feedback, which is contrary to our hypothesis that we should see PE effects on the N170 especially for delayed feedback that refers to stimuli. A possible explanation might be that the N170 reflects not only MTL activity (as suggested by Arbel et al., 2017) or visual reactivation (as we suggest in the introduction), but both processes: PE coding in the MTL might appear in parallel to PE coding in the striatum, irrespective of association type only for immediate feedback. However, visual reactivation in extrastriate cortex might be stronger for delayed feedback (and influence the N170 more in this condition) because of the need of credit assignment, and would only happen for stimulus–feedback associations, not action–feedback associations.

Integration of Findings for the FRN/RewP and the N170

Looking at FRN/RewP and N170 results together, it is evident that the relative recruitment of different learning and memory systems during feedback learning in the present study depended on the type of association learned. Post hoc, we suggest that the cooperation of the striatal and the hippocampal system worked better for stimulus–feedback associations, for which better learning

was found, with PE coding for N170 and FRN/RewP in all timing conditions. The cooperation may be less effective for action–feedback associations, where it may have worked only for immediate feedback. In line with this, Palombo, Hayes, Reid, and Verfaellie (2019) found significantly impaired learning for patients with hippocampal damage compared with healthy controls in a probabilistic learning task involving stimulus–feedback associations (although see Foerde et al., 2013, for comparable results at least with immediate feedback). In 2002, White and McDonald suggested that both the striatal and hippocampal system share access to the same information, but they specialize in encoding different types of relationships. Indeed, midbrain dopamine neurons project to the striatum (Chuhma et al., 2023; Oldehinkel et al., 2022; Schultz et al., 1997) and also to the MTL (Schott et al., 2004, 2008; Lisman & Grace, 2005). In line with this, both the striatum and the hippocampus have been found to reflect a PE during feedback learning (Dickerson et al., 2011). Shohamy and Adcock (2010) suggest that the interactions between midbrain dopamine regions and the MTL enhance memory for rewarding and novel episodes and build memory representations that inform future decision-making.

Feedback Delay Effects and Temporal Predictability

Kimura and Kimura (2016) proposed that feedback delay effects on the FRN/RewP are a function of diminished temporal predictability for delayed feedback and found comparable patterns between immediate and delayed feedback when delayed feedback was made similarly predictable via a regular tone during the delay. In the present study, we did not replicate this result and found mostly similar feedback delay effects for temporally predictable and unpredictable delayed feedback. The participants in the study by Kimura and Kimura could not learn during the study, so the FRN/RewP effect found might not represent a learning signal, but specifically PEs about the temporal characteristics of the feedback stimuli. In the current study, learning was possible, and FRN/RewP and N170 could thus represent learning signals, which might explain the differences in the result pattern.

Limitations

The primary limitation of this study is that passive stimulus–feedback associations were not learned consistently. Participants of the PSFC, which aimed to assess stimulus–feedback learning without actions, did not learn, on average, leading to difficulties in comparing the respective EEG results from this condition with the AFC and ASFC. Problems with learning might be attributed to a number of reasons, for example, a potential need of higher contingencies in passive (meaning no own actions were conducted) compared with active learning (Bellebaum, Brodmann, & Thoma, 2014) or difficulty in learning passively when the events to be learned from are not executed by another human (differences in processing of human and nonhuman actions have for example been found by Fukushima & Hiraki, 2009). A closer look at the accuracy rates in the PSFC revealed that although participants did not choose the more rewarding stimulus more frequently, on average, over the entire experiment, they did choose it exclusively in the test trials of some blocks. In some cases, accuracy rates fluctuated between 0% and 100% from block to block. The block design, in which 20 learning trials are followed by 20 test trials, is based on observational learning studies (e.g., Bellebaum et al., 2014). This type of design, however, sometimes leads to participants choosing only one stimulus within a block. In the case of a false belief regarding the more rewarding stimulus, this results in an accuracy of 0%. Together with accuracy rates of 100% in other blocks, this leads to an average accuracy of around 50%, which is what we observed in the PSFC. We thus primarily base our interpretations on the comparison of action–feedback associations with the stimulus–feedback associations learned in the ASFC and not in the PSFC. Although the ASFC also contained actions (i.e., button presses), feedback was only related to the chosen stimuli and not to the actions. On the contrary, in the AFC, visual stimuli (i.e., rectangles) were presented on the screen, but the feedback referred only to the actions and not to the stimuli. The two conditions are therefore compatible but differ in one crucial point: In the ASFC, stimulus identity predicted the reward, whereas in the AFC, motor aspects of the task predicted the reward. Better learning for active stimulus–feedback associations compared with action–feedback associations could indicate that this association type allowed the striatal and MTL systems to cooperate in the most effective way. However, findings from the PSFC also contributed to the interpretation of the role of the N170 in feedback learning: Despite overall inconsistent learning, participants in the PSFC may nevertheless have formed a (false) expectation regarding the stimuli's feedback contingencies, allowing meaningful PE analyses. In this sense, adding the PE to the statistical model may, at least to some extent, take differences in learning performance between the groups into account. Indeed, data from the PSFC revealed larger N170 amplitudes for delayed than immediate feedback, as hypothesized. Nevertheless, future studies should try to separate action–feedback and stimulus–feedback associations even more by overcoming learning problems in the PSFC. Due to our relatively small sample size, it would be interesting to see whether the effects reported here can be replicated.

Conclusion

In conclusion, our results suggest that the type of the learned association, action–feedback or stimulus–feedback, does play a role in feedback processing and feedback learning. This was evident in both the

FRN/RewP and N170 ERP components, which are thought to represent feedback processing in the striatal or MTL system. However, no clear-cut pattern was found in the sense that one of the systems takes over sole control in specific learning conditions. Instead, the learned association, together with feedback timing, seems to modulate how well the systems cooperate during learning. Furthermore, a modulation of the N170 by the PE (reversed to that of the RewP/FRN) provides new evidence that the component is associated with feedback processing, especially for delayed feedback. The study results challenge previous views of a competitive relationship between MTL- and striatal-based learning systems, suggesting a cooperation that is modulated by learning contexts.

APPENDIX
Section A1

Statistical Comparison of Accuracy Variance between AFC, ASFC, and PSFC

To statistically investigate variability between blocks in the three association type groups, we first calculated the mean accuracy (in % correct responses) for each participant across all blocks. In a next step, we calculated the (absolute) accuracy deviation from this mean for each block and used this as the accuracy variance measure for this block. We then defined an LME model with accuracy variance as dependent variable and block (1–4, scaled to lie between −0.5 and + 0.5), association type (AFC, ASFC, and PSFC, modulated in a simple coding matrix with PSFC as baseline), and feedback timing (modulated in a simple coding matrix with immediate feedback as baseline). Fitting the model with the procedure described in the main articles resulted in the following model:

$$Variance \sim Block \times Association\ Type \times Feedback\ Timing + (1|Subject)$$

We found a significant effect of association type, $F(2, 59.01) = 23.02, p < .001$. Variance was significantly higher in the PSFC compared with both the AFC ($t = -4.91, p < .001, b = -15.55$) and ASFC ($t = -6.45, p < .001, b = -19.40$). Recalculating the model with AFC as baseline for the association type, we found no difference between AFC and ASFC ($p = .230$). No other main or interaction effects were significant (all $p \geq .058$). For descriptive data of the accuracy variance of the three association type groups, see Table A1.1. Only five participants of the PSFC had a lower or equal mean accuracy variance than the overall mean of the AFC (only four PSFC participants had a lower mean variance than the ASFC mean).

Table A1.1. Mean, Standard Deviation, Minimum, and Maximum of Block-wise Accuracy Variance (in %)

AssociationType	*M*	*SD*	*Min*	*Max*
AFC	18.04	6.30	6.94	33.23
ASFC	14.17	8.82	0.40	31.88
PSFC	30.66	13.25	6.09	47.50

M = mean; *SD* = standard deviation; Min = minimum; Max = maximum.

Section A2

Results for the AFC Compared with the PSFC

PE modeling. For comparability, we used the same model constraints as for the PE calculation of the main analysis, where we used the following model (M_2 in the main text):

$$Q_{c,t+1} = Q_{c,t} + \alpha_{con/dis} \times \delta_{c,t}$$

Each action or stimulus was initially assigned a value of 0.5 that was iteratively updated in every trial t in which the action or stimulus was chosen. The value of the unchosen action or stimulus, Q_u, was updated as $1\text{-}Q_c$ in each trial. We allowed different learning rates for learning from positive (α_{con}) and negative feedback (α_{dis}).

Because learning trials in the PSFC did not entail own choice actions, the data from the learning trials could not be used to calculate the model's error term. Instead, the error (i.e., the deviance between the model's predictions and the values measured, which is $-LL$ in the PE calculation for the main analysis) was calculated using the probabilities derived from the test trials for both conditions, AFC and PSFC: After the first learning block, the Q values of the two stimuli should equal the frequency in which they were chosen during the following test trials. Frequency was calculated as the number of test trials in which one stimulus was chosen divided by the total number of trials in the respective test block. We defined the residual error as the absolute difference between the Q

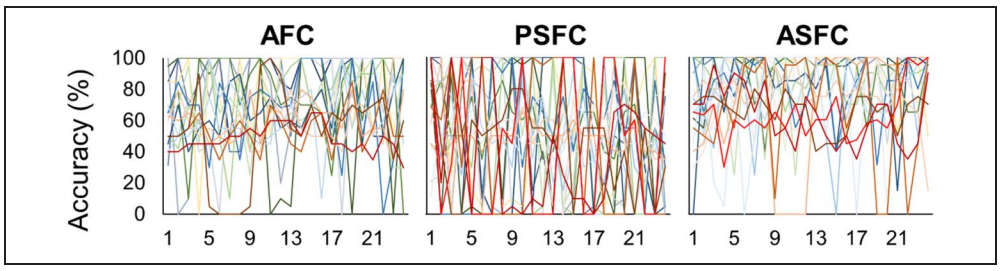


Figure A1. Single-subject representation of accuracy rates across all 24 test blocks without feedback. Each line represents one participant of the respective association type group.

value calculated by the model of the "correct" stimulus or action (the one that was more associated with wins) after the last trial of each learning block and the frequency of its choice during the following test block. We only used one of the stimuli or actions of each block because, as the values were inverted, using both would not add any information. Note that this calculation of the residual error term did not consider the exploration parameter β. To further adapt the model to the one used for the main analysis, we chose the minimum and maximum of α_{con} and α_{dis} from the model of the main analysis of the AFC condition as constraints for the respective values (α_{con}: [0.00–1], α_{dis}: [0.00–0.62]). For the subsequent analyses, the absolute values of the PE for each trial were used.

Behavioral results. Because participants in the PSFC did not make active choices during the learning trials, we used the test trials to compare behavioral results between the AFC and PSFC. We used a model including block, association type, and feedback timing as fixed effects and the main effects of block and feedback timing as well as their interaction as random effects. See the table of results (Table A2.1) as well as the descriptive data (Figure A2.1) depicted below. There was a main effect of association type (see Table A2.1) with higher accuracy in the AFC than PFSC. All other effects were not significant. Only including the first and last block for an additional post hoc analysis focusing on learning effects in the different conditions, we also found a main effect of association type, $z = -3.60$, $p < .001$, $b = -0.94$ (see above), and again no significant main effect of block ($p = .758$). However, there was a significant interaction between block and association type, $z = -2.01$, $p = .045$. In both the first block, $z = -2.29$, $p = .022$, $b = -0.62$, as well as the last block, $z = -3.76$, $p < .001$, $b = -1.26$, participants in the AFC performed significantly better than in the PSFC, but the effect was more pronounced in the last block. Analyzing learning (i.e., block) effects separately for the different conditions, there was a trend for immediate feedback in the PSFC ($p = .073$, $b = -0.73$; decreasing accuracy from Blocks 1–4) and for delayed feedback without tones in the AFC ($p = .091$, $b = 0.89$; increasing accuracy from Blocks 1–4).

EEG results. Due to the differences in learning performance between the PSFC and the AFC, differences in the neural signals between the groups are difficult to interpret. Although the inclusion of the PE in the statistical model may, to some extent, account for learning differences, PE modeling for the observed behavioral learning pattern in the PSFC (mainly 0% and 100% correct in the test blocks) is not ideal. Nevertheless, the results give some insight into the general influences of feedback valence and feedback timing in the PSFC, and we can carefully compare effects of these between the groups.

FRN. For the analyses, we used a model including association type, feedback timing, feedback valence, and PE as fixed effect factors, as well as the main effects of feedback timing and feedback valence as random effects. The tables of results for the model, the grand averages, and the descriptive data are depicted below (Figures A2.2 and A2.3, Tables A2.2 and A2.3). As for the analyses reported in the main article, we will focus on effects of the factors association type and feedback timing in interaction with the other factors.

Table A2.1. *b* Values, Confidence Intervals, and *t*-test Results for the GLME Analysis on Accuracy for the AFC and PSFC

Effects	*b*	*SE*	*z*	*p*	*2.5% CI*	*97.5% CI*
Intercept	0.63	0.09	6.70	**<.001**	0.45	0.82
Block	−0.05	0.15	−0.33	.740	−0.33	0.27
Association type	−0.97	0.19	−5.23	**<.001**	−1.34	−0.60
Delayed feedback with tone	−0.23	0.17	−1.32	.185	−0.59	0.15
Delayed feedback without tone	−0.24	0.13	−1.85	*.064*	−0.49	0.03
Block × Association Type	−0.37	0.31	−1.22	.223	−0.95	0.22
Block × Delayed Feedback With Tone	0.46	0.42	1.10	.273	−0.38	1.27
Block × Delayed Feedback Without Tone	0.71	0.49	1.44	.150	−0.17	1.75
Association Type × Delayed Feedback With Tone	−0.10	0.34	−0.28	.776	−0.77	0.60
Association Type × Delayed Feedback Without Tone	0.13	0.26	0.49	.624	−0.41	0.70
Block × Association Type × Delayed Feedback With Tone	−0.19	0.81	−0.23	.819	−1.99	1.58
Block × Association Type × Delayed Feedback Without Tone	1.45	0.95	1.54	.125	−0.71	3.44

The sign of the *b*-estimates indicates the direction of main effects for the fixed-effects predictors block (1 [−0.5], 2 [−0.167], 3 [0.167], 4 [0.5]), association type (AFC [−0.5] vs. PSFC [0.5]), and feedback timing (simple coding contrast matrix with immediate feedback set as baseline that is compared with delayed feedback with tone and delayed feedback without tone).

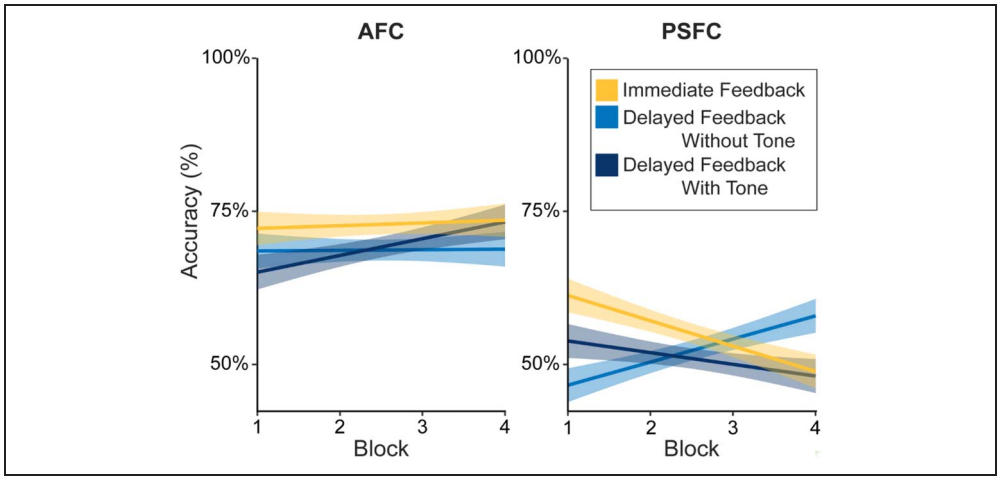


Figure A2.1. Accuracy in the test parts for the AFC and PSFC. Error margins represent 95% confidence intervals.

As for the analysis comparing AFC and ASFC in the main text, there was a significant main effect of feedback valence with more negative amplitudes for negative compared with positive feedback (see Table A2.2 and A2.3). Instead of an interaction between feedback timing and feedback valence, as for the AFC – ASFC comparison, we found a significant interaction involving these two factors and the factor association type (feedback timing, feedback valence, and association type; see Figure A2.4). Only for immediate feedback, we found a significant interaction between association type and feedback valence, $F(1, 136.88) = 5.23$, $p = .024$ ($p = .118$ for delayed feedback without tones and $p = .180$ for delayed feedback with tones). For immediate feedback, a significant valence effect emerged for the AFC, $F(1, 119.02) = 47.60$, $p < .001$, $b = 2.04$, and PSFC, $F(1, 160.31) = 14.40$, $p < .001$, $b = 1.09$. In both association types, negative feedback led to more negative FRN/RewP amplitudes, but the effect was weaker for the PSFC.

As for the analysis reported in the main article, there was an interaction between feedback valence and PE, which was further modulated by association type (interaction between association type, feedback valence, and PE). The underlying pattern differed, however. While a significant interaction between feedback valence and PE showed for the AFC, $F(1, 17782.20) = 13.43$, $p < .001$, indicating PE coding, as in the analysis in the main text, FRN amplitude did not appear to vary with the PE for the PSFC ($p = .75$ for the interaction between feedback valence and PE). The pattern for the AFC was as reported in the main text (note that the PE was modeled differently, see above): For negative feedback in the AFC, there

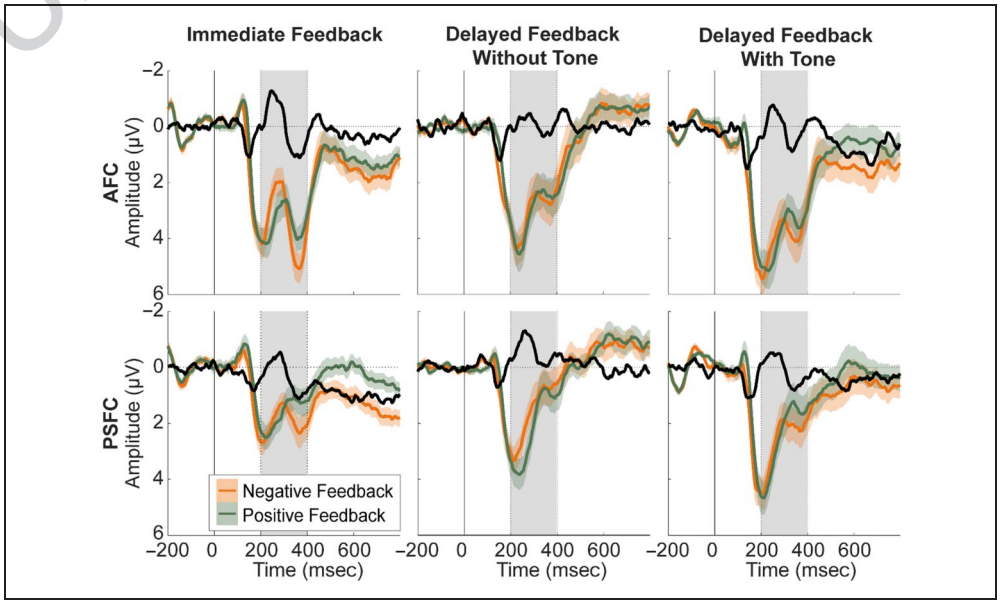


Figure A2.2. Grand averages of the FRN for the AFC and PSFC. Error margins represent standard errors.

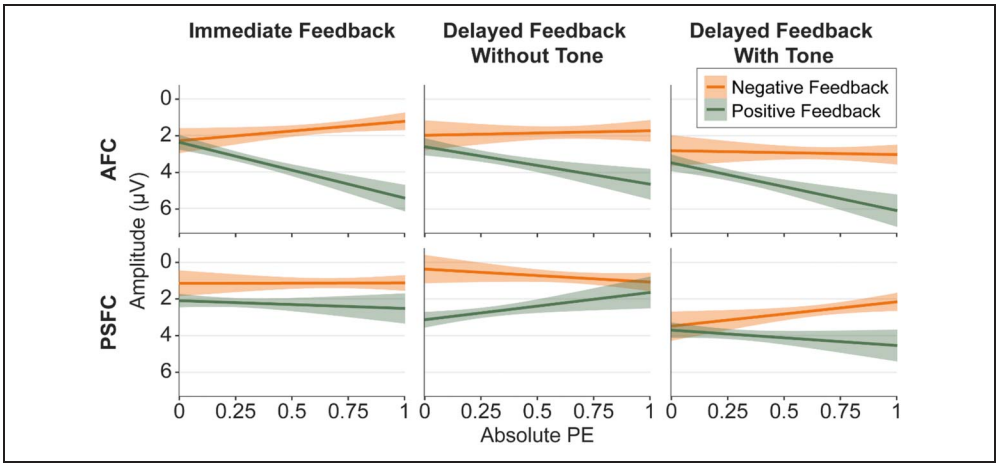


Figure A2.3. Descriptive statistics for the FRN model for the AFC and PSFC. Error margins represent 95% confidence intervals. Absolute PE = absolute (unsigned) PE.

was no effect of PE ($p = .92$), but there was an effect for positive feedback in the AFC, $F(1, 15295.69) = 26.84$, $p < .001$, $b = 1.67$. Participants showed more negative amplitudes for low PE values than for high PE values.

As hypothesized, we thus found that the effect of the factor feedback valence was modulated by feedback timing and association type. Only for immediate feedback, the valence effect (i.e., more negative amplitude for negative than positive feedback) was stronger for action–feedback than for stimulus–feedback associations. This pattern emerged, however, because the feedback valence effect was reduced for stimulus–feedback associations for immediate feedback compared with all other conditions (see Figure A2.4).

N170. For the analysis of the N170, we used a model including association type, feedback timing, feedback valence, and PE as fixed effect factors, as well as the main effects of hemisphere and feedback timing and their interaction as random effects. The tables of results for the model, the grand averages, and the descriptive data are depicted below (Figures A2.5 and A2.6, Tables A2.4 and

Table A2.2. *F* and *p* Values for the LME Analysis on the FRN/RewP Amplitude for the AFC and PSFC

Effects	*Num DF*	*Den DF*	*F*	*p*
Association type	1.00	38.48	1.40	.244
Feedback timing	2.00	40.33	5.11	**.011**
Feedback valence	1.00	47.60	102.19	**<.001**
PE	1.00	9588.29	7.12	**.008**
Association Type × Feedback Timing	2.00	40.33	0.17	.840
Association Type × Feedback Valence	1.00	47.60	0.90	.349
Feedback Timing × Feedback Valence	2.00	17734.65	0.72	.489
Association Type × PE	1.00	9588.29	6.51	**.011**
Feedback Timing × PE	2.00	17712.54	0.08	.926
Feedback Valence × PE	1.00	17785.19	8.09	**.004**
Association Type × Feedback Timing × Feedback Valence	2.00	17734.65	6.53	**.001**
Association Type × Feedback Timing × PE	2.00	17712.54	1.41	.244
Association Type × Feedback Valence × PE	1.00	17785.19	5.75	**.017**
Feedback Timing × Feedback Valence × PE	2.00	16682.57	1.22	.295
Association Type × Feedback Timing × Feedback Valence × PE	2.00	16682.57	0.04	.957

Association type (AFC vs. PSFC), feedback timing (immediate, delayed without tone, delayed with tone), feedback valence (negative vs. positive), and PE (unsigned PE)

Table A2.3. *b* Values, Confidence Intervals, and *t*-test Results of the LME Analysis on the FRN/RewP Amplitude for the AFC and PSFC

Effects	*b*	*SE*	*df*	*t*	*p*	*2.5% CI*	*97.5% CI*
Intercept	2.67	0.34	38.48	7.75	**<.001**	2.01	3.37
Association type	−0.82	0.69	38.48	1.18	.244	−2.14	0.63
Delayed feedback with tone	−0.02	0.35	40.96	0.05	.958	−0.67	0.59
Delayed feedback without tone	1.29	0.47	39.78	2.73	**.009**	0.45	2.28
Feedback valence	1.62	0.16	47.60	0.11	**<.001**	1.32	1.93
PE	0.42	0.16	9588.29	2.67	**.008**	0.11	0.76
Association Type × Delayed Feedback With Tone	−0.02	0.69	40.96	0.03	.979	−1.35	1.51
Association Type × Delayed Feedback Without Tone	0.47	0.95	39.78	0.50	.623	−1.29	2.46
Association Type × Feedback Valence	−0.30	0.32	47.60	0.95	.349	−0.93	0.30
Delayed Feedback With Tone × Feedback Valence	0.25	0.23	17735.14	1.09	.276	−0.20	0.73
Delayed Feedback Without Tone × Feedback Valence	0.03	0.23	17732.12	0.11	.913	−0.43	0.49
Association Type × PE	−0.80	0.31	9588.29	2.55	**.011**	−1.40	−0.21
Delayed Feedback With Tone × PE	0.06	0.36	17720.46	0.17	.865	−0.61	0.78
Delayed Feedback Without Tone × PE	−0.08	0.37	17721.36	0.23	.819	−0.87	0.69
Feedback Valence × PE	0.93	0.33	17785.19	2.84	**.004**	0.30	1.61
Association Type × Delayed Feedback With Tone × Feedback Valence	1.58	0.46	17735.14	3.47	**.001**	0.65	2.46
Association Type × Delayed Feedback Without Tone × Feedback Valence	0.39	0.47	17732.12	0.82	.409	−0.52	1.32
Association Type × Delayed Feedback With Tone × PE	0.57	0.73	17720.46	0.78	.436	−1.09	2.04
Association Type x Delayed Feedback Without Tone × PE	−0.68	0.73	17721.36	0.93	.354	−2.26	0.76
Association Type × Feedback Valence × PE	−1.56	0.65	17785.19	2.40	**.017**	−2.80	−0.19
Delayed Feedback With Tone × Feedback Valence × PE	−1.03	0.78	16213.38	1.33	.182	−2.58	0.48
Delayed Feedback Without Tone × Feedback Valence × PE	−1.06	0.79	17477.54	1.34	.180	−2.75	0.39
Association Type × Delayed Feedback With Tone × Feedback Valence × PE	0.07	1.55	16213.38	0.04	.966	−2.93	3.08
Association Type × Delayed Feedback Without Tone × Feedback Valence × PE	0.44	1.58	17477.54	0.28	.779	−2.83	3.40

b = beta estimate; *SE* = standard error; *CI* = confidence interval. The sign of the *b*-estimates indicates the direction of main effects for the fixed-effects predictors association type (action [−0.5] vs. stimuli active [0.5]), feedback timing (simple coding contrast matrix with immediate feedback set as baseline that is compared with delayed feedback with tone and delayed feedback without tone), feedback valence (negative [−0.5] vs. positive [0.5]), and mean centered unsigned PE.

A2.5). Again, we focus on the factors of main interest, association type, and feedback timing, in interaction with other factors.

As for the analysis reported in the main text, there were significant main effects of feedback timing, $F(2, 43.53) = 24.88$, $p < .001$, and feedback valence $F(1, 47.82) = 10.56$, $p = .002$. However, there was a significant two-way interaction between association type and feedback timing, $F(2, 43.53) = 10.63$, $p < .001$, which did not emerge in the analysis reported in the main article. For a display of the interaction, see Figure A2.7 (A). Although there was a main effect of feedback timing for the AFC, $F(2, 42.25) = 5.49$, $p = .008$, there was only a trend for the difference between delayed feedback with tone and immediate feedback in the AFC ($p = .083$, $b = -0.89$) and no difference between delayed feedback without tone and immediate feedback in the AFC ($p = .890$). For the PSFC, the feedback timing effect was stronger, $F(2, 45.11) = 32.56$, $p < .001$, with significantly more negative amplitudes for delayed feedback without tone compared with immediate feedback in the PSFC ($p < .001$, $b = -2.52$) and significantly more negative amplitudes for delayed

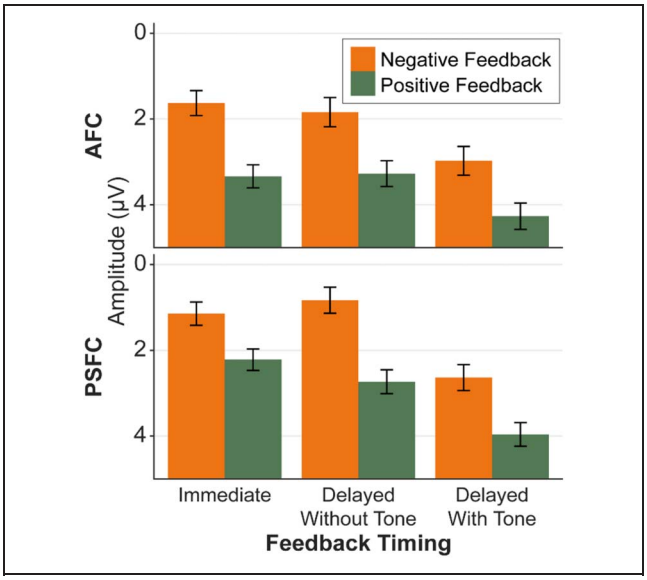


Figure A2.4. Interaction between association type (AFC vs. PSFC), feedback timing, and feedback valence on the FRN/RewP. Error bars represent 95% confidence intervals.

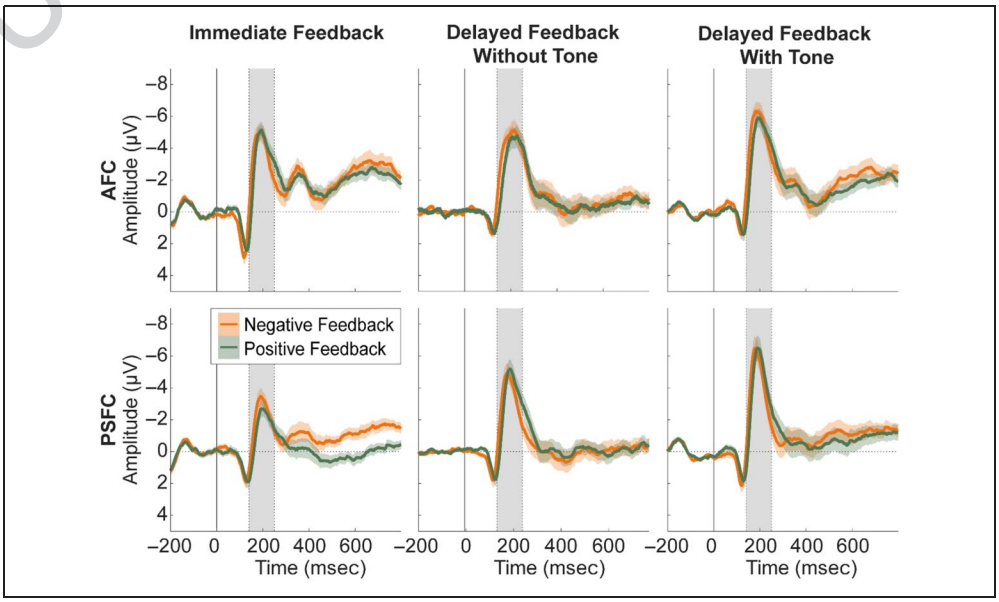
feedback with tone compared with immediate feedback in the PSFC ($p < .001$, $b = -3.71$).

This interaction could be further explained by a three-way interaction between association type, feedback timing, and feedback valence (see Figure A2.7, B). In the resolution of this interaction, however, no differential pattern of valence coding was found for any of the conditions ($p = .27$ for the Feedback Timing × Feedback Valence interaction in the PSFC, $p = .054$ in the AFC).

We additionally found a three-way interaction between association type, feedback valence, and PE, as in the analysis reported in the main text. A significant interaction between feedback valence and PE emerged for both the AFC, $F(1, 35258.96) = 5.73$, $p = .017$, and the PSFC, $F(1, 34840.33) = 12.71$, $p < .001$, but the effects were reversed in the two groups: For the AFC, a PE effect emerged not for negative ($p = .28$) but only for positive feedback, $F(1, 24746.49) = 5.59$, $p = .018$, $b = -0.67$. The higher the PE, the more negative the N170 amplitude, which was the same pattern as reported in the main analysis. For the PSFC, there was a PE effect for negative feedback, $F(1, 20122.58) = 6.51$, $p = .011$, $b = -0.71$, and positive feedback, $F(1, 22965.52) = 6.66$, $p = .010$, $b = 0.73$. For negative feedback, higher PE values led to more negative N170 amplitudes, but for positive feedback, higher PE values led to more positive N170 amplitudes, thus resembling the pattern described for the FRN/RewP.

Finally, the four-way interaction between association type, feedback timing, feedback valence, and PE was significant, as in the analysis on the AFC and the ASFC in the main article. For the AFC, there was (only) a trend for an interaction between feedback timing, feedback valence, and PE. Although the pattern of PE coding in the N170, as described in the main article, was visible in all feedback timing conditions, it seemed to be most pronounced for immediate feedback for action–feedback associations, so that the pattern, despite the nonsignificant interaction, is roughly comparable between the results reported in the main article and the ones reported here. For the PSFC, however, there was a significant interaction between feedback timing, feedback valence, and PE, $F(2, 15704.16) = 3.04$, $p = .048$, in contrast to what was reported in the main article. For the PSFC, with delayed feedback without tone, there was no significant interaction between feedback valence and PE ($p = .92$). Such an interaction did emerge for delayed feedback with tone, $F(1, 17919.60) = 7.85$, $p =$

Figure A2.5. Grand averages of the N170 for the AFC and PSFC. Error margins represent standard errors.



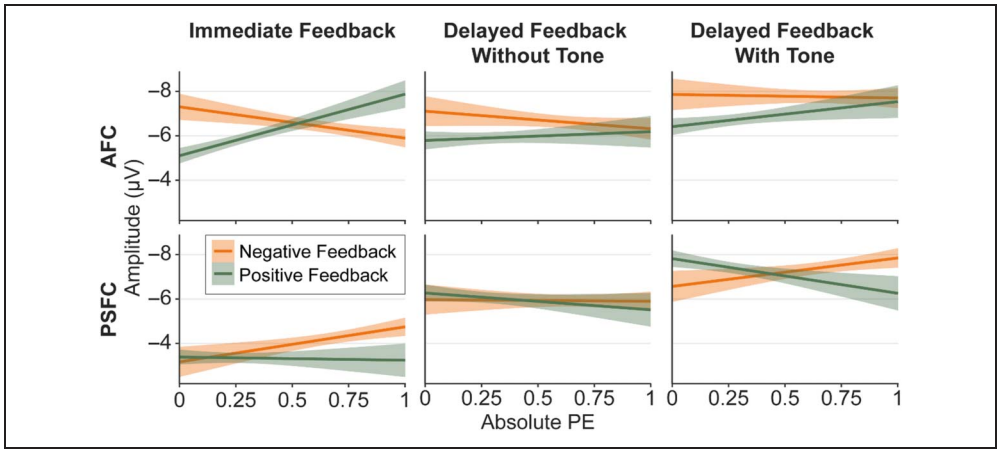


Figure A2.6. Descriptive statistics for the N170 model for the AFC and PSFC. Error margins represent 95% confidence intervals. Absolute PE = absolute (unsigned) PE.

.005, and immediate feedback, $F(1, 33075.55) = 11.51$, $p = .001$. A significant main effect of PE showed for the PSFC with positive delayed feedback with tone, $F(1, 24266.87) = 7.18$, $p = .007$, $b = 1.28$. The larger the PE, the more positive the N170 amplitude. No PE effect emerged for negative feedback ($p = .15$). For the PSFC with positive immediate feedback, no PE effect emerged ($p = .133$), but for negative feedback, $F(1, 33451.01) = 11.57$, $p = .001$, $b = -1.56$. The larger the PE, the more negative the N170 amplitude.

To summarize, our analyses revealed the hypothesized interaction between association type and feedback timing, with higher N170 amplitudes for delayed than immediate feedback in the PSFC, but not in the AFC. As in the analysis reported in the main article, also the four-way interaction reached significance, indicating that PE coding was modulated by feedback timing and association type. Due to the differences in learning and thus PE distribution between the AFC and the PSFC, we will focus on the pattern found in the analysis in the main article.

Table A2.4. *F* and *p* Values for the LME Analysis on the N170 Amplitude for the AFC and PSFC

Effects	*Num DF*	*Den DF*	*F*	*p*
Association type	1.00	38.27	1.84	.183
Feedback timing	2.00	43.53	24.88	**<.001**
Feedback valence	1.00	47.82	10.56	**.002**
PE	1.00	11919.56	0.38	.537
Association Type × Feedback Timing	2.00	43.53	10.63	**<.001**
Association Type × Feedback Valence	1.00	47.82	2.40	.128
Feedback Timing × Feedback Valence	2.00	35495.17	0.25	.782
Association Type × PE	1.00	11919.60	0.48	.488
Feedback Timing × PE	2.00	35223.42	1.97	.139
Feedback Valence × PE	1.00	35091.40	0.64	.425
Association Type × Feedback Timing × Feedback Valence	2.00	35495.16	3.97	**.019**
Association Type × Feedback Timing × PE	2.00	35223.43	1.17	.310
Association Type × Feedback Valence × PE	1.00	35091.44	17.71	**<.001**
Feedback Timing × Feedback Valence × PE	2.00	18049.55	1.84	.159
Association Type × Feedback Timing × Feedback Valence × PE	2.00	18049.69	3.96	**.019**

Association type (AFC vs. PSFC), feedback timing (immediate, delayed without tone, delayed with tone), feedback valence (negative vs. positive), and PE (unsigned PE).

Table A2.5. *b* Values, Confidence Intervals, and *t*-test Results for the LME Analysis on the N170 Amplitude for the AFC and PSFC

Effects	*b*	*SE*	*df*	*t*	*p*	*2.5% CI*	*97.5% CI*
Intercept	−6.19	0.71	2.29	−8.71	**.008**	−7.59	−4.80
Association type	1.13	0.83	38.27	1.36	.183	−0.59	2.68
Delayed feedback with tone	−1.23	0.28	41.34	−4.36	**<.001**	−1.76	−0.67
Delayed feedback without tone	−2.30	0.34	40.72	−6.75	**<.001**	−2.95	−1.63
Feedback valence	0.47	0.14	47.82	3.25	**.002**	0.20	0.75
PE	−0.09	0.14	11919.56	−0.62	.537	−0.39	0.20
Association Type × Delayed Feedback With Tone	−2.58	0.57	41.34	−4.56	**<.001**	−3.63	−1.48
Association Type × Delayed Feedback Without Tone	−2.81	0.68	40.72	−4.13	**<.001**	−4.19	−1.52
Association Type × Feedback Valence	−0.44	0.29	47.82	−1.55	.128	−0.98	0.10
Delayed Feedback With Tone × Feedback Valence	0.13	0.20	35547.34	0.63	.530	−0.28	0.54
Delayed Feedback Without Tone × Feedback Valence	0.01	0.21	35469.87	0.04	.964	−0.39	0.43
Association Type × PE	0.19	0.28	11919.60	0.69	.488	−0.39	0.71
Delayed Feedback With Tone × PE	0.64	0.32	35208.39	1.98	**.047**	−0.03	1.30
Delayed Feedback Without Tone × PE	0.30	0.32	35277.54	0.93	.351	−0.31	0.89
Feedback Valence × PE	0.23	0.29	35091.41	0.80	.425	−0.33	0.77
Association Type × Delayed Feedback With Tone × Feedback Valence	−1.12	0.40	35547.34	−2.78	**.005**	−1.97	−0.40
Association Type × Delayed Feedback Without Tone × Feedback Valence	−0.74	0.41	35469.85	−1.80	.072	−1.57	0.10
Association Type × Delayed Feedback With Tone × PE	−0.07	0.65	35208.42	−0.11	.911	−1.30	1.12
Association Type × Delayed Feedback Without Tone × PE	0.83	0.65	35277.52	1.28	.201	−0.49	2.26
Association Type × Feedback Valence × PE	2.42	0.57	35091.44	4.21	**<.001**	1.31	3.66
Delayed Feedback With Tone × Feedback Valence × PE	−0.31	0.68	23113.18	−0.46	.646	−1.71	1.06
Delayed Feedback Without Tone × Feedback Valence × PE	0.98	0.69	27046.50	1.42	.157	−0.35	2.32
Association Type × Delayed Feedback With Tone × Feedback Valence × PE	−3.76	1.37	23113.39	−2.75	**.006**	−6.39	−1.15
Association Type × Delayed Feedback Without Tone × Feedback Valence × PE	−2.54	1.39	27046.44	−1.83	.067	−5.12	−0.11

The sign of the *b*-estimates indicates the direction of main effects for the fixed-effects predictors association type (action [−0.5] vs. stimuli active [0.5]), feedback timing (simple coding contrast matrix with immediate feedback set as baseline that is compared with delayed feedback with tone and delayed without tone), feedback valence (negative [−0.5] vs. positive [0.5]), and mean centered unsigned PE.

Section A3

PE Model Comparisons

M_2 had the lowest Akaike information criterion (AIC) value, AIC = 448.74. To determine whether the model performed indeed significantly better as the other models, we performed one-sided dependent-samples *t* tests to compare the negative log likelihood values of each participant for each of the other three models with Model 2. Because this meant three statistical tests, results were interpreted on a Bonferroni-corrected alpha value of .017. M_2 had significantly smaller negative log likelihood values than all other models. For detailed statistical results, see the table below.

Section A4

Statistical Comparison of the Reinforcement Learning Models between AFC, ASFC, and PSFC

To determine if the enhanced accuracy variance in the test trials that we found for the PSFC (see Section A1) also

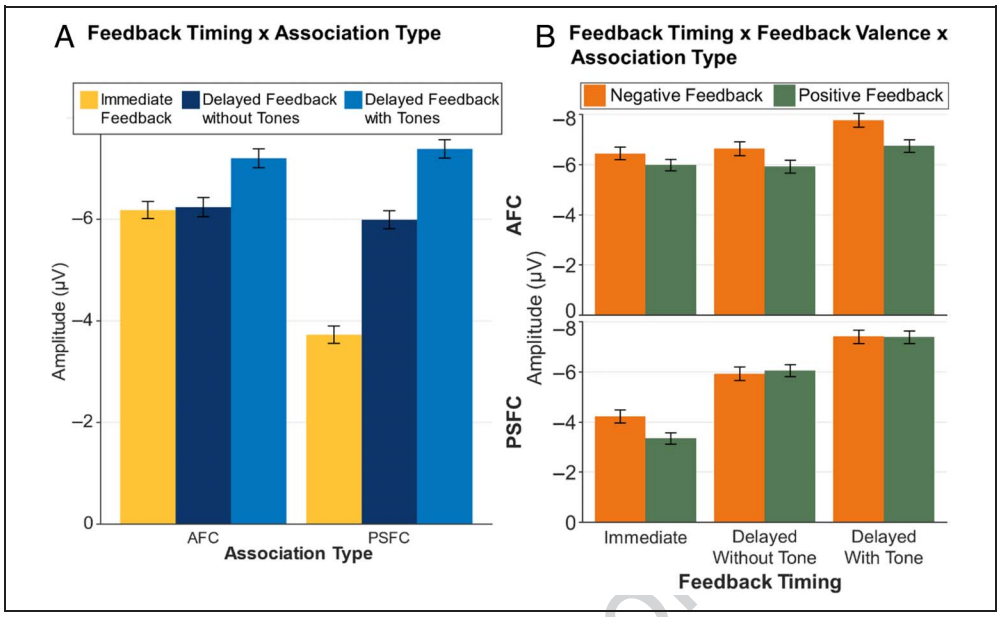


Figure A2.7. Significant interactions involving association type (AFC vs. PSFC) on the FRN/RewP. Error bars represent 95% confidence intervals.

affects the reinforcement learning models and thus the PEs, we compared the residual errors for the calculation of the subjective values of the stimuli based on the test trials (see Section A2 for details) between the three association type conditions (across feedback timings) using a one-factor ANOVA. Although the reinforcement learning model could also be calculated based on the learning trials in the ASFC and the AFC, these conditions also entailed test trials without feedback so that the reinforcement learning model could also be based on accuracy in the test trials, as in the PSFC (see Section A2 of the Appendix). We found a significant effect of association type on the residual errors of the reinforcement learning model, $F(2, 58) = 33.60$, $p < .001$. Bonferroni-corrected pairwise comparisons revealed that the error in the PSFC was significantly higher than in the AFC ($p < .001$) and in the ASFC ($p < .001$), but no difference could be observed between AFC and ASFC ($p > .999$). The subjective stimulus values, and with them the PEs, could thus be calculated less accurately in the PSFC compared with the other conditions.

Table A3.1. Statistical Comparison of the Three Other Models to Model 2

Model	*AIC*	*Mean Difference to M_2*	*df*	*t*	*p*
M_2	448.74				
M_1	490.28	−21.77	39	−4.08	**<.001**
M_3	473.04	−12.15	39	−4.97	**<.001**
M_4	521.35	−32.31	39	−4.43	**<.001**

Table A4.1. *b* Values, Confidence Intervals, and *t*-test Results for the GLME Analysis on Accuracy

Effects	*b*	*SE*	*z*	*p*	*2.5% CI*	*97.5% CI*
Intercept				**<.001**	0.79	1.22
Block	0.50	0.14	3.46	**<.001**	0.21	0.77
Association type	0.71	0.23	3.04	**.002**	0.25	1.21
Delayed feedback without tone	0.01	0.13	0.12	.907	−0.22	0.26
Delayed feedback with tone	0.06	0.09	0.68	.497	−0.10	0.24
Block × Association Type	0.36	0.29	1.25	.212	−0.20	0.89
Block × Delayed Feedback Without Tone	0.18	0.18	0.95	.343	−0.29	0.64
Block × Delayed Feedback With Tone	0.09	0.25	0.35	.725	−0.47	0.58
Association Type × Delayed Feedback Without Tone	−0.02	0.25	−0.09	.926	−0.49	0.39
Association Type × Delayed Feedback With Tone	0.04	0.19	0.23	.819	−0.40	0.36
Block × Association Type × Delayed Feedback Without Tone	−0.68	0.36	−1.89	*.059*	−1.38	0.18
Block × Association Type × Delayed Feedback With Tone	−0.33	0.50	−0.66	.510	−1.37	1.00

The sign of the *b*-estimates indicates the direction of main effects for the fixed-effects predictors block (1 [−0.5], 2 [−0.167], 3 [0.167], 4 [0.5]), association type (AFC [−0.5] vs. ASFC [0.5]), and feedback timing (simple coding contrast matrix with immediate feedback set as baseline that is compared with delayed feedback with tone and delayed feedback without tone).

Table A4.2. *b* Values, Confidence Intervals, and *t*-test Results of the LME Analysis on the FRN/RewP Amplitude

Effects	*b*	*SE*	*df*	*t*	*p*	*2.5% CI*	*97.5% CI*
Intercept	2.67	0.32	38.56	8.31	**<.001**	2.06	3.30
Association type	−0.82	0.64	38.56	−1.28	.207	−2.12	0.39
Delayed feedback without tone	0.29	0.35	40.94	0.84	.407	−0.45	0.87
Delayed feedback with tone	0.70	0.34	41.31	2.07	**.045**	0.04	1.36
Feedback valence	1.58	0.13	53.27	11.71	**<.001**	1.32	1.81
PE	0.59	0.15	6695.48	3.80	**<.001**	0.28	0.89
Association Type × Delayed Feedback Without Tone	0.56	0.70	40.94	0.80	.427	−0.81	1.85
Association Type × Delayed Feedback With Tone	−0.69	0.68	41.31	−1.01	.316	−1.97	0.70
Association Type × Feedback Valence	−0.48	0.27	53.27	−1.78	*.080*	−1.04	0.07
Delayed Feedback Without Tone × Feedback Valence	−0.68	0.23	17796.59	−3.00	**.003**	−1.15	−0.24
Delayed Feedback With Tone × Feedback Valence	−0.30	0.23	17798.47	−1.30	.192	−0.77	0.12
Association Type × PE	−0.60	0.31	6695.48	−1.94	*.052*	−1.24	−0.01
Delayed Feedback Without Tone × PE	−0.55	0.37	17747.66	−1.49	.136	−1.30	0.15
Delayed Feedback With Tone × PE	−0.31	0.36	17781.7	−0.86	.388	−1.04	0.40
Feedback Valence × PE	2.52	0.32	17851.58	7.86	**<.001**	1.94	3.18
Association Type × Delayed Feedback Without Tone × Feedback Valence	−0.53	0.46	17796.59	−1.16	.246	−1.49	0.31
Association Type × Delayed Feedback With Tone × Feedback Valence	−0.25	0.46	17798.47	−0.55	.584	−1.19	0.55
Association Type × Delayed Feedback Without Tone × PE	−1.11	0.74	17747.66	−1.51	.130	−2.53	0.41
Association Type × Delayed Feedback With Tone × PE	−1.05	0.73	17781.70	−1.44	.149	−2.57	0.39
Association Type × Feedback Valence × PE	1.42	0.64	17851.58	2.20	**.028**	0.21	2.83
Delayed Feedback Without Tone x Feedback Valence × PE	−0.74	0.78	17165.48	−0.95	.343	−2.32	0.82
Delayed Feedback With Tone × Feedback Valence × PE	−1.92	0.77	16844.95	−2.49	**.013**	−3.46	−0.33
Association Type × Delayed Feedback Without Tone × Feedback Valence × PE	0.27	1.56	17165.48	0.18	.861	−2.48	3.30
Association Type × Delayed Feedback With Tone × Feedback Valence × PE	−1.24	1.55	16844.95	−0.80	.422	−4.15	1.90

The sign of the *b*-estimates indicates the direction of main effects for the fixed-effects predictors association type (AFC [−0.5] vs. ASFC [0.5]), feedback timing (simple coding contrast matrix with immediate feedback set as baseline that is compared with delayed feedback with tone and delayed feedback without tone), feedback valence (negative [−0.5] vs. positive [0.5]), and mean centered unsigned PE.

Table A4.3. *b* Values, Confidence Intervals, and *t*-test Results for the LME Analysis on the N170 Amplitude

Effects	*b*	*SE*	*df*	*t*	*p*	*2.5% CI*	*97.5% CI*
Intercept	−6.33	0.59	2.87	−10.80	**.002**	−7.47	−5.09
Association type	0.81	0.76	38.30	1.08	.289	−0.71	2.26
Delayed feedback without tone	−0.28	0.32	40.47	−0.88	.384	−0.95	0.33
Delayed feedback with tone	−1.22	0.32	40.75	−3.82	**<.001**	−1.82	−0.64
Feedback valence	0.62	0.17	44.32	3.61	**.001**	0.28	0.97
PE	−0.21	0.13	21005.49	−1.61	.107	−0.47	0.05
Association Type × Delayed Feedback Without Tone	−0.77	0.64	40.47	−1.20	.239	−2.00	0.58
Association Type × Delayed Feedback With Tone	−0.69	0.64	40.75	−1.08	.285	−1.96	0.73
Association Type × Feedback Valence	−0.24	0.34	44.32	−0.69	.492	−0.90	0.49
Delayed Feedback Without Tone × Feedback Valence	0.53	0.19	35716.30	2.75	**.006**	0.11	0.93
Delayed Feedback With Tone × Feedback Valence	0.07	0.20	35714.45	0.33	.738	−0.32	0.47
Association Type × PE	−0.09	0.27	21005.60	−0.34	.732	−0.69	0.44
Delayed Feedback Without Tone × PE	0.84	0.31	35684.79	2.71	**.007**	0.23	1.44
Delayed Feedback With Tone × PE	0.22	0.31	35681.95	0.73	.467	−0.38	0.86
Feedback Valence × PE	−1.57	0.27	35460.14	−5.77	**<.001**	−2.09	−1.05
Association Type × Delayed Feedback Without Tone × Feedback Valence	−0.37	0.39	35716.30	−0.95	.340	−1.18	0.35
Association Type × Delayed Feedback With Tone × Feedback Valence	−0.72	0.39	35714.45	−1.83	.067	−1.45	0.09
Association Type × Delayed Feedback Without Tone × PE	0.50	0.62	35684.79	0.80	.421	−0.81	1.75
Association Type × Delayed Feedback With Tone × PE	0.32	0.62	35681.94	0.53	.599	−0.97	1.51
Association Type × Feedback Valence × PE	−1.48	0.54	35460.08	−2.72	**.007**	−2.54	−0.46
Delayed Feedback Without Tone × Feedback Valence × PE	0.24	0.65	27098.51	0.37	.711	−0.92	1.40
Delayed Feedback With Tone × Feedback Valence × PE	0.48	0.65	27326.96	0.74	.461	−0.78	1.70
Association Type × Delayed Feedback Without Tone × Feedback Valence × PE	−2.39	1.31	27098.28	−1.83	.068	−4.90	0.22
Association Type × Delayed Feedback With Tone × Feedback Valence × PE	−3.64	1.30	27326.66	−2.79	**.005**	−6.14	−1.09

The sign of the *b*-estimates indicates the direction of main effects for the fixed-effects predictors association type (AFC [−0.5] vs. ASFC [0.5]), feedback timing (simple coding contrast matrix with immediate feedback set as baseline that is compared with delayed feedback with tone and delayed without tone), feedback valence (negative [−0.5] vs. positive [0.5]), and mean centered unsigned PE.

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Data Availability Statement

The study was preregistered on https://doi.org/10.17605/OSF.IO/GVMWP. All data supporting the findings are openly accessible through the Open Science Framework at https://doi.org/10.17605/OSF.IO/E6X9G.

Author Contributions

Madita Röhlinger: Data curation; Formal analysis; Investigation; Methodology; Project administration; Visualization; Writing—Original draft. Christine Albrecht: Data curation; Formal analysis; Investigation; Methodology; Project administration; Software; Visualization; Writing—Original draft. Marta Ghio: Methodology; Project administration; Resources; Supervision; Validation; Writing—Review & editing. Christian Bellebaum: Funding acquisition; Methodology; Project administration; Resources; Supervision; Validation; Writing—Review & editing.

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Diversity in Citation Practices

Retrospective analysis of the citations in every article published in this journal from 2010 to 2021 reveals a persistent pattern of gender imbalance: Although the proportions of authorship teams (categorized by estimated gender identification of first author/last author) publishing in the *Journal of Cognitive Neuroscience* (*JoCN*) during this period were M(an)/M = .407, W(oman)/M = .32, M/W = .115, and W/W = .159, the comparable proportions for the articles that these authorship teams cited were M/M = .549, W/M = .257, M/W = .109, and W/W = .085 (Postle and Fulvio, *JoCN*, 34:1, pp. 1–3). Consequently, *JoCN* encourages all authors to consider gender balance explicitly when selecting which articles to cite and gives them the opportunity to report their article's gender citation balance. The authors of this paper report its proportions of citations by gender category to be: M/M = .570; W/M = .244; M/W = .058; W/W = .128.

REFERENCES

Albrecht, C., van de Vijver, R., & Bellebaum, C. (2023). Learning new words via feedback-association between feedback-locked ERPs and recall performance—An exploratory study. *Psychophysiology*, *60*, e14324. https://doi.org/10.1111/psyp.14324, PubMed: 37144796

Arbel, Y., Hong, L., Baker, T. E., & Holroyd, C. B. (2017). It's all about timing: An electrophysiological examination of feedback-based learning with immediate and delayed feedback. *Neuropsychologia*, *99*, 179–186. https://doi.org/10.1016/j.neuropsychologia.2017.03.003, PubMed: 28284986

Aubert, I., Ghorayeb, I., Normand, E., & Bloch, B. (2000). Phenotypical characterization of the neurons expressing the D1 and D2 dopamine receptors in the monkey striatum. *Journal of Comparative Neurology*, *418*, 22–32. PubMed: 10701753

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, *67*, 1–48. https://doi.org/10.18637/jss.v067.i01

Becker, M. P. I., Nitsch, A. M., Miltner, W. H. R., & Straube, T. (2014). A single-trial estimation of the feedback-related negativity and its relation to BOLD responses in a time-estimation task. *Journal of Neuroscience*, *34*, 3005–3012. https://doi.org/10.1523/JNEUROSCI.3684-13.2014, PubMed: 24553940

Bellebaum, C., Brodmann, K., & Thoma, P. (2014). Active and observational reward learning in adults with autism spectrum disorder: Relationship with empathy in an atypical sample. *Cognitive Neuropsychiatry*, *19*, 205–225. https://doi.org/10.1080/13546805.2013.823860, PubMed: 23998722

Bellebaum, C., & Colosio, M. (2014). From feedback- to response-based performance monitoring in active and observational learning. *Journal of Cognitive Neuroscience*, *26*, 2111–2127. https://doi.org/10.1162/jocn_a_00612, PubMed: 24666168

Bellebaum, C., & Daum, I. (2008). Learning-related changes in reward expectancy are reflected in the feedback-related negativity. *European Journal of Neuroscience*, *27*, 1823–1835. https://doi.org/10.1111/j.1460-9568.2008.06138.x, PubMed: 18380674

Bellebaum, C., Jokisch, D., Gizewski, E. R., Forsting, M., & Daum, I. (2012). The neural coding of expected and unexpected monetary performance outcomes: Dissociations between active and observational learning. *Behavioural Brain Research*, *227*, 241–251. https://doi.org/10.1016/j.bbr.2011.10.042, PubMed: 22074898

Bentin, S., Allison, T., Puce, A., Perez, E., & McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *Journal of Cognitive Neuroscience*, *8*, 551–565. https://doi.org/10.1162/jocn.1996.8.6.551, PubMed: 20740065

Brain Products GmbH. (2018). *BrainVision analyzer* (Version 2.2) [Computer software]. Gilching, Germany: Brain Products GmbH.

Burnside, R., Fischer, A. G., & Ullsperger, M. (2019). The feedback-related negativity indexes PE in active but not observational learning. *Psychophysiology*, *56*, e13389. https://doi.org/10.1111/psyp.13389

Calabresi, P., Picconi, B., Tozzi, A., Ghiglieri, V., & Di Filippo, M. (2014). Direct and indirect pathways of basal ganglia: A critical reappraisal. *Nature Neuroscience*, *17*, 1022–1030. https://doi.org/10.1038/nn.3743, PubMed: 25065439

Carlson, J. M., Foti, D., Mujica-Parodi, L. R., Harmon-Jones, E., & Hajcak, G. (2011). Ventral striatal and medial prefrontal BOLD activation is correlated with reward-related electrocortical activity: A combined ERP and fMRI study. *Neuroimage*, *57*, 1608–1616. https://doi.org/10.1016/j.neuroimage.2011.05.037, PubMed: 21624476

Cavanagh, J. F., Eisenberg, I., Guitart-Masip, M., Huys, Q., & Frank, M. J. (2013). Frontal theta overrides pavlovian learning biases. *Journal of Neuroscience*, *33*, 8541–8548. https://doi.org/10.1523/JNEUROSCI.5754-12.2013, PubMed: 23658191

Chuhma, N., Oh, S. J., & Rayport, S. (2023). The dopamine neuron synaptic map in the striatum. *Cell Reports*, *42*, 112204. https://doi.org/10.1016/j.celrep.2023.112204, PubMed: 36867530

Cohen, M. X., & Ranganath, C. (2007). Reinforcement learning signals predict future decisions. *Journal of Neuroscience*, *27*, 371–378. https://doi.org/10.1523/JNEUROSCI.4421-06.2007, PubMed: 17215398

Cooper, J. C., Dunne, S., Furey, T., & O'Doherty, J. P. (2012). Human dorsal striatum encodes prediction errors during observational learning of instrumental actions. *Journal of Cognitive Neuroscience*, *24*, 106–118. https://doi.org/10.1162/jocn_a_00114, PubMed: 21812568

Daniel, R., & Pollmann, S. (2014). A universal role of the ventral striatum in reward-based learning: Evidence from human studies. *Neurobiology of Learning and Memory*, *114*, 90–100. https://doi.org/10.1016/j.nlm.2014.05.002, PubMed: 24825620

Deffke, I., Sander, T., Heidenreich, J., Sommer, W., Curio, G., Trahms, L., et al. (2007). MEG/EEG sources of the 170-ms response to faces are co-localized in the fusiform gyrus. *Neuroimage*, *35*, 1495–1501. https://doi.org/10.1016/j.neuroimage.2007.01.034, PubMed: 17363282

Dickerson, K. C., Li, J., & Delgado, M. R. (2011). Parallel contributions of distinct human memory systems during probabilistic learning. *Neuroimage*, *55*, 266–276. https://doi.org/10.1016/j.neuroimage.2010.10.080, PubMed: 21056678

Eichenbaum, H., Otto, T., & Cohen, N. J. (1992). The hippocampus—What does it do? *Behavioral and Neural Biology*, *57*, 2–36. https://doi.org/10.1016/0163-1047(92)90724-I, PubMed: 1567331

Fischer, A. G., & Ullsperger, M. (2013). Real and fictive outcomes are processed differently but converge on a common adaptive mechanism. *Neuron*, *79*, 1243–1255. https://doi.org/10.1016/j.neuron.2013.07.006, PubMed: 24050408

Foerde, K., Race, E., Verfaellie, M., & Shohamy, D. (2013). A role for the medial temporal lobe in feedback-driven learning: Evidence from amnesia. *Journal of Neuroscience*, *33*, 5698–5704. https://doi.org/10.1523/JNEUROSCI.5217-12.2013, PubMed: 23536083

Foerde, K., & Shohamy, D. (2011). Feedback timing modulates brain systems for learning in humans. *Journal of Neuroscience*, *31*, 13157–13167. https://doi.org/10.1523/JNEUROSCI.2701-11.2011, PubMed: 21917799

Foti, D., Weinberg, A., Dien, J., & Hajcak, G. (2011). Event-related potential activity in the basal ganglia differentiates rewards from nonrewards: Temporospatial principal components analysis and source localization of the feedback negativity. *Human Brain Mapping*, *32*, 2207–2216. https://doi.org/10.1002/hbm.21182, PubMed: 21305664

Frank, M. J., Seeberger, L. C., & O'reilly, R. C. (2004). By carrot or by stick: Cognitive reinforcement learning in parkinsonism. *Science*, *306*, 1940–1943. https://doi.org/10.1126/science.1102941, PubMed: 15528409

Fu, W.-T., & Anderson, J. R. (2008). Solving the credit assignment problem: Explicit and implicit learning of action sequences with probabilistic outcomes. *Psychological Research*, *72*, 321–330. https://doi.org/10.1007/s00426-007-0113-7, PubMed: 17447083

Fukushima, H., & Hiraki, K. (2009). Whose loss is it? Human electrophysiological correlates of non-self reward processing. *Social Neuroscience*, *4*, 261–275. https://doi.org/10.1080/17470910802625009, PubMed: 19191194

Gao, C., Conte, S., Richards, J. E., Xie, W., & Hanayik, T. (2019). The neural sources of N170: Understanding timing of activation in face-selective areas. *Psychophysiology*, *56*, e13336. https://doi.org/10.1111/psyp.13336, PubMed: 30710345

Gauthier, J. L., & Tank, D. W. (2018). A dedicated population for reward coding in the hippocampus. *Neuron*, *99*, 179–193. https://doi.org/10.1016/j.neuron.2018.06.008, PubMed: 30008297

Gerfen, C. R. (1992). The neostriatal mosaic: Multiple levels of compartmental organization in the basal ganglia. *Annual Review of Neuroscience*, *15*, 285–320. https://doi.org/10.1146/annurev.ne.15.030192.001441, PubMed: 1575444

Guitart-Masip, M., Duzel, E., Dolan, R., & Dayan, P. (2014). Action versus valence in decision making. *Trends in Cognitive Sciences*, *18*, 194–202. https://doi.org/10.1016/j.tics.2014.01.003, PubMed: 24581556

Guitart-Masip, M., Fuentemilla, L., Bach, D. R., Huys, Q. J. M., Dayan, P., Dolan, R. J., et al. (2011). Action dominates valence in anticipatory representations in the human striatum and dopaminergic midbrain. *Journal of Neuroscience*, *31*, 7867–7875. https://doi.org/10.1523/JNEUROSCI.6376-10.2011, PubMed: 21613500

Guitart-Masip, M., Huys, Q. J. M., Fuentemilla, L., Dayan, P., Duzel, E., & Dolan, R. J. (2012). Go and no-go learning in reward and punishment: Interactions between affect and effect. *Neuroimage*, *62*, 154–166. https://doi.org/10.1016/j.neuroimage.2012.04.024, PubMed: 22548809

Haber, S. N., & Knutson, B. (2010). The reward circuit: Linking primate anatomy and human imaging. *Neuropsychopharmacology*, *35*, 4–26. https://doi.org/10.1038/npp.2009.129, PubMed: 19812543

Hajcak, G., Moser, J. S., Holroyd, C. B., & Simons, R. F. (2007). It's worse than you thought: The feedback negativity and violations of reward prediction in gambling tasks. *Psychophysiology*, *44*, 905–912. https://doi.org/10.1111/j.1469-8986.2007.00567.x, PubMed: 17666029

Haruno, M., & Kawato, M. (2006). Different neural correlates of reward expectation and reward expectation error in the putamen and caudate nucleus during stimulus–action–reward association learning. *Journal of Neurophysiology*, *95*, 948–959. https://doi.org/10.1152/jn.00382.2005, PubMed: 16192338

Hernández-López, S., Bargas, J., Surmeier, D. J., Reyes, A., & Galarraga, E. (1997). D1 receptor activation enhances evoked discharge in neostriatal medium spiny neurons by modulating an L-type Ca2+ conductance. *Journal of Neuroscience*, *17*, 3334–3342. https://doi.org/10.1523/JNEUROSCI.17-09-03334.1997, PubMed: 9096166

Hernández-López, S., Tkatch, T., Perez-Garci, E., Galarraga, E., Bargas, J., Hamm, H., et al. (2000). D2 dopamine receptors in striatal medium spiny neurons reduce L-type Ca2+ currents and excitability via a novel PLCβ1–IP3–calcineurin-signaling cascade. *Journal of Neuroscience*, *20*, 8987–8995. https://doi.org/10.1523/JNEUROSCI.20-24-08987.2000, PubMed: 11124974

Hiebert, N. M., Vo, A., Hampshire, A., Owen, A. M., Seergobin, K. N., & MacDonald, P. A. (2014). Striatum in stimulus-response learning via feedback and in decision making. *Neuroimage*, *101*, 448–457. https://doi.org/10.1016/j.neuroimage.2014.07.013, PubMed: 25038436

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*, 679–709. https://doi.org/10.1037/0033-295X.109.4.679, PubMed: 12374324

Holroyd, C. B., Krigolson, O. E., & Lee, S. (2011). Reward positivity elicited by predictive cues. *NeuroReport*, *22*,

249–252. https://doi.org/10.1097/WNR.0b013e328345441d, PubMed: 21386699
Holroyd, C. B., Larsen, J. T., & Cohen, J. D. (2004). Context dependence of the event-related brain potential associated with reward and punishment. *Psychophysiology*, *41*, 245–253. https://doi.org/10.1111/j.1469-8986.2004.00152.x, PubMed: 15032989
Höltje, G., & Mecklinger, A. (2020). Feedback timing modulates interactions between feedback processing and memory encoding: Evidence from event-related potentials. *Cognitive, Affective, & Behavioral Neuroscience*, *20*, 250–264. https://doi.org/10.3758/s13415-019-00765-5, PubMed: 31900874
Jocham, G., Brodersen, K. H., Constantinescu, A. O., Kahn, M. C., Ianni, A. M., Walton, M. E., et al. (2016). Reward-guided learning with and without causal attribution. *Neuron*, *90*, 177–190. https://doi.org/10.1016/j.neuron.2016.02.018, PubMed: 26971947
Kim, S., & Arbel, Y. (2019). Immediate and delayed auditory feedback in declarative learning: An examination of the feedback related event related potentials. *Neuropsychologia*, *129*, 255–262. https://doi.org/10.1016/j.neuropsychologia.2019.04.001, PubMed: 30981647
Kimura, K., & Kimura, M. (2016). Temporal prediction restores the evaluative processing of delayed action feedback: An electrophysiological study. *NeuroReport*, *27*, 1061–1067. https://doi.org/10.1097/WNR.0000000000000657, PubMed: 27513200
Kirsch, F., Kirschner, H., Fischer, A. G., Klein, T. A., & Ullsperger, M. (2022). Disentangling performance-monitoring signals encoded in feedback-related EEG dynamics. *Neuroimage*, *257*, 119322. https://doi.org/10.1016/j.neuroimage.2022.119322, PubMed: 35577025
Kloth, N., Itier, R. J., & Schweinberger, S. R. (2013). Combined effects of inversion and feature removal on N170 responses elicited by faces and car fronts. *Brain and Cognition*, *81*, 321–328. https://doi.org/10.1016/j.bandc.2013.01.002, PubMed: 23485023
Knowlton, B. J., Mangels, J. A., & Squire, L. R. (1996). A neostriatal habit learning system in humans. *Science*, *273*, 1399–1402. https://doi.org/10.1126/science.273.5280.1399, PubMed: 8703077
Knowlton, B. J., & Patterson, T. K. (2016). Habit formation and the striatum. In R. E. Clark & S. Martin (Eds.), *Behavioral neuroscience of learning and memory* (Vol. 37, pp. 275–293). Springer. https://doi.org/10.1007/7854_2016_451
Kobza, S., Ferrea, S., Schnitzler, A., Pollok, B., Südmeyer, M., & Bellebaum, C. (2012). Dissociation between active and observational learning from positive and negative feedback in Parkinsonism. *PLoS One*, *7*, e50250. https://doi.org/10.1371/journal.pone.0050250, PubMed: 23185586
Lisman, J. E., & Grace, A. A. (2005). The hippocampal-VTA loop: Controlling the entry of information into long-term memory. *Neuron*, *46*, 703–713. https://doi.org/10.1016/j.neuron.2005.05.002, PubMed: 15924857
Lukie, C. N., Montazer-Hojat, S., & Holroyd, C. B. (2014). Developmental changes in the reward positivity: An electrophysiological trajectory of reward processing. *Developmental Cognitive Neuroscience*, *9*, 191–199. https://doi.org/10.1016/j.dcn.2014.04.003, PubMed: 24879113
Meteyard, L., & Davies, R. A. (2020). Best practice guidance for linear mixed-effects models in psychological science. *Journal of Memory and Language*, *112*, 104092. https://doi.org/10.1016/j.jml.2020.104092
Miltner, W. H., Braun, C. H., & Coles, M. G. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience*, *9*, 788–798. https://doi.org/10.1162/jocn.1997.9.6.788, PubMed: 23964600
Myers, C. E., Shohamy, D., Gluck, M. A., Grossman, S., Kluger, A., Ferris, S., et al. (2003). Dissociating hippocampal versus basal ganglia contributions to learning and transfer. *Journal of Cognitive Neuroscience*, *15*, 185–193. https://doi.org/10.1162/089892903321208123, PubMed: 12676056
Neurobehavioral Systems Inc. (2020). *Presentation* (Version 22) [Computer software]. Albany, CA, USA: Neurobehavioral Systems Inc. https://www.neurobs.com/
Nikooyan, A. A., & Ahmed, A. A. (2015). Reward feedback accelerates motor learning. *Journal of Neurophysiology*, *113*, 633–646. https://doi.org/10.1152/jn.00032.2014, PubMed: 25355957
O'Doherty, J., Dayan, P., Schultz, J., Deichmann, R., Friston, K., & Dolan, R. J. (2004). Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science*, *304*, 452–454. https://doi.org/10.1126/science.1094285, PubMed: 15087550
Oldehinkel, M., Llera, A., Faber, M., Huertas, I., Buitelaar, J. K., Bloem, B. R., et al. (2022). Mapping dopaminergic projections in the human brain with resting-state fMRI. *eLife*, *11*, e71846. https://doi.org/10.7554/eLife.71846, PubMed: 35113016
Palombo, D. J., Hayes, S. M., Reid, A. G., & Verfaellie, M. (2019). Hippocampal contributions to value-based learning: Converging evidence from fMRI and amnesia. *Cognitive, Affective & Behavioral Neuroscience*, *19*, 523–536. https://doi.org/10.3758/s13415-018-00687-8, PubMed: 30767129
Paul, K., Vassena, E., Severo, M. C., & Pourtois, G. (2020). Dissociable effects of reward magnitude on fronto-medial theta and FRN during performance monitoring. *Psychophysiology*, *57*, e13481. https://doi.org/10.1111/psyp.13481, PubMed: 31578739
Peterburs, J., Albrecht, C., & Bellebaum, C. (2022). The impact of social anxiety on feedback-based go and nogo learning. *Psychological Research*, *86*, 110–124. https://doi.org/10.1007/s00426-021-01479-5, PubMed: 33527222
Peterburs, J., Kobza, S., & Bellebaum, C. (2016). Feedback delay gradually affects amplitude and valence specificity of the feedback-related negativity (FRN). *Psychophysiology*, *53*, 209–215. https://doi.org/10.1111/psyp.12560, PubMed: 26459164
Plachti, A., Eickhoff, S. B., Hoffstaedter, F., Patil, K. R., Laird, A. R., Fox, P. T., et al. (2019). Multimodal parcellations and extensive behavioral profiling tackling the hippocampus gradient. *Cerebral Cortex*, *29*, 4595–4612. https://doi.org/10.1093/cercor/bhy336, PubMed: 30721944
Pleger, B., Blankenburg, F., Ruff, C. C., Driver, J., & Dolan, R. J. (2008). Reward facilitates tactile judgments and modulates hemodynamic responses in human primary somatosensory cortex. *Journal of Neuroscience*, *28*, 8161–8168. https://doi.org/10.1523/JNEUROSCI.1093-08.2008, PubMed: 18701678
Pleger, B., Ruff, C. C., Blankenburg, F., Klöppel, S., Driver, J., & Dolan, R. J. (2009). Influence of dopaminergically mediated reward on somatosensory decision-making. *PLoS Biology*, *7*, e1000164. https://doi.org/10.1371/journal.pbio.1000164, PubMed: 19636360
Poldrack, R. A., Clark, J., Paré-Blagoev, E. J., Shohamy, D., Creso Moyano, J., Myers, C., et al. (2001). Interactive memory systems in the human brain. *Nature*, *414*, 546–550. https://doi.org/10.1038/35107080, PubMed: 11734855
Proudfit, G. H. (2015). The reward positivity: From basic research on reward to a biomarker for depression. *Psychophysiology*, *52*, 449–459. https://doi.org/10.1111/psyp.12370, PubMed: 25327938
Qin, S., Piekema, C., Petersson, K. M., Han, B., Luo, J., & Fernández, G. (2007). Probing the transformation of discontinuous associations into episodic memory: An

event-related fMRI study. *Neuroimage*, *38*, 212–222. https://doi.org/10.1016/j.neuroimage.2007.07.020, PubMed: 17804259
Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and non-reinforcement. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 64–99). Appleton-Century-Crofts.
Rossion, B. (2014). Understanding face perception by means of human electrophysiology. *Trends in Cognitive Sciences*, *18*, 310–318. https://doi.org/10.1016/j.tics.2014.02.013, PubMed: 24703600
Rossion, B., & Jacques, C. (2012). *The N170: Understanding the time course of face perception in the human brain*. Oxford University Press. https://doi.org/10.1093/oxfordhb/9780195374148.013.0064
Sambrook, T. D., & Goslin, J. (2015). A neural reward prediction error revealed by a meta-analysis of ERPs using great grand averages. *Psychological Bulletin*, *141*, 213–235. https://doi.org/10.1037/bul0000006, PubMed: 25495239
Schiffer, A.-M., Muller, T., Yeung, N., & Waszak, F. (2014). Reward activates stimulus-specific and task-dependent representations in visual association cortices. *Journal of Neuroscience*, *34*, 15610–15620. https://doi.org/10.1523/JNEUROSCI.1640-14.2014, PubMed: 25411489
Schott, B. H., Minuzzi, L., Krebs, R. M., Elmenhorst, D., Lang, M., Winz, O. H., et al. (2008). Mesolimbic functional magnetic resonance imaging activations during reward anticipation correlate with reward-related ventral striatal dopamine release. *Journal of Neuroscience*, *28*, 14311–14319. https://doi.org/10.1523/JNEUROSCI.2058-08.2008, PubMed: 19109512
Schott, B. H., Sellner, D. B., Lauer, C.-J., Habib, R., Frey, J. U., Guderian, S., et al. (2004). Activation of midbrain structures by associative novelty and the formation of explicit memory in humans. *Learning & Memory*, *11*, 383–387. https://doi.org/10.1101/lm.75004, PubMed: 15254215
Schultz, W. (2000). Multiple reward signals in the brain. *Nature Reviews Neuroscience*, *1*, 199–207. https://doi.org/10.1038/35044563, PubMed: 11257908
Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, *275*, 1593–1599. https://doi.org/10.1126/science.275.5306.1593, PubMed: 9054347
Schultz, W., & Dickinson, A. (2000). Neuronal coding of prediction errors. *Annual Review of Neuroscience*, *23*, 473–500. https://doi.org/10.1146/annurev.neuro.23.1.473, PubMed: 10845072
Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery & Psychiatry*, *20*, 11–21. https://doi.org/10.1136/jnnp.20.1.11, PubMed: 13406589
Shohamy, D. (2011). Learning and motivation in the human striatum. *Current Opinion in Neurobiology*, *21*, 408–414. https://doi.org/10.1016/j.conb.2011.05.009, PubMed: 21658933
Shohamy, D., & Adcock, R. A. (2010). Dopamine and adaptive memory. *Trends in Cognitive Sciences*, *14*, 464–472. https://doi.org/10.1016/j.tics.2010.08.002, PubMed: 20829095
Shohamy, D., Myers, C. E., Grossman, S., Sage, J., Gluck, M. A., & Poldrack, R. A. (2004). Cortico-striatal contributions to feedback-based learning: Converging data from neuroimaging and neuropsychology. *Brain*, *127*, 851–859. https://doi.org/10.1093/brain/awh100, PubMed: 15013954
Singer, A. C., & Frank, L. M. (2009). Rewarded outcomes enhance reactivation of experience in the hippocampus. *Neuron*, *64*, 910–921. https://doi.org/10.1016/j.neuron.2009.11.016, PubMed: 20064396
Squire, L. R., Shimamura, A. P., & Amaral, D. G. (1989). Memory and the hippocampus. In *Neural models of plasticity* (pp. 208–239). Academic Press. https://doi.org/10.1016/B978-0-12-148955-7.50016-3
Staresina, B. P., & Davachi, L. (2009). Mind the gap: Binding experiences across space and time in the human hippocampus. *Neuron*, *63*, 267–276. https://doi.org/10.1016/j.neuron.2009.06.024, PubMed: 19640484
Steiner, H., & Tseng, K. Y. (Eds.). (2016). *Handbook of basal ganglia structure and function*. Academic Press.
Stolyarova, A. (2018). Solving the credit assignment problem with the prefrontal cortex. *Frontiers in Neuroscience*, *12*, 182. https://doi.org/10.3389/fnins.2018.00182, PubMed: 29636659
Sugar, J., & Moser, M.-B. (2019). Episodic memory: Neuronal codes for what, where, and when. *Hippocampus*, *29*, 1190–1205. https://doi.org/10.1002/hipo.23132, PubMed: 31334573
The MathWorks, Inc. (2021). *MATLAB* (Version R2021a) [Computer software]. Natick, MA: The MathWorks, Inc.
The R Foundation. (2021). *R: A language and environment for statistical computing* (Version 4.2.2) [Computer software]. Vienna, Austria: R Foundation for Statistical Computing. https://www.R-project.org/
Tversky, A., & Kahneman, D. (1992). Advances in prospect theory: Cumulative representation of uncertainty. *Journal of Risk and Uncertainty*, *5*, 297–323. https://doi.org/10.1007/BF00122574
Vassiliadis, P., Beanato, E., Popa, T., Windel, F., Morishita, T., Neufeld, E., et al. (2024). Non-invasive stimulation of the human striatum disrupts reinforcement learning of motor skills. *Nature Human Behaviour*, *8*, 1581–1598. https://doi.org/10.1038/s41562-024-01901-z, PubMed: 38811696
Voeten, C. (2020). Using 'buildmer' to automatically find & compare maximal (mixed) models.
Weber, C., & Bellebaum, C. (2024). Prediction-error-dependent processing of immediate and delayed positive feedback. *Scientific Reports*, *14*, 9674. https://doi.org/10.1038/s41598-024-60328-8, PubMed: 38678065
Weinberg, A., Luhmann, C. C., Bress, J. N., & Hajcak, G. (2012). Better late than never? The effect of feedback delay on ERP indices of reward processing. *Cognitive, Affective, & Behavioral Neuroscience*, *12*, 671–677. https://doi.org/10.3758/s13415-012-0104-z, PubMed: 22752976
Weismüller, B., & Bellebaum, C. (2016). Expectancy affects the feedback-related negativity (FRN) for delayed feedback in probabilistic learning. *Psychophysiology*, *53*, 1739–1750. https://doi.org/10.1111/psyp.12738, PubMed: 27565454
Wessel, M. J., Beanato, E., Popa, T., Windel, F., Vassiliadis, P., Menoud, P., et al. (2023). Noninvasive theta-burst stimulation of the human striatum enhances striatal activity and motor skill learning. *Nature Neuroscience*, *26*, 2005–2016. https://doi.org/10.1038/s41593-023-01457-7, PubMed: 37857774
White, N. M., & McDonald, R. J. (2002). Multiple parallel memory systems in the brain of the rat. *Neurobiology of Learning and Memory*, *77*, 125–184. https://doi.org/10.1006/nlme.2001.4008, PubMed: 11848717
Yagishita, S., Hayashi-Takagi, A., Ellis-Davies, G. C. R., Urakubo, H., Ishii, S., & Kasai, H. (2014). A critical time window for dopamine actions on the structural plasticity of dendritic spines. *Science*, *345*, 1616–1620. https://doi.org/10.1126/science.1255514, PubMed: 25258080
Yovel, G. (2016). Neural and cognitive face-selective markers: An integrative review. *Neuropsychologia*, *83*, 5–13. https://doi.org/10.1016/j.neuropsychologia.2015.09.026, PubMed: 26407899
Zaghloul, K. A., Blanco, J. A., Weidemann, C. T., McGill, K., Jaggi, J. L., Baltuch, G. H., et al. (2009). Human substantia

nigra neurons encode unexpected financial rewards. *Science*, *323*, 1496–1499. https://doi.org/10.1126/science.1167342, PubMed: 19286561

Zeithamova, D., Gelman, B. D., Frank, L., & Preston, A. R. (2018). Abstract representation of prospective reward in the hippocampus. *Journal of Neuroscience*, *38*, 10093–10101. https://doi.org/10.1523/JNEUROSCI.0719-18.2018, PubMed: 30282732

Zhang, Y., Wu, I.-W., Buckley, S., Coffey, C. S., Foster, E., Mendick, S., et al. (2015). Diffusion tensor imaging of the nigrostriatal fibers in Parkinson's disease. *Movement Disorders*, *30*, 1229–1236. https://doi.org/10.1002/mds.26251, PubMed: 25920732

Zottoli, T. M., & Grose-Fifer, J. (2012). The feedback-related negativity (FRN) in adolescents. *Psychophysiology*, *49*, 413–420. https://doi.org/10.1111/j.1469-8986.2011.01312.x, PubMed: 22091835

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The Role of the N170 in Linking Stimuli to Feedback—Effects of Stimulus Modality and Feedback Delay

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ABSTRACT

With increasing feedback delay, feedback processing appears to shift from the striatum to the hippocampus. In addition, higher-order sensory areas might be involved in bridging a temporal gap between stimulus and feedback by reactivating the representation of the feedback-predicting stimulus during feedback processing. We hypothesized that the feedback-locked N170, an occipito-temporal event-related potential (ERP) component linked to higher-order visual processing, is more pronounced when delayed feedback is provided for choices between visual compared to auditory stimuli. 35 subjects completed a probabilistic feedback learning task with immediate (1 s) and delayed (7 s) monetary feedback for choices between visual or auditory stimuli. Participants successfully learned to choose the more rewarding stimuli irrespective of stimulus modality. For the N170 amplitude over the right hemisphere, we found an interaction between feedback timing and the modality of the chosen stimulus. Only for delayed feedback, the N170 was more pronounced for choices between visual than auditory stimuli. Moreover, in this condition, the N170 amplitude particularly reflected the reward prediction error (PE), with larger amplitudes for positive PEs and lower amplitudes for negative PEs. This suggests that the N170 reflects feedback-locked reactivations in higher-order visual areas mediated by the reward PE. While these effects need to be studied further, we discuss the N170 as a counterpart to the feedback-related negativity (FRN) regarding interacting influences of feedback valence, feedback timing, and PE.

1 | Introduction

Numerous studies underpin the involvement of a dopaminergic, striatal, mesocorticolimbic reward system in processing performance feedback, that is, when human study participants receive positive or negative outcomes for their choice actions (for reviews, see Delgado 2007; Haber and Knutson 2010; Wang et al. 2016). However, neural mechanisms involved in feedback processing are affected by the temporal proximity of an action and its outcome (Jocham et al. 2016). A study by Foerde and Shohamy (2011) underlined the role of striatal activity in processing immediate feedback but found pronounced hippocampal activity in processing delayed feedback (after a couple of seconds). Causal inferences concerning the neural mechanisms of processing immediate and delayed feedback could be drawn from lesion studies: Parkinson's disease patients suffering from striatal dysfunctions (Damier et al. 1999) had problems learning from immediate, but not from delayed feedback (Foerde and Shohamy 2011). Conversely, amnestic patients with presumed lesions in the medial temporal lobe (MTL) including the hippocampus had problems learning from delayed, but not from immediate feedback (Foerde et al. 2013). Staresina and Davachi (2009) suggested that the role of the hippocampus is to bind representations separated

by space and time to bridge gaps in our experience. Several researchers have suggested that, in the absence of immediate feedback to the striatum, the MTL may be recruited to bind an individual's response with the delayed feedback, despite their separation in time (Arbel et al. 2017; Foerde et al. 2013; Peterburs et al. 2016).

In studies assessing neural feedback processing by means of electroencephalography (EEG), delays have been found to differentially affect two event-related potential (ERP) components that have been associated with the reward system and the MTL, respectively (Arbel et al. 2017; Höltje and Mecklinger 2020; Kim and Arbel 2019; Peterburs et al. 2016). The feedback-related negativity (FRN) peaks around 250 to 300 ms after feedback presentation at frontocentral electrode sites and is more pronounced for negative than positive feedback (Becker et al. 2014; Bellebaum and Daum 2008; Foti et al. 2011; Holroyd and Coles 2002; Miltner et al. 1997; Nieuwenhuis et al. 2004), possibly because a positive component referred to as Reward Positivity (RewP; for a review see Proudfit 2015) drives the signal toward positive amplitudes for positive feedback. The amplitude of the signal in the FRN/RewP time window reflects a prediction error (PE) that indicates whether feedback is better or worse than expected (Burnside et al. 2019; Fischer and Ullsperger 2013; Kirsch et al. 2022; Sambrook and Goslin 2015; Weber and Bellebaum 2024). A PE is encoded by midbrain dopaminergic neurons, for instance in the substantia nigra (Schultz et al. 1997; Zaghloul et al. 2009), suggesting that the FRN indirectly reflects activity of the midbrain dopamine system (Foti et al. 2015; Hauser et al. 2014; Holroyd and Coles 2002). Williams et al. (2020) provide evidence that the FRN reflects an underlying learning process that drives behavioral adaptation based on PEs. Having been linked to striatal activity (Becker et al. 2014; Carlson et al. 2011; Foti et al. 2011), and thus a dopamine projection site (Chuhma et al. 2023; Oldehinkel et al. 2022; Zhang et al. 2015), the FRN difference wave for negative –positive feedback better differentiates feedback valence when feedback is presented immediately (Arbel et al. 2017; Höltje and Mecklinger 2020; Peterburs et al. 2016; Weinberg et al. 2012; Weismüller and Bellebaum 2016). Evidence suggests, however, that the FRN is not directly generated by the striatum (Cohen et al. 2011), but by the medial prefrontal cortex, more specifically, the anterior cingulate cortex (Nieuwenhuis et al. 2005; Hauser et al. 2014; Becker et al. 2014; Oerlemans et al. 2025), which in turn receives projections from the striatum (Chau et al. 2018; Hauser et al. 2014).

In contrast, the N170, a negative deflection about 170 ms after visual stimulus presentation at lateral temporal electrode sites (Bentin et al. 1996), was repeatedly found to be more pronounced for delayed than immediate feedback (Arbel et al. 2017; Höltje and Mecklinger 2020; Kim and Arbel 2019; but see Albrecht et al. 2023, for the opposite pattern). Arbel et al. (2017) and Kim and Arbel (2019) hypothesized that the N170 is generated by a delayed reward signal to reinforce a memory representation of a stimulus stored in the MTL. In this line, Baker and Holroyd (2009) demonstrated that the spatial location of feedback stimuli elicited a pronounced N170 response associated with right MTL activation in a navigational feedback learning task. In subsequent studies, Baker and Holroyd (2013) and Baker et al. (2015) localized the N170 in this task to the right parahippocampal region, proposing that the parahippocampal cortex encodes salient information essential for spatial navigation.

With the present work we aim to investigate an alternative explanation regarding larger N170 amplitudes for delayed feedback: The N170 is usually investigated in the context of higher visual processing, being particularly pronounced for faces (Bentin et al. 1996; Itier and Taylor 2004; for a review see Yovel 2016) and words (for a review see Carreiras et al. 2014), but also cars (Kloth et al. 2013). For faces and words, an origin in the fusiform gyrus was found (Brem et al. 2006; Deffke et al. 2007; Gao et al. 2019; Iidaka et al. 2006), which contains specialized regions for diverse stimulus categories (Cohen et al. 2002; Kanwisher et al. 1997; for an overview see Weiner and Zilles 2016). Thus, a pronounced N170 after delayed feedback may indicate the activation of higher-order visual areas during the processing of (delayed) feedback, possibly mediated by the MTL.

If feedback is delayed, a reactivation of the representations of the associated stimulus might be the mechanism to bridge the temporal gap between stimulus and feedback. Support for this assumption comes from several fMRI studies: For example, participants in a study by Pleger et al. (2008) had to discriminate somatosensory stimuli regarding their frequency (high vs. low) and were rewarded for correct judgments. Notably, the primary somatosensory cortex was reactivated when reward was presented, an effect mediated by dopamine (Pleger et al. 2009). In a study by Schiffer et al. (2014), reward activated stimulus-category-specific representations of reward-associated stimuli in visual association cortices.

In the present study, we want to examine whether the N170 for delayed feedback represents a reactivation of a previously selected visual stimulus to bridge the temporal gap and assign credit to the stimulus. To test this, we manipulate the modality of the stimuli between which participants have to choose in a feedback learning task. More specifically, participants receive visual feedback for choices between two visual or two auditory stimuli. We hypothesize that the N170 has a larger amplitude when the feedback is associated with visual than with auditory stimuli and that this effect is stronger for delayed compared to immediate feedback. Given that the right hemisphere plays a dominant role in processing certain visual stimuli, such as faces (Rossion 2014), and in N170 generation in different contexts (Baker and Holroyd 2009; Baker and Holroyd 2013; Baker et al. 2015; Kim and Arbel 2019), we were particularly interested in whether the effects would be stronger over the right hemisphere. In addition, we explore whether the PE is represented in the N170, possibly depending on stimulus modality, feedback timing, and hemisphere. For this purpose, we will model trial-by-trial fluctuations of the PE using the behavioral learning data. Given that the hippocampus shows PE-related activity (Dickerson et al. 2011) and that the N170 may be mediated by MTL processing, it is conceivable that the PE is reflected in the N170 amplitude, especially following delayed feedback for the choice between visual stimuli and over the right hemisphere. Regarding the FRN, we aimed to replicate previous effects for PE coding and

effects of the timing of feedback and explore effects of the modality of the stimulus associated with the feedback in interaction with these factors, without a specific hypothesis.

2 | Method

2.1 | Participants

The sample size was planned a priori and based on the number of participants in previous studies investigating the effects of feedback timing on FRN and N170: Arbel et al. (2017) found a significant effect of feedback timing on the N170 in a study with 21 subjects. In the planned study, we were particularly interested in the interaction between feedback timing and stimulus modality and also in higher-order interactions (see Data Analysis for details), which suggests that a larger sample size was needed to reach adequate power. We thus preregistered to recruit 40 healthy young adults (18–40 years) for participation in the experiment. Exclusion criteria were a history of neurological or psychiatric disorders, the regular or acute consumption of substances affecting the central nervous system, knowledge about Hiragana-Characters, uncorrected impaired vision, and impaired hearing. Of 40 acquired participants, we excluded five participants, three of them because they fulfilled at least one of our exclusion criteria, one because of bad EEG data quality due to alpha waves, and one due to technical problems. The final sample included in the analyses thus consisted of 35 participants, 30 women and 5 men, 2 left-handed and 33 right-handed. The mean age was 23.2 years (SD = 4.5 years, Min = 19 years, Max = 35 years).

2.2 | Experimental Task and Conditions

Participants underwent a probabilistic feedback learning task, in which they could learn associations between stimuli and positive or negative monetary feedback (feedback valence: +4 ct vs. −2 ct). The task comprised the two within-subject factors Stimulus Modality and Feedback Timing: On every trial, each participant could choose between two stimuli. In half of the trials of the experiment, the choice was between two visual stimuli; in the other half of the trials, the choice was between two auditory stimuli (factor Stimulus Modality). Figure 1A shows an exemplary trial for the choice between visual and Figure 1B for the choice between auditory stimuli. Furthermore, feedback appeared 1 s (immediate feedback) or 7 s (delayed feedback) after participants' choice and was always presented visually on the screen (factor Feedback Timing). Participants completed four learning phases with stimuli of one modality (either visual or auditory) before switching to stimuli of the other modality, again for four learning phases, with the order of modalities counterbalanced across participants. In each learning phase, a new stimulus pair was presented, and there were thus eight stimulus pairs in total, four visual and four auditory pairs. Feedback timing (immediate or delayed) remained consistent throughout the phase. The feedback timing changed only at the beginning of a new learning phase, coinciding with the presentation of a new stimulus pair. Thus, feedback timing varied across phases, with the starting condition counterbalanced across participants. Each

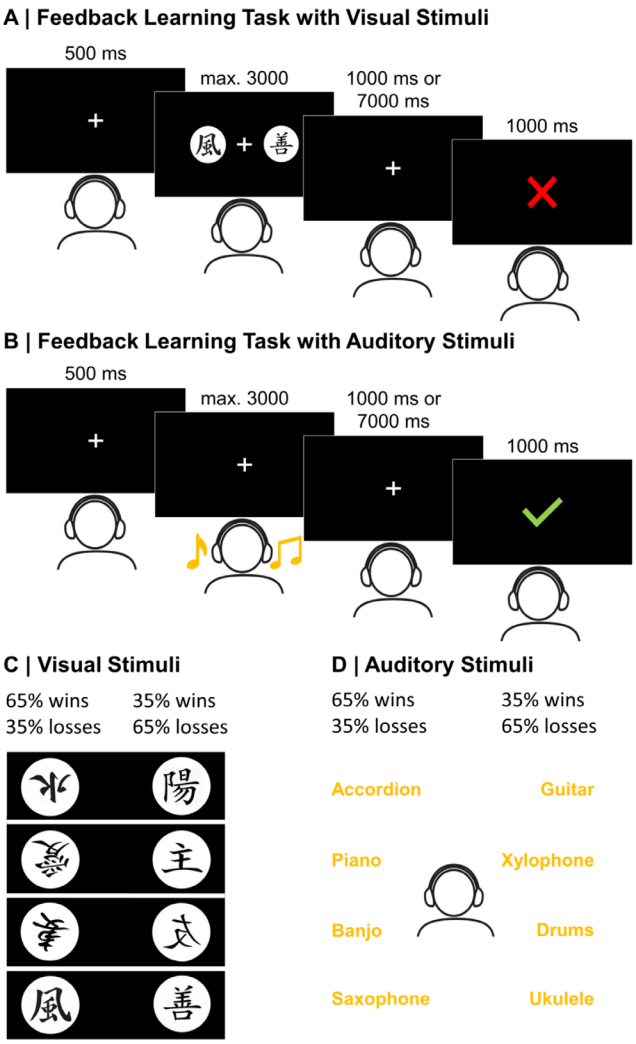

FIGURE 1 | Stimuli and time course of the probabilistic feedback learning tasks. Participants were instructed that the red cross represented a loss of −2 ct while the green tick represented a gain of +4 ct. (A) Feedback learning task with visual stimuli: The assignment of visual stimuli to the left and right sides of the screen was counterbalanced. In this way, feedback could clearly be associated with a stimulus and not with a response side. (B) Feedback learning task with auditory stimuli: The assignment of auditory stimuli to the left and right ears was counterbalanced. In this way, feedback could clearly be associated with a stimulus and not with a response side. (C) Visual stimuli: The neighboring stimuli form the four pairs used for all participants. The more rewarding stimulus (65% wins, 35% losses) was determined randomly when a new stimulus pair was presented. (D) Auditory stimuli: The neighboring stimuli form the four pairs used for all participants. The more rewarding stimulus (65% wins, 35% losses) was determined randomly when a new stimulus pair was presented.

learning phase consisted of 80 trials and was further divided into 4 blocks of 20 trials. Overall, each participant thus completed 640 trials.

In the visual condition, in every trial a pair of visual stimuli was presented on screen for maximally 3000 ms, one on the left and one on the right side of a centrally presented fixation cross. As stimuli, we used Hiragana-like characters (see Figure 1C) that cannot easily be verbalized (see Frank

et al. 2004). Participants could choose one of the two stimuli by pressing the corresponding (left vs. right) control key on a computer keyboard. The assignment of visual stimuli to the left and right side of the screen was counterbalanced. In this way, feedback could clearly be associated with a stimulus and not with a response side.

In the auditory condition, a pair of auditory stimuli was presented simultaneously via headphones for maximally 3000 ms, one to the left and one to the right ear, while participants' eyes rested on a fixation cross on the screen. As stimuli, we used different melodies played by different instruments to increase distinctiveness (see Figure 1D and listen to an example https://tinyurl.com/mrxtjvt2). Auditory stimuli were downloaded from Pixabay (https://pixabay.com/) and edited with Audacity (https://www.audacityteam.org/). Participants could choose one of the two stimuli by pressing the corresponding (left vs. right) control key on a computer keyboard. The assignment of auditory stimuli to the left and right ear was counterbalanced. In this way, feedback could clearly be associated with a stimulus and not with a response side.

After their choice, feedback was presented. Unbeknown to the participants, one stimulus of each pair was associated with reward in 65% of the trials and with punishment in 35%, while probabilities were reversed for the other stimulus. We chose these contingencies to prevent ceiling effects, as learning with just one stimulus pair at a time in an 80-trial learning phase might be too easy with higher contingencies. Additionally, these contingencies ensured relatively balanced frequencies of positive and negative feedback, minimizing the risk that one type of feedback would elicit different ERPs simply due to its lower occurrence frequency. The participants' task was to learn which stimulus was more likely to be rewarded and thereby maximize reward through their choices. Both wins and losses contributed to the overall sum of money.

2.3 | Procedure and Data Acquisition

Upon arrival in the laboratory, participants were informed about the experimental procedure and gave written informed consent to participate in the study, followed by a demographic questionnaire. Afterwards, we attached EEG electrodes and placed participants in front of a 27 in, 1920 × 1080 px W-LED monitor (BENQ EW2740L) with a refresh rate of 60 Hz, where the experimental task began, lasting about 60 min. Auditory stimuli were presented via dynamic stereo headphones (Sennheiser HD 201). Participants were informed prior to the experiment that they would receive 25 € or, in the case of psychology students, course credit. The money earned in the feedback learning task was thus not paid out in the end and was only virtual. The study was approved by the ethics committee of the Faculty of Mathematics and Natural Sciences at Heinrich Heine University Düsseldorf, Germany, and is in accordance with the declaration of Helsinki.

The software Presentation (Neurobehavioral Systems Inc 2020.) controlled the timing of stimulation and the recording of responses. Responses were performed on a standard computer keyboard (Logitech K120) where participants could press the left and right control keys to choose between the stimuli.

2.3.1 | EEG Data

EEG data was acquired from 60 active scalp electrodes, fixed with an actiCap textile softcap (BrainProducts, Germany) and evenly distributed on the scalp based on the extended 10–20 system. Electrodes were attached to the scalp sites AF3, AF4, AF7, AF8, C1, C2, C3, C4, C5, C6, CP1, CP2, CP3, CP4, CP5, CP6, CPz, Cz, F1, F2, F3, F4, F5, F6, F7, F8, FC1, FC2, FC3, FC4, FC5, FC6, FT10, FT7, FT8, FT9, Fz, O1, O2, Oz, P1, P2, P3, P4, P5, P6, P7, P8, PO10, PO3, PO4, PO7, PO8, PO9, POz, Pz, T7, T8, TP7, and TP8. The online reference was placed at the position FCz. Two further electrodes were placed over the left and right mastoids to cover as much of the scalp as possible for the calculation of the average reference (see below). Two electrodes (vEOG) were attached above (at Fp1 position) and below the left eye to measure vertical eye movements and blinks (yielding 65 electrodes in total). The ground electrode was attached to the AFz position. For data recording, a BrainAmp DC amplifier (BrainProducts, Germany) and the Brain Vision Recorder software (BrainProducts, Germany) were used, with a sampling rate of 1000 Hz and an online lowpass filter of 100 Hz. Impedances were kept below 15 kΩ.

2.4 | Data Analysis

2.4.1 | Behavioral Data Analysis

The dependent variable for behavioral data analysis was response accuracy, with correct responses coded as 1 and incorrect responses as 0 for the statistical analysis (see below). Correct responses were defined as the choice of the stimulus associated with the higher reward probability. We applied generalized linear mixed-effects models (GLME) suitable for binomial distributions and single-trial data by means of the lme4 package (version 1.1.34; Bates et al. 2015) in R to analyze the behavioral data (The R Foundation 2021). Descriptive data visualizations were adapted with the assistance of OpenAI's GPT-4 (OpenAI 2023). The model comprised fixed-effect predictors of the categorical factors Stimulus Modality (visual [−0.5] vs. auditory [0.5]) and Feedback Timing (immediate [−0.5] vs. delayed [0.5]), as well as the continuous factor learning block (1 [−0.5], 2 [−0.167], 3 [0.167], 4 [0.5]) and all possible interactions between the factors. Participants were included as random intercepts. For the inclusion of random-effect slopes per participant, we followed best practice (Meteyard and Davies 2020): all within-subject main and interaction effects were included as random slopes, unless their inclusion led to non-successful model fit. The best possible model was determined by using the buildmer (Version 2.11; Voeten 2020) function and resulted in the model presented in Table S1 of the Supporting Information.

2.4.2 | Modeling of PEs

We derived single-trial values of the PE for each participant by fitting a reinforcement learning model to the behavioral

data using MATLAB version R2021a (The MathWorks, Inc 2021; for a similar approach see Burnside et al. 2019; Lefebvre et al. 2017; Weber and Bellebaum 2024). Aiming for a model whose predicted choices deviate the least from our participants' behavior, we compared two models of different complexity. Starting point was each participants' sequence of choices and the received feedback. The PE $\delta_{c,t}$ was calculated as:

$$\delta_{c,t} = r_t - Q_{c,t}$$

where in a given trial t the reward r_t is 1 for positive feedback and 0 for negative feedback, and $Q_{c,t}$ is the value of the chosen stimulus. Separately for each of the eight stimulus pairs (four containing visual and four containing auditory stimuli), both stimuli were initially assigned a stimulus value of 0.5, that was iteratively updated in every trial t in which the stimulus pair was presented. In a first model (M_1) the stimulus value of the chosen stimulus, Q_c, was updated based on the deviation between the prior value and the received outcome, i.e., the PE δ, and a learning rate α (specific for each stimulus pair), which indicates the extent to which the PE was used to update the stimulus value.

$$Q_{c,t+1} = Q_{c,t} + \alpha * \delta_{c,t}$$

As both stimuli of a pair were always presented together, we expected participants to draw conclusions about the unchosen stimulus from feedback for the chosen stimulus. Therefore, the value of the unchosen stimulus, Q_u, equaled $1 - Q_c$ and was updated accordingly.

For each trial, $t_{1,\ldots,n_{trials}}$, the probability p that the model would choose the stimulus which was indeed chosen by the participant was calculated using the softmax function based on prior stimulus values of the two stimuli that were available, i.e., values of the chosen stimulus, $Q_{c,t}$, and the unchosen stimulus in trial t, $Q_{u,t}$, and an exploration parameter β:

$$p_{c,t} = \frac{e^{Qc,t*\beta}}{e^{Qc,t*\beta} + e^{Qu,t*\beta}}$$

with β indicating the impact of prior stimulus values on a subject's choices. A larger β indicates that a participant utilized prior stimulus values (i.e., a larger impact of prior values), whereas a smaller β indicates rather explorative choice behavior (i.e., a smaller impact of prior values).

In a next step, the probabilities p were used to calculate the negative summed log-likelihood ($-LL$) as measure for the model's goodness of fit:

$$-\sum \log\left(p_{c,t_1,\ldots,n_{trials}}\right)$$

We used the optimization function fmincon from the Optimization Toolbox of MATLAB (R2021a, The MathWorks, Inc 2021) to minimize the $-LL$ value by estimating values for the free parameters $(\alpha, \beta)/(\alpha_{con}, \alpha_{dis}, \beta$, see below) that result in the least deviation between the model's predicted choices and the participant's behavior. We fit the model repeatedly (50 iterations) to the subjects' behavior to avoid local minima. As start values for the free parameters, we allowed random numbers within the interval [0; 1]. We set value constraints for the free parameters to [0; 1] for the learning rate, and to [0; 100] for the exploration parameter β.

In a second model (M_2), we allowed different learning rates for learning from positive feedback and negative feedback. The stimulus value of the chosen stimulus was updated with the learning rate α_{con} for trials with positive feedback that confirms the choice as follows:

$$Q_{c,t+1} = Q_{c,t} + \alpha_{con} * \delta_{c,t}$$

Analogously, for trials with negative feedback that disconfirms the choice, the stimulus value of the chosen stimulus was updated with the learning rate α_{dis}:

$$Q_{c,t+1} = Q_{c,t} + \alpha_{dis} * \delta_{c,t}$$

Everything else stayed the same compared to M_1.

The two models were compared based on their negative summed log-likelihood ($-LL$) by a paired samples t-test. M_2 resulted in significantly lower $-LL$ values (M = 360.88, SD = 246.73) than M_1 (M = 381.21, SD = 242.18), $t(34) = 9.18$, $p < 0.001$, indicating a better model fit. Furthermore, a lower Bayesian Information Criterion (BIC) indicated that M_2 (BIC = 751.73) provides a better balance between model fit and complexity compared to M_1 (BIC = 782.40). Eventually, M_2 was used to extract stimulus values and trial-by-trial PEs. Single-subject $-LL$ values are illustrated in Figure S1A of the Supporting Information. Furthermore, we visualized the learning rates for positive and negative feedback (α_{con} and α_{dis}) to ensure that they do not systematically converge to values of 0 or 1 (see Figures S1B and S2 of the Supporting Information). Finally, we examined participants' win-stay and lose-shift behavior to determine whether participants were using the PE to adapt their behavior. The results are presented in the Supporting Information, in the section titled Win-stay vs. lose-shift analysis accompanied by Figure S3. All visualizations and analyses supported that the PE modeling resulted in meaningful data.

2.4.3 | EEG Data Analysis

BrainVision Analyzer 2.2 (Brain Products GmbH 2018), MATLAB R2021a (The MathWorks, Inc 2021) and R (The R Foundation 2021) were used for EEG data analysis. Trials in which participants failed to answer (M = 1.67%, SD = 2.59%, Min = 0.16%, Max = 13.44%) were excluded from any further EEG analyses.

2.4.3.1 | Preprocessing. In a first step, we re-referenced the data to the average of all 63 scalp electrodes including the mastoids (see above; the signal at the online reference site FCz was calculated; see Arbel et al. 2017; Höltje and Mecklinger 2020, for similar procedures). The reduction of ERP effects that can result as a consequence of using an average reference (see Luck 2014) is minimized for high-density EEG acquisition as in our study. In a second step, the data were

filtered, using a 30 Hz low cut-off and a 0.1 Hz high cut-off filter (as proposed by Luck 2014) as well as a 50 Hz Notch Filter. In order to correct for blink artifacts, an independent component analysis (ICA) and reverse ICA was performed on single-subject EEG data (see Peterburs et al. 2016; Weismüller et al. 2019 for a similar procedure). We created segments from 200 ms before to 800 ms after feedback onset and performed a baseline correction relative to the first 200 ms. Then, segments with artifacts in electrodes used to measure the FRN and N170 (see below) were removed (for a similar approach see Albrecht et al. 2023; all segments containing voltage steps > 50 μV/ms, differences between values > 80 μV or < 0.1 μV within an interval of 100 ms or amplitudes > 80 μV or < −80 μV; M = 1.09%, SD = 2.23%, Min = 0.00%, Max = 12.97%). This way, we aimed to include as much data as possible for our single-trial analysis, as linear mixed-effects (LME) models that we applied for the analyses (see below) are tailored for managing data variability (Bates et al. 2015; Quené and Van den Bergh 2004). On average, per participant, 156.6 trials (SD = 5.2, Min = 130, Max = 160) from the visual task with immediate feedback and 155.6 trials (SD = 5.3, Min = 136, Max = 160) with delayed feedback entered the analysis. From the auditory task, on average 155.6 trials (SD = 7.6, Min = 122, Max = 160) with immediate feedback and 154.2 trials (SD = 8.5, Min = 112, Max = 160) with delayed feedback entered the analysis per participant.

The remaining segments were grouped and averaged for each of the eight conditions (positive and negative immediate feedback and delayed feedback for the tasks involving visual and auditory stimuli), yielding eight averages per participant. Subsequently, all single-trial segment data as well as all averages per condition and participant were exported for later analysis. For further preprocessing steps, MATLAB scripts (MathWorks, MA) were utilized, which were adapted with the assistance of OpenAI's GPT-4 (OpenAI 2023) to extract single-trial data.

For the N170, single-trial amplitudes (see Albrecht et al. 2023) were derived from electrodes P7 and P8 (see Arbel et al. 2017; Höltje and Mecklinger 2020; Kim and Arbel 2019), as outlined in the preregistration for the study (osf.io/fu2gy). First, the maximum negative peak amplitude between 130 and 230 ms post-feedback was determined in each participant's average, at both electrode sites and for all eight conditions separately (see above). Then, for each single trial, the mean amplitude in a time window of ±10 ms around the condition-specific N170 peak latency was calculated. Because grand averages revealed differences between the conditions already in the preceding positive peak (see Figure 3), we additionally extracted the single-trial mean amplitude in a time window of ±10 ms around the preceding positive peak (P1). As for the negative peak, the latency of the P1 was determined based on the condition-specific average at each electrode site. The P1 was determined as the maximum positivity in a time window starting 80 ms after feedback onset to the respective condition-specific negative peak. For the analysis, we used the N170 defined as the peak-to-peak amplitude by subtracting the single-trial amplitude value of the preceding P1 from the single-trial value of the negative peak.

For the FRN, single-trial amplitudes were derived from an electrode cluster consisting of Fz, FCz, Cz, FC1, and FC2, for which the signal was pooled. Previous studies showed that the FRN was maximal at FCz but also pronounced at neighboring channels (Arbel et al. 2017; Kim and Arbel 2019; Maurer et al. 2022; Mushtaq et al. 2022). To account for individual differences, we decided to measure FRN amplitudes in the pooled signal of a group of five frontocentral electrode sites (for a similar approach see Zottoli and Grose-Fifer 2012), including FCz and neighboring electrodes (see Weber and Bellebaum 2024). For each participant, we used their mean waveform for both positive and negative feedback separately for each of the four conditions (immediate feedback in the visual task, delayed feedback in the visual task, immediate feedback in the auditory task, and delayed feedback in the auditory task). Then, we computed the difference wave by subtracting the mean positive feedback waveform from the mean negative feedback waveform for each of these four conditions. For each participant, we identified the maximum negative peak amplitude in each of the four difference waves within a time window of 230–360 ms post-feedback, i.e., the peak latency was determined separately for each condition. Next, for each single trial, we extracted the mean amplitude within a ±10 ms window around the condition-specific difference wave peak latency. It is important to emphasize that our dependent variable is not derived from the difference wave itself. Rather, the difference wave was only used to identify the latency at which the difference between the processing of positive and negative feedback is maximal. This latency was then used to extract the single-trial ERP data. Therefore, our actual dependent variable was derived from the ERPs for positive and negative feedback in each condition.

2.4.3.2 | Statistical Analysis

2.4.3.2.1 | N170. The single-trial N170 amplitude was analyzed as a dependent variable by applying an LME analysis in R (Bates et al. 2015). The model comprised fixed-effect predictors of the categorical factors feedback timing (immediate [−0.5] vs. delayed [0.5]), stimulus modality (visual [−0.5] vs. auditory [0.5]) and feedback valence (negative [−0.5] vs. positive [0.5]). Furthermore, the PE was used as a continuous predictor. However, as the signed PE is confounded by valence, we used the unsigned or absolute PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) indicating general expectation violations or surprise. Finally, the factor electrode (P7 [−0.5] vs. P8 [0.5]) was added. Furthermore, we added all possible interactions between the factors. Although this adds complexity to the model, we believe that this is justified due to our hypotheses and the interrelated nature of the predictors. Our hypothesis concerning the N170 already involves an interaction between the factors stimulus modality, feedback timing, and electrode, as we expected its amplitude to be most pronounced for delayed feedback following choices between visual stimuli and over the right hemisphere. The more exploratory analysis, whether the N170 encodes a PE, aims at the question of whether there is an interaction between feedback valence and the absolute PE. Moreover, this interaction may again be modulated by the three factors Stimulus Modality, feedback timing, and electrode. Given that all of the predictors are thus closely linked and may influence each other, we decided to include all interactions when planning the study (as was also preregistered). Participant was included as a random-effect factor. Random slopes per participant were added as described for the behavioral GLME above (see Table S1 of the Supporting Information for the resulting

model). Simple slope analyses were performed to resolve significant interactions, with Bonferroni-corrected p-values (multiplied by the number of conducted tests).

2.4.3.2.2 | FRN. The single-trial FRN amplitude was analyzed as a dependent variable by applying an LME analysis in R (Bates et al. 2015). The model comprised fixed-effect predictors of the categorical factors feedback timing (immediate [−0.5] vs. delayed [0.5]), stimulus modality (visual [−0.5] vs. auditory [0.5]) and feedback valence (negative [−0.5] vs. positive [0.5]) and as a continuous factor the mean centered unsigned PE, as well as all possible interactions between the factors. For the FRN, it has been shown that its amplitude reflects a (signed) PE, indicated by the interaction between the factors feedback valence and (unsigned) PE. Moreover, effects of feedback timing have been found, which may also interact with PE coding (Weber and Bellebaum 2024). In this study, we aimed to explore whether stimulus modality affects the FRN, alone or in interaction with the mentioned factors. Participant was included as a random-effect factor. Random slopes per participant were added as described for the behavioral GLME above (see Table S1 of the Supporting Information for the resulting model). Significant interactions were resolved as described for the N170 (see above).

3 | Results

3.1 | Behavioral Results

With the GLME analysis of the behavioral data, we first aimed to determine whether participants learned to increasingly select the more frequently rewarded stimulus across the four learning blocks. Second, we examined whether there were any differences in learning between the tasks involving choices between visual and auditory stimuli, or between the conditions with immediate and delayed feedback, or between any combinations of these two factors.

Descriptive data are presented in Figure 2. Table S2 in the Supporting Information lists β-estimates and effect-specific z-tests for the GLME analysis investigating effects of feedback timing, feedback valence, and stimulus modality on the behavioral data. The analysis revealed a significant effect of Block ($p < 0.001$) on response accuracy, driven by an increasing number of correct responses across the four learning Blocks. Figure 2 suggests that this effect is due in particular to an increase in correct responses from block 1 to block 2. No other significant effects were observed (all $ps \geq 0.140$), indicating that learning was comparable for immediate and delayed feedback and for the tasks involving choices between visual and auditory stimuli.

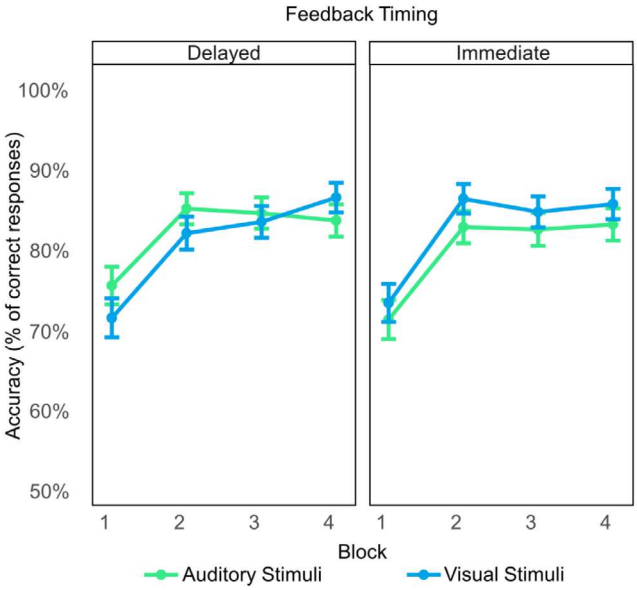

FIGURE 2 | Descriptive pattern of performance improvement during the feedback learning task. Mean accuracy (% of correct responses) for the four learning blocks of the probabilistic feedback learning task, separately for immediate and delayed feedback and for the tasks involving choices between visual and auditory stimuli. Error bars represent 95% confidence intervals.

3.2 | EEG Results

3.2.1 | N170

With the LME analysis of the N170 single-trial data, we aimed to test our hypothesis that the N170 is most pronounced for delayed feedback referring to the choice of visual stimuli, with a possibly more pronounced effect over the right hemisphere. This would be reflected in an interaction between the factors stimulus modality, feedback timing, and electrode. Moreover, we aimed to investigate if the N170 reflects a signed PE, which would be reflected in an interaction between feedback valence and the unsigned PE, and whether this effect is modulated by the other factors stimulus modality, feedback timing, and electrode. The analyses thus focused on interaction effects of the involved predictors, and main effects will not be reported in the following. Grand averages for the ERPs following positive and negative immediate and delayed feedback for the choice between visual and auditory stimuli at electrode sites P7 and P8 are presented in Figure 3. In addition, the Supporting Information contains grand averages separately for low and high absolute PE values (expected vs. unexpected; Figure S4). Table S3 in the Supporting Information lists β-estimates and effect-specific t-tests for all effects of the LME analysis investigating the N170 amplitude. In the following, more negative N170 amplitudes are described as more pronounced or larger, respectively.

Regarding our hypothesis, we indeed found a significant stimulus modality × feedback timing interaction ($p < 0.001$) that was further explained by a significant stimulus modality × feedback timing × electrode interaction ($p < 0.001$) which we thus resolved. The descriptive pattern behind the three-way interaction is presented in Figure 4A. A simple slope analysis revealed that for the P7, the effect of Stimulus Modality was neither significant for immediate ($\beta = -0.08$, $SE = 0.33$, $t = -0.23$, $p > 0.999$) nor for delayed feedback ($\beta = 0.72$, $SE = 0.42$, $t = 1.72$, $p = 0.363$). For the P8, there was a significant effect of stimulus modality following immediate feedback with larger N170 amplitudes for auditory compared to visual stimuli, $\beta = -1.29$, $SE = 0.33$, $t = -3.93$, $p < 0.001$. For delayed feedback, this effect was reversed with significantly larger N170 amplitudes for visual compared to auditory stimuli, $\beta = 1.31$, $SE = 0.42$, $t = 3.14$, $p = 0.010$.

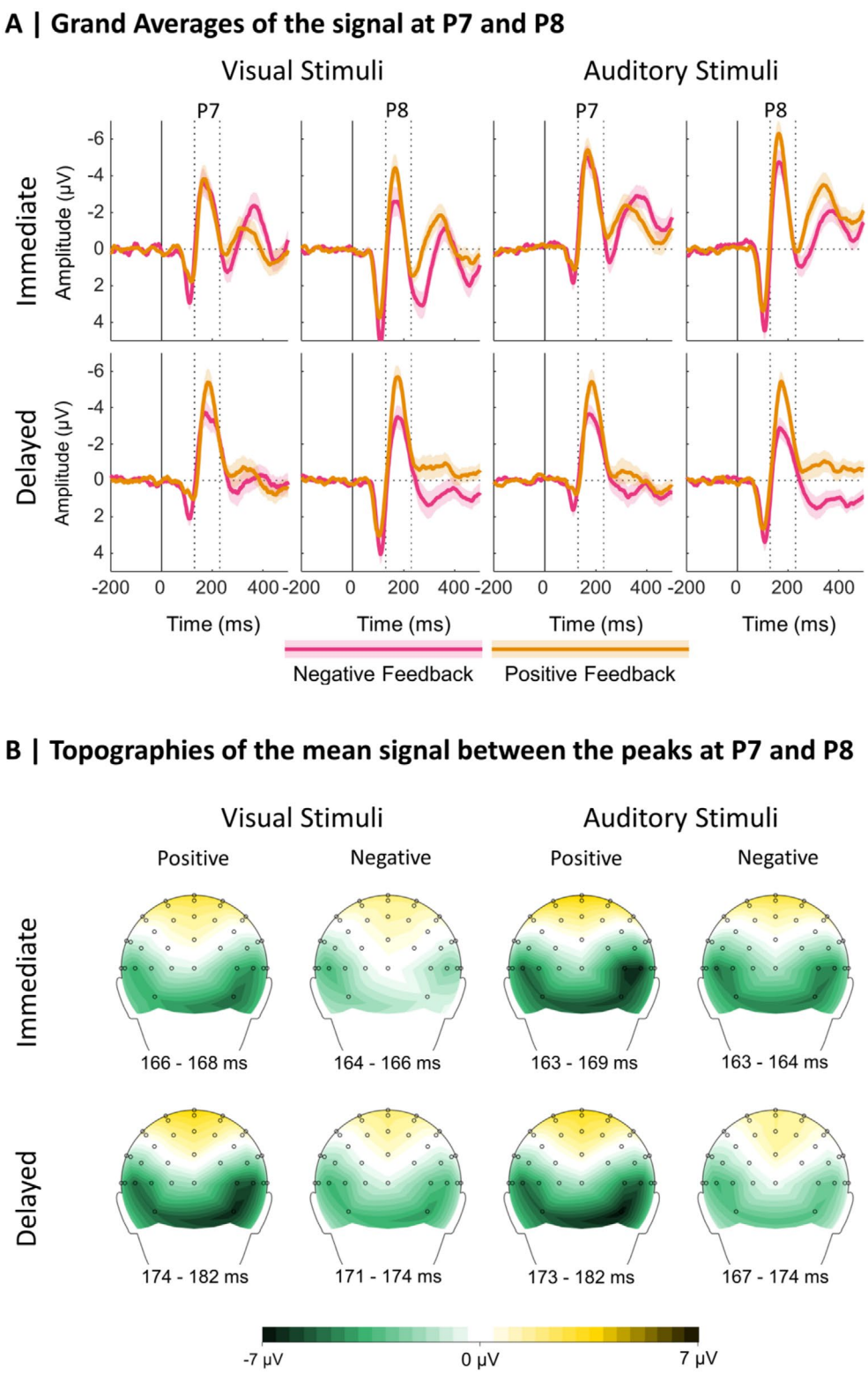

FIGURE 3 | Grand averages at P7 and P8 and topographical maps at the respective peaks. (A) Grand Averages: Dotted lines indicate the time window used for the N170 peak detection. Shaded areas indicate standard errors. (B) Topographies: The maps are based on the condition-specific N170 peaks.

With regard to more exploratory results, we found a significant interaction between PE and feedback valence ($p < 0.001$), indicating that the N170 indeed reflects the signed PE. As this interaction was further explained by a significant three-way interaction between PE, feedback valence, and electrode ($p = 0.001$), we decided to resolve the three-way interaction with simple slope analyses. The underlying descriptive data are presented in Figure 4B. For P7, the PE had no significant effect on the N170, neither for negative ($\beta = 0.18$, SE = 0.40, $t = 0.46$, $p > 0.999$) nor for positive feedback ($\beta = -0.66$, SE = 0.41, $t = -1.60$, $p = 0.439$).

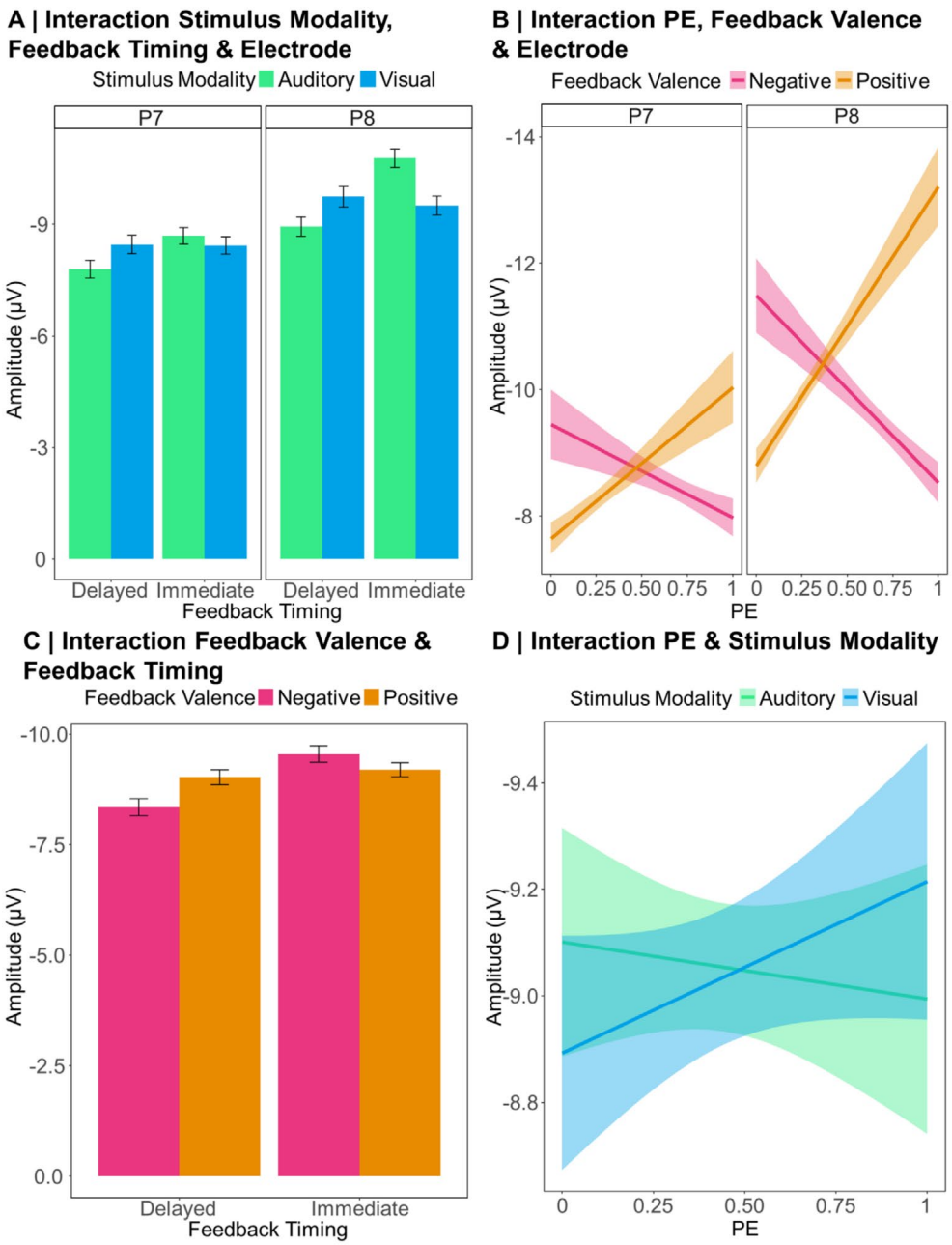

FIGURE 4 | Descriptive data patterns underlying the N170 analysis. Error bars indicate a 95% confidence interval. Shaded areas indicate standard errors.

For P8, the PE had a significant effect on the N170 following negative feedback, with larger amplitudes for expected compared to unexpected feedback ($\beta = 1.23$, SE = 0.40, $t = 3.11$, $p = 0.008$). For positive feedback, the effect was reversed, with significantly larger N170 amplitudes for unexpected compared to expected feedback ($\beta = -2.27$, SE = 0.41, $t = -5.52$, $p < 0.001$).

While we found two further two-way interactions, one between feedback valence and feedback timing ($p = 0.002$, the underlying descriptive data are presented in Figure 4C) and one between PE and stimulus modality ($p = 0.026$, the underlying descriptive data are presented in Figure 4D), a significant five-way interaction between all included predictors was of main interest ($p = 0.042$). The underlying descriptive pattern is presented in Figure 5.

To resolve this interaction, we split the dataset based on the electrode and repeated the LME analysis separately for P7 and P8. There was a significant four-way interaction between the remaining factors feedback timing, feedback valence, stimulus modality, and PE for the P8 ($\beta = 4.88$, SE = 2.36, $t(2251.33) = 2.07$, $p = 0.039$), but not for the P7 ($\beta = -2.68$, SE = 2.18, $t(2767.86) = -1.23$, $p = 0.218$). To resolve the four-way interaction at P8, we again split the dataset, but this time according to Feedback Timing. For delayed feedback, the three-way interaction between feedback valence, stimulus modality, and PE reached significance ($\beta = 5.07$, SE = 1.76, $t(7454.36) = 2.87$, $p = 0.004$), unlike for immediate feedback ($\beta = 0.98$, SE = 1.67, $t(5272.01) = -0.58$, $p = 0.560$). To resolve the three-way interaction for delayed feedback, we finally split the dataset according to stimulus modality. We found a significant interaction between PE and feedback valence for visual

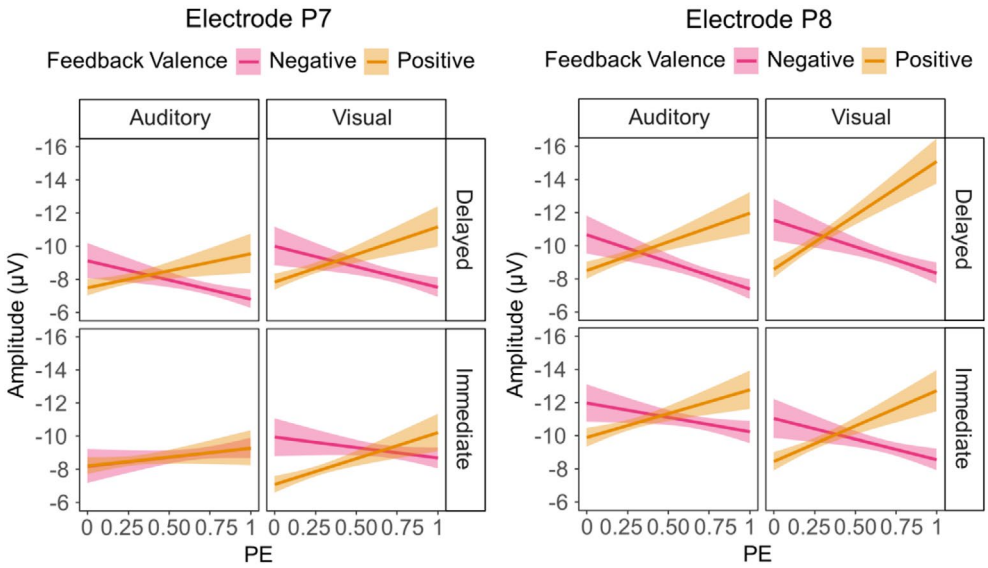

FIGURE 5 | Descriptive data pattern underlying the PE × Feedback Valence × Modality × Feedback Timing × Electrode interaction for the N170. Shaded areas indicate standard errors.

stimuli ($\beta = -7.12$, SE = 1.30, $t(4870.20) = -5.45$, $p < 0.001$), but not for auditory stimuli ($\beta = -1.36$, SE = 1.23, $t(4300.97) = -1.11$, $p = 0.267$). We resolved the two-way interaction found for visual stimuli with a simple slope analysis. For negative feedback, larger PE values led to significantly less pronounced N170 amplitudes ($\beta = 2.02$, SE = 0.87, $t = 2.32$, $p = 0.046$). For positive feedback, larger PE values led to significantly more pronounced N170 amplitudes ($\beta = -5.09$, SE = 0.93, $t = -5.47$, $p < 0.001$). To conclude, the five-way interaction is driven by a reflection of the PE in the N170 measured over the right hemisphere, especially following delayed feedback that refers to visual stimuli. In the Supporting Information, the descriptive data underlying the N170 interaction effects described in the main text are represented with a detailed overview of data distribution and variance (see Figures S5 and S6).

3.2.2 | FRN

With the LME analysis of the FRN single-trial data, we aimed to replicate that the FRN is sensitive to feedback valence, especially following immediate feedback. Furthermore, we aimed to replicate that the amplitude reflects a signed PE signal, reflected in an interaction between (unsigned) PE and feedback valence. In an exploratory manner, we were also interested in the effects of stimulus modality, alone or in interaction with the other predictors. Grand averages for the ERPs following positive and negative immediate and delayed feedback for the choice between visual and auditory stimuli pooled over the frontocentral cluster of electrodes are presented in Figure 6. In addition, the Supporting Information contains grand averages separately for expected and unexpected feedback (Figure S7). For β-estimates of the LME analysis on the FRN amplitude and effect-specific *t*-tests, see Table S4 in the Supporting Information. Descriptive statistics can be found in Figure 7A. In the following, more negative FRN amplitudes are described as more pronounced or larger, respectively.

As expected, we could replicate previous findings of a significant main effect of feedback valence (more pronounced FRN for negative compared to positive feedback, $p < 0.001$) and a significant feedback timing × feedback valence interaction ($p = 0.006$, see Figure 7B). Resolving this interaction using simple slope analyses showed a more pronounced FRN for negative compared to positive feedback for both immediate ($\beta = 2.94$, SE = 0.39, $t = 7.60$, $p < 0.001$) and delayed feedback ($\beta = 1.86$, SE = 0.26, $t = 7.16$, $p < 0.001$), with a larger feedback valence effect for immediate feedback.

Another replication concerned a significant feedback valence × PE interaction ($p < 0.001$, see Figure 7C). Resolving this interaction via simple slope analyses resulted in a significant effect of the PE for negative feedback ($\beta = -3.55$, SE = 0.63, $t = -5.60$, $p < 0.001$) with larger (i.e., more negative) FRN amplitudes for unexpected feedback. For positive feedback, there was a significant effect of the PE with smaller (i.e., more positive) amplitudes for unexpected feedback ($\beta = 3.89$, SE = 0.58, $t = 6.73$, $p < 0.001$).

Regarding more exploratory results involving the factor stimulus modality, the analysis revealed a significant main effect ($p = 0.014$), which was further explained by a significant feedback timing × stimulus modality interaction ($p = 0.002$, see Figure 7D). We resolved this interaction using a simple slope analysis that yielded a significant effect of Stimulus Modality, with larger amplitudes for visual than auditory stimuli, for immediate ($\beta = 1.33$, SE = 0.31, $t = 4.33$, $p < 0.001$), but not for delayed feedback ($\beta = -0.04$, SE = 0.34, $t = -0.13$, $p > 0.999$). All other main and interaction effects were not significant (all $ps \geq 0.171$). In the Supporting Information, the descriptive data underlying the FRN interaction effects described in the main text are represented with a detailed overview of data distribution and variance (see Figure S8).

4 | Discussion

The present study aimed to investigate whether the N170 ERP component is modulated by the modality of the associated

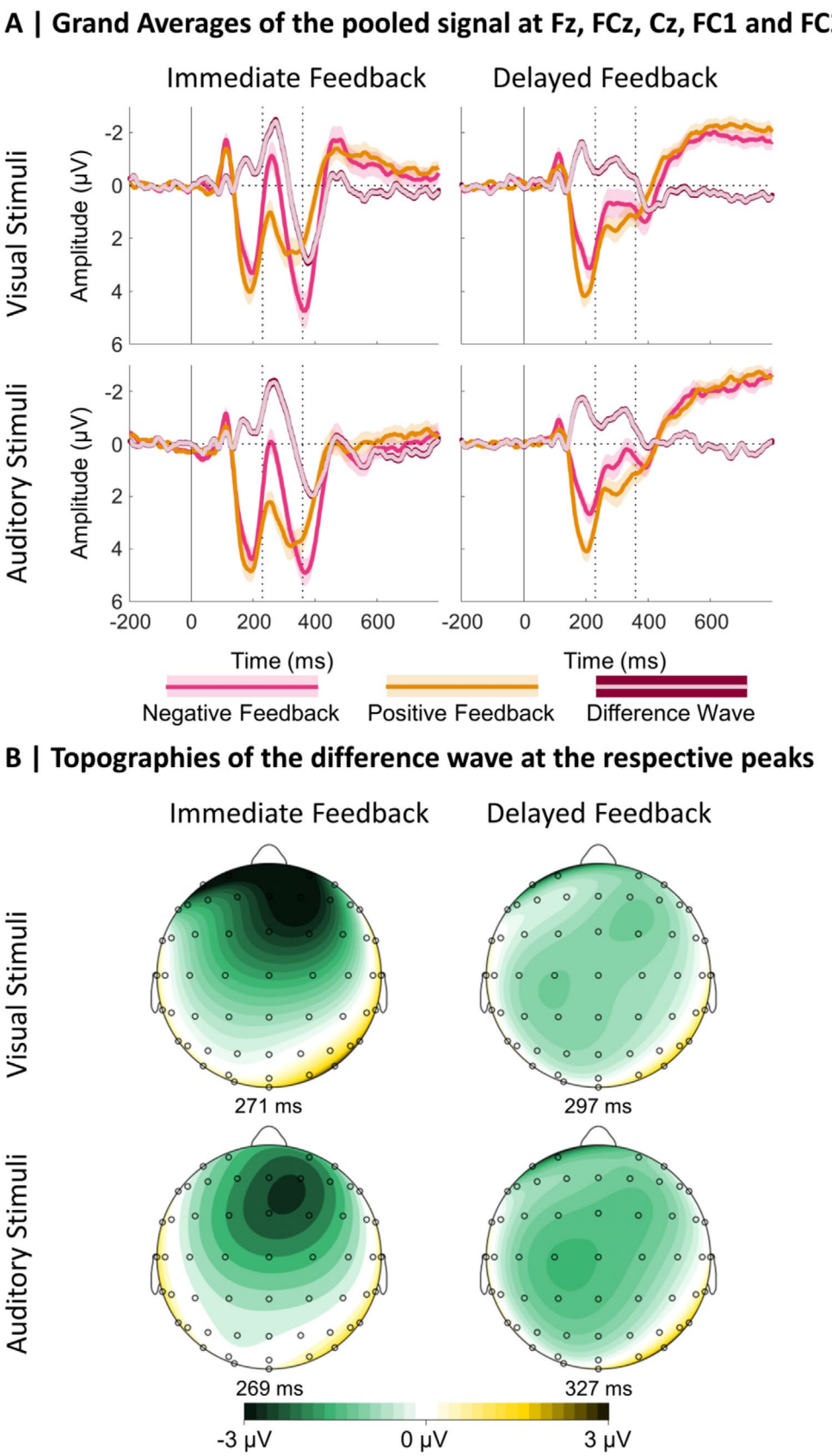

FIGURE 6 | Grand averages and topographical maps of the FRN. (A) Grand Averages: Dotted lines indicate the time window used for the peak detection in the difference wave (negative—positive feedback). Shaded areas indicate standard errors. (B) Topographies: The maps are based on the condition-specific difference wave.

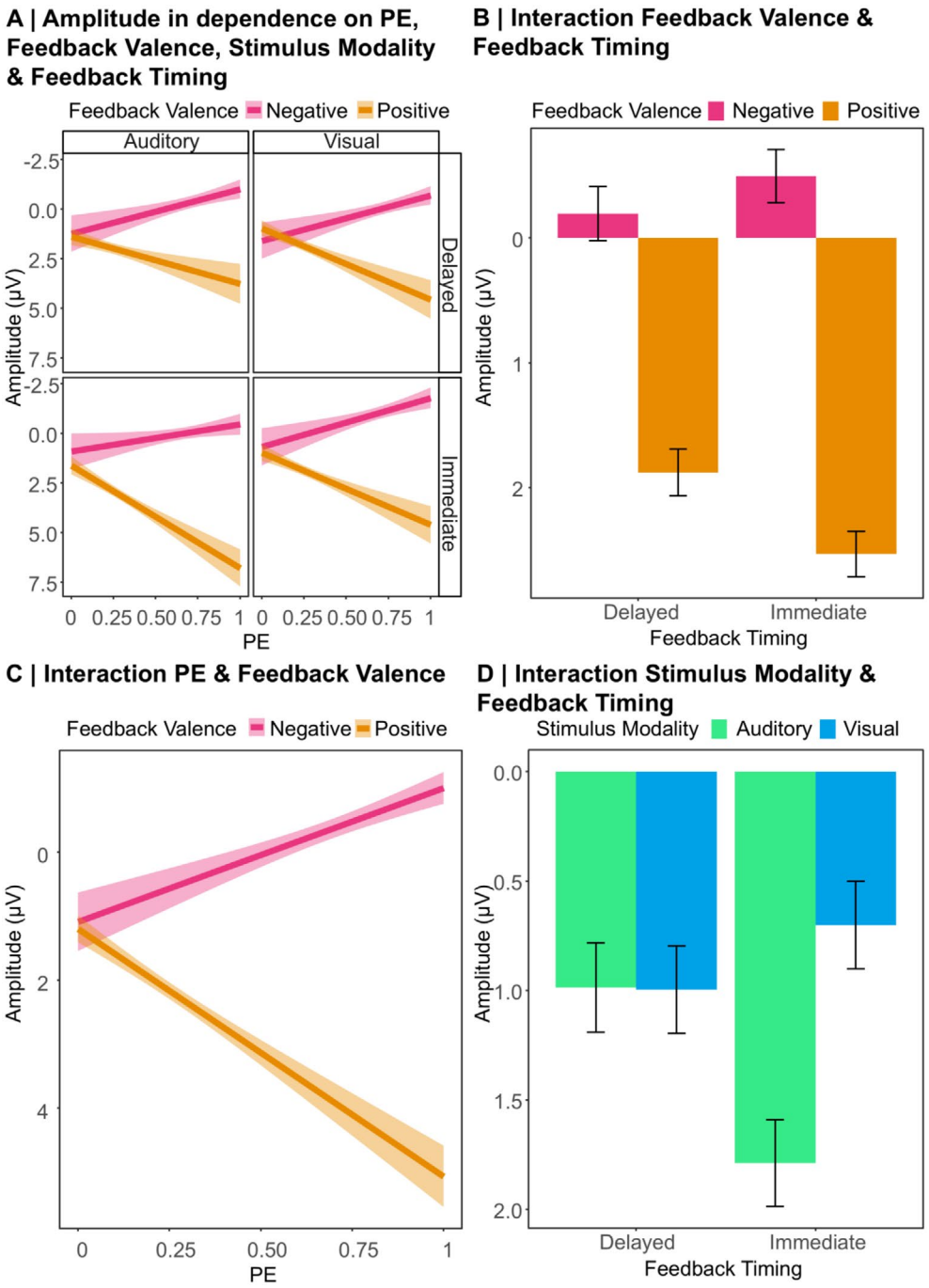

FIGURE 7 | Descriptive data patterns underlying the FRN analysis. Error bars indicate a 95% confidence interval. Shaded areas indicate standard errors.

stimulus during feedback processing in a reinforcement learning task. While previous studies have examined the influence of feedback modality (visual vs. auditory, see Kim and Arbel 2019), our study is the first to manipulate the sensory modality of the stimuli (visual vs. auditory) between which participants make their choices before receiving visual feedback. More specifically, we hypothesized that the N170 reflects a process that bridges the temporal gap between the choice of a stimulus and feedback, especially for delayed feedback for visual stimuli that are associated with the feedback and over the right hemisphere. Indeed, we found that delayed feedback related to the choice of visual stimuli led to significantly larger N170 amplitudes than feedback following the choice of auditory stimuli over the right lateral hemisphere. Furthermore, we found pronounced effects of the PE on the N170 measured over the right hemisphere, again especially for delayed feedback related to the choice of visual stimuli. For immediate feedback, however, an unexpected pattern emerged, with larger N170 amplitudes for feedback following the choice between auditory compared to visual stimuli. Regarding the FRN, we also found a modality effect, specifically for immediate feedback: it was more pronounced when the feedback was related to the choice between visual stimuli than auditory stimuli. Despite the differences in feedback processing depending on Feedback Timing and Stimulus Modality,

our participants appeared to learn equally well from immediate and delayed feedback, as well as in the tasks involving visual or auditory stimuli.

4.1 | The Role of the Modality of the Associated Stimulus for Feedback Processing

Based on previous studies, a clear functional interpretation of the N170 in the context of (delayed) feedback processing is not yet possible. In studies investigating delayed feedback processing, the stimuli associated with feedback were always visual (Arbel et al. 2017; Höltje and Mecklinger 2020; Kim and Arbel 2019). We hypothesized that the modality of the stimulus that is associated with the feedback modulates the amplitude of the N170. Since this component has been linked to visual processing in the extrastriate cortex (Brem et al. 2006; Deffke et al. 2007; Gao et al. 2019; Iidaka et al. 2006), we assumed that the N170 reflects a reactivation of a visual stimulus associated with feedback and should be more pronounced when feedback is given for a choice between visual stimuli, especially when feedback is delayed. Given that the right hemisphere plays a dominant role in processing certain visual stimuli, such as faces (Rossion 2014), and in N170 generation in different contexts (Baker and Holroyd 2009, 2013; Baker et al. 2015; Kim and Arbel 2019), we were particularly interested in whether the effects would be stronger over the right hemisphere.

The research question of the present study thus addresses the implementation of the so-called credit assignment problem within the brain. For immediate feedback, the temporal proximity of the reward signal from the dopaminergic midbrain and the activation of cortical areas representing, e.g., a visual stimulus probably suffice to establish a connection. (Schultz 2002; Jocham et al. 2016) found heuristic time-based learning mechanisms related to activity in circuits including the striatum. Furthermore, reward signals coded by dopamine drive synaptic connections—the molecular basis of learning—in the striatum in a narrow time window of up to 2s (Yagishita et al. 2014). However, if feedback is presented after a longer delay, the representation of the selected stimulus might be reactivated at the time of feedback presentation. The present study provides first evidence that the modality of the associated stimulus affects the N170: In the right hemisphere, we found larger N170 amplitudes following delayed feedback for the choice of visual compared to auditory stimuli. While a study by Herholz et al. (2012) found an overlap of melody perception and imagery in secondary auditory areas, supporting the existence of auditory reactivation processes, the N170 has been specifically linked to stimulus processing in the visual domain (Bentin et al. 1996; Itier and Taylor 2004; Kloth et al. 2013; for reviews see Yovel 2016; Carreiras et al. 2014). Our results thus support the hypothesis that the N170 reflects stimulus reactivations in higher-order visual areas, which may mirror an association mechanism in which reactivated representations of a selected stimulus are used to bridge the temporal gap to delayed feedback. This interpretation is in line with fMRI studies that revealed post-reward reactivation mechanisms in visual (Schiffer et al. 2014) as well as somatosensory areas (Pleger et al. 2008, 2009) as a way to assign credit to a stimulus for an obtained reward. Finding this potential reactivation for the N170 only over the right hemisphere may be due to the functional specialization of the right hemisphere for visuo-spatial processing (e.g., Thiebaut de Schotten et al. 2011), as the visual stimuli used in our study (hiragana characters) had a visuo-spatial character. Furthermore, studies investigating the N170 in the context of navigational feedback learning particularly linked it to activity within the right MTL, or more precisely the right parahippocampal cortex (Baker and Holroyd 2009, 2013; Baker et al. 2015). It is important to note that the functional meaning of the N170 could be different in contextually different tasks.

Against our expectation, we found a larger feedback-locked N170 for choices between auditory than visual stimuli for immediate feedback. One explanation could be that the N170 reflects overlapping activity of MTL and extrastriate visual areas in feedback processing. Indeed, the hippocampus has been found to be involved in feedback processing even for short feedback delays of only two seconds (Dickerson et al. 2011). Integrating information about feedback and the associated stimulus, hippocampal processing demands for the auditory condition may have been particularly high, as this condition required cross-modal associations, which activates the hippocampus more than unimodal associations (Butler and James 2011). For delayed feedback, the extrastriate visual cortex contribution to the N170 may have been higher.

For the FRN, which has been investigated much more extensively in the context of feedback processing, the fact that we found larger FRN amplitudes following immediate feedback for the choice between visual compared to auditory stimuli was also surprising. FRN effects are mainly interpreted with respect to feedback valence and/or the PE. As stimulus modality did not affect the effects of feedback valence or the reflection of the PE in the FRN, it is questionable whether stimulus modality exerted a significant influence on the processes underlying the FRN.

4.2 | Effects of Feedback Valence and PE for Immediate and Delayed Feedback

In contrast to previous studies, we did not find a main effect of feedback timing (Arbel et al. 2017; Kim and Arbel 2019; Höltje and Mecklinger 2020) or feedback valence (Kim and Arbel 2019) for the N170, but an interaction between the two: a valence effect was only detectable when feedback was delayed. In this regard, the N170 formed a kind of counterpart to the FRN, for which there was an enhanced differentiation between immediate positive and negative feedback compared to delayed (for similar results see Arbel et al. 2017; Höltje and Mecklinger 2020; Peterburs et al. 2016; Weinberg et al. 2012; Weismüller and Bellebaum 2016).

This complementary processing is further evident considering the PE effects on the two components. Effects of reward PEs on the N170 have not been reported before. We found that the N170 reflects the whole range of PEs, which is in line with recent findings by Baker et al. (2021, 2023), who reported more pronounced N170 amplitudes for unpredictable compared to predictable stimuli during the perceptual processing of visual stimuli, linking the N170 to surprise in general. While the FRN also reflected the whole range of PEs in the present study, the

N170, especially over the right hemisphere, was enhanced for unexpected positive feedback and reduced for unexpected negative feedback, and the pattern of PE coding was reversed for the FRN that became more negative when negative feedback was unexpected and more positive when positive feedback was unexpected. Regarding the N170, enhanced amplitudes following unexpected positive feedback might indicate that representations of unexpectedly rewarded stimuli are especially reactivated. Put simply, this means that it is especially important to remember which stimulus brought the reward and strengthen that relationship. Remembering what led to the reward can be very helpful for survival, and a form of reactivation following rewards could be a way to bind them to preceding situations (Singer and Frank 2009).

Correlates of the PE in the N170 could be interpreted as reflecting PE-related hippocampal activity (Dickerson et al. 2011; Foerde and Shohamy 2011). The midbrain dopamine system contains neurons that have widespread projections and could send reinforcement signals not only to the striatum and frontal cortex (Schultz 2002) but also to the hippocampus (Schott et al. 2004). Zaghloul et al. (2009) observed that the firing rate of neurons in the human substantia nigra was higher for unexpected gains compared to losses as early as 150 ms after feedback presentation. This finding supports the possibility that the PE effects observed in the N170, which had a latency of about 160 ms to 180 ms in the present study, could reflect the influence of the dopaminergic midbrain on the MTL, specifically in the context of feedback-based learning. However, alternative explanations are also possible. For instance, the locus coeruleus (LC), which plays a key role in norepinephrine release, also reacts to unexpected events that evoke attention like rewards, sending PE signals to other areas of the brain, for example via axons diverging to the cerebral cortex (for a review see Schultz and Dickinson 2000). Importantly, the LC also projects to the hippocampus, where its norepinephrine projections have been shown to modulate synaptic plasticity, playing a crucial role in regulating behavioral control (for reviews, see Sara 2009, 2015; Schultz and Dickinson 2000).

Nevertheless, finding a pronounced PE effect on the N170 for the prior choice between visual and not auditory stimuli and especially for delayed feedback supports the role of the N170 in the processing of visual stimuli and the idea that it specifically represents a reactivation of visual areas during feedback processing. Since signals from the MTL may evoke the reactivation of an internal representation of an event, allowing it to be linked to a later event such as the feedback in our task (Qin et al. 2007), we propose that the N170 reflects overlapping activity of the MTL and extrastriate visual areas.

For the signal in the FRN time window, accumulating evidence suggests that it is specifically modulated by positive feedback. Early studies showed that the ERP response to losses and breaking even (neither winning nor losing) can be understood as the baseline response, while rewards evoke a relative positivity (Holroyd et al. 2006; Kujawa et al. 2013). This suggests the unfolding of a positivity on gain trials more than a negativity during loss trials, in accordance with the conception of the RewP (Proudfit 2015). In line with this, it was reported that the PE affected positive feedback, while no effect emerged for negative feedback (Weber and Bellebaum 2024; Kirsch et al. 2022). In the present study, however, the signal in the FRN/RewP time range also reflected the full range of PEs, irrespective of feedback delay. The differential contribution of PE signals reflected in the FRN/RewP and the N170, and thus of the activity in neural structures underlying these components, to learning remains to be explored in future studies.

4.3 | Limitations

One aspect that limits the generalizability of our results is our predominantly female sample. A previous study found, for example, increased punishment sensitivity for women that might lead to sex differences in negative feedback processing also in our study (Santesso et al. 2011). However, the main interest in our study was in how far the modality of the feedback-preceding stimulus affects feedback processing in interaction with feedback timing, and we have no reason to believe that the effects related to this research question are affected by sex. Nevertheless, potential sex differences could be investigated in future studies.

Another concern is that the reported valence effects may partly be driven by perceptual differences between positive and negative feedback. The feedback color was not counterbalanced across participants, and this difference in saliency may have affected the FRN (Liu et al. 2014; Pfabigan et al. 2015) or, even more likely, the N170, which is associated with visual processing. However, the focus in our study was on interaction effects, which can hardly be caused by perceptual differences between negative and positive feedback. To rule out confounds of visual processing, future studies could consider using abstract feedback stimuli that are not inherently associated with valence, as implemented by Höltje and Mecklinger (2020), who used indoor vs. outdoor pictures to signal positive and negative feedback.

4.4 | Conclusions

The fact that we can use feedback to adapt our behavior, even if presented after a temporal delay, is crucial for learning and progression in our complex world. A more pronounced N170 following delayed feedback related to the choice of visual compared to auditory stimuli over the right hemisphere, combined with a representation of the PE after delayed feedback for choices of visual stimuli, supports our assumption that this component reflects modality-specific activity within higher-order visual areas of the brain. The reactivation of the chosen stimulus' representation in visual areas, possibly initiated by regions within the MTL, could be a mechanism to establish an association between the selection of a stimulus and the temporally delayed feedback.

Author Contributions

Madita Röhlinger: conceptualization, data curation, formal analysis, investigation, methodology, project administration, visualization, writing – original draft, writing – review and editing. **Christine Albrecht:** data curation, methodology, supervision, validation, writing – review and editing. **Christian Bellebaum:** conceptualization, funding acquisition, project administration, resources, supervision, validation, writing – review and editing.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

All data supporting the findings are openly accessible through the Open Science Framework at https://osf.io/brpy7/?view_only=117f83d7d4b44448aa6aa8d7e02913d2.

References

Albrecht, C., R. van de Vijver, and C. Bellebaum. 2023. "Learning New Words via Feedback-Association Between Feedback-Locked ERPs and Recall Performance-An Exploratory Study." *Psychophysiology* 60: e14324. https://doi.org/10.1111/psyp.14324.

Arbel, Y., L. Hong, T. E. Baker, and C. B. Holroyd. 2017. "It's All About Timing: An Electrophysiological Examination of Feedback-Based Learning With Immediate and Delayed Feedback." *Neuropsychologia* 99: 179–186. https://doi.org/10.1016/j.neuropsychologia.2017.03.003.

Baker, K. S., P. Johnston, N. Yamamoto, and A. J. Pegna. 2023. "Event-Related Potentials Index Prediction Error Signalling During Perceptual Processing of Emotional Facial Expressions." *Brain Topography* 36, no. 3: 419–432. https://doi.org/10.1007/s10548-023-00951-2.

Baker, K. S., A. J. Pegna, N. Yamamoto, and P. Johnston. 2021. "Attention and Prediction Modulations in Expected and Unexpected Visuospatial Trajectories." *PLoS One* 16, no. 10: e0242753. https://doi.org/10.1371/journal.pone.0242753.

Baker, T. E., and C. B. Holroyd. 2009. "Which Way Do I Go? Neural Activation in Response to Feedback and Spatial Processing in a Virtual T-Maze." *Cerebral Cortex* 19, no. 8: 1708–1722. https://doi.org/10.1093/cercor/bhn223.

Baker, T. E., and C. B. Holroyd. 2013. "The Topographical N170: Electrophysiological Evidence of a Neural Mechanism for Human Spatial Navigation." *Biological Psychology* 94, no. 1: 90–105. https://doi.org/10.1016/j.biopsycho.2013.05.004.

Baker, T. E., A. Umemoto, A. Krawitz, and C. B. Holroyd. 2015. "Rightward-Biased Hemodynamic Response of the Parahippocampal System During Virtual Navigation." *Scientific Reports* 5, no. 1: 9063. https://doi.org/10.1038/srep09063.

Bates, D., M. Mächler, B. Bolker, and S. Walker. 2015. "Fitting Linear Mixed-Effects Models Using lme4." *Journal of Statistical Software* 67, no. 1: 1–48. https://doi.org/10.18637/jss.v067.i01.

Becker, M. P. I., A. M. Nitsch, W. H. R. Miltner, and T. Straube. 2014. "A Single-Trial Estimation of the Feedback-Related Negativity and Its Relation to BOLD Responses in a Time-Estimation Task." *Journal of Neuroscience* 34, no. 8: 3005–3012. https://doi.org/10.1523/JNEUROSCI.3684-13.2014.

Bellebaum, C., and I. Daum. 2008. "Learning-Related Changes in Reward Expectancy Are Reflected in the Feedback-Related Negativity." *European Journal of Neuroscience* 27, no. 7: 1823–1835. https://doi.org/10.1111/j.1460-9568.2008.06138.x.

Bentin, S., T. Allison, A. Puce, E. Perez, and G. McCarthy. 1996. "Electrophysiological Studies of Face Perception in Humans." *Journal of Cognitive Neuroscience* 8, no. 6: 551–565. https://doi.org/10.1162/jocn.1996.8.6.551.

Brain Products GmbH. 2018. *BrainVision Analyzer 2.2 [Computer Software]*. Brain Products GmbH.

Brem, S., K. Bucher, P. Halder, et al. 2006. "Evidence for Developmental Changes in the Visual Word Processing Network Beyond Adolescence." *NeuroImage* 29, no. 3: 822–837. https://doi.org/10.1016/j.neuroimage.2005.09.023.

Burnside, R., A. G. Fischer, and M. Ullsperger. 2019. "The Feedback-Related Negativity Indexes PE in Active but Not Observational Learning." *Psychophysiology* 56, no. 9: e13389. https://doi.org/10.1111/psyp.13389.

Butler, A. J., and K. H. James. 2011. "Cross-Modal Versus Within-Modal Recall: Differences in Behavioral and Brain Responses." *Behavioural Brain Research* 224, no. 2: 387–396. https://doi.org/10.1016/j.bbr.2011.06.017.

Carlson, J. M., D. Foti, L. R. Mujica-Parodi, E. Harmon-Jones, and G. Hajcak. 2011. "Ventral Striatal and Medial Prefrontal BOLD Activation Is Correlated With Reward-Related Electrocortical Activity: A Combined ERP and fMRI Study." *NeuroImage* 57, no. 4: 1608–1616. https://doi.org/10.1016/j.neuroimage.2011.05.037.

Carreiras, M., B. C. Armstrong, M. Perea, and R. Frost. 2014. "The What, When, Where, and How of Visual Word Recognition." *Trends in Cognitive Sciences* 18, no. 2: 90–98. https://doi.org/10.1016/j.tics.2013.11.005.

Chau, B. K., H. Jarvis, C. K. Law, and T. T. J. Chong. 2018. "Dopamine and Reward: A View From the Prefrontal Cortex." *Behavioural Pharmacology* 29, no. 7: 569–583. https://doi.org/10.1097/FBP.0000000000000424.

Chuhma, N., S. J. Oh, and S. Rayport. 2023. "The Dopamine Neuron Synaptic Map in the Striatum." *Cell Reports* 42, no. 3: 112204. https://doi.org/10.1016/j.celrep.2023.112204.

Cohen, L., S. Lehéricy, F. Chochon, C. Lemer, S. Rivaud, and S. Dehaene. 2002. "Language-Specific Tuning of Visual Cortex? Functional Properties of the Visual Word Form Area." *Brain* 125, no. 5: 1054–1069. https://doi.org/10.1093/brain/awf094.

Cohen, M. X., J. F. Cavanagh, and H. A. Slagter. 2011. "Event-Related Potential Activity in the Basal Ganglia Differentiates Rewards From Nonrewards: Temporospatial Principal Components Analysis and Source Localization of the Feedback Negativity: Commentary." *Human Brain Mapping* 32, no. 12: 2270–2271. https://doi.org/10.1002/hbm.21358.

Damier, P., E. C. Hirsch, Y. Agid, and A. M. Graybiel. 1999. "The Substantia Nigra of the Human Brain. II. Patterns of Loss of Dopamine-Containing Neurons in Parkinson's Disease." *Brain* 122, no. 8: 1437–1448. https://doi.org/10.1093/brain/122.8.1437.

Deffke, I., T. Sander, J. Heidenreich, et al. 2007. "Meg/Eeg Sources of the 170-Ms Response to Faces Are Co-Localized in the Fusiform Gyrus." *NeuroImage* 35, no. 4: 1495–1501. https://doi.org/10.1016/j.neuroimage.2007.01.034.

Delgado, M. R. 2007. "Reward-Related Responses in the Human Striatum." *Annals of the New York Academy of Sciences* 1104, no. 1: 70–88. https://doi.org/10.1196/annals.1390.002.

Dickerson, K. C., J. Li, and M. R. Delgado. 2011. "Parallel Contributions of Distinct Human Memory Systems During Probabilistic Learning." *NeuroImage* 55, no. 1: 266–276. https://doi.org/10.1016/j.neuroimage.2010.10.080.

Fischer, A. G., and M. Ullsperger. 2013. "Real and Fictive Outcomes Are Processed Differently but Converge on a Common Adaptive Mechanism." *Neuron* 79, no. 6: 1243–1255. https://doi.org/10.1016/j.neuron.2013.07.006.

Foerde, K., E. Race, M. Verfaellie, and D. Shohamy. 2013. "A Role for the Medial Temporal Lobe in Feedback-Driven Learning: Evidence From

Amnesia." *Journal of Neuroscience* 33, no. 13: 5698–5704. https://doi.org/10.1523/JNEUROSCI.5217-12.2013.

Foerde, K., and D. Shohamy. 2011. "Feedback Timing Modulates Brain Systems for Learning in Humans." *Journal of Neuroscience* 31, no. 37: 13157–13167. https://doi.org/10.1523/JNEUROSCI.2701-11.2011.

Foti, D., A. Weinberg, E. M. Bernat, and G. H. Proudfit. 2015. "Anterior Cingulate Activity to Monetary Loss and Basal Ganglia Activity to Monetary Gain Uniquely Contribute to the Feedback Negativity." *Clinical Neurophysiology* 126, no. 7: 1338–1347. https://doi.org/10.1016/j.clinph.2014.08.025.

Foti, D., A. Weinberg, J. Dien, and G. Hajcak. 2011. "Event-Related Potential Activity in the Basal Ganglia Differentiates Rewards From Nonrewards: Temporospatial Principal Components Analysis and Source Localization of the Feedback Negativity." *Human Brain Mapping* 32, no. 12: 2207–2216. https://doi.org/10.1002/hbm.21182.

Frank, M. J., L. C. Seeberger, and R. C. O'reilly. 2004. "By Carrot or by Stick: Cognitive Reinforcement Learning in Parkinsonism." *Science* 306, no. 5703: 1940–1943. https://doi.org/10.1126/science.1102941.

Gao, C., S. Conte, J. E. Richards, W. Xie, and T. Hanayik. 2019. "The Neural Sources of N170: Understanding Timing of Activation in Face-Selective Areas." *Psychophysiology* 56, no. 6: e13336. https://doi.org/10.1111/psyp.13336.

Haber, S. N., and B. Knutson. 2010. "The Reward Circuit: Linking Primate Anatomy and Human Imaging." *Neuropsychopharmacology* 35, no. 1: 4–26. https://doi.org/10.1038/npp.2009.129.

Hauser, T. U., R. Iannaccone, P. Stämpfli, et al. 2014. "The Feedback-Related Negativity (FRN) Revisited: New Insights Into the Localization, Meaning and Network Organization." *NeuroImage* 84: 159–168. https://doi.org/10.1016/j.neuroimage.2013.08.028.

Herholz, S. C., A. R. Halpern, and R. J. Zatorre. 2012. "Neuronal Correlates of Perception, Imagery, and Memory for Familiar Tunes." *Journal of Cognitive Neuroscience* 24, no. 6: 1382–1397. https://doi.org/10.1162/jocn_a_00216.

Holroyd, C. B., and M. G. H. Coles. 2002. "The Neural Basis of Human Error Processing: Reinforcement Learning, Dopamine, and the Error-Related Negativity." *Psychological Review* 109, no. 4: 679–709. https://doi.org/10.1037/0033-295X.109.4.679.

Holroyd, C. B., G. Hajcak, and J. T. Larsen. 2006. "The Good, the Bad and the Neutral: Electrophysiological Responses to Feedback Stimuli." *Brain Research* 1105: 93–101. https://doi.org/10.1016/j.brainres.2005.12.015.

Höltje, G., and A. Mecklinger. 2020. "Feedback Timing Modulates Interactions Between Feedback Processing and Memory Encoding: Evidence From Event-Related Potentials." *Cognitive, Affective, & Behavioral Neuroscience* 20, no. 2: 250–264. https://doi.org/10.3758/s13415-019-00765-5.

Iidaka, T., A. Matsumoto, K. Haneda, T. Okada, and N. Sadato. 2006. "Hemodynamic and Electrophysiological Relationship Involved in Human Face Processing: Evidence From a Combined fMRI-ERP Study." *Brain and Cognition* 60, no. 2: 176–186. https://doi.org/10.1016/j.bandc.2005.11.004.

Itier, R. J., and M. J. Taylor. 2004. "N170 or N1? Spatiotemporal Differences Between Object and Face Processing Using ERPs." *Cerebral Cortex* 14, no. 2: 132–142. https://doi.org/10.1093/cercor/bhg111.

Jocham, G., K. H. Brodersen, A. O. Constantinescu, et al. 2016. "Reward-Guided Learning With and Without Causal Attribution." *Neuron* 90, no. 1: 177–190. https://doi.org/10.1016/j.neuron.2016.02.018.

Kanwisher, N., J. McDermott, and M. M. Chun. 1997. "The Fusiform Face Area: A Module in Human Extrastriate Cortex Specialized for Face Perception." *Journal of Neuroscience* 17, no. 11: 4302–4311. https://doi.org/10.1523/JNEUROSCI.17-11-04302.1997.

Kim, S., and Y. Arbel. 2019. "Immediate and Delayed Auditory Feedback in Declarative Learning: An Examination of the Feedback Related Event Related Potentials." *Neuropsychologia* 129: 255–262. https://doi.org/10.1016/j.neuropsychologia.2019.04.001.

Kirsch, F., H. Kirschner, A. G. Fischer, T. A. Klein, and M. Ullsperger. 2022. "Disentangling Performance-Monitoring Signals Encoded in Feedback-Related EEG Dynamics." *NeuroImage* 257: 119322. https://doi.org/10.1016/j.neuroimage.2022.119322.

Kloth, N., R. J. Itier, and S. R. Schweinberger. 2013. "Combined Effects of Inversion and Feature Removal on N170 Responses Elicited by Faces and Car Fronts." *Brain and Cognition* 81, no. 3: 321–328. https://doi.org/10.1016/j.bandc.2013.01.002.

Kujawa, A., E. Smith, C. Luhmann, and G. Hajcak. 2013. "The Feedback Negativity Reflects Favorable Compared to Nonfavorable Outcomes Based on Global, Not Local, Alternatives." *Psychophysiology* 50: 134–138. https://doi.org/10.1111/psyp.12002.

Lefebvre, G., M. Lebreton, F. Meyniel, S. Bourgeois-Gironde, and S. Palminteri. 2017. "Behavioural and Neural Characterization of Optimistic Reinforcement Learning." *Nature Human Behaviour* 1, no. 4: 0067. https://doi.org/10.1038/s41562-017-0067.

Liu, Y., L. D. Nelson, E. M. Bernat, and W. J. Gehring. 2014. "Perceptual Properties of Feedback Stimuli Influence the Feedback-Related Negativity in the Flanker Gambling Task." *Psychophysiology* 51, no. 8: 782–788. https://doi.org/10.1111/psyp.12216.

Luck, S. J. 2014. *An Introduction to the Event-Related Potential Technique*. MIT Press.

Maurer, L. K., M. Joch, M. Hegele, H. Maurer, and H. Müller. 2022. "Relevance of Predictive and Postdictive Error Information in the Course of Motor Learning." *Neuroscience* 486: 77–90. https://doi.org/10.1016/j.neuroscience.2021.05.007.

Meteyard, L., and R. A. Davies. 2020. "Best Practice Guidance for Linear Mixed-Effects Models in Psychological Science." *Journal of Memory and Language* 112: 104092. https://doi.org/10.1016/j.jml.2020.104092.

Miltner, W. H., C. H. Braun, and M. G. Coles. 1997. "Event-Related Brain Potentials Following Incorrect Feedback in a Time-Estimation Task: Evidence for a "Generic" Neural System for Error Detection." *Journal of Cognitive Neuroscience* 9, no. 6: 788–798. https://doi.org/10.1162/jocn.1997.9.6.788.

Mushtaq, F., S. D. McDougle, M. P. Craddock, et al. 2022. "Distinct Neural Signatures of Outcome Monitoring After Selection and Execution Errors." *Journal of Cognitive Neuroscience* 34, no. 5: 748–765. https://doi.org/10.1162/jocn_a_01824.

Neurobehavioral Systems Inc. 2020. *Presentation (Version 22) [Computer Software]*. Neurobehavioral Systems Inc. https://www.neurobs.com/.

Nieuwenhuis, S., C. B. Holroyd, N. Mol, and M. G. H. Coles. 2004. "Reinforcement-Related Brain Potentials From Medial Frontal Cortex: Origins and Functional Significance." *Neuroscience and Biobehavioral Reviews* 28, no. 4: 441–448. https://doi.org/10.1016/j.neubiorev.2004.05.003.

Nieuwenhuis, S., H. A. Slagter, N. J. A. Von Geusau, D. J. Heslenfeld, and C. B. Holroyd. 2005. "Knowing Good From Bad: Differential Activation of Human Cortical Areas by Positive and Negative Outcomes." *European Journal of Neuroscience* 21, no. 11: 3161–3168. https://doi.org/10.1111/j.1460-9568.2005.04152.x.

Oerlemans, J., R. J. Alejandro, D. Van Roost, et al. 2025. "Unravelling the Origin of Reward Positivity: A Human Intracranial Event-Related Brain Potential Study." *Brain* 148, no. 1: 199–211. https://doi.org/10.1093/brain/awae259.

Oldehinkel, M., A. Llera, M. Faber, et al. 2022. "Mapping Dopaminergic Projections in the Human Brain With Resting-State fMRI." *eLife* 11: e71846. https://doi.org/10.7554/eLife.71846.

OpenAI. 2023. *GPT-4* [*Large Language Model*]. https://openai.com/gpt-4.

Peterburs, J., S. Kobza, and C. Bellebaum. 2016. "Feedback Delay Gradually Affects Amplitude and Valence Specificity of the Feedback-Related Negativity (FRN)." *Psychophysiology* 53, no. 2: 209–215. https://doi.org/10.1111/psyp.12560.

Pfabigan, D. M., U. Sailer, and C. Lamm. 2015. "Size Does Matter! Perceptual Stimulus Properties Affect Event-Related Potentials During Feedback Processing." *Psychophysiology* 52, no. 9: 1238–1247. https://doi.org/10.1111/psyp.12458.

Pleger, B., F. Blankenburg, C. C. Ruff, J. Driver, and R. J. Dolan. 2008. "Reward Facilitates Tactile Judgments and Modulates Hemodynamic Responses in Human Primary Somatosensory Cortex." *Journal of Neuroscience* 28, no. 33: 8161–8168. https://doi.org/10.1523/JNEUROSCI.1093-08.2008.

Pleger, B., C. C. Ruff, F. Blankenburg, S. Klöppel, J. Driver, and R. J. Dolan. 2009. "Influence of Dopaminergically Mediated Reward on Somatosensory Decision-Making." *PLoS Biology* 7, no. 7: e1000164. https://doi.org/10.1371/journal.pbio.1000164.

Proudfit, G. H. 2015. "The Reward Positivity: From Basic Research on Reward to a Biomarker for Depression." *Psychophysiology* 52, no. 4: 449–459. https://doi.org/10.1111/psyp.12370.

Qin, S., C. Piekema, K. M. Petersson, B. Han, J. Luo, and G. Fernández. 2007. "Probing the Transformation of Discontinuous Associations Into Episodic Memory: An Event-Related fMRI Study." *NeuroImage* 38, no. 1: 212–222. https://doi.org/10.1016/j.neuroimage.2007.07.020.

Quené, H., and H. Van den Bergh. 2004. "On Multi-Level Modeling of Data From Repeated Measures Designs: A Tutorial." *Speech Communication* 43, no. 1–2: 103–121. https://doi.org/10.1016/j.specom.2004.02.004.

Rossion, B. 2014. "Understanding Face Perception by Means of Human Electrophysiology." *Trends in Cognitive Sciences* 18, no. 6: 310–318. https://doi.org/10.1016/j.tics.2014.02.013.

Sambrook, T. D., and J. Goslin. 2015. "A Neural Reward Prediction Error Revealed by a Meta-Analysis of ERPs Using Great Grand Averages." *Psychological Bulletin* 141, no. 1: 213–235. https://doi.org/10.1037/bul0000006.

Santesso, D. L., A. Dzyundzyak, and S. J. Segalowitz. 2011. "Age, Sex and Individual Differences in Punishment Sensitivity: Factors Influencing the Feedback-Related Negativity." *Psychophysiology* 48, no. 11: 1481–1489. https://doi.org/10.1111/j.1469-8986.2011.01229.x.

Sara, S. J. 2009. "The Locus Coeruleus and Noradrenergic Modulation of Cognition." *Nature Reviews Neuroscience* 10, no. 3: 211–223. https://doi.org/10.1038/nrn2573.

Sara, S. J. 2015. "Locus Coeruleus in Time With the Making of Memories." *Current Opinion in Neurobiology* 35: 87–94. https://doi.org/10.1016/j.conb.2015.07.004.

Schiffer, A. M., T. Muller, N. Yeung, and F. Waszak. 2014. "Reward Activates Stimulus-Specific and Task-Dependent Representations in Visual Association Cortices." *Journal of Neuroscience* 34, no. 47: 15610–15620. https://doi.org/10.1523/JNEUROSCI.1640-14.2014.

Schott, B. H., D. B. Sellner, C.-J. Lauer, et al. 2004. "Activation of Midbrain Structures by Associative Novelty and the Formation of Explicit Memory in Humans." *Learning & Memory (Cold Spring Harbor, N.Y.)* 11, no. 4: 383–387. https://doi.org/10.1101/lm.75004.

Schultz, W. 2002. "Getting Formal With Dopamine and Reward." *Neuron* 36, no. 2: 241–263. https://doi.org/10.1016/S0896-6273(02)00967-4.

Schultz, W., P. Dayan, and P. R. Montague. 1997. "A Neural Substrate of Prediction and Reward." *Science* 275, no. 5306: 1593–1599. https://doi.org/10.1126/science.275.5306.1593.

Schultz, W., and A. Dickinson. 2000. "Neuronal Coding of Prediction Errors." *Annual Review of Neuroscience* 23, no. 1: 473–500. https://doi.org/10.1146/annurev.neuro.23.1.473.

Singer, A. C., and L. M. Frank. 2009. "Rewarded Outcomes Enhance Reactivation of Experience in the Hippocampus." *Neuron* 64, no. 6: 910–921. https://doi.org/10.1016/j.neuron.2009.11.016.

Staresina, B. P., and L. Davachi. 2009. "Mind the Gap: Binding Experiences Across Space and Time in the Human Hippocampus." *Neuron* 63, no. 2: 267–276. https://doi.org/10.1016/j.neuron.2009.06.024.

The MathWorks, Inc. 2021. *MATLAB R2021a [Computer Software]*. MathWorks, Inc.

The R Foundation. 2021. *R: A Language and Environment for Statistical Computing (Version 4.2.2) [Computer Software]*. R Foundation for Statistical Computing. https://www.R-project.org/.

Thiebaut de Schotten, M., F. Dell'Acqua, S. Forkel, et al. 2011. "A Lateralized Brain Network for Visuo-Spatial Attention." *Nature Neuroscience* 14, no. 10: 1245–1246. https://doi.org/10.1038/npre.2011.5549.1.

Voeten, C. 2020. *Using 'Buildmer' to Automatically Find & Compare Maximal (Mixed) Models*.

Wang, K. S., D. V. Smith, and M. R. Delgado. 2016. "Using fMRI to Study Reward Processing in Humans: Past, Present, and Future." *Journal of Neurophysiology* 115, no. 3: 1664–1678. https://doi.org/10.1152/jn.00333.2015.

Weber, C., and C. Bellebaum. 2024. "Prediction-Error-Dependent Processing of Immediate and Delayed Positive Feedback." *Scientific Reports* 14, no. 1: 9674. https://doi.org/10.1038/s41598-024-60328-8.

Weinberg, A., C. C. Luhmann, J. N. Bress, and G. Hajcak. 2012. "Better Late Than Never? The Effect of Feedback Delay on ERP Indices of Reward Processing." *Cognitive, Affective, & Behavioral Neuroscience* 12, no. 4: 671–677. https://doi.org/10.3758/s13415-012-0104-z.

Weiner, K. S., and K. Zilles. 2016. "The Anatomical and Functional Specialization of the Fusiform Gyrus." *Neuropsychologia* 83: 48–62. https://doi.org/10.1016/j.neuropsychologia.2015.06.033.

Weismüller, B., and C. Bellebaum. 2016. "Expectancy Affects the Feedback-Related Negativity (FRN) for Delayed Feedback in Probabilistic Learning." *Psychophysiology* 53, no. 11: 1739–1750. https://doi.org/10.1111/psyp.12738.

Weismüller, B., J. Kullmann, M. Hoenen, and C. Bellebaum. 2019. "Effects of Feedback Delay and Agency on Feedback-Locked Beta and Theta Power During Reinforcement Learning." *Psychophysiology* 56, no. 10: e13428. https://doi.org/10.1111/psyp.13428.

Williams, C. C., C. D. Hassall, T. Lindenbach, and O. E. Krigolson. 2020. "Reward Prediction Errors Reflect an Underlying Learning Process That Parallels Behavioural Adaptations: A Trial-To-Trial Analysis." *Computational Brain & Behavior* 3: 189–199. https://doi.org/10.1007/s42113-019-00069-4.

Yagishita, S., A. Hayashi-Takagi, G. C. Ellis-Davies, H. Urakubo, S. Ishii, and H. Kasai. 2014. "A Critical Time Window for Dopamine Actions on the Structural Plasticity of Dendritic Spines." *Science* 345, no. 6204: 1616–1620. https://doi.org/10.1126/science.1255514.

Yovel, G. 2016. "Neural and Cognitive Face-Selective Markers: An Integrative Review." *Neuropsychologia* 83: 5–13. https://doi.org/10.1016/j.neuropsychologia.2015.09.026.

Zaghloul, K. A., J. A. Blanco, C. T. Weidemann, et al. 2009. "Human Substantia Nigra Neurons Encode Unexpected Financial Rewards." *Science* 323, no. 5920: 1496–1499. https://doi.org/10.1126/science.1167342.

Zhang, Y., I. W. Wu, S. Buckley, et al. 2015. "Diffusion Tensor Imaging of the Nigrostriatal Fibers in Parkinson's Disease: DTI of the Nigrostriatal

Fibers in PD." *Movement Disorders* 30, no. 9: 1229–1236. https://doi.org/10.1002/mds.26251.

Zottoli, T. M., and J. Grose-Fifer. 2012. "The Feedback-Related Negativity (FRN) in Adolescents." *Psychophysiology* 49, no. 3: 413–420. https://doi.org/10.1111/j.1469-8986.2011.01312.x.

Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Supplementary Material

Table S1

Maximal (G)LME models for the analysis of behavioral and EEG data

Analysis	Model formula
GLME behavioral analysis	
	*Accuracy ~ 1 + Feedback Timing * Stimulus Modality * Block + (1 + Feedback Timing * Stimulus Modality * Block | Subject)*
LME N170 analysis	
	*N170 ~ 1 + Feedback Timing * Feedback Valence * Stimulus Modality * PE * Electrode + (1 + Electrode + Feedback Timing + Stimulus Modality + Feedback Valence + Feedback Timing : Electrode + Feedback Valence : Electrode + Feedback Timing : Stimulus Modality + Feedback Timing : Feedback Valence | Subject)*
LME FRN analysis	
	*FRN ~ 1 + Feedback Timing * Feedback Valence * Stimulus Modality * PE + (1 + Feedback Timing + Stimulus Modality + Feedback Valence + Feedback Timing : Stimulus Modality + Feedback Timing : Feedback Valence + PE + Feedback Valence : PE | Subject)*

Note. GLME = (generalized) linear mixed effects. Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), Stimulus Modality (visual [-0.5] vs. auditory [0.5]), PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) and Electrode (P7 [-0.5] vs. P8 [0.5]).

Table S2

Results for the GLME analysis on accuracy

Effect	β-estimate	*SE*	*z*	*p*	
Block	1.41	0.21	6.71	< .001	***
Modality	- 0.14	0.20	- 0.69	.493	
Timing	0.16	0.11	1.46	.144	
Block x Modality	- 0.40	0.27	- 1.48	.140	
Modality x Timing	0.25	0.25	0.99	.324	
Block x Timing	0.18	0.21	0.84	.399	
Block x Modality x Timing	- 0.52	0.48	- 1.08	.279	

Note. GLME = generalized linear mixed effects, *SE* = standard error, Modality = Stimulus Modality, Timing = Feedback Timing. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Block (1 [-0.5], 2 [-0.167], 3 [0.167], 4 [0.5]), Stimulus Modality (visual [-0.5] vs. auditory [0.5]) and Feedback Timing (immediate [-0.5] vs. delayed [0.5]).

*** $p < 0.001$

Table S3

Results for the LME analysis on the N170 amplitude

Effect	β-estimate	*SE*	*df*	*t*	*p*	
Timing	0.52	0.36	38.41	1.45	.156	
Valence	-0.39	0.23	45.02	-1.66	.104	
Modality	0.15	0.28	41.95	0.55	.583	
PE	-0.38	0.20	13196.94	-1.90	.057	.
Electrode	-1.67	0.58	35.55	-2.85	.007	**
Timing x Valence	-0.91	0.28	68.40	-3.22	.002	**
Timing x Modality	1.67	0.40	51.49	4.17	< .001	***
Valence x Modality	0.22	0.23	38090.27	0.92	.356	
Timing x PE	0.26	0.38	1842.60	0.69	.491	
Valence x PE	-2.18	0.42	25580.32	-5.17	< .001	***
Modality x PE	0.85	0.38	28767.85	2.23	.026	*
Timing x Electrode	0.33	0.44	45.89	0.76	.453	
Valence x Electrode	-0.75	0.43	47.26	-1.75	.087	.
Modality x Electrode	-0.33	0.23	43198.93	-1.41	.159	
PE x Electrode	-0.28	0.40	14337.18	-0.72	.472	
Timing x Valence x Modality	0.35	0.46	25994.03	0.77	.444	
Timing x Valence x PE	-1.55	0.83	15617.27	-1.86	.063	.
Timing x Modality x PE	0.70	0.75	11251.67	0.93	.351	
Valence x Modality x PE	1.23	0.82	8791.51	1.50	.135	
Timing x Valence x Electrode	-0.16	0.46	42974.28	-0.35	.730	
Timing x Modality x Electrode	1.87	0.46	41676.48	4.03	< .001	***
Valence x Modality x Electrode	0.27	0.46	41751.62	0.59	.553	
Timing x PE x Electrode	-1.00	0.75	42470.22	-1.35	.178	
Valence x PE x Electrode	-2.66	0.81	41145.40	-3.28	.001	**
Modality x PE x Electrode	-0.16	0.75	36239.92	-0.22	.828	
Timing x Valence x Modality x PE	1.57	1.64	6468.07	0.96	.339	
Timing x Valence x Modality x Electrode	-0.93	0.92	43204.28	-1.01	.313	
Timing x Valence x PE x Electrode	-0.35	1.59	13032.80	-0.22	.824	
Timing x Modality x PE x Electrode	1.05	1.49	42818.31	0.71	.480	
Valence x Modality x PE x Electrode	0.52	1.53	42657.98	0.34	.734	
Timing x Valence x Modality x PE x Electrode	6.19	3.04	35302.83	2.04	.042	*

Note. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, Modality = Stimulus Modality, PE = PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), Stimulus Modality (visual [-0.5] vs. auditory [0.5]), PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) and Electrode (P7 [-0.5] vs. P8 [0.5]).
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, $p < 0.1$

Table S4

Results of the LME analysis on the FRN amplitude

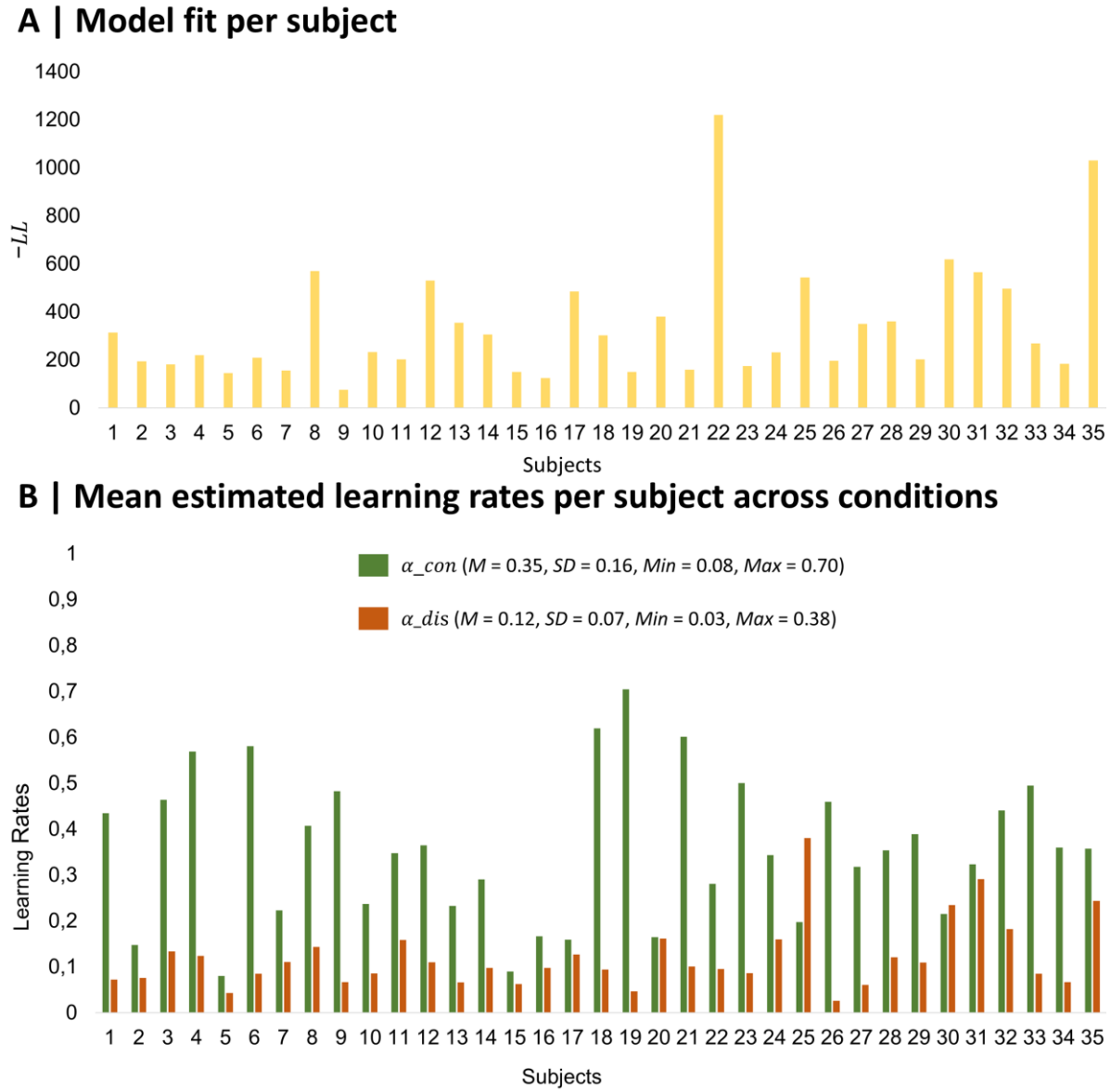
Effect	β-estimate	*SE*	*df*	*t*	*p*	
Timing	0.07	0.30	42.71	0.23	.822	
Valence	2.40	0.27	31.69	8.88	< .001	***
Modality	0.62	0.24	48.93	2.55	.014	*
PE	0.17	0.33	22.67	0.51	.613	
Timing x Valence	-1.07	0.38	57.33	-2.85	.006	**
Timing x Modality	-1.38	0.43	54.65	-3.20	.002	**
Valence x Modality	0.32	0.27	13460.59	1.19	.236	
Timing x PE	-0.19	0.45	2707.83	-0.43	.664	
Valence x PE	7.44	1.01	26.42	7.36	< .001	***
Modality x PE	0.20	0.44	10712.29	0.46	.644	
Timing x Valence x Modality	0.08	0.53	16207.28	0.16	.876	
Timing x Valence x PE	1.18	0.97	11120.41	1.22	.222	
Timing x Modality x PE	-1.09	0.87	10237.58	-1.26	.207	
Valence x Modality x PE	1.32	0.96	6536.76	1.37	.171	
Timing x Valence x Modality x PE	-2.38	1.90	6027.52	-1.25	.212	

Note. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, Modality = Stimulus Modality, PE = PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), Stimulus Modality (visual [-0.5] vs. auditory [0.5]) and the PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean).

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Figure S1

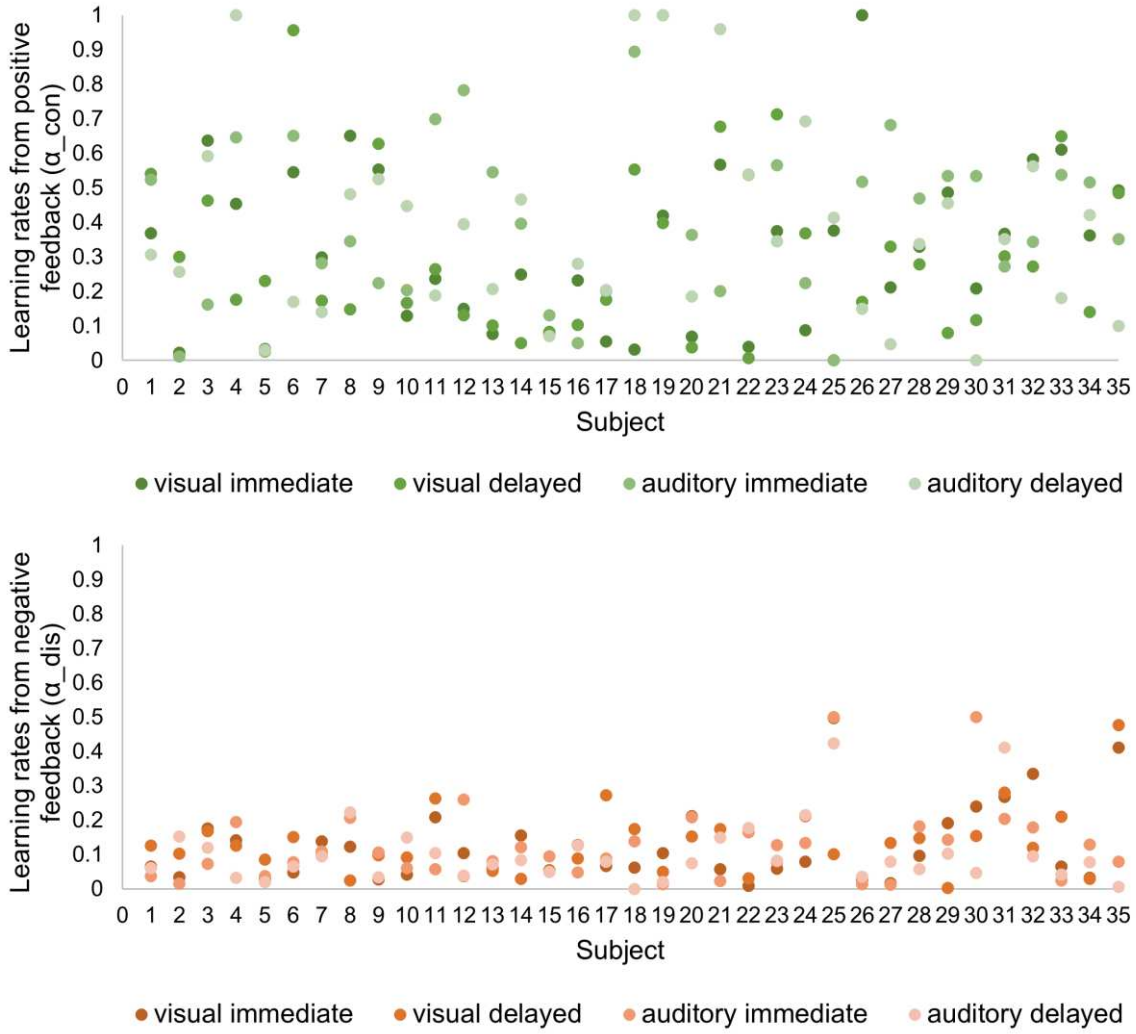
Descriptive data for the computational prediction error modeling



Note. **A** Model fit: $-LL$ = negative summed log-likelihood as measure for the model's goodness of fit per subject across conditions. **B** Learning Rates: α_{con} = estimated learning rate from positive feedback that confirms the choice, α_{dis} = estimated learning rate from negative feedback that disconfirms the choice.

Figure S2

Estimated learning rates from computational prediction error modeling

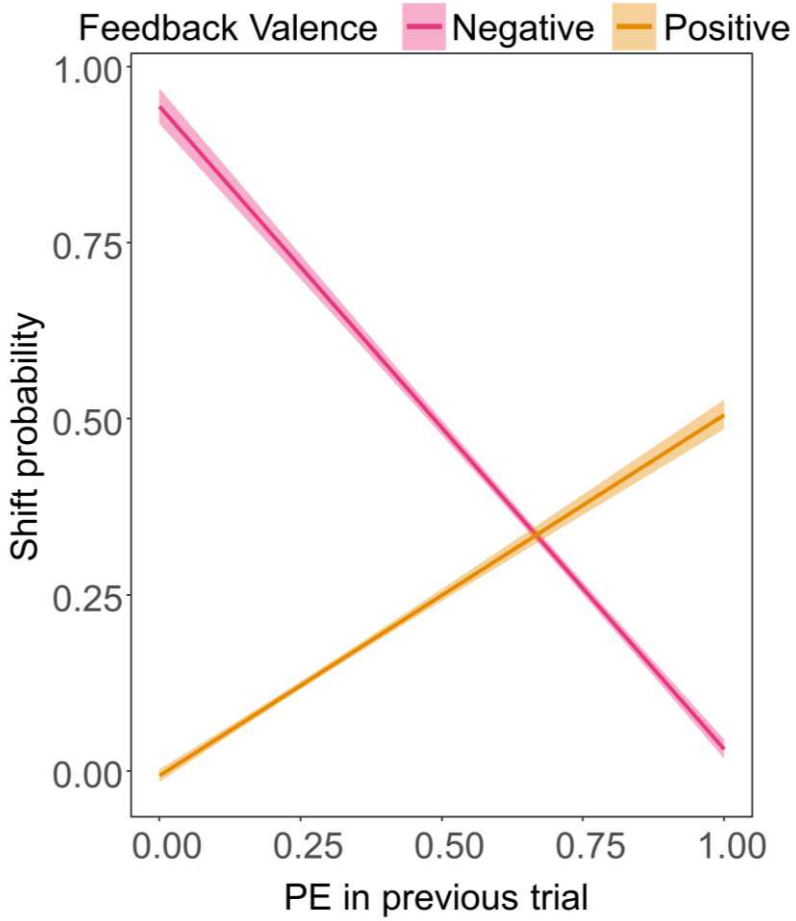


Note. Illustrated are the mean learning rates per subject for learning from positive feedback (α_{con}) and negative feedback (α_{dis}) for the respective Stimulus Modality (visual vs. auditory) and Feedback Timing (immediate vs. delayed) conditions as estimated by the computational prediction error modeling.

Win-stay vs. lose-shift analysis

Our participants generally tended to stick with their previous stimulus choice: In 80% of trials, they selected the same stimulus as in the preceding trial and switched to the alternative stimulus only 20% of the time. Notably, their behavior was influenced by prior feedback: After losing, they maintained their choice in 72.62% of cases, whereas after winning, this percentage increased to 86.28%, which means that they switched more often after a loss or negative feedback. This effect was indeed significant, as revealed by a GLME analysis on the probability of switch responses, including Feedback Valence and absolute PE in the previous trial, as well as their interaction, as fixed factors (random effects were specified as described for the GLME model of the behavioral data in the main text): main effect of Feedback Valence, $z = -11.23$, $p < .001$, $b = -2.30$. There also was a significant main effect of the absolute PE in the previous trial, $z = -3.68$, $p < .001$, $b = -0.81$, which was further explained by a significant interaction between Feedback Valence and absolute PE in the previous trial, $z = 7{,}34$, $p < .001$, $b = 14.88$. Figure S3 illustrates the underlying pattern in the data: The more unexpected the negative feedback in the previous trial, the greater the tendency of participants to persist with their previous choice. Conversely, the more unexpected positive feedback in the previous trial, the higher the probability of a shift.

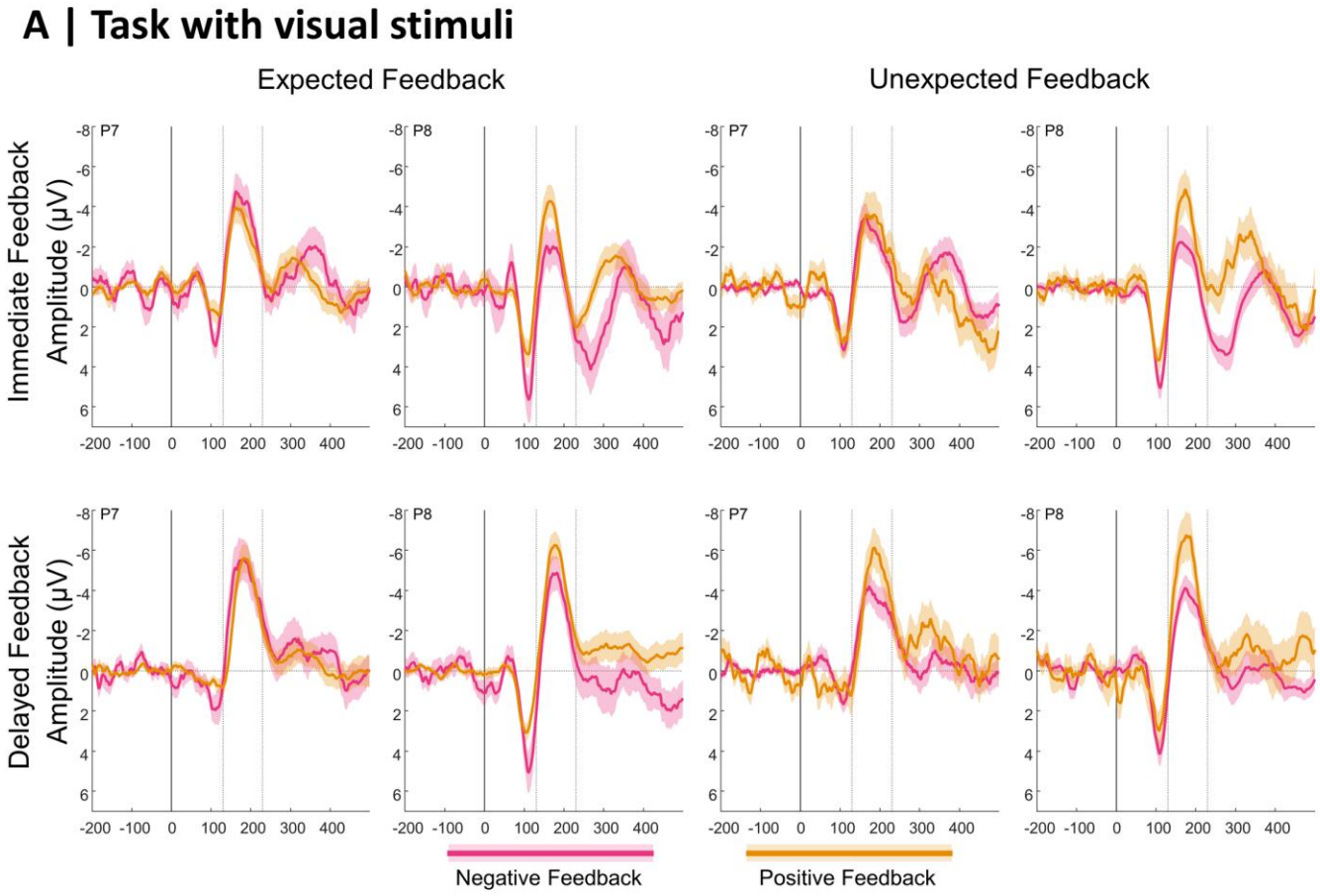
Figure S3
Win-stay and lose-shift behavior

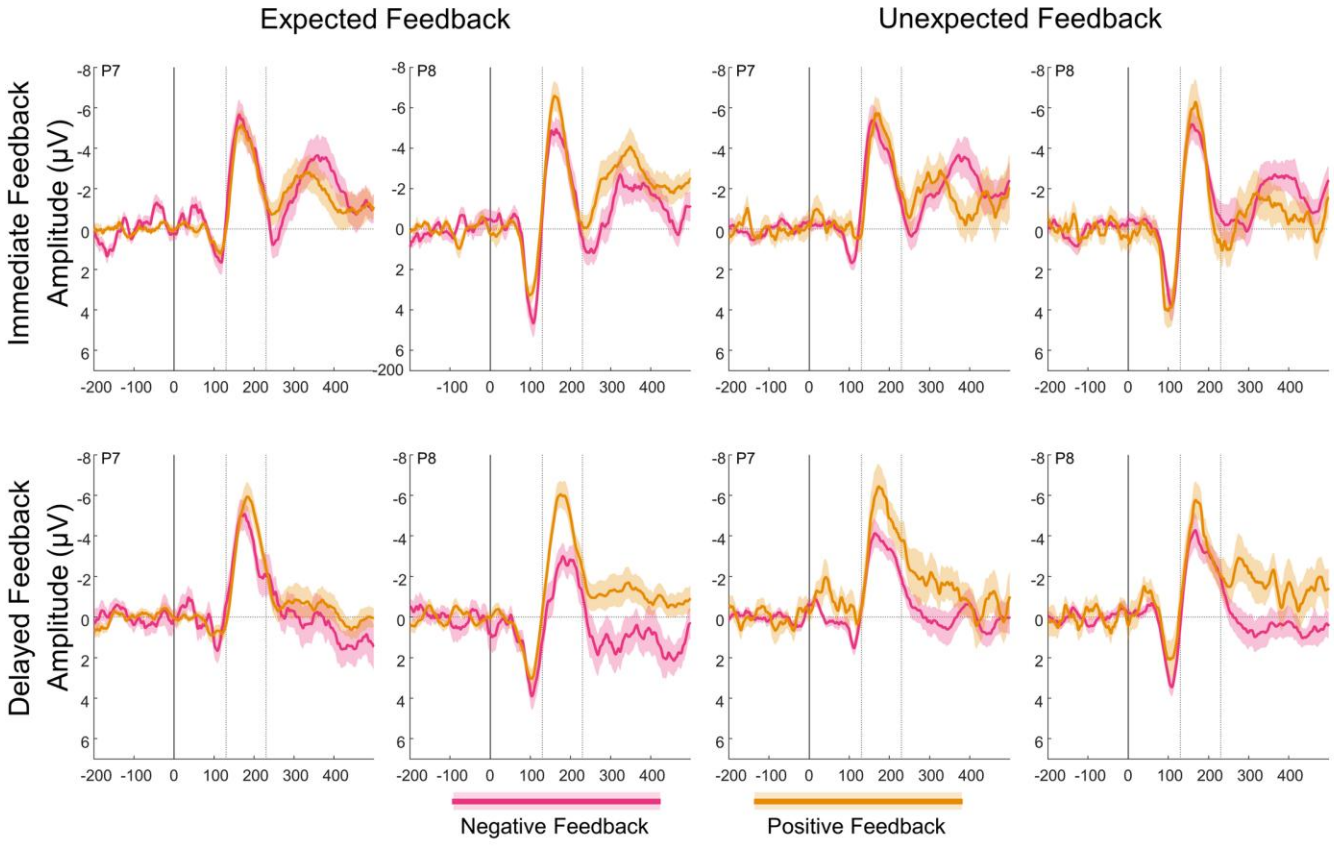


Note. PE = prediction error. The y-axis indicates the probability of a shift. A shift value = 0 means that participants stayed with the stimulus they had chosen in the previous trial, a shift value = 1 means that they chose the other stimulus.

Figure S4

Grand Averages for the N170 following Expected and Unexpected Feedback

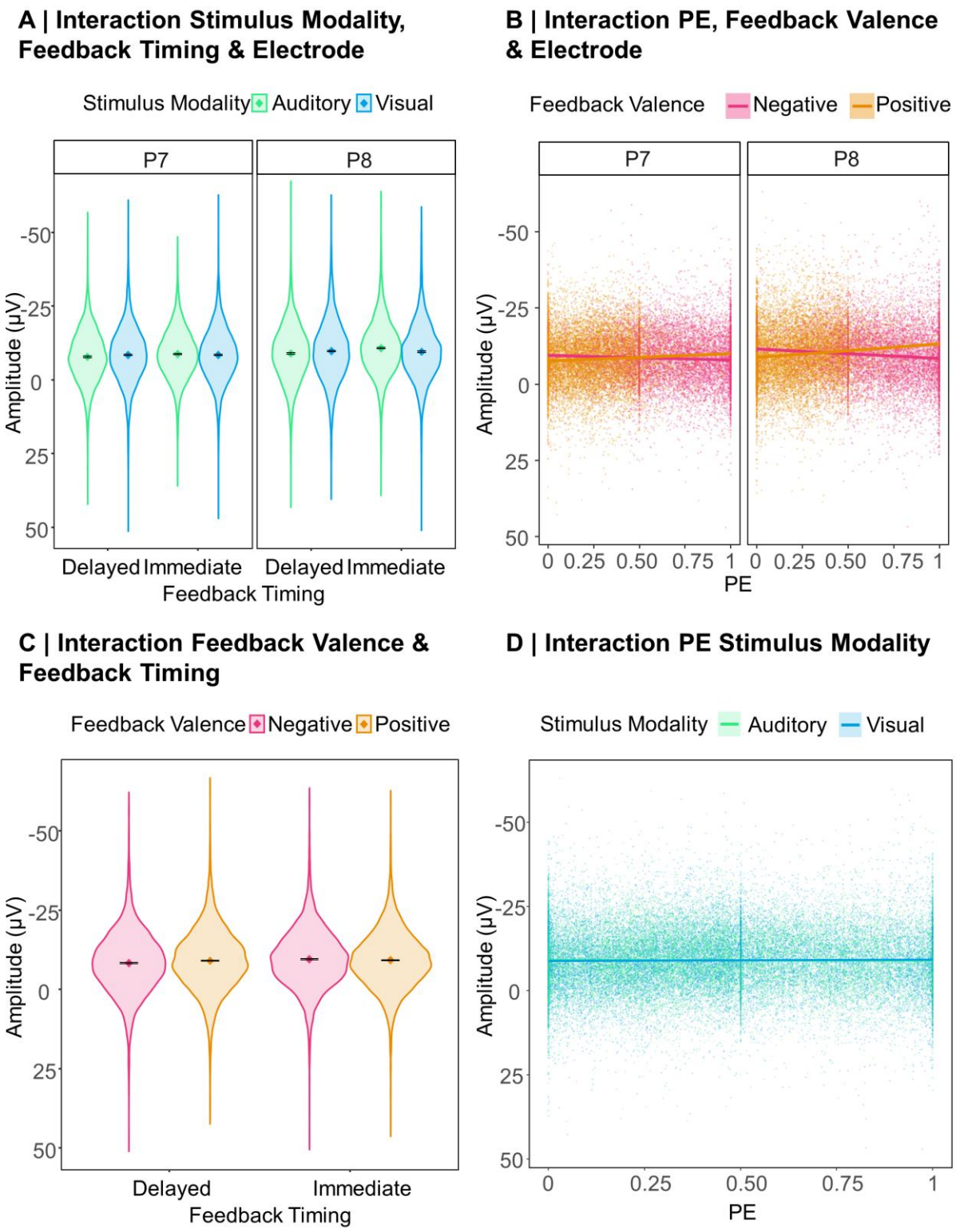

A | Task with visual stimuli
Expected Feedback
Unexpected Feedback
Immediate Feedback Amplitude (μV)
Delayed Feedback Amplitude (μV)
P7
P8
Negative Feedback
Positive Feedback


B | Task with auditory stimuli
Expected Feedback
Unexpected Feedback
Immediate Feedback Amplitude (μV)
Delayed Feedback Amplitude (μV)
P7
P8
Negative Feedback
Positive Feedback

Note. Dotted lines indicate the time window used for the N170 peak detection. Shaded areas indicate standard errors. Unexpected feedback corresponds to absolute PE values > 0.50, while expected feedback corresponds to absolute PE values ≤ 0.50.

Figure S5

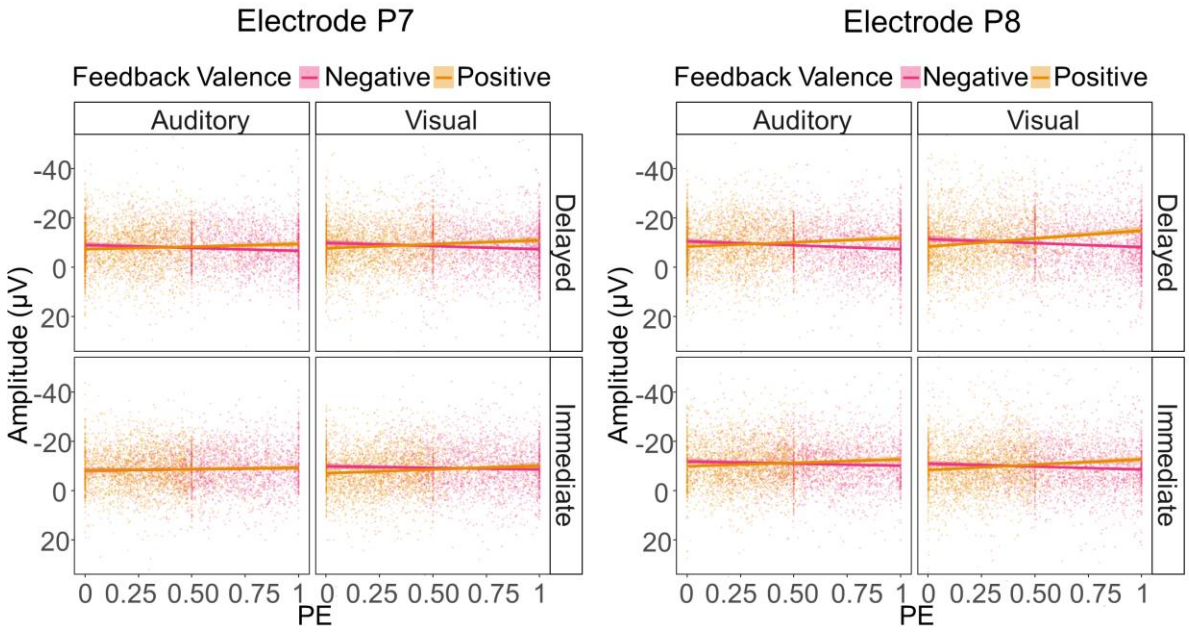
Descriptive data patterns and distributions underlying the N170 analysis



Error bars indicate a 95% confidence interval. Dots represent single-trial data points.

Figure S6

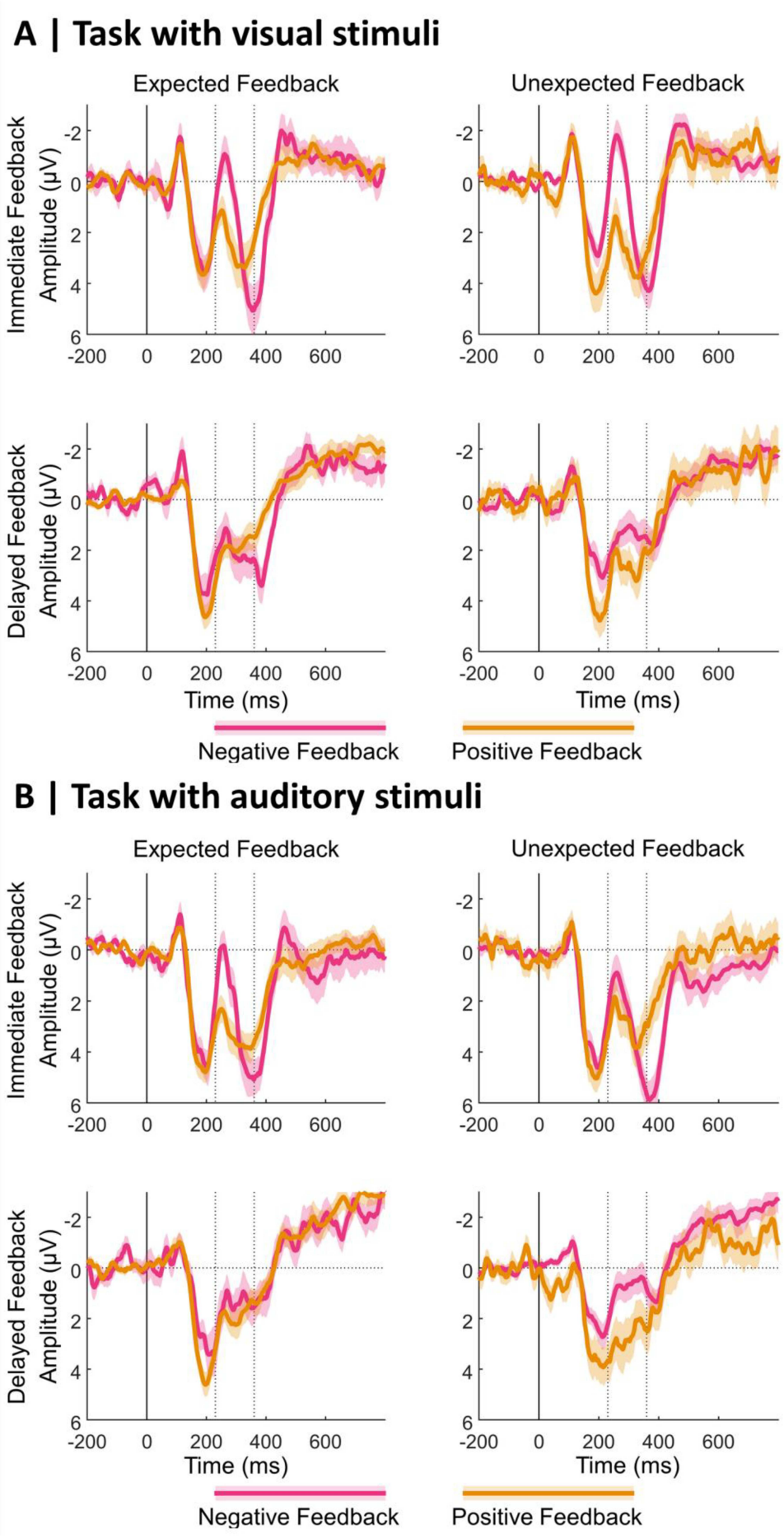
Descriptive data pattern and distribution underlying the PE x Feedback Valence x Modality x Feedback Timing x Electrode interaction for the N170



Dots represent single-trial data points.

Figure S7

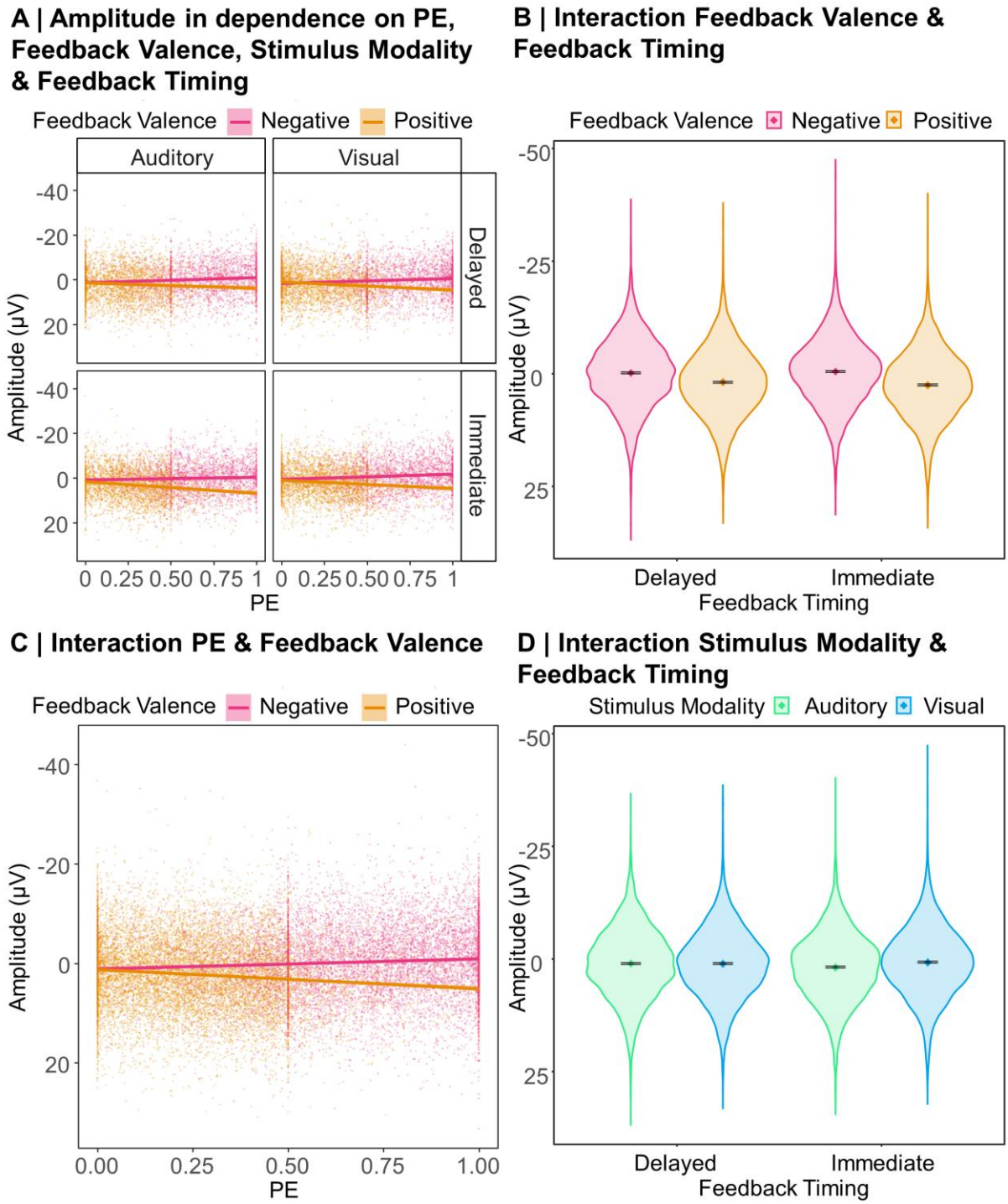
Grand Averages for the FRN following Expected and Unexpected Feedback


A | Task with visual stimuli
Expected Feedback
Unexpected Feedback
Immediate Feedback Amplitude (μV)
Delayed Feedback Amplitude (μV)
-2
0
2
4
6
-200
0
200
400
600
Time (ms)
Negative Feedback
Positive Feedback
B | Task with auditory stimuli
Expected Feedback
Unexpected Feedback
Immediate Feedback Amplitude (μV)
Delayed Feedback Amplitude (μV)
Time (ms)
Negative Feedback
Positive Feedback

Note. Dotted lines indicate the time window used for the peak detection in the difference wave (negative – positive feedback). Shaded areas indicate standard errors. Unexpected feedback corresponds to absolute PE values > 0.50, while expected feedback corresponds to absolute PE values ≤ 0.50.

Figure S8

Descriptive data patterns and distributions underlying the FRN analysis



Dots represent single-trial data points. Error bars indicate a 95% confidence interval.

Links between Altered Feedback Learning and Symptoms of Depression: Insights from an EEG Study on FRN and N170

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Abstract

Blunted electrophysiological and striatal responses to reward have been suggested as biomarkers or endophenotypes for depression. However, previous studies did not differentiate between learning from immediate and learning from delayed feedback, which involves different neural structures. The aim of the present study was to clarify whether depression alters learning from both immediate and delayed feedback. We investigated the influence of current and past depressive symptom severity and familial history of depression in a mixed clinical and non-clinical sample of 45 individuals on two event-related potential (ERP) components, namely the feedback-related negativity (FRN) and N170, which are associated with immediate and delayed feedback processing, respectively. Performance in a probabilistic feedback learning task with immediate and delayed feedback was reduced for more severe depressive symptoms, regardless of feedback timing. Surprisingly, the FRN was not affected by current or past depressive symptom severity or familial vulnerability to depression. However, we found depression-related changes in the N170 for both immediate and delayed feedback processing: currently experienced depressive symptoms were associated with poorer encoding of prediction errors in the N170. In addition, a family history of depression was associated with lower sensitivity to the valence of feedback in the N170. In summary, the N170 may emerge as a novel, important biomarker in clinical research on depression and feedback-based learning.

Keywords: Depression, N170, FRN/RewP, Delayed Feedback, Prediction Error

Links between Altered Feedback Learning and Symptoms of Depression: Insights from an EEG Study on FRN and N170

While modern living conditions seem to feed the incidence of depression, many questions concerning its pathophysiology are still unresolved (Hidaka, 2012). Researchers try to find structural and functional alterations in the brain that may help explain the underlying mechanisms of depression. Given the heterogeneity of the symptoms, it is likely that multiple brain regions and mechanisms are involved (Nestler et al., 2002, Thompson, 2023). The etiological diversity of depression is so complex that it can hardly be studied in its entirety (Kendler et al., 2002). Therefore, recent research has focused on identifying endophenotypes, hoping to better understand biological mechanisms contributing to depression. Endophenotypes are inheritable traits that allow linking observable symptoms with genetic predispositions and thereby help to develop tailored interventions (Luking et al., 2016).

In this context, it was suggested that dysfunctional reward processing is a crucial aspect in the pathophysiology of depression (Admon & Pizzagalli, 2015) and that blunted neural responses to reward might be an endophenotype for depression (Bress et al., 2015; Luking et al., 2016). The dopaminergic midbrain forms the core of the brain's reward system (Björklund & Dunnett, 2007; Glimcher, 2011; Haber & Knutson, 2010; Schultz & Dickinson, 2000). However, midbrain dopaminergic neurons do not signal reward itself; rather, they reflect whether an outcome is better or worse than expected, encoding a prediction error ([PE]; Schultz et al., 1997). The PE signal has been found to be reflected in brain activity measured via electroencephalography (EEG). More precisely, the feedback-related negativity (FRN) is an event-related potential (ERP) component that peaks between 230 and 330 ms after feedback onset (Miltner et al., 1997). While its amplitude was reported to be larger for losses than gains, more recent research suggests that the negative going waveform is the baseline response and that amplitude modulations of the component rather reflect a positivity

following rewards, leading to the conceptualization of the reward positivity ([RewP]; for a review see Proudfit, 2015). In this manuscript, however, we refer to the component within the FRN/RewP time window as FRN. Undoubtedly the electrophysiological activity in this time window distinguishes between positive and negative feedback, which is why it is often presented as a difference wave for negative minus positive feedback-related ERP (FRN_{diff}). Remarkably, the FRN is not only sensitive to the valence of feedback but is also modulated by PEs (Burnside et al., 2019; Fischer & Ullsperger, 2013; Röhlinger et al., 2025; Weber & Bellebaum, 2024). Accordingly, it is associated with the mesencephalic dopaminergic reward system influencing the anterior cingulate cortex, which is a likely generator of the signal in the FRN/RewP time window (Bellebaum & Daum, 2008; Foti et al., 2011; Holroyd et al., 2004; Holroyd & Coles, 2002; Oerlemans et al., 2025).

Numerous studies describe a link between depression and reduced feedback valence sensitivity in the FRN, driven by reduced (less positive) amplitudes following rewards (Bress et al., 2012, 2013, 2015; Foti et al., 2014; Klawohn et al., 2021; for a meta-analytic review see Keren et al., 2018), even in young preschool-age children (Belden et al., 2016). In line with changes in the FRN, altered and mainly impaired feedback learning has been reported for depressed individuals (Admon et al., 2017; Bakic et al., 2017; Kumar et al., 2018; Kunisato et al., 2012; Macoveanu et al., 2014; Pechtel et al., 2013; Pizzagalli et al., 2005, 2008; for a review see Chen et al., 2015 and Eshel & Roiser, 2010). However, previous studies did not differentiate between learning from immediate and learning from delayed feedback, the processing of which involves different neuronal structures. The processing of immediate feedback is based on the striatum (Foerde et al., 2013; Foerde & Shohamy, 2011), and striatal hypo-functioning is decisive for dysfunctional reward processing in depression (Pizzagalli et al., 2009; Takamura et al., 2017; for a review see Admon & Pizzagalli, 2015 and Luking et al., 2016). Within non-depressed and depressed individuals, The ERPs in the

FRN time window and striatal activation were correlated, indicating convergence across the two measures (Becker et al., 2014; Carlson et al., 2011; Foti et al., 2014).

In contrast, the processing of delayed feedback has been suggested to rely less on the striatum (Foerde et al., 2013, Foerde & Shohamy, 2011). Several ERP studies (Arbel et al., 2017; Höltje & Mecklinger, 2020; Peterburs et al., 2016, Weinberg et al., 2012; Weismüller & Bellebaum, 2016) support this by describing a decrease in the amplitude difference between positive and negative feedback for the FRN following delayed feedback. At the same time an increase in the amplitude of the N170 ERP component has been described with increased feedback delay (Arbel et a., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019), which may be related to a stronger role of the hippocampus for delayed feedback processing (Foerde et al., 2013, Foerde & Shohamy, 2011). The feedback-locked N170 is most pronounced over the occipitotemporal cortex in a time window between 100 and 200 ms and was therefore interpreted as reflecting processes in the medial temporal lobe ([MTL]; Arbel et a., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019). A recent study suggested that the pronounced N170 following delayed feedback reflects the reactivation of visual stimulus representations in visual brain areas, possibly initiated by regions within the MTL, to link the selection of a visual stimulus to temporally delayed feedback (Röhlinger et al., 2025). The hippocampus, as part of the MTL, is a central structure for memory functions (Scoville & Milner, 1957) but also receives information from and sends information to several brain regions in the reward system (for a review see Heshmati & Russo, 2015). Accordingly, in a previous study we found that the N170 reflects the whole range of the PE, with more pronounced amplitudes for unexpected positive feedback and smaller amplitudes following unexpected negative feedback (Röhlinger et al., 2025).

Interestingly, the hippocampus also plays a role in etiological models linking depression to chronic stress: Stress, whether acute or chronic, activates the hypothalamic-

pituitary-adrenal axis, and extreme or long-lasting stress can cause damage to the hippocampus (for a review, see Nestler et al., 2002). Accordingly, depression can be accompanied by hippocampal atrophy, which in turn is linked to memory impairment and might contribute to some of the cognitive distortions seen in depression (Fairhall et al., 2010; Nestler et al., 2002; Thompson, 2023). Besides weaknesses in recollection, depressed individuals tend to have a reduced memory for positive events, while their memory for negative events is increased (Shah et al., 1998; for a review see Dillon & Pizzagalli, 2018). Findings by Hager et al. (2021) suggest that depression is associated with dysfunctional source memory for rewards but not losses. Given the broad symptoms of depression, it is no surprise that the pathophysiology involves a variety of brain regions, including the hippocampus and striatum (Nestler et al., 2002).

Although the processing of immediate and delayed feedback seems to primarily rely on different structures in the brain, it is conceivable that also for delayed feedback depression is accompanied by behavioral and neurophysiological changes. Based on the changes in the hippocampus and related functions, we expected that depressed individuals would also show alterations in learning from and processing of delayed feedback, with the latter being reflected in different neurophysiological signals compared to immediate feedback (see above). With the present work, we aimed to investigate the link between depression and the behavioral performance as well as the electrophysiological processing in a probabilistic feedback learning task with immediate and delayed feedback.

It must be taken into account that endophenotypes are state-independent, i.e., they can be detected in a person even if the disease is not active (Gottesman and Gould, 2003). Accordingly, blunted striatal activity was found in remitted depressed individuals (McCabe et al., 2009) as well as a blunted (more negative) FRN amplitudes following rewards in “healthy” siblings of depressed individuals (Weinberg et al., 2015). There is also an increased

risk of developing depression for the children of depressed mothers (Halligan et al., 2007; Raposa et al., 2014), accompanied by blunted responses to reward within the dorsal and ventral striatum, relative to children of non-depressed mothers (for an extensive review see Luking et al., 2016). In summary, depressed individuals, remitted individuals and those at high risk show blunted striatal responses to the receipt of (immediate) rewards. Therefore, we assessed current depressive symptoms, past depression episodes, and the family history of depression as predictors for feedback learning and processing. Since dichotomizing a continuous variable leads to a loss of information and reduced statistical power (see Clayson et al., 2020), we approach depression not as a binary state (healthy vs. depressed), but as a dimensional construct, operationalized as a continuous variable (see also Hager et al., 2021) in a mixed clinical and non-clinical sample.

Because there is evidence for a publication bias and only a weak relationship between FRN and depression has been found in previous studies (Clayson et al., 2020; Moran et al., 2017), the first aim of the planned study is to replicate findings of reduced learning performance and FRN amplitude with immediate feedback in the context of depression. In several studies, the association between depression and reduced feedback valence sensitivity in the FRN was mainly driven by the response to reward (Belden et al., 2016; Bress et al. 2012, 2013, 2015; Brush et al., 2018; Foti et al., 2014). Therefore, we expected a reduced sensitivity to feedback valence in the FRN in individuals with an increased familial vulnerability for depression, individuals that have experienced depressive episodes in the past and participants that currently experience depressive symptoms, especially in the immediate feedback condition. In addition, we aimed to investigate whether these individuals also show alterations like reduced amplitudes for the N170, especially following delayed feedback. Finally, for both ERP components, we aimed to explore depression related changes in the neural processing of the PE, possibly in interaction with feedback timing.

Method

Participants

The sample size was based on the number of participants in previous studies. Bress et al. (2015) found a significant, moderate to strong correlation ($r = .41$, $p < .010$) between depressive symptomatology and the FRN_{diff} in a sample of 41 individuals. Taking dropouts due to EEG artifacts or exclusion criteria (see below) into account, we preregistered to recruit 50 participants (18 to 40 years). Exclusion criteria were current or former neurological disorders, acute psychotic conditions, the regular or acute consumption of substances affecting the central nervous system, knowledge about Hiragana-Characters, and uncorrected impaired vision. To consider acute depressive symptoms as a continuous factor in the analysis and therefore cover sufficient variance we promoted the study on the campus of the Heinrich Heine University Düsseldorf, via social media platforms and at the Outpatient Psychotherapy Unit of the LVR Clinic for Psychosomatic Medicine and Psychotherapy in Düsseldorf. In total, we acquired data from 50 participants. We excluded five participants, three of them because they fulfilled at least one of our exclusion criteria, one because of bad EEG data quality determined during visual inspection of the raw data, and one due to improper setup of the EEG system during the acquisition. The final sample included in the analyses thus consisted of 45 participants: 37 reported being women and eight men; 41 were right-handed, three left-handed and one ambidextrous. The mean age was 24.87 years (*SD* = 5.54 years, *Min* = 18 years, *Max* = 39 years).

Procedure

Upon arrival in the laboratory, participants were informed about the experimental procedure and gave written informed consent to participate in the study, followed by a short clinical interview (see below) lasting about 30 minutes. Afterwards, participants were placed in front of a 27 in, 1920 * 1080 px W-LED monitor (BENQ EW2740L) with a refresh rate of

60 Hz and filled in a demographic questionnaire that additionally contained questions about past depressive episodes (see below) and whether a first-degree relative is or was affected by depression (see below), followed by a questionnaire to assess the participants current depressive symptoms (see below). Then we attached EEG electrodes and started the experimental task (see below) which lasted about 45 minutes. Participation was compensated with 5€ per 30 minutes or course credit for psychology students. Additionally, participants received the money they earned during the feedback learning task (see below) rounded up to 6€. The study was approved by the ethics committee of the Faculty of Mathematics and Natural Sciences at Heinrich Heine University Düsseldorf, Germany, and in accordance with the declaration of Helsinki.

Preceding Interview

To detect psychiatric disorders apart from or comorbid with depression, a trained experimenter conducted parts of the Mini-DIPS (Margraf & Cwik, 2017; Margraf et al., 2017) with all participants (for a similar approach see Bress et al., 2013 and Foti et al., 2014). The Mini-DIPS is an abbreviated version of the Diagnostic Interview for Psychological Disorders (DIPS) and, according to Margraf and Cwik (2017), offers an efficient and reliable diagnosis of psychological disorders according to DSM-5 and ICD-10 for research questions. The interview begins with a set of questions designed to screen for symptoms, and if any are affirmed, additional questions are asked to evaluate if the symptoms fulfill the necessary criteria for a diagnosis. We used the Mini-DIPS to detect the following conditions: anxiety disorders, substance addiction disorders, eating disorders, obsessive-compulsive disorders, affective disorders, and suicidality. We decided to screen for these disorders because they pose common comorbidities in depression (Jacobi et al., 2014; Lamers et al., 2011; Zimmermann et al., 2002), were in some cases associated with the FRN (Aarts & Pourtois, 2012; Bellato et al., 2021; Forester et al., 2024; Gu et al., 2010; Jiang et al., 2018; Ryu et al.,

2017; Sehrig et al., 2019; Takács et al., 2015; Tobias & Ito, 2021) and are some of the most common mental disorders in adulthood in Germany (Suhr, 2020).

Assessment of Depression

Familial vulnerability

We asked participants whether a first-degree relative, i.e., parent or sibling (excluding half-siblings), has ever been diagnosed with depression, providing "yes", "no" and "I'm not sure" as response options.

Past depressive episodes

Adapted from the approach by Bress et al. (2013), we used a modified version of the mood module of the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001; German version: Gräfe et al., 2004) to evaluate past depressive episodes. The modified version by Cannon et al. (2007) contains 9 items (e.g., *Little interest or pleasure in doing things*) that can be rated on a 4-point-scale ((0) *not at all* to (3) *almost every day*). Importantly, the modified version refers to the two weeks in the participants' lives when they were feeling most blue, sad, or depressed. The sum score can range from 0 to 27, with higher values indicating more severe depressive symptoms. High correspondence with lifetime diagnosis based on the Structured Clinical Interview for DSM–IV (SCID; First & Gibbon, 2004) makes the modified PHQ-9 an efficient measure of lifetime depression (Cannon et al., 2007).

Current depressive symptoms

We used the Beck Depression Inventory ([BDI-II]; Beck et al., 1996; German version: Hautzinger et al., 2006) as a measure for acute depression severity (for a similar account, see Bress et al., 2013). It contains 21 items, each consisting of 4 statements reflecting values from 0 to 3. For example, the first item addresses sadness with the following statements: (0) *I do not feel sad* (1) *I feel sad much of the time* (2) *I am sad all of the time* (3) *I am so sad or unhappy that I can't stand it*. We asked participants to indicate which of the four statements

has most likely been true for them in the past two weeks. The values of the ticked statements were summed up and built a score ranging from 0 (no depressive symptomatology) to 63 (severe symptomatology).

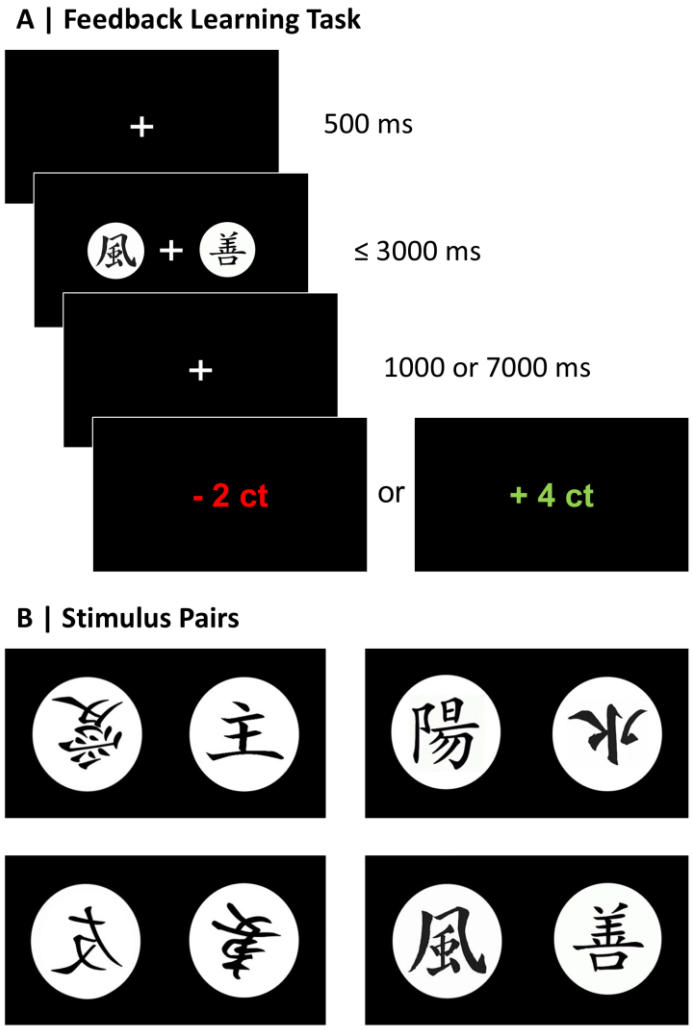
Experimental Task and Conditions

Participants performed a probabilistic feedback learning task, where they could learn associations between stimuli and positive (+4ct) or negative (-2ct) monetary feedback (see Figure 1A). The task contained the within-subject factor feedback timing: Feedback appeared either 1 s (immediate feedback) or 7 s (delayed feedback) after the participant's choice. The experiment consisted of four learning phases, two with immediate and two with delayed feedback. Learning phases with immediate vs. delayed feedback alternated and it was counterbalanced which feedback timing condition was presented first.

Figure 1A shows the temporal sequence of a trial, from the presentation of two available stimuli to the selection and feedback. The software Presentation (Neurobehavioral Systems Inc., Albany, CA, USA) controlled the timing of stimulation and the recording of responses. Responses were performed on a standard computer keyboard (Logitech K120) where participants could press the left and right control keys to choose between the stimuli. One stimulus of each pair was associated with reward in 65 % of the trials and with punishment in 35 %, while the probabilities were reversed for the other stimulus. The participant's task was to learn which stimulus is more likely to be rewarded and thus maximize the reward. Participants were instructed that wins and losses contribute to the total amount of money paid out at the end. A new pair of stimuli was presented in each of the four learning phases (see Figure 1B). Each learning phase consisted of 4 blocks of 20 trials with short breaks in between, i.e., 80 trials per learning phase and 320 trials in total.

Figure 1

Stimuli and time course of the probabilistic feedback learning task



Note. **A** Feedback Learning Task: The assignment of visual stimuli to the left and right side of the screen, as well as the side on which the more rewarding stimulus was presented, was counterbalanced to ensure that feedback could clearly be associated with a stimulus and not with a response side. **B** Stimulus Pairs: The neighboring stimuli build the four pairs used for all participants. The stimulus associated with higher reward probability was randomly determined when a new pair was presented.

EEG Data Acquisition

We acquired EEG data from a total of 60 electrodes, fixed with an actiCap textile softcap (BrainProducts, Germany), and evenly distributed on the scalp based on the extended 10–20 system. Electrodes were attached to the scalp sites AF3, AF4, AF7, AF8, C1, C2, C3, C4, C5, C6, CP1, CP2, CP3, CP4, CP5, CP6, CPz, Cz, F1, F2, F3, F4, F5, F6, F7, F8, FC1, FC2, FC3, FC4, FC5, FC6, FT10, FT7, FT8, FT9, Fz, O1, O2, Oz, P1, P2, P3, P4, P5, P6, P7, P8, PO10, PO3, PO4, PO7, PO8, PO9, POz, Pz, T7, T8, TP7, TP8. In addition, the ground

electrode was attached to the AFz position and an online reference to the position FCz. We placed two further electrodes over the left and right mastoids to cover as much of the scalp as possible for the average reference (see below). Finally, we attached two more electrodes (vEOG) above (at Fp1 position) and below the left eye to keep track of vertical eye movements and blinks. A BrainAmp DC amplifier (BrainProducts, Germany) and the Brain Vision Recorder software (BrainProducts, Germany) were used for data recording with a sampling rate of 1000 Hz and an online lowpass filter of 100 Hz. We kept impedances below 15 kΩ.

Data Analysis

Behavioral Analysis

We performed generalized linear mixed-effects models (GLME) analyses suitable for binomial distributions and single-trial data using the lme4 package (version 1.1.34; Bates et al., 2015) in R (The R Foundation, 2021). The dependent variable was response accuracy, with correct responses (defined as the choice of the stimulus associated with the higher reward probability) coded as 1 and incorrect responses as 0. We calculated three separate models, one for each measure of depression as predictor. The first model comprised as fixed-effect predictors the BDI (between-subjects: severity of current depressive symptoms measured via the BDI-II), Feedback Timing (within-subjects: immediate vs. delayed) and Block (1-4; because learning is indicated by an increase in the number of correct responses within the same learning phase), together with all interactions. Participants were included as random intercepts. For the inclusion of random-effect slopes per participant, we considered best practice guidelines (Meteyard & Davies, 2020): we included all within-subject main and interaction effects as random slopes, unless their inclusion compromised model fit. The maximal model was determined by using the buildmer (Version 2.11; Voeten, 2020) function.

The other two models were constructed based on the same principle, with the only difference being the depression measure used as fixed effect predictor. The second model contained the PHQ (a measure of past depressive episodes assessed through the modified PHQ-9) and the third model included Familial Vulnerability for depression as a predictor (binary categorical variable indicating whether a first degree relative has ever been diagnosed with depression). The resulting model formulas for all three models are presented in Table S1 of the Supporting Information.

Modelling of Prediction Errors

We inferred single trial values of the PE for each participant by fitting a reinforcement learning model to the behavioral data in MATLAB (version R2021a, The MathWorks, Inc., 2021; for a similar approach see Burnside et al., 2019; Lefebvre et al., 2017; Röhlinger et al., 2025; Weber & Bellebaum, 2024). The basis for the application of the reinforcement learning model was each participants' sequence of choices and the feedback they received. The PE $\delta_{c,t}$ was conceptualized as:

$$\delta_{c,t} = r_t - Q_{c,t}$$

where in a given trial t the reward r_t is 1 for positive feedback and 0 for negative feedback, and $Q_{c,t}$ is the value of the stimulus the participant chose. Separately for each of the four stimulus pairs, we primarily assigned both stimuli a stimulus value of 0.5, that was iteratively adjusted in every trial t in which the stimulus pair was displayed. The stimulus value of the chosen stimulus, Q_c, was adjusted based on the difference between the previous value and the obtained outcome (the PE δ), together with a learning rate α that mirrors how much the participant used the PE to adjust the stimulus value. For each of the four stimulus pairs, we modeled different learning rates for learning from positive feedback and negative feedback. We adjusted the stimulus value of the chosen stimulus with the learning rate α_{con} for trials with positive feedback that confirms the choice as follows:

$$Q_{c,t+1} = Q_{c,t} + \alpha_{con} * \delta_{c,t}$$

For trials with negative feedback that disconfirms the choice, the stimulus value of the chosen stimulus was adjusted with the learning rate α_{dis}:

$$Q_{c,t+1} = Q_{c,t} + \alpha_{dis} * \delta_{c,t}$$

As both stimuli of a pair were always displayed together, we expected that participants would form assumptions about the unchosen stimulus from feedback for the chosen stimulus. Therefore, the value of the unchosen stimulus, Q_u, was 1-Q_c and was updated accordingly.

For each trial, $t_{1,\dots,n_{trials}}$, we calculated the probability p that the model would choose the stimulus which was indeed chosen by the participant with the help of the softmax function. This calculation was based on prior stimulus values of both stimuli that were displayed, namely values of the chosen stimulus, $Q_{c,t}$, and the unchosen stimulus in trial t, $Q_{u,t}$, along with an exploration parameter β:

$$p_{c,t} = \frac{e^{Qc,t * \beta}}{e^{Qc,t * \beta} + e^{Qu,t * \beta}}$$

with β indicating how much prior stimulus values affected the participants choices. A larger β indicates that a participant relied more on earlier stimulus values, whereas a smaller β indicates that the participant was more explorative in the choice behavior.

In a next step, we used the probabilities p to calculate the negative summed log-likelihood ($-LL$) as measure for the model's goodness of fit:

$$-\Sigma \log (p_{c,t_1,\dots,n_{trials}})$$

The optimization function fmincon from the Optimization Toolbox of MATLAB (R2021a, The MathWorks, Inc., 2021) minimized the $-LL$ value by estimating values for the free parameters ($\alpha_{con}, \alpha_{dis}, \beta$) that led to the least difference between the model's predicted choices and the participant's actual behavior. The model was fit repeatedly (50 iterations) to the participants' behavior to prevent convergence to local minima. We allowed random

numbers within the interval [0; 1] as start values for the free parameters. We set boundaries of [0; 1] for the learning rates ($\alpha_{con}, \alpha_{dis}$), and [0; 100] for the exploration parameter (β).

EEG Data Analysis

BrainVision Analyzer 2.2 (Brain Products GmbH, 2018), MATLAB R2021a (The MathWorks, Inc., 2021) and R (The R Foundation, 2021) were used for EEG data analysis. Trials in which participants failed to answer (*M* = 1.00%, *SD* = 1.60%, *Min* = 0.00 %, *Max* = 8.44%) were excluded from any further EEG analyses.

Preprocessing. We first re-referenced the data to the average of all scalp electrodes and calculated the signal at the online reference site FCz (for similar procedures see Arbel et al., 2017; Höltje & Mecklinger, 2020; Röhlinger et al., 2025). To minimize the reduction of ERP effects that can result by using an average reference (see Luck, 2014) we used high-density EEG acquisition including data from 63 scalp electrodes (see above, including mastoids) into the average reference. Afterwards, we filtered the data with a 30 Hz low cut-off and a 0.1 Hz high cut-off filter (as proposed by Luck, 2014) as well as a 50 Hz Notch Filter. Then, we performed an independent component analysis (ICA) followed by a reversed ICA on single-subject EEG data to remove blinking artifacts. In a next step, we created segments from 200 ms before to 800 ms after feedback onset followed by a baseline correction relative to the first 200 ms of the segment. Then, we excluded segments with artifacts in the electrodes of our interest (for similar approaches see Albrecht et al., 2023; Röhlinger et al., 2025), i.e., electrodes used to measure the FRN (Fz, FCz, Cz, FC1 & FC2) and N170 (P7 and P8). Precisely, all segments containing voltage steps > 50 µV/ms, differences between values > 80 µV or < 0.1 µV within an interval of 100 ms or amplitudes > 80 µV or < -80 µV were removed (*M* = 1.25%, *SD* = 3.25%, *Min* = 0.00%, *Max* = 17.41%). We grouped and averaged the remaining segments according to the conditions (positive and negative immediate feedback and delayed feedback), yielding four averages per participant.

Eventually, we exported all single-trial segment data as well as all averages per condition and participant for further analysis in MATLAB to extract single-trial ERP data (MathWorks, MA).

For the N170, we retrieved single-trial amplitudes (see Albrecht et al., 2023) from electrodes P7 and P8 (for similar approaches see Arbel et al., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019; Röhlinger et al., 2025) as preregistered. First, we identified the maximum negative peak amplitude between 130 and 230 ms after feedback presentation in each participant's averages, at both electrode sites and for all four conditions separately (see above). Then, for each single trial, we calculated the mean amplitude in a time window of ± 10 ms around the condition- and electrode-specific N170 peak latency. In addition, we extracted the mean amplitude in a time window of ± 10 ms around the preceding positive peak (P1). Similar to the approach used for the negative peak, P1 latency was determined using the condition-specific average at each electrode site. We determined the P1 as the maximum positivity in a time window starting 75 ms after feedback onset to the respective condition-specific negative peak. As dependent variable for the analysis, we used the N170 defined as the peak-to-peak amplitude by subtracting the single-trial amplitude value corresponding to the preceding P1 from the single-trial value corresponding to the negative peak (for a similar approach see Röhlinger et al., 2025).

For the FRN, we retrieved single-trial amplitudes from the pooled signal of an electrode cluster consisting of Fz, FCz, Cz, FC1, and FC2 (for a similar approach see Röhlinger et al., 2025), as preregistered. First, we calculated the difference wave (FRN_{diff}) for negative – positive feedback separately for immediate and delayed feedback for each participant. We used the two difference waves to determine the maximum negative peak amplitude latency between 230 and 360 ms post-feedback for each participant. Then, for each single trial, we calculated the mean amplitude in a time window of ± 10 ms around the

condition-specific $FRNd_{iff}$ peak latency. Importantly, the difference waves were only used to derive the latency of the time point when the difference between positive and negative feedback was maximal, but single-trial values based on this latency were extracted from the original waveforms.

Statistical Analysis.

N170. We analyzed the single-trial N170 amplitude as the dependent variable in an LME analysis using R (Bates et al., 2015). Similar to the analysis of the behavioral data, we constructed three separate models, one for each of the three different depression variables as predictor (BDI, PHQ and Familial Vulnerability). The first model contained the fixed-effect predictors BDI (severity of current depressive symptoms measured via the BDI-II), as well as Feedback Timing (immediate vs. delayed), Feedback Valence (negative vs. positive) and the unsigned PE (indicating general expectation violations or surprise, independent of feedback valence). Because the N170 has been found to show hemispheric differences (see Röhlinger et al., 2025), we also added Electrode (P7 vs. P8) as a fixed-effect predictor, in addition with all possible interactions between the factors. As random effect factor, we included Participant. Random slopes per participant were added as described for the behavioural GLME above.

The other two models were created based on the same approach, with the only variation being the depression measure used as a fixed effect predictor. The second model contained the PHQ (a measure of past depressive episodes assessed through the modified PHQ-9) and the third model included Familial Vulnerability for depression as a predictor (binary categorical variable indicating whether a first degree relative has ever been diagnosed with depression). The resulting model formulas for all three models are presented in Table S2 of the Supporting Information. To resolve significant interactions, simple slope analyses were performed with Bonferroni corrected *p*-values (multiplied by the number of conducted tests).

FRN. We analyzed the single-trial FRN amplitude as the dependent variable in an LME analysis using R (Bates et al., 2015). We followed the same approach as in the N170 analysis (see above), building three models that varied only regarding the depression variable used as fixed-effect predictor (BDI, PHQ or Familial Vulnerability). As additional fixed-effect predictors in each of the three models we added Feedback Timing (immediate vs. delayed), Feedback Valence (negative vs. positive), and the unsigned PE (indicating general expectation violations or surprise, independent of feedback valence), as well as all possible interactions between these factors. As random intercepts, we included Participant. Random slopes per participant were added as described for the behavioral GLME above. The formulas for the three resulting models are presented in Table S3 of the Supporting Information. Significant interactions were resolved as described for the N170 (see above).

Results

Mini-Dips, BDI-II, modified PHQ-9 and Familial Vulnerability

According to the Mini-DIPS, depressive symptoms were the most frequent psychiatric symptoms in our sample. Of the 45 participants in our sample, 18 met the diagnostic criteria for a major depressive episode, either currently or in the past. 13 participants fulfilled the criteria for social anxiety disorder, and 11 participants met the criteria for generalized anxiety disorder, each either currently or in the past. Diagnostic criteria for other mental disorders were met less frequently (see Table 1). Additionally, 9 participants reported having received outpatient or inpatient psychotherapy either currently or in the past. Participants reached a mean BDI score of 9.89 (*SD* = 9.34; *Min* = 0, *Max* = 39) and a mean PHQ score of 9.64 (*SD* = 5.68, *Min* = 0, *Max* = 26). Finally, 8 participants reported that a first degree relative has been diagnosed with depression, while this was not the case for 29 participants and another 8 participants were not sure.

Table 1

Descriptive statistics resulting from the Mini-DIPS

Diagnosis	*n*	Percent (%)
Anxiety Disorders		
Panic Disorder	4	8
Agoraphobia	4	8
Social Anxiety Disorder	13	26
Specific Phobia	4	8
Gerneralized Anxiety Disorder	11	22
Affective Disorders		
Manic Episode	0	0
Hypomanic Episode	6	12
Major Depression	18	36
Persistent Depressive Disorder	3	6
Eating Disorder		
Anorexia Nervosa	3	6
Bulimia Nervosa	5	10
Binge Eating Disorder	3	6
Obsessive Compulsive Disorder	4	8
Substance Addiction	1	2
Suicidal Tendencies	2	4

Note. N = 45. Mini-Dips = abbreviated version of the Diagnostic Interview for Psychological Disorders. Listed are the numbers of individuals who, according to their own reports during the interview, met the criteria for the psychiatric disorders presented, either currently or in the past.

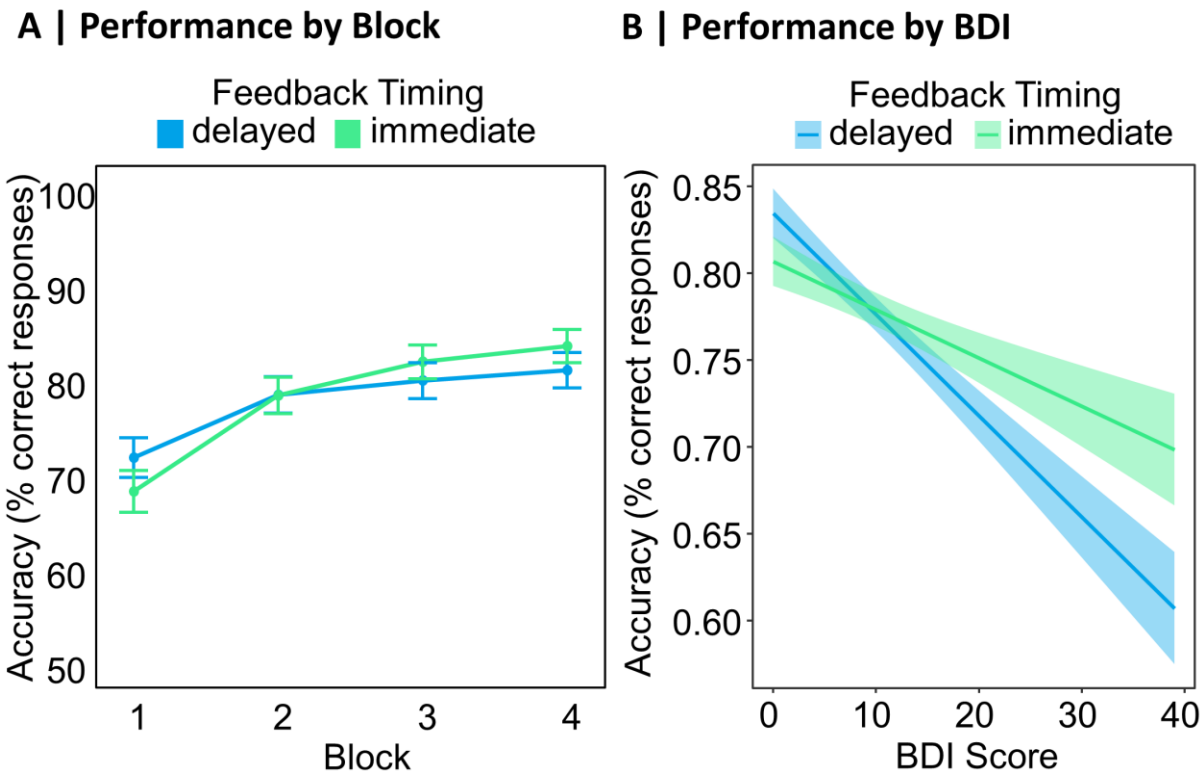
Behavioral Results

Table S4 in the Supporting Information lists β-estimates and effect specific *z*-tests for the three conducted GLME analyses, one for each depression measure (BDI, PHQ and Familial Vulnerability). First, we report results from the GLME analysis including the BDI, together with Block and Feedback Timing. The analysis revealed a significant effect of Block ($p < .001$) on response accuracy, with an increasing number of correct responses (selection of the more frequently rewarded stimulus) across the four learning Blocks. Descriptive data for this effect are presented in Figure 2A. Furthermore, we found a significant effect of BDI ($p = .011$) in the direction that higher BDI scores led to a reduced performance in the experimental feedback learning task. Descriptive data underlying this effect are presented in Figure 2B. No other significant effects were observed (all $ps \geq .106$). The models containing

the PHQ and Familial Vulnerability both replicated the significant effect of Block ($p < .001$) found in the analysis including the BDI, while they did not reveal other significant effects (all $ps \geq .073$ for the analysis involving PHQ and all $ps \geq .285$ for the analysis involving Familial Vulnerability).

Figure 2

Descriptive pattern of learning performance during the feedback learning task



Note. The plots are based on descriptive data. **A** Performance by Block: Mean accuracy (% of correct responses) for the four learning blocks of the probabilistic feedback learning task, separately for immediate and delayed feedback. Error bars represent 95% confidence intervals. **B** Performance by BDI: Mean accuracy (% of correct responses) in the probabilistic feedback learning task depending on BDI-II Scores and Feedback Timing. Shaded areas represent 95% confidence intervals.

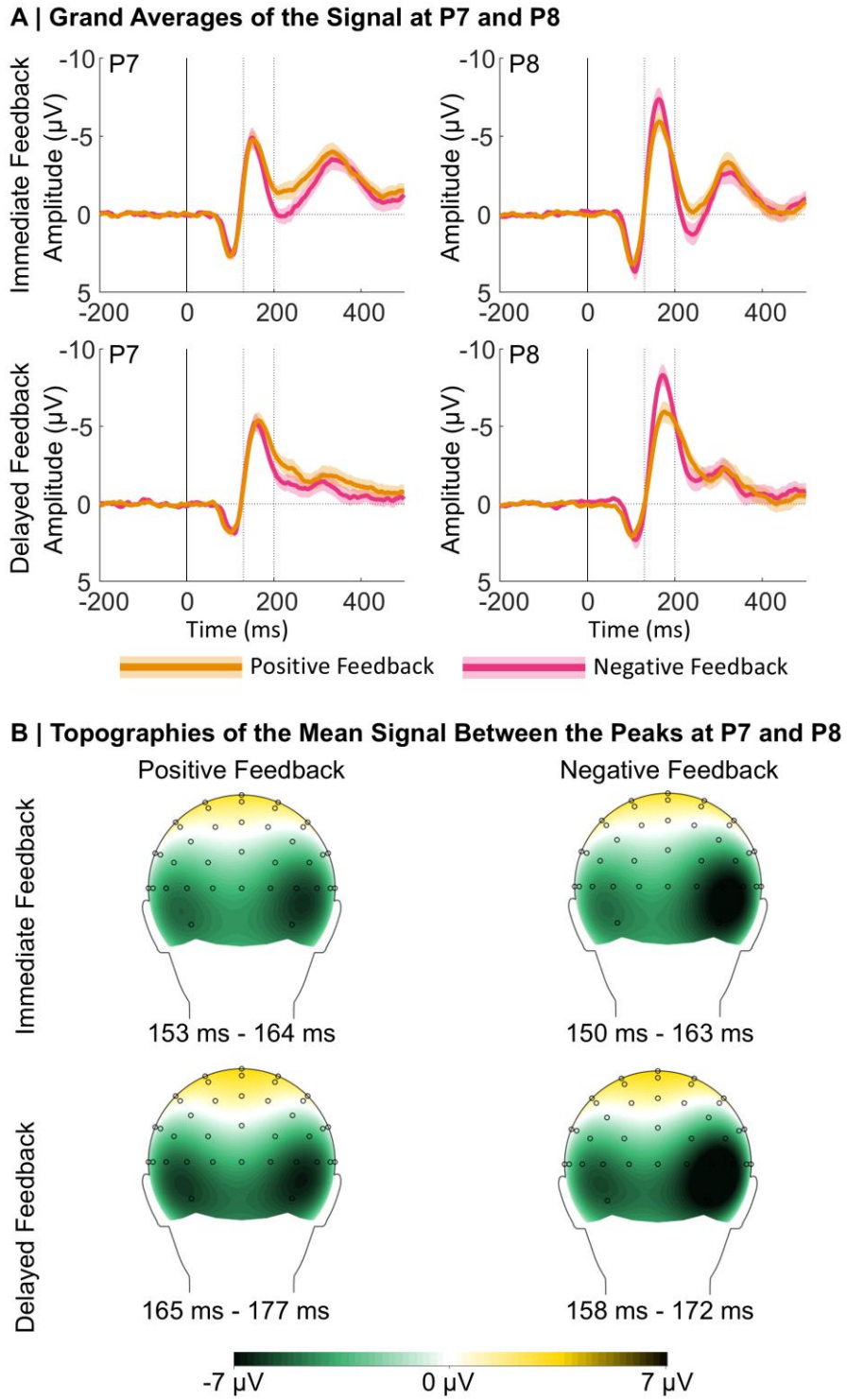
EEG Results

N170

Grand averages for the ERPs following positive and negative immediate and delayed feedback at electrode sites P7 and P8 are presented in Figure 3. In the following, we describe more negative N170 amplitudes as more pronounced or larger.

Figure 3

Grand averages at P7 and P8 and topographical maps at the respective peaks

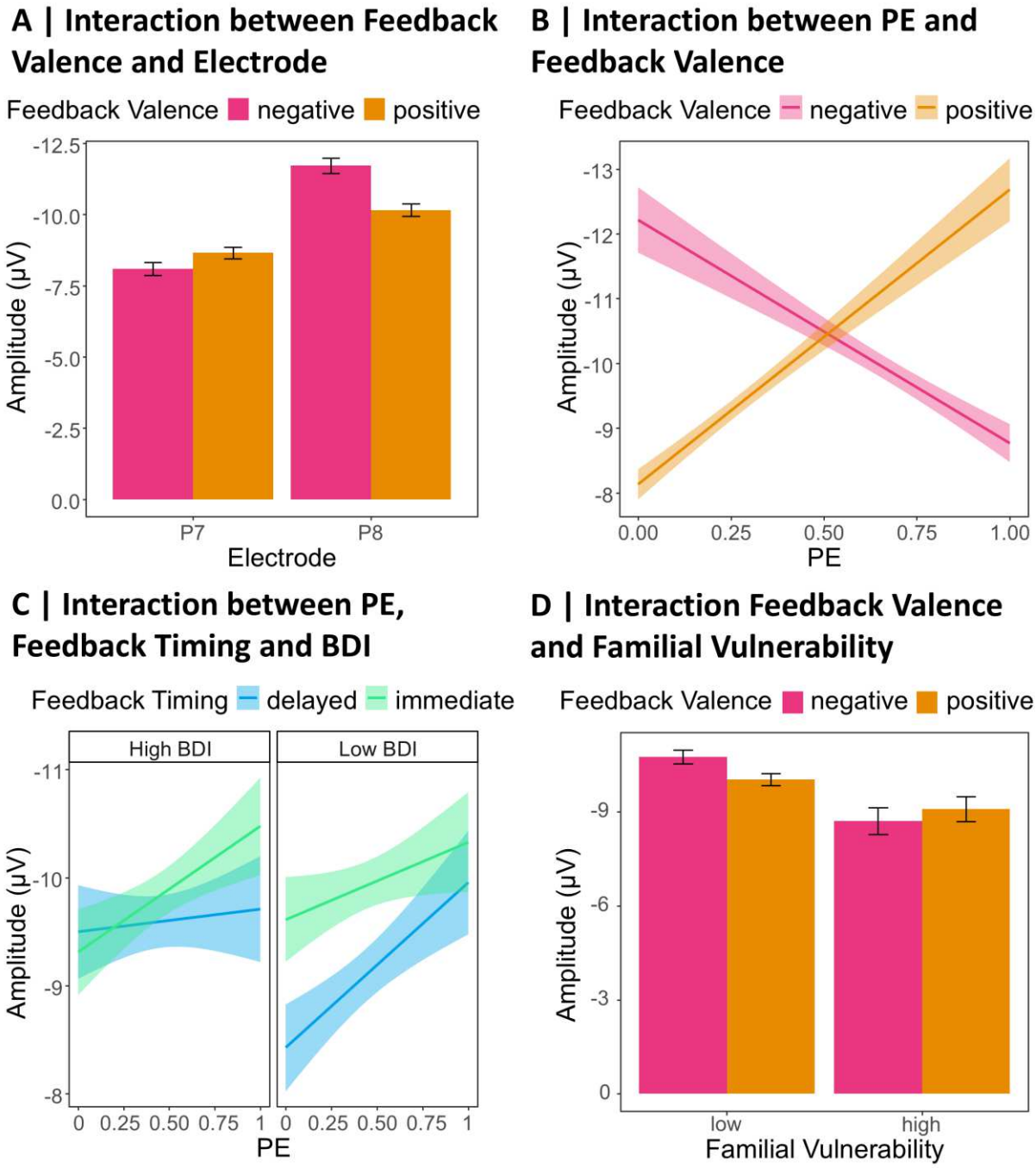


Note. **A** Grand Averages: Dotted lines indicate the time window we used for the N170 peak detection. Shaded areas represent standard errors. **B** Topographies: The maps were constructed on the basis of the condition-specific N170 peaks.

First, we report results from the LME analysis on the N170 including the BDI, Feedback Timing, Feedback Valence, PE and Electrode as predictors. Table S5 in the Supporting Information lists β-estimates and effect specific *t*-tests. The analysis revealed a significant main effect of the Electrode ($p = .009$), with more pronounced amplitudes over the right (P8) than the left hemisphere (P7). This main effect was further explained by a two-way interaction between Electrode and Feedback Valence ($p < .001$), which we resolved with simple slope analyses. The underlying descriptive data are presented in Figure 4A. We found a significant effect of Feedback Valence only for P8 ($\beta = 1.55$, $SE = 0.33$, $t = 4.67$, $p < .001$) with larger amplitudes following negative compared to positive feedback, but not for P7 ($\beta = -0.61$, SE = 0.30, t = -2.02, p = .100). Furthermore, there was a significant interaction between Feedback Valence and PE ($p < .001$), which we resolved with simple slope analyses. The underlying descriptive data are presented in Figure 4B. For negative feedback, the PE had a significant effect on the N170, with more pronounced amplitudes for expected compared to unexpected feedback ($\beta = 0.95$, $SE = 0.35$, $t = 2.69$, $p = .020$). For positive feedback, the effect was reversed, with significantly larger N170 amplitudes for unexpected compared to expected feedback ($\beta = -1.86$, $SE = 0.36$, $t = -5.17$, $p < .001$).

Figure 4

Descriptive data patterns underlying the N170 analyses

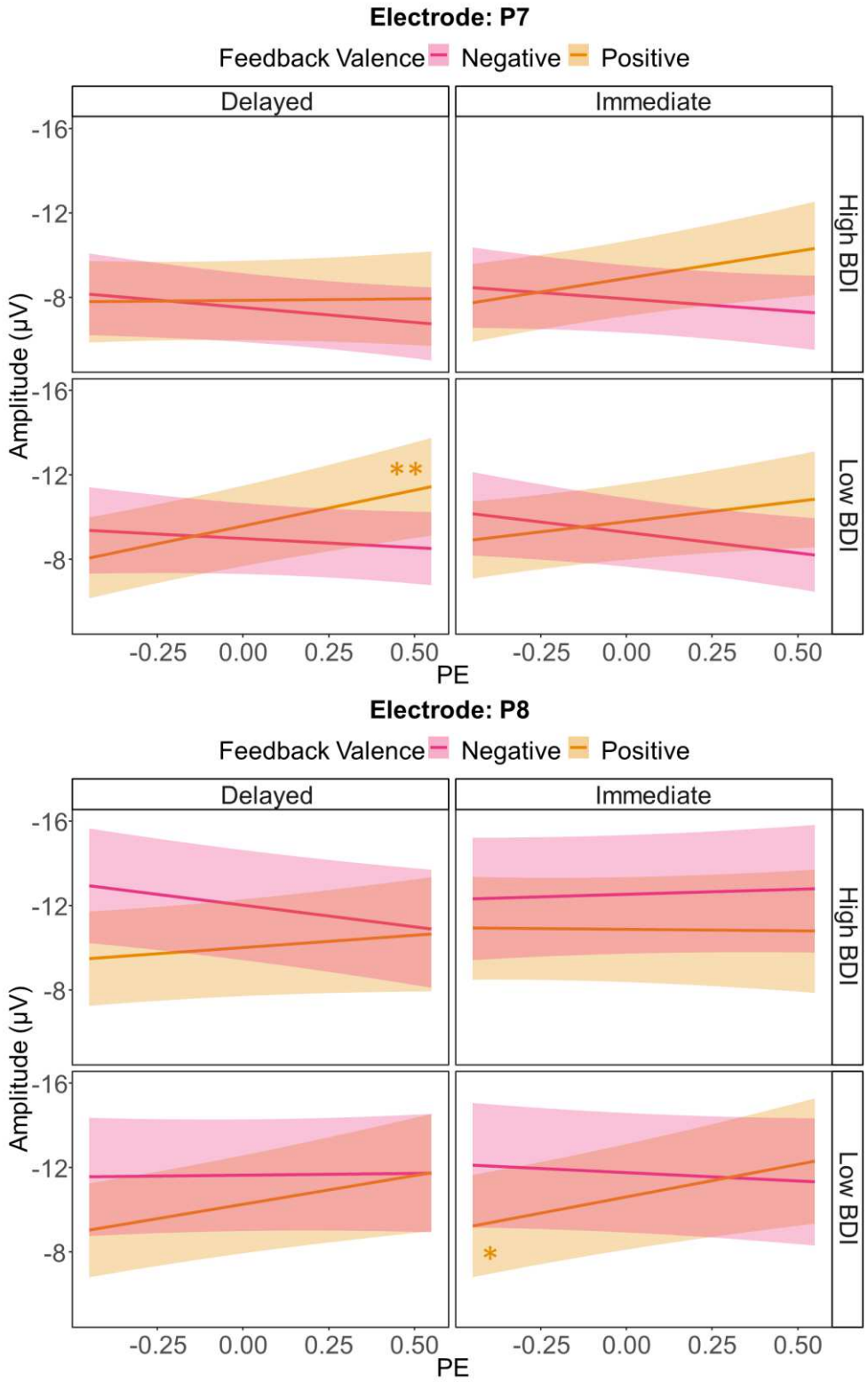


Note. The plots are based on descriptive data. **A** Feedback Valence x Electrode: $n = 45$. Error bars represent 95% confidence intervals. **B** PE x Feedback Valence: $n = 45$. Shaded areas represent 95% confidence intervals. **C** PE x Feedback Timing x BDI: $n = 45$. The high BDI graph represents data from participants with BDI-II scores ≥ median, the low BDI graph represents data from participants with BDI-II scores < the median. Shaded areas represent 95% confidence intervals. **D** Feedback Valence x Familial Vulnerability: $n = 37$. Low Familial Vulnerability represents descriptive data from participants with first-degree relatives without a history of depression, high Familial Vulnerability represents data from participants with first-degree relatives with a depression diagnosis. Error bars represent 95% confidence intervals.

Regarding effects of the BDI, we found a significant three-way interaction between BDI, Feedback Timing and PE (p = .047). The underlying descriptive data are presented in Figure 4C. Simple slope analyses revealed that the PE only had a significant effect on the N170 following delayed feedback in participants with low BDI values (i.e., BDI = -1SD; β = -1.58, SE = 0.48, t = -3.30, p = .012). In individuals with no or only minimal depressive symptoms, the N170 amplitude increased for more unexpected delayed feedback. All other simple slope analyses did not reach significance (all ps ≥ .330; see Table S6 in the Supporting Information for β-estimates and effect specific t-tests). The three-way interaction was further explained by a significant five-way interaction between all predictors included in the analysis (p = .041), which we resolved using simple slope analyses. Model plots for P7 and P8 separately are presented in Figure 5, Table S7 in the Supporting Information lists β-estimates and effect specific t-tests. The simple slope analyses revealed a significant effect of the PE on the N170 for the P7 in participants with low BDI scores after receiving delayed positive feedback (β = -3.36, SE = 0.92, t = -3.67, p = .004), with larger amplitudes for more unexpected feedback. For the P8, the simple slope analysis also revealed a significant PE effect on the N170, again in participants with low BDI scores and following positive feedback, but this time for immediate feedback (β = -3.09, SE = 1.00, t = -3.09, p = .034). Again, more unexpected positive feedback was associated with larger N170 amplitudes. Apart from these two significant slopes, the simple slope analyses did not reveal any further significant effects (all ps ≥ .051). All other main and interaction effects of the LME analysis including the BDI did not reach significance (all ps ≥ .063).

Figure 5

Model plots for the BDI x PE x Feedback Valence x Feedback Timing x Electrode interaction for the N170



Note. Depicted are linear mixed effects model-based predictions for the N170 amplitude at electrodes P7 and P8. Shaded areas indicate 95% confidence intervals. Stars indicate significance levels for the Bonferroni-corrected *p*-values that resulted from simple slopes analyses.
*$p < .050$, **$p < .010$

For the analysis containing the PHQ instead of the BDI as a predictor, Table S8 in the Supporting Information lists β-estimates and effect specific *t*-tests. To avoid redundancies, we only focus on effects involving the PHQ, while other effects (mainly replications of effects that were already described for the BDI analysis) are reported in the section titled N170 Analysis Including PHQ of the Supporting Information. The analysis revealed neither a significant main effect of PHQ, nor significant interactions involving PHQ (all *p*s ≥ .054).

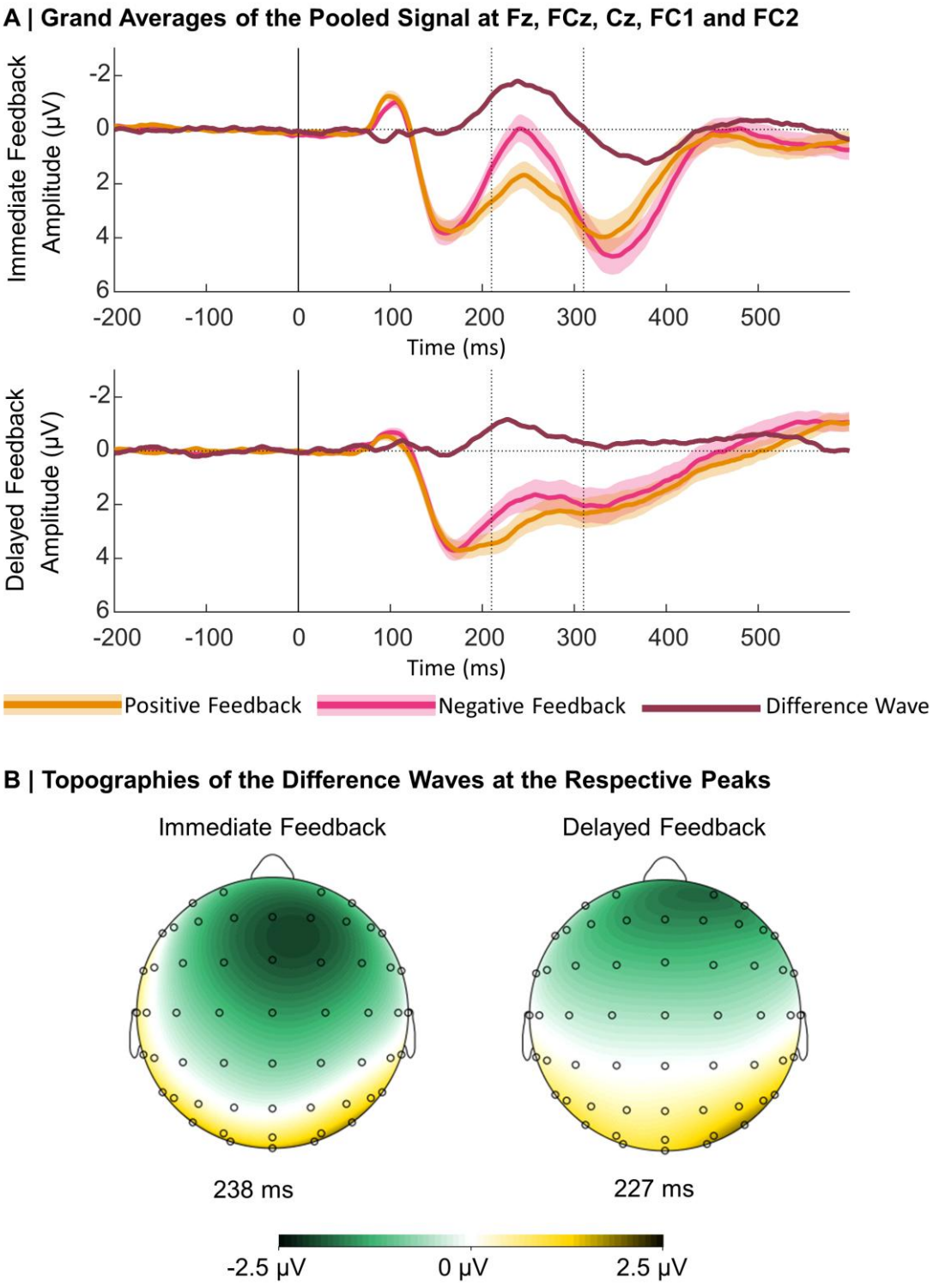
For the analysis containing Familial Vulnerability, Table S9 in the Supporting Information lists β-estimates and effect specific *t*-tests. As above, here we only report effects involving Familial Vulnerability, while other effects (again mainly replications already described for the BDI model above) are reported in the Supporting Information under the section titled N170 Analysis Including Familial Vulnerability. The analysis revealed a significant interaction between Feedback Valence and Familial Vulnerability ($p = .011$). The underlying descriptive data are presented in Figure 4D. Only participants without first-degree relatives with a history of depression showed significantly larger amplitudes for negative compared to positive feedback ($\beta = 0.93$, $SE = 0.29$, $t = 3.20$, $p = .006$), while there was no effect of Feedback Valence for participants with first-degree relatives with a diagnosed depression ($\beta = -0.74$, $SE = 0.54$, $t = -1.38$, $p = .358$). Apart from that, there were no other significant effects involving Familial Vulnerability (all *p*s ≥ .064).

FRN

Grand averages for the ERPs following positive and negative immediate and delayed feedback pooled over the frontocentral cluster of electrodes are presented in Figure 6.

Figure 6

Grand Averages and topographical maps of the FRN



Note. **A** Grand Averages: Dotted lines indicate the time window we used for the peak detection in the difference wave (negative – positive feedback). Shaded areas represent standard errors. **B** Topographies: The maps were constructed on the basis of the condition-specific difference waves.

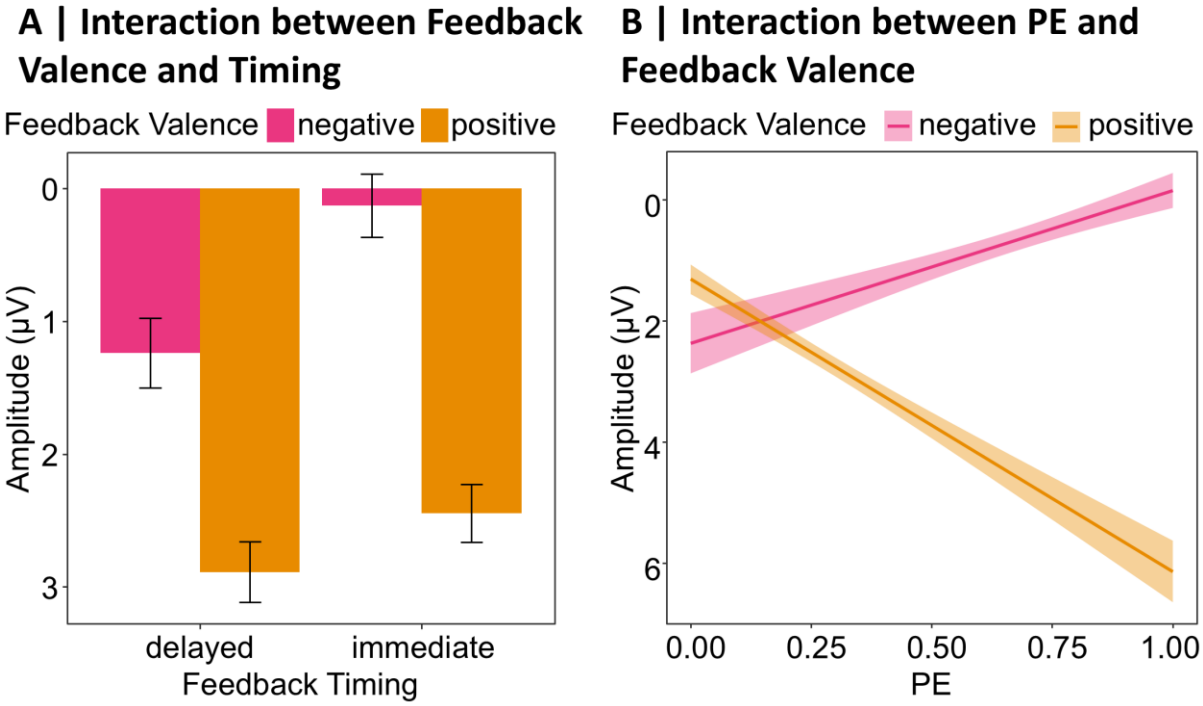
Similar to the results of the N170 analysis, we begin by reporting the results of the LME analysis on the FRN, including BDI, Feedback Timing, Feedback Valence, and PE as

predictors. Table S10 in the Supporting Information lists β-estimates and effect specific *t*-tests. The analysis revealed a significant effect of Feedback Valence ($p < .001$), with more negative amplitudes following negative compared to positive feedback. In addition, there was a significant effect of Feedback Timing ($p = .004$), indicating more negative amplitudes following immediate compared to delayed feedback. A significant interaction between Feedback Valence and Feedback Timing ($p = .008$) explained these effects further: Negative feedback was associated with more negative amplitudes for both immediate ($\beta = 2.59$, $SE = 0.26$, $t = 9.85$, $p < .001$) and delayed feedback ($\beta = 1.85$, $SE = 0.27$, $t = 6.80$, $p < .001$), but the effect was stronger for immediate feedback. Descriptive data underlying this interaction are presented in Figure 7A. Furthermore, we found a significant effect of PE ($p = .008$) that was further explained by a significant interaction between PE and Feedback Valence ($p < .001$), which we thus resolved. Descriptive data underlying this interaction are presented in Figure 7B. There was a significant effect of PE on the FRN amplitude for negative feedback, with more negative amplitudes for more unexpected feedback ($\beta = -1.98$, $SE = 0.49$, $t = -4.01$, $p = .001$). For positive feedback, this effect was reversed with more positive amplitudes for more unexpected feedback ($\beta = 3.75$, $SE = 0.57$, $t = 6.56$, $p < .001$). All other effects (including effects involving the BDI) were not significant (all ps $\geq .065$).

Tables S11 and S12 in the Supporting Information list β-estimates and effect specific t-tests for the two models in which BDI was replaced by either PHQ or Familial Vulnerability. For both models, we could replicate the findings described for the BDI model—they are reported in detail in the Supporting Information under the sections titled FRN Analysis Including PHQ and FRN Analysis Including Familial Vulnerability, respectively. Apart from that, we did not find significant effects, neither of PHQ nor of Familial Vulnerability (all ps $\geq .271$ for the model including PHQ and all ps $\geq .056$ for the model including Familial Vulnerability).

Figure 7

Descriptive data patterns underlying the FRN analyses



Note. The plots are based on descriptive data. **A** Feedback Valence x Feedback Timing: n = 45. Error bars represent 95% confidence intervals. **B** PE x Feedback Valence: n = 45. Shaded areas represent 95% confidence intervals.

Discussion

The present study aimed to investigate links between depressive symptoms and feedback learning and feedback processing using insights from two ERP components, FRN and N170, that have primarily been associated with immediate and delayed feedback processing, respectively. Previous studies reported a reduced differentiation between responses to gains and losses in FRN amplitudes in combination with reduced striatal processing in depression (Bress et al., 2012, 2015; Foti et al., 2014; Klawohn et al., 2021; Pizzagalli et al., 2009; Takamura et al., 2017; for reviews see Admon & Pizzagalli, 2015, Keren et al., 2018 and Luking et al., 2016). However, the striatum seems to be particularly important for immediate feedback processing, while for delayed feedback processing hippocampal activity is increased, which has been linked to the N170 ERP component (Arbel

et al., 2017; Foerde et al., 2013; Foerde & Shohamy, 2011; Höltje & Mecklinger, 2020; Kim & Arbel, 2019; Peterburs et al., 2016, Weinberg et al., 2012; Weismüller & Bellebaum, 2016). This is the first study to explicitly address effects of depressive symptoms on learning from and processing of immediate and delayed feedback. Besides trying to replicate findings of impaired learning (Admon et al., 2017; Bakic et al., 2017; Kumar et al., 2018; Kunisato et al., 2012; Macoveanu et al., 2014; Pechtel et al., 2013; Pizzagalli et al., 2005, 2008) and reduced differentiations between positive and negative feedback in FRN amplitudes following immediate feedback in depression (Bress et al., 2012, 2015; Foti et al., 2014; Klawohn et al., 2021), we hypothesized to find alterations for learning from delayed feedback and also delayed feedback processing as measured via the N170. As hypothesized, we found that learning performance decreased with more severe currently experienced depressive symptoms, irrespective of feedback delay. While we could not replicate findings of a reduced differentiation between responses to gains and losses in FRN amplitude for more severe depressive symptoms, we found PE coding in the N170 only in participants with low BDI scores, mainly driven by responses to (delayed) positive feedback. In addition, familial vulnerability for depression was linked to a reduced sensitivity for feedback valence encoded in the N170.

Effects of current depressive symptoms on feedback learning, FRN and N170

The ability to use positive and negative consequences of our actions to learn and shape future decisions is fundamental for human intelligent behavior (Silver et al., 2021). Accordingly, participants in our study successfully learned to choose the more rewarding out of two stimuli throughout a probabilistic feedback learning task that required an accumulation of experiences over time (Fu & Anderson, 2008). Previous studies indicated that depression interferes with the ability to learn from feedback (Admon et al., 2017; Bakic et al., 2017; Kumar et al., 2018; Kunisato et al., 2012; Macoveanu et al., 2014; Pechtel et al., 2013;

Pizzagalli et al., 2005, 2008; for reviews see Chen et al., 2015 and Eshel & Roiser, 2010). In line with this and our hypothesis, we found that participants currently experiencing more severe depressive symptoms showed worse learning performance in the probabilistic feedback learning task, regardless of feedback timing. This is in line with our assumption that both, learning from immediate and delayed feedback, is affected by depression, possibly caused by changes in the striatum and hippocampus (Admon & Pizzagalli, 2015; Fairhall et al., 2010; Luking et al., 2016; Nestler et al., 2002; Pizzagalli et al., 2009; Takamura et al., 2017; Thompson, 2023) that are both involved in feedback processing (Foerde et al., 2013; Foerde & Shohamy, 2011).

However, these behavioral alterations were not reflected in the FRN: Against our hypothesis, we did not find reduced feedback valence sensitivity in FRN amplitudes in participants currently experiencing depressive symptoms, which is not in line with previous findings (Bress et al., 2012, 2015; Foti et al., 2014; Klawohn et al., 2021). An explorative investigation revealed that PE coding in the FRN was also not affected by the severity of currently experienced depressive symptoms. In contrast, a study by Jackson and Cavanagh (2023) indicated that low mood can influence reward learning via poorer prediction error coding in the RewP. On the one hand, our results seem to support studies that found no or only weak, task-dependent relationships between the FRN and depression (Hager et al., 2021; Clayson et al., 2020; Moran et al., 2017). On the other hand, the EEG signal is influenced by many cognitive processes, and their spatiotemporal overlaps can make it difficult to link individual performance to ERP deflections (Ullsperger, 2024). Thus, dissociations between the FRN and behavior are not an uncommon finding (Ullsperger, 2024), indicating that other neural processes, reflected in other feedback-locked ERP components, might be more closely linked to behavior.

The present study is the first to investigate depression-related alterations in the feedback-locked N170. Previous studies found more pronounced N170 amplitudes following delayed feedback compared to immediate feedback (Arbel et al., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019). Because of the role of the hippocampus for delayed feedback processing (Foerde et al., 2013; Foerde & Shohamy, 2011), the N170 has been interpreted to reflect MTL activity (Arbel et a., 2017; Höltje & Mecklinger, 2020; Kim & Arbel, 2019). Depression can be accompanied by changes in hippocampal structure and functioning, possibly explaining memory impairments and some of the cognitive symptoms seen in depression (Fairhall et al., 2010; Nestler et al., 2002; Thompson, 2023). Therefore, we expected reduced N170 amplitudes for participants currently experiencing more severe depressive symptoms, especially following delayed feedback. While we could not find the hypothesized pattern, currently experienced depressive symptoms affected PE coding reflected in the N170. More specifically, we found reflections of the PE in the N170 amplitude only in participants with low levels of depressive symptoms and especially following (delayed) positive feedback. For these participants, more unexpected positive feedback was linked to more pronounced N170 amplitudes. While this is not exactly what we had hypothesized, it still matches our assumption that structures and processes that are involved in generating the N170 are altered in depression.

For the feedback-locked N170, it was only once previously described that it reflects the entire range of the PE (Röhlinger et al., 2025), with an opposite pattern compared to the FRN (Burnside et al., 2019; Fischer & Ullsperger, 2013; Weber & Bellebaum, 2024): the more unexpected positive feedback was, the more negative (pronounced) the N170 became and the more unexpected negative feedback was, the more positive it became. In the present study we could replicate this finding. Since midbrain dopamine neurons seem to send information not only to striatal and fronto-cortical areas of the brain (Schultz, 2002), but also

to the hippocampus (Calabresi et al., 2013; Tsetsenis et al., 2013), the described pattern could mean that the MTL is especially involved in reactivating representations of unexpectedly rewarded stimuli to link them to (temporally delayed) feedback (Röhlinger et al., 2025). While in healthy individuals, midbrain dopamine regions and the MTL seem to enhance memory representations of rewarded stimuli to adapt future behavior (Shohamy & Adcock, 2010), our results suggest that this process might be disrupted by acute depression. This would be in line with findings of hippocampal atrophy and changes in cognitive functions like memory (Fairhall et al., 2010; Nestler et al., 2002; Thompson, 2023). In addition, acute depressive symptoms come along with anhedonia, reduced reward responsiveness and altered feedback learning processes (Admon et al., 2017; Bakic et al., 2017; Huys et al., 2013; Kumar et al., 2018; Kunisato et al., 2012; Rizvi et al., 2016), which might be related to the reduced PE coding following positive feedback in the present study.

Effects of past depressive episodes on feedback learning, FRN and N170

Since individuals who have recovered from depression still show blunted responses to reward in the striatum (McCabe et al., 2009), we intended to not only look at effects of currently experienced depressive symptoms, but also investigate effects of past depressive symptom severity on feedback learning and processing, with a new focus on delayed feedback processing and potentially reduced N170 amplitudes. However, we did not find any effects of past depressive episodes as measured via a modified version of the PHQ-9. The modified version of the PHQ-9 was introduced as a measure of lifetime depression (Cannon et al., 2007) in which participants rate how strongly they have experienced a list of depressive symptoms in the two weeks in their life in which they have felt most sad or depressed. However, scoring high on this questionnaire does not necessarily indicate that a person went through a major depressive episode in the past. For example, losing a beloved person can cause a feeling of sadness or emptiness, a loss of pleasure or interest in activities, or one of

the other symptoms that are among the diagnostic criteria for a major depressive episode according to the International Classification of Diseases (ICD-11, World Health Organization, 2019) and that are assessed by the modified PHQ-9. Apparently, there is an overlap of symptoms between grief and depression and although grief can culminate in a major depression, it is usually not pathological (Shear et al., 2011; Zisook & Shear, 2009). We assume that the modified PHQ-9 used in this study might have been unsuitable to reliably and validly assess clinically relevant past depressive episodes.

Effects of familial vulnerability on feedback learning, FRN and N170

A familial history of depression, for example having a depressed mother, poses a risk of developing depression (Halligan et al., 2007; Raposa et al., 2014). Accordingly, blunted FRN amplitudes following rewards were found in siblings of depressed individuals who were not depressed themselves (Weinberg et al., 2015). Blunted responses to reward within the dorsal and ventral striatum relative to children of no-depressed mothers serve as neurophysiological explanation for the increased risk and altered FRN amplitudes (for an extensive review see Luking et al., 2016). We intended to find out whether familial vulnerability for depression, besides affecting the FRN following immediate feedback, also affects the processing of delayed feedback, for example indicated by reduced N170 amplitudes. First of all, familial vulnerability did not affect behavioral response accuracy in the probabilistic feedback learning task and we did not find vulnerability-related changes in the FRN in terms of a reduced sensitivity for feedback valence. However, we observed such an effect for the N170, independent of feedback timing: In general, the N170 in this study was more pronounced following negative than positive feedback (for similar results see Kim & Arbel, 2019), but only over the right hemisphere. This sensitivity for feedback valence was not found in participants with a familial history of depression. In other words, the amplitude difference between positive and negative feedback in the N170 diminished for participants at

increased risk for depression. In this line, an fMRI study on 10- to 14-year-old girls with a familial history of depression found reduced striatal activity in response to rewards and increased activation in the dorsal anterior cingulate cortex following losses (compared to peers without such a family history) even before the onset of depressive symptoms (Gotlib et al., 2010). Similar alterations in the activity of structures underlying the N170 could account for the altered processing of positive and negative feedback observed in this study.

Limitations and Future Studies

While it seems to be justified to consider depression not as a dichotomous variable, there are also limitations of this approach. The participants in our study reached a mean BDI score of 9.89 (SD = 9.34), which is below the cut-off score for depression (von Glischinski et al., 2019). Some participants scored really low (Min = 0) and the maximum score was 39 in our sample (with respect to a theoretically possible score of 63), indicating that the variance in currently experienced depressive symptomatology was limited. Even though Bress et al. (2012, 2015) found correlations between the FRN amplitude and self-rated depressive symptoms in a non-clinical sample, their sample cannot be compared to ours, as it consisted of 8- to 13-year-old children/adolescents, and depression-related alterations in the FRN seem to be most pronounced in individuals under age 18 (Keren et al., 2018). A lack of participants with very high BDI scores may have prevented us from finding such a relationship in an adult sample (Clayson et al., 2020). In addition, self-reported depression assessed via the BDI-II in our mostly undergraduate academic participants may have been confounded by academic or peer-related stressors and therefore less indicative of a major depressive disorder (Hager et al., 2021). On the contrary, the variance in BDI scores in our sample was sufficient to detect effects on behavioral response accuracy and the N170. Other studies that found effects of depression on the FRN compared clinical samples, i.e., participants that met the criteria for a clinical diagnosis of unipolar depression (and reached a BDI score $\geq$ 13), with healthy

controls (Foti et al., 2014; Klawohn et al., 2021). Future studies could try to overcome variance issues by combining group designs (clinical vs. non-clinical sample) with the inclusion of depression as a continuous variable in the analysis.

Another sample-related limitation of our results involves the prevalence of other mental disorders apart from depression, which were identified by the Mini-DIPS. For example, 26% of our participants met the criteria for a social anxiety disorder (currently or in the past). Heightened responses to negative feedback in the FRN have been observed in anxiety (Tobias & Ito, 2021), which may blur effects of depressive symptoms. Grabowska et al. (2024) explain that due to evidence for ERP components being modulated by various interindividual differences, focusing on a small set of them might be problematic: Mental disorders like anxiety and depression are interconnected and their influence on an individual's way of processing feedback may be either direct or indirect. Furthermore, depression encompasses a wide variety of emotional, cognitive, behavioral and neurovegetative symptoms (see ICD-11, World Health Organization, 2019) and understanding the complex relationships between them and feedback processing requires further investigation. For example, it is possible that only a specific subtype of depression—particularly characterized by anhedonia—exhibits alterations at the level of the FRN. Finally, heterogeneity in the etiology of depression (Kendler et al., 2002) may account for the difficulties of replicating effects on feedback processing. Grabowska et al. (2024) suggest to tackle the challenges in research on relationships between ERP components and psychopathologies with network analysis techniques.

Conclusion

In the present study, we found that performance in a learning task decreased with more severe depressive symptoms, for learning from both immediate and delayed feedback. While the FRN was unaffected by acute depressive symptom severity, past depressive

episodes, and familial vulnerability for depression, we found depression-related changes in the N170, remarkably for both immediate and delayed feedback processing. Currently experienced depressive symptoms were associated with poorer encoding of prediction errors in the N170, possibly explaining reduced learning performance. In addition, a family history of depression was associated with reduced sensitivity to feedback valence in the N170. Thus, the N170 emerges as a novel, important biomarker alongside the FRN in clinical research on depression and feedback-based learning processes.

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Author Contributions

Madita Röhlinger: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Validation, Visualization, Writing - original draft, Writing - review & editing; Christian Bellebaum: Conceptualization, Methodology, Project administration, Resources, Supervision, Validation, Writing - review & editing.

Data and Code Availability

All data supporting the findings are openly accessible through the Open Science Framework at https://osf.io/qu7td/.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors used OpenAI's GPT-4 and DeepL Translator in order to translate text, improve readability of the article and adapt data analysis scripts. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

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References

Aarts, K., & Pourtois, G. (2012). Anxiety disrupts the evaluative component of performance monitoring: An ERP study. *Neuropsychologia*, *50*(7), 1286-1296. https://doi.org/10.1016/j.neuropsychologia.2012.02.012

Admon, R., Kaiser, R. H., Dillon, D. G., Beltzer, M., Goer, F., Olson, D. P., Vitaliano, G., & Pizzagalli, D. A. (2017). Dopaminergic Enhancement of Striatal Response to Reward in Major Depression. *The American Journal of Psychiatry*, *174*(4), 378–386. https://doi.org/10.1176/appi.ajp.2016.16010111

Admon, R., & Pizzagalli, D. A. (2015). Dysfunctional Reward Processing in Depression. *Current Opinion in Psychology*, *4*, 114–118. https://doi.org/10.1016/j.copsyc.2014.12.011

Albrecht, C., van de Vijver, R., & Bellebaum, C. (2023). Learning new words via feedback-Association between feedback-locked ERPs and recall performance-An exploratory study. *Psychophysiology*, e14324-e14324. https://doi.org/10.1111/psyp.14324

Arbel, Y., Hong, L., Baker, T. E., & Holroyd, C. B. (2017). It's all about timing: An electrophysiological examination of feedback-based learning with immediate and delayed feedback. *Neuropsychologia*, *99*, 179–186. https://doi.org/10.1016/j.neuropsychologia.2017.03.003

Bakic, J., Pourtois, G., Jepma, M., Duprat, R., Raedt, R. de, & Baeken, C. (2017). Spared internal but impaired external reward prediction error signals in major depressive disorder during reinforcement learning. *Depression and Anxiety*, *34*(1), 89–96. https://doi.org/10.1002/da.22576

Bates D, Mächler M, Bolker B, Walker S (2015). "Fitting Linear Mixed-Effects Models Using lme4." *Journal of Statistical Software*, *67*(1), 1–48. https://doi.org/10.18637/jss.v067.i01

Beck, A. T., Steer, R. A., & Brown, G. (1996). Beck depression inventory–II. *Psychological assessment*. https://psycnet.apa.org/doi/10.1037/t00742-000

Becker, M. P., Nitsch, A. M., Miltner, W. H., & Straube, T. (2014). A single-trial estimation of the feedback-related negativity and its relation to BOLD responses in a time-estimation task. *Journal of Neuroscience*, *34*(8), 3005-3012. https://doi.org/10.1523/JNEUROSCI.3684-13.2014

Belden, A. C., Irvin, K., Hajcak, G., Kappenman, E. S., Kelly, D., Karlow, S., ... & Barch, D. M. (2016). Neural correlates of reward processing in depressed and healthy preschool-age children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *55*(12), 1081-1089. https://doi.org/10.1016/j.jaac.2016.09.503

Bellato, A., Norman, L., Idrees, I., Ogawa, C. Y., Waitt, A., Zuccolo, P. F., ... & Shephard, E. (2021). A systematic review and meta-analysis of altered electrophysiological markers of performance monitoring in Obsessive-Compulsive Disorder (OCD), Gilles de la Tourette Syndrome (GTS), Attention-Deficit/Hyperactivity disorder (ADHD) and Autism. *Neuroscience & Biobehavioral Reviews*, *131*, 964-987. https://doi.org/10.1016/j.neubiorev.2021.10.018

Bellebaum, C., & Daum, I. (2008). Learning-related changes in reward expectancy are reflected in the feedback-related negativity. *European Journal of Neuroscience*, *27*(7), 1823-1835. https://doi.org/10.1111/j.1460-9568.2008.06138.x

Björklund, A., & Dunnett, S. B. (2007). Dopamine neuron systems in the brain: an update. *Trends in neurosciences*, *30*(5), 194-202. https://doi.org/10.1016/j.tins.2007.03.006

Brain Products GmbH. (2018). BrainVision Analyzer 2.2 [Computer software]. Gilching, Germany: Brain Products GmbH.

Bress, J. N., Foti, D., Kotov, R., Klein, D. N., & Hajcak, G. (2013). Blunted neural response to rewards prospectively predicts depression in adolescent girls. *Psychophysiology*, *50*(1), 74-81. https://doi.org/10.1111/j.1469-8986.2012.01485.x

Bress, J. N., Meyer, A., & Proudfit, G. H. (2015). The stability of the feedback negativity and its relationship with depression during childhood and adolescence. *Development and Psychopathology*, *27*(4 Pt 1), 1285–1294. https://doi.org/10.1017/S0954579414001400

Bress, J. N., Smith, E., Foti, D., Klein, D. N., & Hajcak, G. (2012). Neural response to reward and depressive symptoms in late childhood to early adolescence. *Biological Psychology*, *89*(1), 156–162. https://doi.org/10.1016/j.biopsycho.2011.10.004

Brush, C. J., Ehmann, P. J., Hajcak, G., Selby, E. A., & Alderman, B. L. (2018). Using multilevel modeling to examine blunted neural responses to reward in major depression. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *3*(12), 1032-1039. https://doi.org/10.1016/j.bpsc.2018.04.003

Burnside, R., Fischer, A. G., & Ullsperger, M. (2019). The feedback-related negativity indexes PE in active but not observational learning. *Psychophysiology*, *56*(9), e13389. https://doi.org/10.1111/psyp.13389

Calabresi, P., Castrioto, A., Di Filippo, M., & Picconi, B. (2013). New experimental and clinical links between the hippocampus and the dopaminergic system in Parkinson's disease. *The Lancet Neurology*, *12*(8), 811-821. https://doi.org/10.1016/S1474-4422(13)70118-2

Cannon, D. S., Tiffany, S. T., Coon, H., Scholand, M. B., McMahon, W. M., & Leppert, M. F. (2007). The PHQ-9 as a brief assessment of lifetime major depression.

Psychological assessment, *19*(2), 247-251. https://psycnet.apa.org/doi/10.1037/1040-3590.19.2.247

Carlson, J. M., Foti, D., Mujica-Parodi, L. R., Harmon-Jones, E., & Hajcak, G. (2011). Ventral striatal and medial prefrontal BOLD activation is correlated with reward-related electrocortical activity: a combined ERP and fMRI study. *Neuroimage*, *57*(4), 1608-1616. https://doi.org/10.1016/j.neuroimage.2011.05.037

Chen, C., Takahashi, T., Nakagawa, S., Inoue, T., & Kusumi, I. (2015). Reinforcement learning in depression: A review of computational research. *Neuroscience and Biobehavioral Reviews*, *55*, 247–267. https://doi.org/10.1016/j.neubiorev.2015.05.005

Clayson, P. E., Carbine, K. A., & Larson, M. J. (2020). A registered report of error-related negativity and reward positivity as biomarkers of depression: P-Curving the evidence. *International Journal of Psychophysiology*, *150*, 50-72. https://doi.org/10.1016/j.ijpsycho.2020.01.005

Dillon, D. G., & Pizzagalli, D. A. (2018). Mechanisms of Memory Disruption in Depression. *Trends in Neurosciences*, *41*(3), 137–149. https://doi.org/10.1016/j.tins.2017.12.006

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

Fairhall, S. L., Sharma, S., Magnusson, J., & Murphy, B. (2010). Memory related dysregulation of hippocampal function in major depressive disorder. *Biological Psychology*, *85*(3), 499–503. https://doi.org/10.1016/j.biopsycho.2010.09.002

First, M. B., & Gibbon, M. (2004). The structured clinical interview for DSM-IV axis I disorders (SCID-I) and the structured clinical interview for DSM-IV axis II disorders (SCID-II). In M. J. Hilsenroth & D. L. Segal (Eds.), *Comprehensive handbook of psychological assessment, Vol. 2. Personality assessment* (pp. 134–143).

Fischer, A. G., & Ullsperger, M. (2013). Real and fictive outcomes are processed differently but converge on a common adaptive mechanism. *Neuron*, *79*(6), 1243-1255. https://doi.org/10.1016/j.neuron.2013.07.006

Foerde, K., Race, E., Verfaellie, M., & Shohamy, D. (2013). A role for the medial temporal lobe in feedback-driven learning: Evidence from amnesia. *Journal of Neuroscience*, *33*(13), 5698–5704. https://doi.org/10.1523/JNEUROSCI.5217-12.2013

Foerde, K., & Shohamy, D. (2011). Feedback timing modulates brain systems for learning in humans. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *31*(37), 13157–13167. https://doi.org/10.1523/JNEUROSCI.2701-11.2011

Forester, G., Schaefer, L. M., Johnson, J. S., Amponsah, T., Dvorak, R. D., & Wonderlich, S. A. (2024). Neurocognitive reward processes measured via event-related potentials are associated with binge-eating disorder diagnosis and ecologically-assessed behavior. *Appetite*, *193*, 107151. https://doi.org/10.1016/j.appet.2023.107151

Foti, D., Carlson, J. M., Sauder, C. L., & Proudfit, G. H. (2014). Reward dysfunction in major depression: Multimodal neuroimaging evidence for refining the melancholic phenotype. *NeuroImage*, *101*, 50–58. https://doi.org/10.1016/j.neuroimage.2014.06.058

Foti, D., Weinberg, A., Dien, J., & Hajcak, G. (2011). Event-related potential activity in the basal ganglia differentiates rewards from nonrewards: Temporospatial principal components analysis and source localization of the feedback negativity. *Human brain mapping*, *32*(12), 2207-2216. https://doi.org/10.1002/hbm.21182

Frank, M. J., Seeberger, L. C., & O'reilly, R. C. (2004). By carrot or by stick: Cognitive reinforcement learning in parkinsonism. *Science*, *306*(5703), 1940–1943. https://doi.org/10.1126/science.1102941

Fu, W. T., & Anderson, J. R. (2008). Solving the credit assignment problem: explicit and implicit learning of action sequences with probabilistic outcomes. *Psychological research*, *72*, 321-330. https://doi.org/10.1007/s00426-007-0113-7

Glimcher, P. W. (2011). Understanding dopamine and reinforcement learning: the dopamine reward prediction error hypothesis. *Proceedings of the National Academy of Sciences*, *108*(supplement_3), 15647-15654. https://doi.org/10.1073/pnas.1014269108

Gotlib, I. H., Hamilton, J. P., Cooney, R. E., Singh, M. K., Henry, M. L., & Joormann, J. (2010). Neural processing of reward and loss in girls at risk for major depression. *Archives of general psychiatry*, *67*(4), 380-387. https://doi.org/10.1001/archgenpsychiatry.2010.13

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *The American Journal of Psychiatry*, *160*(4), 636–645. https://doi.org/10.1176/appi.ajp.160.4.636

Grabowska, A., Sondej, F., & Senderecka, M. (2024). A network analysis of affective and motivational individual differences and error monitoring in a non-clinical sample. *Cerebral Cortex*, *34*(10), bhae397. https://doi.org/10.1093/cercor/bhae397

Gräfe, K., Zipfel, S., Herzog, W., & Löwe, B. (2004). Screening psychischer Störungen mit dem “Gesundheitsfragebogen für Patienten (PHQ-D)“. *Diagnostica*, *50*(4), 171-181. https://doi.org/10.1026/0012-1924.50.4.171

Gu, R., Ge, Y., Jiang, Y., & Luo, Y. J. (2010). Anxiety and outcome evaluation: the good, the bad and the ambiguous. *Biological psychology*, *85*(2), 200-206. https://doi.org/10.1016/j.biopsycho.2010.07.001

Haber, S. N., & Knutson, B. (2010). The reward circuit: linking primate anatomy and human imaging. *Neuropsychopharmacology*, *35*(1), 4-26. https://doi.org/10.1038/npp.2009.129

Hager, N. M., Judah, M. R., & Rawls, E. (2021). Win, lose, or draw: Examining salience, reward memory, and depression with the reward positivity. *Psychophysiology*, *59*(1), e13953. https://doi.org/10.1111/psyp.13953

Halligan, S. L., Murray, L., Martins, C., & Cooper, P. J. (2007). Maternal depression and psychiatric outcomes in adolescent offspring: A 13-year longitudinal study. *Journal of Affective Disorders*, *97*(1-3), 145–154. https://doi.org/10.1016/j.jad.2006.06.010

Hautzinger, M., Keller, F., & Kühner, C. (2006). *BDI II*. Frankfirt a.M.: Harcourt Test Services

Heshmati, M., & Russo, S. J. (2015). Anhedonia and the brain reward circuitry in depression. *Current Behavioral Neuroscience Reports*, *2*(3), 146–153. https://doi.org/10.1007/s40473-015-0044-3

Hidaka, B. H. (2012). Depression as a disease of modernity: Explanations for increasing prevalence. *Journal of Affective Disorders*, *140*(3), 205–214. https://doi.org/10.1016/j.jad.2011.12.036

Holroyd, C. B., & Coles, M. G. (2002). The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. *Psychological review*, *109*(4), 679-709. https://psycnet.apa.org/doi/10.1037/0033-295X.109.4.679

Holroyd, C. B., Larsen, J. T., & Cohen, J. D. (2004). Context dependence of the event-related brain potential associated with reward and punishment. *Psychophysiology*, *41*(2), 245–253. https://doi.org/10.1111/j.1469-8986.2004.00152.x

Höltje, G., & Mecklinger, A. (2020). Feedback timing modulates interactions between feedback processing and memory encoding: Evidence from event-related potentials. *Cognitive, Affective, & Behavioral Neuroscience*, *20*(2), 250–264. https://doi.org/10.3758/s13415-019-00765-5

Huys, Q. J., Pizzagalli, D. A., Bogdan, R., & Dayan, P. (2013). Mapping anhedonia onto

reinforcement learning: a behavioural meta-analysis. *Biology of mood & anxiety disorders*, *3*, 1-16. https://doi.org/10.1186/2045-5380-3-12

Jackson, T. C., & Cavanagh, J. F. (2023). Reduced positive affect alters reward learning via reduced information encoding in the Reward Positivity. *Psychophysiology*, *60*(8), e14276. https://doi.org/10.1111/psyp.14276

Jacobi, F., Höfler, M., Siegert, J., Mack, S., Gerschler, A., Scholl, L., ... & Wittchen, H. U. (2014). Twelve-month prevalence, comorbidity and correlates of mental disorders in Germany: the Mental Health Module of the German Health Interview and Examination Survey for Adults (DEGS1-MH). *International journal of methods in psychiatric research*, *23*(3), 304-319. https://doi.org/10.1002/mpr.1439

Jiang, D., Zhang, D., Chen, Y., He, Z., Gao, Q., Gu, R., & Xu, P. (2018). Trait anxiety and probabilistic learning: Behavioral and electrophysiological findings. *Biological Psychology*, *132*, 17-26. https://doi.org/10.1016/j.biopsycho.2017.10.010

Kendler, K. S., Gardner, C. O., & Prescott, C. A. (2002). Toward a comprehensive developmental model for major depression in women. *American Journal of Psychiatry*, *159*(7), 1133-1145. https://doi.org/10.1176/appi.ajp.159.7.1133

Keren, H., O'Callaghan, G., Vidal-Ribas, P., Buzzell, G. A., Brotman, M. A., Leibenluft, E., Pan, P. M., Meffert, L., Kaiser, A., Wolke, S., Pine, D. S., & Stringaris, A. (2018). Reward Processing in Depression: A Conceptual and Meta-Analytic Review Across fMRI and EEG Studies. *The American Journal of Psychiatry*, *175*(11), 1111–1120. https://doi.org/10.1176/appi.ajp.2018.17101124

Kim, S., & Arbel, Y. (2019). Immediate and delayed auditory feedback in declarative learning: An examination of the feedback related event related potentials. *Neuropsychologia*, *129*, 255–262. https://doi.org/10.1016/j.neuropsychologia.2019.04.001

Klawohn, J., Burani, K., Bruchnak, A., Santopetro, N., & Hajcak, G. (2021). Reduced neural response to reward and pleasant pictures independently relate to depression. *Psychological Medicine*, *51*(5), 741-749. https://doi.org/10.1017/S0033291719003659

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9: validity of a brief depression severity measure. *Journal of general internal medicine*, *16*(9), 606-613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x

Kumar, P., Goer, F., Murray, L., Dillon, D. G., Beltzer, M. L., Cohen, A. L., Brooks, N. H., & Pizzagalli, D. A. (2018). Impaired reward prediction error encoding and striatal-midbrain connectivity in depression. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *43*(7), 1581–1588. https://doi.org/10.1038/s41386-018-0032-x

Kunisato, Y., Okamoto, Y., Ueda, K., Onoda, K., Okada, G., Yoshimura, S., Suzuki, S., Samejima, K., & Yamawaki, S. (2012). Effects of depression on reward-based decision making and variability of action in probabilistic learning. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(4), 1088–1094. https://doi.org/10.1016/j.jbtep.2012.05.007

Lamers, F., van Oppen, P., Comijs, H. C., Smit, J. H., Spinhoven, P., van Balkom, A. J., ... & Penninx, B. W. (2011). Comorbidity patterns of anxiety and depressive disorders in a large cohort study: the Netherlands Study of Depression and Anxiety (NESDA). *The Journal of clinical psychiatry*, *72*(3), 341-348.

Lefebvre, G., Lebreton, M., Meyniel, F., Bourgeois-Gironde, S., & Palminteri, S. (2017). Behavioural and neural characterization of optimistic reinforcement learning. *Nature Human Behaviour*, *1*(4), 0067. https://doi.org/10.1038/s41562-017-0067

Luck, S. J. (2014). *An introduction to the event-related potential technique*. MIT press.

Luking, K. R., Pagliaccio, D., Luby, J. L., & Barch, D. M. (2016). Reward Processing and Risk for Depression Across Development. *Trends in Cognitive Sciences*, *20*(6), 456–468. https://doi.org/10.1016/j.tics.2016.04.002

Macoveanu, J., Knorr, U., Skimminge, A., Søndergaard, M. G., Jørgensen, A., Fauerholdt-Jepsen, M., Paulson, O. B., Knudsen, G. M., Siebner, H. R., & Kessing, L. V. (2014). Altered reward processing in the orbitofrontal cortex and hippocampus in healthy first-degree relatives of patients with depression. *Psychological Medicine*, *44*(6), 1183–1195. https://doi.org/10.1017/S0033291713001815

Margraf, J. & Cwik, J. C. (2017). Mini-DIPS Open Access: Diagnostisches Kurzinterview bei psychischen Störungen. Bochum: Forschungs- und Behandlungszentrum für psychische Gesundheit, Ruhr-Universität Bochum. https://doi.org/10.13154/rub.102.91

Margraf, J., Cwik, J. C., Pflug, V., & Schneider, S. (2017). Strukturierte klinische Interviews zur Erfassung psychischer Störungen über die Lebensspanne. *Zeitschrift für Klinische Psychologie und Psychotherapie*. 176-186. https://doi.org/10.1026/1616-3443/a000430

McCabe, C., Cowen, P. J., & Harmer, C. J. (2009). Neural representation of reward in recovered depressed patients. *Psychopharmacology*, *205*(4), 667–677. https://doi.org/10.1007/s00213-009-1573-9

Meteyard, L., & Davies, R. A. (2020). Best practice guidance for linear mixed-effects models in psychological science. *Journal of Memory and Language*, *112*, 104092. https://doi.org/10.1016/j.jml.2020.104092

Miltner, W. H., Braun, C. H., & Coles, M. G. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a “generic” neural system

for error detection. *Journal of Cognitive Neuroscience*, *9*(6), 788–798. https://doi.org/10.1162/jocn.1997.9.6.788

Moran, T. P., Schroder, H. S., Kneip, C., & Moser, J. S. (2017). Meta-analysis and psychophysiology: A tutorial using depression and action-monitoring event-related potentials. *International Journal of Psychophysiology*, *111*, 17-32. https://doi.org/10.1016/j.ijpsycho.2016.07.001

Nestler, E. J., Barrot, M., DiLeone, R. J., Eisch, A. J., Gold, S. J., & Monteggia, L. M. (2002). Neurobiology of depression. *Neuron*, *34*(1), 13-25. https://doi.org/10.1016/S0896-6273(02)00653-0

Oerlemans, J., Alejandro, R. J., Van Roost, D., Boon, P., De Herdt, V., Meurs, A., & Holroyd, C. B. (2025). Unravelling the origin of reward positivity: a human intracranial event-related brain potential study. *Brain*, *148*(1), 199-211. https://doi.org/10.1093/brain/awae259

Pechtel, P., Dutra, S. J., Goetz, E. L., & Pizzagalli, D. A. (2013). Blunted reward responsiveness in remitted depression. *Journal of Psychiatric Research*, *47*(12), 1864–1869. https://doi.org/10.1016/j.jpsychires.2013.08.011

Peterburs, J., Kobza, S., & Bellebaum, C. (2016). Feedback delay gradually affects amplitude and valence specificity of the feedback-related negativity (FRN). *Psychophysiology*, *53*(2), 209–215. https://doi.org/10.1111/psyp.12560

Pizzagalli, D. A., Holmes, A. J., Dillon, D. G., Goetz, E. L., Birk, J. L., Bogdan, R., Dougherty, D. D., Iosifescu, D. V., Rauch, S. L., & Fava, M. (2009). Reduced caudate and nucleus accumbens response to rewards in unmedicated individuals with major depressive disorder. *The American Journal of Psychiatry*, *166*(6), 702–710. https://doi.org/10.1176/appi.ajp.2008.08081201

Pizzagalli, D. A., Iosifescu, D., Hallett, L. A., Ratner, K. G., & Fava, M. (2008). Reduced hedonic capacity in major depressive disorder: Evidence from a probabilistic reward task. *Journal of Psychiatric Research*, *43*(1), 76–87. https://doi.org/10.1016/j.jpsychires.2008.03.001

Pizzagalli, D. A., Jahn, A. L., & O'Shea, J. P. (2005). Toward an objective characterization of an anhedonic phenotype: A signal-detection approach. *Biological Psychiatry*, *57*(4), 319–327. https://doi.org/10.1016/j.biopsych.2004.11.026

Proudfit, G. H. (2015). The reward positivity: From basic research on reward to a biomarker for depression. *Psychophysiology*, *52*(4), 449-459. https://doi.org/10.1111/psyp.12370

Raposa, E., Hammen, C., Brennan, P., & Najman, J. (2014). The long-term effects of maternal depression: Early childhood physical health as a pathway to offspring depression. *The Journal of Adolescent Health: Official Publication of the Society for Adolescent Medicine*, *54*(1), 88–93. https://doi.org/10.1016/j.jadohealth.2013.07.038

Rizvi, S. J., Pizzagalli, D. A., Sproule, B. A., & Kennedy, S. H. (2016). Assessing anhedonia in depression: Potentials and pitfalls. *Neuroscience & Biobehavioral Reviews*, *65*, 21-35. https://doi.org/10.1016/j.neubiorev.2016.03.004

Röhlinger, M., Albrecht, C., & Bellebaum, C. (2025). The Role of the N170 in Linking Stimuli to Feedback—Effects of Stimulus Modality and Feedback Delay. *Psychophysiology*, *62*(4), e70050. https://doi.org/10.1111/psyp.70050

Ryu, V., Ha, R. Y., Lee, S. J., Ha, K., & Cho, H. S. (2017). Behavioral and electrophysiological alterations for reinforcement learning in manic and euthymic patients with bipolar disorder. *CNS neuroscience & therapeutics*, *23*(3), 248-256. https://doi.org/10.1111/cns.12671

Schultz, W. (2002). Getting formal with dopamine and reward. *Neuron*, *36*(2), 241-263. https://doi.org/10.1016/S0896-6273(02)00967-4

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, *275*(5306), 1593–1599. https://doi.org/10.1126/science.275.5306.1593

Schultz, W., & Dickinson, A. (2000). Neuronal coding of prediction errors. *Annual review of neuroscience*, *23*(1), 473-500. https://doi.org/10.1146/annurev.neuro.23.1.473

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of neurology, neurosurgery, and psychiatry*, *20*(1), 11-21. https://doi.org/10.1136%2Fjnnp.20.1.11

Sehrig, S., Weiss, A., Miller, G. A., & Rockstroh, B. (2019). Decision-and feedback-related brain potentials reveal risk processing mechanisms in patients with alcohol use disorder. *Psychophysiology*, *56*(12), e13450. https://doi.org/10.1111/psyp.13450

Shah, P. J., Ebmeier, K. P., Glabus, M. F., & Goodwin, G. M. (1998). Cortical grey matter reductions associated with treatment-resistant chronic unipolar depression: controlled magnetic resonance imaging study. *The British journal of psychiatry*, *172*(6), 527-532. https://doi.org/10.1192/bjp.172.6.527

Shear, M. K., Simon, N., Wall, M., Zisook, S., Neimeyer, R., Duan, N., ... & Keshaviah, A. (2011). Complicated grief and related bereavement issues for DSM‐5. *Depression and anxiety*, *28*(2), 103-117. https://doi.org/10.1002/da.20780

Shohamy, D., & Adcock, R. A. (2010). Dopamine and adaptive memory. *Trends in Cognitive Sciences*, *14*(10), 464–472. https://doi.org/10.1016/j.tics.2010.08.002

Silver, D., Singh, S., Precup, D., & Sutton, R. S. (2021). Reward is enough. *Artificial Intelligence*, *299*, 103535. https://doi.org/10.1016/j.artint.2021.103535

Suhr, F. (8. Juli, 2020). Die häufigsten psychischen Erkrankungen [Digital Picture]. Retrieved January 31, 2024, from https://de.statista.com/infografik/22218/haeufigste-psychische-erkrankungen-in-deutschland/

Takács, Á., Kóbor, A., Janacsek, K., Honbolygó, F., Csépe, V., & Németh, D. (2015). High trait anxiety is associated with attenuated feedback-related negativity in risky decision making. *Neuroscience letters*, *600*, 188-192. https://doi.org/10.1016/j.neulet.2015.06.022

Takamura, M., Okamoto, Y., Okada, G., Toki, S., Yamamoto, T., Ichikawa, N., Mori, A., Minagawa, H., Takaishi, Y., Fujii, Y., Kaichi, Y., Akiyama, Y., Awai, K., & Yamawaki, S. (2017). Patients with major depressive disorder exhibit reduced reward size coding in the striatum. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *79*(Pt B), 317–323. https://doi.org/10.1016/j.pnpbp.2017.07.006

The MathWorks, Inc. (2021). MATLAB R2021a [Computer software]. Natick, MA: The MathWorks, Inc.

The R Foundation. (2021). R: A language and environment for statistical computing (Version 4.2.2) [Computer software]. Vienna, Austria: R Foundation for Statistical Computing. https://www.R-project.org/

Thompson, S. M. (2023). Plasticity of synapses and reward circuit function in the genesis and treatment of depression. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *48*(1), 90–103. https://doi.org/10.1038/s41386-022-01422-1

Tobias, M. R., & Ito, T. A. (2021). Anxiety increases sensitivity to errors and negative feedback over time. *Biological psychology*, *162*, 108092. https://doi.org/10.1016/j.biopsycho.2021.108092

Tsetsenis, T., Broussard, J. I., & Dani, J. A. (2023). Dopaminergic regulation of hippocampal

plasticity, learning, and memory. *Frontiers in behavioral neuroscience*, *16*, 1092420. https://doi.org/10.3389/fnbeh.2022.1092420

Ullsperger, M. (2024). Beyond peaks and troughs: Multiplexed performance monitoring signals in the EEG. *Psychophysiology*, *61*(7), e14553. https://doi.org/10.1111/psyp.14553

Voeten, C. Using 'buildmer' to automatically find & compare maximal (mixed) models (2020).

von Glischinski, M., von Brachel, R., & Hirschfeld, G. (2019). How depressed is "depressed"? A systematic review and diagnostic meta-analysis of optimal cut points for the Beck Depression Inventory revised (BDI-II). *Quality of Life Research*, *28*, 1111-1118. https://doi.org/10.1007/s11136-018-2050-x

Weber, C., & Bellebaum, C. (2024). Prediction-error-dependent processing of immediate and delayed positive feedback. *Scientific Reports*, *14*(1), 9674. https://doi.org/10.1038/s41598-024-60328-8

Weinberg, A., Liu, H., Hajcak, G., & Shankman, S. A. (2015). Blunted neural response to rewards as a vulnerability factor for depression: Results from a family study. *Journal of Abnormal Psychology*, *124*(4), 878–889. https://doi.org/10.1037/abn0000081

Weinberg, A., Luhmann, C. C., Bress, J. N., & Hajcak, G. (2012). Better late than never? The effect of feedback delay on ERP indices of reward processing. *Cognitive, Affective, & Behavioral Neuroscience*, *12*(4), 671–677. https://doi.org/10.3758/s13415-012-0104-z

Weismüller, B., & Bellebaum, C. (2016). Expectancy affects the feedback-related negativity (FRN) for delayed feedback in probabilistic learning. *Psychophysiology*, *53*(11), 1739–1750. https://doi.org/10.1111/psyp.12738

World Health Organization. (2019). *International classification of diseases 11th revision (ICD-11)*. World Health Organization. https://icd.who.int/

Zimmerman, M., Chelminski, I., & McDermut, W. (2002). Major depressive disorder and axis I diagnostic comorbidity. *Journal of Clinical Psychiatry*, *63*(3), 187-193.

Zisook, S., & Shear, K. (2009). Grief and bereavement: what psychiatrists need to know. *World psychiatry*, *8*(2), 67-74. https://doi.org/10.1002/j.2051-5545.2009.tb00217.x

Supporting Information

Links between Altered Feedback Learning and Symptoms of Depression: Insights from an EEG Study on FRN and N170

Madita Röhlinger & Christian Bellebaum

Table S1

Maximal GLME models for the analysis of behavioral data

Model	Formula
Current depressive symptoms	*Accuracy ~ 1 + Block + BDI + Block:BDI + Timing + BDI:Timing + Block:Timing + Block:BDI:Timing + (1 + Block + Timing + Timing:Block | Participant)*
Past depressive episodes	*Accuracy ~ 1 + Block + PHQ + Block:PHQ + Timing + Block:Timing + PHQ:Timing + Block:PHQ:Timing + (1 + Block + Timing + Timing:Block | Participant)*
Familial vulnerability	*Accuracy ~ 1 + Block + Vulnerability + Timing + Block:Timing + Vulnerability:Timing + Block:Vulnerability + Block:Vulnerability:Timing +(1 + Block + Timing + Timing:Block | Participant)*

Note. GLME = generalized linear mixed effects. BDI = BDI-II (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), PHQ = modified PHQ-9 (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), Vulnerability = Familial Vulnerability (first-degree relatives without a history of depression [−0.5] vs. first-degree relatives with a depression diagnosis [0.5]), Timing = Feedback Timing (immediate [-0.5] vs. delayed [0.5]).

Table S2

Maximal LME models for the N170 analysis

Model	Formula
Current depressive symptoms (BDI-II)	*N170 Amplitude ~ 1 + Timing + Valence + Timing:Valence + BDI + Timing:BDI + Valence:BDI + Timing:Valence:BDI + PE + Timing:PE + Valence:PE + Timing:Valence:PE + BDI:PE + Timing:BDI:PE + Valence:BDI:PE + Timing:Valence:BDI:PE + Electrode + Timing:Electrode + Valence:Electrode + Timing:Valence:Electrode + BDI:Electrode + Timing:BDI:Electrode + Valence:BDI:Electrode + Timing:Valence:BDI:Electrode + PE:Electrode + Timing:PE:Electrode + Valence:PE:Electrode + Timing:Valence:PE:Electrode + BDI:PE:Electrode + Timing:BDI:PE:Electrode + Valence:BDI:PE:Electrode + Timing:Valence:BDI:PE:Electrode + (1 + Electrode + Timing + Valence + Valence:Electrode + Timing:Electrode + PE | Participant)*
Past depressive episodes (modified PHQ)	*N170 amplitude ~ 1 + Timing + Valence + Timing:Valence + PHQ + Timing:PHQ + Valence:PHQ + Timing:Valence:PHQ + PE + Timing:PE + Valence:PE + Timing:Valence:PE + PHQ:PE + Timing:PHQ:PE + Valence:PHQ:PE + Timing:Valence:PHQ:PE + Electrode + Timing:Electrode + Valence:Electrode + Timing:Valence:Electrode + PHQ:Electrode + Timing:PHQ:Electrode + Valence:PHQ:Electrode + Timing:Valence:PHQ:Electrode + PE:Electrode + Timing:PE:Electrode + Valence:PE:Electrode + Timing:Valence:PE:Electrode + PHQ:PE:Electrode + Timing:PHQ:PE:Electrode + Valence:PHQ:PE:Electrode + Timing:Valence:PHQ:PE:Electrode + (1 + Electrode + Timing + Valence + Valence:Electrode + Timing:Electrode + PE | Participant)*
Familial vulnerability	*N170 amplitude ~ 1 + Timing + Valence + Timing:Valence + Vulnerability + Timing:Vulnerability + Valence:Vulnerability + Timing:Valence:Vulnerability + PE + Timing:PE + Valence:PE + Timing:Valence:PE + Vulnerability:PE + Timing:Vulnerability:PE + Valence:Vulnerability:PE + Timing:Valence:Vulnerability:PE + Electrode + Timing:Electrode + Valence:Electrode + Timing:Valence:Electrode + Vulnerability:Electrode + Timing:Vulnerability:Electrode + Valence:Vulnerability:Electrode + Timing:Valence:Vulnerability:Electrode + PE:Electrode + Timing:PE:Electrode + Valence:PE:Electrode + Timing:Valence:PE:Electrode + Vulnerability:PE:Electrode + Timing:Vulnerability:PE:Electrode + Valence:Vulnerability:PE:Electrode + Timing:Valence:Vulnerability:PE:Electrode + (1 + Electrode + Timing + Valence + Valence:Electrode + Timing:Electrode + PE + Valence:PE | Participant)*

Note. LME = linear mixed effects. BDI = BDI-II (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), PHQ = modified PHQ-9 (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), Vulnerability = Familial Vulnerability (first-degree relatives without a history of depression [−0.5] vs. first-degree relatives with a depression diagnosis [0.5]), Timing = Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Valence = Feedback Valence (negative [-0.5] vs. positive [0.5]), PE = unsigned PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean), Electrode (P7 [-0.5] vs. P8 [0.5]).

Table S3

Maximal LME models for the FRN analysis

Model	Formula
Current depressive symptoms (BDI-II)	*FRN Amplitdue ~ 1 + Timing + Valence + Timing:Valence + BDI + Timing:BDI + Valence:BDI + Timing:Valence:BDI + PE + Timing:PE + Valence:PE + Timing:Valence:PE + BDI:PE + Timing:BDI:PE + Valence:BDI:PE + Timing:Valence:BDI:PE + (1 + Timing + Valence + PE + Valence:PE | Participant)*
Past depressive episodes (PHQ)	*FRN Amplitdue ~ 1 + Timing + Valence + Timing:Valence + PHQ + Timing:PHQ + Valence:PHQ + Timing:Valence:PHQ + PE + Timing:PE + Valence:PE + Timing:Valence:PE + PHQ:PE + Timing:PHQ:PE + Valence:PHQ:PE + Timing:Valence:PHQ:PE + (1 + Timing + Valence + PE + Valence:PE | Participant)*
Familial vulnerability	*FRN amplitude ~ 1 + Timing + Valence + Timing:Valence + Vulnerability + Timing:Vulnerability + Valence:Vulnerability + Timing:Valence:Vulnerability + PE + Timing:PE + Valence:PE + Timing:Valence:PE + Vulnerability:PE + Timing:Vulnerability:PE + Valence:Vulnerability:PE + Timing:Valence:Vulnerability:PE + (1 + Timing + Valence + PE | Participant)*

Note. LME = linear mixed effects. BDI = BDI-II (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), PHQ = modified PHQ-9 (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), Vulnerability = Familial Vulnerability (first-degree relatives without a history of depression [−0.5] vs. first-degree relatives with a depression diagnosis [0.5]), Timing = Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Valence = Feedback Valence (negative [-0.5] vs. positive [0.5]), PE = unsigned PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean).

Table S4

Results for the GLME analyses on accuracy

Model	Effect	β-estimate	*SE*	*z*	*p*	
Current depressive symptoms (BDI)	Block	1.42	0.23	6.04	<.001	***
	BDI	-0.04	0.02	-2.55	.011	*
	Timing	0.11	0.13	0.81	.419	
	Block:BDI	-0.04	0.02	-1.62	.106	
	BDI-II:Timing	-0.02	0.01	-1.41	.160	
	Block:Timing	-0.34	0.32	-1.08	.280	
	Block:BDI:Timing	0.01	0.03	0.16	.870	
Past depressive episodes (PHQ)	Block	1.40	0.24	5.97	<.001	***
	PHQ	-0.05	0.03	-1.79	.073	
	Timing	0.10	0.13	0.72	.474	
	Block:PHQ	-0.02	0.04	-0.54	.593	
	Block:Timing	-0.37	0.32	-1.18	.239	
	PHQ:Timing	0.00	0.02	-0.01	.989	
	Block:PHQ:Timing	0.09	0.05	1.65	.099	
Familial vulnerability	Block	1.24	0.33	3.76	<.001	***
	Vulnerability	-0.48	0.45	-1.07	.285	
	Timing	-0.09	0.16	-0.56	.573	
	Block:Timing	-0.46	0.43	-1.05	.294	
	Vulnerability:Timing	-0.23	0.31	-0.73	.467	
	Block:Vulnerability	-0.55	0.65	-0.84	.399	
	Block:Vulnerability:Timing	0.48	0.85	0.57	.569	

Note. $n = 45$. GLME = generalized linear mixed effects, *SE* = standard error, Timing = Feedback Timing, BDI = BDI-II, PHQ = modified PHQ-9, Vulnerability = Familial Vulnerability. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors BDI (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), PHQ (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), Familial Vulnerability (first-degree relatives without a history of depression [−0.5] vs. first-degree relatives with a depression diagnosis [0.5]), Block (1 [-0.5], 2 [-0.167], 3 [0.167], 4 [0.5]), and Feedback Timing (immediate [-0.5] vs. delayed [0.5]).
***$p < .001$, *$p < .050$

Table S5

Results for the LME analysis on the N170 amplitude including BDI

Effect	β-estimate	*SE*	*df*	*t*	*p*	
Timing	0.47	0.36	49.05	1.31	.198	
Valence	0.47	0.25	38.14	1.91	.063	
BDI	0.03	0.06	43.34	0.46	.649	
PE	-0.45	0.26	15.42	-1.74	.102	
Electrode	-2.49	0.90	43.95	-2.75	.009	**
Timing:Valence	0.28	0.26	23157.78	1.06	0.291	
Timing:BDI	0.03	0.04	46.99	0.65	.518	
Valence:BDI	0.01	0.03	35.96	0.48	.632	
Timing:PE	0.09	0.42	24156.31	0.21	.832	
Valence:PE	-2.80	0.48	22604.02	-5.85	<.001	***
BDI:PE	0.06	0.03	16.94	1.89	.077	
Timing:Electrode	-0.02	0.40	66.47	-0.06	.954	
Valence:Electrode	2.16	0.40	64.66	5.46	<.001	***
BDI:Electrode	-0.09	0.10	43.64	-0.92	.363	
PE:Electrode	-0.26	0.45	3464.48	-0.58	.565	
Timing:Valence:BDI	0.02	0.03	26194.06	0.83	.404	
Timing:Valence:PE	-0.15	0.94	17003.06	-0.16	.870	
Timing:BDI:PE	0.10	0.05	22050.20	1.98	.047	*
Valence:BDI:PE	0.09	0.05	24532.31	1.68	.093	
Timing:Valence:Electrode	0.03	0.52	27505.85	0.05	.957	
Timing:BDI:Electrode	0.00	0.04	59.16	-0.02	.981	
Valence:BDI:Electrode	0.04	0.04	57.95	0.90	.370	
Timing:PE:Electrode	0.12	0.84	26241.62	0.15	.884	
Valence:PE:Electrode	1.10	0.95	25900.45	1.16	.246	
BDI:PE:Electrode	0.05	0.05	4822.82	0.91	.364	
Timing:Valence:BDI:PE	-0.07	0.11	20110.07	-0.66	.507	
Timing:Valence:BDI:Electrode	-0.03	0.05	27695.23	-0.61	.539	
Timing:Valence:PE:Electrode	-2.18	1.82	4225.98	-1.20	.232	
Timing:BDI:PE:Electrode	-0.09	0.10	26779.66	-0.94	.345	
Valence:BDI:PE:Electrode	0.03	0.11	26754.40	0.28	.780	
Timing:Valence:BDI:PE:Electrode	-0.42	0.21	5828.78	-2.05	.041	*

Note. $n = 45$. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, BDI = BDI-II, PE = unsigned PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), BDI (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) and Electrode (P7 [-0.5] vs. P8 [0.5]).
*** $p < .001$, ** $p < .010$, * $p < .050$

Table S6

Simple slope analyses for the interaction between BDI, Feedback Timing and PE

BDI Scores	Feedback Timing	β-estimate	*SE*	*t*	*p*	
Low (= -1 *SD*)	Immediate	-0.82	0.50	-1.64	.648	
	Delayed	-1.58	0.48	-3.30	.012	*
Medium (= Mean)	Immediate	-0.68	0.35	-1.97	.330	
	Delayed	-0.60	0.33	-1.80	.474	
High (= +1 *SD*)	Immediate	-0.54	0.50	-1.07	>.999	
	Delayed	0.38	0.47	0.80	>.999	

Note. $n = 45$. PE = unsigned PE, BDI = BDI-II, *SD* = standard deviation, *SE* = standard error. Presented *p*-values were Bonferroni-corrected. The sign of the β-estimates indicates the direction of unsigned PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) effects on N170 amplitudes in the respective condition.

*$p < .050$

Table S7

Simple slope analyses for the interaction between Electrode, BDI, Feedback Timing, Feedback Valence and PE found in the LME analysis on the N170

Electrode	BDI	Timing	Valence	β-estimate	*SE*	*df*	*t*	*p*	
P7	High	Delayed	Positive	-0.15	0.91	641.56	-0.16	>.999	
			Negative	1.41	0.89	553.91	1.58	>.999	
		Immediate	Positive	-2.58	0.98	745.34	-2.64	.134	
			Negative	1.19	0.93	613.52	1.28	>.999	
	Low	Delayed	Positive	-3.36	0.92	437.12	-3.67	.004	**
			Negative	0.86	0.94	626.81	0.91	>.999	
		Immediate	Positive	-1.92	0.99	688.34	-1.94	.853	
			Negative	1.95	0.95	563.98	2.05	.650	
P8	High	Delayed	Positive	-1.16	0.91	612.82	-1.28	>.999	
			Negative	2.04	0.90	530.50	2.26	.384	
		Immediate	Positive	0.14	0.98	772.55	0.14	>.999	
			Negative	-0.48	0.94	575.24	-0.52	>.999	
	Low	Delayed	Positive	-2.73	0.92	418.39	-2.97	.051	
			Negative	-0.18	0.95	618.58	-0.19	>.999	
		Immediate	Positive	-3.09	1.00	736.17	-3.09	.034	*
			Negative	0.80	0.96	508.64	0.83	>.999	

Note. $n = 45$. PE = unsigned PE, BDI = BDI-II, Timing = Feedback Timing, Valence = Feedback Valence, *SE* = standard error, *df* = degrees of freedom. Presented *p*-values were Bonferroni-corrected. The sign of the β-estimates indicates the direction of unsigned PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) effects on N170 amplitudes in the respective condition.

**$p < .010$, *$p < .050$

Table S8

Results for the LME analysis on the N170 amplitude including PHQ

Effect	β-estimate	*SE*	*df*	*t*	*p*	
Timing	0.48	0.37	48.73	1.31	.197	
Valence	0.53	0.24	39.25	2.19	.034	*
PHQ	0.07	0.11	43.52	0.66	.516	
PE	-0.27	0.28	22.56	-0.96	.348	
Electrode	-2.50	0.92	43.88	-2.74	.009	**
Timing:Valence	0.33	0.26	24621.51	1.26	.207	
Timing:PHQ	0.01	0.07	49.02	0.14	.887	
Valence:PHQ	-0.06	0.04	40.42	-1.44	.157	
Timing:PE	0.06	0.42	24861.91	0.14	.891	
Valence:PE	-2.73	0.48	25107.91	-5.70	<.001	***
PHQ:PE	0.02	0.05	19.29	0.50	.623	
Timing:Electrode	-0.07	0.40	65.51	-0.18	.861	
Valence:Electrode	2.18	0.39	64.67	5.56	<.001	***
PHQ:Electrode	0.06	0.16	43.91	0.35	.730	
PE:Electrode	-0.23	0.45	3181.98	-0.51	.611	
Timing:Valence:PHQ	-0.02	0.05	24719.79	-0.37	.714	
Timing:Valence:PE	-0.09	0.95	19006.54	-0.09	.926	
Timing:PHQ:PE	-0.01	0.07	26354.21	-0.07	.942	
Valence:PHQ:PE	0.15	0.08	22986.82	1.93	.054	
Timing:Valence:Electrode	0.00	0.52	27523.97	0.01	.993	
Timing:PHQ:Electrode	0.02	0.07	67.95	0.25	.801	
Valence:PHQ:Electrode	0.09	0.07	67.25	1.25	.216	
Timing:PE:Electrode	0.18	0.84	26131.73	0.22	.830	
Valence:PE:Electrode	1.07	0.95	25862.22	1.13	.258	
PHQ:PE:Electrode	0.07	0.07	5801.38	0.90	.368	
Timing:Valence:PHQ:PE	0.03	0.15	22968.14	0.21	.834	
Timing:Valence:PHQ:Electrode	-0.01	0.09	27680.67	-0.08	.934	
Timing:Valence:PE:Electrode	-2.16	1.82	4002.55	-1.19	.234	
Timing:PHQ:PE:Electrode	0.01	0.14	27049.30	0.05	.957	
Valence:PHQ:PE:Electrode	0.24	0.15	27105.95	1.59	.113	
Timing:Valence:PHQ:PE:Electrode	-0.13	0.30	7134.45	-0.43	.670	

Note. $n = 45$. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, PHQ = modified PHQ-9, PE = unsigned PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), PHQ (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) and Electrode (P7 [-0.5] vs. P8 [0.5]).
*** $p < .001$, ** $p < .010$, * $p < .050$

N170 Analysis Including PHQ

The LME analysis on the N170 amplitude including PHQ as a predictor alongside Feedback Timing, Feedback Valence, PE and Electrode replicated all of the effects described for the model containing the BDI in the main text. Again, there was a significant effect of electrode ($p = .009$) with more pronounced amplitudes over P8, as well as a significant two-way interaction between Feedback Valence and Electrode ($p < .001$). As for the BDI model, we resolved the interaction via simple slope analyses and found no significant effect of Feedback Valence for P7 ($\beta = -0.56$, $SE = 0.29$, $t = -1.93$, $p = .118$), but for P8 negative feedback led to significantly larger N170 amplitudes than positive feedback ($\beta = 1.62$, $SE = 0.33$, $t = 4.90$, $p < .001$). In addition, the analysis also replicated the significant interaction between PE and Feedback Valence ($p < .001$), with amplitudes increasing for more unexpected positive feedback ($\beta = -1.64$, $SE = 0.38$, $t = -4.36$, $p < .001$) and decreasing for more unexpected negative feedback ($\beta = 1.09$, $SE = 0.37$, $t = 2.97$, $p = .008$). In addition, the model revealed a significant effect of Feedback Valence ($p = .034$), with more pronounced amplitudes for negative compared to positive feedback. All other effects were not significant (all $ps \geq .054$, see Table S8 above for β-estimates and effect-specific *t*-tests).

Table S9

Results for the LME analysis on the N170 amplitude including Familial Vulnerability

Effect	β-estimate	*SE*	*df*	*t*	*p*	
Timing	0.43	0.53	37.68	0.82	.415	
Valence	0.10	0.31	30.62	0.34	.739	
Vulnerability	1.61	1.68	34.89	0.96	.343	
PE	-0.82	0.40	20.55	-2.08	.050	
Electrode	-2.64	1.31	35.45	-2.02	.051	
Timing:Valence	0.18	0.34	21408.96	0.54	.589	
Timing:Vulnerability	-0.45	1.05	37.68	-0.43	.669	
Valence:Vulnerability	-1.67	0.62	30.62	-2.71	.011	*
Timing:PE	-0.14	0.53	21449.25	-0.26	.799	
Valence:PE	-2.69	1.00	17.62	-2.70	.015	*
Vulnerability:PE	-1.56	0.79	20.55	-1.96	.064	
Timing:Electrode	0.68	0.49	49.16	1.38	.173	
Valence:Electrode	2.52	0.52	47.41	4.84	<.001	***
Vulnerability:Electrode	-0.11	2.62	35.45	-0.04	.967	
PE:Electrode	-0.04	0.56	4260.73	-0.08	.938	
Timing:Valence:Vulnerability	0.38	0.67	21408.96	0.57	.572	
Timing:Valence:PE	-0.24	1.18	16148.09	-0.20	.840	
Timing:Vulnerability:PE	-0.39	1.07	21449.25	-0.36	.716	
Valence:Vulnerability:PE	1.17	2.00	17.62	0.59	.564	
Timing:Valence:Electrode	-0.04	0.67	22686.77	-0.06	.954	
Timing:Vulnerability:Electrode	1.82	0.99	49.16	1.84	.072	
Valence:Vulnerability:Electrode	-0.39	1.04	47.41	-0.37	.711	
Timing:PE:Electrode	0.62	1.06	21846.76	0.58	.560	
Valence:PE:Electrode	0.99	1.18	21180.96	0.84	.400	
Vulnerability:PE:Electrode	-0.12	1.13	4260.73	-0.11	.912	
Timing:Valence:Vulnerability:PE	-1.60	2.36	16148.10	-0.68	.498	
Timing:Valence:Vulnerability:Electrode	0.67	1.34	22686.77	0.50	.616	
Timing:Valence:PE:Electrode	-1.55	2.25	3450.50	-0.69	.492	
Timing:Vulnerability:PE:Electrode	2.54	2.13	21846.76	1.20	.232	
Valence:Vulnerability:PE:Electrode	0.92	2.36	21180.96	0.39	.696	
Timing:Valence:Vulnerability:PE:Electrode	1.64	4.50	3450.50	0.36	.716	

Note. $n = 37$. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, Vulnerability = Familial Vulnerability, PE = unsigned PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), Vulnerability (first-degree relatives without a history of depression [−0.5] vs. first-degree relatives with a depression diagnosis [0.5]), PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean) and Electrode (P7 [-0.5] vs. P8 [0.5]).
*** $p < .001$, * $p < .050$

N170 Analysis Including Familial Vulnerability

The LME analysis on the N170 amplitude including Familial Vulnerability as a predictor alongside Feedback Timing, Feedback Valence, PE and Electrode replicated the significant interaction between Feedback Valence and Electrode ($p < .001$) that was also described for the analysis involving the BDI reported in the main text. Simple slope analyses revealed significantly larger amplitudes following negative compared to positive feedback for P8 ($\beta = 1.88$, $SE = 0.34$, $t = 5.54$, $p < .001$), but not for P7 ($\beta = -0.75$, $SE = 0.33$, $t = -2.26$, $p = .060$). The analysis also replicated the significant interaction between PE and Feedback Valence ($p = .015$), however, this time amplitudes significantly increased for more unexpected positive feedback ($\beta = -1.90$, $SE = 0.56$, $t = -3.36$, $p = .004$), but did not significantly decrease for more unexpected negative feedback ($\beta = 1.13$, $SE = 0.52$, $t = 2.18$, $p = .086$). All other effects (apart from the interaction between Feedback Valence and Familial Vulnerability reported in the main text) did not reach significance (all ps $\geq .050$; see Table S9 above for β-estimates and effect-specific *t*-tests).

Table S10

Results for the LME analysis on the FRN amplitude including BDI

Effect	β-estimate	*SE*	*df*	*t*	*p*	
Timing	0.87	0.29	54.59	3.02	.004	**
Valence	2.22	0.23	41.42	9.74	<.001	***
BDI	-0.07	0.05	42.15	-1.29	.205	
PE	0.89	0.31	25.44	2.90	.008	**
Timing:Valence	-0.74	0.28	13417.45	-2.65	.008	**
Timing:BDI	-0.06	0.03	50.64	-1.89	.065	
Valence:BDI	0.00	0.02	38.37	0.10	.920	
Timing:PE	-0.28	0.45	13147.85	-0.63	.528	
Valence:PE	5.73	0.88	26.47	6.54	<.001	***
BDI:PE	0.02	0.03	27.25	0.47	.643	
Timing:Valence:BDI	-0.01	0.03	13808.02	-0.32	.749	
Timing:Valence:PE	0.15	0.99	7036.57	0.15	.882	
Timing:BDI:PE	0.01	0.05	12738.57	0.16	.871	
Valence:BDI:PE	-0.10	0.10	26.87	-1.07	.293	
Timing:Valence:BDI:PE	-0.12	0.11	8801.38	-1.04	.300	

Note. $n = 45$. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, BDI = BDI-II, PE = unsigned PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), BDI (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean) and PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean).
*** $p < .001$, ** $p < .010$

Table S11

Results for the LME analysis on the FRN amplitude including PHQ

	β-estimate	*SE*	*df*	*t*	*P*	
Timing	0.85	0.29	53.93	2.91	.005	**
Valence	2.22	0.23	40.65	9.83	<.001	***
PHQ	-0.08	0.09	42.67	-0.90	.374	
PE	0.89	0.31	25.17	2.88	.008	**
Timing:Valence	-0.73	0.28	13469.55	-2.63	.009	**
Timing:PHQ	-0.06	0.05	54.60	-1.11	.271	
Valence:PHQ	-0.02	0.04	42.34	-0.58	.562	
Timing:PE	-0.30	0.45	13144.80	-0.67	.504	
Valence:PE	5.73	0.89	28.96	6.42	<.001	***
PHQ:PE	-0.01	0.05	21.86	-0.10	.924	
Timing:Valence:PHQ	-0.03	0.05	13466.57	-0.67	.503	
Timing:Valence:PE	0.04	0.99	7177.33	0.04	.971	
Timing:PHQ:PE	-0.03	0.07	13597.04	-0.34	.737	
Valence:PHQ:PE	-0.12	0.15	26.12	-0.81	.425	
Timing:Valence:PHQ:PE	-0.03	0.16	9540.55	-0.16	.873	

Note. $n = 45$. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, PHQ = modified PHQ-9, PE = unsigned PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), PHQ (mean centered, yielding negative values for scores below the mean vs. positive values for scores above the mean), and PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean).
*** $p < .001$, ** $p < .010$, * $p < .050$

FRN Analysis Including PHQ

The LME analysis on the FRN amplitude including PHQ as a predictor alongside Feedback Timing, Feedback Valence and PE replicated all of the effects described for the model containing the BDI in the main text. The analysis replicated the significant effect of Feedback Valence ($p < .001$), with more negative amplitudes following negative compared to positive feedback. In addition, the significant effect of Feedback Timing ($p = .005$), indicating more negative amplitudes following immediate compared to delayed feedback, was replicated. Again, a significant interaction between Feedback Valence and Feedback Timing ($p = .009$) explained these effects further: Negative feedback was associated with more negative amplitudes for both immediate ($\beta = 2.59$, $SE = 0.26$, $t = 9.90$, $p < .001$) and delayed feedback ($\beta = 1.86$, $SE = 0.27$, $t = 6.88$, $p < .001$), but the effect was stronger for immediate feedback. Furthermore, we could replicate a significant effect of PE ($p = .008$) that was again further explained by a significant interaction between PE and Feedback Valence ($p < .001$), which we thus resolved. There was a significant effect of PE on the FRN amplitude for negative feedback, with more negative amplitudes for more unexpected feedback ($\beta = -1.98$, $SE = 0.51$, $t = -3.91$, $p = .002$). For positive feedback, this effect was reversed with more positive amplitudes for more unexpected feedback ($\beta = 3.75$, $SE = 0.58$, $t = 6.51$, $p < .001$). All other effects (including effects involving the PHQ) were not significant (all $ps \geq .271$; see Table S11 above for β-estimates and effect-specific *t*-tests).

Table S12

Results for the LME analysis on the FRN amplitude including Familial Vulnerability

Effect	β-estimate	*SE*	*df*	*t*	*p*	
Timing	1.19	0.41	40.75	2.87	.007	**
Valence	2.36	0.29	29.38	8.24	<.001	***
Vulnerability	2.47	1.25	35.59	1.97	.056	
PE	0.80	0.38	15.57	2.13	.050	*
Timing:Valence	-1.00	0.36	11014.83	-2.77	.006	**
Timing:Vulnerability	1.31	0.83	40.75	1.58	.122	
Valence:Vulnerability	-0.26	0.57	29.38	-0.46	.647	
Timing:PE	-0.37	0.57	11135.99	-0.65	.518	
Valence:PE	4.60	0.64	11259.45	7.22	<.001	***
Vulnerability:PE	-1.27	0.76	15.57	-1.69	.111	
Timing:Valence:Vulnerability	-0.92	0.72	11014.83	-1.28	.202	
Timing:Valence:PE	0.58	1.26	8485.11	0.46	.645	
Timing:Vulnerability:PE	0.20	1.15	11135.99	0.18	.859	
Valence:Vulnerability:PE	0.84	1.27	11259.45	0.66	.509	
Timing:Valence:Vulnerability:PE	1.72	2.51	8485.11	0.69	.492	

Note. $n = 37$. LME = linear mixed effects, *SE* = standard error, *df* = degrees of freedom, Timing = Feedback Timing, Valence = Feedback Valence, Vulnerability = Familial Vulnerability, PE = unsigned PE. The sign of the β-estimates indicates the direction of main effects for the fixed-effects predictors Feedback Timing (immediate [-0.5] vs. delayed [0.5]), Feedback Valence (negative [-0.5] vs. positive [0.5]), Vulnerability (first-degree relatives without a history of depression [−0.5] vs. first-degree relatives with a depression diagnosis [0.5]), and PE (scaled and mean centered, yielding negative values for PE values below the mean vs. positive values for PE values above the mean).
*** $p < .001$, * $p < .050$

FRN Analysis Including Familial Vulnerability

The LME analysis on the FRN amplitude including Familial Vulnerability as a predictor alongside Feedback Timing, Feedback Valence and PE replicated all of the effects described for the model containing the BDI in the main text. The analysis replicated the significant effect of Feedback Valence ($p < .001$), with more negative amplitudes following negative compared to positive feedback. In addition, the significant effect of Feedback Timing ($p = .007$), indicating more negative amplitudes following immediate compared to delayed feedback, was replicated. Again, a significant interaction between Feedback Valence and Feedback Timing ($p = .006$) explained these effects further: Negative feedback was associated with more negative amplitudes for both immediate ($\beta = 2.82$, $SE = 0.28$, $t = 10.03$, $p < .001$) and delayed feedback ($\beta = 2.08$, $SE = 0.29$, $t = 7.23$, $p < .001$), but the effect was stronger for immediate feedback. The effect of PE was at the threshold of significance ($p = .050$), but we were able to replicate the significant interaction between PE and Feedback Valence ($p < .001$), which we thus resolved. There was a significant effect of PE on the FRN amplitude for negative feedback, with more negative amplitudes for more unexpected feedback ($\beta = -1.01$, $SE = 0.43$, $t = -2.37$, $p = .044$). For positive feedback, this effect was reversed with more positive amplitudes for more unexpected feedback ($\beta = 3.35$, $SE = 0.43$, $t = 7.72$, $p < .001$). All other effects (including effects involving Familial Vulnerability) were not significant (all $ps \geq .056$; see Table S12 above for β-estimates and effect-specific t-tests).