The Impact of Motivational and Affective Context on Error-Induced Learning

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Kerstin Unger

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Der Dekan:

Prof. Dr. Roland Brünken

Berichterstatter:

Prof. Dr. J. Kray, Universität des Saarlandes

Prof. Dr. Axel Mecklinger, Universität des Saarlandes

Prof. Dr. Gesine Dreisbach, Universität Regensburg

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Table of Content

List of Tables

List of Figures

I Theoretical Part

1. Introduction

One of the most remarkable achievements of evolution is the human's ability to learn:

Learning is an enduring change in the mechanisms of behaviour involving specific stimuli and/or responses that results from prior experience with those or similar stimuli and responses. (Domjan, 2003, p. 14).

As becomes evident from this definition, learning allows an organism to flexibly adapt to changing environmental conditions. In doing so, an individual has to continuously monitor their performance to detect discrepancies between intended and actual responses (i.e., errors) and to adjust behaviour accordingly. Experience- (or feedback-) based learning thus requires the ability to evaluate the outcomes of one's behaviour. The motivational and affective significance of action outcomes, however, can vary considerably. For instance, some errors place the individual in serious danger or threaten a person's self-worth whilst others have virtually no consequences. In order to meet specific situational demands, an efficient performance monitoring system should take into account the affective and motivational context of an action. Nevertheless, the question of how motivational and affective processes interact with performance monitoring has received surprisingly little attention thus far (Pessoa, 2008, 2009). Evidence from electrophysiological research suggests that the motivational and affective value of on-going events has a substantial impact on error- and feedback processing (Falkenstein, Hoormann, Christ & Hohnsbein, 2000; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Hajcak, Moser, Yeung, & Simons, 2005; Olvet & Hajcak, 2011; Wiswede, Münte, Goschke, & Rüsseler, 2009a; Wiswede, Münte, & Rüsseler, 2009b). However, it remains largely unknown from the existing literature how these context-specific modulations in error- and feedback processing relate to flexible behavioural adaptation. The present thesis addressed this essential question by utilizing the high temporal resolution of event-related potentials (ERPs) to track the impact of motivational and affective manipulations on the neural mechanisms of error and feedback processing during reinforcement learning.

This thesis compromises a theoretical and an empirical part. The theoretical part first gives an overview of contemporary accounts on reinforcement learning and its neural underpinnings. This overview is followed by a chapter highlighting the role of emotional and motivational processes in reinforcement learning and action selection. Afterwards, I will review empirical evidence indicating how theoretical approaches to performance monitoring and learning are informed by electrophysiological research. I conclude with a summary of the general aims of this thesis and an outline of the three experiments it includes. The empirical part starts with a formulation of the specific research goals of Experiments 1 and 2, followed by the deduction of the research hypotheses and a description of study design and methods. The results will be presented consecutively for Experiment 1 and 2. After an interim discussion of the combined findings of Experiment 1 und 2, I will give an outline of Experiment 3, including the corresponding research goals and hypotheses. The description of design and methods is followed by a presentation of the results and a discussion of the findings of Experiment 3. The empirical part closes with a general discussion of the main findings from the three experiments in the context of the relevant literature.

2. Review of Literature

Overview

The literature review is structured into five main parts: In the first part, I will introduce basic theoretical concepts and theories of reinforcement learning. The second part addresses the neural underpinnings of reinforcement learning, particularly the role that has been attributed to dopaminergic mechanisms in coding a learning signal that is referred to as "reward prediction error". In the third section, I will summarize theoretical considerations and empirical findings emphasising the significance of motivational and affective processes in learning and adaptive decision making. Part four describes components in the event-related potential (ERP) that are thought to reflect the activity of a generic performance monitoring system: the error negativity (Ne), the feedback-related negativity (FRN), and the error positivity (Pe). As will be outlined in the fifth section, integrative neurocomputational models on performance monitoring conceptualize the Ne and the FRN in terms of learning or conflict signals mediating goal-directed behavioural adjustments.

Theoretical Accounts on Reinforcement Learning

Towards a Definition of Reinforcement Learning

Imagine a child coming home from kindergarten and showing their mother a picture painted by themselves. The mother will most likely praise the child and this makes them feel proud and happy. A few days later, however, the child is painting the wall of the living room with crayons while the mother is preparing dinner. Probably to the child's surprise, the mother's reaction to their work will now be less pleasant. Obviously, the consequences of the child's action are more or less desirable, depending upon the specific situation in which the behaviour occurs. Thus, in order to satisfy their needs and desires as well as to avoid harm and punishment, an individual has to learn which action to select in a given situation. This fundamental process is commonly referred to as reinforcement learning (RL).

The foundations of modern theories on RL were laid by the pioneering work of E. L. Thorndike. On the basis of his laboratory studies on "animal intelligence", Thorndike

formulated the “Law of Effect” (Thorndike, 1911). According to this “law”, the association between a situation and a certain behaviour is strengthened if the behaviour is followed by a satisfying state. By comparison, if the behaviour is followed by discomfort, the association between situation and behaviour is weakened. Although Thorndike’s assertion that behaviour is controlled by the association between situation and response has been challenged by later studies, the Law of Effect highlights two key characteristics of RL (Sutton & Barto, 1998). First, it states that RL involves the selection of a particular behaviour, that is, the subject has to discover rewarding responses by trial and error, enabling them to adapt to unknown situations. Second, the Law of Effect implies that RL is associative in nature. The associationistic view is still inherent to most neurophysiological models of RL. On the neural level, synaptic plasticity is widely assumed to be a substrate of associative learning (Stefan et al., 2000). In the following sections, I will provide essential information about the basic elements of RL and the different types of associations that can be learned. Then I will introduce the contemporary computational theories of RL this thesis relies on.

States, Actions, and Reinforcements

The associative structure of RL comprises relations between states, actions[1], and outcomes. In the RL framework, the term ‘state’ refers to the representation of the current situation (e.g., Sutton & Barto, 1998). It should be noted that the state potentially involves a broad range of relevant information that are not restricted to a specific set of sensory stimuli. Specifically, states may include motivational and emotional aspects of the situation. Given a particular state, an individual has to choose between several potential actions, each of which is usually followed by specific consequences and hence results in a new state. If the outcome of an action increases the future probability for the same action to be performed, it is called a *reinforcer*[2] (Skinner, 1953) or *reward*. Note that this definition includes both the occurrence of pleasant states (positive reinforcement) and the avoidance or termination of

[1] The term ‘action’ refers to intentional, goal-directed behaviours, whereas the term ‘response’ emphasizes that a certain behaviour is elicited by a stimulus.

[2] While early researchers thought of reinforcers as specific stimuli, the term has been expanded to responses that have been assigned a specific role in the adaptation to environmental constraints.

unpleasant states (negative reinforcement). Conversely, an outcome that decreases the future probability for the action to be performed is termed a *punisher* (Azrin & Holz, 1966), either denoting the occurrence of an unpleasant outcome (positive punishment) or the withdrawal of pleasant consequences (negative punishment).

Instrumental vs. Classical Conditioning

Learned associations have traditionally been divided into two basic categories: (1) associations between different stimuli or events and (2) associations between stimuli/events and behaviour. This distinction is closely related to the investigation of learning processes using the paradigms of classical vs. instrumental conditioning. In classical (or Pavlovian) conditioning two types of stimuli are distinguished based on the behavioural response they elicit on first presentation, that is, without prior learning (cf. Domjan, 2003). The unconditioned stimulus (US) effectively evokes a specific response called unconditioned response (UR). Hence, the association between US and UR is an innate reflex (Pavlov, 1927). The second class of stimuli is referred to as conditioned stimuli (CSs). In contrast to the US, the CS does not evoke a specific behavioural reaction on first presentation, but comes to do so after repeated pairing with the US. This reaction upon presentation of the previously “neutral” CS is called the conditioned response (CR). For conditioning to occur the CS has to be *predictive* of the US, that is, the CS has to be presented before the US or simultaneously with it (e.g., Bower & Hilgard, 1981). Crucially, learning about the relation between stimuli allows the subject to predict future events and to engage in anticipatory responses.

However, successful behavioural adaptation in a changing environment does not only require the ability to predict an event but also to control the results of one’s own behaviour (cf. Balleine, 2001). This latter form of behaviour is usually called goal-directed or instrumental. Instrumental conditioning includes three basic components: (1) a response, (2) a response outcome (or reinforcer), and (3) a relation between response and outcome, also termed response-reinforcer (or instrumental) contingency (cf. Domjan, 2003). The instrumental contingency denotes the extent to which a particular reinforcer is more likely to

occur if it is preceded by the instrumental response. In case of a perfect contingency, the reinforcer is not delivered unless the instrumental response is made. Thus, in contrast to UR and CR in classical conditioning, the instrumental response is controlled by its consequences (Grindley, 1932). Yet, Thorndike already emphasized the importance of the situation or stimulus context for determining which instrumental response should occur. As will be pointed out below, evidence has accumulated for the notion that stimulus context, instrumental response, and response outcome are connected in instrumental conditioning. In particular, the expectation of future reinforcements has been ascribed a pivotal role in motivating instrumental behaviour. Contemporary theories of RL thus rely on research from the field of both classical and instrumental conditioning.

Motivational Mechanisms in Instrumental Learning

Goal-Directed Actions vs. Habits

Early theories of learning were inspired by the study of animal behaviour and focused mainly on the establishment of stimulus-response (S–R) associations, thereby implying that a specific behaviour is directly elicited by a stimulus. This notion contrasts with the commonly held view that instrumental behaviour is controlled by its consequences. Indeed, evidence from outcome devaluation studies has strongly suggested that instrumental conditioning also results in the learning of response-outcome (R-O) associations (for a review, see Dickinson & Balleine, 1995). In these studies, the instrumental conditioning procedure is followed by a manipulation of the outcome value. For example, a food- and water-deprived rat is trained to press a lever to obtain food pellets and to pull a chain to obtain a sucrose solution (or vice versa). Subsequently, the rat gains direct access to either the food or the liquid and a taste aversion is conditioned by injecting the animal with a toxic substance. In the final test phase, the rat is exposed to the two response devices again, but pressing the button or pulling the chain does result in any outcome. Despite the fact that the outcome devaluation should not affect the previously learned S-R association, the rat produces fewer lever presses if the pellets have been associated with illness, but pulls the chain less often if the liquid has been associated with illness (Colwill & Rescorla, 1985). This finding clearly indicates that the

animal has learned to associate the action with its specific consequences. Similarly, instrumental behaviour is generally sensitive to degrading the response-outcome contingency and vanishes if it is no longer followed by the reinforcing outcome (extinction; e.g., Domjan, 2003). Even in case of unchanged reward probability, instrumental behaviour has been shown to become less frequent if the contingency between response and reinforcer is reduced (Hammond, 1980). Behaviour that is (a) guided by the knowledge of the relation to some outcome and (b) susceptible to alterations in the value of those outcomes is called *goal-directed* (cf. Balleine, Liljeholm, & Ostlund, 2009).

Devaluation studies have also shown that instrumental conditioning can result in the learning of S-R associations that are insensitive to changes in outcome value (Dickinson, 1985). These associations arise, for example, from extensive overlearning as behaviour becomes more and more *habitual* (Adams, 1981) and thus are commonly referred to as habits. In contrast to goal-directed actions, habits are relatively unaffected by changes in the response-outcome contingency (e.g., Dickinson, Squire, Varga, & Smith, 1998) and may persist even if followed by negative consequences, as illustrated by drug seeking behaviour (Nelson & Killcross, 2006; Schoenbaum & Setlow, 2005). Importantly, accumulating evidence indicates that habitual and goal-directed learning constitute distinct processes that are dissociable at both the functional and the neural level (Balleine et al., 2009; Redgrave et al., 2010; Yin & Knowlton, 2002). This distinction is critical for the current study because the learning task applied has been shown to recruit both the habitual and the goal-directed system (Doll et al., 2009; Huys & Dayan, 2009).

Expectancy of Outcomes: The Role of S-O Associations or Pavlovian-Instrumental Transfer

Given the principles of learning that have been inferred from classical conditioning, it is reasonable to assume that the associative structure in instrumental learning is not restricted to S-R and R-O associations, but also includes the development of stimulus-outcome (S-O) associations. Indeed, it has been suggested that an organism learns to predict a particular outcome during instrumental conditioning by means of an association between a situation and an outcome (Hull, 1930; Spence, 1956). The two-process theory

(Rescorla & Solomon, 1967) states that the predictive stimulus (cue) induces the instrumental response by eliciting an outcome-specific affective state such as hope for food or fear of pain. Alternatively, it has been suggested that an S-(O)-R association is formed (Trapold & Overier, 1972). According to this view, the expectancy of the outcome acts as an internal stimulus and hence becomes directly associated with the response. Research on Pavlovian-instrumental transfer has provided ample evidence for the notion that instrumental performance depends on S-O associations (Domjan, 1993). There is also evidence, however, that this may only be the case if the cue conveys reliable information about the upcoming outcome (e.g., Delamater, 1995). More recently, Berridge (2001) posited that the cue does not only trigger the process of "wanting" the outcome, but in itself acquires an incentive value as well. It has been shown that this kind of Pavlovian incentive value of the cue can be distinguished from a more complex cognitive expectation of the incentive value of the outcome, i.e., "the desire for the outcome" (Berridge, 2001).

Competition and Cooperation of Goal-directed and Habitual Control

From our every-day life we know that there are many instances in which the goal-directed action-outcome (A-O) system and the S-R habit system compete to gain control of behaviour. For example, if a deer suddenly jumps out in front of your car you have to suppress the habitual tendency to swerve. Instead, you should switch to a goal-directed mode of control that enables you to straighten the steering wheel, to brake firmly, and to take the foot off the brake as you impact. The relative dominance of habitual vs. goal-directed control has been found to depend on several conditions. As I pointed out above, habits are established as a result of overlearning. The stronger the S-R association grows, the more likely the stimulus becomes to guide response selection, particularly under time pressure and other stressful conditions (Schwabe, Wolf, & Oitzl, 2010). Furthermore, acute and chronic stress has been found to favour the habit learning system over the goal-directed learning system (Dias-Ferreira et al., 2009; Schwabe & Wolf, 2009). Conversely, the goal-directed system can quickly exert control over habitual response tendencies in the face of unexpected events, i.e., if habitual control turns out to be inappropriate (Hikosaka & Isoda, 2010).

In most cases, however, the two learning systems appear to work in a complementary rather than competitive fashion. Under stable environmental conditions, habitual control can be highly efficient, as it requires only few processing resources and thus allows for the simultaneous execution of several behavioural routines. Indeed, it has been suggested that action selection is a multi-step process in which stimulus-response associations (including O-R associations) largely determine the initial choice of a response option and thereby initiate the evaluation of this option through the retrieval of the corresponding response-outcome association. Feed-forward response selection and feedback evaluative processes finally converge in the supra-threshold activation of a specific response representation (Balleine et al., 2009). This raises the question how disambiguation is accomplished if the two action controllers generate conflicting response tendencies. Adopting a computational RL framework, Daw, Niv, and Dayan (2005, 2006) proposed that the relative uncertainty of the predictions of the two learning systems determines whether habitual or goal-directed control is favoured. While habits are assumed to be implemented by a computational simple but inflexible *model-free* RL algorithm, the goal-directed system is assumed to instantiate a flexible and adaptive but computationally costly *model-based* algorithm. Both RL methods are discussed in more detail below.

Computational Models of Reinforcement Learning

Computational approaches to RL where first considered in the 50s of the last century and have proven to be powerful tools for both explaining and predicting behavioural and neural correlates of learning over the last decades (Niv & Montague, 2008). Within the framework of RL algorithms, behavioural choice relies on the establishment of an optimal *policy* that maps a particular state *s* on the optimal action *a*, i.e., the action that leads to the largest expected sum of future rewards (Maia, 2009). A policy *p(s,a)* indicates the probability of choosing action a in state s and hence represents the strength of the S-R association. The learning of optimal policies can be accomplished via either model-free or model-based approaches. Model-based RL involves the development of an experience-based internal model of how the environment changes upon an agent' s actions. Specifically, the agent

learns the conditional probability that an initial state s_1 is followed by a state s_2 when performing an action *a* (transition function, $\mathrm{T}(\mathrm{s}_1, \mathrm{a}, \mathrm{s}_2)$) as well as the conditional probability of the corresponding reinforcements (reward function, $\mathrm{R}(s_1, \mathrm{a}, \mathrm{s}_2)$). The knowledge and continuous updating of the environmental dynamics do not only allow the agent to select appropriate actions, i.e., to find an optimal policy, but also enable them to flexibly adapt to changes in outcome value or transition contingencies (Dayan & Niv, 2008). In model-free RL, an optimal policy is estimated via trial-and-error, that is, without prior learning of an environmental model. Instead, behavioural choice relies on a recency-weighted average across successive "samples" of state-action-state sequences (Maia, 2009). Since transition probabilities and reward function are not explicitly learned, model-free RL does not allow for flexible adjustments to outcome revaluation or dynamic contingencies (Dayan & Niv, 2008). Model-free methods of behavioural choice involve error-correcting learning mechanisms that operate on inconsistencies between what has been predicted and what is actually happening. This concept can be traced back to the influential model of animal learning by Robert Rescorla and Allan Wagner (Rescorla & Wagner, 1972).

The Rescorla-Wagner Model and the Importance of Expectancy in Learning

The Rescorla-Wagner model (Rescorla & Wagner, 1972) significantly advanced theoretical accounts on learning in highlighting the importance of expectancy. Specifically, Rescorla and Wagner suggested that learning only occurs if an event differs from what has been expected. More formally, the model states that changes in the associative strength *V* of a CS can be described by the following rule (Niv & Montague, 2008):

$$\Delta V(CS_i) = \eta(CS_i, US)\left[\lambda(US) - \sum_i V(CS_i)\right].$$

According to this equation, learning only occurs if a difference exists between the prediction of the US due to all conditioned stimuli present in the situation ($\sum_{\mathrm{i}} \mathrm{V}(\mathrm{CS_i})$) and the maximal associative strength possible with the given US ($\lambda(\mathrm{US})$). The parameter $\eta(\mathrm{CS_i}, \mathrm{US})$ denotes the learning rate that depends on the salience of both CS and US ($\eta \leq 1$). Although the Rescorla-Wagner model made an important contribution to the field of RL by assuming

that expectancy deviations are necessary for learning to take place, some findings are difficult to reconcile with its assumptions (for examples, see Domjan, 2003). In particular, the model does not address the critical aspect of temporal factors in conditioning. This decisive drawback led to the proposal of the so-called *temporal-difference* learning rule (Sutton & Barto, 1988) that extended the Rescorla-Wagner model to the time domain.

Temporal Difference Learning and Prediction Errors

In temporal-difference (TD) learning, the agent estimates the value of states, i.e., the average sum of future rewards that can be obtained when choosing appropriate actions in the given state. The standard TD learning principle can be described by the following equation (Niv & Montague, 2008):

$$\Delta V(S_t) = \eta \left[r(t) + \gamma \sum_{S_{k,t+1}} V\left(S_{k,t+1}\right) - \sum_{S_{j,t}} V\left(S_{j,t}\right) \right].$$

In contrast to the Rescorla-Wagner model, each time point *t* within a trial constitutes a specific state for which a value $\mathrm{V}(\mathrm{S_t})$ is learned. Moreover, the stimuli $\mathrm{S_j}$ present at time t are assumed to predict not only the immediate reward $\mathrm{r(t)}$ but also the value of the following state $\mathrm{V}(\mathrm{S_{t+1}})$, i.e., future rewards that are themselves predicted by the stimuli $\mathrm{S_k}$ present at $\mathrm{t+1}$. The parameter $\gamma \leq 1$ denotes a discounting factor. Learning progresses as differences are evident between predicted ($\sum_{\mathrm{S_{j,t}}} \mathrm{V(S_{j,t})}$) and actually obtained ($\mathrm{r(t)} + \gamma \sum_{\mathrm{S_{k,t+1}}} \mathrm{V}\left(\mathrm{S_{k,t+1}}\right)$) rewards. Therefore, the term $\mathrm{r(t)} + \gamma \sum_{\mathrm{S_{k,t+1}}} \mathrm{V}\left(\mathrm{S_{k,t+1}}\right) - \sum_{\mathrm{S_{j,t}}} \mathrm{V}\left(\mathrm{S_{j,t}}\right)$ is also referred to as *prediction error (PE)* $\delta(\mathrm{t})$. Note that the PE reflects both the reinforcement obtained at time *t* as well as the difference in value between state $\mathrm{S_t}$ and the subsequent state $\mathrm{S_{t+1}}$. Hence, a positive PE can indicate either the unexpected occurrence of a reward or the transition to a state with a higher value than was predicted. That is, external reinforcement signals are not necessary for the PE to be different from zero. Conversely, a negative PE simply means that an event is "worse than expected". At the beginning of learning $\mathrm{V}(\mathrm{S_t})$ is set to some initial value and then iteratively improved or "updated" across successive trials ($\triangleq$ samples) according to (Niv & Montague, 2008):

$$V(S_t)_{new} = V(S_t)_{old} + \eta\delta(t).$$

Thus, the PE is zero and no further learning occurs if both the immediate reinforcement and the value of $V(S_t)_{new} = V(S_t)_{old} + \eta\delta(t)$ are predicted exactly. In order to select optimal actions, however, the agent needs to know not only the value of a particular state but also what subsequent states the available actions lead to. In model-free RL, these transition probabilities remain unknown. One solution to this fundamental problem was provided by *actor-critic* methods that used the PE to improve both state value predictions and policies (Barto, 1995; Barto, Sutton, & Anderson, 1983).

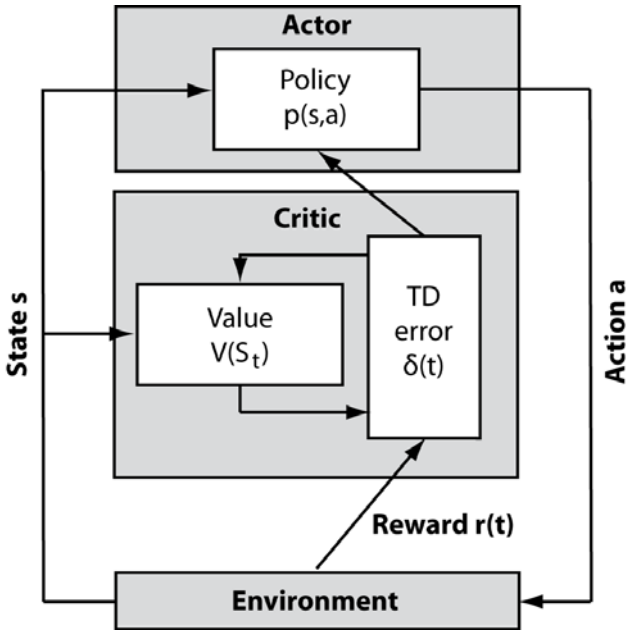

Figure 1: Schematic illustration of the actor/critic architecture. The critic computes the PE ($\delta(t)$) based on information about the state (S_t) and the reward ($r(t)$). The PE is used by the critic to improve its own state value predictions $V(S_t)$ and by the actor to improve the policy $p(s,a)$. Figure adapted from Niv (2009).

The Actor-Critic Architecture

The actor-critic consists of two basic units: the "adaptive critic" learns the state-value function according to TD principles and the "actor" learns action preferences for a given state, i.e., the policy (Maia, 2009). The critic calculates a PE that is used by the actor to improve action selection. A positive PE increases the probability of a recently chosen action *a* to be performed in state S_t, whereas a negative PE decreases this probability. The updating of the policy can be described as follows (Niv & Montage, 2008),

$$p(a|S_t)_{new} = p(a|S_t)_{old} + \eta_p \delta(t),$$

with the parameter $\eta_p \leq 1$ denoting the policy-specific learning rate. As can be seen from the equation, the actor applies the same TD rule to improve the policy as the critic does to update value estimation. Figure 1 illustrates the basic principles underlying actor-critic architectures.

Computational Accounts of Model-based Reinforcement Learning

The computational principles underlying goal-directed action selection in model-based RL have been explored far less extensively than model-free methods, such as the TD algorithms described above (Botvinick & An, 2009; Hasselmo, 2005). As Daw and colleagues (Daw, Niv, & Dayan, 2005) have pointed out, model-based RL requires the agent to estimate the parameters of both the transition function and the reward function that in conjunction with the corresponding sets of states and actions constitute a model of the environment. Action selection is then achieved through exploring the model, i.e., searching for the optimal path in a complex tree-like structure of possible successor states (Dayan & Niv, 2008). The acquisition and application of this knowledge can be modelled within a Bayesian framework in which experience about transitions and rewards is used to update prior distributions over the parameters of the functions *T* and *R* (Tenenbaum, Griffiths, & Kemp, 2006; Toussaint & Storkey, 2006). The posterior distribution over *T* and *R* for any parameter values within the defined range indicates how likely they are to represent the true parameter values given the data observed so far. In the next iteration, this posterior distribution serves as the prior distribution and is updated again. Since prior and posterior distributions converge very quickly, the Bayesian approach provides appropriate estimates of transition and reward probabilities even in case of unstable environments.

Summary and Implications for the Present Study

Research on both classical and instrumental conditioning has indicated that individuals rely on expectancies about the potential outcomes of situations in the course of RL. According to the Rescorla-Wagner model (Rescorla & Wagner, 1972) these expectancies are of primary importance as only surprising events that defy expectations can lead to successful learning. Modern computational accounts of RL have extended this basic claim to the time domain by invoking TD learning rules that define learning more broadly in terms of evaluating differences between predicted and actual values of subsequent states. Unexpectedly rewarding outcomes or high state values lead to a positive PE whereas a negative PE indicates that an outcome was worse than expected. Actions associated with a positive PE will consequently be performed more frequently in the future while the likelihood of actions corresponding to a negative PE decreases.

The present thesis is largely based on a neurocomputational model, has establishes a link between the PE and an ERP-components that are elicited when participants make mistakes and are presented with error feedback during RL tasks (Holroyd & Coles, 2002). Specifically, the model asserts that these components directly reflect negative PEs, making them suitable tools to examine the time-course of RL.

Reinforcement Learning in the Brain

Computational models of RL have gained increasing popularity among neuroscientists during the last two decades, as the brain appears to implement some of the basic mechanisms and elements of these normative models (Niv & Montague, 2008). Most notably, electrophysiological recordings in non-human primates have revealed that phasic activity of midbrain dopamine (DA) neurons seems to code a PE signal during classical and instrumental conditioning (for reviews see Schultz, 2000, 2006, 2007, 2010). Moreover, functional neuroimaging studies in humans have provided evidence for PE signals in main cortical and subcortical projection areas of the midbrain DA system such as the prefrontal cortex (PFC) and the basal ganglia (BG) (O'Doherty et al., 2004; Pessiglione et al. 2006; Schönberg et al., 2007). The objective of the following section is to give an overview of the

dopamine reward prediction error hypothesis (Montague et al., 1996; Schultz et al., 1997) and how it relates to the neural implementation of RL.

The Dopamine Reward Prediction Error Hypothesis

The dopamine reward prediction error (DA-RPE) hypothesis was largely inspired by the work of Wolfram Schultz and colleagues that demonstrated a striking similarity between the phasic firing patterns of single dopaminergic neurons in the ventral tegmental area (VTA) of monkeys and the characteristics of TD prediction errors (Schultz et al. 1997). After the presentation of unexpected primary rewards or reward-predicting stimuli these neurons typically show a phasic increase in activation. Importantly, the response of these DA neurons does not appear to primarily relate to general processes of attention or arousal since it differentiates between rewards and salient non-rewarding events such as aversive stimuli (e.g., Mirenowicz & Schultz, 1996). The most striking finding, however, was that the dopaminergic firing pattern changed systematically over the course of learning in simple Pavlovian or instrumental conditioning tasks.

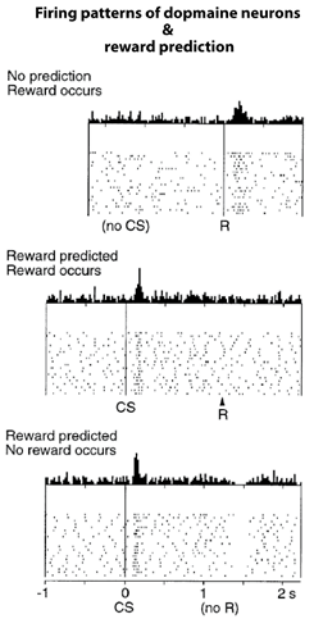

Figure 2: Firing patterns of DA neurons from the VTA during instrumental learning. *Top:* Phasic increase in activity of DA neurons after an unpredicted reward (R). *Middle:* After learning, the phasic increase in activity of DA neurons occurs after the presentation of reward-predicting stimulus (CS), whereas DA neurons are no longer activated by the reward (R) itself. *Bottom:* If the predicted reward is not delivered (no R), activity of DA neurons is phasically depressed at the same time the reward should have occurred. (Figure adapted from Schultz, 1997).

Figure 2 illustrates that during the initial stages of instrumental learning the unpredicted reward elicits a phasic burst of DA neurons. After some practise with the task, cells respond to both the reward and the reward-predicting cue. Once learning is completed, DA neurons are only activated by the cue and no longer by the fully predicted reward. Instead, dopaminergic activity transiently drops below baseline if the predicted reward is unexpectedly omitted. This shift of the phasic DA response from the primary reward to the reward-predicting cue exactly mirrors the properties of a TD reward prediction error in that phasic increases and decreases code whether states are “better or worse than expected” (see also Bayer & Glimcher, 2005; Bayer, Lau, & Glimcher, 2007). Learning-related phasic dopaminergic signals have been shown to occur with latencies of less than 100 ms and durations of less than 200 ms (for reviews, see Schultz, 2007, 2010) and are thought to be functionally distinct from slower changes in tonic levels of extra-synaptic DA (e.g., Niv, 2007; Seamans & Yang, 2004). More recent studies have provided additional support for the DA-RPE hypothesis by showing that the dopaminergic response reflects the magnitude and probability of expected rewards (Fiorillo et al. 2003, Tobler et al. 2005) as well as temporal discounting of delayed rewards (Roesch et al. 2007), and is consistent with behavioural phenomena like the blocking effect (Waelti et al., 2001).

In humans, most evidence for the existence of a dopaminergic RPE signal has been inferred from functional magnetic resonance imaging (fMRI) studies on reward learning. Correlates of RPEs have been found in areas that constitute major targets of dopaminergic afferents, such as the ventral and dorsal striatum (O’Doherty et al., 2004; Tanaka et al., 2006) and prefrontal areas, including the orbitofrontal, dorsolateral, and ventrolateral cortex (Cohen, 2007; McClure et al., 2003; Rolls, McCabe, & Redoute, 2008). Moreover, it has been shown that RPE-related activity in the striatum correlates with learning performance (Schönberg et al., 2007) and that both the RPE-signal in the striatum and behavioural choices are modulated by the administration of DA agonists and antagonists (Pessiglione, Seymour, Flandin, Dolan, & Frith, 2006). Recent advances in imaging techniques have also revealed RPE-like activity directly in the human midbrain dopaminergic nuclei (D’Ardenne, McClure, Nystrom, & Cohen, 2008) However, dopaminergic signals have been shown to subserve dissociable functions not only at different time-courses but also in different target

areas (Schultz, 2000; 2007), with distinct roles for phasic DA input to the PFC and the BG (Cools & D'Esposito, 2011; van Schouwenburg, Aaarts, & Colls, 2010).

Dissociable Roles of Dopamine in the Basal Ganglia and the Prefrontal Cortex

Midbrain dopaminergic activity is thought to influence neural processing through three major pathways (Björklund & Dunnett, 2007). The *nigrostriatal* pathway arises from DA cells in the zona compacta of the substantia nigra (SNc) that project mainly to the dorsal striatum. The *mesolimbic* and *mesocortical* pathways denote projections from the VTA to limbic regions, including the nucleus accumbens (ventral striatum), amygdala, and hippocampus (mesolimbic system) and frontal cortical regions, including the medial prefrontal, cingulate, and perirhinal cortex (mesocortical system).

The Basal Ganglia and Action Selection

Research on RL largely focused on dopaminergic projections to the striatum that is considered one of the two main input structures of the BG. The BG are a group of subcortical nuclei that have been assigned a pivotal role in action selection, specifically in the acquisition and expression of habits (for reviews, see Redgrave et al., 2010; Seger & Spiering, 2011; Yin & Knowlton, 2006). Indeed, early accounts assumed that the BG and reward-related dopaminergic input directly implement S-R learning as described by the Law of Effect, with reward-related information conveyed by dopaminergic input (e.g., Mishkin, Malamut, & Bachevalier, 1984). One of the most influential views on the BG architecture and function holds that the intrinsic connectivity pattern of the nuclei comprises a 'direct', a 'indirect', and a 'hyperdirect' pathway (Albin, Young, & Penney, 1989). Crucially, it has been suggested that the 'direct' pathway facilitates the execution of appropriate actions in a given state, whereas the 'indirect' pathway inhibits inappropriate actions. Dopaminergic signals from VTA and SNr are thought to control the relative dominance of the two pathways (Albin et al., 1989; Redgrave et al., 2010). Recent computational models established a direct link between the 'gating' function of the BG and the DA-RPE hypothesis in proposing that phasic DA bursts

and dips mediate RL in the BG circuits by strengthening or weakening synaptic plasticity within the 'direct' and 'indirect' pathway, respectively (Maia & Frank, 2011).

Different Cortico-Basal Ganglia-Cortical Loops Contribute to Reinforcement Learning

The BG are connected with the cerebral cortex through parallel albeit partly overlapping cortico-basal ganglia-cortical loops, constituting functionally distinct networks processing motivational and affective (limbic network), cognitive (associative network), and sensorimotor-related information (Alexander, DeLong, & Strick, 1986; Draganski et al., 2008; see Figure 3). In the limbic network, the orbitofrontal cortex (OFC), ventral PFC, and anterior cingulate cortex (ACC) project to more ventromedial regions of the striatum. The associative network involves projections from the prefontal and parietal association cortices (most notably the dorsolateral prefrontal cortex, dlPFC) to the dorsomedial striatum, whereas the dorsolateral striatum receives major input from sensorimotor cortices (Draganski et al., 2008; Postuma & Dagher, 2006; Saint-Cyr, 2003).

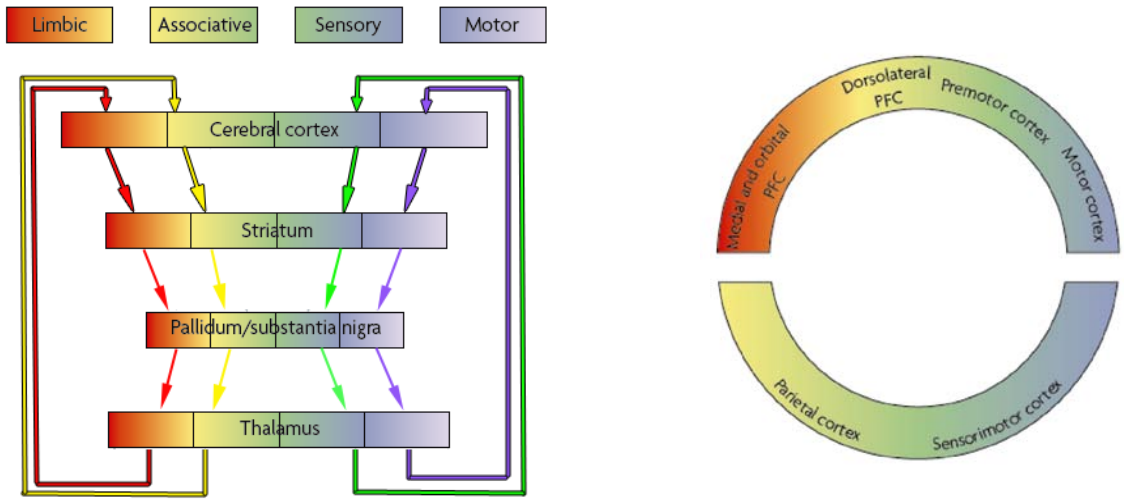

Figure 3: Corticobasal ganglia-cortical loops. *Left*: Schematic illustration of the parallel loops, conveying limbic (red), associative (yellow-green), and sensorimotor (blue-white) information. *Right*: The ring shows limbic (red), associative (yellow-green), and sensorimotor (blue-white) cortical regions. Within the ring the subdivisions of the striatum are depicted. Colors code subregions that receive the strongest input from the corresponding cortical regions. (Figure adapted from Redgrave et al., 2010).

In line with this structural organization, sensorimotor and associative systems are thought to mediate habitual S-R learning and goal-directed A-O learning, respectively (Yin & Knowlton, 2006). The limbic network has been implicated mainly in the learning of Pavlovian state values and is assumed to mediate motivational influences on both types of learning via inputs to the sensorimotor and associative loop (Balleine & O'Doherty, 2010). According to a related view (Ito & Doya, 2011), the ventral and dorsomedial striatum are critically involved in implementing model-based RL, while the dorsolateral striatum is considered a primary locus in mediating model-free RL.

In support of this notion, human fMRI studies have revealed increased activation of the dorsomedial striatum, the OFC, and the medial prefrontal cortex (mPFC) for training on schedules promoting goal-directed responding (high A-O contingency) compared to training on schedules promoting habitual responding (low A-O contingency) (Hampton, Bossaerts, & O'Doherty, 2006; Tanaka, Balleine, & O'Doherty, 2008). Moreover, Valentin and colleagues (2007) demonstrated that activity in the OFC exhibited sensitivity to outcome devaluation in instrumental learning, which is consistent with the presumed role of this region in representing the value of goals and action outcomes (Kringelbach et al., 2003; O'Doherty et al., 2001; Plassmann et al., 2007). Conversely, activity in the dorsolateral striatum has been shown to track the progression from goal-directed to habitual responding (Tricomi et al., 2009).

Within the framework of actor-critic architectures, the dorsolateral striatum has been assigned the role of the actor that learns the policy (S-R associations). The critic, which learns state values (S-O associations) and calculates the RPE has been associated with the ventral striatum (O'Doherty et al., 2004), possibly working in tandem with the OFC and the amygdala (Maia, 2009) - two structures that are closely connected to both the ventral striatum and the midbrain DA system (Rempel-Clower, 2007). This implies that ventral and dorsolateral striatum should be differentially engaged in Pavlovian and instrumental conditioning, as only the latter involves outcome-guided action selection. In support of the assertion that the ventral and dorsal striatum are concerned with outcome prediction and instrumental responding, respectively, a number of fMRI studies found RPE-like signals in the ventral striatum during both types of learning whereas RPE-like signals in the dorsal

striatum arose only during instrumental learning (O' Doherty et al., 2004; Tricomi, et al., 2009; Valentin & O'Doherty, 2009). Moreover, recent evidence indicated that the ventral striatum was involved in both reward-based and punishment-based RL (Robinson et al., 2010, Delgado, Jou, & Phelps, 2011). Although the actor/critic RL model implies that the computational function of dopaminergic signals is confined to habit learning, the involvement of the dorsomedial striatum in action-outcome learning as well as the strong dopaminergic projections to the PFC suggest that DA also contributes to goal-directed behavioural control.

The Role of Dopamine in the Prefrontal Cortex

The PFC is comprised of a collection of heterogeneous structures that have been assigned distinct functional roles in RL. Cytoarchitecturally, the PFC can be divided into the following subregions (Ridderinkhof, van Wildenberg, Segalowitz, & Carter, 2004): (1) the OFC, (2) the lateral PFC, including the dorsolateral PFC (BA[3]9/46, 46, and 8a), and (3) medial frontal cortex (MFC), including the ACC (BA 24, 25 and 32). The PFC, including orbitofrontal, dorsolateral, and anterior cingulate regions, is richly innervated by midbrain DA neurons via the mesocortical pathway (for reviews, see Arnsten, 1998; Floresco & Magyar, 2006; Cools & D'Esposito, 2011). Dopamine neurotransmission in prefrontal regions has been shown to exert strongly modulatory influences on a variety of cognitive and executive functions such as working memory, behavioural flexibility, decision making, and attentional control (for reviews, see Cohen, Braver, & Brown, 2002; & D'Esposito, 2011; Floresco & Magyar, 2006).

It should be noted that DA-mediated effects on PFC neurons have been shown to be relatively long-lasting and thus do not appear to match the functional properties of a PE signal (Seamans & Yang, 2004). Theoretical and empirical work has instead indicated complementary functions for DA in the BG and the PFC (Frank & Claus, 2006; Hazy, Frank; & O'Reilly, 2006; Leber, Turk-Browne, & Chun, 2008; McNab & Klingberg, 2008). Specifically, it has been suggested that striatal DA may promote flexible updating of mental

[3] Brodmann Area

representations in the PFC, whereas frontal DA has been linked to stable maintenance of current representations (Cools & D'Esposito, 2011).

Substantial evidence for a role of frontal DA in the stabilization of working memory representations has been provided by human fMRI studies which demonstrated that functionally selective improvements and impairments of working memory performance due to DA agonists and antagonists are accompanied by activity changes in dlPFC, cingulate cortex, and insula (e.g., Gibbs & D'Esposito, 2005). At the cellular level, DA receptor stimulation in the PFC has been proposed to increase the signal-to-noise ratio in terms of attenuating neuronal firing associated with all but the most strongly activated memory states (Seamans & Yang, 2004; Thurley, Senn, & Luscher, 2008; Williams & Goldman-Rakic, 1995). DA transmission to the PFC might hence support the stable maintenance of task-relevant information, protecting those representations from interference by task-irrelevant distractors. In support of this notion, the Val/Met polymorphism in the catechol-*O*-methyltransferase (COMT) gene, which is thought to determine prefrontal DA levels in humans, has been shown to predict individual differences in working memory performance. Met-allele carriers which are characterized by low COMT activity and high prefrontal DA levels perform better in tasks requiring the persistent stabilization of task-relevant presentations than Val-allele carriers characterized by high COMT activity and low prefrontal DA (Frank, Moustafa, Haughey, Curran, & Hutchison, 2007a; Meyer-Lindenberg et al., 2005).

As I pointed out earlier, the BG are thought to subserve a dynamic gating function for information flow to the cortex via cortico-basal ganglia-cortical loops. In particular, phasic bursts and dips in DA are assumed to increase and decrease the likelihood of an action being performed through modulations of plasticity in the direct and indirect pathway, respectively (Frank, 2005). The described architecture of the cortico-basal ganglia-cortical circuits renders it likely that the BG gate not only motor programs (sensorimotor loop), but also cognitive information (associative loop) to the PFC (Frank, 2005). In line with this view, increased striatal activation has been observed during tasks requiring rapid updating of cognitive representations such as reversal learning and task switching (Leber, Turk-Browne, & Chun, 2008; Cools, Clark, & Robbins, 2004). Notably, performance in those tasks is also susceptible to DA agonists and antagonists that appear to selectively influence BG activity

during flexible updating in response to new input and PFC activity during active maintenance (Cools et al., 2007; Dodds et al., 2009). Consistent with the proposed functional dichotomy, a reciprocal relationship has been observed between DA levels in the striatum and the PFC (Meyer-Lindenberg et al., 2005).

Alternatively, it has been suggested that the balance between stable maintenance and appropriate updating of task-relevant representations may be regulated by phasic vs. tonic dopaminergic activity (Cohen, Braver, & Brown, 2002). According to this view, phasic increases in DA trigger the transient gating of new information into the PFC and thereby also provide a RL signal for learning when to update, whereas DA dips rapidly deactivate the PFC and thus "clear" working memory contents. By comparison, tonic DA levels are assumed to stabilize currently active working memory representations. Biophysically sophisticated models, however, have challenged this account by emphasizing the prevalence of tonic DA in PFC functioning (Brunel & Wang, 2001; Durstewitz & Seamans, 2002). In particular, several researchers have posited that the kinetics of DA transmission in the mesocortical pathway do not match the requirements of a phasic learning signal that triggers rapid updating of PFC representations[4] (Jocham & Ullsperger, 2009; Seaman & Yang, 2004).

Either way, the PFC has been strongly implicated in model-based learning and is assumed to exert a top-down biasing influence on the activity of the model-free RL system, possibly mediated by hierarchical interactions between dorsomedial and dorsolateral cortico-striatal loops (Daw et al., 2005; 2006a; Frank et al., 2007a; Miller & Cohen, 2001). This is in line with the observation that value-related signals in OFC and MFC vary as a function of response-outcome contingencies and outcome devaluation (Hampton et al., 2006; Tanaka et al., 2008). Moreover, it has been suggested that the PFC, most notably the ACC, is not only a *recipient* of phasic dopaminergic effects but also *drives* phasic activity of the midbrain DA

[4] In an attempt to resolve these inconsistencies, Cohen and colleagues (2002) argued that two distinct receptor types mediate phasic and tonic effects of DA in the PFC. The authors associated the tonic maintenance effects with the slow acting D1 receptor type and the phasic updating and RL effects with the more rapidly acting D2 receptor type (Seamans, Gorelova, Durstewitz, & Yang, 2001; Semans & Yang, 2004). Given that the number of D2 receptors is much larger in the striatum than in the PFC (Seamans & Yang, 2004), however, it seems reasonable to assign a pivotal role in updating PFC representations to the cortico-striatal circuits rather than the "direct" mesocortical DA pathway.

system (Frank et al., 2005; Jocham & Ullsperger, 2009; Strafella, Paus, Barrett, & Dagher, 2001). The following section summarizes some compelling demonstrations of how activity in the PFC might impact RL in a top-down (or model-based) manner.

Rapid Trial-To-Trial Learning in the Hippocampus-Prefrontal Cortex System

On the basis of the theoretical and empirical work that has established a close link between the PFC and adaptive action selection, Frank and colleagues (Doll, Jacobs, Sanfey, & Frank, 2009; Frank & Claus, 2006) suggested that the active maintenance of task-relevant information in the PFC is likely to exert a top-down influence on response selection mediated by the more slowly learning BG system. Specifically, they proposed that the OFC maintains information concerning the magnitude of recent response outcomes in working memory, thereby biasing behavioural adaptation on a trial-by-trial basis. By comparison, the dlPFC is usually assigned a key role in the maintenance of task set, particularly in the implementation of behavioural goals and rules (Miller & Cohen, 2001; Tanji & Hoshi, 2008).

In several genetics studies, Frank and colleagues demonstrated that COMT genotype determined trial-to-trial behavioural adjustments in probabilistic reinforcement learning tasks (Frank et al., 2007a; Frank, D'Lauro, & Curran, 2007b; Frank et al., 2009). For example, Met-allele carriers, which are characterized by higher prefrontal DA levels, showed a greater tendency to slow down and switch their response after negative feedback than did Val-allele carriers with lower prefrontal DA levels (Frank et al., 2007a). The authors explained this finding in terms of Met-allele carriers being able to better maintain the outcome of a particular response across several intervening trials. Furthermore, a recent study used spectral Granger causality analyses to demonstrate a stronger top-down-directed functional connectivity between the MFC, most likely the ACC, and the ventral striatum when rewards were anticipated compared to a no-reward condition (Cohen et al, 2011). The notion of a top-down biasing influence of the PFC is also consistent with reports of performance deficits during early stages of probabilistic learning in patients with OFC lesions (Chase et al., 2008). Rapid behavioural adaptation after the reversal of the response-outcome contingencies is assumed to rely strongly on robust working memory representations of recent outcomes.

Nonetheless, the OFC-lesioned patients were able to respond appropriately after extensive training on the new rule, suggesting that the habitual learning system is spared.

A similar dissociation has been observed for patients with lesions of the medial temporal lobe (MTL) memory system (Bayley, Frascino, & Squire, 2005; Shohamy, Myers, Kalanithi, & Gluck, 2008). The hippocampus and the surrounding MTL have been extensively studied within the framework of episodic memory (Eichenbaum & Cohen, 2001) and are thought to be significantly involved in explicit, non-incremental learning processes (Shohamy et al., 2008; Shohamy & Adcock, 2010). In particular, the MTL memory system supports the rapid ("one-trial") formation of conjunctive representations linking multiple aspects of an event. Interestingly, hippocampal function also appears to be directly modulated by dopaminergic input, reflected in a DA-dependent facilitation of episodic memory formation (Adcock et al., 2006; Krebs et al., 2009). In RL tasks, hippocampus and MTL might thus support explicit memorizing of correct and incorrect choices after a single trial in which a certain stimulus-response pairing is followed by a rewarding vs. non-rewarding outcome. Indeed, several findings have indicated that during early stages of learning the MTL memory system contributes to the development of explicit rules for responding (Frank et al., 2004; Frank, O'Reilly, & Curran, 2006; Poldrack et al., 2001).

Whereas hippocampus and MTL have been associated with the encoding and storage of these rules, the PFC is assumed to be critically involved in the retrieval and active maintenance of abstract rules and explicit contingencies[5] (Badre, Kayser, & D'Esposito, 2011; Bunge & Souza, 2008). Combining computational modelling with analyses of genetic variants of dopaminergic neurotransmission, Doll and colleagues (Doll et al. 2009; Doll, Hutchison, & Frank, 2011) showed that rule-like representations in the PFC might lead to a confirmatory bias, so that rule-consistent evidence is overweighed, whereas rule-inconsistent evidence is discounted by the BG system. Moreover, these cognitive strategies are accompanied by systematic changes in striatal activity in reward-based learning (Delgado, Gillis, & Phelps, 2008). In line with the above findings, recent evidence from human

[5] Note, however, that the PFC has also been implicated in other aspects of episodic memory, e.g. during encoding (for review, see Paller & Wagner, 2002).

neuroimaging indicated that the PE signal in the ventral striatum reflected not only model-free but also model-based evaluation (i.e., learned rules) indicating close interactions between the different learning systems (Daw et al., 2011).

In sum, potential dopaminergic influences on RL are apparently not restricted to PE signalling in the striatum and model-free habitual behavioural control. Instead, DA also modulates working memory functions and explicit rule learning and acts on regions like the PFC and the hippocampus that have been associated with model-based, goal-directed control. Moreover, a highly integrated architecture underlies RL, implemented by corticostriatal interactions through hierarchical spiraling connections between corticostriatal loops from ventromedial (limbic) via dorsomedial (associative) to dorsolateral (sensorimotor) subregions (Haber, Fudge, & MacFarland, 2000).

The Integration of Cognition, Emotion, and Action in the Anterior Cingulate Cortex

Alongside the BG, the ACC is considered one of the key structures implementing RL and adaptive decision-making. One of the most characteristic features of this brain region is its involvement in a broad range of functions, including emotion and motivation, autonomic control, pain, and action selection (for reviews, see Bush, Luu, & Posner, 2000; Rushworth & Behrens, 2008; Paus, 2001; Shackman et al., 2011). Neuroanatomically, the ACC is ideally positioned to synthesize behaviourally relevant information from multiple sources through its reciprocal interconnections with OFC, ventral striatum, hippocampus, amygdala, insula, and the midbrain DA nuclei. In addition, the ACC's strong connectivity with the lateral PFC, premotor and motor cortices implies that this information can be directly integrated with current behavioural goals in order to guide instrumental behaviours. Indeed, it is increasingly acknowledged that the ACC uses reinforcement information to implement adaptive behavioural control (Rushworth & Behrens, 2008; Holroyd & Coles, 2002; Holroyd & Yeung, 2011; Shackman et al., 2011).

Considerable evidence for an evaluative function of the ACC has been inferred from human electrophysiological studies on performance monitoring and learning. In particular, significant insight has been gained from examining two components in the scalp-recorded

electroencephalogram (EEG), sometimes termed the error negativity (Falkenstein, Hohnsbein, & Hoormann, 1990) and feedback-related negativity (Miltner et al., 1997), that are elicited when participants make mistakes and are presented with error feedback, respectively. Both components are thought to be generated in the ACC (Debener et al., 2005) and might reflect PE-like signals that are used to flexibly adjust behaviour after response errors or negative feedback (Chase et al., 2011; Cohen & Ranganath, 2007; Holroyd & Coles, 2002; van der Helden, Boksem, & Blom, 2010). The ACC thus appears to be critically involved in the detection and correction of discrepancies between the intended or anticipated and the actual outcomes of an action – a key component of cognitive control that is commonly referred to as performance (or action) monitoring.

The Anterior Cingulate Cortex Encodes Action Values

Theoretical accounts on ACC functioning, however, disagree with respect to what exactly is monitored, with proposals including errors, error likelihood, response conflict, and several other phenomena (Brown, 2009; Holroyd & Coles, 2002; Yeung, Botvinick, & Cohen, 2004). Moreover, there is some debate as to whether the functional role of the ACC is restricted to detecting the need for control that is mediated by other brain regions, e.g., the dlPFC (Gehring & Knight, 2000; Yeung et al., 2004) or directly involves the implementation of control in order to optimize behavioural choice (Holroyd & Coles, 2002; Holroyd & Yeung, 2011; Mansouri, Tanaka, & Buckley, 2009). The latter view is supported by the ACC's dense connectivity with motor areas as well as human neuroimaging and lesion studies indicating its fundamental contribution to voluntary action generation (Paus, 2001). Notably, the ACC has been proposed to provide the neural substrate for uncertainty-based arbitration between model-free and model based control (Daw et al., 2005) and high-level behavioural option selection (Holroyd & Yeung, 2011).

These controversies notwithstanding, previous research demonstrated that the ACC encodes a type of PE signal in a variety of learning tasks in both humans and monkeys (Amiez, Joseph, & Procyk, 2006; Hester, Barre, Murphy, Silk, & Mattingley, 2008; Jocham, Neumann, Klein, Danielmeier, & Ullsperger, 2009; Kennerley, Walton, Behrens, Buckley, &

Rushworth, 2006; Matsumoto, Matsumoto, Abe & Tanaka, 2007; Schiller, Levy, Niv, LeDoux, & Phelps, 2008). In support of this notion, human fMRI studies showed that error-related ACC activation predicted future performance in an associative learning task (Hester et al., 2008). Moreover, negative PE-related activity in the ACC was directly related to learning rate, i.e., to the weight of the PE in updating action values (Jocham et al., 2009). Interestingly, faster learning rates have been observed in rapidly changing environments, when subjects had to rely more strongly on recent response outcomes (Rushworth & Behrens, 2008). In addition, Behrens and colleagues (2007) observed a correlation between individual estimates of this environmental volatility and outcome-related ACC activation. Hence, the ACC might encode the behavioural impact or informative value of a given action outcome in accordance with the specific environmental demands.

Further evidence for a specific link between the PE-like activity in the ACC and action selection comes from studies comparing the effects of OFC vs. ACC lesions on Pavlovian vs. instrumental reversal-learning (Kennerley & Walton, 2011). In one of these studies, Rudebeck and colleagues (2008) found that ACC-lesioned animals were severely impaired in learning instrumental reversals but showed virtually no deficits when the task required an update of stimulus-reward associations. By contrast, animals with OFC lesions showed the opposite pattern in that their performance was unaffected when action-reward associations were to be learned but strongly disrupted in the stimulus-reinforcement reversal-learning task. These findings substantiate the notion that ACC and OFC differentially contribute to adaptive decision-making: whereas the ACC appears to be more strongly involved in linking action-reinforcement history to future action selection, the OFC seems to be critical for the representation of the expected value of stimuli (Rushworth, Behrens, Rudebeck, & Walton, 2007). In line with the cited animal research, ACC lesions in humans are generally accompanied by deficits in error detection and error-related remedial actions (Di Pellegrino, Ciaramelli, & Làdavas, 2007; Stemmer, Segalowitz, Witzke, & Schonle, 2004; Swick & Turken, 2002). Patients with OFC damage, in contrast, are principally impaired in detecting changes in the reward value of stimuli (Hornak et al., 2004). However, recent evidence from human neuroimaging indicated that the medial OFC also encodes action-related value

representations and is involved in action-based reversal learning, suggesting a general role for this structure in goal-directed behaviour (Gläscher, Hampton, & O'Doherty, 2009).

The above section has highlighted the ACC's potential contribution to reinforcement-guided action selection. Yet, this region is also known to play an important part in emotion and motivation. In the following section I will first sketch a highly influential account on functional segregation within the ACC. Then I will discuss the 'adaptive control hypothesis' that has been formulated by Shackman and colleagues (2011) in an attempt to provide an integrative account on the ACC's activity based on recent challenges to the segregationist view.

The Anterior Cingulate Cortex Contributes to Emotional and Motivational Processing

The ACC is not a homogeneous cortical structure, but can be divided into different subdivisions with distinct cytoarchitectural properties and connectivity patterns (Vogt et al., 1992). A prominent view on functional segregation within the ACC holds that the rostral-ventral division (rACC) is mainly involved affective processing, whereas the dorsal division (dACC) subserves cognitive functions (Bush, Luu, & Posner, 2000; Devinsky et al., 1995; see Figure 4).

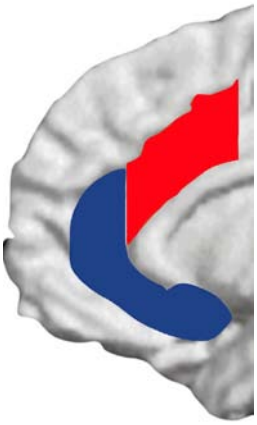

Figure 4: The segregationist model of ACC functioning: cognitive division (red) vs. the affective division (blue) (Figure adapted from Bush et al., 2000)

The segregationist model draws on neuroanatomical and functional neuroimaging evidence. As Bush and colleagues (2000) pointed out, the rostral 'affective' subdivision receives massive input from limbic regions, including associated structures like the OFC and the ventral striatum, and projects to the autonomic and visceral system. Hence, the rACC is well positioned to process the affective and motivational salience of an event and to regulate emotional responses. By comparison, the dACC has been suggested to maintain only few connections to "affect- and motivation-related" brain regions and instead is richly interconnected with lateral prefrontal, parietal, and motor regions. Accordingly, the dACC is assumed to subserve a variety of executive control functions, including performance monitoring, working memory, and effort-related decision-making. Bush and colleagues (2000) reviewed several fMRI findings and meta-analyses that support their proposal. More recently, two fMRI studies reported increased phasic rACC activation in response to errors that were financially penalized compared to errors that did not result in monetary losses, whereas phasic dACC activation did not differentiate between these error types (Simões-Franklin, Hester, Shpaner, Foxe, & Garavan, 2010; Taylor et al., 2006).

Using the advanced technique of coordinate-based meta-analysis, Shackman and colleagues (2011), however, provided evidence for a considerable overlap of neural activation foci for a large database including 192 imaging studies of cognitive control, negative affect, and pain. The identified cluster of activation overlap roughly corresponded to the rostral cingulate zone (RCZ), a premotor area lying in the vicinity of the cingulate sulcus (Morecraft & Tanji, 2009). Note that the RCZ belongs to the 'cognitive' division of the ACC according to the segregationist model of Bush and coworkers (2000). Shackman and colleagues (2011) reviewed neuroanatomical and functional evidence indicating that the ACC, including the RCZ, is a 'hub-like' convergence zone in which affectively and motivationally relevant information is linked to motor areas involved in implementing and synchronizing instrumental motor output. Specifically, their 'adaptive control hypothesis' holds that the ACC implements a domain-general function of integrating punishment-related information in order to bias instrumental responding and to arbitrate between competing motor controllers, particularly in unstable and threatening environments when habitual control fails to effectively guide behaviour. Consistent with this notion, the ACC has been found to

encode a PE signal in several studies using aversive learning procedures (Menon et al., 2007; Schiller et al., 2008; Seymour et al., 2004). Considerable evidence, however, points to an additional involvement of the ACC in reward-related processing and positive affect (for reviews, see Haber & Knutson, 2010; Liu, Hairston, Schrier, & Fan, 2010), encouraging the view that this structure might contribute to the evaluation of both positive and negative response outcomes in the service of signalling the need for behavioural change (cf. Magno, Simões-Franklin, Robertson, & Garavan, 2008).

Summary and Implications for the Present Study

The DA-RPE hypothesis posits that phasic activity of midbrain DA neurons code a PE signal during classical and instrumental conditioning (Schultz, 2000; 2006; 2007; 2010). Functional neuroimaging research in humans corroborated the notion that activity in a network of cortical and subcortical dopaminergic systems reflects RL mechanisms involved in the estimation and utilization of PE. Whereas sensorimotor and associative cortico-striatal loops are thought to mediate habitual S-R learning and goal-directed A-O learning (Yin & Knowlton, 2006), the ventral striatum and the OFC have been implicated in the learning of Pavlovian state values and the computation of PEs (O'Doherty et al., 2004; Tricomi, et al., 2009; Valentin & O'Doherty, 2009). It has been suggested that phasic dopaminergic transmission to the BG promotes flexible updating of mental representations in the PFC, whereas frontal DA has been linked to stable maintenance of current representations (Cools & D'Esposito, 2011). Moreover, prefrontal and MTL regions have been associated with explicit rule learning (model-based learning) and are thought to exert a top-down biasing influence on model-free incremental RL in the BG (Daw et al., 2005; Doll, Jacobs, Sanfey, & Frank, 2009; Frank & Claus, 2006). In particular, neuroimaging and electrophysiological research has indicated a prominent role for the ACC in RL, which acts as an interface between affective/motivational and cognitive processing and can thus mediate the flexible adjustment of behaviour in response to errors and/or negative feedback according to specific situational demands. Of note, the ACC is also thought to underlie the generation of the ERP-correlates of error and feedback processing this thesis focuses on.

On the Significance of Affective and Motivational Context in Learning and Adaptive Decision Making

The above discussion on the functional properties of the ACC underlines the need to explore the performance-monitoring system in terms of both cognitive and motivational-emotional mechanisms. Indeed, researchers have increasingly acknowledged the close interaction between cognitive, motivational, and emotional processes in mediating adaptive behavioural control (Etkin & Egner, & Kalisch, 2010; Pessoa, 2008; 2009; Pessoa & Engelmann, 2010). Moreover, it has been suggested that there is no clear distinction between cognition and emotion at both the functional and the neuroanatomical level (Pessoa, 2008; Salzman & Fusi, 2008). In the following sections, I will briefly introduce the concepts of 'motivation' and 'emotion'. This overview is followed by a sketch of how motivational and emotional processes are incorporated into cognitive control and learning.

Basic Concepts

The terms 'emotion' and 'motivation' denote two closely linked, complex constructs referring to abstract inner states of an individual (LeDoux, 2002; Roseman, 2008). Emotions are commonly thought to provide an evaluation of the relationship between an organism and its environment whereas motivation concerns temporal aspects (initiation, maintenance, stopping), intensity, and direction of behavioural responses (LeDoux, 2002; Pessoa, 2009; Roseman, 2008; Rothermund & Eder, 2011). Emotions have been assigned a critical role in the regulation of motivated behaviours and are assumed to activate specific behavioural tendencies, most notably appetitive and aversive motivational circuits (Lang & Bradley, 2008). Hence, emotions themselves appear to be 'motivators'. Since it has been proven difficult to find clear definitions of both motivation and emotion and to distinguish these concepts from cognition, I will use the terms for partly overlapping processes in the following.

Concepts of Motivation and their Relevance for Learning

Early theories of motivation were intimately linked to the concepts of *drive* and *homeostasis* (Berridge, 2004; Rothermund & Eder, 2011). According to these theoretical

accounts, unspecific internal states of tension (drives) push an organism to engage in behaviours that results in the reduction of this tension (drive reduction), thereby restoring a state of physiological equilibrium that has survival value. Drives are thought to arise from specific, biologically determined needs (physiological depletion states) and are perceived as aversive by an organism, whereas drive reduction is associated with satisfaction and pleasure. Learning theorists soon recognized the usefulness of the drive concept as an intervening "organism" variable in S-R explanations of behaviour (e.g., Hull, 1943; Tolman, 1932). For instance, Hull's motivation theory (1943) proposed that frequency and intensity of a behavioural response to a specific stimulus depend on both the association strength and the drive strength. In later versions of the model, the concept of incentive expectations, i.e., learned expectations of hedonic reward, was additionally incorporated as a motivational component (Hull, 1952). Hull assumed that drives only have an unspecific energizing function, whereas the learned S-R association determines the direction of behaviour. However, some kind of motivation is still necessary for RL to occur. Thorndike (1913) already introduced the concept of "preparedness" to explain the observation that food does not support the establishment of new S-R associations, unless the animal has been deprived of nutrition. As I have pointed out above, learned S-O expectations play a critical role in motivating instrumental behaviour. Modern concepts of incentive motivation suggest that physiological depletion states specifically increase the incentive value of the corresponding rewards and reward-predicting stimuli (e.g., Toates, 1986).

Incentive motivation is closely related to the concept of valence that can be traced back to Lewin's Field Theory of Learning (Lewin, 1942). According to Lewin, behaviour is a function of both the person and the environment in which learning takes place. Objects in the environment gain affordance character - or positive valence - if they have the potential to satisfy psychological needs. Conversely, elements of the environment can also repel an individual and hence have negative valence. This dichotomy roughly corresponds to the fundamental distinction of approach vs. avoidance motivation thought to underlie adaptive decisions (Elliott, 2008). The concepts of valence and expectancy have been integrated in expectancy-value theories of motivation (Atkinson, 1957; Eccles et al., 1983; Wigfield, 1994). A common denominator of these cognitive-rational accounts is the assumption that the

resulting behavioural tendency is a function of (1) the value an individual assigns to the predicted outcome of an action, including the perceived instrumentality of an action to attain current and future goals, and (2) the subjective degree of confidence that the action will actually lead to the predicted outcome. These and similar ideas highlight the close relation between contemporary theories of motivation and goal-directed, model-based learning.

Emotions as Dispositions for Actions

Several attempts have been made to distinguish emotions from other affective [6] phenomena, such as feelings, mood, and affect dispositions (Rothermund & Eder, 2011; Scherer, 2005). Central to these efforts are so-called 'component-process' definitions that conceptualize emotions as multi-dimensional constructs (e.g., Scherer, 2005). Specifically, it has been suggested that emotion episodes include (1) a cognitive component (appraisal), (2) a neurophysiological component (body states), (3) a motivational component (behavioural tendencies), (4) a motor expression component, and (5) a subjective feeling component (emotional experience) (Scherer, 2005). According to this view, feelings constitute only a specific subcomponent of emotions, whereas moods refer to rather diffuse and enduring affective states that cannot be directly associated with specific appraisals and usually affect cognition rather than action (e.g. Davidson, 1994). Affect disposition denotes an individual's tendency to react with certain emotions to an eliciting event or to preferentially experience particular kinds of moods, as will be discussed in more detail below.

The cognitive component of emotions refers directly to the information they convey about the specific value that is assigned to any object, including states and actions (Clore & Huntsinger, 2007). Hence, it signifies the critical role of emotions in guiding instrumental behaviours. Moreover, affective states have been proposed to promote specific modes of information processing (Clore & Huntsinger, 2007; Gray, 2004). For instance, the 'affect-as-information'-hypothesis posits that positive affect signals the absence of threat and hence results in more heuristic or global processing, whereas negative affect promotes more careful, analytic processing (Clore & Huntsinger, 2007; Mitchell & Philipps, 2007).

Importantly, the component-process definition implies striking similarities of emotion and motivation at a functional level. Adopting a phylogenetic perspective, Lang and Bradley (2008) described emotions as "survival-based dispositions to actions". According to this, emotions are elicited by the activation of appetitive motivational circuits, promoting positive affect, and defensive motivational circuits, promoting negative affect. These motivational systems have been hypothesized to directly enhance attention and perceptual processing as well as influence motor output systems. This becomes particularly evident in the automatic engagement and/or modulation of somatic and autonomic reflexes, such as orienting and startle reflexes, upon the presentation of rewarding or threatening stimuli in both animals and humans (e.g., Bradley et al., 2001; Lang et al., 1997). In complex organisms, the adaptive value of emotion-mediated automatic evaluation is complemented by more deliberate, rational processing (Frank, Cohen, & Sanfey, 2009; Miller & Cohen, 2001).

Specifically, it has been suggested that emotion and motivation are integrated with cognitive control processes so as to bias information processing and behaviour selection according to specific situational demands (Gray, 2004; Pessoa, 2009). Affective and motivational significance are thought to influence competition between limited processing resources and to arbitrate between multiple conflicting modes of information processing and response tendencies, i.e., to resolve control dilemmas. This may be reflected in prioritized processing of emotional stimuli (see also Vuilleumier, 2005) and enhanced engagement of effortful control mechanisms, e.g., leading to improved error or conflict detection and resolution. However, task-irrelevant emotional distractors typically have a negative effect on task performance (e.g., Blair et al., 2007; Verbruggen & De Houwer, 2007). Task-irrelevant affect-related processing is thought to consume limited resources, resulting in a reduction in available cognitive capacity (e.g., Seibert & Ellis, 1991). Moreover, intense emotional reactions prompt down-regulatory mechanisms that are thought to strongly rely on brain areas associated with cognitive control functions (Gross & Thompson, 2007; Ochsner & Gross, 2005), which may result in further depletion of shared resources (Inzlicht & Gutsell, 2007). Motivational and affective significance can hence either improve or impair behavioural

[6] The term 'affect' is used to denote several states involving relatively rapid valence appraisals and thus share

performance, depending on several factors, such as task-relevance and intensity of affective-motivational content (Pessoa, 2009; Pessoa & Engelmann, 2010).

Personality Traits Related to Motivation and Emotion

Dispositional concepts of motivation and emotion have been considered useful in explaining interindividual variability in behaviour in the face of identical external stimuli (Berridge, 2004; Heckhausen & Heckhausen, 2006). Motivation and affect dispositions (or traits) refer to the relatively stable tendency of an individual to show particular motivations and emotions in particular situations (Asendorpf, 2007; Heckhausen & Heckhausen, 2006). In the following sections I will discuss the neurophysiological underpinnings of approach and avoidance (or withdrawal) that do not only regulate appetitive and aversive motivation, but also form the substrate of human personality (for review, see Carver, Sutton, & Scheier, 2000).

The Reinforcement Sensitivity Theory of J.A. Gray: A Framework for Individual Differences in Reinforcement Processes

In an influential account, Gray (1972) suggested that traits reflect interindividual differences in two complementary motivational systems: sensitivity to rewards and punishments (for similar proposals, see Cloninger, 1987; Davidson, 1998; Depue & Colllins, 1999; Fowles, 1980). Gray's Reinforcement Sensitivity Theory (RST; Gray, 1982) originally proposed that the so-called behavioural approach system (BAS) is activated by conditioned appetitive stimuli, i.e., signals of reward or omission/termination of punishment, and promotes reward-directed approach behaviour. By comparison, sensitivity to punishment is mediated by two behavioural systems. First, the behavioural inhibition system (BIS) is activated by conditioned aversive stimuli, i.e., signals of punishment or non-reward, as well as extreme novelty, and high-intensity stimuli. It thus supports the inhibition of on-going behaviour in the service of avoidance or extinction. Second, the fight-flight system (FFS) is activated by unconditioned aversive stimuli and facilitates defensive behaviours. According to

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certain (e.g. attentional) processes (cf. Scherer, 1984).

Gray (1982), there are several behavioural systems underlying reactivity to different classes of unconditioned appetitive stimuli. Crucially, the RST states that the BAS is related to trait impulsivity and gives rise to state positive affect, whereas the BIS is associated with trait anxiety and elicits fear (Gray, 1990). Moreover, activation of the BIS is thought to result in increased arousal and enhanced attention and information processing. Activity in the FFS is thought to underlie the trait dimension of psychoticism (Eysenck & Eysenck, 1976), and to trigger state negative affect as well as panic and rage[7].

Gray conceptualized BAS, FFS, and BIS as fundamental emotion systems to be specified at the behavioural, neural, and computational (or "cognitive") level (e.g., Gray, 1990). Although Gray did not fully detail the neural substrate of the BAS, he strongly linked it to activity in the nigrostriatal and mesolimbic DA system as well as the cortico-striatal loops (Gray, 1987). Specifically, he suggested that the ventral (limbic) striatum – with major inputs from amygdala and the hippocampal formation – mediates incentive motivation, whereas the dorsal (sensorimotor) striatum is concerned with more specific sensorimotor aspects of behaviour. The BIS has been hypothesized to include the septo-hippocampal system, including its projections to the frontal lobe and its afferents from the brainstem. Importantly, the ACC is thought to be a core component of the BIS as well. According to RST, the PFC maintains overarching plans and goals and exerts top-down control on both BAS and BIS. The neural basis of the FFS remains mostly unknown. Candidate regions include the medial hypothalamus and the periaqueductal grey, a midbrain region that has been implicated in the modulation of defensive behaviour (Watt, 2000).

Empirical tests of the RST provided only partial support for some of its basic claims. For instance, Gallagher & Hall (1992) compared the performance of participants who were particularly sensitive to reward or punishment cues in a proof-reading task under either reward (potential gain) or punishment (potential loss) conditions. Consistent with the predictions of the RST, punishment-sensitive individuals identified significantly more errors

[7] In a revised version of the RST, Gray & McNaughton (2000) hypothesized that the BAS is sensitive to all classes of appetitive stimuli (conditioned and unconditioned). Moreover, the revised RST postulated a fight-flight-freeze system (FFFS), mediating responses to all classes of aversive stimuli, whereas the BIS is thought to solve

than did reward-sensitive individuals in the punishment condition. Contrary to the predictions of the RST, however, both groups showed comparable performance in the reward condition. Moreover, the authors found no evidence that reward- and punishment sensitive participants experienced different emotional reactions in the two incentive conditions.

Other studies confirmed a higher emotional reactivity of punishment-sensitive individuals to aversive stimuli (e.g., Corr et al., 1995). Available evidence on reward-mediated emotional reactivity is similarly inconclusive (for a review, see Corr, 2004).

In particular, Gray's model implies that high sensitivity to aversive stimuli should lead to enhanced learning from punishments. In line with this notion, it has been shown that high punishment sensitivity improved implicit learning in a punishment compared to a control condition. In contrast, low punishment sensitivity was associated with impaired learning under punishment and improved learning in the control condition (Corr, Pickering, & Gray, 1997). Still, less consistent findings have been reported in studies using explicit learning tasks (e.g., Zinbarg & Revelle, 1989). Interestingly, a recent study found that individuals who were highly sensitive to punishment showed improved probabilistic learning from negative feedback (punishment learning) and impaired probabilistic learning from positive feedback (reward learning) when under social evaluative stress compared to a neutral condition (Cavanagh, Frank, & Allen, 2011a). By contrast, in less punishment sensitive individuals, stress was associated with impaired punishment learning and improved reward learning. Moreover, stress-induced negative affect reliably predicted superior punishment learning performance in participants characterized by high punishment sensitivity, but poor punishment learning performance in less sensitive participants.

Thus, the theoretical conceptualization of the BIS has received comparatively more empirical support, whereas findings concerning the BAS remain controversial. Nonetheless, the RST has proven a useful framework for exploring the neurophysiological basis of personality.

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conflicts between BAS and FFFS. Accordingly, the FFFS is associated with fear, whereas the BIS is associated with anxiety as well as worry and rumination.

Trait-Level Positive and Negative Affect

The RST defines BIS and BAS as emotion systems underlying both aversive vs. appetitive behaviour and negative vs. positive affective states. Indeed, many theoretical models of personality structure involve the basic trait dimensions of positive and negative emotionality. Most notably, Watson and Tellegen (1985) identified two orthogonal dimensions in factor analytic studies of self-rated mood: Positive Affect (PA) refers to the cross-situationally consistent tendency to experience pleasant affective states, such as pride, delight, and enjoyment. Whereas high PA is associated with activity and optimistic engagement, low PA is characterized by sadness and lack of energy. By comparison, negative affect (NA) denotes the extent to which individuals are prone to experience various unpleasant affective states, such as anger, disgust, guilt and fear. Accordingly, high NA related to distress and aversively motivated engagement, while low NA was associated with calmness (Watson, Clark, & Tellegen, 1988).

Consistent with the proposed link to reward and punishment sensitivity, measures of PA and NA correlated moderately with measures of BAS and BIS, respectively (Heubeck, Wilkinson, & Cologon, 1998). On the basis of those findings, it has been proposed that the neural substrate of BAS and BIS might also underlie PA and NA (e.g., Carver, Sutton, & Scheier, 2000). Furthermore, NA predicted measures of state anxiety, depression, and general psychological distress. In contrast, PA was inversely related to these variables (Watson, Clark, & Tellegen, 1988). Similarly, intra-individual variations in perceived stress were related to fluctuations in NA but not PA, whereas intra-individual variations in social activity were primarily associated with fluctuations in PA (Watson, 1988).

Importantly, individuals differed not only in the propensity to experience positive and negative affect, but also in the ability to *regulate* these affective responses (Gross & Thompson, 2007). As I have outlined above, affective reactions can interfere with task-relevant processing. A prominent example is the detrimental effect of uncontrollable failure experiences on subsequent instrumental learning (Mikulincer, 1994; Seligman, 1975; Wortman & Brehm, 1975). Notably, the ability to cope with failure-induced negative affect

(Kuhl, 1981) has been shown to moderate the consequences of failure on learning performance. This finding will be discussed in more detail below.

Uncontrollable Failure Experiences and Learning – A Special Case of a Deficit in Motivation or Affect Regulation?

How failure experiences affect subsequent performance has been a subject of intense debate in psychological research for several decades (Brunstein & Gollwitzer, 1996). Much of this work has been inspired by the learned helplessness theory (Seligman, 1975), which focuses on deficits in learning performance after the exposure to uncontrollable failure. According to helplessness researchers (e.g., Mikulincer, 1994; Seligman, 1975), the repetitive experience of uncontrollable aversive stimuli, such as inescapable painful stimuli or repeated failure feedback, results in a motivational deficit that is associated with impaired task performance, and generalizes to new tasks and environments (Hiroto & Seligman, 1975; Maier & Watkins, 2005; Roth & Kubal, 1975). Moreover, uncontrollable failure is typically followed by a heightened state of negative affect that is typically associated with reduced reward responsivity or 'anhedonia' (Bogdan & Pizzagalli, 2006; Henn & Vollmayer, 2005). The learned-helplessness effect is thus assumed to involve a cognitive, motivational and affective component (Pryce et al., 2011).

The notion that a motivational deficit underlies the cognitive effects of uncontrollable failure has received considerable support from animal research. These studies showed that the exposure to inescapable electric shocks appeared to specifically reduce the amount of effort rats were willing to exert in an instrumental learning task with high response demands, while memory acquisition and retrieval in a spatial learning task were not affected (e.g. Vollmayer et al., 2004). Moreover, this view is consistent with expectancy-value theories (see above) that predict a failure-related decrease in motivation as a consequence of reduced outcome expectancies. A purely 'motivational helplessness' hypothesis of failure experiences, however, has been challenged: (1) Trait and state variations in action- vs. state orientation (i.e., the capacity to regulate the affective reaction to failure, see below) moderated the effects of failure on subsequent task performance (e.g., Kuhl, 1981) and (2)

failure may also result in increased motivation, particularly in initial attempts to cope with the aversive event (e.g., Wortmann & Brehm, 1975). Furthermore, several findings point to the importance of self-relevance in determining the effects of failure on subsequent task performance (Brunstein, 2000). Brunstein and Gollwitzer (1996) demonstrated that participants exposed to failure feedback on a task critical for their self-definition subsequently showed improved performance on another identity-relevant task but impaired performance on an unrelated task.

The concepts of action- vs. state orientation refer to individual differences in the ability to control affect and thought in the service of goal-directed behaviour, especially under high cognitive demands (Kuhl, 1994). State-orientation is characterized by unintended rumination, prolonged preoccupation with the aversive event, and hesitation. In a state-oriented mode, individuals hence are unlikely to change their current mental state and to implement action plans. Action orientation, in contrast, refers to efficient affect-regulation, resulting in an improved detachment from thoughts about the aversive event and a focus on task execution and goal implementation. Thus, highly action-oriented individuals are expected to show superior task performance following failure experiences or changes in task settings. Accordingly, Kuhl (1981) showed that failure-related performance decrements can primarily be attributed to state-oriented cognitions. Participants exposed to an unsolvable cognitive task reported reduced outcome expectations regarding the unsolvable training task - but not an unrelated test task. Nonetheless, the experimental induction of state orientation after the failure-experience resulted in performance deficits on a subsequent concentration test (d-2; Brickenkamp, 1962). Likewise, participants characterized by a disposition for state orientation performed worse after failure than action-oriented participants. Based on these findings, Kuhl (1981) concluded that valence and outcome expectancy – as proposed by expectancy-value theories of motivation – cannot fully predict the consequences of failure experiences. Instead, he argued that the dimensions of action- vs. state orientation have to be considered as well.

Furthermore, recent findings from studies examining the effects of social-evaluative stress on instrumental learning indicated that stress had an impact on learning strategies rather than on overall accuracy (Petzold, Plessow, Goschke, & Kirschbaum, 2010; Schwabe

et al., 2007; Schwabe & Wolf, 2009). Interestingly, stress appeared to promote habitual (S-R) learning in detriment of (a) more explicit hippocampus-dependent spatial learning strategies (Schwabe et al., 2007) and (b) goal-directed instrumental learning (Schwabe & Wolf, 2009). Interestingly, in the latter study, the stress-related strategy effect was also reflected in reduced explicit knowledge about the S-R mappings (Schwabe & Wolf, 2009). Indeed, it has been suggested that stress selectively impairs explicit learning and memory systems, while implicit, habitual learning is not affected (Schwabe & Wolf, 2011). It should be noted, however, that mild stress has also been shown to improve explicit learning, presumably mediated by moderately increased arousal (Roozendaal et al., 2009; Wolf, 2009). Moreover, Petzold and colleagues (2010) reported that stress exposure selectively lessened the ability to efficiently use negative feedback during subsequent feedback-based learning. The authors suggested that stress might induce an attentional bias towards positive and away from negative, threatening information. Yet, findings by Cavanagh and colleagues (2011a) strongly suggested that individual differences in punishment sensitivity determine whether acute stress results in impaired vs. improved learning from negative feedback. In this study, only the participants reporting low punishment sensitivity showed stress-induced impairments in punishment learning, while the opposite pattern was observed for highly sensitive participants. Given that both failure experiences and social-evaluative stress are thought to elicit strong negative affect, their effects on learning may bear some similarities.

In sum, the induction of failure is associated with complex changes in cognitive, affective and motivational processes. Although the specific mechanisms underlying the impact of failure experiences on subsequent task performance are not entirely clear, most researchers agree that failure outcomes are experienced aversively and trigger negative affective states that individuals have to cope with. Moreover, acute failure feedback has been shown to promote reactive engagement in a subsequent task, possibly reflecting compensatory efforts.

Brain Mechanisms Underlying the Interaction of Motivation, Emotion, and Cognition

Traditionally, motivational and emotional processes have been distinguished from cognitive processes both on a functional and a neuroanatomical level. The amygdala and the PFC are two prominent examples for structures that have been associated with emotional and cognitive processing, respectively (LeDoux, 2000; Miller & Cohen, 2001; Ochsner & Gross, 2005). Considerable evidence, however, indicated that this modular view is no longer tenable. Previous research demonstrated a close interaction between brain regions supposedly involved in emotional and cognitive functions (for reviews, see Davidson, Pizzagalli, Nitschke, & Kalin, 2003; Pessoa, 2008; Phelps, 2006; Salzman & Fusi, 2010). Moreover, it has been suggested that emotional and cognitive parameters are integrated within dynamic brain networks, involving limbic and prefrontal structures (Pessoa, 2008; Salzman & Fusi, 2010). The next paragraph summarizes several critical findings this suggestion is based on.

The amygdala is structurally heterogeneous group of nuclei within the anterior medial temporal lobe. It has been described as a core affective region that is critically involved in fear conditioning (Antoniadis, Winslow, Davis, & Amaral, 2009; Pessoa, 2010), the representation of a reinforcer's value (Murray & Izquierdo, 2007), and the processing of emotional valence and intensity (LeDoux, 2007; Machado, Kazama, & Bachevalier, 2009). In particular, amygdala activation is related to vigilance and arousal, which lead some researchers to suggest that this region acts as a "detector" for the biological relevance of on-going events (Sander, Grafman, & Zalla, 2003). In line with this notion, the amygdala has been shown to support the prioritized processing of affectively significant stimuli (Phelps & LeDoux, 2005). Moreover, Salzman and Fusi (2010) posited that the amygdala might encode the value of the current state that is used to regulate the engagement of appetitive and aversive behavioural systems. In support of this view, Prevost and colleagues (2011) showed that the basolateral and centromedial complexes of the amygdala played important roles in instrumental reward and avoidance learning, respectively. Importantly, these findings demonstrated that the amygdala contributed not only to affective but also to cognitive functions, such as associative learning, attention, and decision making (Pessoa, 2010).

The representation of state value has also been associated with the OFC that is strongly interconnected with the amygdala. Indeed, there is ample evidence for a functional overlap between the two structures (Murray & Izquierdo, 2007). However, the OFC is not the only prefrontal subregion engaged in affective processing. Further regions include the ACC, the ventromedial prefrontal cortex (vmPFC) and parts of the inferior frontal gyrus, i.e., structures that are on the top of the hierarchy of the limbic cortico-basal ganglia network. Notably, emotion and cognition also appear to be integrated in regions that have been characterized as purely cognitive, such as the dlPFC (Pessoa, 2010). For instance, Savine and Braver (2010) showed that performance-contingent reward incentives increased behavioural efficiency during task-switching. Importantly, the reward-related performance modulations were associated with interactive incentive effects on switch-related activity in the dlPFC. The incentive manipulation also affected cue-related preparatory activity, which in turn predicted performance. These findings suggest that motivational influences on task performance are unlikely to reflect an unspecific "energizing" of behaviour, as would be indicated by "simple" additive effects of reward manipulations. Instead, motivation appears to sharpen and/or enhance task-specific processing in the service of behavioural optimization (cf. Pessoa & Engelmann, 2010).

Central to the integration of affective and motivational information are key nodes occupying a "hub-position" within the neural networks, i.e., regions on which inputs from multiple brain areas converge. The ACC has already been characterized as a suitable candidate for the implementation of this function. This was further corroborated by the observation that the recruitment of the ACC directly relates to task-engagement and effortful control (Boksem, Meijman, & Lorist, 2006a; Paus, Koski, Caramanos, & Westbury, 1998). Another potential mechanism for mediating affective and motivational influences on task performance is the neuromodulatory influence of DA. Drawing on the crucial role of DA in reward processing and approach motivation (Schultz, 2007), dopaminergic dynamics have also been hypothesized to underlie the impact of affective state on goal-directed behaviour and adaptive control (Aarts, Custers, & Veltkamp, 2008; Ashby, Isen, & Turken, 1998). However, there are several findings indicating that reward and positive affect exert dissociable effects on cognitive functioning (Chiew & Braver, 2011). The precise nature of

the relationships between affect, motivation, and dopaminergic activity thus remains to be determined.

The Neurophysiological Effects of Failure

In human neuroimaging studies, the exposure to uncontrollable, aversive stimuli has consistently been shown to engage the ACC (Pryce et al., 2011). In line with this, the ACC was typically found to be activated in response to acute stress (Dedovic, D'Aguiar, & Pruessner, 2009). Moreover, evidence from human and animal research suggested that multiple neurochemical systems are involved in the response to uncontrollable failure and other stressors (Joels & Baram, 2009; Maier & Watkins, 2005). First of all, stress exposure is associated with activation of the hypothalamus-pituitary adrenal (HPA) axis (de Kloet, Joëls, & Holsboer, 2005), resulting in an increased release of cortisol. Acute stress-induced enhancement of cortisol levels is accompanied by improved associative learning (e.g., fear conditioning) and declarative memory consolidation, particularly for emotionally arousing material (Roozendaal et al., 2009; Wolf, 2009).

These effects are most likely mediated by the regulatory influence of the amygdala on other brain regions implicated in associative learning and memory formation, such as the hippocampus. Specifically, cortisol has been suggested to facilitate the effects of noradrenaline that is released into the amygdala during aversive and stressful events (Roozendaal et al., 2009). Interestingly, there is also evidence indicating that stress hormones like cortisol might directly and indirectly affect dopaminergic activity and hence RL (e.g., Minton et al., 2009). In addition, the exposition to aversive events typically results in a short-lived increase of DA release in the ventral striatum (e.g., Cabib & Puglisi-Allgra, 1994).

Notably, the PFC, including the ACC, showed the highest density of stress hormone receptors (e.g., Pathel et al., 2000). Accumulating evidence indicated that the ACC might control the stress-induced activation of the HPA-axis (Herman et al., 2003) as well as the DA response in the ventral striatum (Pascucci et al., 2007). Specifically, Pascucci and colleagues showed that acute stress triggered a short-lived increase of DA and noradrenaline in the medial PFC of rats. Whereas prefrontal noradrenaline enhancement

determined the increase of DA release in the ventral striatum, continued mesocortical DA transmission due to sustained stressor exposition inhibited DA release in the ventral striatum. The opposing effects of prefrontal DA and noradrenaline on striatal DA are likely to reflect adaptive mechanisms supporting active coping or withdrawal as a function of perceived controllability of an event, which in turn is crucially dependent on the ACC (Amat et al., 2005).

Summary and Implications for the Present Study

Adaptive behavioural control is mediated by the interaction of cognitive mechanisms with motivational and emotional processes. The concepts of emotion and motivation are closely interrelated and both appear to govern RL by affecting information processing as well as instrumental behaviours. Interactions between motivation, emotion, and cognition can be integrated within dynamic brain networks involving limbic and prefrontal structures, particularly the ACC, the amygdala as well as multiple neurochemical systems. In line with this notion, the ACC is activated by aversive stimuli and appears to evaluate the controllability of stressors, thereby determining stress-related neurochemical responses, e.g. DA responses in the striatum. By comparison, the amygdala has been hypothesized to mediate prioritized processing of affectively significant stimuli and to regulate the engagement of appetitive and aversive behavioural systems. The amygdala is also believed to exert regulatory influence on other brain regions implicated in associative learning and memory formation, e.g. in response to stressors such as failure experiences and exposure to social-evaluative stress that have been shown to significantly impact RL mechanisms. Crucially, recent evidence suggests that stress-related variations in negative affect influence neural learning mechanisms (e.g. reflected in an enhanced sensitivity to errors) rather than overall performance (Cavanagh et al., 2011a; Petzold et al., 2010; Schwabe & Wolf, 2009). Importantly, the effects of stress- and failure-induced negative affective states on behavioural indices of learning appear to be modulated by interindividual differences in sensitivity to reward (BAS) and punishments (BIS) (Gray, 1972) as well as the disposition towards action vs. state orientation (Kuhl, 1981).

Electrophysiological Correlates of Reinforcement Learning

Studies measuring scalp recorded event-related potentials (ERPs) to examine neural processes underlying performance monitoring have greatly advanced our knowledge on how the brain implements RL. In particular, two components associated with the processing of response errors and performance feedback, respectively, have been hypothesized to track learning-related changes in the evaluation and utilization of information about action outcomes (e.g., Holroyd & Coles, 2002). First, the *error negativity* (Ne; Falkenstein et al., 1991) or error-related negativity (ERN; Gehring et al., 1993) has been linked to the activity of an internal error monitoring system. Second, a morphologically and functionally similar component can be observed in response to error-feedback stimuli and is commonly referred to as the *feedback-related negativity* (FRN; Miltner et al., 1997). Furthermore, the Ne is usually followed by a positive-going deflection, termed the *error positivity,* which is assumed to reflect error-related processes that are functionally dissociable from the Ne (Falkenstein, Hohnsbein, & Hoorman, 1990). In the subsequent sections, I will review a number of key experimental findings concerning the three ERP-components (Ne, FRN, Pe), with a specific focus on the neural underpinnings of learning.

The Error Negativity (Ne)

The Ne is a negative deflection in the ERP that starts shortly before an individual's erroneous response and peaks within 100 ms thereafter. The Ne has been observed in broad range of speeded-response tasks involving several stimulus and response modalities (for reviews, see Falkenstein, 2004; Falkenstein et al., 2000; Nieuwenhuis, Holroyd, Mol, & Coles, 2004). As can be seen in Figure 5, the scalp distribution of the Ne is maximal at fronto-central recording sites. Converging evidence from EEG source localizing, magnetoencephalographic, and fMRI studies in humans (for reviews, see Hester, Fassbender, & Garavan, 2004; Olvet & Hajcak; 2008; Ridderinkhof, Ullsperger, & Crone, 2004; Taylor, Stern, & Gehring, 2007) as well as from intracranial recordings in primates (Emeric et al., 2008; Ito, Stuphorn, Brown, & Schall, 2003) indicates that the Ne originates in the ACC. Although the source of the Ne is most consistently localized within the dorsal ACC,

considerable variation between different studies indicates that alternative and/or additional regions might contribute to Ne generation, most notably, the pre-supplementary motor area (pre-SMA) and the rostral ACC (Brazdil, Roman, Daniel, & Rektor, 2005; Luu, Tucker, Derryberry, Reed, & Poulson, 2003; Ridderinkhof et al., 2004). Given that the Ne has also been characterized as part of on-going theta and delta rhythms (Luu & Tucker, 2001; Trujillo & Allen, 2007; Yordanova, Falkenstein, Hohnsbein, & Kolev, 2004), it is interesting to note that error-related theta-band activity over the MFC predicted white matter connectivity with the ventral striatum, ventrolateral PFC, and motor cortex (Cohen, 2011). Available evidence thus suggests that a widespread network underlies action monitoring.

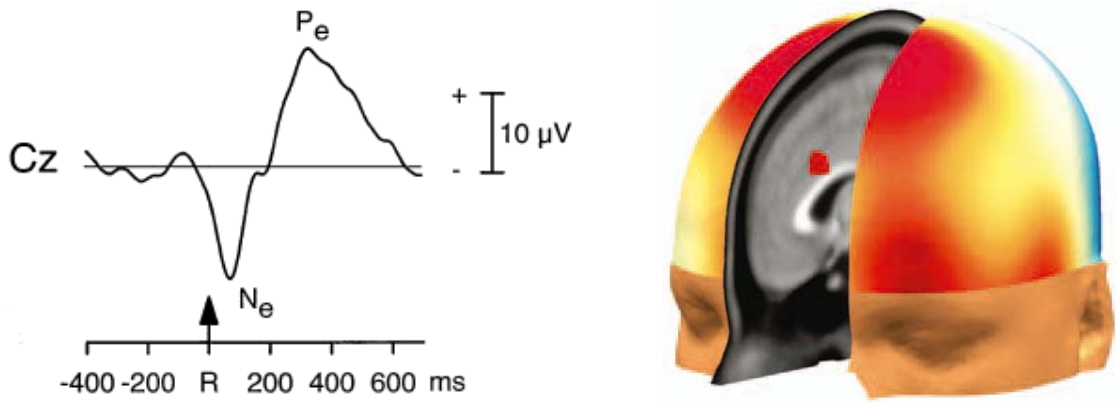


Figure 5: *Left*: The Ne peaks roughly within 100 ms after an individula's erroneous response and is typically followed by a positive-going deflection, termed the error positivity (Pe). *Right:* The topographical map demonstrates a fronto-central maximum of the Ne (red focus). The red diamant corresponds to the estimated source of the Ne in the dACC (dipole model). (Figures adapted from Falkenstein et al., 2000 and Taylor et al., 2007)

One of the earliest findings with respect to the Ne is its sensitivity to speed vs. accuracy instructions (Falkenstein et al., 1990; Gehring et al. 1993). Gehring and colleagues (1993) instructed participants to respond as quickly as possible (speed emphasis) or to make as few errors as possible (accuracy emphasis). The accuracy emphasis resulted in larger Ne amplitudes and more efficient post-error compensatory behaviour, including post-error slowing, and error correction rate. Although the comparison relied on error trials matched for response latency, the finding remains somewhat ambiguous because the speed-accuracy manipulation triggered a different weighting of what exactly is considered an error: slow

responses or wrong button presses. Moreover, the accuracy instruction by definition implies lower error rates. Studies examining the relation between accuracy and Ne amplitude have typically found larger Ne amplitudes when participants show better performance (e.g., Amodio, Jost, Master, & Yee, 2007; Hajcak, McDonald, & Simons, 2003; but see Falkenstein et al., 2000; Mathewson, Dywan, & Segalowitz, 2005). Interestingly, previous studies using probabilistic learning paradigms suggested that individual differences in accuracy related to ERPs on correct trials rather than the Ne (Eppinger et al., 2008; Eppinger, Mock, & Kray, 2009). Either way, one should consider performance differences when interpreting variations in Ne amplitude across experimental conditions (cf. Yeung, 2004).

An association between Ne magnitude and overall task performance would be consistent with the idea that the Ne reflects the activity of an internal performance monitoring system. More specifically, one might expect a direct link between Ne magnitude and error-related behavioural adjustments (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Gehring et al., 1993; Holroyd & Coles, 2002). Although this issue has been addressed by several studies, no consistent picture has emerged so far. Most reports focused on post-error slowing, i.e., strategic adjustments in reaction time (RT) on trials following an error, thought to reflect the increased recruitment of cognitive control processes (Botvinick et al., 2001). Using a single-trial measure of the Ne, Debener and colleagues (2005) demonstrated a significant relation between the Ne amplitude and the amount of post-error slowing (see also Gehring et al., 1993). However, a number of further studies – mostly using cross-trial averaging to quantify the Ne – failed to observe this kind of association (e.g., Dudschig & Jentzsch; 2009; Gehring & Fencsik, 2001), suggesting that more fine-grained analyses at a single-trial level might be critical. Supporting this notion, Cavanagh and colleagues (2009) showed that single-trial dynamics in error-related theta-power over the posterior MFC reliably predicted post-error slowing. Alternatively, it has been suggested that post-error RT changes might reflect processes other than control, such as sustained error processing interfering with stimulus-related processing on the next trial (cf. Gehring et al., in press). Research that aimed to establish a relation between Ne and immediate error correction has yielded similarly inconclusive findings (e.g., Burle et al., 2008; Falkenstein et al., 1996; Fiehler et al., 2005; Gehring et al., 1993).

Surprisingly few studies have examined whether the Ne relates to error-related behavioural adaptation in RL. Findings by Frank and colleagues (2005) point to a link between inter-individual differences in the Ne amplitude and a bias to avoid responses that have been learned to result in unfavourable outcomes (see also Cavanagh et al., 2011a; Frank et al., 2007b). Intriguingly, a recent study showed that state-level negative affect and inter-individual differences in punishment sensitivity modulated the tendency to learn more from errors or to correct choices as well as error-related neural processing (Cavanagh et al., 2011a). This is in line with ample evidence for affective and motivational influences on action monitoring reflected in the Ne, as detailed in one of the following sections.

The Feedback-Related Negativity (FRN)

While the Ne occurs around the time of an erroneous response, the FRN is a negative deflection elicited ~250-300 ms following the presentation of a feedback stimulus in a wide variety of experimental paradigms (Gehring & Willoughby, 2002; Miltner et al., 1997; for a review, see Nieuwenhuis, Holroyd, Mol, & Coles, 2004) (see Figure 6). As noted by Miltner and coworkers (1997), the FRN shares several characteristics with the response-locked Ne, which led some researchers to conclude that they might reflect the same cognitive and neural process (Holroyd & Coles, 2002; Nieuwenhuis, Yeung, Holroyd, Schurger, & Cohen, 2004).

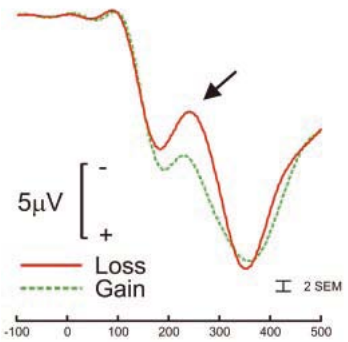


Figure 6: *Left:* The FRN can be oberserved as a relatively more negative-going deflection ~250-300 ms following the presentation of a feedback stimulus *Right:* Topographical map and estimated source in the dACC (dipole model). (Figure adapted from Gehring & Willoughby, 2002)

Similar to the Ne, the FRN has a fronto-central scalp distribution, suggesting that neural sources of the two components overlap. Indeed, the ACC and adjacent MFC have been identified as most likely generators of the FRN (Doñamayor et al., 2011; Holroyd et al., 2004; Ruchsow, Grothe, Spitzer, & Kiefer, 2002), along with the posterior cingulate cortex (Müller et al., 2005; Nieuwenhuis et al., 2005) and the right frontal cortex (Christie & Tata, 2009).

Several studies showed that the FRN is more pronounced after negative compared to positive feedback, indicating the component is sensitive to the valence of an outcome (e.g., Gehring & Willoughby, 2002; Nieuwenhuis, Yeung, et al., 2004; Sato et al., 2005; Yeung & Sanfey, 2004). Moreover, it has been suggested that the FRN may reflect a coarse evaluative mechanism that classifies on-going events in a binary manner as "good" or "bad" (e.g., Hajcak, Moser, Holroyd, & Simons, 2006; Sato et al., 2005; Toyomaki & Murohashi, 2005). For instance, Yeung and Sanfey (2004) applied a gambling task to examine whether the magnitude (small vs. large) of gains and losses affects the FRN amplitude. While the FRN differentiated only between gains and losses, independently of their magnitude, a later-occurring positive slow wave known as the P300 showed sensitivity to the magnitude but not the valence of outcomes. Although this finding has been confirmed by a number of other reports (e.g., Hajcak et al., 2006; Holroyd, Hajcak, & Larsen, 2006), there are also demonstrations that the FRN is sensitive to the magnitude of the outcome (e.g., Goyer, Woldorff, & Huettel, 2008; Kreussel et al., 2011).

In addition, there is evidence supporting of the notion that the evaluative process reflected in the FRN might operate in a context-dependent fashion (Holroyd, Larsen, & Cohen, 2004). When the task included a range of possible outcomes, the FRN appeared to track their relative rather than absolute value. Accordingly, in the Holroyd et al.'s study (2004), a zero outcome elicited a larger FRN if it was the worst possible outcome (i.e., in the context of potential gains), but a smaller FRN if it was the best possible outcome (i.e., in the context of potential losses). Furthermore, a study by Nieuwenhuis and colleagues (2004) revealed that monitoring processes associated with the FRN rely on the most salient information the feedback stimulus conveys. In this study, participants' choices on each trial in a gambling task either resulted in winning or losing 5 vs. 25 Cent. The authors showed that

the modulation of the FRN depended on whether colour coding emphasized the utilitarian (gain vs. loss) or performance aspect (correct vs. incorrect) of feedback information.

Interestingly, a recent study indicated that feedback salience can facilitate learning, particularly if the feedback was partially ambiguous (Herbert, Eppinger, & Kray, 2011). Although the relation between FRN and learning has rarely been directly tested, various findings point to a functional link between the FRN and the utilization of feedback information for behavioural adaptation (e.g., Cavanagh, Klein, Frank, & Allen, 2010a; Holroyd & Coles, 2008; Santesso et al., 2008). Most notably, van der Helden and colleagues (2010) demonstrated that the FRN amplitude predicted whether a mistake was repeated or learned from, implying that this component might reflect a PE. Further support for this notion comes from a study by Cohen and Ranganath (2007) showing that the FRN was larger on trials preceding behavioural switches, which is consistent with the magnitude of the negative PE derived from a computational RL model. A subsequent study that aimed to disentangle the PE-related mechanism and explicit rule-based decision-making only found an association between FRN and PE, whereas rule-based behavioural switches were related to P300 amplitude (Chase et al., 2011). On the basis of their findings, the authors conclude that the FRN reflects the activity of an incremental habitual (model-free) RL system, while the P300 relates to fast model-based RL mediating rule-based behavioural adjustments.

It is a matter of current debate whether the FRN represents PEs monotonically, combining magnitude and valence into a single scalar. Some reports suggested that the FRN varies as a function of expectancy deviation rather than feedback valence and hence represents the magnitude but not the direction of the PE (Chase et al., 2011; Oliveira, McDonald, & Goodman, 2007). Alternatively, variations in FRN amplitude have been characterized as positive deflections that scale with the magnitude of positive PEs (Foti, Weinberg, Dien, & Hajcak, 2011; Holroyd, Pakzad-Vaezi, Krigolson, & Krigolson, 2008). Furthermore, some researchers have argued against a monotonic representation of the PE in the FRN and instead proposed a sequential and spatially distributed processing of feedback information (Philastides, Biele, Vavatzanides, Kazzer, & Heekeren, 2010). According to this notion, an early ‘good-bad’ categorization is followed by a more fine-grained outcome evaluation that incorporates both PE valence and magnitude.

In summary, Ne and FRN are two morphologically and topographically similar ERP components thought to reflect the activity of a generic performance monitoring system. In particular, these components have been hypothesized to evaluate on-going events in the service of behavioural adaptation. Although a number of studies provided empirical support for a specific link between Ne and FRN and RL, this relation has rarely been addressed explicitly and so far remains under-explored. Importantly, the evaluative process underlying Ne and FRN appears to be sensitive to the specific context of behaviour. Indeed, several findings suggest that the two components reflect the motivational significance of response outcomes rather than their objective value, as detailed below.

The Error Positivity (Pe)

The Pe is a positive slow wave in the response-locked ERP that reaches its maximum between 200 and 400 ms after response-onset (Falkenstein et al., 1990; for a review, see Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; see Figure 6). The Pe is more pronounced after erroneous compared to correct responses and exhibits a centroparietal scalp distribution. Accumulating evidence, however, indicated that two dissociable subcomponents occur in the time-range of the Pe: (1) an early frontocentrally distributed subcomponent (early Pe) and (2) a later centroparietally distributed subcomponent (late or 'classic' Pe) (Arbel & Donchin, 2009; van Boxtel, van der Molen, & Jennings, 2005; van Veen & Carter, 2002). The early Pe may originate from brain regions in the MFC that are also thought to underlie the Ne generation, suggesting that this component is actually part of the oscillatory process underlying the Ne (Arbel & Donchin, 2009; Falkenstein et al., 1991; O'Connell et al., 2007). However, the early Pe shows a somewhat more central scalp distribution, possibly indicating that different albeit neighbouring regions of the ACC contribute to Ne and early Pe. By contrast, the late Pe[8] has been associated with two distinct neural sources in the rACC and in the vicinity of the posterior cingulate and the precuneus (O'Connell et al., 2007). The latter region has been implicated in consciousness, self-related processing, and the experience of agency (Cavanna & Trimble, 2006). This nicely fits the finding that the Pe – as opposed to

[8] The present thesis focuses exclusively on the late Pe.

the Ne – varies as a function of error awareness (Endrass, Reuter, & Kathmann, 2007; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; O'Connell et al., 2007). Likewise, the Pe is increased for cognitively or affectively more salient errors (Leutholt & Sommer, 1999; Stemmer, Witzke, & Schönle, 2001). Importantly, O'Connell and colleagues (2007) observed the early Pe for both aware and unaware errors, but the late Pe for aware errors only. These findings strongly indicate that the late Pe is associated with the conscious recognition of an error. This view is further corroborated by a recent study that demonstrated a strong link between the Pe and error detection (Steinhauser & Yeung, 2010).

Studies relating the Pe amplitude to task performance yielded inconsistent findings. Some investigators reported a smaller Pe for low-performing compared to high-performing participants (Dywan, Mathewson, & Segalowitz, 2004; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000) which has been considered evidence that this component reflects the affective appraisal of an error or its consequences. Other studies, however, did not find a relation between accuracy and Pe magnitude (Hajcak, McDonald, & Simons, 2003; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004). Mixed evidence has also been obtained regarding the association between Pe and post-error behavioural adjustments. For instance, Hajcak and coworkers (2003) found that larger Pe amplitudes predicted the degree of post-error slowing (see also Nieuwenhuis et al, 2001). Nevertheless, most studies failed to show an association between the Pe and immediate error-corrective behaviour (Falkenstein et al., 2000; Fiehler et al., 2005; Ullsperger & von Cramon, 2006). Hence, there is no clear-cut support for a 'behavioural-adaptation hypothesis' of the Pe. Nonetheless, the observed functional dissociations between Ne and (late) Pe suggest that a more slowly operating, deliberative performance monitoring system that is triggered by salient errors might underlie the Pe (cf. Overbeek et al., 2005).

In addition, several researchers have noted that the Pe bears strong resemblance to the stimulus-locked P300, which is thought to reflect the stimulus-inherent motivational or emotional significance (e.g., Keil et al., 2007; Nieuwenhuis, Aston-Jones, & Cohen, 2005; Yeung & Sanfey, 2004). According to this view, the Pe may indicate the updating of the representation of task-context in response to errors (Leutholt & Sommer, 1999) or the mobilization of resources for task-relevant processing in the service of immediate, i.e., within-

trial, remedial actions (Nieuwenhuis et al., 2005). The latter hypothesis is consistent with the observation that both the Pe and the P300 correlate with arousal (O'Connell et al. 2007).

Moreover, the Pe has been shown to increase with learning and feedback validity in a probabilistic RL task (Eppinger et al., 2009). In contrast, the Ne did not change over the course of learning, supporting the notion that Pe and Ne reflect dissociable processes. The finding that the Pe grows larger as participants are better able to represent the correctness of their responses is compatible with accounts linking this component to conscious aspects of error processing as well as context updating.

The Susceptibility of Error Negativity, Feedback-Related Negativity, and Error Positivity to Affective and Motivational Influences

Given that the consequences of an action can remarkably depend on the specific situation, one should expect the evaluative functions underlying Ne, FRN, and Pe to be sensitive to the motivational and affective significance of an on-going event. Much evidence in support of this notion comes from (1) studies with patients suffering from affective and other neuropsychiatric disorders that are characterized by negative emotionality and (2) studies investigating state and trait variations in affect and motivation.

Deviant Performance Monitoring and Learning in Neuropsychiatric Populations

Patients with Major Depressive Disorder (MDD) typically show increased sensitivity to performance errors and negative feedback (Eshel & Roiser, 2010; Steffens, Wagner, Levy, Horn, & Krishnan, 2001). Accordingly, increased Ne and FRN amplitudes have been observed for moderately depressed compared to healthy individuals, particularly when errors are penalized (Chiu & Deldin, 2007; Hajcak & Foti, 2009; Holmes & Pizzagalli, 2008; Santesso et al., 2008; Tucker et al., 2003; but see Ruchsow et al., 2004, 2006). Although such an increase on the level of neurophysiological correlates of error processing should be associated with more efficient error-related behavioural adaptation (Holroyd & Coles, 2002; Frank et al., 2005; van der Helden et al., 2010), depressed subjects typically show poor performance following mistakes in response competition task (Compton et al., 2008; Holmes

& Pizzagalli, 2007). However, two recent studies using probabilistic RL paradigms failed to obtain any performance impairments in depressed participants (Cavanagh, Bismark, Frank, & Allen, 2011b; Chase et al., 2010). Instead, in the Cavanagh et al.'s study, the association between error-related ACC activity (reflected in FRN amplitude and error-related theta power) and avoidance learning was stronger for MDD patients, suggesting that the ACC might mediate specific behavioural effects of increased affective reactivity to negatively valenced events in depression. Moreover, Holmes and Pizgalli (2008b) found increased error-related rACC and medial prefrontal cortex (mPFC) responses in patients with MDD compared to controls, but failure to subsequently recruit regions implementing control (dlPFC). In contrast, rACC, mPFC, and dlPFC activation was positively correlated in healthy participants, suggesting a dynamic interplay between these regions in adaptive behaviour regulation.

Studies examining the Pe in depressed patients yielded mixed results. While some researchers observed diminished Pe amplitudes in depressed individuals compared to healthy controls (Schrijvers et al., 2008, 2009), others failed to obtain significant differences (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008). As Schrijvers and colleagues (2009) point out, the divergent findings might be partly due to differences in symptom severity. Whereas increased levels of negative affect might account for enhanced Ne amplitudes in mild depression, this effect might be attenuated by extreme levels of anhedonia and apathy in more severely depressed patients, which in turn are characterized by decreased Pe amplitudes.

Hyperresponsivity to response errors and increased Ne amplitudes has also been found in patients suffering from obsessive compulsive disorder (OCD) (Endrass, Klawohn, Schuster, & Kathmann, 2008; Gehring, Himle, & Nisenson, 2000; Johannes et al., 2001). This finding is consistent with the notion that excessive concerns and repetitive behaviour of these individuals are associated with deviant action monitoring. However, recent evidence indicated that the OCD-related Ne modulation was task-specific, with non-patients suffering from high OC-symptomatology showing enhanced amplitudes in response competition tasks but not in RL tasks (Gründler, Cavanagh, Figueroa, Frank, & Allen, 2009). On the basis of this task-specific dissociation, the authors concluded that dissociable neural systems

underlie adaptive decision making in learning and speeded response competition tasks. Moreover, OC symptomatology selectively affected the Ne amplitude, whereas the FRN did not vary as a function of symptom score (Gründler et al., 2009). Likewise, no differences in Pe amplitude have been reported for OCD patients compared to controls (Endrass et al., 2008; Ruchsow et. al, 2005).

Despite some inconsistencies, the overall pattern of findings supports the notion that psychopathological changes in affective processing are accompanied by deviant action monitoring as reflected in Ne, FRN, and Pe. In particular, the observed dissociation between Ne and Pe suggests that the two components reflect different aspects of error processing.

Influence of Affective and Motivational States and Traits on Performance Monitoring

Consistent with the idea that healthy individuals who are particularly concerned with the correctness or social appropriateness of their actions should be characterized by a hyperactive performance monitoring system, the Ne amplitude has been shown to relate to trait differences in negative emotionality and anxiety (Dennis & Chen, 2009; Hajcak, McDonald, & Simons, 2003; 2004; Tops, Boksem, Wester, Lorist, & Meijman, 2006; Vocat, Pourtois, & Vuilleumier, 2008). Likewise, individuals with high levels of negative affectivity exhibited larger FRN amplitudes to negative – but not positive – feedback than those with low levels of negative affectivity (Santesso et al., 2011; Sato, Yasuda, & Ohiro, 2005). Studies focusing on the relation between negative affect and Pe are scarce. However, there is evidence indicating that higher negative affectivity was associated with decreased Pe amplitudes (Hajcak et al., 2004).

Interestingly, the Ne has been found to correlate with punishment sensitivity, whereas reward sensitivity correlates with the Pe (e.g., Boksem, Tops, Kostermans, & De Cremer, 2008; Boksem, Tops, Wester, Meijman, & Lorist, 2006a). In addition, the relation between punishment vs. reward sensitivity and Ne amplitude was modulated by the motivational context. Individuals that were highly sensitive to punishment showed larger Ne amplitudes on errors associated with losses compared to those associated with omissions of gain. In contrast, individuals that were highly sensitive to reward showed the opposite pattern.

Moreover, high reward sensitivity was related to greater Pe amplitudes in gain omission compared to loss conditions (Boksem et al., 2008). Similarly, highly punishment sensitive individuals showed larger FRN amplitudes in response to external performance feedback (Balconi & Crivelli, 2010; De Pascalis, Varriale, & D'Antuono, 2010) and monetary losses (Santesso, Dzyundzyak, & Segalowitz, 2011). It should be noted, however, that other studies have failed to obtain a difference in the Ne, FRN or Pe as a function of punishment or reward sensitivity (Cavanagh & Allen, 2008; Van den Berg, Franken, & Muris, 2011).

Drawing on previous research showing that the Ne was affected by mental fatigue (Boksem, Meijman, & Lorist, 2006b; Tops et al., 2006), Tops and Boksem (2010) recently proposed that the relation between Ne and personality measures, such as punishment sensitivity or negative affectivity, might be mediated by the motivational trait persistence. The authors demonstrated that high levels of constraint predicted less pronounced decreases in both behavioural measures and Ne amplitude during prolonged task performance, suggesting that trait-related differences in task engagement may underlie variations of the Ne amplitude.

An important drawback of studies using an individual differences approach is that they cannot exclude that non-affect related, a priori group differences account for the observed differences in error- and feedback-related ERP components. Therefore, it is important to test the degree to which experimental manipulations of affective and motivational states are accompanied by modulations of ERP-correlates of action monitoring and learning. Several studies indicated that the Ne was related to the salience or significance of an error. For instance, larger Ne amplitudes have been observed when accuracy is emphasized over speed (Falkenstein et al., 2000; Gehring et al., 1993). Furthermore, Hajcak and colleagues (2005) showed in two independent experiments that the Ne was increased on incorrect trials associated with high monetary value and under conditions of social evaluation. In the first experiment, the motivational significance of errors in a Flankers task was manipulated on a trial-to-trial basis by means of high vs. low monetary incentive cues. While high-value errors were associated with a greater Ne than low-value errors, participants showed comparable overall performance in both conditions. In the second experiment, participants were told that an experimenter would monitor and evaluate their performance.

Similar to the first study, the Ne amplitude was increased in the evaluation condition compared to a control condition, without any differences in overall performance. However, the design of the Hajcak et al.'s study did not differentiate between the consequences of error-related loss and error-related failure to gain. This distinction might be of special importance given that according to a commonly held view losses loom larger than gains ("loss aversion"; Kahneman & Tversky, 1979). Indeed, a recent report found larger Ne amplitudes for errors associated with monetary losses compared to errors associated with failure to obtain monetary rewards (Potts, 2011). Furthermore, Pailing and Segalowitz (2004) demonstrated that personality variables moderated the relation between motivational manipulations and Ne amplitude. In their study, high neuroticism scores predicted larger incentive-related modulations in Ne amplitude, whereas high conscientiousness predicted smaller incentive effects.

Studies that induced short-term positive and negative affect yielded mixed findings. In one report, pleasant, unpleasant, or neutral pictures were presented prior (700 ms) to each imperative stimulus in a flankers task (Wiswede, Münte, Goschke, & Rüsseler, 2009a). While the Ne amplitude was larger in the unpleasant than in the neutral condition, no difference was found between the pleasant and the neutral condition. In contrast, Larson and colleagues (2006) reported increased Ne amplitudes to flanker stimuli that were superimposed on pleasant pictures but no difference between neutral and unpleasant backgrounds. In a further study, Wiswede and coworkers (2009b) investigated how encouraging or derogatory feedback that was based on participants' reaction time influenced performance monitoring during a flanker task. The authors found larger Ne amplitudes in subjects that were provided derogatory feedback compared to encouraging feedback, whereas the Pe was unaffected by the feedback manipulation. However, a subsequent study using the same paradigm failed to obtain differences in Ne amplitude between the two feedback groups (Clayson, Clawson, & Larson, 2011), leading the authors to suggest that alterations in affective state do not influence action monitoring as reflected in the Ne. Yet, the results of the latter study raised some doubts regarding the effectiveness of the feedback manipulation in eliciting negative affect. First, the two groups reported equal levels of negative affect after the experiment, suggesting that the affect-induction was not effective.

Second, in contrast to the Wiswede et al.'s study, reaction times did not differ between the two feedback conditions, that is, participants in the derogatory-feedback group did not increased their response speed over the course of the task. Instead, they made more errors over time and hence performed worse than the encouraging-feedback group, making the interpretation of the null findings for the Ne (which has been shown to decrease with reduced accuracy) difficult. Clearly, further research is needed to clarify the relation between affective states and action monitoring indices. Importantly, the Pe was unaffected by the feedback manipulation in both studies.

Summary and Implications for the Present Study

ERP studies investigating the electrophysiological correlates of reinforcement learning have yielded important insights into the neural processes underlying error and feedback processing. In the context of the present study, three ERP components are of particular interest: The Ne and the FRN, assumed to reflect activity of a generic error-processing system (Falkenstein et al., 1990; Gehring et al., 1993; Holroyd & Coles, 2002; Miltner et al., 1997), and the Pe (error positivity), which has been associated with the conscious recognition and affective/motivational appraisal of errors. Of major importance for this study are demonstrations that the Ne (and potentially also the FRN and the Pe) is susceptible to affective and motivational influences, indicating that the significance of on-going events has a substantial impact on action monitoring. Yet, most of the studies cited above used response competition or gambling tasks to examine the influence of affective and motivational variables on error- and feedback-related ERP components. Reports of task-specific dissociations in Ne amplitude (e.g. Gründler et al., 2009) suggest that the described findings cannot readily be generalized to studies of RL.

Integrative Theoretical Accounts on Performance Monitoring

A number of theories have been put forward to account for the ERP-correlates of error- and feedback processing. Most theories primarily addressed the functional significance of the Ne. An intense debate has emerged as to whether the Ne reflects the detection of a

mismatch between the actual and the required response (Falkenstein et al., 1990; Gehring et al. 1993), conflict arising from simultaneous activation of multiple response tendencies (Yeung, et al., 2004), differences between expected and obtained action outcomes (Holroyd & Coles, 2002), or the affective and motivational evaluation of an error (Hajcak & Foti, 2008; Luu et al., 2003). Importantly, these accounts are neither mutually exclusive nor can one of them fully explain the majority of empirical data. However, two computational models have largely dominated action monitoring research during the last decade: the Reinforcement-Learning (R-L) theory (Holroyd & Coles, 2002) and the conflict monitoring theory (Botvinick et al., 2001).

The Reinforcement Learning Theory of Holroyd and Coles – An Integrative Theoretical Account on Error Processing and Learning

The reinforcement learning (R-L) theory (Holroyd & Coles, 2002) provides a computational model that accounts for both the Ne and the FRN by integrating the DA-RPE hypothesis with the action selection functions attributed to the ACC. The R-L theory conceptualizes the Ne and FRN in terms of negative RPE signals indicating that the outcome of an action is “worse than expected”. Accordingly, the model posits that the Ne is elicited on the basis of internal response representations, whereas the FRN is elicited by external feedback stimuli. The ACC was proposed to use these learning signals to update associations between states, actions, and outcomes. Hence, the model directly links the two ERP components to the acquisition and optimization of goal-directed behaviour.

Drawing on the work of Schultz and colleagues as well as on computational models of RL, Holroyd & Coles (2002) suggested an actor-critic architecture in which the TD PE is coded by the phasic activity of the mesencephalic DA system (see Figure 7). More specifically, the authors assumed that transient pauses in DA firing (i.e., negative PEs) disinhibit motor neurons in the ACC, leading to the generation of Ne and FRN. In accordance with previous research on the neural basis of RL (cf. Dayan & Niv, 2008; Maia, 2009; Niv & Montague, 2008), the R-L theory assigns the role of the adaptive critic to the basal ganglia. Additionally, the model suggests that multiple actors operate in parallel, including dlPFC,

OFC, and amygdala, each of which might use the PE to improve action selection. Importantly, the ACC is thought to act as a “supervisor” at the top of the hierarchy that uses the dopaminergic PE signal to arbitrate between the different motor controllers. In a way, the ACC thus learns a “controller-policy”, mapping states to actors, which in turn learn their own policies.

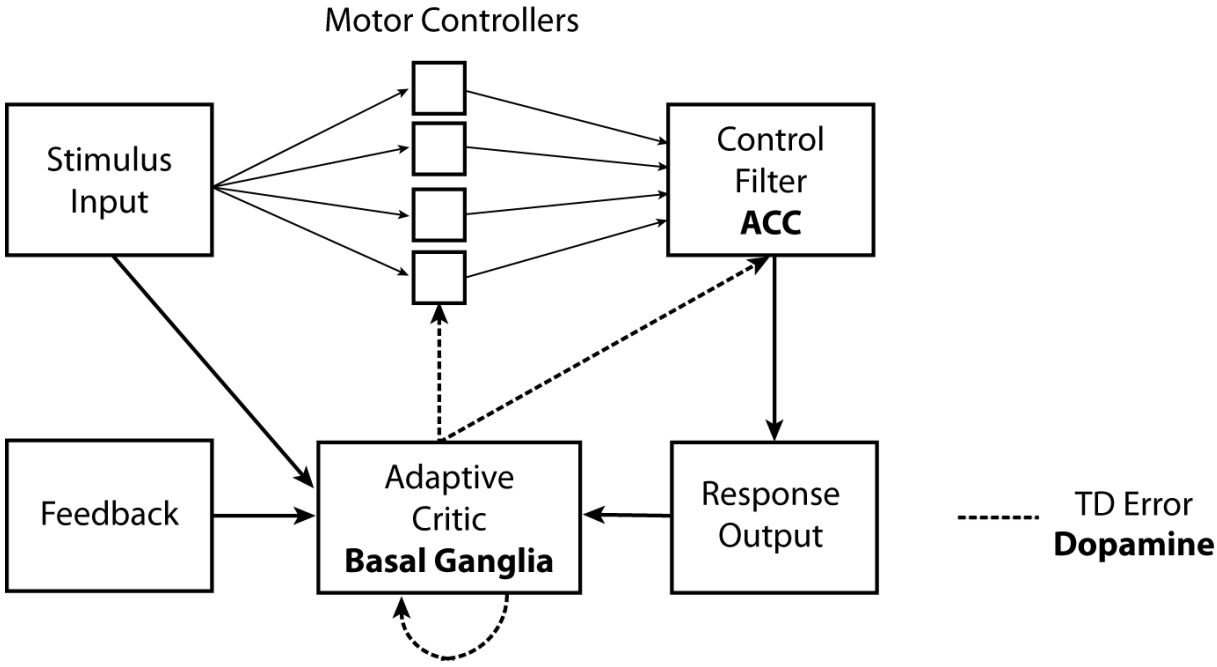


Figure 7: Schematic illustration of the R-L theory. (Figure adapted from Holroyd & Coles, 2002)

Given that feedback-based learning is accompanied by a transition from an external to an internal reference for action evaluation, a core prediction of the R-L theory concerns modulations of Ne and FRN over the course of learning. Specifically, the theory postulates that the Ne should increase with learning, reflecting the development of an internal representation of the correct response. In contrast, the FRN should decrease with learning, indicating reduced reliance on external feedback to determine the correctness of a response. Note that negative feedback after an erroneous response is fully expected once an individual has learned the response-outcome contingencies and hence does not result in a PE. Instead, the PE is computed around the time of the response, reflected in the Ne. Another important prediction following from the R-L theory is that larger PEs, i.e., larger Ne/FRN amplitudes, should be associated with a stronger tendency to subsequently avoid the same

maladaptive response. In the following section, I will review evidence on the central claim that both the Ne and the FRN are neural manifestations of a dopaminergic PE signal and hence indicators of a RL mechanism.

Evaluation of the Reinforcement-Learning Theory. Consistent with the proposed involvement of the DA system, the Ne is sensitive to changes in dopaminergic transmission. For instance, altered Ne amplitudes have been reported in neuropsychiatric disorders that are characterized by altered DA levels, including Parkinson's disease (Falkenstein, et al., 2001; Stemmer, Segalowitz, Dywan, Panisset, & Melmed, 2007; Willemsen, Müller, Schwarz, Hohnsbein, & Falkenstein, 2008) and Huntington's disease (Beste et al., 2007, 2008). Furthermore, the Ne amplitude is increased following the administration of a DA agonist compared to a placebo (de Bruijn et al., 2004), whereas DA antagonists lead to an attenuation of the Ne (de Bruijn et al., 2004; Zirnheld et al., 2004). Notably, a recent study reported impaired reward learning and more negative FRN amplitudes to reward feedback for participants receiving a DA agonist compared to a control group (Santesso et al., 2009). In contrast, the Pe is typically unaffected by modulations of the dopaminergic activity (Overbeek et al., 2005). Examining genetic polymorphisms of the COMT gene, which determines DA levels in the PFC, Frank and coworkers (2007b) found no difference in Ne amplitude between met/met and val/val carriers. Another study, however, reported larger Ne amplitudes for individuals homozygous for a certain allele of the DA D4 receptor gene that determines prefrontal receptor responsiveness to DA (Krämer et al., 2007).

Although these findings are mostly consistent with the R-L theory, substantial evidence indicates that other neurotransmitters, such as serotonin, noradrenaline, and GABA, are additionally involved in the generation of the Ne and the FRN (cf. Jocham & Ullsperger, 2009). Moreover, it has been argued that DA plays a different role in RL than originally proposed by Holroyd and Coles (2002). As was discussed in the context of the DA-RPE hypothesis, several researchers have pointed out that the effects of phasic DA activity in the PFC take seconds or even minutes to resolve and are therefore unlikely to fulfil the requirements of a PE signal and to underlie rapid electrophysiological responses such as Ne and FRN (Durstewitz & Seamans, 2002; Jocham & Ullsperger, 2009). Instead, it has been

suggested that the performance monitoring functions of the ACC might exert a top-down modulatory influence on the DA system (Frank et al., 2005; Jocham & Ullsperger, 2009).

If Ne and FRN are neural manifestations of PEs, they should reflect response-outcome contingency (reward probability) and valence. Convergent evidence indicates that both the Ne and the FRN are sensitive to feedback validity. Consistent with PEs derived from TD learning rules, learning conditions involving deterministic response-outcome contingencies (valid feedback) were associated with larger Ne and smaller FRN amplitudes than those involving probabilistic contingencies (partly invalid feedback) (Eppinger et al., 2008, 2009; Holroyd & Coles, 2002; Nieuwenhuis et al., 2002). Although the R-L theory does not explicitly specify to which degree expectancy and value are jointly represented by the FRN, the original proposal suggests a scalar value, i.e., the more unexpected *and* unfavourable the outcome, the more negative the amplitude. Unfortunately, the empirical evidence is still inconclusive. While some findings suggested that the FRN is sensitive to expectancy deviation rather than feedback valence (Chase et al., 2011; Oliveira, McDonald, & Goodman, 2007), others indicated that the FRN is unaffected by expectancy violations (Hajcak et al., 2007). Similarly, it remains unclear whether the feedback value is coded in a binary (Holroyd et al., 2004; 2006) or more graded fashion (Bellebaum et al., 2010; Goyer et al., 2008; Kreussel et al., 2011).

Furthermore, the R-L theory has been challenged by reports of dissociations between Ne and FRN. For instance, the Ne appears to be modulated by affective and motivational variables, whereas very few studies reported corresponding effects for the FRN. Crucially, studies that measured both the Ne and the FRN found distinctive influences of factors such as OC symptomatology (Gründler et al., 2009) and age (Eppinger et al., 2008). A related problem concerns learning-related changes of the two components. In line with the predictions of the R-L theory, a number of studies showed that the Ne increases with learning (Eppinger et al., 2008, 2009; Holroyd & Coles, 2002; Morris et al., 2008; Nieuwenhuis et al., 2002; Pietschmann et al., 2008). The pattern of findings for the FRN, however, is less consistent. Several studies failed to obtain the predicted decrease of the FRN over the course of learning (e.g., Cohen & Ranganath, 2007; Holroyd & Coles, 2002; Pietschmann et al., 2008). Importantly, Eppinger and colleagues (2008, 2009) showed

pronounced learning-related changes in the ERPs to correct feedback and responses rather than in the ERPs to incorrect feedback and errors. A similar result was reported by Cohen and Ranaganath (2007). On the basis of their findings, Eppinger and coworkers (2008) concluded that the so-called 'response/feedback-locked positivity' might reflect dopaminergic modulations of ACC activity, possibly associated with a positive PE. The idea of a reward-related positivity has also been put forward by Holroyd and coworkers (2008, 2011). Notably, a recent attempt to isolate to reward-related positivity by means of principal component analysis yielded promising results (Foti et al., 2011).

Surprisingly few studies have tested whether Ne and FRN magnitude predict behavioural adjustments during learning – as should be expected if the two components reflect a PE. Consistent with a RL framework, previous studies demonstrated that the magnitude of the FRN predicted strategic action selection (Cohen & Ranganath, 2007). Yet, in this study participants played a strategic game in which feedback was based on an algorithm that ensured equal distribution of wins and losses. Hence, there was no learnable response-outcome contingency. A subsequent study provided more compelling evidence for a link between FRN amplitude and learning-related behavioural changes (van der Helden, Boksem, & Blom, 2010). In a feedback-based sequence learning task, the FRN amplitude was more negative on incorrect trials that were corrected on the next encounter of the same item compared to those that were not corrected. Moreover, findings by Frank and coworkers point to a link between individual differences in Ne amplitude and a bias to avoid responses that have been learned to result in unfavourable outcomes (Cavanagh et al., 2011a; Frank et al., 2005, 2007b).

Alternative Accounts on the Error Negativity and Related ERP-Components

The Error/Mismatch Detection Theory. Early accounts have discussed the Ne in terms of an error detection mechanism (Coles et al., 2001; Falkenstein et al., 1991; Gehring et al., 1993). According to this view, the processing of the imperative stimulus activates a representation of the correct (or intended) response, which is compared with an efference copy of the outgoing motor command, i.e., the executed response. The Ne reflects an error signal that indicates a mismatch between the two representations. Most variants of the error

detection account agree on the assumption that the mechanism underlying the Ne gives rise to remedial actions such as immediate error correction. The error detection account directly implies that greater deviations of the actual response from the intended one should result in larger Ne amplitudes. Studies testing the effects of response and stimulus similarity on the Ne, however, yielded inconclusive results (e.g., Bernstein et al, 1995; Falkenstein et al., 1996; Gehring & Fencsik, 2001; Yeung et al., 2007). For instance, Falkenstein and coworkers (1996) reported larger Ne amplitudes for dissimilar response representations, whereas Gehring and Fencsik (2001) found larger Ne amplitudes for similar response representations. However, these studies defined response similarity only based on effector side, which might be an inappropriate or insufficient characterization on a conceptual level. Moreover, the effects of stimulus and response similarity were often confounded with performance differences, rendering definitive conclusions difficult.

The Conflict-Monitoring Theory. Another prominent computational approach to the Ne holds that this component reflects a monitoring process in the ACC that tracks the degree to which mutually incompatible responses are co-activated[9] (Botvinick et al., 2001; Yeung, Botvinick, & Cohen, 2004). According to the conflict monitoring theory, the Ne reflects post-error conflict due to the simultaneous activation of incorrect and correct responses, the latter arising from continued processing of the target stimulus after the erroneous response. The conflict signal is thought to be conveyed to the dlPFC which implements top-down control by recruiting task-set related attentional mechanisms in order to bias information processing. A central claim of the conflict monitoring account is that higher degrees of response conflict are associated with larger Ne amplitudes. However, empirical tests provided only limited (or no) support for this assertion (e.g., Burle et al., 2008; Carbonnell & Falkenstein, 2006; Yeung et al., 2004), possibly reflecting an inappropriate modelling of motor representations in these studies and/or the conflict monitoring theory itself (cf. Gehring et al., in press). In addition, the conflict monitoring theory refers to another medial frontal negativity: The N200, a stimulus-locked ERP-component that is typically larger on incompatible compared to compatible trials

[9] Response conflict is quantified as the Hopfield (1982) energy of simultaneously activated response units, i.e., the product of the activation of each unit, weighted by the strength of the inhibitory connections between the units.

in conflict tasks. Given that high response conflict does not necessarily result in errors, the N200 reflects pre-response conflict on correct trials. Indeed, there is evidence indicating that Ne and N200 bear strong resemblance both morphologically and functionally (Ferdinand, Mecklinger, & Kray, 2008; Folstein & Van Petten, 2008). However, dissociations of Ne and N200 have been reported as well (e.g., Davies et al., 2001; Ridderinkhof et al., 2002; but see Yeung & Cohen, 2006).

Of note, recent modelling work suggests that both conflict monitoring in the ACC and reinforcement learning in the BG can be integrated within a single model that accounts not only for the Ne but also for the FRN (Cockburn & Frank, 2011). This is of particular importance as the conflict monitoring theory presumes instructed task rules and does not explain how task-specific response mappings are acquired.

Affective/Motivational Theories. An important omission of the above-discussed models on ACC functioning is that they do not explicitly account for the susceptibility of the Ne (and potentially the FRN and Pe) to affective and motivational variables. Although those influences are not denied, a commonly held – but usually implicit notion – is that higher affective and motivational significance leads to an increased recruitment of cognitive control mechanisms (cf. Yeung, 2004). Considerable evidence suggested that the affective and motivational significance of on-going events has a substantial impact on action monitoring. However, the existing computational models do not specify how these effects are implemented in the brain.

Luu and colleagues (2003) addressed this issue by assuming that the Ne might reflect the joint activity of several structures in a broader corticolimbic circuit. In line with the segregationist view on ACC functioning (Bush et al., 2000), the authors stated that distinct subregions of the ACC differentially contribute to action regulation with the dorsal part (dACC) being more strongly implicated in cognitive aspects of action monitoring and the rostral part (rACC) primarily processing the affective significance of a response outcome (see also van Veen & Carter, 2002). Interestingly, Luu et al. (2003) found that both the dACC and the rACC contributed to the Ne, whereas only the dACC contributed to the FRN. Although the functional segregation of the ACC into a cognitive and an affective subdivision has been

seriously challenged (Shackman et al., 2011), a large number of studies pointed to a functional dissociation between rACC and dACC in error processing and learning (e.g., Cavanagh, Gründler, Frank, & Allen, 2010b; Simões-Franklin, Hester, Shpaner, Foxe, & Garavan, 2010; Taylor et al., 2006).

Furthermore, Boksem and coworkers (2006b) posited that there is a strong correspondence between the BIS (Gray, 1987) and the sensitivity of the Ne to mistakes, punishment, and negative affect. They further reasoned that the Ne might reflect aversively motivated, reactive engagement aimed at avoiding punishment. In line with this view, the Ne has been directly linked to defensive behaviours. Hajcak and Foti (2008) showed that the Ne amplitude predicted the potentiation of the startle reflex following error trials. On the basis of this finding, the authors reasoned that the Ne is associated with an error detection mechanism. The outcome of this mechanism serves to trigger defensive reactions in order to protect the organism against potential dangers. Moreover, Boksem and colleagues (2006a) found a correlation between the Pe and reward sensitivity BAS. From their findings, the authors concluded that high BIS scores and enhanced Ne amplitudes reflect a bias towards reactive control, denoting the tendency to recruit control processes after a (negative) event has occurred (Braver, Gray, & Burgess, 2007; Tops, Boksem, Luu, & Tucker, 2010). By contrast, high BAS scores and large Pe amplitudes are thought to indicate a bias towards proactive control, i.e., the tendency to engage in preparatory attention to maintain context and goal representations and to allocate resources in order to obtain rewards and to prevent negative events.

Integrative Accounts. Recently, several attempts have been made to integrate the empirical findings on the Ne and related components into a more comprehensive view on performance monitoring and adaptive control (Alexander & Brown, 2010; Taylor, Stern, & Gehring, 2007; Tops et al., 2010). The prediction of response-outcome (PRO) theory (Alexander & Brown, 2010) synthesizes core ideas and mechanisms of existing models. Similar to the R-L theory, action monitoring is conceptualized within a RL framework. However, the PRO model is thought to learn R-O associations rather than S-R or S-O associations. Specifically, the PRO-model involves two functional components, implemented

by the mPFC: the first component is trained by a RL mechanism to predict the potential outcomes of an action, whereas the second component compares predicted and actually obtained outcomes. The first component subsequently uses detected discrepancies to improve its own predictions. In contrast to the R-L theory, the PRO model states that the mPFC signals discrepancies, i.e., PEs, for both positive and negative outcomes and allows for encoding of multiple PEs. Moreover, the PRO model does not presume that the mPFC is trained by a dopaminergic PE signal.

Tops and coworkers (Tops et al., 2010; Tops & Boksem, 2011) provided an even more comprehensive and general scheme of behavioural systems underlying psychological processes. Drawing on the basic distinction between proactive and reactive behavioural programs (e.g., Braver, Gray, & Burgess, 2007; Koolhaas et al., 2007) the model proposes four distinct behavioural control systems: proactive approach, proactive avoidance, reactive approach, and reactive avoidance. Crucially, these systems are hypothesized to be under dopaminergic (reactive approach), cholinergic (reactive avoidance), noradrenergic (proactive approach), and serotonergic (proactive avoidance) neuromodulatory control. Moreover, the proactive and reactive systems are linked to a dorsomedial and ventrolateral cortical system, respectively. The authors reviewed evidence in support of the notion that proactive control is more adaptive in stable environments, in which behaviour is guided by context models whereas reactive control is more adaptive in unstable, rapidly changing environments that require fast, feedback-driven behavioural adjustments.

Summary and Implications for the Present Study

The R-L theory (Holroyd & Coles, 2002) provides a neurocomputational model of error processing that accounts both for the Ne and the FRN. It integrates the DA-RPE hypothesis with the action selection functions attributed to the ACC and implies a direct link between both ERP-components and learning-related behavioural adaptation. Specifically, it is assumed that negative PE signals are used by the ACC to update associations between states, actions, and outcomes. Other theories have also linked the Ne to remedial actions, but discussed the Ne in terms of a “mere” error/mismatch detection mechanism and a conflict

signal indicating the need for enhanced cognitive control. In addition, a few recent frameworks have aimed at accounting for the susceptibility of action monitoring to affective and motivational variables. However, these affective/motivational “theories” clearly lack the computational specificity of the R-L and conflict monitoring models, which in turn are more or less ignorant to the role of motivational and affective variables in performance monitoring. In particular, surprisingly little is known on how affect- and motivation-related modulations of ERP-correlates of performance monitoring relate to adaptive behavioural changes during RL.

3. Statement of Problem and Overview of Studies

Despite considerable differences in their conceptualization of the ERP-correlates of error and feedback processing, most theories on performance monitoring assert a functional link between the neural mechanisms underlying these components and flexible behavioural adjustments. Most notably, the R-L theory (Holroyd & Coles, 2002) states that Ne and FRN are neural manifestations of PEs, which train the ACC to select appropriate motor controllers, i.e., to learn adaptive response rules for the task at hand. Although this claim has not thoroughly been supported by the literature, previous studies showed that the FRN amplitude predicts subsequent strategic choices and (Cohen & Ranganath, 2005) and learning-related behavioural adjustments (van der Helden et al., 2010). In addition, the Ne amplitude relates to interindividual variations in a bias to avoid responses that have been learned to result in unfavourable outcomes (Frank et al., 2005).

At the same time, the evidence reviewed above strongly suggests that performance monitoring – as reflected in the Ne – is sensitive to the affective and motivational context of an action. The susceptibility of the FRN and the Pe to those influences is much less substantiated. Yet, recent findings indicate that highly punishment sensitive individuals are characterized by larger FRN amplitudes (Balconi & Crivelli, 2010; De Pascalis, Varriale, & D'Antuono, 2010; Santesso et al., 2011). So far, research focused almost exclusively on conflict and gambling paradigms to examine affective-motivational modulations of Ne and FRN, respectively. To my knowledge, only one prior study has explicitly tested how affective-manipulations influence the activity of medial prefrontal performance monitoring system during reinforcement learning. Cavanagh and colleagues (2011a) found that social-evaluative stress leads increased response-related mediofrontal EEG theta power but decreased theta power to negative feedback. Interestingly, the changes in mPFC activity were associated with relatively better punishment and worse reward learning in highly punishment sensitive individuals, but worse punishment learning and improved reward learning in less punishment sensitive individuals. These findings suggest that trait vulnerability to punishment significantly moderates the impact of stress-induced negative affect on action monitoring and learning.

Given that Ne, FRN, and Pe have been proposed to reflect neural signals that are used to guide subsequent behavioural adjustments, the main goals of this thesis were to determine (1) the degree to which manipulations of motivational-affective state result in modulations of the Ne, FRN, and Pe during reinforcement learning, (2) how these modulations are reflected in the ability to use error signals for learning-related behavioural adaptation, and whether the impact of the affective-motivational manipulations on the electrophysiological and behavioural indices of action monitoring changes (3) over the course of learning and (4) as a function of affect-related traits.

A further aim of this thesis was to address the on-going debate as to whether the Ne is an index of deviant error processing associated with trait – but not state – variations in negative affect (e.g., Clayson et al., 2011). Indeed, it has been suggested that variations in Ne amplitude might reflect an 'endophenotype' for internalizing and externalizing disorders, respectively (Olvet & Hajcak, 2008). Such a conclusion appears premature for two important reasons: First, thus far, only a very small number of studies used experimental manipulations to examine the impact of motivational and affective factors on performance-monitoring processes. Second, employing an individual difference approach, one cannot exclude that non-affect-related variables account for the observed differences in Ne amplitude. Moreover, it remains to be determined whether experimentally induced state variations in negative affect and trait level negative affect are accompanied by similar changes in the functioning of the action monitoring system.

These issues were addressed in three experiments. The *first experiment* was designed to investigate how an episode of self-relevant failure affects adaptive behavioural adjustments and ERP-correlates of performance monitoring during a subsequent feedback-based learning task. To this end, two phases (pre- and posttest) of a probabilistic learning task were applied. Between pre- and posttest, participants performed a visual search task that was described as diagnostic of intellectual abilities. In this task, participants were assigned to one of two conditions in which they received either failure feedback (failure-feedback-group) or no feedback (no-feedback-group).

Failure experiences are associated with prominent affective and motivational consequences. First of all, failure is an acute stressor that triggers negative affective states

(Westermann, Spies, Stahl, & Hesse, 1996). Crucially, recent evidence suggests that stress-related variations in negative affect influence learning strategy rather than overall performance (Cavanagh et al., 2011a; Petzold et al., 2010; Schwabe & Wolf, 2009). In particular, it has been shown that stress-manipulations affect the processing of response errors and negative feedback information. Moreover, personality traits such as punishment sensitivity might be important factors in mediating the specific behavioural consequences of these manipulations. The first experiment thus specifically aimed to determine (1) whether failure affects the reliance on internal vs. external cues for action evaluation during the time-course of learning, reflected in learning-related dynamics of Ne vs. FRN, (2) the degree to which failure promotes a bias towards enhanced processing of errors/negative feedback or correct responses/positive feedback, and (3) whether affect-related traits moderate the effects of failure.

Uncontrollable failure experiences also induce significant motivational changes (Brunstein, 2000; Kuhl, 1987; Seligmann, 1975). Previous research has demonstrated that self-relevance of the task and individual differences in action vs. state orientation are critical in determining whether failure leads to motivational deficits or reactive engagement. Importantly, the Ne has been shown to be sensitive to task engagement in that high persistence is associated with smaller reductions in Ne amplitude across the course of the task (Boksem et al., 2006a; Tops & Boksem, 2010). However, individual differences in negative affectivity appear to modulate the time course of motivational engagement during prolonged task performance. Luu and colleagues (2000) demonstrated that individuals characterized by high negative affectivity showed increased motivational engagement at the beginning of the task but impaired engagement during later stages of the task, reflected in initially enhanced but subsequently attenuated Ne amplitudes compared to individuals low in trait negative affect. Hence, motivational disengagement might disguise more specific consequences of failure on performance monitoring and learning (e.g., alterations in learning strategy).

In an attempt to disentangle the effects of failure induction and motivational disengagement due to prolonged task performance, two experimental conditions were realized in the first and the second experiment. In the first experiment, the posttest was

described as indicative of intellectual abilities to ensure high self-relevance. By contrast, in the *second experiment*, the posttest was described in neutral terms. In both experiments, pre-task questionnaires assessed individual differences in punishment sensitivity, trait negative and positive affect, and action vs. state orientation.

While Experiments 1 and 2 induced self-relevant failure to examine the consequences of short-term changes in affective and motivational state on performance monitoring and learning, the *third experiment* had a slightly different focus. Building up on previous demonstrations that the Ne tracks trial-by-trial variations in motivational significance (Hajcak et al., 2005; Potts, 2011), Experiment 3 aimed to determine whether manipulations of appetitive vs. aversive motivation by means of monetary incentives modulate error and feedback processing – as reflected in the Ne, FRN, and Pe – and how these modulations relate to behavioural adaptations during reinforcement learning. Given that a number of studies point to a functional dissociation between rACC and dACC in error processing (e.g., Cavanagh et al., 2010b; Simões-Franklin, et al., 2010, Taylor et al., 2006), a further goal was to explore whether the two cingulate subregions differentially contribute to error processing and error-related behavioural adjustments, depending on the motivational significance of an error.

II Empirical Part

4. Research Goals: Experiment 1 and 2

In Experiment 1 and 2, participants were exposed to self-relevant failure during a visual search task that was linked to participants' intellectual capability. The main goal of the two experiments was to examine the extent to which failure-induction influences the participants' ability to use error signals for behavioural adaptation in a subsequent feedback-based learning task and whether these differences in performance monitoring are reflected in modulations of the Ne, FRN, and Pe. In order to avoid that motivational disengagement due to prolonged task performance disguises the effect of failure-induction, different posttest instructions were given to manipulate participants' motivation. In Experiment 1, the posttest was linked to intelligence, whereas a neutral instruction was given in Experiment 2.

Previous research showed that the induction of negative affect due to derogatory feedback (Wiswede et al., 2009) or social-evaluative stress (Cavanagh et al., 2011a) leads to increased responsivity to internal indicators of performance errors, reflected in enhanced Ne amplitudes and mediofrontal theta power, respectively. Given the implication of the R-L theory (Holroyd & Coles, 2002) that larger prediction errors, i.e., larger Ne and FRN amplitudes, are associated with a stronger tendency to avoid the same maladaptive response subsequently, it would be predicted that failure-induced negative affective states are accompanied by improved learning from errors and negative feedback.

However, available evidence indicates that personality may be an important factor in mediating behavioural manifestations of failure-induction. In particular, the findings by Cavanagh et al. (2011a) suggest that high and low punishment sensitivity mediates different biases towards punishment vs. reward learning. Moreover, individual differences in action vs. state orientation that have been linked to the ability to cope with failure-induced negative affect (Kuhl, 1987), might explain why stress has sometimes been found to compromise the use of negative feedback for learning-related behavioural adjustments (e.g. Petzold et al., 2010). Thus, a specific aim of Experiment 1 and 2 was to test for moderating influences of

individual differences in punishment sensitivity, trait level negative and positive affect as well as action vs. state orientation on failure-induced changes in RL.

Given that social-evaluative stress has been shown to differentially affect habitual learning and goal-directed learning as well as explicit knowledge of response rules, it is furthermore conceivable that the effects of failure-induction differ for deterministic and probabilistic A-O-contingencies, promoting explicit and implicit learning strategies, respectively. Therefore, the employed learning task included a deterministic and a probabilistic condition.

A last issue concerned the on-going debate as to whether the response- and feedback-locked positivity are primary determinants of experimental variations in Ne and FRN, respectively (Eppinger et al., 2008; Foti et al., 2011; Holroyd et al., 2008). In particular, Eppinger and coworkers (2008; 2009) found more pronounced learning-related dynamics in the ERPs on correct trials, possibly reflecting neural activity associated with positive PEs. If this was the case, one might reason that failure affects the ERPs to correct responses and feedback rather than the ERPs to errors and negative feedback.

5. Experiment 1

Study Design

The experiment involved two consecutive phases (pre- and posttest) of a feedback-based probabilistic learning task. In order to investigate whether the effects of failure induction are sensitive to response-outcome contingency, the learning task included three different conditions of feedback validity: in the deterministic learning condition feedback was always valid, in the probabilistic learning condition feedback was valid in 80% of trials but invalid in 20% of trials, and in the chance condition, which served as control condition, feedback was delivered randomly. After the pretest, subjects performed a visual search task that they were informed was diagnostic of their intellectual abilities. This was incorporated in order to enhance self-relevance, which is assumed to be an essential feature of efficient failure induction techniques (cf. Brunstein, 2000). One half of the subjects were exposed to failure feedback (failure-feedback group) while the other half received no feedback during this task (no-failure-feedback group). Subsequently, both groups performed the posttest, which tested for the effects of prior failure manipulation. While the learning task was described in neutral terms at pretest, it was linked to intellectual abilities at posttest. In addition, to assess individual differences in cognitive as well as motivational and affective variables, a number of psychometric tests and questionnaires were administered at the start of the experiment that included measures of fluid and crystallized intelligence, working memory capacity, punishment and reward sensitivity, positive and negative affect as well as action and state orientation.

Research Predictions

The outline of research predictions is structured into three parts. The first part addresses learning-related effects in Ne, FRN, and Pe. Although the present study did not focus on the impact of learning per se, the analysis of these modulations is important for the interpretation of failure-related effects. Moreover, the present research aimed to replicate findings by Eppinger and colleagues (2008, 2009) indicating that learning does not only

affect the processing of errors and negative feedback, but also the processing of correct responses and positive feedback. The second part contains hypotheses concerning the impact of failure on error and feedback processing, including the implications for learning-related behavioural adaptation. The third part subsumes predictions regarding the role of personality in determining the failure-related effects.

Learning-related Modulations in Ne, FRN, and Pe

The R-L theory (Holroyd & Coles, 2002) asserts that the Ne increases as learning progresses, whereas the FRN should decrease with learning, reflecting the transition from reliance on internal vs. external feedback to evaluate the outcome of an action. Several studies have reported a learning-related increase in Ne amplitude (Eppinger et al., 2008, 2009; Holroyd & Coles, 2002; Morris et al., 2008; Nieuwenhuis et al., 2002; Pietschmann et al., 2008), whereas the evidence for learning-related modulations of the FRN is mixed (Cohen & Ranganath, 2007; Holroyd & Coles, 2002; Pietschmann et al., 2008; Walsh & Anderson, 2011a). Importantly, recent findings by Eppinger and colleagues (2008, 2009) suggest that learning-related changes occur in the ERPs to correct responses and positive feedback, rather than in the ERPs to incorrect feedback and errors.

Moreover, the R-L theory predicts that both the Ne and the FRN should be sensitive to action-outcome-contingency as the PE tracks deviations from “cached” estimates of expected value stored by the actor-critic (Maia, 2009; Niv & Montague, 2008). The expected value of an action, hence, is smaller if this action does not consistently lead to favourable outcomes. Consistent with this prediction, the Ne and the FRN have been shown to be smaller and larger, respectively, when partly invalid feedback interferes with learning (Eppinger et al., 2008; 2009; Holroyd & Coles, 2002; Nieuwenhuis et al., 2002). According to the R-L theory, Ne and FRN reflect teaching signals that train the ACC to learn adaptive response rules. In support of this notion, previous studies demonstrated a link between the magnitude of the FRN and subsequent behavioural adjustments (Cohen & Ranganath, 2007; van der Helden et al., 2010). In addition, the Ne amplitude has been shown to predict

individual differences in learning from errors and negative feedback (Frank et al., 2005). On the basis of these findings, the following predictions were made:

Prediction 1: *(a)* The Ne increases over the course of learning in the deterministic and probabilistic learning condition, but not in the chance condition. However, when correct and erroneous responses are analysed separately, more pronounced learning-related modulations are evident in the 'correct response-related positivity'. *(b)* The Ne amplitude is sensitive to feedback validity and should increase from the chance condition over the probabilistic to the deterministic learning condition. As for the learning-related changes, this effect should be present both in the ERPs to correct and incorrect responses. *(c)* The Ne amplitude predicts error-related behavioural adjustments, i.e., higher Ne amplitudes are associated with higher post-error accuracy.

Prediction 2: *(a)* The FRN decreases over the course of learning, mainly reflected in modulations in the ERPs to positive feedback. *(b)* The FRN is sensitive to feedback validity and decreases from the chance condition over the probabilistic to the deterministic learning condition[10].

The Pe is not assumed to reflect a PE and has not consistently been implicated in learning-related behavioural adaptation. Nonetheless, most theories on the functional significance of this component such as the 'error awareness-hypothesis' and the 'context-updating hypothesis' imply that the Pe should grow larger with learning (cf. Overbeek et al., 2005). To my knowledge, only one study examined learning-related modulations in Pe amplitude thus far. In line with accounts that conceptualize the Pe in terms of conscious aspects of error processing as well as context updating, Eppinger and cowokers (2009) demonstrated that the Pe increased as participants became better able to represent the correctness of their responses. Thus, the prediction was:

[10] In contrast to the Ne, the FRN amplitude was not expected to correlate with post-error accuracy. According to the R-L theory, larger FRN amplitudes should predict more efficient learning from negative feedback only in the very beginning of learning. With the development of an internal representation of the correct responses, the FRN is assumed to decrease and the Ne should predict behavioural adjustments. During later stages of the task, larger FRN amplitudes hence are thought to reflect *poor* learning.

Prediction 3: The Pe increases *(a)* over the course of learning and *(b)* with feedback validity.

Effects of Failure on Behavioural and Electrophysiological Indices of Learning

The impact of uncontrollable failure experiences on subsequent performance has been shown to depend on motivational characteristics both of the failure-induction and the subsequent training task (Brunstein & Gollwitzer, 1996). Helplessness research suggested that a motivational deficit underlies the negative effects of failure on instrumental learning (Mikulincer, 1994; Seligmann, 1975; Vollmayer et al., 2004). Brunstein and Gollwitzer (1996), however, demonstrated that participants exposed to self-relevant failure showed improved performance on a subsequent self-relevant task but impaired performance on an unrelated task. At the same time, it has been suggested that the need to cope with failure-induced negative emotions depletes limited control resources and hence leads to performance impairments on tasks relying on the same resources (Inzlicht & Gutsell, 2007).

Moreover, social-evaluative stress has been found to affect learning strategies rather than overall learning performance (Cavanagh et al., 2011a; Petzold et al., 2010, Schwabe & Wolf, 2009). Findings by Schwabe and colleagues (2011) suggested that stress specifically promotes implicit, habitual learning at the expense of explicit learning and memory strategies. Given the contradictory pattern of findings regarding behavioural effects of uncontrollable failures, the following hypotheses are more tentative:

Prediction 4: *(a)* Participants in the failure-feedback group should be more motivated to perform well at posttest than those in the no-failure-feedback group. The increased motivation should counteract the potentially detrimental effects of resource-consuming emotion regulation demands. Hence, both groups were expected to show similar overall posttest performance. *(b)* Given that stress has been shown to promote slow, habitual learning, failure-induction may differentially affect learning of deterministic and probabilistic contingencies. Thus, if anything, failure should affect performance in the deterministic learning condition in which explicit strategies are more helpful. In addition, a failure-induced bias towards habitual control should be reflected in a slower time course of learning.

Critically, available evidence on whether stress alters the ability to learn from past mistakes is inconclusive. While Petzold and colleagues (2010) reported stress-related impairments in the ability to use negative feedback for behavioural adaptation, others found stress-related improvements in punishment learning in highly punishment sensitive individuals (Cavanagh et al., 2011a). However, both studies assessed the stress-related effects in a test phase, which followed the actual learning phase. This test phase required participants to select the 'best option' among different pairs of stimuli that were associated with different reward probabilities in the previous learning phase. In choosing this indirect approach, the studies did not directly track error- and feedback-related behavioural adjustments during learning.

Previous findings indicated that the Ne is sensitive to the motivational and affective significance of an error (Falkenstein et al., 2000; Gehring et al., 1993; Hajcak et al., 2005; Potts, 2011). Moreover, the induction of short-term negative affect has been shown to be associated with an increase in Ne amplitude (Wiswede et al., 2009a,b). Similarly, Cavanagh and colleagues (2011a) showed that the exposure to social evaluative stress lead to heightened response-related mediofrontal theta-power. However, others failed to obtain negative affect-related enhancements in Ne amplitude (Clayson et al., 2011; Larson et al., 2006). So far, the effects of affective/motivational manipulations on performance monitoring have been examined using response competition or gambling tasks with known response contingencies. There is evidence indicating that the processing of 'errors of commission' during these tasks and suboptimal choices during probabilistic learning might be differentially influenced by affective variables (Gründler et al., 2009; Cavanagh et al., 2010b). Since most researchers agree that uncontrollable failure gives rise to strong negative feelings, the following predictions concerning the Ne and post-error behavioural adjustments were made:

Prediction 5[11]: *(a)* Failure-induction leads to an increase of the Ne at posttest. *(b)* This affect-related enhancement was expected to be more pronounced if participants were better able to represent the correctness of their responses, i.e., the pre-post Ne differences should

[11] As was already stated above, this study specifically focused on differential modulations of ERPs to error/negative feedback vs. correct responses/positive feedback.

be larger towards the end compared to the beginning of the learning process. *(c)* In addition, dissociations in Ne amplitude between tasks with fixed response rules and probabilistic learning tasks suggest that the effects of failure may be sensitive to feedback validity. This could be reflected in a more pronounced failure-related Ne modulation in the deterministic compared to the probabilistic learning condition.

Prediction 6: Although previous research indicated that social-evaluative stress alters the processing of errors and feedback, no study has explicitly examined the impact affective/motivational manipulations on behavioural adjustments during learning. Yet, the R-L theory implies that affect-related modulations in the Ne (see Prediction 5) should be reflected in adaptive action selection. Therefore, the failure-related enhancement in Ne amplitude was expected to be associated with higher post-error accuracy.

The susceptibility of FRN and Pe to motivational and affective influences is less substantiated. In neuropsychiatric disorders such as major depression and OCD, the Pe is often unaffected (Overbeek et al., 2005). Reduced Pe amplitudes in severely depressed patients have been linked to apathy rather than negative affect (Schrijvers et al., 2009). However, Hajcak and colleagues (2004) reported that high trait level negative affect was associated with a reduced Pe. Larger FRN amplitudes have been found in moderately depressed patients (Cavanagh et al., 2011b; Santesso et al., 2008; Tucker et al., 2003). Furthermore, recent findings point to a positive relationship between punishment sensitivity and FRN magnitude (Balconi & Crivelli, 2010; De Pascalis, Varriale, & D'Antuono, 2010; Santesso et al., 2011). Yet, reduced mediofrontal theta-power in response to negative feedback has been found when participants were exposed to stress (Cavanagh et al., 2011a). The described pattern of findings made a clear prediction regarding failure-related effects on the FRN and the Pe difficult:

Prediction 7: *(a)* Given the findings that the FRN is enhanced in depressed and highly punishment sensitive individuals, failure-induction may result in larger FRN amplitudes. This prediction also seems consistent with the R-L theory, which states that Ne and FRN reflect basically the same process. However, no prior study has examined the impact of

experimental manipulations of affective/motivational variables on the FRN. Moreover, stress has been found to reduce sensitivity to negative feedback, making a straightforward prediction difficult. *(b)* According to the R-L theory, Ne and FRN track the reliance on internal and external feedback, respectively, over the course of learning. It was reasoned that the experience of uncontrollable self-relevant failure might affect the dynamics of learning-related changes in Ne and FRN. Specifically, participants in the failure-feedback group were expected to rely more strongly on external feedback. This should be reflected in a less pronounced reduction in FRN amplitude as learning progressed.

Prediction 8: Given the limited number of studies that experimental manipulations of affective and motivational variables to examine later stages of error processing as reflected in the Pe, the susceptibility of this component to failure-induction remains an open question.

The Modulatory Role of Personality

Individual differences in the disposition towards state vs. action orientation have been shown to moderate the effect of uncontrollable failure on subsequent performance (Kuhl, 1981). Thus, it follows that:

Prediction 9: State-orientated participants perform worse after failure-induction than action-oriented participants.

Moreover, high punishment sensitivity predicted better implicit learning in a punishment compared to a control condition (Corr, Pickering, & Gray, 1997) as well as improved probabilistic learning from negative feedback and impaired learning from positive feedback under stress (Cavanagh et al., 2011a). The opposite pattern has been observed in low punishment sensitive individuals. Yet, less consistent findings have been reported in studies using explicit learning tasks with deterministic contingencies (e.g., Zinbarg & Revelle, 1989) and the effects in Cavanagh et al.'s study were observed *after* but not *during* learning. Nonetheless the findings suggest:

Prediction 10: *(a)* Punishment sensitivity moderates the effects of failure on subsequent learning performance. *(b)* The influence of individual differences in punishment sensitivity may differ for the deterministic and probabilistic learning condition.

Pailing and Segalowitz (2004) demonstrated that high neuroticism scores predicted larger incentive-related modulations in Ne amplitude, whereas high conscientiousness predicted smaller incentive effects. In the study by Cavanagh and colleagues (2011a), however, stress-induced negative affect predicted higher response-related but reduced negative feedback-related mediofrontal theta power both in highly and less punishment sensitive individuals[12]. To my knowledge, no further study has investigated to what extent personality factors moderate the effect of affective/motivational manipulations on electrophysiological correlates of error and feedback processing. Nonetheless, the sensitivity of the Ne and - albeit to a lesser extent - of Pe and FRN to motivation- and affect-related traits suggests that:

Prediction 11: Individuals differences in state vs. action orientation and punishment sensitivity moderate the impact of failure on the Ne. It is an open issue to what extent trait variables determine failure-related modulations in FRN and Pe.

Methods

Participants

Forty-two undergraduate students with normal or corrected to normal vision, free of neurological or psychological disorder and free from psychoactive medication use, completed pre- and posttest. A further 22 participants had to be excluded after the pretest because they did not commit enough errors to obtain reliable ERP-measures (>14 error trials in both halves of pre- and posttest). Data from seven participants were discarded due to poor learning performance[13] (3), excessive artifacts (2), and technical problems during EEG recording (2).

[12] Yet, the neural responses differentially predicted behavioural indices of punishment learning in the low vs. high BIS group.

[13] Less than 55% correct trials in the deterministic learning condition

The effective sample thus consisted of 17 participants (12 women; mean age = 22.6 years; age range = 19 to 33 years) in the failure feedback group and 18 participants (13 women, mean age = 21.7 years; age range = 19 to 27 years) in the no-failure-feedback group. They were paid 8 Euros per hour or received course credit. Informed written consent was required in accordance with the protocols approved by the local ethics committee of Saarland University, and participants were thoroughly debriefed after the experiment.

Overview of the Experimental Procedure

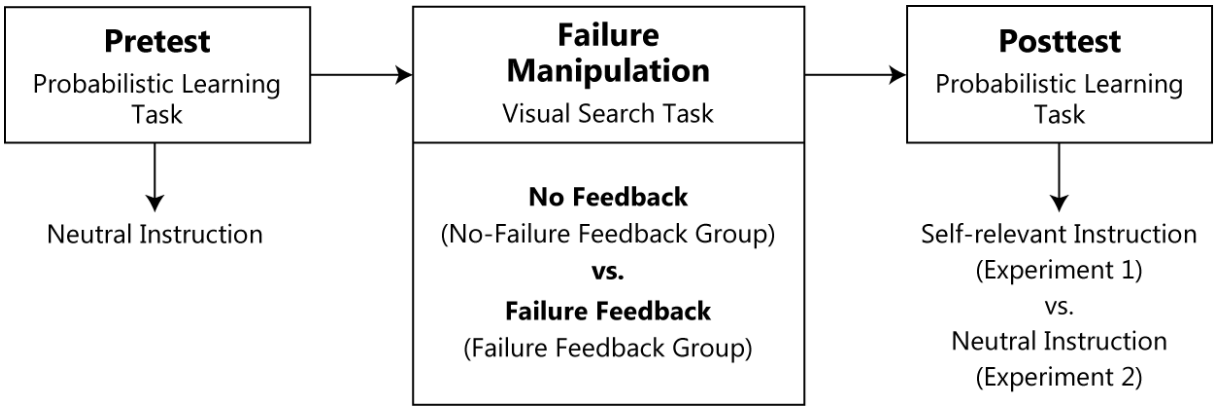


Figure 8: Schematic illustration of the experimental procedure.

A schematic overview of the experimental procedure is outlined in Figure 8. After a brief description of the experiment, participants filled out a consent form and a short demographic questionnaire. Then they completed the psychometric tests and personality scales. The following experiment involved three consecutive phases: the first probabilistic learning phase (pretest), failure manipulation (visual search task), and the second probabilistic learning phase (posttest). After completion of the pretest, one half of the participants were assigned to the no-failure-feedback group, and the other half were assigned to the failure-feedback group. Both groups were matched for performance in the pretest. In order to test the effect of failure manipulation, participants' mood state was assessed with a short questionnaire immediately after the visual search task. At posttest, participants were informed that they were going to perform the probabilistic learning task again. In contrast to the pretest, it was now stressed that the task was indicative of intellectual abilities. At the end of the experiment, participants were given a brief final

questionnaire that focused on the motivational involvement and the emotional experience associated with performing the two learning phases and the visual search task (see Appendix). Afterwards, participants were informed of the actual purpose of the study and the nature of the experimental manipulations. Particularly, participants in the failure feedback group were told that the negative feedback in the visual search task was totally independent of their actual performance.

Stimuli and Tasks

Probabilistic learning task (Pre- and posttest). The stimulus material consisted of 24 colored images of objects (Snodgrass & Vanderwart, 1980). On a given trial participants were presented with a target stimulus and had to press one of two response keys. Following the response either the word “RICHTIG” (“correct”), “FALSCH” (“incorrect”) or “ZU LANGSAM” (“too slow”) was presented. Participants were instructed to infer the correct stimulus-response mappings on the basis of the feedback. In order to maintain motivation throughout the experiment, participants were also told that they would gain a point for each correct answer and lose a point for each incorrect or too slow response. At the end of the experiment a monetary bonus of up to 10 Euros was awarded based upon the total amount of points. Stimuli were associated with three different conditions of feedback validity (100%, 80 %, and 50%). Four stimuli were assigned to each condition, yielding a total of 12 different target stimuli. In the deterministic learning condition, feedback was always valid (100% validity). Two stimuli were mapped to the left and the right response key, respectively. In the probabilistic learning condition, feedback was valid in 80% of the trials. For the two stimuli that were mapped to the left response key, participants thus received ‘Correct’ feedback in 80% and ‘Incorrect’ feedback in 20% of left button presses (and vice versa for right button presses and the other two stimuli mapped to the right response key). In the chance condition, ‘Correct’ and ‘Incorrect’ feedback was delivered at random (50% validity). Each of the 12 target stimuli were presented 50 times in pseudo-randomized order throughout the task, resulting in a total of 600 trials. The same individual stimulus appeared at most three times in a row and within a block of 60 trials the number of stimuli associated with the three learning

conditions was equal. The assignment of stimuli to learning condition and response key was counterbalanced across participants.

Each trial started with a central fixation cross presented on a light gray background for a variable interval of 250 to 500 ms, followed by the centrally presented target stimulus that was displayed for 500 ms. In order to obtain a sufficient number of error trials, we applied an adaptive response deadline ranging from a minimum value of 400 ms to a maximum value of 1000 ms (for a similar procedure, see Eppinger et al., 2008). At the beginning of the task, the response time window was set to an initial value of 800 ms. After the completion of the first 100 trials, the response window was individually adjusted on a trial-to-trial basis, depending on the proportion of time out trials, i.e., the total number of time-out trials divided by the total number of trials performed up to that point. If the proportion of time-outs was less than two (or more than eight) percent, the response time window was decremented (incremented) by 200 ms. If the proportion of time-outs varied between two and four (six and eight) percent, the response deadline was decremented (incremented) by 100 ms. In the interval from four to six percent of time-outs, the response deadline remained unchanged. After the response, a blank screen was presented for 500 ms, followed by the feedback displayed for 500 ms. A randomly jittered 1250- to 2000-ms interval separated each trial. Participants first worked through 60 practice trials. Pre- and posttest consisted of 20 blocks of 30 trials. During the breaks, they received feedback about the total amount of points they had collected up to that point.

Visual search task (Failure manipulation). Ten pairs of natural-scene pictures served as stimuli for the failure manipulation task. Each picture pair comprised the “original” and a modified “copy” of a colored photograph that were presented on the left (original) and right (copy) side of the screen. The “copies” were created by changing six to ten subtle details in each picture (see Figure 9 for an example stimulus pair). All photographs were scaled to 9 × 12 cm. The task was described as a mental speed test predictive of intelligence. This was incorporated to enhance self-relevance, which is assumed to be an essential feature of efficient failure induction techniques (cf. Brunstein, 2000). Each picture pair was presented for 60 s, and participants were asked to indicate by mouse-click all differences in the

modified picture as quickly as possible. Participants in the failure feedback group received predominantly negative feedback indicating the number of differences that have not been detected. Feedback was delivered according to a fixed schedule that has been shown to induce a strong failure experience (Brunstein & Olbrich, 1985). An initial sequence of success and failure (0 − 2 − 0 − 3) was followed by continuous failure (4 − 4 − 3 − 5 − 4 − 6). Thereafter, a brief protocol was presented which provided participants in the failure feedback group with a spurious evaluation of their performance: The total number of differences missed by the individual (31) and the average number of differences missed by other participants (19). In the no-failure feedback group no feedback was provided.

Figure 9: Example stimulus pair presented in the visual search task. Differences are marked in the rightmost picture.

Questionnaires. The participants performed two psychometric tests assessing fluid intelligence (Digit-Symbol Substitution test, DSS; adapted from Wechsler, 1981), and crystallized intelligence (Spot-a-Word test; adapted from Lehrl, 1977). A modified version of the Digit Ordering Test (Cooper, Sagar, Jordan, Harvey, & Sullivan, 1991) was used to measure working memory capacity. Participants also completed German versions of the following questionnaires: The Positive Affect Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988; Krohne, Egloff, Kohlmann, & Tausch, 1996), the Carver and White (1994)

Behavioral Inhibition Scale/Behavioral Activation Scale (BIS/BAS), and the Action Control Scales (ACS-90, Kuhl, 1994). The PANAS assesses the predisposition to experience positive or negative affective state. The BIS/BAS scales were used as measures of punishment and reward sensitivity. The ACS-90 was used to assess the general tendency toward action- vs. state-oriented behaviour after failure experiences (HOM). The final questionnaire focused on the motivational involvement and the emotional experience associated with performing the learning tasks and the visual search task (see Appendix). To check the effect of failure manipulation we administered the German questionnaire "Befindlichkeitsskala" (BfS). The BfS (von Zerssen, 1976) comprises 28 pairs of opposite adjectives (e.g., "self-confident/insecure"). For each pair, subjects are required to indicate the adjective that better represents their current feelings.

EEG Recording

The electroencephalogram (EEG) was recorded with Ag/AgCl electrodes from 59 sites according to the extended 10-20 system, referenced to the left mastoid, at a sampling rate of 500 Hz (filtered online from DC to 70 Hz). The horizontal and vertical electrooculograms were recorded from electrodes placed on the left and right canthi of both eyes and at the infra- and supra-orbital ridges of the left eye. Electrode impedances were kept below 5 kΩ.

Data Analyses

Behavioural data analyses. Response latencies of less than 244 ms (> 2SD) or exceeding the response deadline were excluded from further analyses[14]. Mean accuracy rates were computed separately for each learning condition in pre- and posttest by averaging the data into six bins of 100 trials each, i.e., Bin 1 contained Trials 1-100, Bin 2 contained

[14] The deadline was exceeded by 2.2% (no-failure-feedback group) and 2.1% (failure-feedback group) of the responses. At pretest, mean RTs on correct and incorrect trials were 421 ms (*SD* = 25 ms) and 417 ms (*SD* = 30 ms) for the no-failure feedback group and 422 ms (*SD* = 26 ms) and 416 ms (*SD* = 29 ms) for the failure feedback group. At posttest, mean RTs on correct and incorrect trials were 405 ms (*SD* = 30 ms) and 398 ms (*SD* = 31 ms) for the no-failure feedback group and 396 ms (*SD* = 32 ms) and 388 ms (*SD* = 31 ms) for the failure feedback group.

Trials 101-200, and so on. Note that for the probabilistic learning condition only valid trials were included. To examine error-related behavioural changes, I additionally computed post-error accuracy, i.e., the proportion of correct choices on the next presentation of a particular item, separately for each learning condition and test phase.

ERP data analyses. Offline, the data were re-referenced to the linked mastoid and band-pass filtered from 0.05 to 30 Hz. Eye movement artefacts were removed using independent component analysis from the BrainVision Analyzer Software Package (Brain products); remaining artefacts were eliminated with a semiautomatic artefact rejection procedure (amplitudes over ± 100 µV, changing more than 50 µV between samples or more than 200 µV within single epochs, or containing baseline drifts). Artefact-free EEG data were segmented relative to response and feedback onset to extract response-related and feedback-related ERPs. The response-locked and feedback-locked epochs were baseline corrected with respect to the average voltage during a -200 to -50-ms-pre-response interval and a 100-ms-pre-stimulus interval, respectively.

Following previous studies using probabilistic learning tasks (e.g., Eppinger et al., 2008; Frank, et al., 2005), the Ne was quantified after 15 Hz low-pass filtering at electrode FCz as the peak-to-peak difference in voltage between the most negative peak between -50 and 100 ms and the largest positive peak in the prior 100 ms. Peak-to-peak voltage was measured to determine baseline-independent amplitudes and to minimize distortions due to the positivity on which the Ne is superimposed. Since a negative peak could not be reliably determined for correct trials, mean amplitudes in a 0-100 ms post-response time window at electrode FCz were computed to analyse the positivity following correct responses. I decided to analyse correct and incorrect responses separately rather than to compute difference waves (error-correct), because former studies revealed larger learning-related modulations in the positivity on correct trials than in the Ne (e.g., Eppinger et al., 2008), whereas affective-motivational manipulations have been shown to specifically affect the Ne (e.g., Hajcak et al., 2005).

The FRN was quantified twofold. In a first step, I determined the FRN as peak-to-peak voltage difference between the most negative peak in a 200 to 400 ms time window

after feedback onset and the preceding positive peak in a 150 to 300 ms post-feedback interval at electrode FCz (see Frank et al., 2005; Yeung & Sanfey, 2004). However, as participants learned quickly, the ERP-averages contained relatively few trials for negative feedback, particularly in the deterministic learning condition. Whereas just six trials are necessary to obtain stable averages for the Ne (Olvet & Hajcak, 2009b), 20 trials are needed to ensure a reliable FRN (Marco-Pallares et al., 2011). Of note, peak-amplitude measures are more sensitive to noise-induced fluctuations than average amplitude measures (Luck, 2005). Therefore, in a second step, the FRN was defined as mean amplitude in a 50-ms-time-window centered on the individual negative peaks of the FRN. As in previous studies (Hajcak et al., 2004; Wiswede et al., 2009), the Pe was measured as the mean amplitude between 200 and 400 ms after the response at electrode Pz.

To examine learning-related changes in Ne, FRN, and Pe, ERP averages were computed for each learning condition, separately for the first and second half of pre- and posttest. Thus, Bin 1 (first half) of the ERP analysis comprised Bins 1-3 of the behavioral analysis, and Bin 2 (second half) of the ERP analysis comprised Bins 4-6 of the behavioral analysis.

Statistical analyses. Accuracy and ERP data were analyzed using repeated measures analyses of variance (ANOVAs). In a further step, each personality measure (BIS, BAS, NA, and HOM) was included as a continuous moderator in the ANOVAs to examine the modulatory role of trait variables. Separate analyses of covariance (ANCOVAs) were conducted for each of the four variables. Whenever necessary, the Geisser-Greenhouse correction was applied (Geisser & Greenhouse, 1958) and corrected p values are reported together with the uncorrected degrees of freedom and the epsilon values (ε). Pearson's correlations were calculated to examine the relation between Ne amplitude and post-error accuracy. In order to check for a-priori group differences in personality measures (control analyses) as well as group differences in post-manipulation mood rating and final questionnaire (manipulation check), the corresponding measures were analyzed using multivariate analysis of variance (MANOVA).

Results

Control Analyses/Manipulation Check

Table 1 shows the results of the psychometric tests and the questionnaires for the no-feedback and the failure feedback group.

Tabelle 1. Results of the psychometric measures, the mood scale (BfS), self-evaluation for the visual search task and motivational involvement/rumination at posttest (means and standard deviations) for the no-failure feedback group and the failure feedback group (Experiment 1)

Measure	No-failure feedback group	Failure feedback group
Cognitive variables		
DSS	63.67 (10.62)	62.18 (9.70)
Spot-a-word	18.56 (6.61)	18.76 (4.89)
Digit ordering	9.33 (2.06)	8.94 (1.92)
Affect & action control		
PA	34.28 (4.38)	34.47 (5.51)
NA	23.17 (6.30)	20.06 (5.93)
BIS	2.87 (0.45)	2.91 (0.49)
BAS	2.99 (0.49)	3.11 (0.33)
HOM[a]	4.50 (3.01)	3.82 (3.36)
Mood (post-manipulation)		
BfS[b]	9.72 (3.56)	18.47 (3.34)
Final questionnaire		
Self-evaluation	3.47 (0.74)	2.71 (0.44)
Involvement	3.33 (0.78)	3.80 (0.61)
Rumination	2.03 (1.01)	2.06 (0.92)

a Note that higher scores indicate action orientation while lower scores indicate state-orientated behavior.

b Higher scores indicate more negative feelings.

Importantly, the two groups did not differ significantly with respect to psychometric measures (all *p*-values > .14). There were, however, differences in current mood state after failure manipulation. The failure feedback group reported more negative feelings, as indicated by a higher BfS score, [F (1,33) = 56.07, p < .001]. Moreover, participants' self-reports on the final questionnaire showed that they were less satisfied with their performance on the visual search task as compared to the no-failure feedback group [F (1,33) = 15.78, p < .001], and also tended to indicate higher posttest involvement [F (1,33) = 3.90, p < .058].

Accuracy Data

Accuracy data (see Figure 10) were analysed using an ANOVA with the between-subjects factor *feedback group* (failure feedback vs. no feedback), and the within-subject factors *test phase* (pretest vs. posttest), *learning condition* (deterministic, probabilistic and chance condition), and *bin* (Bins 1-6). As expected, the analysis yielded a significant main effect of learning condition [F(2,66) = 231.26, p < .001, ε = .80]. Contrasts revealed accuracy to be higher for the deterministic and probabilistic learning condition compared to the chance condition, as well as for the deterministic compared to the probabilistic learning condition (*p*-values < .01).

Learning-related effects. The analysis yielded a reliable main effect of bin [F(5,165) = 22.81, p < .001, ε = .66] that was qualified by significant interactions between learning condition and bin [F(10,330) = 4.83, p < .001, ε = .82], and test phase, learning condition, and bin [F(10,330) = 3.28, p < .01, ε = .85] suggesting that the course of learning differed between pre- and posttest as a function of feedback validity. To decompose the interaction, separate analyses were conducted for pre- and posttest. At pretest, polynomial contrasts showed that accuracy increased over the course of the task following a linear trend for the deterministic as well as the probabilistic learning condition (*p*-values < .001; see Figure 12). At posttest, a significant linear trend across bins was obtained only for the probabilistic learning condition (p < .01). In contrast, a predominantly cubic trend for the deterministic learning condition (p < .001) reflected that after an initial enhancement, accuracy dropped and finally increased again.

Effects of failure manipulation. Visual inspection of the posttest data suggested that the transient decrease in accuracy emerged for the failure feedback group only (see Figure 10). However, the four-way interaction of feedback group, test phase, learning condition, and bin failed to reach significance (p = .10).

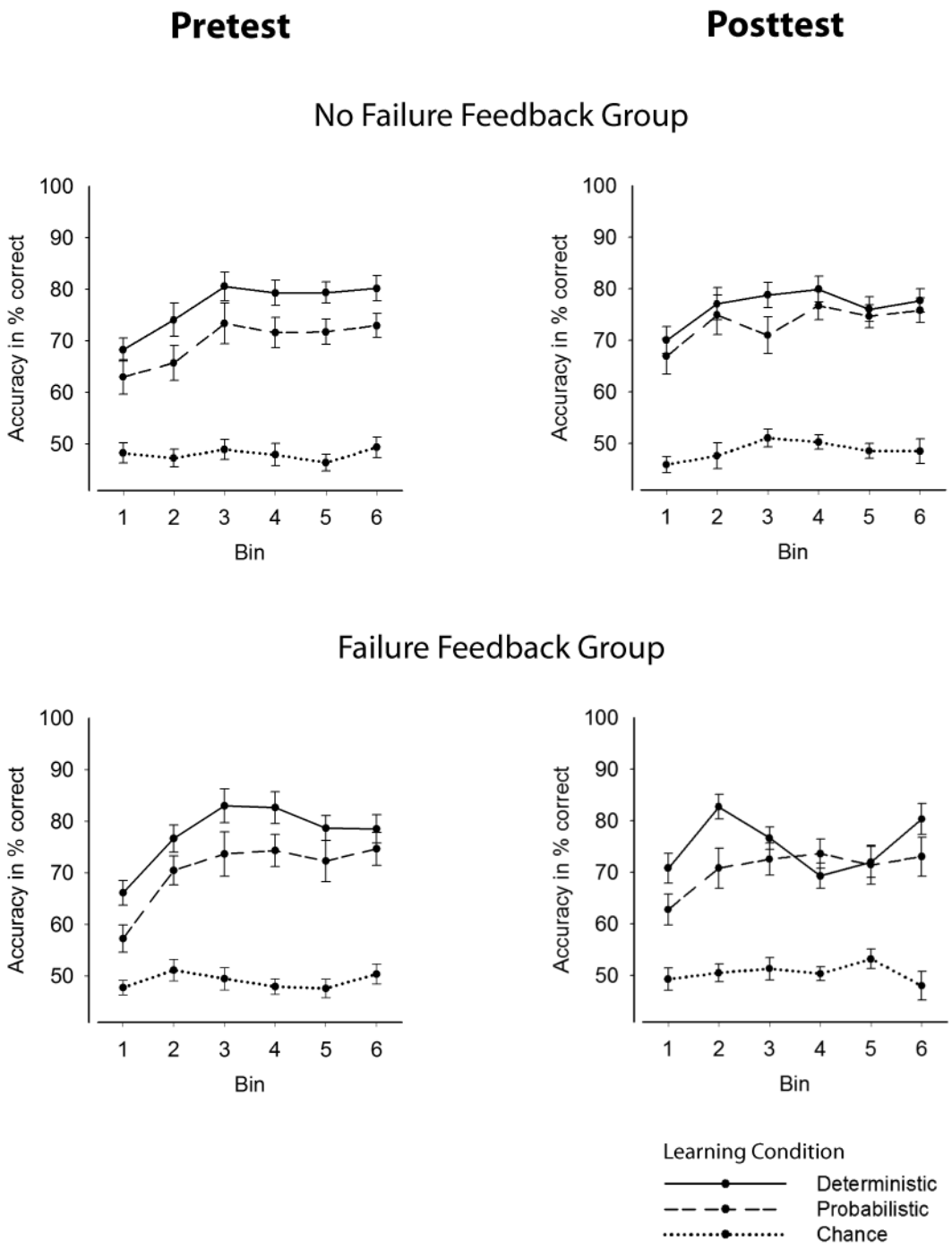


Figure 10: Mean accuracy learning curves for the three learning conditions displayed separately for the no-failure feedback group and failure feedback group at pretest (left) and posttest (right). Error bars indicate standard error.

Post-error Accuracy Data

In order to determine whether failure induction affects the ability to learn from past mistakes and thus modulates error-related behavioural changes, post-error accuracy rates were subjected to an ANOVA with the between-subjects factor *feedback group*, and the within-subject factors *test phase*, and *learning condition*. Consistent with the results for the total accuracy, a significant main effect of learning condition was obtained [$F(2,66) = 78.80$, $p < .001$], indicating that post-error accuracy was lowest in the chance condition and highest in the deterministic learning condition (all p-values < .001) (see Figure 11).

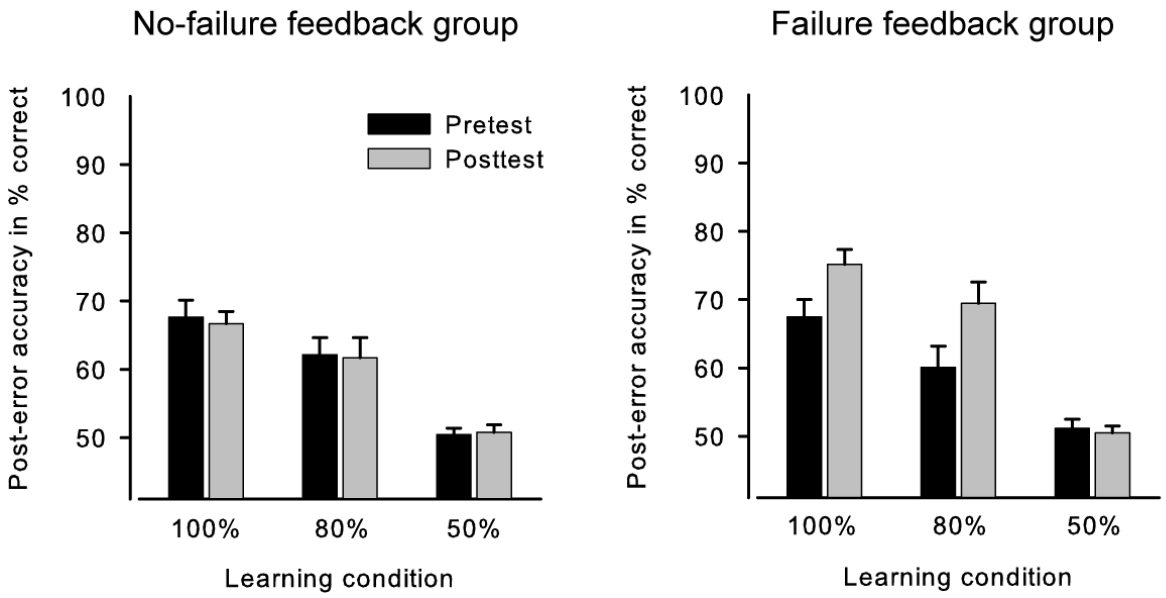


Figure 11: Mean post-error accuracy rates for the three learning conditions at pretest and posttest displayed separately for the no-failure feedback group (left) and failure feedback group (right). Error bars indicate standard error.

Effects of failure manipulation. The analysis revealed a reliable main effect of test phase [$F(1,33) = 4.27$, $p < .05$] that was qualified by a significant interaction between feedback group and test phase [$F(1,33) = 5.53$, $p < .05$], and a marginally significant interaction between feedback group, test phase, and learning condition [$F(2,66) = 2.85$, $p = .065$]. Separate analyses for the two groups showed that post-error accuracy increased from pre- to posttest for the failure feedback group ($p < .01$) but not for the no-failure feedback group ($p = .85$). To examine whether the post-error accuracy differences are due to the failure feedback group adopting a more conservative response strategy, i.e., more accurate

at the expense of slower post-error responding, post-error correct vs. incorrect reaction times (RT) were analyzed (see Appendix). This analysis did not reveal significant interactions involving the factor feedback group and test phase (*p*-values > .15). Thus, the failure-related change in post-error accuracy does not appear to reflect a speed-accuracy trade-off.

Does Personality Moderate the Effects of Failure on Error Processing and Learning?

To investigate whether personality differences modulates the effects of failure-induction on learning, accuracy rates were averaged across the six bins of pre- and posttest, respectively. Mean accuracy rates were subjected to 2 (*feedback group*) × 2 (*test phase*) × 3 (*learning condition*) ANCOVAs with the trait measures as continuous moderators. The analyses focused on interaction terms involving the continuous moderator variable and the factors feedback group and test phase.

High punishment sensitivity predicts failure-induced learning impairments. The analysis of the moderating influence of punishment sensitivity yielded a marginally significant four-way-interaction of feedback group, test phase, learning condition, and BIS [$F(2,62) = 2.67$, $p = .089$, $\varepsilon = .82$]. Follow-up ANCOVAs that were split by test phase revealed a significant interaction of feedback group, learning condition, and BIS for posttest [$F(2,62) = 3.14$, $p = .05$] but not for pretest ($p = .29$). Of note, a marginally significant interaction of learning condition and bin was found in the failure-feedback-group only [$F(2,30) = 3.14$, $p = .058$]. In the failure-feedback-group, higher BIS-scores predicted worse learning performance in the deterministic learning condition at posttest [$r(17) < -.54$, $p < .05$]. Furthermore, a marginally significant negative correlation between BIS and overall posttest performance was obtained for the probabilistic learning condition [$r(17) < -.44$, $p = .077$]. There was, however, no evidence for influences of punishment sensitivity on failure-related differences in post-error accuracy (*F*-values < 1.7, *p*-values > .20). No further trait variable reliably modulated the effects of failure-induction on learning performance (*F*-values < 2.1, *p*-values > .16).

Summary of Behavioural Findings

Not surprisingly, accuracy was found to increase with feedback validity. Moreover, accuracy increased in a linear function over the course of pretest for both learning conditions, but this was only the case for the probabilistic learning condition at posttest. For the deterministic learning condition considerable learning occurred at the beginning of posttest, but accuracy decreased with time on task. As participants were likely to become quickly aware of the response contingencies in the deterministic learning condition, this finding suggests a higher amount of "slips" in cognitive control resulting in motor errors of commission – rather than weak reinforcement learning – during later stages of posttest. Consistent with this notion, RT decreased from pre- to posttest, and erroneous responses were faster in the deterministic compared to the probabilistic learning condition (*p*-values < .001) (see Appendix). There were no between-group differences in overall performance at either pretest or posttest. However, high punishment sensitivity predicted worse posttest performance in the failure-feedback group. By contrast, punishment sensitivity did not appear to affect learning in the no-failure-feedback group. Furthermore, I did not obtain pre-post changes in overall accuracy for either of the two feedback groups. Instead, participants in the failure feedback group were more likely to correct their errors on the next repetition of a given stimulus, as was shown by an increase in post-error accuracy from pre- to posttest.

ERP data

Error Negativity

As illustrated in Figure 12, the Ne was evident as a fronto-centrally distributed negative deflection in the deterministic and probabilistic learning condition for both groups at pre- and posttest. Correct responses were followed by a pronounced positivity that, unlike the negativity on incorrect trials, clearly increased over the course of learning. To test for group differences in the peak-to-peak amplitude of the Ne and the mean amplitude of the correct response-related positivity, I used separate 2 (*feedback group:* failure feedback vs. no feedback) × 2 (*test phase:* pretest vs. posttest) × 3 (*learning condition:* deterministic, probabilistic and chance condition) × 2 (*bin:* Bin 1 vs. Bin 2) ANOVAs. Both the Ne and the

correct response-related positivity increased with feedback validity [$F(2,66) = 49.14$ and 49.50, $p < .001$ and .01, respectively]. Below, I will first report the failure- and learning-related effects for the Ne, followed by the results for correct responses.

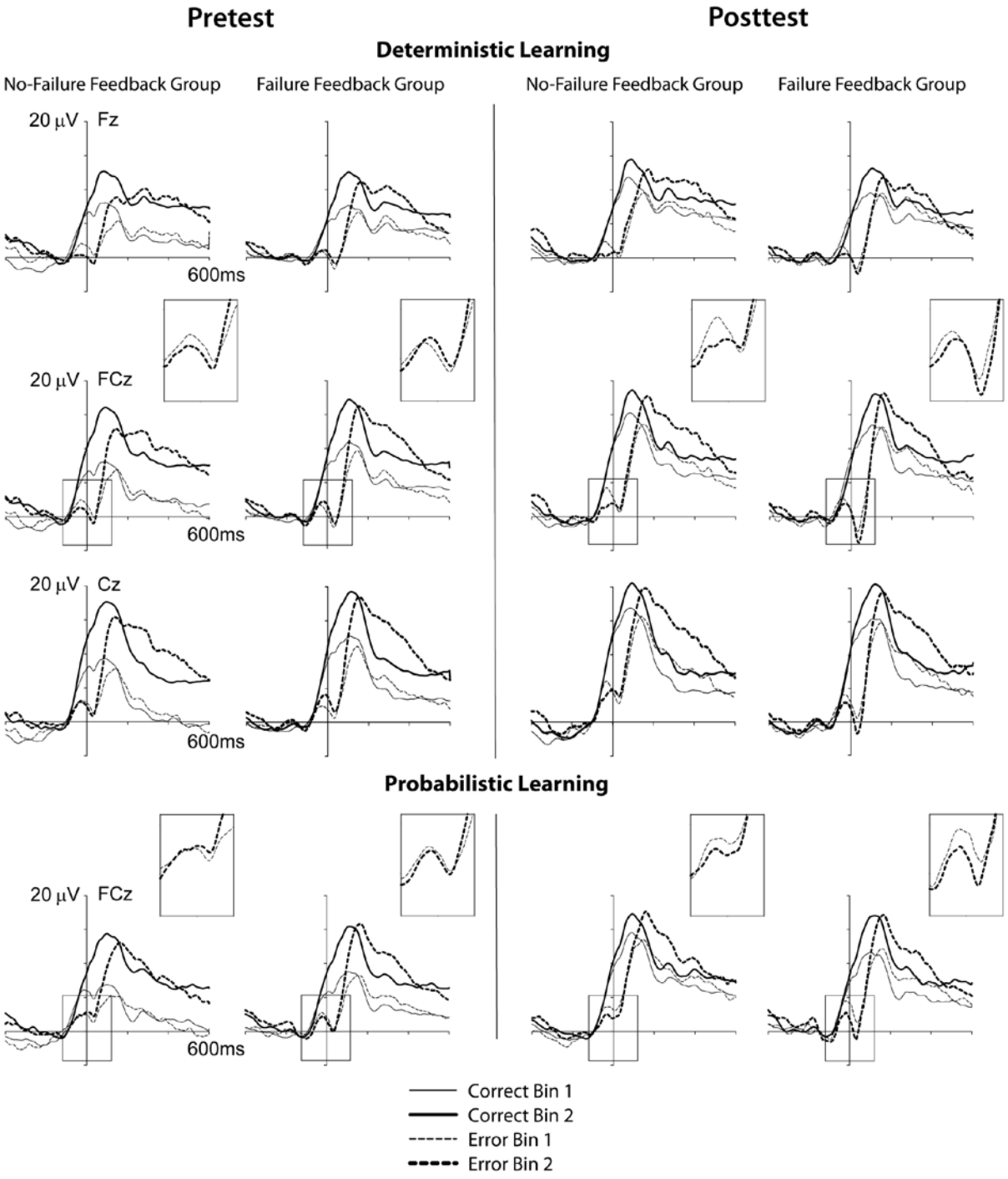


Figure 12: Response-locked ERPs to correct (solid lines) and incorrect responses (dashed lines) displayed separately for the no-failure feedback group and failure feedback group and the two halves of pretest (left) and posttest (right). The upper panels show the ERPs in the deterministic learning condition at electrode sites Fz, FCz, and Cz. Small boxes highlight the Ne effect at electrode FCz. The lowest panel shows the ERPs in the probabilistic learning condition at FCz. Note that the Ne amplitude was quantified peak-to-peak.

General effects of failure on the Ne. The analysis yielded a reliable main effect of feedback group [$F(1,33) = 8.30$, $p < .01$] and significant interactions between feedback group and test phase [$F(1,33) = 10.01$, $p < .01$] and feedback group, learning condition and test phase [$F(2,66) = 4.29$, $p < .05$, $\varepsilon = .75$]. A follow-up ANOVA that was split by test phase confirmed that there were no group differences at pretest (p-values > .34), whereas the Ne amplitude was larger for the failure feedback group than for the no-failure feedback group at posttest [$F(1,33) = 15.26$, $p < .001$]. As illustrated in Figures 12 and 13, the failure-related Ne modulation was more pronounced in the deterministic and probabilistic learning condition than in the chance condition, reflected in an interaction between feedback group and learning condition [$F(2,66) = 5.64$, $p < .01$, $\varepsilon = .80$]. Nonetheless, separate analyses revealed significant differences for the deterministic ($p < .01$) and probabilistic learning condition ($p < .001$) as well as for the chance condition ($p < .05$). Figure 13 also shows that the Ne increased from pre- to posttest for the deterministic and probabilistic learning condition in the failure feedback group ($p < .05$ and .01, respectively), but not in the no-failure feedback group (p-values >.17).

Learning-related effects of failure on the Ne. The analysis of learning-related changes in the Ne revealed significant interactions between learning condition and bin [$F(2,66) = 7.18$, $p < .01$] and feedback group, learning condition, and bin [$F(2,66) = 5.29$, $p < .01$], as well as a marginally significant interaction between test phase, feedback group, learning condition, and bin [$F(2,66) = 2.88$, $p = .065$]. Follow-up ANOVAs for the two test phases yielded a reliable interaction between feedback group, learning condition, and bin for posttest [$F(2,66) = 9.66$, $p < .001$], but not for pretest ($F < 1$), indicating group differences in the modulation of the Ne across posttest only. Decomposing the interaction within each group revealed a significant main effect of bin [$F(1,17) = 11.57$, $p < .01$] and a significant interaction between learning condition and bin [$F(2,34) = 7.72$, $p < .01$, $\varepsilon = .70$] for the no-failure feedback group as well as for the failure-feedback group [$F(1,16) = 4.66$, $p < .05$ and $F(2,32) = 7.97$, $p < .01$, respectively]. Figure 13 illustrates that the Ne decreased from the first to the second half of posttest for the deterministic learning condition in the no-failure feedback group ($p < .01$) but

increased for deterministic and probabilistic learning condition in the failure feedback group (*p*-values < .05).

It should be noted that there was no evidence for learning-related changes in the Ne for either deterministic or probabilistic learning condition at pretest (*p*-values > .61). However, more negative Ne amplitudes reliably predicted higher post-error accuracy for the deterministic and probabilistic learning condition at both pretest [$r(35) < -.46$ and $-.36$, $p <$ .01 and .05, respectively] and posttest [$r(35) < -.52$ and $-.44$, respectively, *p*-values < .01].[15]

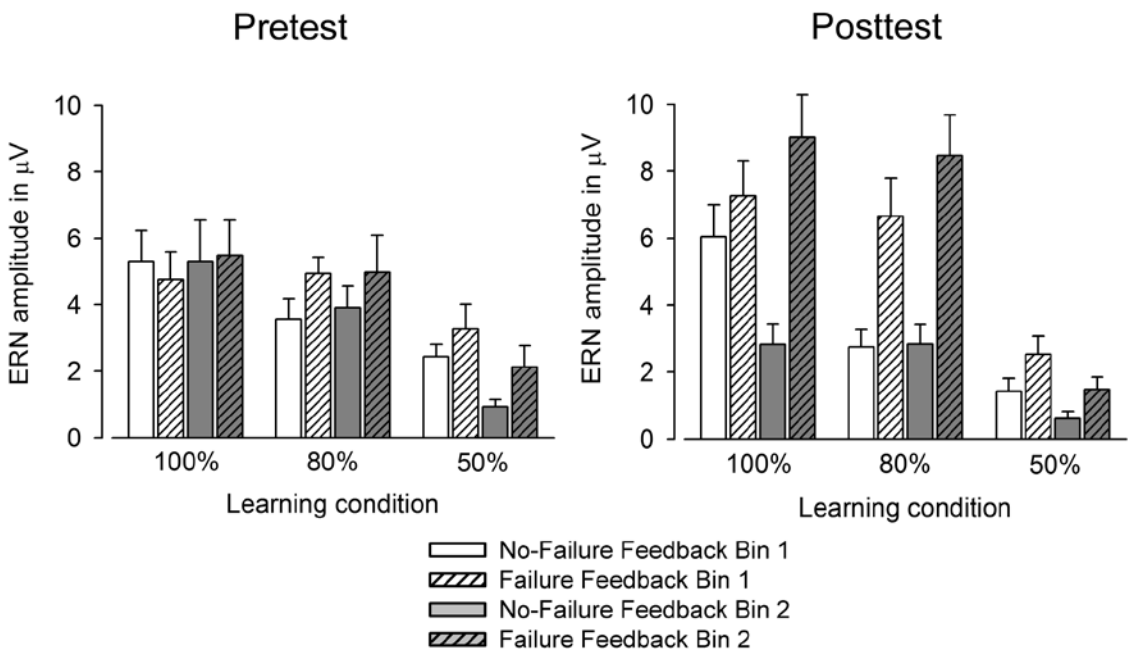


Figure 13: Bar graphs show the mean Ne amplitude at electrode FCz, separately for the three learning conditions within each group at pretest (left) and posttest (right). Error bars indicate standard error. Note that the amplitude difference in comparison to the waveforms shown in Figure 4 is due to the latency jitter across participants causing a reduction of the Ne in the grand average ERP.

[15] At posttest, significant correlations between Ne amplitude and post-error accuracy were found for the failure-feedback group (*r*-values > −.65, *p*-values < .01) but not for the no-failure-feedback group (*r*-values < −.16, *p*-values > .52). In contrast, at pretest, (marginally) significant correlations were found for the no-failure-feedback group (*r*-values > −.44, *p*-values < .07) but not for the failure feedback group (*r*-values < −.39, *p*-values > .12). Importantly, the correlation coefficients did not significantly differ between the two feedback groups at pretest (*z*-values < .80, *p*-values > .21), whereas the coefficients were significantly larger in the failure feedback group compared to the no-failure-feedback group at posttest (*z*-values > 1.8, *p*-values < .07). These findings further support the notion that failure induction promoted aversively motivated behavioural control.

Learning-related effects in the correct response-related positivity. The analysis of the positivity following correct responses yielded reliable main effects of bin [$F(1,33) = 97.03$, $p < .001$] and test phase [$F(1,33) = 21.77$, $p < .001$], indicating that the correct response-related positivity increased across the two bins and from pre- to posttest. As shown in Figure 13, the pre-post difference was greater in Bin 1 than in Bin 2, reflected in a significant interaction between test phase and bin [$F(1,33) = 18.88$, $p < .001$]. No main effect or interaction involving the factor feedback group approached significance (p-values > .20).

Moderating Influences of Personality on the Failure-related Ne Increase

To examine the role of personality, peak-to-peak amplitudes of the Ne were averaged across the two bins of pre- and posttest, respectively, and subjected to 2 (*feedback group*) × 2 (*test phase*) × 3 (*learning condition*) ANCOVAs with the trait measures as continuous moderators.

State orientation attenuates the failure-related differences in Ne amplitude. A marginally significant interaction of feedback group, test phase, learning condition, and HOM was obtained [$F(2,62) = 2.69$, $p = .076$]. Follow-up ANCOVAs that were split by test phase revealed a significant interaction of feedback group, learning condition, and HOM only for the posttest [$F(2,62) = 3.28$, $p < .05$] but not for the pretest ($p = .20$). Separate analyses for the three learning conditions found a marginally reliable interaction of feedback group and HOM for the probabilistic learning condition only ($p = .085$). State orientation[16] was associated with relatively larger Ne amplitudes in the no-failure-feedback group (action orientation: -2.13 μV, state orientation: -4.71 μV) but not in the failure-feedback-group (action orientation: -5.39 μV, state orientation: -5.01 μV). No significant correlation between HOM and Ne amplitude was found (p-values > .12). Similar ANCOVAs for BIS, BAS, and NA did not yield reliable moderating effects of the personality measures (F-values < 2.8, p-values > .10).

[16] Action and state orientation were defined by means of a median-split of HOM.

Summary of Ne Findings

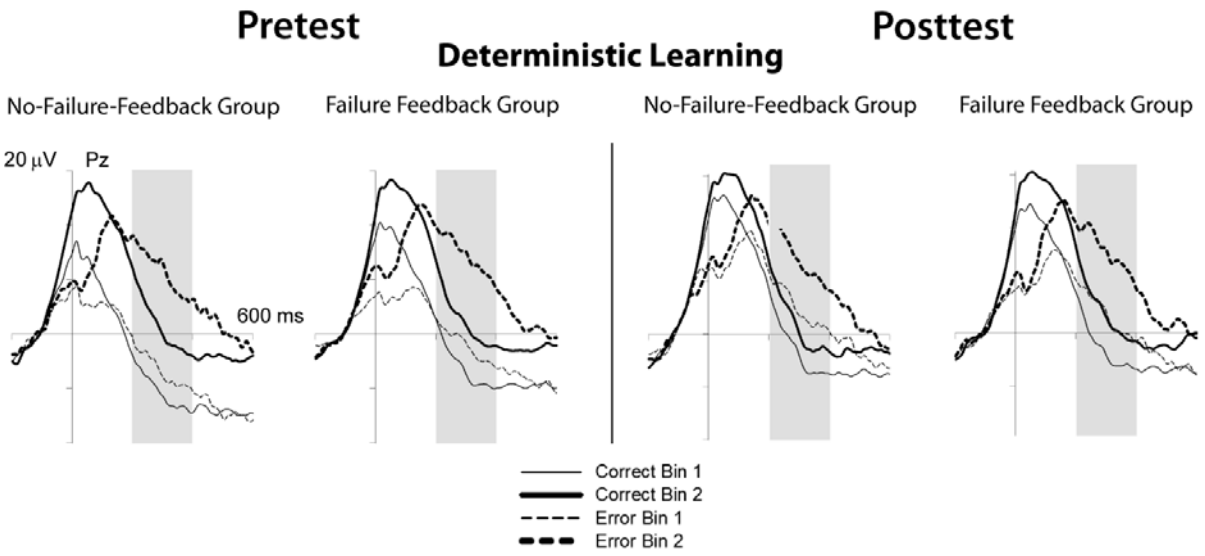
As predicted, failure induction was accompanied by an increase of the Ne for the deterministic and probabilistic learning condition. Moreover, the Ne increased across posttest both in the deterministic and probabilistic learning condition for the failure feedback group, but decreased in the deterministic learning condition for the no-failure feedback group. The failure-related Ne enhancement was not modulated by interindividual differences in punishment sensitivity or trait negative affect. State-orientation was associated with reduced between-group differences in Ne amplitude in the probabilistic learning condition at posttest. This effect primarily reflected larger Ne amplitudes for state- compared to action-oriented participants in the no-failure-feedback group. Importantly, the failure manipulation did not reliably affect the positivity following correct responses.

Error Positivity

There was a pronounced amplitude difference between erroneous and correct responses in the Pe time window for the deterministic and probabilistic learning condition with a maximum at centro-parietal sites (see Figure 15). The Pe amplitudes were analyzed using an ANOVA with the factors *feedback group* (failure feedback vs. no feedback), *test phase* (pretest vs. posttest), *learning condition (*deterministic, probabilistic, and chance condition), *bin* (Bin 1 vs. Bin 2), and *correctness* (correct vs. incorrect responses).

The ANOVA revealed significant main effects of feedback type [$F(1,33) = 65.31$, $p < .001$] and learning condition [$F(2,66) = 28.92$, $p < .001$] that were qualified by a significant interaction of feedback type and learning condition [$F(2,66) = 38.13$, $p < .001$]. As illustrated in Figure 16, these findings reflect that the Pe was evident as a more positive-going slow wave following erroneous compared to correct responses in the deterministic and probabilistic learning condition, but not in the chance condition. Contrasts confirmed that the Pe was greater in the deterministic compared to the probabilistic learning condition ($p < .05$) and in the two learning conditions compared to the chance condition ($p < .001$). Furthermore, the analysis yielded a reliable main effect of test phase [$F(1,33) = 27.14$, $p < .001$] and significant interactions of test phase and feedback type [$F(1,33) = 8.43$, $p < .01$] and test

phase, feedback type and learning condition [(2,66) = 3.51, $p < .05$]. Separate analyses for each learning condition revealed significant interactions of test phase and feedback type for the deterministic and probabilistic learning condition only (p-values < .05). Although the ERPs to both erroneous and correct responses were more positive-going at posttest (p-values < .01), these interactions indicated that the increase was more pronounced for erroneous responses (see Figure 14).



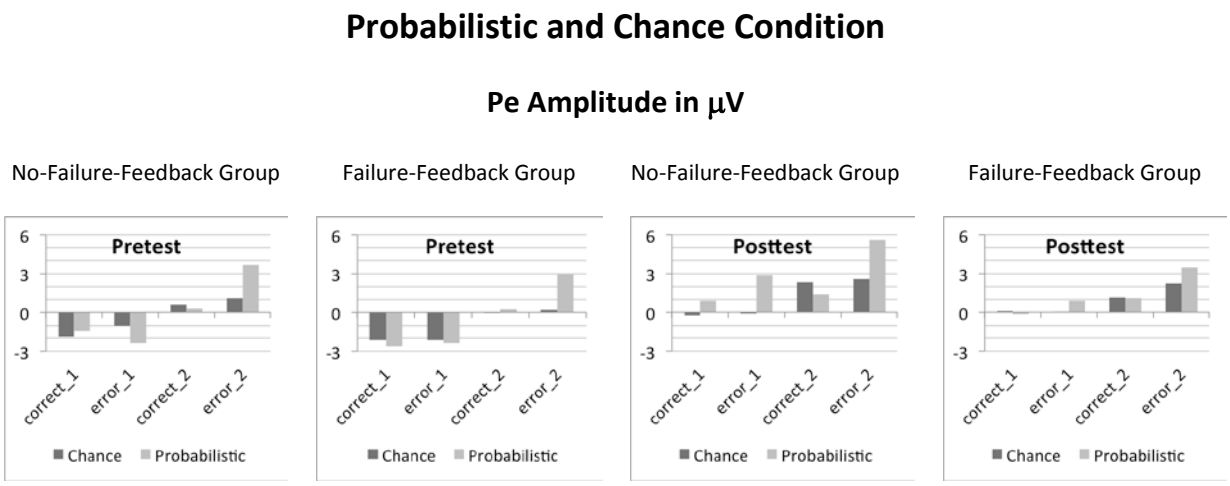


Figure 14: The upper panel shows the response-locked ERPs in the deterministic learning condition at electrode Pz. Grey bars highlight the time window used for Pe analysis. The lower panel shows the mean Pe amplitdes (in μV) in the probabilistic learning and chance condition, separarely for correct and incorrect trials.

Learning-related effects in the Pe. The analysis yielded a reliable main effect of bin [$F(1,33) = 113.32$, $p < .001$] and significant interactions of bin and feedback type [$F(1,33) = 40.47$, $p < .001$] and bin, feedback type, and learning condition [$F(2,66) = 10.45$, $p < .001$]. Decomposing the interaction within each learning condition revealed that the Pe increased with learning in the deterministic ($p < .001$) and probabilistic learning condition ($p < .001$) but not in the chance condition ($p = .66$) (see Figure 14). However, there was no significant relationship between the Pe and post-error accuracy at either pre- or posttest (rs < .14, p-values > .44).

Summary of Pe findings

The Pe increased both with feedback validity and learning. Furthermore, the Pe increased from pre-to posttest. Most critically, however, no effects of failure-induction on the Pe were obtained. Moreover, ANCOVAs for BIS, BAS, NA, and HOM indicated that failure-related effects did not rely on influences of personality (F-values < 2.4, p-values > .13).

Feedback-related Negativity

Figure 15 shows the feedback-locked ERPs in the three learning conditions at pre- and posttest, separately for the two experimental groups. At pretest, the FRN was clearly evident as a fronto-centrally distributed negative-going deflection that was larger following positive compared to negative feedback. This difference between correct and incorrect-feedback trials was most pronounced in the chance condition. Both the peak-to-peak amplitude and the mean amplitude measures of the FRN were subjected to an ANOVA with the factors *feedback group* (failure feedback vs. no feedback), *test phase* (pretest vs. posttest), *learning condition (*deterministic, probabilistic, and chance condition), *bin* (Bin 1 vs. Bin 2), and *feedback type* (positive vs. negative feedback). Below, I will first report the results for the peak-to-peak measures, followed by the results for the mean amplitudes measures.

Peak-to-peak Analysis of the FRN

The analysis revealed a significant main effect of feedback type [$F(1,33) = 46.15$, $p < .001$] that was qualified by an interaction of learning condition and feedback type [$F(2,66) = 4.53$, $p < .05$], suggesting that the amplitude difference between positive and negative feedback was the larger the more invalid the feedback (see Figure 15). However, contrasts of deterministic vs. probabilistic learning condition as well as deterministic and probabilistic learning condition vs. chance condition failed to obtain significant differences (p-values > .17). No further effects approached significance.

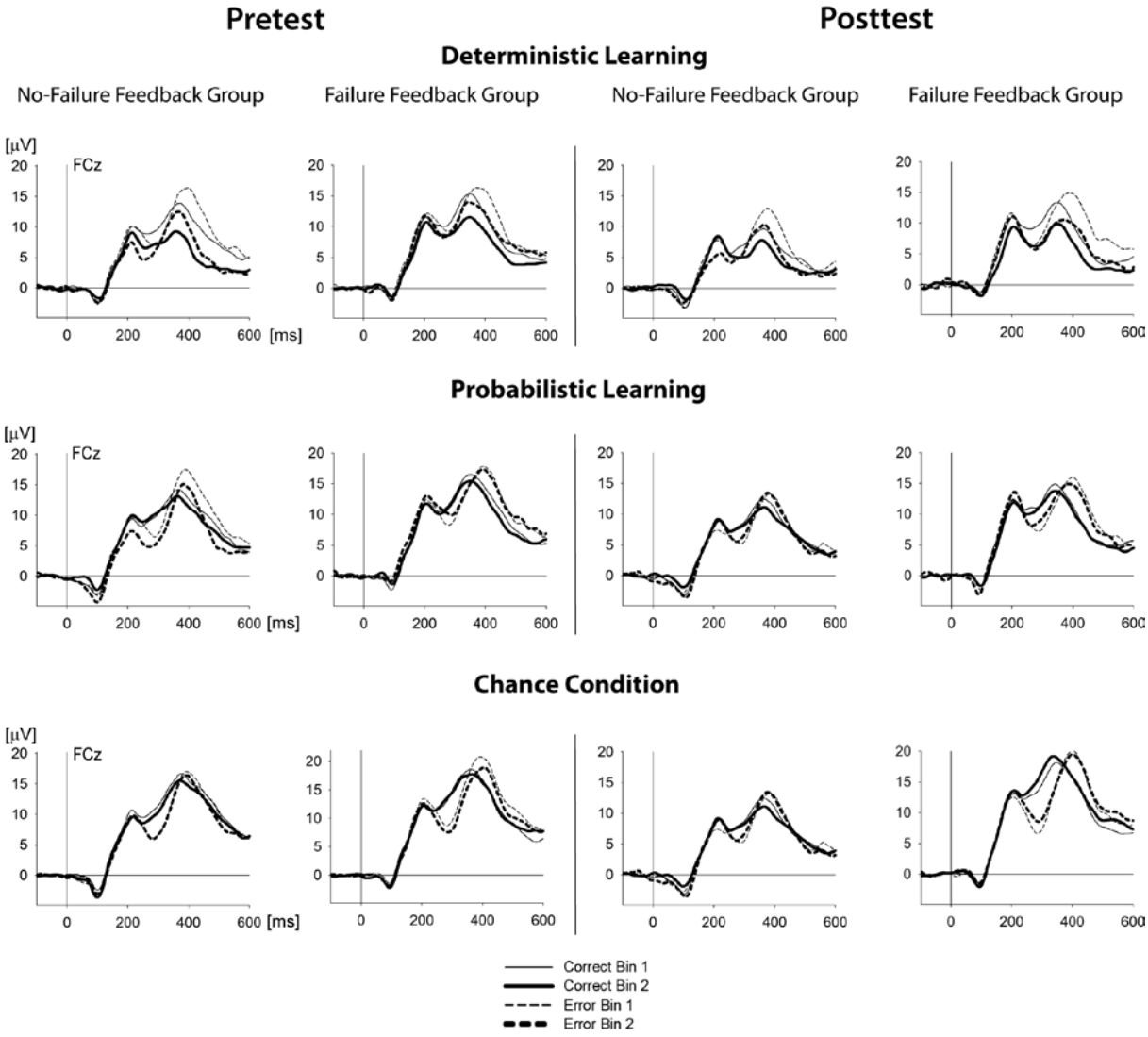


Figure 15: Feedback-locked ERPs to correct (solid lines) and incorrect responses (dashed lines) displayed separately for the no-failure feedback group and failure feedback group and the two halves of pretest (left) and posttest (right).

Mean Amplitude Analysis of the FRN

Similar to the peak-to-peak amplitudes, the analysis of mean amplitudes revealed a reliable main effect of learning condition [$F(2,66) = 20.97$, $p < .001$], a reliable main effect of feedback type [$F(1,33) = 46.15$, $p < .001$], and a significant interaction between learning condition and feedback type [$F(2,66) = 9.12$, $p < .01$, $\varepsilon = .64$]. Contrasts showed that the amplitude difference between positive and negative feedback, i.e., the FRN, was larger for the probabilistic compared to the deterministic learning condition [$F(1,33) = 5.02$, $p < .05$] as well as for the chance condition compared to deterministic and probabilistic learning condition [$F(1,33) = 14.27$, $p < .01$]. Follow-up ANOVAs that were split by feedback type yielded a significant main effect of learning condition for positive ($p < .001$) but not for negative feedback ($p = .12$).

General effects of failure on the FRN. A significant interaction between feedback group, feedback type, and test phase [$F(1,33) = 5.60$, $p < .05$] indicated that the FRN was affected by the failure-manipulation. As could be seen from Figure 15, at posttest, the difference between FRN and feedback-locked positivity was larger for the failure-feedback group than for the no-failure-feedback-group. Separate analyses for the two test phases, however, failed to obtain a significant interaction between feedback group and feedback type both at pretest ($p = .89$) and posttest ($p = .12$). In addition, decomposing the interaction within each group did not reveal significant interactions of test phase and group for either positive ($p = .13$) or negative feedback ($p = .75$).

Learning-related effects of failure on the FRN. The analysis yielded a significant main effect of bin [$F(1,33) = 4.68$, $p < .05$] and a significant interaction between learning condition and bin [$F(2,66) = 10.23$, $p < .001$, $\varepsilon = .80$], reflecting a more pronounced amplitude reduction in the ERPs to positive and negative feedback across the two bins in the deterministic learning condition (see Figure 15). This was confirmed by contrasts that revealed a significant larger difference between Bin 1 and 2 in the deterministic compared to the probabilistic learning condition [$F(1,33) = 14.88$, $p < .01$] as well as in the two learning conditions compared to the chance condition [$F(1,33) = 5.45$, $p < .05$]. Moreover, marginally

significant interactions between learning condition, bin, and test phase [$F(2,66) = 2.48$, $p = .092$] and feedback group, learning condition, bin, and test phase [$F(2,66) = 3.04$, $p = .054$] suggested that the amplitude modulation across bins was affected by failure-manipulation. A follow-up ANOVA that was split by test phase yielded a significant interaction between feedback group, learning condition, and bin for the posttest [$F(2,66) = 3.71$, $p < .05$] but not for the pretest ($p = .87$). Figure 15 illustrates that only the failure-feedback-group showed a marked amplitude decrease across bins in the deterministic learning condition at posttest, reflected in a significant interaction between learning condition and bin ($p < .01$). This interaction was not observed for the no-failure-feedback group ($p = .37$), which instead showed reduced amplitudes in both halves of posttest. It should be noted that the learning-related effects did not differ for positive and negative feedback. Moreover, in contrast to the Ne, the FRN did not reliably predict post-error accuracy posttest (rs < .27, p-values > .12).

Summary of FRN Findings

The mean amplitude analysis of the FRN suggested that failure-induction resulted in enhanced sensitivity to feedback, reflected in relatively larger amplitude differences between the ERPs to positive and negative feedback in the failure-feedback group at posttest. Moreover, amplitude of the ERPs to both feedback types was larger in the failure-feedback-group compared to the no-failure-feedback-group in the deterministic learning condition during the first half of posttest. The latter finding possibly indicates that participants in the failure-feedback-group paid relatively more attention to the feedback at the beginning of learning. ANCOVAs with BIS, BAS, NA, and HOM as continuous moderator did not reveal significant influences of personality on failure-related changes in feedback processing[17] (F-values < 2.7, p-values > .11).

Summary Experiment 1

The first experiment revealed two main findings concerning the failure feedback group and the no-failure feedback group. First, it was found that failure induction resulted in an

[17] ANCOVAs were conducted both for peak-to-peak and mean amplitude measures of the FRN.

increase of the Ne for the deterministic and probabilistic learning condition at posttest. Importantly, the amplitude enhancement was not accompanied by higher overall accuracy and therefore cannot simply be explained by changes in error expectancy. Instead, the Ne increase was associated with higher post-error accuracy, that is, the participants were more likely to correct an erroneous response on the next presentation of a stimulus. Thus, failure induction appears to increase the impact of error signals on behavioral adaptation during subsequent feedback-based learning. Furthermore, punishment sensitivity moderated the effects of failure on subsequent learning performance. High punishment sensitivity predicted reduced posttest accuracy in the failure-feedback group. By contrast, no significant relationship between punishment sensitivity and posttest performance was found in the no-failure-feedback group. There was also no evidence that punishment sensitivity modulated the impact of failure on neural mechanisms of error processing as reflected in the Ne.

Second, a pronounced decrease in Ne amplitude was observed in the second half of posttest in the deterministic learning condition for the no-failure-feedback group. At the same time, there was no significant pre-post accuracy decrease for either learning condition in this group. The latter result suggests that linking posttest performance to intelligence successfully motivated participants to maintain task engagement. Moreover, the present data indicate that lower responsivity of the error monitoring system – as reflected in the decrease of the Ne from the first to the second half of posttest – is not necessarily associated with performance deficits.

In addition, the analysis of mean-amplitude measures of the FRN indicated that failure-induction lead not only to an increase of the Ne but also to enhanced sensitivity to external performance feedback, reflected in a relative increase in the FRN, i.e., a more pronounced differentiation between positive and negative feedback. By contrast, no failure-related modulations in Pe amplitude were observed. Moreover, the analyses did not reveal clear evidence in support of the notion that individual differences in trait level punishment sensitivity, negative affect, and action vs. state orientation moderate the effects of the failure manipulation on error- and feedback processing.

6. Experiment 2

The results of Experiment 1 show that failure induction results in an enhanced responsivity to internal indicators of errors as well as external performance feedback during a subsequent learning task. However, it remains unknown to what extent the observed effects reflect the increased self-relevance of posttest that was linked to intelligence. To explore this issue, the second experiment used a different posttest instruction: participants were simply told that they were going to perform the same task as in pretest.

Research Predictions

Based on the findings by Boksem and colleagues on mental fatigue due to sustained task performance (Boksem et al., 2006a; Tops and Boksem, 2010), it was predicted that subjects in the no-failure-feedback group disengage from task over the course of posttest. Thus:

Prediction 1: In contrast to Experiment 1, accuracy as well as Ne amplitude was expected to decrease from pre- to posttest in the no-failure-feedback group, since the cognitive system becomes less efficient in monitoring ongoing behavior.

It seemed plausible to assume that motivational disengagement also results in less differentiated processing of positive and negative feedback, i.e., in a decrease in FRN amplitude. Yet, impaired learning should be associated with larger feedback-related PEs. According to the R-L theory this would lead to a *larger* FRN.

Prediction 2: If disengagement affects the FRN, this should be particularly evident in the chance condition, which is not subject to learning-related changes.

Boksem and colleagues (2006a) did not report whether the Pe is affected by mental fatigue, but they found an increase in latency of the stimulus-locked P300 with time on task. Given that the Pe has been proposed to reflect a response-related P300 (cf. Overbeek et al., 2005), one might reason that the Pe is also sensitive to disengagement. This prediction is

consistent with other theoretical accounts that link the Pe to the motivational significance or conscious recognition of an error.

Prediction 3: It is conceivable that the Pe decreases from pre-to posttest in the no-failure-feedback group.

Although Luu et al. (2000) reported a relationship between negative affect and motivational disengagement, threatening participants' self-worth by self-relevant failure appears to result in an "inherent" motivational boost at posttest (Brunstein, 2000). Hence, action monitoring should be less affected by motivational disengagement in the failure feedback group than in the no-failure feedback group:

Prediction 4: (a) At posttest, the failure-feedback group shows better performance and larger Ne amplitudes than the no-failure-feedback group. *(b)* The hypotheses concerning the FRN and Pe were more tentative. Generally, effects of disengagement on the FRN and the Pe (see Predictions 2 and 3) should be less pronounced in the failure-feedback group compared to the no-failure-feedback group.

Moreover, the findings from Experiment 1 suggest that failure feedback specifically promotes a reactive, error-driven motivational engagement. It remains an open question, however, whether there are also pre-post increases in Ne amplitude, FRN amplitude, and post-error accuracy when posttest is described in neutral terms.

Personality did not appear to moderate the impact of failure on the ERP-measures in Experiment 1. However, personality measures, such as punishment sensitivity, have been shown to preserve task engagement (Tops & Boksem, 2010). Thus, the corresponding predictions are:

Prediction 6: (a) High punishment sensitivity is associated with less pronounced pre-post-decreases in performance and Ne amplitudes in the no-failure-feedback group. *(b)* As in Experiment 1, high punishment sensitivity should predict worse performance in the failure-feedback group.

Methods

Participants

Sixty-five undergraduate students were recruited for participation in this study by applying the same criteria as in Experiment 1. None of them had participated in the first experiment. Twenty-three participants quit the experiment after the pretest because they did not commit enough errors to obtain reliable ERP-measures (>14 error trials in both halves of pre- and posttest). Data from a further 9 participants had to be excluded because of poor performance in the learning task (4), excessive artifacts (1), and technical problems during EEG recording (4). The final sample thus consisted of 16 participants (11 women, mean age = 21.0 years; age range = 19 to 28 years) in the failure feedback group and 17 participants (12 women, mean age = 22.4 years; age range = 18 to 29 years) in the no-failure feedback group.

Stimuli, Tasks, and Procedure

Stimuli and tasks were the same as in the first experiment. Procedural details were also identical to those in Experiment 1, except for one important difference: Before starting with the learning task at posttest participants received the same instruction as at pretest, i.e., the task was not linked to intelligence, but was described in neutral terms.

Results

Control Analyses/Manipulation Check

Table 2 shows the results of the psychometric tests and the questionnaires for the no-feedback and the failure feedback group. There were no group differences with respect to the psychometric measures (all p-values > .37). As in Experiment 1, participants in the failure feedback group reported more negative feelings [F (1,31) = 14.49, p < .002] and were less satisfied with their performance on the visual search task [F (1,31) = 13.28, p < .002].

Tabelle 2. Results of the psychometric measures, the mood scale (BfS), self-evaluation for the visual search task and motivational involvement/rumination at posttest (means and standard deviations) for the no-failure feedback group and the failure feedback group (Experiment 2)

Measure	No-failure feedback group	Failure feedback group
Cognitive variables		
DSS	61.59 (9.01)	61.88 (8.41)
Spot-a-word	21.06 (4.49)	19.81 (3.31)
Digit ordering	8.53 (2.43)	8.94 (2.05)
Affect & action control		
PA	36.29 (4.78)	37.38 (4.76)
NA	21.47 (4.61)	23.19 (7.83)
BIS	2.88 (0.49)	2.84 (0.41)
BAS	3.16 (0.29)	3.18 (0.31)
HOM[a]	4.12 (2.00)	4.75 (2.65)
Mood (post-manipulation)		
BfS[b]	10.71 (5.55)	18.06 (5.54)
Final questionnaire		
Self-evaluation	3.53 (0.82)	2.59 (0.64)
Involvement	3.43 (0.62)	3.32 (0.69)
Rumination	2.09 (0.92)	1.59 (0.73)

a Note that higher scores indicate action orientation while lower scores indicate state-orientated behaviour.

b Higher scores indicate more negative feelings.

Accuracy Data

Response latencies faster than 256 ms (> 2SD) or exceeding the response deadline were excluded from further analyses[18]. Accuracy data were analysed using the same ANOVA design as in Experiment 1. As can be seen from Figure 16, a significant main effect of learning condition [$F(2,62) = 250.66$, $p < .001$] indicated that accuracy was highest for the deterministic learning condition and lowest for the chance condition (p-values < .001).

Learning-related effects. The analysis yielded a significant main effect of bin [$F(5,155) = 26.30$, $p < .001$, $\varepsilon = .68$] that was qualified by an interaction between learning condition and bin [$F(10,310) = 4.38$, $p < .001$]. Moreover, an interaction between test phase and bin [$F(5,155) = 4.29$, $p < .01$, $\varepsilon = .82$] and a marginally significant interaction between test phase, bin, and learning condition [$F(10,310) = 1.83$, $p = .088$, $\varepsilon = .66$] indicated that the course of learning differed between pre- and posttest. Polynomial contrasts revealed linear increases in accuracy for both learning conditions across pretest only (p-values < .01). At posttest, accuracy varied across the bins following a predominantly cubic ($p < .001$) and quadratic ($p < .01$) trend for the deterministic and probabilistic learning condition, respectively (see Figure 16).

Effects of failure manipulation. In contrast to Experiment 1, a significant interaction between feedback group and test phase was obtained [$F(1,31) = 8.32$, $p < .01$]. Separate analyses for the two groups revealed that accuracy significantly decreased from pre- to posttest in the no-failure feedback group [$F(1,16) = 9.85$, $p < .01$], whereas no pre-post difference in accuracy was found for the failure feedback group ($p = .23$).

[18] The deadline was exceeded by 2.2% (no-failure feedback group) and 2.3% (failure feedback group) of the responses. At pretest, mean RTs on correct and incorrect trials were 446 ms (*SD* = 33 ms) and 440 ms (*SD* = 30 ms) for the no-failure feedback group and 427 ms (*SD* = 23 ms) and 423 ms (*SD* = 22 ms) for the failure feedback group. At posttest, mean RTs on correct and incorrect trials were 404 ms (*SD* = 32 ms) and 393 ms (*SD* = 26 ms) for the no-failure feedback group and 411 ms (*SD* = 27 ms) and 403 ms (*SD* = 26 ms) for the failure feedback group.

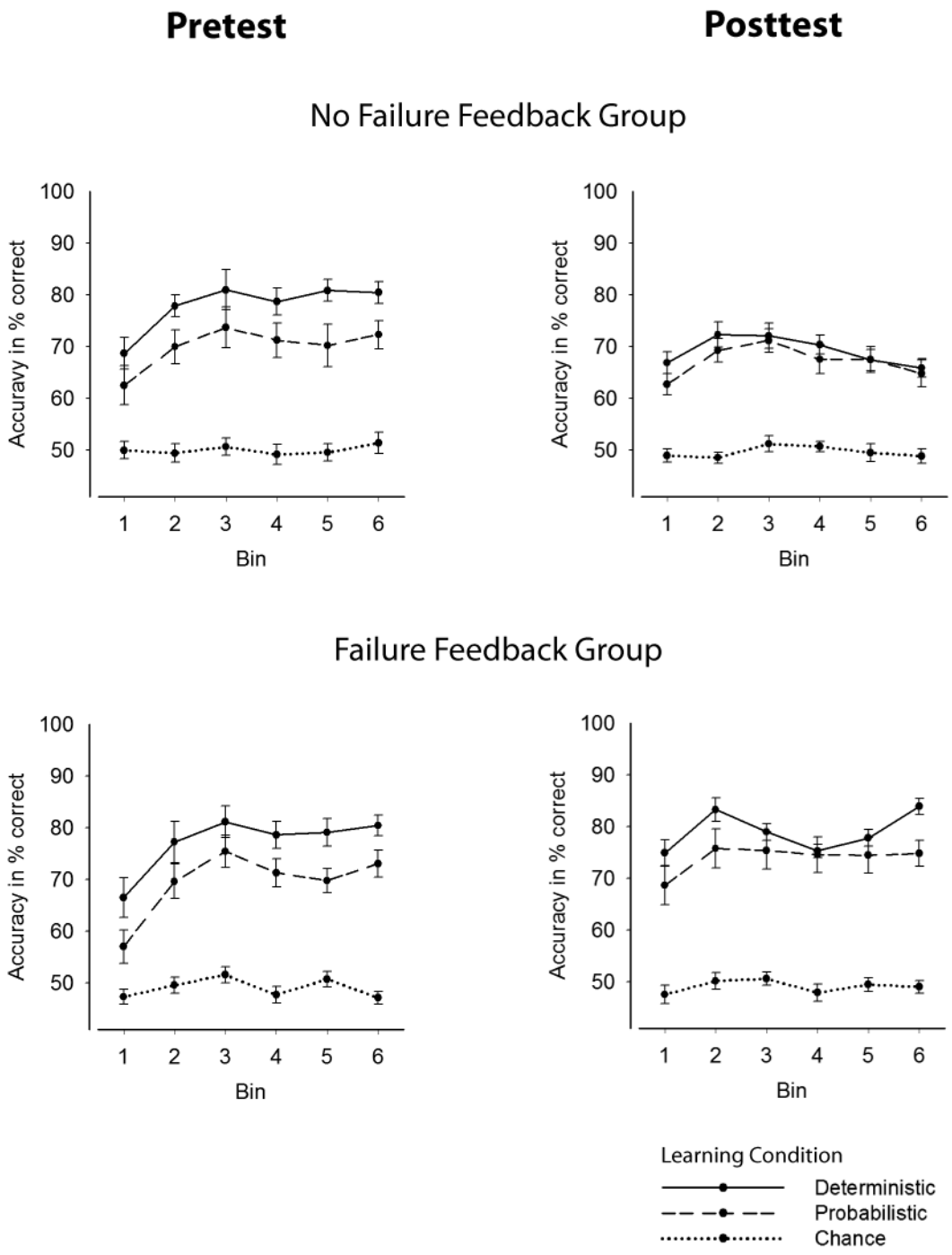


Figure 16: Mean accuracy learning curves for the three learning conditions displayed separately for the no-failure feedback group and failure feedback group at pretest (left) and posttest (right). Error bars indicate standard error.

Post-error Accuracy Data

As in Experiment 1, post-error accuracy rates were subjected to an ANOVA with the factors *feedback group*, *test phase*, and *learning condition*. Figure 17 shows that post-error accuracy increased with feedback validity [$F(2,62) = 82.86$, $p < .001$].

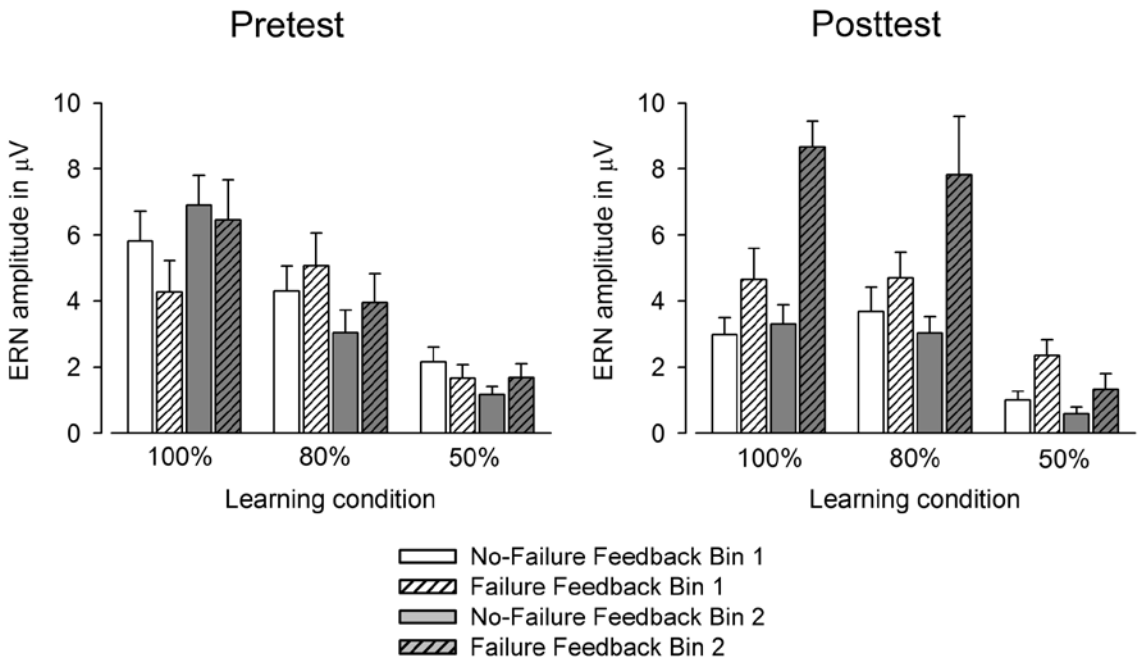


Figure 17: Mean post-error accuracy rates for the three learning conditions at pretest and posttest displayed separately for the no-failure feedback group (left) and failure feedback group (right). Error bars indicate standard error.

Effects of failure manipulation. The analysis yielded a significant interaction between feedback group and test phase [$F(1,31) = 11.14$, $p < .01$]. Decomposing the interaction revealed that post-error accuracy reliably decreased from pre- to posttest for the no-failure feedback group [$F(1,16) = 5.05$, $p < .05$], but increased for the failure feedback group [$F(1,15) = 6.25$, $p < .05$] (see Figure 18). In contrast to Experiment 1, the analysis of post-error RT revealed a significant interaction between feedback group and test phase [$F(1,31) = 9.46$, $p < .01$], reflecting a smaller pre-post decrease in post-error RT for the failure feedback group than for the no-failure feedback group. Although there were no reliable between-group differences at pre- or posttest (p-values > .10), this finding suggests that the failure feedback group increased post-error accuracy at the cost of relatively longer post-error RT.

Summary of Behavioural Findings

Whereas the no-failure feedback group showed worse performance at posttest, overall accuracy did not differ between pre- and posttest in the failure feedback group. Similar to Experiment 1, non learning-related dynamics of performance became prevalent for both groups at posttest. While these findings are likely to reflect reduced task engagement towards the end of posttest in the no-failure feedback group, the pattern of performance in the failure feedback group suggests that participants produced relatively more errors of commission during posttest, particularly in the deterministic learning condition. In support of this, RTs decreased from pre- to posttest and were faster in the deterministic than in the probabilistic learning condition on error trials (*p*-values < .001). Consistent with Experiment 1, failure induction was specifically associated with increased error-correction rates. Whereas failure-induction was associated with learning impairments in highly punishment sensitive individuals in Experiment 1, trait variables did not reliably moderate the effects of failure on overall or post-error accuracy in Experiment 2 (*F*-values < 2.6, *p*-values > .12).

ERP data

Error Negativity

Figure 18 shows the ERPs to correct and incorrect responses, separately for both feedback groups in the first and second half of pre- and posttest (see also Figure 20). The peak-to-peak measures of the Ne and the mean amplitude of the correct response-related positivity were analyzed using the same ANOVA design as in Experiment 1. Consistent with Experiment 1, both the Ne and the correct response-related positivity increased with feedback validity [$F(2,62) = 47.89$ and 61.42, respectively, *p*-values < .001]. Below, the failure-related effects on the Ne are reported first, followed by the results for the correct-response related positivity.

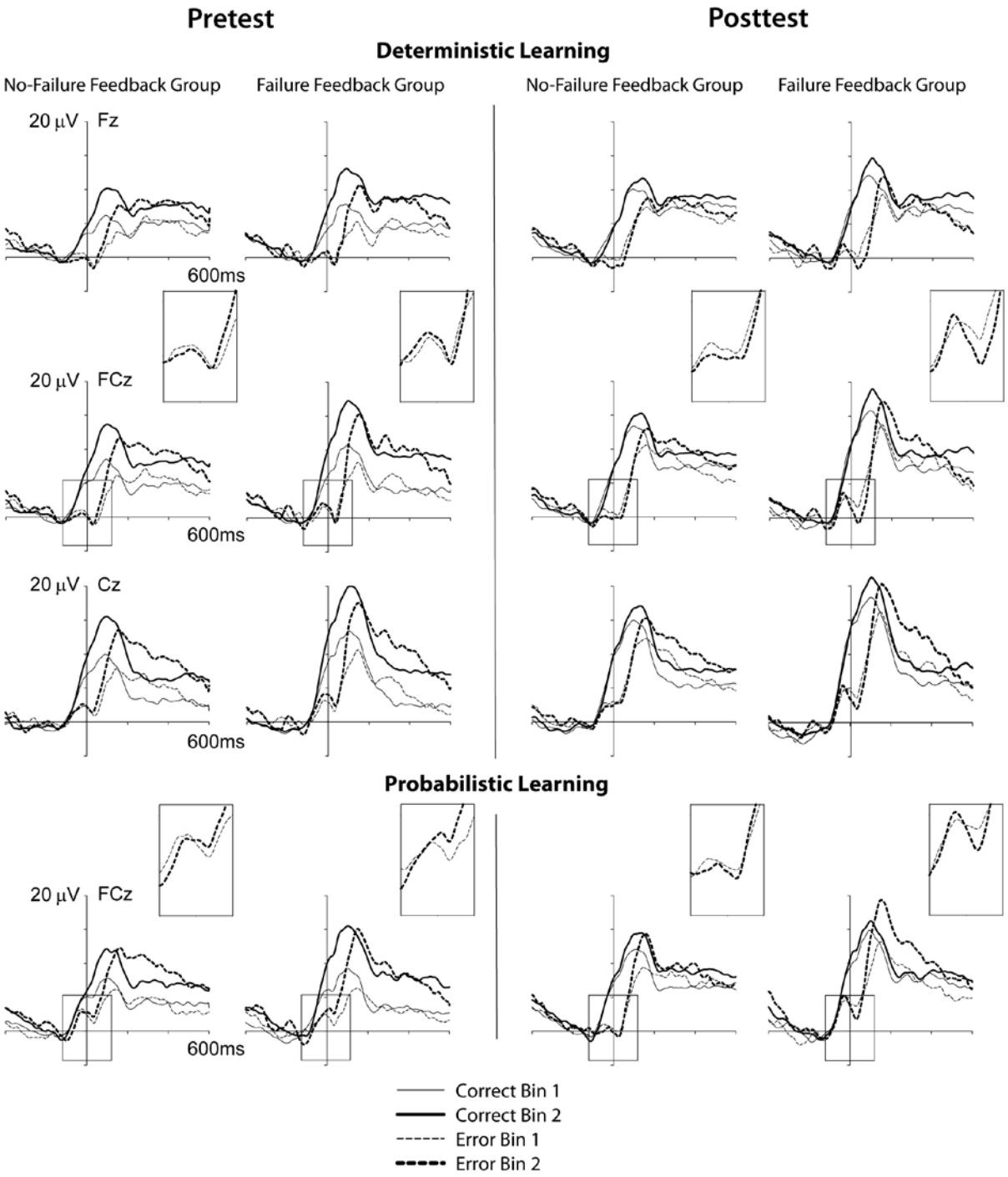


Figure 18: Response-locked ERPs to correct (solid lines) and incorrect responses (dashed lines) displayed separately for the no-failure feedback group and failure feedback group and the two halves of pretest (left) and posttest (right). The upper panels show the ERPs in the deterministic learning condition at electrode sites Fz, FCz, and Cz. Small boxes highlight the Ne effect at FCz. The lowest panel shows the ERPs in the probabilistic learning at electrode FCz.

General effects of failure. The analysis revealed a reliable main effect of feedback group [$F(1,31) = 4.74$, $p < .05$] that was qualified by a significant interaction between feedback group and test phase [$F(1,31) = 17.41$, $p < .001$]. As illustrated in Figure 18 (see

also Figure 19), the Ne decreased from pre- to posttest for the no-failure feedback group [$F(1,16) = 10.97$, $p < .01$], but increased from pre- to posttest for the failure feedback group [$F(1,15) = 6.14$, $p < .05$].

Learning-related effects of failure. Similar to Experiment 1, I found significant interactions between learning condition and bin [$F(2,62) = 11.88$, $p < .001$] and test phase, feedback group, learning condition, and bin [$F(2,62) = 3.34$, $p < .05$]. Separate pre- and posttest analyses yielded a significant interaction between feedback group, learning condition, and bin for posttest [$F(2,62) = 4.93$, $p < .05$, $\varepsilon = .84$] but not for pretest ($F < 1$). At posttest, a significant interaction between learning condition and bin was obtained for the failure feedback group only [$F(2,30) = 6.56$, $p < .01$; no-failure feedback group: $p = .30$]. Figure 19 shows that the Ne increased over the course of posttest for the deterministic and probabilistic learning condition (p-values < .05) but tended to decrease for the chance condition ($p = .056$).

Consistent with Experiment 1, greater Ne amplitudes predicted higher post-error accuracy for deterministic and probabilistic learning condition at both pretest [$r(33) = -.38$ and $-.40$, respectively, p-values< .05] and posttest [$r(33) = -.54$ and $-.67$, respectively, p-values< .01][19].

Effects of learning on the correct response-related positivity. The positivity on correct trials increased from Bin 1 to Bin 2 [$F(1,31) = 70.61$, $p < .001$] and from pre- to posttest [$F(1,31) = 16.13$, $p < .001$]. As was indicated by a significant interaction between bin and test

[19] However, separate analyses for the two groups and the two test phases revealed a more inconsistent pattern than in Experiment 1. For the no-failure-feedback group, (marginally) significant correlations between Ne and post-error accuracy were found both at pre- and posttest in the deterministic learning condition, [$r(17) = -.48$ and $-.67$, $p = .053$ and <.01, respectively], but only at posttest in the probabilistic learning condition [$r(17) = -.32$ and $-.53$, $p = .21$ and <.05, respectively]. For the failure-feedback group, in contrast, no reliable correlation between Ne and post-error accuracy was found at either pre- or posttest in the deterministic learning condition [$r(16) = -.07$ and $-.36$, $p = .80$ and .17, respectively], but there were (marginally) significant relations both at pre- and posttest in the probabilistic learning condition [$r(16) = -.49$ and $-.77$, $p = .056$ and <.001, respectively]. Notably, the correlation coefficients did not significantly differ between the two feedback groups at either pre- or posttest (z-values < 1.19, p-values > .23).

phase [$F(1,31) = 20.83$, $p < .001$], the amplitude difference between the two bins was larger at pretest (see Figure 18). No effect of failure manipulation was found (p-values > .18).

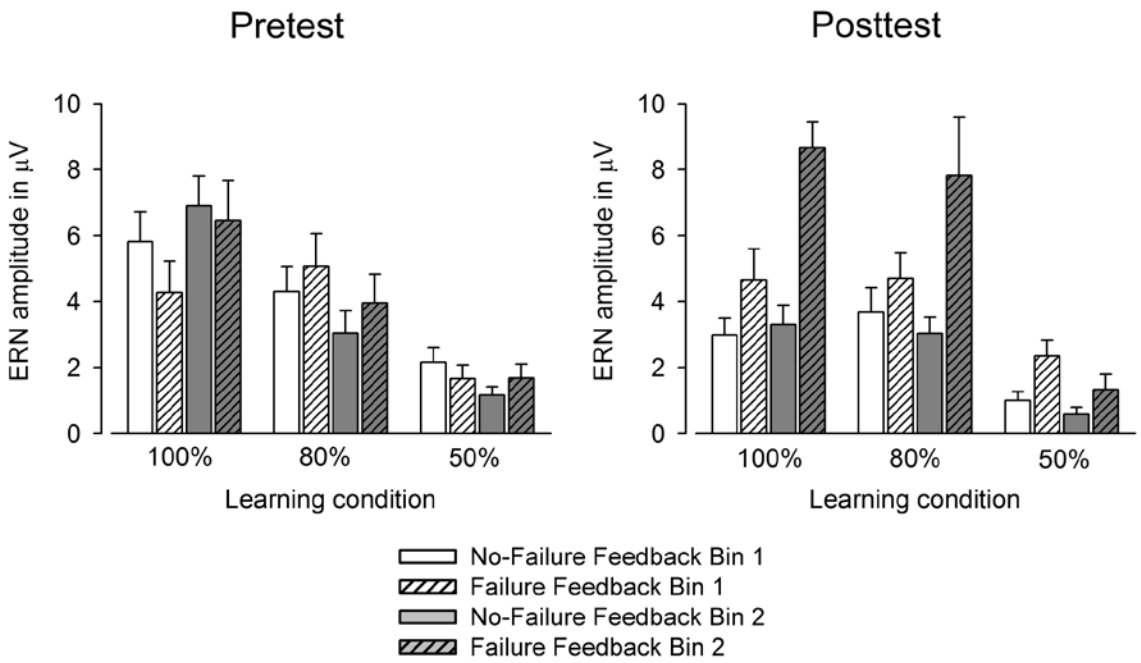


Figure 19: Bar graphs show the mean Ne amplitude at electrode FCz, separately for the three learning conditions within each group at pretest (left) and posttest (right). Error bars indicate standard error. Note that the amplitude difference in comparison to the waveforms shown in Figure 7 is due to the latency jitter across participants, resulting in a reduction of the Ne in the grand average ERP.

Summary of Ne Findings

In line with Experiment 1, these findings demonstrate that failure feedback resulted in an enhancement of the Ne. Moreover, there was an increase in Ne amplitude over the course of posttest for the deterministic and probabilistic learning condition in the failure feedback group. In contrast, the Ne decreased from pre- to posttest for the no-failure feedback group. This decrease was much more pronounced than in Experiment 1, where the no-failure group showed a reduced Ne for the deterministic learning condition in the second half of posttest only. In contrast to Experiment 1, no evidence for a moderating role of state orientation (or any other trait variable) was found (F-values < 2.4, p-values > .10).

Error Positivity

Figure 20 shows the ERPs to correct and incorrect responses at electrode Pz, separately for the three learning conditions and the two feedback groups at pre- and posttest. The Pe was clearly evident as a centro-parietally distributed slow-wave that was more positive-going after erroneous compared to correct responses in the deterministic and probabilistic learning condition. The Pe was analyzed using the same ANOVA design as in Experiment 1. The analysis yielded reliable main effects of feedback type [$F(1,31) = 51.17$, $p < .001$] and learning condition [$F(2,62) = 47.90$, $p < .001$, $\varepsilon = .87$]. Similar to Experiment 1, a significant interaction of feedback type and learning condition [$F(2,62) = 42.50$, $p < .001$, $\varepsilon = .70$] indicated that the Pe was sensitive to feedback validity. Contrasts showed that the Pe was larger in the deterministic compared to the probabilistic learning condition [$F(1,31) = 4.38$, $p < .05$] and in the two learning conditions compared to the chance condition [$F(1,31) = 51.78$, $p < .001$], in which no Pe was observed (see Figure 21). Moreover, a marginally significant interaction of feedback type, learning condition, test phase, and feedback group was obtained [$F(2,62) = 2.88$, $p = .075$, $\varepsilon = .82$]. Decomposing the interaction within each group revealed a marginally significant interaction of feedback type, learning condition, and test phase in the failure-feedback group only [$F(2,30) = 2.61$, $p = .090$]. As can be seen from Figure xx, this finding reflects that the Pe tended to increase from pre- to posttest in the failure-feedback group.

Learning-related effects of failure. Figure 20 illustrates that the Pe increased over the course of pretest in the deterministic and probabilistic condition (but not in the chance condition) in both feedback groups. At posttest, however, a learning-related increase in Pe amplitude was only observed for the failure-feedback group. Accordingly, the analysis revealed a significant main effects of bin [$F(1,31) = 66.53$, $p < .001$] as well as significant interactions of bin and feedback type [$F(1,31) = 25.63$, $p < .001$], bin, feedback type, and learning condition [$F(2,62) = 4.49$, $p < .05$, $\varepsilon = .85$], bin, feedback type, learning condition, and feedback group [$F(2,62) = 5.94$, $p < .01$, $\varepsilon = .85$], bin, feedback type, learning condition, feedback group, and test phase [$F(2,62) = 3.81$, $p < .05$]. Follow-up ANOVAs that were split

by test phase revealed a significant interaction of bin, feedback type, learning condition, feedback group for the posttest [$F(2,62) = 10.14$, $p < .01$, $\varepsilon = .78$], but not for the pretest ($p = .77$). Decomposing the interaction within each group yielded a significant interaction of bin, feedback type, and learning condition for the failure-feedback group only [$F(2,30) = 10.14$, $p < .01$, $\varepsilon = .71$]. As shown in Figure 20, this interaction indicates that the Pe increases over the course of posttest in the two learning conditions for the failure-feedback group. As in Experiment 1, there were no significant correlations between Pe amplitude and post-error accuracy at pre- or posttest (rs < .18, p-values > .30).

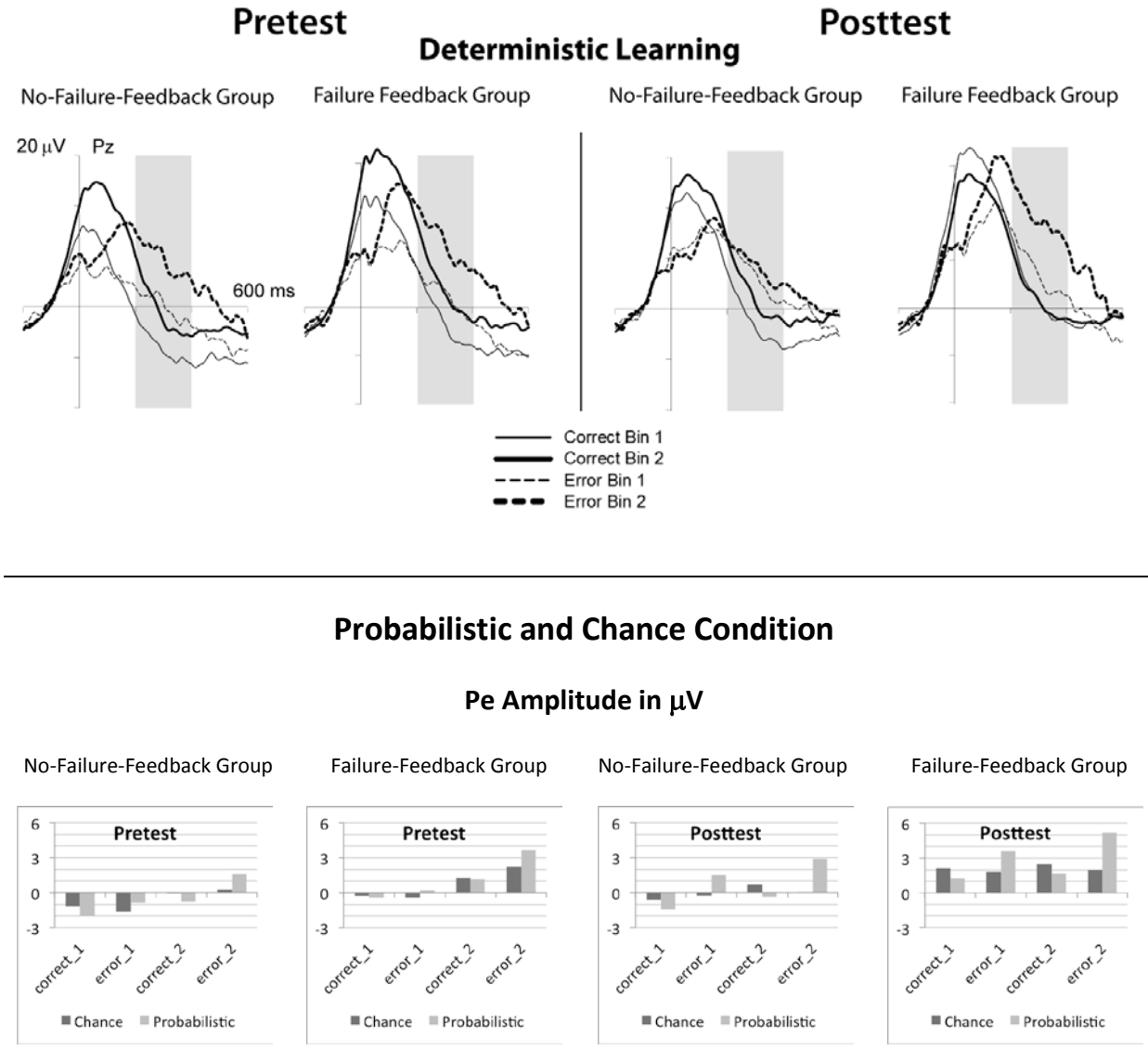


Figure 20: The upper panel shows the response-locked ERPs in the deterministic learning condition at electrode Pz. Grey bars highlight the time window used for Pe analysis. The lower panel shows the mean Pe amplitudes (in μV) in the probabilistic learning and chance condition, separately for correct and incorrect trials.

Summary of Pe Findings

In line with Experiment 1, the Pe increased with feedback validity. Moreover, the Pe increased with learning in both groups at pretest, but this was only the case for the failure-feedback group at posttest. As in Experiment 1, trait variables did not moderate the effects on Pe-amplitude (*F*-values < 2.1, *p*-values > .14).

Feedback-related Negativity

Feedback-locked ERPs are presented in Figure 21. The FRN was evident as a more negative-going deflection following negative compared to positive feedback at fronto-central sites and decreased with feedback validity. Similar to the first experiment, visual inspection suggested that there was an overall amplitude reduction in the feedback-locked ERPs over the course of learning as well as from pre- to posttest, particularly in the deterministic learning condition. However, Figure 21 also shows that the amplitude of the feedback-locked ERPs was generally reduced in the no-failure-feedback group both at pre- and posttest. As in Experiment 1, I will first report the analyses of peak-to-peak amplitudes, followed by the results for the mean amplitude measures of the FRN.

Peak-to-peak Analysis of the FRN

Failure-related effects. The analysis of peak-to-peak amplitudes yielded a reliable main effect of feedback type [$F(1,31) = 66.32$, $p < .001$], and marginally significant interactions of feedback type and test phase [$F(1,31) = 3.68$, $p = .064$] and feedback type, test phase and feedback group [$F(1,31) = 3.86$, $p = .058$]. Separate analyses for each group yielded a significant interaction of feedback type and test phase for the no-failure-feedback group [$F(1,17) = 17.09$, $p < .01$], whereas no effect was found for the failure-feedback group ($p = .98$). Figure 21 illustrates that the FRN – defined as the difference between positive and negative feedback[20] – decreased from pre- to posttest in the no-failure-feedback group. Moreover, the analysis revealed a significant interaction of feedback group, test phase,

[20] Separate analyses failed to obtain significant pre-post differences both for ERPs to positive ($p = .25$) and negative feedback ($p = .10$).

learning condition, feedback type, and bin [$F(2,62) = 3.49$, $p < .05$]. Follow-up ANOVAs within each group yielded a significant interaction of test phase, learning condition, feedback type, and bin for the no-failure-feedback group only [$F(2,32) = 4.97$, $p < .05$]. As can be seen from Figure 22, this interaction indicated that for the chance condition, the pre-post decrease in FRN amplitude was more pronounced in Bin 2.

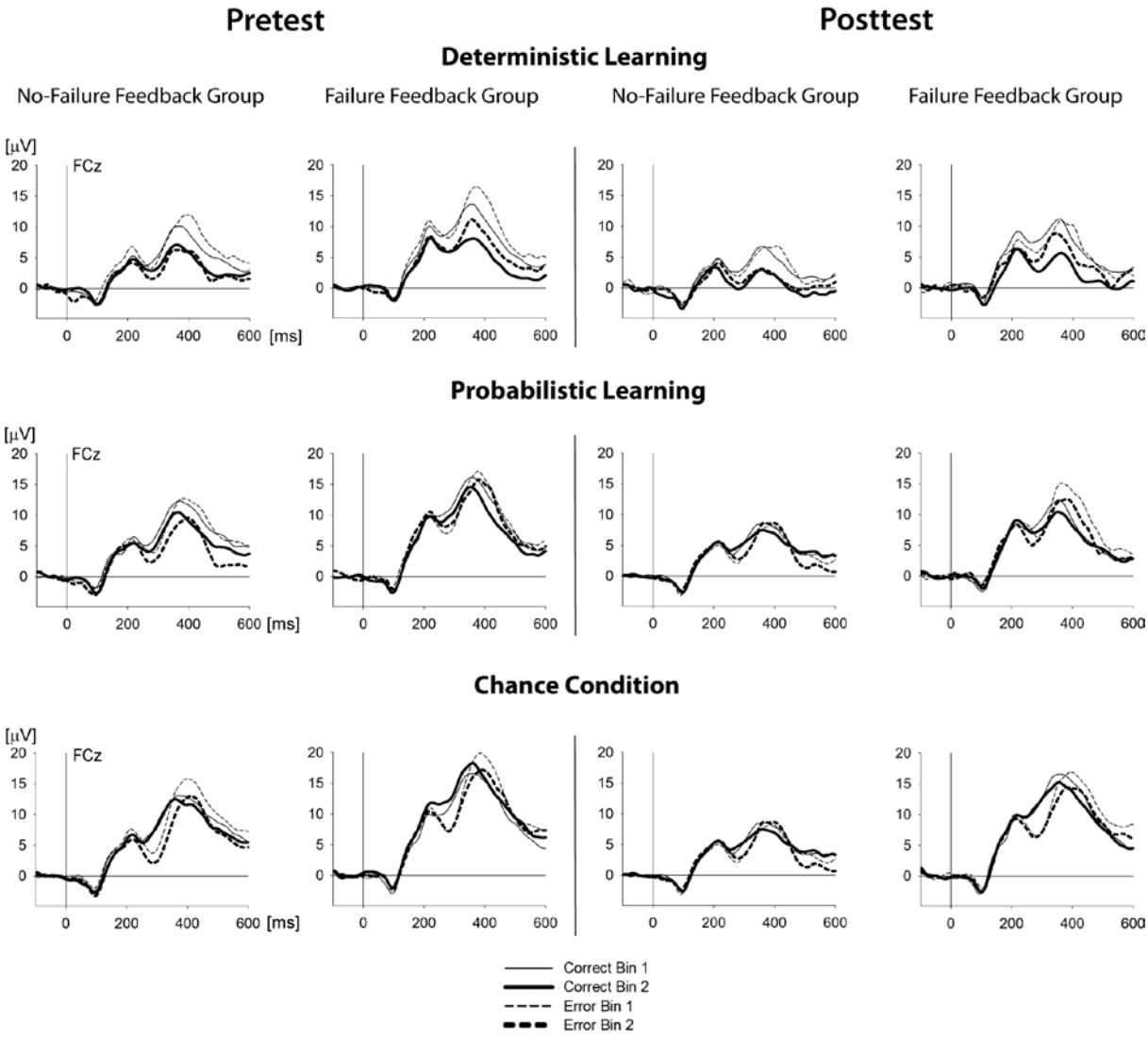


Figure 21: Feedback-locked ERPs to correct (solid lines) and incorrect responses (dashed lines) displayed separately for the no-failure feedback group and failure feedback group and the two halves of pretest (left) and posttest (right).

Mean Amplitude Analysis of the FRN

The analysis yielded reliable main effects of feedback type [$F(1,31) = 24.15$, $p < .001$] and learning condition [$F(2,62) = 20.36$, $p < .001$] that were qualified by an interaction of

feedback type and learning condition [$F(2,62) = 5.55$, $p < .01$]. Contrasts showed that the FRN was smaller in the two learning conditions compared to the chance condition [$F(1,31) = 7.63$, $p < .05$], whereas the difference between deterministic and probabilistic learning condition failed to reach significance ($p = .22$). Separate analyses for each feedback type yielded a significant main effect of learning condition both for positive feedback ($p < .001$) and negative feedback ($p < .05$). Positive feedback elicited more negative amplitudes in the deterministic compared to the probabilistic learning condition and in the two learning conditions compared to the chance condition (p-values < .001). By comparison, for negative feedback, a significant difference was found between deterministic and probabilistic learning condition ($p < .05$) but not between the two learning conditions and the chance condition ($p = .23$). In addition, the analysis yielded a reliable main effect of test phase [$F(1,31) = 8.89$, $p < .01$], reflecting a general attenuation of ERP amplitudes from pre- to posttest. Moreover, a marginally significant main effect of feedback group [$F(1,31) = 3.16$, $p = .085$], indicated an overall difference in ERP amplitude between the two groups. As can be seen from Figure 22, the ERPs were more positive-going both at pre- and posttest in the failure-feedback group.

Learning-related effects. A significant main effect of bin [$F(1,31) = 8.74$, $p < .01$] and a significant interaction of bin and learning condition [$F(2,62) = 10.90$, $p < .001$] were obtained. As illustrated in Figure 22, these effects reflect a learning-related decrease in the amplitude of the feedback-locked ERPs that was more pronounced in the deterministic compared to the probabilistic learning condition [$F(1,31) = F(1,31) = 12.20$, $p < .01$] and in the two learning conditions compared to the chance condition [$F(1,31) = 7.52$, $p < .05$]. Of note, neither the mean amplitude measures nor the peak-to-peak measures of the FRN were significantly correlated with post-error accuracy at pre- or posttest (rs < .27, p-values > .14).

Summary of FRN Findings

For the peak-to-peak measures, the analysis showeda decrease of the FRN from pre- to posttest in the no-failure-feedback group. In contrast, the mean-amplitude analysis revealed an overall pre-post attenuation of ERP amplitude only. In line with Experiment, no

evidence was obtained to indicate a modulatory role of trait variables for either mean amplitude measures or peak-to-peak amplitudes of the FRN (*F*-values < 2.0, *p*-values > .18).

Summary Experiment 2

The results of Experiment 2 confirmed the prediction that prolonged task performance results in motivational disengagement for the no-failure feedback group and provide further support for the notion that failure feedback amplifies sensitivity to internal indicators of errors. First, in contrast to Experiment 1, participants in the no-failure feedback group showed worse performance at posttest. Consistent with former studies (Boksem et al., 2006b; Tops & Boksem, 2010), the performance impairments were accompanied by a decrease of the Ne in the deterministic learning condition. However, in contrast to previous suggestions (Tops & Boksem, 2010), there was no evidence that traits such as punishment sensitivity and negative affectivity interact with the effects of task duration on behavioural and electrophysiological indices of engagement. In line with Experiment 1, the Pe increased with learning in both groups at pretest, but this was only the case for the failure-feedback group at posttest. This finding further supports the notion that participants in the no-failure-feedback group disengaged from task towards the end of posttest. Moreover, the analysis of peak-to-peak amplitudes revealed a decrease in the FRN from pre- to posttest in the no-failure-feedback group. Yet, the mean amplitude analysis of the FRN only yielded an overall pre-post decrease in ERP amplitude that was evident in both groups. This finding may reflect that participants generally paid less attention to the feedback at posttest.

Second, the results for the failure feedback group replicated the findings from Experiment 1. In both experiments, failure induction was associated with an increase in Ne amplitude as well as higher post-error accuracy in the deterministic and probabilistic learning condition. In contrast to Experiment 1, however, the failure manipulation did not affect the FRN nor were there influence of punishment sensitivity of posttest learning performance.

7. Discussion of Experiment 1 and 2

The following section is divided into three parts. The first part summarizes the main findings of the two experiments. In the second part, learning-related changes of the response- and feedback-locked ERPs are discussed. The third part focuses on the most important findings of the present research – the effects of the failure manipulation.

Summary of Main Results

The aim of the present experiments was to examine how exposure to self-relevant failure influences performance monitoring – as reflected in the Ne, FRN, and Pe – and behavioural adaptation during subsequent feedback-based learning. Two phases (pre- and posttest) of a learning task were applied that included three different conditions of feedback validity (100%, 80%, and 50%). Between pre- and posttest, participants were assigned to one of two groups receiving either failure feedback or no feedback during a visual search task that was described as diagnostic of intellectual abilities. To disentangle the effects of failure and motivational disengagement due to prolonged task performance, the posttest was linked to intelligence (Experiment 1) or described in neutral terms (Experiment 2).

Consistent with previous research that has established a link between the Ne and the evaluation of the affective and motivational significance of an error (e.g., Gehring et al., 1993; Hajcak et al., 2005; Luu et al., 2003), the results of both experiments revealed that exposure to uncontrollable failure led to an increase of the Ne in the subsequent learning task. These findings were extended by the observation that the failure-induced Ne amplitude enhancement was accompanied by more efficient error-related behavioural adjustments during learning. Crucially, the increase in Ne amplitude at posttest was not associated with better overall performance, but higher post-error accuracy, i.e., a higher proportion of correct choices on the next presentation of the target on which an erroneous response occurred. It is important to note that this error-related change in behaviour is unlikely to reflect an unspecific increase of attention or arousal since stimuli were presented in random order. Instead, the behavioural adjustment appears to specifically relate to a higher impact of negative reinforcement learning signals at posttest (Frank et al., 2005; Holroyd & Coles, 2002). In line

with this view, the failure-induced Ne increase was more pronounced in the second half of posttest when the participants were better able to internally represent an incorrect response. Moreover, failure feedback affected the Ne amplitude in the two learning conditions but not in the chance condition. In Experiment 1, the analysis of mean-amplitude measures of the FRN revealed a relatively larger difference between positive and negative feedback in the failure-feedback group compared to the no-failure-feedback group at posttest. This finding suggests that failure-induction did not only result in a heightened responsivity to internal indicators of errors but also increased the sensitivity to external performance feedback. However, the results of Experiment 2 failed to confirm a failure-related FRN modulation. Instead, the peak-to-peak amplitude analysis revealed a reduced difference between positive and negative feedback in the *no*-failure-feedback group at posttest. Furthermore, the mean-amplitude analysis showed that the ERPs generally decreased from pre- to posttest. No failure-related modulations in Pe amplitude were observed in Experiments 1 and 2. Yet, there was a lack of learning-related changes in Pe amplitude across posttest in the no-failure-feedback group in Experiment 2.

Corroborating prior findings concerning the effects of prolonged task performance on action monitoring (Boksem et al., 2006a; Tops & Boksem, 2010), Ne amplitude and accuracy decreased with time on task for the no-failure feedback group in Experiment 2. In contrast, both groups in Experiment 1 as well as the failure feedback group in Experiment 2 showed comparable overall performance at pre- and posttest. Thus, increasing the motivational significance of posttest by linking the learning task to intelligence (Experiment 1) or giving prior negative feedback as to participants' intellectual abilities (Experiment 1 and 2) preserved task engagement. However, we observed different fluctuations of the Ne across posttest: the Ne amplitude decreased for the deterministic learning condition in the no-failure feedback group, but increased for deterministic and probabilistic learning condition in the failure feedback groups.

Furthermore, the results of the two experiments did not reveal clear evidence in support of the notion that individual differences in trait level punishment sensitivity, negative affect, and action vs. state orientation modify the effects of the failure manipulation on error- and feedback processing. Only in Experiment 1, punishment sensitivity moderated the

effects of failure on subsequent learning performance. High punishment sensitivity predicted reduced posttest accuracy in the failure-feedback group but not in the no-failure-feedback group. There was no evidence that punishment sensitivity modulated the impact of failure on neural mechanisms of error processing as reflected in the Ne and FRN.

Learning-related Changes in the Response- and Feedback-locked ERPs

There were two main reasons for examining ERP modulations over the course of pretest. First, they provided a baseline that was potentially relevant for the interpretation of failure-related effects. Second, the present research aimed to replicate findings by Eppinger and colleagues (2008, 2009) indicating learning-related effects on the processing of correct responses and positive feedback.

Learning-related changes in the Ne. In line with previous studies (e.g., Eppinger et al., 2008, 2009; Holroyd & Coles, 2002; Nieuwenhuis et al., 2002), the analysis of the response-locked ERPs showed that the Ne was the larger the more valid the feedback. Moreover, the present study confirmed prior findings (Eppinger et al., 2008; 2009), which demonstrated that not only the Ne but also the correct response-related positivity is sensitive to response-outcome contingency. Yet contrary to the predictions of the R-L theory (Holroyd & Coles, 2002), the Ne did not increase with learning in either group. Instead, consistent with the studies by Eppinger and coworkers, there was a pronounced learning-related enhancement in the ERPs to correct responses.

On the first glance, the observed pattern of learning-related modulations in the response-locked ERPs contradicts the predictions of the R-L theory. Instead, the data appear to speak in favour of more recent proposal that focus on variations in the ERPs to correct responses (e.g., Eppinger et al., 2008, 2009; Foti et al., 2011; Holroyd et al., 2008). Yet, there are several reasons to suggest that caution is warranted in drawing this conclusion based on the current data. First, the bins in the present experiments contained a quite large number of trials. Given that accuracy reached asymptote within the first bin (see Figures 11 and 17), averaging across the first vs. second half of the learning task might have obscured an early increase in Ne amplitude. In support of this notion, learning-related changes in the

Ne have been found if the learning task contained a fewer number of item repetitions (e.g. Eppinger & Kray, 2011) or involved more than two response options and hence was more difficult (Pietschmann et al., 2008).

Second, it has been demonstrated the action monitoring as indexed by the Ne is susceptible to mental fatigue due to prolonged task performance (Boksem et al., 2006a; Tops & Boksem, 2010). Of note, in the Boksem et al.'s (2006a) study, the Ne amplitude showed the most pronounced reduction after the first 20 minutes of task performance. This approximately matches the duration of one bin of the learning task in the present experiments. Thus, it is conceivable that the effects of fatigue on Ne amplitude have attenuated those of learning.

Third, the correct response-related positivity increased not only in the deterministic and probabilistic learning condition but also in the chance condition. Furthermore, the positivity increased from pre- to posttest. At the same time, accuracy did not change or even decreased from the first to the second learning phase. This finding seems hard to reconcile with the idea of a reward-related learning signal. It should be noted, however, that the variance in the response-locked positivity might partly reflect stimulus-evoked P300 activity (Hajcak, Vidal, & Simons, 2004; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003). Indeed, Eppinger and coworkers (2008, 2009) reported that the stimulus-evoked P300 increased across the bins in each of the three conditions, including the chance condition. In contrast to the present study, the authors did not find an increase in the response-locked positivity in the chance condition, suggesting that this component is dissociable from the stimulus-evoked P300. Nonetheless, component overlap is a serious problem in interpreting learning-related modulations in Ne and correct response-related positivity.

It might be of particular interest, therefore, that a similar investigation failed to obtain differential learning-related modulations in the ERPs to correct and erroneous responses (Pietschmann et al., 2008). Importantly, the learning task used in this study involved four (instead of two) possible S-R mappings. Since no response deadline was applied, this manipulation resulted in comparatively long response latencies (about 900 ms) and probably in a less substantial overlap with the stimulus-evoked P300. Although the mean accuracy rates in the study were comparable to those obtained in the present experiments, the authors

observed moderate learning-related changes in the ERPs to correct as well as erroneous responses. In addition, Pietschmann and coworkers found a pronounced correct-response negativity (CRN) – rather a response-related positivity – that decreased as learning progressed. As the CRN has been linked to response uncertainty (Coles et al., 2001; Scheffers & Coles, 2001), the authors suggested that their findings reflect the learning-related reduction in uncertainty about the correctness of a response.

Thus both the null finding for the Ne and the pronounced learning-related modulations in the correct response-related positivity need to be interpreted with caution.

Learning-related changes in the FRN. Similar to the Ne, the FRN varied as a function of response-outcome contingency in both experiments. As predicted, the FRN was the smaller the more valid the feedback. In addition, analyses showed that feedback validity affected the ERPs to positive feedback rather than those to negative feedback, which is in line with previous data (Cohen et al., 2007; Eppinger et al., 2008, 2009; Holroyd & Coles, 2002; Nieuwenhuis et al., 2002). However, the present study did not find evidence for learning-related modulations in FRN amplitude. Instead, the ERPs to both positive and negative feedback became less positive as participants learned the mappings rules. In particular, this finding contrasts with the results of Eppinger and colleagues (2008, 2009), who reported a learning-contingent amplitude reduction in the ERPs to positive feedback only. This discrepancy was somewhat surprising, given that basically the same learning paradigm was applied. The most obvious difference to the Eppinger et al.'s studies was the longer temporal extension of the bins created to examine effects of learning[21]. Thus, it seems plausible to assume that differential learning-related modulations of positive vs. negative feedback trials occurred within the first bin only. By contrast, the overall amplitude reduction in the second bin was likely to reflect an attenuation of the feedback-evoked P300. In line with this view, visual inspection suggested that the effect was maximal at posterior sites. The feedback-evoked P300 shows sensitivity to the motivational salience of an outcome (Yeung

[21] Pre- and posttest contained only 600 trials (as opposed to 1500 trials in the Eppinger et al.'s studies) and were divided in two bins only (instead of four). Moreover, the learning blocks contained a larger number of stimuli (12 vs. 6) and a longer inter-trial-interval was applied.

& Sanfey, 2004) and is smaller if feedback does not provide response-related information (Walsh & Anderson, 2011; Yeung, Holroyd, & Cohen, 2005). A feasible explanation of the amplitude reduction in the second bin, then, is that participants paid less attention to the feedback stimulus after they have developed an internal representation of the correct response.

Yet, the lack of learning-related variations in the ERPs to positive feedback contrasts with the findings for the response-locked positivity. If the two components are neural manifestations of a positive PE, as was suggested by Eppinger and coworkers (2008, 2009), they would be expected to show complementary changes over the course of learning. There is no obvious reason why reward prediction indicated by the response-related positivity should continue to increase, while the evaluation of the actual reward indicated by the feedback-locked positivity remains unaltered.

Although the bins in the present study may have been too large to reveal learning-related modulations in the FRN, the current findings further substantiate the notion that expectancy of rewards vs. non-rewards affects the neural responses to positive feedback, while leaving unaffected the processing of negative feedback (Cohen et al., 2007; Eppinger et al., 2008, 2009; Holroyd et al., 2008). Indeed, this idea has received considerable support from a recent study using temporospatial principal components analysis to identify a positive deflection underlying the ERP difference between positive and negative feedback in the FRN time window (Foti et al., 2011). Crucially, the study clearly distinguished the reward-related positivity from the feedback-evoked P300. This is particularly important as the P300 overlaps with the FRN in time and has also been shown to vary as a function of outcome expectancy (Hajcak, Holroyd, Moser, & Simons, 2005; Johnson & Donchin, 1980).

Taken together, the FRN was sensitive to feedback validity but did not change with learning, possibly reflecting that the bins were too large to assess learning-related dynamics.

Learning-related changes in the Pe. In line with the findings by Eppinger et al. (2009), the analysis revealed that the Pe increased with learning and feedback validity. Although the ERPs to erroneous as well as correct responses became more positive as learning progressed, the amplitude enhancement was more pronounced on error trials. The sensitivity

of the Pe to feedback validity and learning is consistent with most theoretical accounts on this component such as the 'error-awareness hypothesis' and the 'error-salience hypothesis' (cf. Overbeek et al., 2005; Steinhauser & Yeung, 2010). If the Pe reflects conscious error recognition, it should grow larger with the development of an internal representation of the correct response. Similarly, the cognitive or affective salience of errors should increase with advanced learning.

The learning-related change in the Pe is also in line with proposals that stress the similarity to the P300 (Leutholt & Sommer, 1999). In particular, the P300 has been proposed to reflect the updating of an internal model of the current environmental context in response to events signalling a mismatch with this model (Donchin & Coles, 1988). This directly implies that the Pe should vary over the course of learning, reflecting the incorporation of the response rules into the context model (cf. Overbeek et al., 2005). Yet, the current findings are not thoroughly consistent with a 'P300 account' of the Pe. According to more recent views, the P300 signifies the motivational significance of the eliciting event and is linked to processes that support goal-directed behavioural adjustments (Nieuwenhuis et al., 2005). In line with this notion, Chase and coworkers (2010) showed that the P300 to negative feedback predicted rule detection and explicit rule-based behavioural adjustments in a reversal learning task. The present experiments, however, did not provide evidence for a relationship between the Pe and error-related behavioural adaptation. In contrast to the Ne, the Pe did not significantly correlate with post-error accuracy. Of course, this finding does not exclude that the Pe may relate to other forms of remedial actions, such as for example immediate error correction (Nieuwenhuis et al., 2005).

Interestingly, the pattern of results for the Pe bears striking resemblance to that for the correct response-related positivity both morphologically and functionally. The two components were maximal at midline centro-parietal electrode sites and showed pronounced changes over the course of learning and from pre- to posttest. This finding further corroborates the view that highly similar neural processes underlie the Pe, the correct response-related positivity, and the stimulus-evoked P300.

In sum, most pronounced learning-related modulations were observed for the correct reponse-related positivity and the Pe. By contrast, Ne and FRN increased with feedback validity only. While the latter the latter finding presumably reflect that the bins were too large to track learning-related dynamics, the effects in the correct response-related positivity might have been partially caused by component overlap with the stimulus-evoked P300.

Effects of Failure on Performance Monitoring and Learning

Of central interest for the present thesis were the effects of failure on behavioural and electrophysiological indices of performance monitoring and learning. Consistent with the findings by Brunstein and Gollwitzer (1996), self-relevant failure did not result in learning impairments. Specifically, the results of Experiment 2 indicate that failure exposure prevented motivational disengagement at posttest. However, enhancing the motivational significance of posttest in Experiment 1 was not associated with an additional performance benefit in the failure-feedback group. By contrast, Brunstein and Gollwitzer (1996) reported better performance in a subsequent task relevant to the self-aspect threatened through prior failure. Yet in their study, the task was very simple and short in duration (< 10 minutes). In the present study, the learning task took about 45 min und was cognitively more demanding. Thus, one possible explanation for the lack of performance improvements is that the need to cope with failure-related negative affect depleted control resources the learning task relied on (Inzlicht & Gutsell, 2007).

This view is in line with the suggestion that participants made more “errors of commission” in the deterministic learning condition during later stages of posttest, particularly in Experiment 1. As I have pointed out earlier, these errors were likely to reflect lapses in focused attention. From visual inspection (see Figure 11), it appears that the corresponding drop in accuracy was more prevalent in the failure-feedback-feedback group than in the no-failure-feedback group. Although the effect failed to reach significance in the overall ANOVA, a separate posttest analysis revealed a significant interaction of feedback group, learning condition, and bin [$F(10,330) = 2.13$, $p < .05$]. The interaction indicated that the course of performance differed between the groups in the deterministic learning condition ($p < .05$) but

not in the probabilistic and chance condition (*p*-values > .77). This finding may suggest that participants in the failure-feedback group allocated less attentional resources to stimuli in the deterministic learning condition after they have learned the contingencies.

Furthermore, selective failure-related performance impairments in the deterministic learning condition seem to fit previous findings that stress primarily affects explicit learning and memory strategies (Schwabe et al., 2011; Schwabe & Wolf, 2009). However, failure exposure did not appear to undermine the acquisition of the correct response mappings. If anything, subsequent retrieval and maintenance of the response rules was compromised. There was also no evidence that failure-induction promoted habitual, incremental learning. Although the overall ANOVA on mean accuracy rates might have been insensitive to subtle changes in learning rate, visual inspection of the learning curves did not suggest that learning progressed more slowly at posttest. Instead, participants in the failure feedback group showed numerically higher performance scores at the beginning of posttest. It should be noted, however, that habitual control was not directly tested in the current study. Thus, it is conceivable that failure-induction resulted in relative insensitivity to contingency degrading and reduced explicit knowledge of the mapping rules. Future studies may probe this possibility.

The Ne is sensitive to prior failure. Most strikingly, the results of Experiment 1 and 2 showed that failure-induction led to an increase in the Ne that was accompanied by more efficient error-related behavioural adjustments during subsequent learning. Importantly, the failure-related modulations in Ne amplitude are not attributable to pre-experimentally existent individual differences in trait-level negative affect or punishment sensitivity which have been related to increased reactivity of the error monitoring system (e.g., Boksem et al., 2006a; Hajcak et al., 2004; Luu et al., 2000). Instead, the results are based on direct manipulations of affective-motivational state. As was indicated by participants' self-reports, the experimental manipulation was successful in inducing self-relevant failure, and by this, negative feelings. Importantly and as expected, there was no evidence for between-group differences in Ne amplitude at pretest in either experiment, whereas clear group differences were obtained at posttest. Furthermore, the observed Ne modulations cannot simply be explained by within- or

between-group differences in overall performance (cf. Yeung, 2004). The findings thus provide strong evidence that the functioning of the ACC, as reflected by the Ne, is sensitive to the affective and motivational context of an action (Luu et al., 2003; Olvet & Hajcak, 2008).

In particular, the present study confirms and extends the finding by Wiswede and colleagues (2009a,b) that short-term manipulations of negative affect are reflected in modulations of the Ne. Whereas in the study by Wiswede et al. (2009b) the Ne was measured during the affective manipulation, the present data show that the effects of failure feedback generalized to a different task. This is an important new finding suggesting that failure-induced negative affective state can bias information processing at a broader task-unspecific level. Moreover, the failure-related increase in Ne amplitude challenges the view that the Ne is a state-independent marker of an endophenotype for internalizing (increased amplitude) vs. externalizing (reduced amplitude) psychopathology (Clayson et al., 2011; Olvet & Hajcak, 2008). Instead, the present results suggest that state and trait variations in negative affect might be associated with similar changes in the functioning of the internal error monitoring system (Boksem et al., 2006a; Hajcak et al., 2003, 2004). However, this does not imply that state and trait variables operate in an independent fashion. Although the present study did not find evidence for a moderating role of personality, findings by Cavanagh and coworkers (Cavanagh & Allen, 2008; Cavanagh et al., 2011a) suggest that influences of negative affective state and trait vulnerability to stress jointly modulate stress-related activity of the medial prefrontal performance monitoring system in an inverted-U type fashion. This might explain why affective-motivational manipulations have not consistently been found to affect error monitoring as reflected in the Ne.

Recently, it has been argued that variations in Ne amplitude due to experimental manipulations of motivational and affective significance “merely” reflect changes in the allocation of attentional resources (Clayson et al, 2011). In particular, Clayson and colleagues proposed that the conflict monitoring theory could easily account for Ne modulations that have been observed in conjunction with motivational/affective manipulations (Botvinick et al., 2001; Yeung, Botvinick, & Cohen, 2004). The conflict monitoring theory posits that the Ne reflects post-error conflict due to the simultaneous activation of incorrect and correct response, with the latter arising from continued processing of the target stimulus

after the erroneous response is produced. Given that participants in the failure feedback group were likely to be highly motivated to perform well at posttest, larger Ne amplitudes might indicate an increased post-error activation of the correct response as a consequence of enhanced target processing (cf. Yeung, 2004). Similarly, it seems plausible to assume that more efficient continued processing of the imperative stimulus would facilitate the detection of mismatch between the actual and the intended response, as suggested by the error detection/mismatch theory (Bernstein, Scheffers, & Coles, 1995; Falkenstein, et al., 1990; Gehring et al., 1993).

However, increased attention to task-relevant information should be associated with improved task performance. Contrary to this prediction, both groups showed comparable overall performance in Experiment 1. Similarly, RT data analyses did not reveal reliable between-group differences in response speed at pre- and posttest (see Appendix). This is important, since Yeung and Nieuwenhuis (2009) showed that fast responses are associated with low conflict and smaller Ne amplitudes, whereas slower responses are associated with high conflict and larger Ne amplitudes. In the present study, there was no evidence indicating that the failure-related Ne modulations reflected a speed-accuracy trade-off. Thus, although attentional mechanisms are likely to play a key role in mediating the effects of motivational/affective variables, the current findings do not support a 'pure' conflict-monitoring account of the failure-related Ne modulation[22].

Rather, the present study suggests that failure induction results in a strategic shift towards reactive control, denoting the tendency to recruit control processes when an (negative) event has already occurred (Braver et al., 2007; Tops et al., 2010). Consistent with the notion that a reactive mode of behaviour control is highly adaptive in uncertain

[22] Moreover, the conflict monitoring theory states that high conflict triggers increased recruitment of cognitive control mechanisms on the next trial. By contrast, the present results revealed that the Ne enhancement was accompanied by delayed, i.e., learning-related, rather than immediate behavioural adjustments. Supplementary analyses did not yield significant correlations between the Ne and accuracy on the immediately following trial (*p*-values > .24). Yet, it should also be noted that the predictions of the conflict-monitoring theory primarily apply to response conflict tasks that usually involve one fixed mapping rule for all trials. It seems reasonable to assume, then, that information processing was biased according to target-specific response rules in the present learning task (for instance, the conflict signal may trigger an update of the correct S-R mapping in working memory). In

environments, failure feedback thus appears to induce a state in which participants are particularly vigilant to potential threats and negative response outcomes. This is in line with previous research that has established a relation between the activity of the medial prefrontal performance monitoring system and sensitivity to negative stimuli and events. In particular, larger Ne amplitudes have been linked to punishment sensitivity (e.g., Boksem et al., 2006a; 2008), trait differences in negative emotionality and anxiety (e.g., Hajcak, McDonald, & Simons, 2003; 2004; Tops, Boksem, Wester, Lorist, & Meijman, 2006), learning from errors (Frank et al., 2005), and defensive motivation (Hajcak & Foti, 2008). Similarly, social stress reactivity has been found to increase the sensitivity to internal indicators of error and conflict (Cavanagh, et al., 2011a).

In contrast to the Ne, the correct response-related positivity was not affected by the failure manipulation. This dissociation seems at odds with a recent proposal according to which the correct response-related positivity rather than the negativity on incorrect trials is subject to experimentally induced change (e.g., Holroyd et al., 2008), and instead suggests that both components reflect separable processes. Holroyd and colleagues (2008) largely draw on findings from animal research that phasic increases in dopaminergic activity in response to unpredicted rewards are typically larger than phasic decreases in response to unpredicted negative events (e.g., Schultz, 2002) and thus may have stronger effects on target structures[23] (for a similar suggestion, see Eppinger et al., 2008). However, this leaves open the question why failure substantially changed the impact of negative events on neural processing while apparently leaving unaffected the response to positive events.

Although the present findings highlight the need to explore the performance monitoring system in terms of both cognitive and affective/motivational mechanisms, the precise nature of the processes that mediate the observed effects of failure feedback remains to be determined. As was already pointed out by Yeung (2004), cognitive and affective accounts on the Ne are not necessarily mutually exclusive. Nevertheless, arguments similar to that made by Clayson and colleagues (2011) (see above) show that a

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support of this notion, Egner and coworkers (2007) demonstrated that different types of conflict (Flanker vs. Simon conflict) recruited distinct conflict resolution mechanisms in an independent fashion.

[23] Note, however, that the magnitude of the negative PE has been shown to be coded by the duration of DA dips

sharp distinction is often drawn between influences of affective/motivational states and attention on performance monitoring. As I have outlined in the theoretical part of this thesis, emotional and motivational processes are thought to bias information processing and behaviour selection according to specific situational demands (Gray, 2004; Pessoa, 2008, 2009). Specifically, emotion and motivation systems have been suggested to selectively enhance attentional and perceptual processing and to influence the recruitment of effortful control mechanisms (e.g. Clore & Hunziker, 2007; Pessoa & Engelman, 2010; Savine & Braver, 2010). Hence, it is conceivable that the impact of emotion, motivation, and attention on performance monitoring relies on similar mechanisms in a highly integrated neural architecture (Pessoa, 2008). The critical question, then, is not whether these findings reflect changes in affective/motivational state *or* attentional processing but how affective and motivational influences are integrated with task-specific processing.

Minor influences of failure on the FRN. Importantly, the R-L theory (Holroyd & Coles, 2002) asserts that Ne and FRN reflect activity of the same generic error processing system. The proposed functional similarity implies that if one component is affected by a certain experimental manipulation, so is the other as well. Indeed, the first experiment provided some evidence for a failure-related relative increase not only of the Ne but also of the FRN. However, the FRN did not differ significantly between the two groups at posttest, suggesting that the effect was rather weak. Critically, the findings of Experiment 2 did not confirm the failure-related FRN modulation. Instead, the analysis of peak-to-peak amplitudes revealed a decrease in the FRN from pre- to posttest in the no-failure-feedback group. Thus, failure-induction appeared to increase the responsivity to performance feedback in Experiment 1, but to prevent diminished processing of feedback cues in Experiment 2.

Previous studies found greater FRN amplitudes in individuals with depression, elevated negative affectivity, and higher punishment sensitivity, possibly reflecting increased concern about negative action outcomes (Balconi & Crivelli, 2010; Santesso et al., 2008, 2011a,b; Sato et al., 2005). However, these groups showed heightened neural responses to negative feedback, while failure-induction was associated with enhanced differentiation between positive and negative feedback in the present study. Notably, the effect of failure

was independent of feedback validity and hence seemed insensitive to the information the feedback provided for appropriate responding. Although the accuracy data and Ne findings indicate that participants in the failure-feedback were not impaired in representing the correctness of their responses, the FRN modulation suggests that they were more reluctant to disengage from feedback over the course of the task. This might reflect that the prior failure experience reduced the participants' reliance on internal cues for action evaluation and promoted the use of external feedback cues to validate the internal judgments. According to this view, heightened vigilance to internal and external feedback – as reflected in increases in Ne and FRN – would indicate an attempt to "recalibrate" the performance monitoring system that was challenged through uncontrollable failure.

At first glance, the heightened neural response to external feedback seems to contrast with findings of the Cavanagh et al.'s (2011a) study, which demonstrated a stress-related shift towards diminished processing of external punishment cues. Yet, in this study, participants were exposed to uncontrollable evaluative stress *during* concurrent learning. In addition, the study did not differentiate between valid and invalid negative feedback. This distinction might be critical as reduced vigilance to invalid negative feedback, would be an adaptive strategy to cope with uncontrollable stressors. By contrast, in the present study, heightened sensitivity to external feedback might reflect participants' efforts to regain control after the stressful experience.

It seems plausible to explain the decrease of the FRN in the no-failure-feedback group in Experiment 2 by reduced attention to feedback stimuli. However, the analysis also revealed an overall pre-post attenuation in ERP amplitude for both groups. As I already have outlined above, this effect is likely to reflect a decrease of the feedback-evoked P300, indicating that participants in *both* groups paid less attention to the feedback at posttest. Notably, the decrease in FRN amplitude in the no-failure-feedback group parallels the Ne findings and hence might indicate alterations in ACC functioning due to mental fatigue (Boksem et al., 2006a; Lorist et al., 2005). Indeed, the ACC has been shown to be critically involved in effort-related decision making (Schweiber, Saft, & Hauber, 2005; Walton, Bannerman, Alterescu, & Rushworth, 2003; Winterer, Adams, Jones, & Knutson, 2002). Specifically, Boksem and Tops (2008) proposed that a network involving ACC, amygdala,

ventral striatum, and insula integrates information about the anticipated costs and benefits of behaviour with information on current physiological state and available resources to guide adaptive action selection. According to this view, mental fatigue is an adaptive mechanism to abandon behaviours that have an unfavourable cost-benefit ratio. Hence, the observed group differences in Ne and FRN could also be explained by failure-related shifts in cost-benefit analyses. One might argue that the failure experience was likely increase the subjective value participants place on high performance during posttest. As a consequence, participants should continue to invest mental effort to maintain high accuracy levels throughout the task, reflected in sustained engagement of the ACC in response to behaviourally relevant events. Although this account is compatible with the findings of Experiment 2, it cannot easily explain why enhanced recruitment of cognitive control resources in the failure-feedback group in Experiment 1 did result in improved performance.

Given the lack of robustness of effects, the failure-related FRN modulations should be interpreted with caution. In either case, the present results indicate that failure has distinct effects on Ne and FRN. First, pronounced failure-related changes in Ne amplitude were evident in both experiments. Second, these effects were specific for erroneous responses and varied as a function of learning. The findings thus add to a growing number of studies that reported Ne/FRN dissociations, for example, in neuropsychiatric disorders (e.g., Borries et al., 2010; Gründler et al., 2009), trait level anxiety (Hajcak et al., 2003), and different age groups (Eppinger et al., 2008, 2009).

Failure does not affect the Pe. Consistent with previous studies (Clayson et al., 2011; Wiswede et al., 2009b), the first experiment did not reveal failure-related changes in the Pe. Furthermore, the second experiment demonstrated only a lack of learning-related changes in Pe amplitude in the no-failure-feedback group towards the end of posttest. As comparatively little is known about the functional significance of the Pe, the interpretation of the latter finding is difficult. It is tempting to attribute the absence of learning-related modulations to motivational disengagement; yet, no prior study has explicitly examined the effects of prolonged task performance on the Pe. Critically, visual inspection of the ERP waveforms in the Lorist et al.'s (2005) study suggests that the Pe did not change with time on task,

although the Ne reduction may have disguised modulations in Pe amplitude. Nonetheless, a relative attenuation in the Pe due to mental fatigue seems consistent with the notion that this component reflects the allocation of resources for remedial actions (cf. Overbeek et al., 2005). Further support for the susceptibility of the Pe to mental fatigue comes from source localization studies indicating that the ACC contributes to Pe generation (O'Connell et al., 2007).

Certainly, the present findings are hard to reconcile with conceptualizations of the Pe in terms of the affective appraisal of an error (Falkenstein et al., 2000). However, the present results also contrast with reports of attenuated Pe amplitudes for subjects with high trait level negative affect (Hajcak et al., 2004) and severe depression (Schrijvers et al., 2009). Hajcak and colleagues (2004) suggested that high-NA individuals were less aware of making mistakes or found their errors less salient. Yet, an important restriction of their finding is that the amplitude was reduced on error and correct trials. Moreover, the amplitude differences between high- and low-NA subjects were more pronounced at fronto-central compared to posterior sites, raising some doubts about the true nature of processes underlying the effect.

Within the framework of the 'error-awareness hypothesis' (cf. Overbeek et al., 2005), the present findings suggest that although participants were more vigilant to internal indicators of maladaptive performance after failure exposure, conscious error recognition did not vary as a function of the affective and motivational context of an action. In line with the observation that that only the Pe but not the Ne correlates with error awareness (Endrass et al., 2007; Nieuwenhuis et al., 2001), this result implies that the processes reflected in the Ne are functionally distinct from those leading to conscious error recognition.

Failure enhances the impact of errors on learning-related behavioural adaptation. According to the R-L theory, the Ne constitutes a predictive error signal that is used by the ACC to select and reinforce appropriate actions (Holroyd & Coles, 2002). The finding that the failure-related Ne enhancement was accompanied by higher post-error accuracy supports this view. Similar conclusions have been drawn from previous studies demonstrating that activity in the medial prefrontal error processing system predicted the correctness of future responses (Hester, Barre, Murphy, Silk, & Mattingley, 2008; van der Helden, Boksem, &

Blom, 2010). Moreover, findings by Frank and colleagues (2005) suggest a specific relation between Ne amplitude and a bias to learn more from bad than from good choices. Applied to the present study, this idea implies that individuals exposed to self-relevant failure use error signals more efficiently to determine which response to avoid on subsequent presentations of a stimulus. In line with these findings, the current data challenge the view that positive events generally have a stronger impact on ACC functioning and learning-related behavioural adaptation than do negative events (Eppinger et al., 2008; Holroyd et al., 2008).

Still, the failure-related increase in post-error accuracy seems inconsistent with the results of the study by Petzold and coworkers (2010), which revealed stress-related impairments in the ability to use negative feedback for behavioural adaptation. However, an apparent difference between their study and the present experiments might account for the divergent findings. Petzold and coworkers applied an indirect measure of feedback-based learning. In their study stress-related effects were assessed in a test phase designed to assess the *outcomes* of slow habitual, putatively BG-mediated learning (Frank et al., 2004). By contrast, rapid trial-to-trial adjustments *during* learning, as examined in the current study, have been shown to rely on the PFC and DA levels therein (Frank et al., 2007a,b). Specifically, Frank and colleagues (2007a,b) suggested that high prefrontal DA levels support rapid model-based learning by stabilizing working memory representations of negative response outcomes.

Of note, acute stressors have been shown to trigger DA release in the ACC, which is thought to evaluate the controllability of the stressor and to regulate subsequent mesocortical and mesolimbic DA transmission accordingly (Amat et al., 2005; Pascucci et al., 2007). Increased DA levels have been linked to active behavioural coping with aversive events (Cabib & Puglisi-Allegra, 1996; Horvitz, 2000). Hence, failure-related modulations in ACC functioning, possibly reflecting the attempt to regain behavioural control after the stressful experience, might have contributed to the present findings. Contrary to the predictions of the R-L theory, however, the above reasoning suggests that the ACC is not only a passive recipient of dopaminergic learning signals from the BG, but also actively biases learning-related striatal processing and mediates behavioural adjustments in a model-based fashion (Doll et al., 2009; Frank et al., 2005; Huys & Dayan, 2009).

The involvement of the ACC in regulating the response to stressors might also explain why no evidence for a failure-induced shift towards habitual control was found in the present study. Ample evidence indicates that stress-related effects on instrumental behaviour are mediated by effects of glucocorticoids and noradrenaline in the amygdala, which then biases processing in brain circuits involved goal-directed control such as the mPFC, the hippocampus, and the dorsomedial striatum (Schwabe & Wolf, 2011). Crucially, one of the control functions implemented by the ACC is to inhibit such stress-related activity (Amat et al., 2005). Thus, the putative failure-related change in ACC functioning might have prevented a strong bias towards habitual learning.

A related aim of this study was to examine whether failure differentially affects error processing in the deterministic and probabilistic learning condition. Building upon on findings of task-specific dissociations in Ne amplitude as a function of OCD symptomatology, Gründler and colleagues (2009) proposed that dissociable neural systems might underlie responsivity to errors on conflict task with fixed response rules and maladaptive choices in probabilistic learning tasks. The present results did not reveal distinct influences of failure manipulation for the two learning conditions. However, there was other evidence to suggest that participants' ability to represent the correctness of their responses did play a role for failure-related effects on error processing. Whereas no learning-related changes in Ne amplitude were found at pretest, failure-induction was associated with an increase of the Ne across posttest in the deterministic and probabilistic learning condition. This finding confirms the prediction that the impact of failure on error processing grows larger as participants are better able to represent the correctness of their responses. Understood in the above context, this may reflect that participants in the failure-feedback group were more vigilant to (negative) performance cues throughout the posttest. Thus, failure feedback appeared to amplify learning-related changes of the Ne, possibly reflecting more efficient reactive monitoring during later stages of learning.

Taken together, the present findings further corroborate the view that the Ne is closely linked to behavioural adaptation. Importantly, the Ne magnitude predicted delayed but not immediate behavioural adjustments, suggesting that the neural mechanisms underlying the Ne are implicated in associative learning. This is in line with a large body of

research indicating that the ACC – which is thought to contribute to the generation of the Ne – integrates information about reinforcements to guide adaptive action selection (Rushworth & Behrens, 2008; Shackman et al., 2011). Although the R-L theory (Holroyd & Coles, 2002) most explicitly frames the Ne in terms of a teaching signal, alternative theories such as the conflict monitoring theory or the error detection/mismatch theory (Bernstein et al., 1995; Botvinick et al., 2001; Falkenstein et al., 1990; Gehring et al., 1993) are not incompatible with a relationship between the Ne and learning-related behavioural adjustments. The conflict-monitoring theory asserts that error-related activity in the ACC serves an alerting function, signalling the need to engage additional control. Originally, the conflict-induced control enhancement was conceptualized as a uniform top-down biasing mechanism, strengthening task-specific processing according to the current task set (Botvinick et al., 2001). However, the current study indicates that the adaptive control mechanism operates in an item-specific fashion. Rather than triggering processes that are relevant to all reinforcement contingencies, such as an unspecific increase in attention, the error signal appears to strengthen a particular S-R mapping only. This in in line with evidence indicating that multiple independent control loops may operate in parallel to resolve different types of conflict (Egner, 2008).

Dissociable effects of failure and self-relevance on the Ne. In an attempt to disentangle the effects of failure and prolonged task performance on error and feedback processing, the present study manipulated the self-relevance of the learning task at posttest. Although behavioural and ERP data in the first half of posttest suggest that the initial effects of the experimental manipulations were comparable for the no-failure feedback group in Experiment 1 and the failure feedback group in Experiment 2, the two groups were characterized by distinct changes in Ne amplitude in the second half of posttest. Notably, a pattern of results similar to that observed for the no-failure feedback group, i.e., large initial Ne amplitudes followed by fast reductions, has been reported for individuals characterized by high habitual intrinsic engagement (Tops & Boksem, 2010). The susceptibility of intrinsic engagement to increasing boredom during prolonged performance of monotonous tasks fits the present finding that the Ne decreased in the deterministic but not in the more challenging

probabilistic learning condition. In contrast, the pattern of sustained monitoring in the failure feedback groups parallels findings for individuals scoring high on constraint (Tops & Boksem, 2010). From this perspective, the Ne enhancement across posttest might reflect negatively motivated engagement resulting from worry and concerns about mistakes that become more salient with learning.

Somewhat surprisingly, the decrease of the Ne in the no-failure-feedback group in Experiment 1 was not accompanied by performance decrements. Still, a previous study by Lorist and coworkers (2005) also found an attenuation of the Ne in conjunction with stable error rates across prolonged task performance. Importantly, the latter effect was not confounded by changes in RT, at least within the first 90 minutes of the task. Certainly, it remains an issue for further research to precisely determine the factors that have counteracted the potentially deteriorating effects of compromised error monitoring on overall accuracy in the present study. It might be revealing, however, that the Ne decreased in the deterministic learning condition only. Given that the mapping rules were most apparent in this condition, overall performance might have been less crucially dependent on the integrity of the medial prefrontal action monitoring system during later stages of learning. This would be consistent with the finding that overtraining promotes the development of habitual (i.e., automatic) responding (e.g., Yin & Knowlton, 2006).

When does personality matter? Although an increasing number of studies have described influences of personality traits on error- and feedback processing (e.g., Balconi & Crivelli, 2010; Boksem et al., 2006a, 2008; De Pascalis et al., 2010; Santesso et al., 2011a; Tops & Boksem, 2010), the present study failed to obtain evidence for punishment sensitivity, trait negative affect, or action vs. state orientation to moderate the impact of failure. Only in Experiment 1, there was a small piece of evidence to suggest punishment sensitivity moderated the effects of failure on subsequent learning performance. While participants in both groups showed comparable overall performance, punishment sensitivity predicted poorer learning performance in the failure-feedback group. In the no-failure-feedback group, by contrast, no relationship between punishment sensitivity and accuracy was found. Critically, the impact of failure on Ne and FRN was not modulated by punishment

sensitivity, making the behavioural finding difficult to interpret. In the study by Cavanagh and colleagues (2011a), high punishment sensitivity was associated with better punishment learning under stress. However, in this study punishment learning was defined as loss-related learning rate. A computational RL algorithm was used to estimate this parameter, which obviously provided different information than overall accuracy rates and post-error accuracy. Further differences that may have contributed to the disparate findings include the nature of the experimental manipulation (social-evaluative stress vs. self-relevant failure), the learning paradigm, and the conditions for the assessment of the effects of stressors (during stress manipulation vs. after failure induction).

Moreover, it is important to note that the effect was not replicated in Experiment 2. On the one hand this might indicate that self-relevance of the learning task acted as an additional stressor that triggered the influence of trait vulnerability on performance. On the other hand, it might simply reflect that the effect in Experiment 1 was an artefact. Rather than punishment sensitivity, state orientation was expected to moderate the behavioural effects of failure (Kuhl, 1981). Contrary to this prediction, however, state orientation modulated the posttest Ne amplitude in the no-failure-feedback group in Experiment 1. State-orientation has been associated with unintended rumination and prolonged preoccupation with an aversive event (Kuhl, 1994). Thus, it is conceivable that state-oriented participants became more vigilant to internal error cues when the task was linked to intelligence. In support of this view, state-oriented individuals are characterized by heightened levels of negative affect, particular in response to challenges (Brunstein & Olbrich, 1985) and are more vulnerable to depressive symptoms (Rholes, Michas, & Shroff, 1989), both of which have been associated with enhanced Ne amplitudes.

No further modulatory influence of personality was obtained. Unexpectedly, trait measures did also not moderate the susceptibility to mental fatigue. This is inconsistent with previous research demonstrating that traits, such as punishment sensitivity, are related to persistence thus preserve task engagement (Tops & Boksem, 2010). However, most of the studies that found a relationship between personality and electrophysiological markers of performance monitoring used response conflict tasks such as the flanker task. In these tasks, accuracy typically accounts for a comparatively small proportion of variance in the Ne

amplitude. By contrast, in the present learning task, performance differences contributed to a considerable amount of variance in the Ne (mean R^2 = .16). Critically, the mean accuracy rates as well as Ne amplitudes in the present study were likely to represent a composite score, intermingling effects of learning and disengagement. So even if there were effects of personality, these effects were probably small and therefore difficult to obtain. Other factors that may have caused the lack of reliable findings include the potentially reduced reliability of the ERP components due to the relatively few number of error trials, the homogeneous sample, and the small sample size. Hence, it is clearly premature to discard the hypothesis that personality may play an important role in determining the impact of affective/motivational challenges on error and feedback processing during subsequent learning.

8. Experiment 3

Statement of Problem and Research Goals

The first experiments suggested that self-relevant failure triggered heightened vigilance to internal indicators of performance errors, possibly reflecting a shift towards a reactive, error-driven mode of behaviour control. Experiment 3 further explored the impact of motivational/affective context by investigating the extent to which the effects of negatively motivated reactive engagement differ from those of positively valenced motivational manipulations. Specifically, the study aimed to determine the impact of trial-by-trial variations of appetitive vs. aversive motivation on error processing and learning.

Reward incentives have been shown to enhance executive control processes and to improve behavioural efficiency (Hübner & Schlösser, 2010; Krawczyk, Gazzaley, & D'Esposito, 2007; Pessoa & Engelmann, 2010; Savine, Beck, Edwards, Chiew, & Braver, 2010). Yet, only comparatively few studies have investigated the effects of punishment (or avoidance) motivation on executive control (Engelmann & Pessoa, 2007; Savine et al., 2010; Small et al., 2005). Available evidence indicates that punishment incentives can be as effective as reward incentives in promoting cognitive performance. However, it has been proposed that distinct brain regions/mechanisms might mediate the motivational effects of rewards vs. punishments on control processes (Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Gray, Braver, & Raichle, 2002; Higgins, 1997; Harmon-Jones, Lueck, Faern, & Harmon-Jones, 2006; Small et al., 2005).

An important aspect in examining the effects of appetitive vs. aversive motivation on learning mechanisms obviously concerns the processing of performance feedback. As was already discussed in the theoretical part of this thesis, several studies addressed the question of how outcome value affects feedback processing. Most studies indicated that the FRN evaluates outcomes in a binary fashion as good or bad (e.g., Gehring & Willoughby, 2002; Hajcak, et al., 2006; Sato et al., 2005; Toyomaki & Murohashi, 2005) and tracks their relative rather than absolute value (Holroyd et al., 2004). By contrast, the feedback-evoked P300 appears to be sensitive to the magnitude (but not valence) of the outcome (Yeung &

Sanfey, 2004). Interestingly, recent findings suggest that feedback processing might proceed from an earlier coarse evaluation of the outcome as good or bad to a more fine-grained analysis incorporating both valence and magnitude (e.g., Goyer, et al., 2008; Kreussel et al., 2011, Philastides et al., 2010). In the cited studies, however, the feedback stimulus itself carried the motivational information. Thus, it remains unknown whether the processing of feedback stimuli indicating "only" the appropriateness of the response is sensitive to motivational/affective manipulations.

Another relevant question concerns the influence of reward vs. punishment motivation on monitoring of internal performance cues. Previous research suggested that the specific impact of appetitive vs. aversive motivational cues on the activity of the medial prefrontal performance monitoring system is largely determined by individual differences in reward vs. punishment sensitivity. For instance, Boksem and coworkers (2008) demonstrated that highly punishment sensitive individuals showed a larger Ne to errors associated with monetary losses compared to those associated with reward omission. By contrast, highly reward sensitive individuals showed a larger Ne in the reward omission condition compared to the loss condition. The authors explained their findings by assuming that highly punishment and reward sensitive individuals experienced errors as more or less aversive in the two conditions. Another study compared to effects of punishment (errors were followed by unpleasant tones) and reward motivation (correct responses resulted in monetary gains) in high- vs. low-socialized individuals (Dikman & Allen, 2000). Whereas the Ne did not differ between the two conditions in high-socialized individuals, low-socialized participants exhibited reduced Ne amplitudes in the punishment condition.

However, there are several factors that may have limited the generalizability of the described findings. First, the findings of Dikman and Allen (2000) were potentially confounded by the fact that rewarding and punishing cues differed on several dimensions, such as modality and quality. Second, in the Boksem et al.'s (2008) study appetitive vs. aversive motivation was manipulated in a between-subjects design. Therefore, the critical contrasts involved different subjects. A further problem was the small sample size of only 14 participants in the punishment and reward condition, respectively. Moreover, a closer inspection of their data suggests that the reported effects were mainly driven by differences

in the punishment condition, whereas personality differences did not appear to modulate the amplitudes in the reward condition. In the punishment condition, high punishment and low reward sensitivity were associated with a larger Ne than low punishment and high reward sensitivity. This was further corroborated by the observation that the Ne significantly correlated with punishment sensitivity[24] in the punishment condition only, whereas no reliable correlations were found between the Ne and either reward or punishment sensitivity in the reward condition.

Notably, Potts (2011) recently reported larger Ne amplitudes for errors resulting in monetary losses compared to errors resulting in failure to obtain monetary rewards, indicating that appetitive and aversive motivation differentially engage the medial prefrontal performance monitoring system. Given that gain and loss anticipation is associated with the induction of positive and negative affective states, respectively, this view is also in line with findings by Wiswede et al. (2009a) discussed earlier. The authors showed that the presentation of negative but not positive affective pictures led to an increase in Ne amplitude. Similarly, a recent study demonstrated that the induction of positive affect was associated with an attenuation of the Ne in a subsequent working memory task (van Wouwe, Band, & Ridderinkhof, 2011).

It should be noted, however, that incentive manipulation has not consistently been found to affect the Ne (Chiu & Deldin, 2007; Potts, George, Martin, & Barratt, 2006b). Critically, two studies that failed to show Ne modulations, suffered from potential methodological weaknesses. Chiu and Deldin (2007) used a block design to compare reward, punishment, and neutral condition. The experiment started with a neutral block, followed by reward and punishment block that were counterbalanced across participants. Hence, it cannot be ruled out that the effects of incentive manipulation were distorted by disengagement[25] (Tops & Boksem, 2010) and context effects (Holroyd et al., 2004). Further potential confounding factors in a block design include habituation, attentional, strategic or

[24]There was also a marginally significant negative correlation between reward sensitivity and Ne amplitude in the punishment condition. The (unexpected) effects of reward sensitivity on the Ne in the punishment condition were potentially driven by a negative correlation between punishment and reward sensitivity obtained in this study.

[25] Indeed, largest Ne amplitudes were found in the neutral condition. Moreover, the findings by Luu et al. (2000) suggest that reward and punishment block might have been differentially affected by disengagement.

anticipatory effects. In the study by Potts and colleagues (2006b), incentive value was coded by the identity of the center letter in a flankers task (T or N). Thus, on each trial, the reward (punishment) cue was flanked by four punishment (reward) cues. It is also arguable whether the incentive information was available early enough to effectively bias early error processing as reflected in the Ne. Furthermore, in both studies, the monetary rewards and punishments were relatively small (5 Cent) and hence rather unlikely to have a strong motivational/affective consequences.

Additional evidence for a differential impact of appetitive and aversive motivation on performance monitoring comes from human fMRI studies. In particular, Taylor and colleagues (2006) found greater activation of the rACC in response to errors associated with losses compared to those associated with failure to gain. By contrast, both error types elicited larger activation of the dACC than did errors in a neutral condition. From their findings, the authors concluded that the loss-related activation of rACC reflected the affective appraisal of more costly errors, whereas the incentive-related activation of the dACC indicated increased motivation. Moreover, Simões-Franklin and coworkers (2010) showed that motivational influences modulate tonic activity in the dACC but phasic error-related activity in the rACC. The authors suggested that the tonic activity in the dACC reflected more cautious performance and increased proactive control, whereas the phasic activity in the rACC was linked to reactive control in conjunction with affectively more salient errors. These findings are broadly consistent with the idea that both the rACC and the dACC contribute to Ne generation, with activity in the rACC reflecting the “affective” component of error processing (Luu et al., 2003; van Veen & Carter, 2002).

In fact, electrophysiological studies provided some evidence for functional dissociations between rACC and dACC in error and feedback processing. Holmes and Pizzagalli (2008) found increased error-related rACC and mPFC responses in the Ne time-window for depressed participants compared to healthy controls. Another study investigated dissociations in error processing as a function of OC-symptomatology using a flankers task and a probabilistic learning task (Cavanagh et al., 2010b). In the flankers task, individuals with high OC-symptomology exhibited larger Ne amplitudes than those with low symptomatology. Source analysis revealed comparable error-related dACC power in both

groups, but greater error-related rACC power in the High-OC group. This result is in line with the notion that greater rACC activation underlies the larger Ne amplitude in high OC individuals. By contrast, the Ne was smaller in the High-OC compared to the Low-OC group during probabilistic learning. Moreover, the Low-OC group showed relatively increased error-related rACC activation and decreased error-related dACC activation. On the basis of these findings, the authors concluded that dissociable medial prefrontal systems may support performance monitoring in the two types of tasks. Furthermore, Santesso and colleagues (2011a) showed that the increase in FRN amplitude to negative feedback in individuals with high trait level negative affect was associated with greater rACC but not dACC activation.

Although important insights have been gained from these studies, critical questions on the role of motivational-affective processes in performance monitoring remain unsolved. On the one hand, ERP studies examining the influences of reward and punishment motivation rarely reported how the putative motivation-related differences in error processing relate to flexible behavioural adaptation. Interestingly, an early study by Gehring and colleagues (1993) found larger Ne amplitudes in conjunction with more pronounced error-related behavioural adjustments (response squeeze, post-error slowing, error correction rate) for errors resulting in higher monetary losses. This finding, however, remains somewhat ambiguous because the incentive manipulation was confounded with error probability and a different weighting of speed vs. response errors. On the other hand, neither of the electrophysiological studies focusing on differential contributions of rACC and dACC involved an experimental manipulation of affective-motivational significance. Therefore, they did not provide a straightforward test of the putative involvement of the rACC in processing the affective salience of an error. Furthermore, due to the relatively low temporal resolution of the BOLD signal, it remains unclear whether the pattern of error-related activity in the rACC observed by Taylor et al. (2006) and Simões-Franklin et al. (2010) indeed reflects processes in the latency range of the Ne.

Thus, Experiment 3 examined whether manipulations of incentive value modify error and feedback processing – as reflected in the Ne, FRN, and Pe – and how these modulations relate to behavioural adaptations during reinforcement learning. Specifically, the study aimed to determine (1) whether penalizing errors in terms of losing vs. not winning

money differentially affects Ne, FRN, and Pe over the course of learning, (2) how error-related neural activities in dorsal and rostral ACC are affected by this incentive manipulation, and (3) whether incentive-related modulations in Ne amplitude and underlying source activity predict goal-directed behavioural adjustments during learning. EEG source localization was performed using standardized Low-Resolution Electromagnetic Tomography (sLORETA, Pascual-Marqui, 2002).

Study Design

A feedback-based learning task was applied that included three different incentive conditions. In the gain condition, correct responses were rewarded with a win (50 Cent), whereas incorrect responses led to a neutral outcome (0 Cent). In the loss condition, incorrect responses were penalized with a loss (50 Cent), whereas correct responses resulted in a neutral outcome (0 Cent). In the neutral condition, both correct and incorrect responses led a neutral outcome (0 Cent). Gain, loss, and neutrals trials were presented in a pseudo-random order throughout the learning task. In order to minimize strategic adjustments in response speed across the incentive conditions, that is, more accurate but slower responding on gain and loss compared to neutral trials, the same adaptive response deadline algorithm as in Experiment 1 and 2 was used. Responses that exceeded the deadline were handled as errors in terms of monetary pay-off. Since the first experiments showed that participants learned very quickly, the number of stimulus repetitions within a learning block was considerably reduced.

Research Predictions

The rationale for the incentive manipulation used in this study was to alter the motivational and affective salience of erroneous and correct responses. Although one would intuitively expect monetary rewards and penalties to improve task performance, the empirical evidence on this issue is mixed (Bonner, Hastie, Sprinkle, & Young, 2000; Camerer & Hogarth, 1999). In fact, a meta-analysis by Bonner and coworkers (2000) found beneficial effects of incentives only in about half of the studies. Notably, Dambacher and colleagues

(2011) showed that in speeded response tasks punishing slow responses rather than punishing erroneous responses resulted in performance improvements. In addition, Chiew & Braver (2011) suggested that preparatory cues are critical for advantageous incentive-related effects to occur. However, it remains unclear whether the cue has to contain response-relevant information to be effective. For instance, Hajcak and coworkers (2005) presented cue indicating the value of points that could be won on the next trial but did no find incentive-related performance improvements. Thus:

Prediction 1: *(a)* Applying a deadline procedure and penalizing errors emphasized both accuracy and speed in the current study. Therefore, given the findings by Dambacher et al. (2011), accuracy should be moderately improved in the gain and loss condition compared to the neutral condition. *(b)* It has been suggested that losses have a higher impact on behavioural decisions than do gains of equivalent magnitude (Kahneman & Tversky, 1979). Still, evidence in support of the notion that penalties have more pronounced influences on cognitive performance than rewards is scarce. Hence, it is unclear whether participants put more emphasis on avoiding losses than obtaining rewards. *(c)* Furthermore, it is an open question whether the impact of the incentive manipulation changes with learning. However, as the task is more challenging at the beginning of learning, it conceivable that limited resources are allocated more efficiently to loss and gain trials during early stages of learning.

The results of Experiment 1 and 2 suggested that participants learned quickly. Thus, the large number of trials contained in the bins as well as motivational disengagement might have disguised the effects of learning on Ne and FRN. As the number of stimulus repetition was considerably smaller in the present study, the predictions were:

Prediction 2: *(a)* The Ne increases as learning progresses. *(b)* The FRN decreases over the course of learning.

Previous research showed that motivationally and affectively more salient errors are associated with a larger Ne than less salient errors (Gehring et al., 1993; Hajcak et al., 2005). Specifically, it has been demonstrated that the Ne is greater for errors resulting in monetary losses compared to those resulting in reward omission (Potts, 2011). Moreover,

the results of the first study point to a relationship between the neural processes underlying the Ne and learning-related behavioural adaptation and indicate that the Ne may be a marker of aversively motivated reactive control. Moreover, the first study showed that the effects of failure were more pronounced the better participants were able to represent the correctness of their responses. It is unclear, however, whether failure to gain leads to changes in Ne amplitude compared to a neutral condition. Whereas a reduction of the Ne has been found after the induction of positive affect (van Wouwe et al., 2011), others failed to obtain such an effect for trial-by-trial manipulations of affective state (Wiswede et al., 2009a). Therefore, the predictions were:

Prediction 2: *(a)* The Ne is larger for errors resulting in losses compared to those resulting in failure to gain or neutral outcomes. *(b)* The Ne for errors resulting in failures to gain is smaller or comparable to the Ne on neutral trials. *(c)* The Ne magnitude predicts error-related behavioural adjustments, i.e., post-error accuracy. *(d)* The relationship between Ne amplitude and post-error accuracy is more pronounced for errors resulting in monetary losses than those resulting in neutral outcomes. *(e)* The effects of incentive motivation on the Ne amplitude are more pronounced during later stages of learning.

To my knowledge, no prior study explicitly addressed the question of how the motivational/affective significance of a cognitive task *per se* influences the processing of performance feedback. Yet, larger FRN amplitudes have been found in individuals scoring high on punishment sensitivity and trait level negative affect (e.g., Balconi & Crivelli; Santesso et al., 2011b). However, Experiment 1 and 2 revealed only little evidence for an influence of self-relevant failure on feedback processing.

Although previous research suggested that the FRN classifies outcome value in a binary and context-dependent fashion, more recent evidence indicates that the FRN might also code aspects related to the magnitude of an outcome (e.g., Hoyer et al., 2008; Kreussel et al., 2011). In particular, it has been shown that larger losses and smaller gains are associated with relatively larger FRNs. However, more pronounced learning-related modulations have been found in the ERPs to positive feedback (e.g., Eppinger et al., 2008, 2009). Therefore, it was reasoned that the effects of reward and punishment motivation on

feedback processing might differ for positive and negative feedback. Hence, the predictions were:

Prediction 3: *(a)* The FRN in terms of the difference between positive and negative feedback is larger in the gain and loss condition compared to the neutral condition. *(b)* The FRN to positive and negative feedback is differentially modulated in gain vs. loss condition. *(c)* The effects of incentive motivation on the FRN change over the course of learning.

Furthermore, previous research and the findings of Experiment 1 and 2 suggest that the Pe is rather unaffected by motivational/affective manipulations. Nonetheless, the Pe has been associated with conscious error detection (Nieuwenhuis et al., 2001; Steinhauser & Yeung, 2010) and the motivational significance of an error (Overbeek et al., 2005). In particular, the Pe is thought to reflect activity of a more slowly operating deliberate error monitoring system that complements neural processes underlying the Ne (Overbeek et al., 2005). Thus, it was difficult to formulate a well-founded hypothesis on the impact of the present incentive manipulation on the Pe:

Prediction 4: *(a)* The Pe increases with learning. *(b)* It is an open question whether the Pe is sensitive to incentive value.

It has been proposed that the rACC is involved in affective aspects of error processing and contributes to variations of the Ne due to affective/motivational manipulations. Source localisation studies have established a link between enhanced error-related activity in the rACC and increased Ne amplitudes in OCD and depression (Cavanagh et al., 2010b; Holmes & Pizzagalli, 2008). At the same time, evidence from fMRI studies points to a link between enhanced activity in the dACC and incentive-related increases in effort and between increased activity in the rACC and affective salience of errors. Thus:

Prediction 5: *(a)* Errors in the loss condition are associated with greater activity in the rACC during the Ne time window than errors in the gain or neutral condition. *(b)* Errors in gain and loss condition are associated with greater activity in the dACC during the Ne time

window compared to errors in the neutral condition. *(c)* Error-related activity in dACC and rACC differentially predict behavioural adjustments in the three incentive conditions.

Methods

Participants

Twenty-two undergraduate students volunteered to participate in this study for payment (8 Euro per hour) or course credit. Participants had normal or corrected-to-normal vision, no self-reported history of neurological or psychiatric diseases and were free from psychoactive medication or drug use. Two participants were excluded because they did not commit enough errors to obtain reliable measures of Ne (at least 15 error trials in each condition). Data from two additional participants had to be discarded due to technical problems during EEG recording. The effective sample thus included 18 subjects (11 females; mean age 24.83 years; age-range 19 to 33 years). All gave informed written consent in accordance with the protocols approved by the local ethics committee of Saarland University prior to the start of the experiment.

Stimuli and Task

During the learning task, participants were presented with different coloured images of objects (Snodgrass & Vanderwart, 1980) and were required to press one of two response keys, after which either the word “RICHTIG” (“correct”), “FALSCH” (“incorrect”) or “ZU LANGSAM” (“too slow”) was shown. Participants had to infer the correct stimulus-response mappings by trial and error on the basis of the feedback information. Stimuli were assigned to one of three incentive conditions (gain, loss, and neutral outcome). Each imperative stimulus was preceded by a cue that indicated the incentive value of the upcoming target. The gain cue informed participants that they would win 50 Euro Cents if they responded correctly but 0 Euro Cents if they responded incorrectly or missed the response deadline (see Trial Procedure). Conversely, the loss cue indicated that participants would lose 0 Euro Cent if they responded correctly but 50 Euro Cents if the response was incorrect or too slow. On neutral trials, there was no chance to gain or lose money (see Table 3). Participants were

instructed to use the cue information in order to maximize their profit. At the end of the experiment, all received the same monetary bonus (7.50 Euro).

Tabelle 3. Overview of the three incentive conditions

	Payoff	
Incentive Condition	*Correct*	*Incorrect*
Gain Condition	+50 Cent	±0 Cent
Loss Condition	±0 Cent	−50 Cent
Neutral Condition	±0 Cent	±0 Cent

Trial Procedure

On each trial the incentive cue appeared in the center of the screen for 400 ms. After a 400 ms delay, a central fixation cross was displayed for a randomly jittered interval of 250 to 500 ms, followed by the presentation of the target stimulus for 500 ms. Stimuli were presented on a light gray background. As in Experiment 1 and 2, an adaptive response deadline was applied. Based on the proportion of time-out trials, the response window was individually adjusted in steps of 100 ms within an overall range of 400 to 1000 ms. After the key press, a blank screen was displayed for 500 ms and then visual feedback was provided for again 500 ms. The next trial started after a randomly jittered 500 to 800 ms interval (see Figure 22 for a schematic overview of the trial procedure).

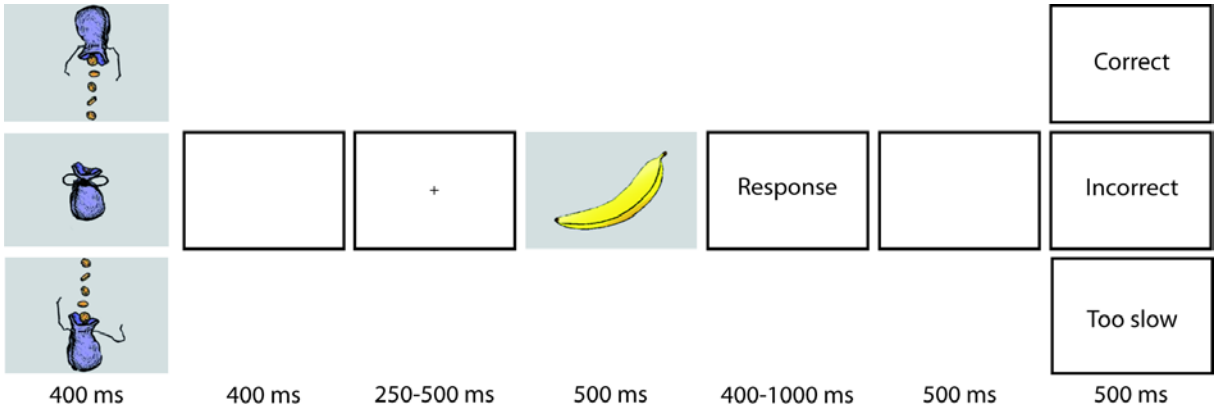

Figure 22: Schematic overview of trial procedure

Experimental Procedure

Prior to the learning task, participants completed a short questionnaire about demographics and health. The learning task consisted of a short practice block (45 trials) and ten experimental blocks, with self-paced breaks every 30 trials. During the breaks, participants were presented with a screen displaying two vertical bars. Participants were told that the height of the left bar represented the maximum amount of money that theoretically could be earned up to that point, whereas the height of the right bar represented the amount of money they had actually earned. They were further told that it was impossible to choose correctly on every trial and that therefore the right bar was always smaller than the left one. Within one block, two stimuli were assigned to each incentive condition, yielding a total of six new stimuli per learning block. Within one block, two stimuli were assigned to each incentive condition, yielding a total of six new stimuli per learning block. For all three incentive conditions, one stimulus was mapped to the left response key and the other one to the right response key. Each stimulus was presented 15 times in pseudo-randomized order throughout the learning block, with the same stimulus appearing not more than two times in a row. The assignment of stimuli to incentive condition and response key was randomized across participants. To avoid ceiling effects in learning, invalid feedback was provided on 12 trials within each block. Only valid trials were included in the analyses.

Electrophysiological Recording

The EEG was recorded from 58 Ag/AgCl electrodes arranged according to the extended 10-20 system, referenced to the left mastoid, using Brain Amp DC Recorder (BrainVision recorder acquisition software). Data were sampled at 500 Hz in DC mode with a low-pass filter at 70 Hz. Impedances were kept below 5 kΩ. Electrodes placed on the outer canthi of the two eyes and on the infra- and supra-orbital ridges of the left eye recorded the horizontal and vertical electrooculograms. The data were re-referenced offline to the linked mastoids and band-pass filtered from 0.1 to 30 Hz. The impact of blinks and eye movements was corrected using an independent component analysis algorithm implemented in the BrainVision Analyzer Software Package (Brain products, Gilching, Germany). Trials

containing EEG activity exceeding ± 100 μV, changing more than 50 μV between samples or containing DC drifts were eliminated by a semiautomatic artefact inspection procedure.

Data Analyses

Behavioural data analyses. Responses exceeding the adaptive deadline were excluded from further analyses (gain: *M* = 0.05, *SD* = 0.01; loss: *M* = 0.05, *SD* = 0.01; neutral: *M* = 0.06, *SD* = 0.01). Mean RTs were 440 ms (*SD* = 45 ms) in the gain condition, 437 ms (*SD* = 43 ms) in the loss condition, and 444 ms (*SD* = 46 ms) in the neutral condition. Neither the proportion of time-out trials nor the RTs did significantly differ between the incentive conditions (*p*-values > .16), suggesting that the adaptive response deadline successfully prevented more accurate at the expense of slower responding in gain and loss condition. To examine the course of learning, each block was split into five bins. The bins were created according to the number of stimulus repetitions, i.e., Bin 1 contained presentations 1-3 of the respective stimuli, Bin 2 presentations 4-6, and so on. Within each bin, mean accuracy rates were computed for the three incentive conditions. To analyse trial-to-trial behavioural adjustments, post-error accuracy ("incorrect-switch" performance) was determined by calculating mean accuracy rates for the next presentation of a given stimulus after an erroneous response, separately for each incentive condition. In addition, post-correct accuracy ("correct-stay" performance) was determined as mean accuracy rate for the next presentation of a given stimulus after a correct response.

ERP analyses. The response-locked and feedback-locked epochs were baseline corrected with respect to the average voltage during a -200 to -50-ms-pre-response interval and a 100-ms-pre-stimulus interval, respectively. As in Experiment 1 and 2, the Ne was quantified after 15 Hz low-pass filtering at electrode FCz as the peak-to-peak difference in voltage between the most negative peak between -50 and 100 ms and the largest positive peak in the prior 100 ms. Similar to the first study, there was no clear negative peak following correct responses. Since visual inspection of the waveforms clearly indicated that the correct response-related positivity did not differ between the there incentive conditions, in a second step, difference waveforms were created by subtracting the activity on correct trials from the

activity on error trials (ΔNe). The ΔNe was defined as the mean amplitude in a 20 to 60 ms post-response time window covering the peak of the difference wave in each incentive condition and each bin. Analogous to Experiment 1 and 2, the Pe was measured as the mean amplitude between 200 and 400 ms after the response at electrode Pz. Similarly to the Ne, the FRN was quantified twofold. In a first step, the FRN was defined as peak-to-peak voltage difference between the most negative peak in a 200 to 400 ms time window after feedback onset and the preceding positive peak in a 150 to 300 ms post-feedback interval at electrode FCz, separately for positive and negative feedback. In a further step, ΔFRN amplitude was determined by subtracting the activity after correct feedback from the activity after negative feedback. Because reliable peak detection proved to be difficult for the difference waveforms, mean voltage in the period from 280 to 320 ms post-feedback was calculated to define the ΔFRN amplitude. This time window was chosen based on the average peak latency of the FRN (300 ms). To examine learning-related changes in Ne, FRN, and Pe, EEG epochs were averaged separately for each incentive condition for the first (Bin 1) and the second half of trials within each block (Bin 2).

In addition, although cue-related preparatory processes were not the focus of this investigation, the cue-locked P300 as well as the contingent negative variation (CNV) that precedes task-relevant stimuli were evaluated. Importantly, both components have been associated with changes in voluntary and effortful control of performance (e.g., Falkenstein, Hoormann, Hohnsbein, & Kleinsorge, 2003; Gevins et al., 1990). To analyse cue-related processing during the foreperiod, epochs covering the cue-target interval were created and baseline-corrected with respect to a 100 ms pre-cue interval. The analysis of the P300 and CNV amplitude in the cue-target interval included a grid of 4 × 5 electrodes over frontal, central, and parietal regions (5 rows from frontal to parietal, each including the midline and two inner/outer left and right electrodes: F7, F3, Fz, F4, F8; FC5, FC3, FCz, FC4, FC6; C5, C3, Cz, C4, C6; CP5, CP3, CPz, CP4, CP6; P5, P3, Pz, P4, P6). The P300 was quantified as mean amplitude measure in a 300 to 600 post-cue interval. The CNV amplitude was calculated as the mean voltage within a 200 ms time window before target onset. I chose to evaluate the terminal (target-locked) CNV to avoid confounding influences of the preceding P300.

sLORETA analysis. Standardized intra-cerebral current density power underlying the response- and feedback-related ERPs were computed using sLORETA (Pascual-Marqui, 2002). This EEG source localization technique provides a solution to the inverse problem by assuming similar activation of adjacent neuronal clusters, without a priori specification of the number of active neural sources. The solution space is limited to cortical gray matter and hippocampi and consists of 6239 voxels (voxel size: 5 mm^3). sLORETA calculates the current density power (in amperes per square meter, A/m^2) at each voxel, expanding the minimum norm inverse solution by taking into account the variance of the actual sources and measurement noise (Hämäläinen & Ilmoniemi, 1984). Before statistical testing, current density power was subject-wise normalized to a total power of 1 and log-transformed at each sampling point. Activity was then averaged within a 20 ms time interval centered on the peak of the Ne (24-44 ms). Within each of the three incentive conditions, error-correct contrasts (voxel-by-voxel) were calculated and subjected to non-parametric permutation tests as implemented in the sLORETA software package. Following Cavanagh and colleagues (2010b), Region of Interest (ROI) analyses were performed for the rostral and dorsal subdivision of the ACC. Voxels were assigned to the dACC (BA 24' and 32') and rACC (BA 24 and 32) based on their (*x,y,z*)- coordinates in the Montreal Neurological Institute (MNI) space according to the following rule: dACC if $z > 15$ and $y < 35$, else rACC (for illustration, see Figure 23). The log-transformed standardized current density values were averaged across all voxels within the two ROIs.

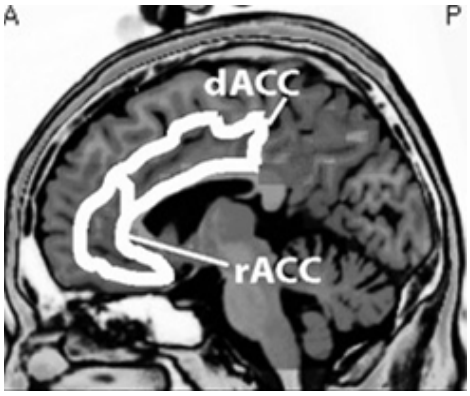

Figure 23: Illustration of the definition of rACC and dACC ROIs (displayed on the sLORETA template)

Statistical analyses. Accuracy and ERP data were analysed using repeated measures analyses of variance (ANOVAs). Whenever necessary, the Geisser-Greenhouse correction was applied (Geisser & Greenhouse, 1958) and corrected p-values are reported together with uncorrected degrees of freedom and the epsilon-values (ε). Pearson's correlations were calculated to examine the relation between Ne amplitude and behavioural measures. Fisher's z tests were used to test the difference between Pearson's correlation coefficients.

Results

Behavioural Data

Overall accuracy. Figure 24 illustrates how mean accuracy rates increased across learning in each of the three incentive conditions. This was confirmed by an *incentive condition* (gain, loss, and neutral) × *bin* (Bins 1-5) ANOVA that yielded a significant main effect of bin [$F(4,68) = 76.60$, $p < .001$, $\varepsilon = .45$]. Moreover, a significant main effect of incentive condition was obtained [$F(2,34) = 6.90$, $p < .01$], indicating that mean accuracy rates differed as a function of trial value. Contrasts revealed accuracy to be higher on gain and loss trials compared to neutral trials [$F(1,17) = 7.94$, $p < .05$], as well as on loss trials compared to gain trials [$F(1,17) = 5.18$, $p < .05$].

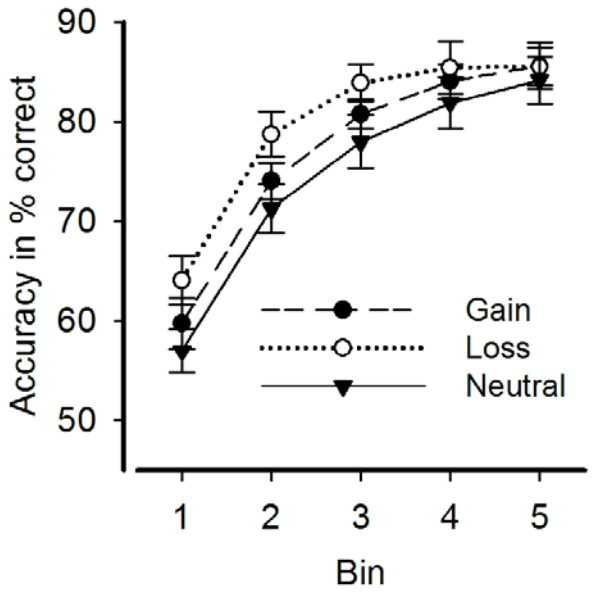

Figure 24: Mean accuracy learning curves for the three incentive conditions. Error bars indicate standard errors.

Trial-to-trial behavioural adaptations. An ANOVA with the factors *correctness* (correct-stay vs. incorrect-switch) and *incentive condition* (gain, loss, and neutral) yielded significant main effects of correctness [$F(1,17) = 143.58$, $p < .001$] and incentive condition [$F(2,34) = 10.24$, $p < .001$] that were qualified by a significant interaction between correctness and incentive condition [$F(2,34) = 4.10$, $p < .05$]. Separate analyses for correct and incorrect choices revealed a significant effect of incentive condition for incorrect-switch performance [$F(2,34) = 8.86$, $p < .01$], but not for correct-stay performance [$F(2,34) = 2.26$, $p = .12$]. Contrasts showed that participants were more likely to switch the response key following incorrect choices in the loss condition compared to gain and neutral condition [$F(1,17) = 17.73$, $p < .01$], whereas there was no significant difference in incorrect-switch performance between gain and neutral condition ($F < 1$) (see Figure 25).

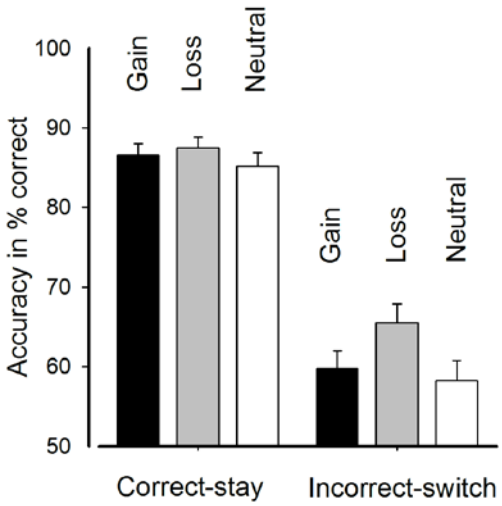

Figure 25: Correct-stay and incorrect-switch performance for the three incentive conditions. Error bars indicate standard errors

In summary, participants showed highest accuracy in the loss condition, followed by gain and neutral condition. This finding indicates that they were able to improve their performance in order to maximize their profit, with being even more motivated to avoid potential losses than to obtain potential rewards. A stronger bias to avoid negative outcomes was also evident from trial-to-trial adjustments in behavioural choices. Participants switched more often to the correct response key when incorrect choices were associated with monetary losses, whereas correct-stay performance was not affected by trial incentive value.

ERP data

Cue-target interval

Figure 26 shows the cue-locked grand-average ERP waveforms for the three incentive conditions at electrode Pz. Motivationally relevant (gain, loss) cues were followed by a larger centro-parietal positivity (P300) than neutral cues. An ANOVA with the factors *incentive condition* (gain, loss, neutral) and *site* on the P300 amplitude revealed a significant main effect of site [$F(14,238) = 49.80$, $p < .001$, $\varepsilon = .17$], indicating that the P300 was largest at centro-parietal midline sites. Moreover, a reliable main effect of incentive condition [$F(2,34) = 54.00$, $p < .001$, $\varepsilon = .80$] showed that the P300 amplitude was greater for the gain and loss condition compared to the neutral condition [$F(1,17) = 71.97$, $p < .001$, $\varepsilon = .80$], whereas no differences were found between gain and loss condition ($F < 1$). The effect of incentive condition was most pronounced at centro-parietal sites, reflected in a significant interaction between incentive condition and site [$F(28,476) = 4.40$, $p < .01$, $\varepsilon = .17$].

Figure 27 presents the target-locked ERP waveforms in the cue-target-interval at electrode Cz. As illustrated in the Figure, a negative slow wave (tCNV) developed about 600 ms after cue onset at fronto-central recording sites. Visual inspection suggested that the tCNV amplitude did not differ for the three incentive conditions. This was confirmed by the statistical analysis that yielded a significant main effect of site only [$F(14,238) = 19.11$, $p < .001$, $\varepsilon = .21$].

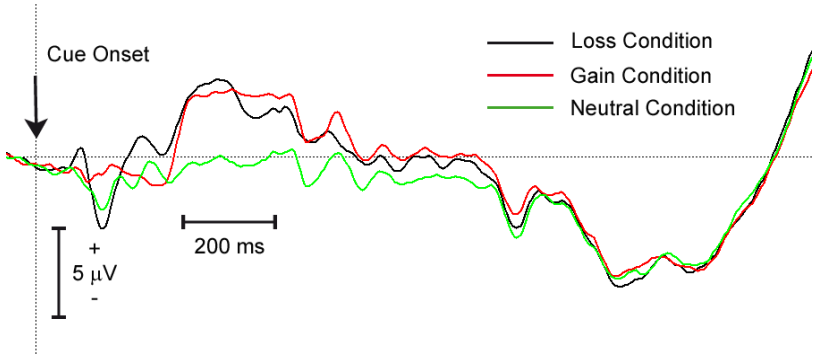

Figure 26: Cue-target-interval: Cue-locked ERPs for the three incentive conditions at electrode Pz

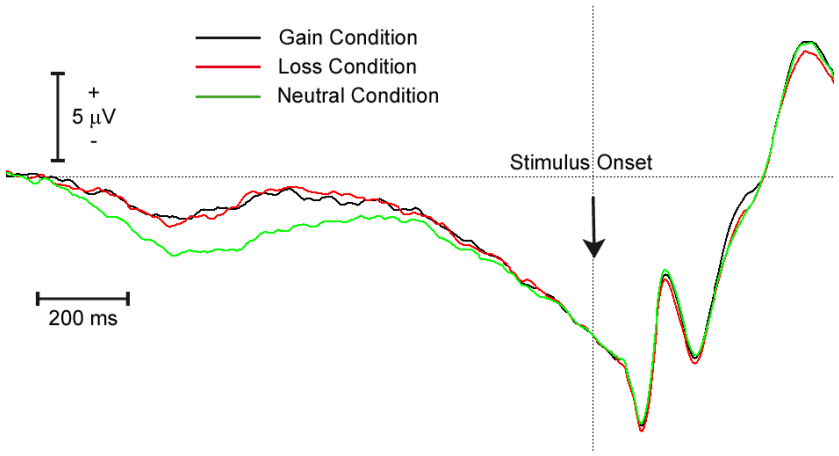

Figure 27: Cue-target-interval: Target-locked ERPs for three incentive conditions in the cue-target-interval at electrode Cz. Note that the difference between neutral condition and gain/loss condition reflects the P300 effect (see Figure 27) that is temporally smeared due to the jittered cue-target-interval.

Response-locked ERPs

Incentive-related modulations in the Ne. Figure 28 presents the ERPs to correct and incorrect responses in Bin 1 and Bin 2 for the three incentive conditions at electrode site FCz. Following incorrect responses the Ne was evident as a negative deflection that increased over the course of learning and appeared larger for loss trials than gain or neutral trials. The peak-to-peak measures of the Ne were subjected to an ANOVA with the factors *incentive condition* (gain, loss, neutral) and *bin* (Bin 1 vs. Bin 2). A significant main effect of bin [$F(1,17) = 6.74$, $p < .05$] confirmed the learning-related increase in Ne amplitude from Bin 1 to Bin 2. Moreover, the analysis yielded a significant main effect of incentive condition [$F(2,34) = 7.63$, $p < .01$]. Contrasts revealed the Ne to be larger on loss trials compared to gain und neutral trials [$F(1,17) = 11.30$, $p < .01$], whereas no amplitude difference was found between gain and neutral condition ($F < 1$).

Analysis of difference waves (ΔNe) corroborated these findings. The ΔNe amplitude reliably varied across bins [$F(1,17) = 27.95$, $p < .001$] and incentive conditions [$F(1,17) = 4.35$, $p < .05$]. Consistent with the peak-to-peak measure, the ΔNe was significantly greater on loss trials compared to gain and neutral trials [$F(1,17) = 8.04$, $p < .05$], but did not differ between gain and neutral condition ($F < 1$). Thus, error processing was specifically affected by outcome valence, reflected in larger Ne amplitudes for errors associated with losses but

not for failures to obtain rewards. For reasons of parsimony, only analyses involving peak-to-peak measures are reported in the following sections.

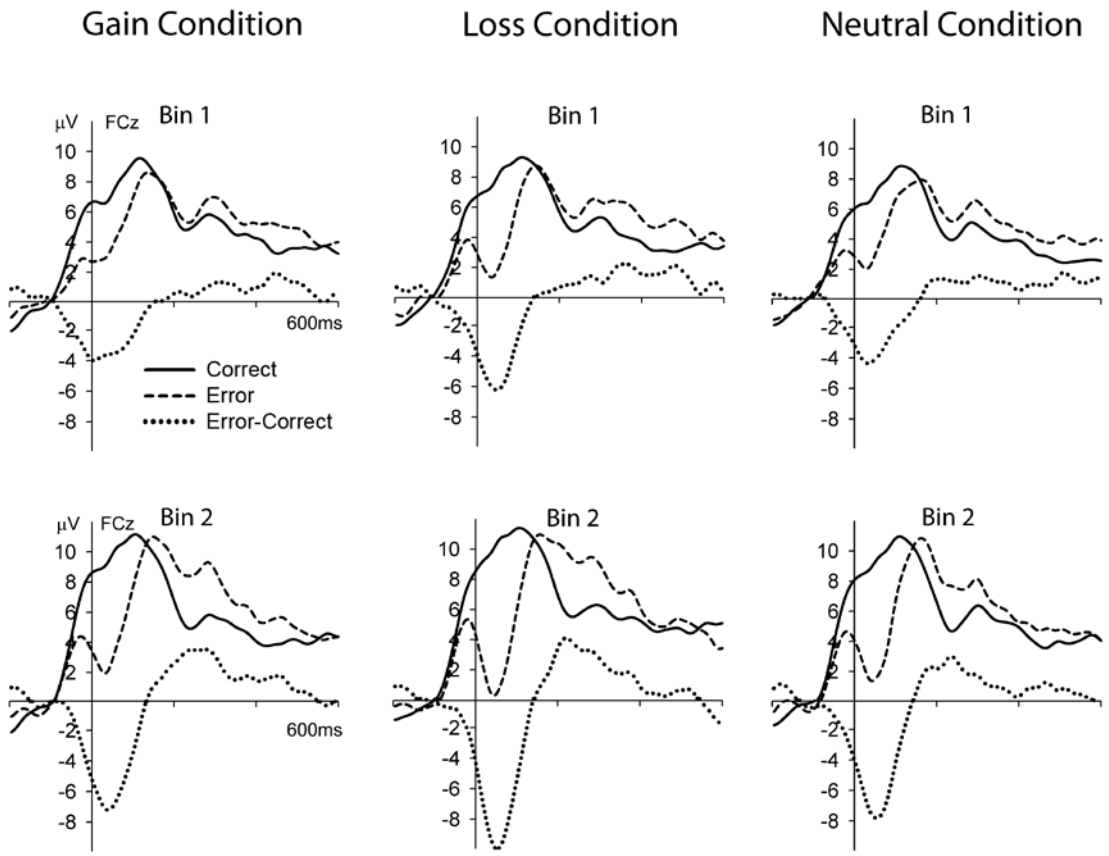

Figure 28: Response-locked ERPs at electrode FCz to correct (solid lines) and incorrect (dashed lines) responses displayed separately for the three incentive conditions and the two bins. Dotted lines represent difference waves (error minus correct)

Loss-related Ne increase does not reflect differences in overall performance. To ensure that the larger loss-related Ne amplitude was not a simple consequence of differences in overall accuracy, a second analysis was run after removing four subjects who clearly performed better in the loss than in the gain condition. The remaining sample showed higher accuracy in gain and loss compared to neutral condition [$F(1,13) = 5.13$, $p < .05$], but performed equally well in gain ($M = 0.78$, $SD = .06$) and loss condition ($M = 0.78$ $SD = .07$) ($F < 1$). Nonetheless, the Ne was significantly larger in the loss condition compared gain and neutral condition [$F(1,13) = 6.18$, $p < .05$], whereas no amplitude difference was found between the latter two conditions (p-values > .12).

Ne amplitude predicts loss avoidance. The analyses of the accuracy and ERP data indicate that loss but not gain anticipation specifically modulates error processing and error-induced learning as reflected in larger Ne amplitude and better incorrect-switch performance. In order to examine the relation between Ne and error-related behavioural adaptations in more detail, correlation analyses between Ne amplitude (averaged across the two bins) and performance scores (overall accuracy, correct-stay and incorrect-switch performance) were conducted, separately for the three incentive conditions (see Figure 29). Reliable correlations were obtained between the Ne on loss trials and incorrect-switch performance in the loss condition [$r = -.59$, $p < .05$] as well as in the gain condition [$r = -.49$, $p < .05$]. The magnitude of the loss-related Ne did not predict correct-stay performance or overall accuracy in any incentive condition (p-values > .14), demonstrating the specificity of the above findings. Furthermore, there were no significant correlations between Ne amplitude in gain or neutral condition and any performance score (p-values > .18). Separate analyses for the two bins, however, revealed a significant correlation between Ne amplitude and incorrect-switch performance during later stages of learning (Bin 2) in the gain condition [$r = -.47$, $p < .05$][26]. By contrast, no reliable relationships between Ne amplitude and performance measures were found for the neutral condition (p-values > .54). Importantly, the correlation between Ne and incorrect-switch performance was significantly larger significantly in the loss condition compared to the neutral condition [$z = -2.12$, $p < .05$]. Yet, the correlation coefficients did not significantly differ between gain and neutral condition[27] [$z = -1.20$, $p = .23$] or gain and loss condition [$z = 1.12$, $p = .26$].

Summary of Ne findings. To summarize, the Ne was larger the loss condition compared to gain and neutral condition. This effect did not change as a function of learning and did not reflect differences in overall performance. Importantly, the Ne reliably predicted error-related behavioural adjustments in the loss condition only. These findings strongly link the neural processes underlying the Ne to aversively motivated behavioural control.

[26] In the loss condition, the Ne correlated significantly with incorrect-switch performance in Bin 1 and 2 (p-values < .05).

[27] However, in Bin 2, the correlation between Ne and incorrect-switch performance differed significantly between gain and neutral ($p < .05$).

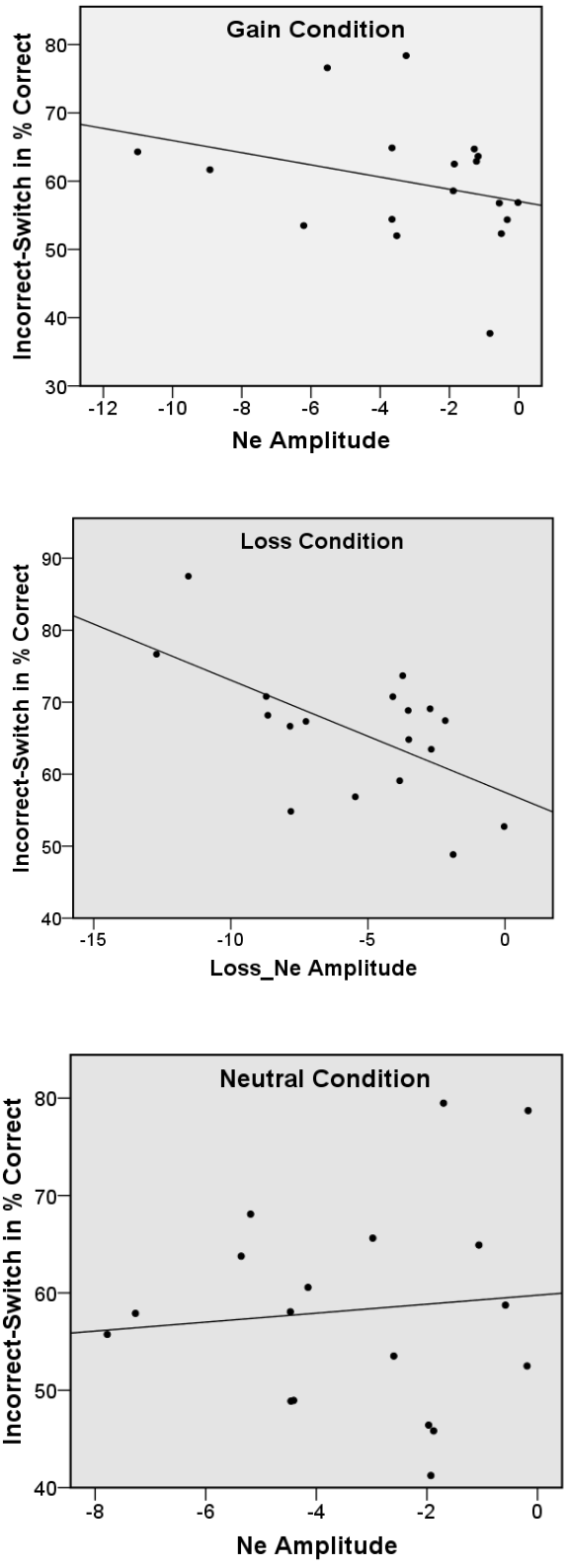

Figure 29: Correlation between Ne amplitude at electrode FCz (averaged across the bins) and incorrect-switch performance, separately for the loss condition ($r = -.59$, $p < .05$), the gain condition ($r = -.27$, n.s.), and the neutral condition ($r = .09$, n.s.).

Error Positivity. The Pe amplitudes (see Figure 30) were subjected to an ANOVA with the factors *incentive condition* (gain, loss, neutral), *bin* (Bin 1 vs. Bin 2) and *feedback type* (positive vs. negative). The analysis yielded a reliable main effect of incentive condition [$F(2,34) = 3.94$, $p < .05$], indicating that the ERPs were more positive-going in the gain and loss condition compared to the neutral condition [$F(1,17) = 4.56$, $p < .05$]. There was also a trend towards larger amplitudes in the loss compared to the gain condition [$F(1,17) = 3.30$, $p = .087$]. Furthermore, the analysis showed that the Pe increased with learning, reflected in significant main effects of feedback type [$F(1,17) = 13.03$, $p < .01$] and bin [$F(1,17) = 7.35$, $p < .05$] and an interaction of feedback type and bin [$F(1,17) = 11.95$, $p < .01$]. Follow-up analyses that were split by feedback type showed that the positivity on error trials ($p < .001$) but not on correct trials ($p = .90$) became larger as learning progressed.

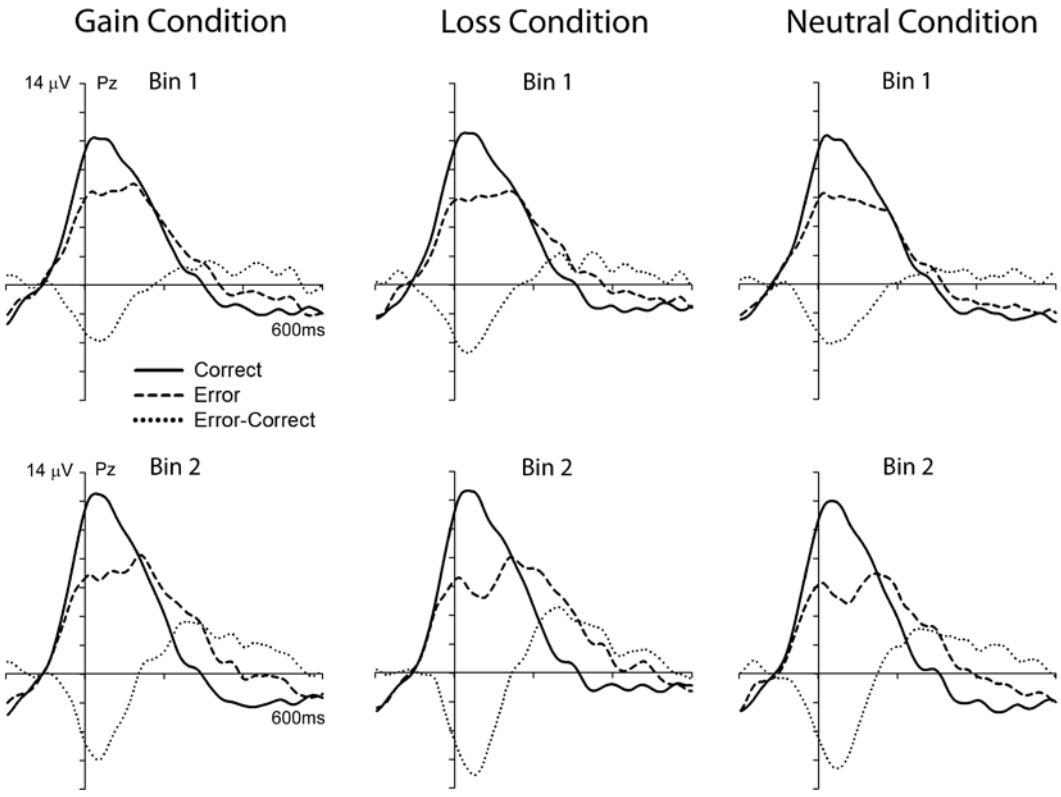

Figure 30: Response-locked ERPs at electrode Pz to correct (solid lines) and incorrect (dashed lines) responses displayed separately for the three incentive conditions and the two bins. Dotted lines represent difference waves (error minus correct)

Thus, the Pe increased with learning. In addition, there was a larger positivity on both erroneous and correct responses in the gain and loss condition compared to the neutral condition.

Feedback-locked ERPs

Feedback-related negativity. The ERPs to positive and negative feedback in the three incentive conditions are presented in Figure 31. From visual inspection, it appeared that there were only small amplitude differences between positive and negative feedback. Irrespective of feedback valence, largest FRN amplitudes were elicited in the neutral condition. The peak-to-peak measures of the FRN were analyzed using an ANOVA with the factors *incentive condition* (gain, loss, neutral), *bin* (Bin 1 vs. Bin 2) and *feedback type* (positive vs. negative). The analysis yielded a reliable main effect of feedback type, indicating that the was larger FRN after negative compared to positive feedback [$F(1,17) = 5.24$, $p < .05$]. Furthermore, a significant main effect of incentive condition was obtained [$F(2,34) = 6.54$, $p < .01$]. Contrasts revealed that the FRN was greater in the neutral condition compared to gain and loss condition [$F(1,17) = 13.41$, $p < .01$], but did not differ between gain and loss condition ($p = .41$). The analysis of the ΔFRN amplitude produced qualitatively similar results to the analysis of the original waveforms and is not reported here.

Control analysis of the P300. Figure 31 illustrates that the FRN amplitude may have been confounded by differences in the feedback-evoked P300. Therefore, mean amplitudes in a 300 to 400 ms post-feedback interval at electrode Pz were analysed using the same ANOVA design as for the FRN amplitudes. The analysis yielded reliable main effects of feedback type [$F(1,17) = 7.94$, $p < .05$] and learning condition [$F(2,34) = 5.64$, $p < .01$] that were qualified by an interaction of feedback type and learning condition [$F(2,34) = 3.93$, $p < .05$]. Separate analyses showed that the P300 to positive feedback did not differ between the three incentive conditions ($p = .72$). By contrast, negative feedback elicited larger P300 amplitudes in the gain condition compared to loss and neutral condition ($p < .01$), whereas no differences were found between the latter two conditions ($p = .38$). Furthermore, the analysis revealed a significant main effect of bin [$F(1,17) = 5.70$, $p < .05$] and an interaction of bin and

feedback type [$F(1,17) = 14.05$, $p < .01$]. Separate analyses showed that the P300 decreased from Bin1 to Bin 2 for positive feedback ($p < .001$) but not for negative feedback ($p = .75$). Hence, if anything, the P300 might have attenuated the FRN after negative feedback in the gain condition. As can be seen from Figure 31, this would decrease rather than increase the differences between the incentive conditions[28].

Thus, better learning in the motivationally significant gain and loss conditions did not appear to be accompanied by more differentiated monitoring of positive and negative feedback as reflected in the FRN.

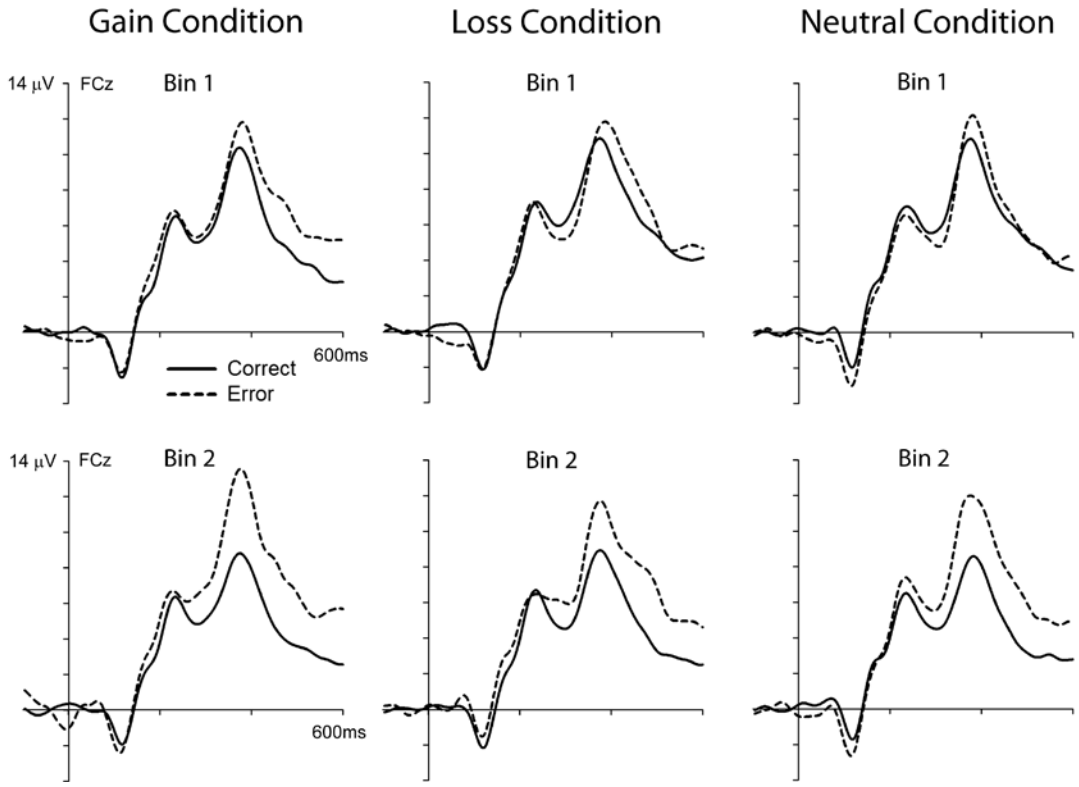

Figure 31: Feedback-locked ERPs at electrode FCz to positive (solids lines) and negative (dashed lines) displayed separately for the three incentive conditions and the two bins.

[28] In a further control analysis, feedback-locked epochs were transformed to current source density (CSD) estimates (Kayser & Tenke, 2006). CSD acts as a spatial filter that amplifies the representation of local potentials and attenuates broadly distributed/distal activities, thereby removing spatial redundancy caused by volume conduction and by this attenuating the influence of overlapping ERP components. However, this analysis yielded a qualitatively similar pattern of findings for the FRN as the analysis of the original waveforms.

sLORETA Analysis

Whole-brain analysis. Figure 32 shows sLORETA images for the error-correct contrast, separately for the three incentive conditions. Whole-brain analysis revealed that parts of the posterior cingulate and precuneus were less active on incorrect than correct trials, particularly in gain condition. Moreover, in the loss condition, greater error-related activity was found for a large cluster of voxels in the midcingulate and the adjacent SMA and pre-SMA. By comparison, on neutral trials, errors were mainly characterized by greater rACC activity (all *p*-values < .05, corrected for multiple comparisons).

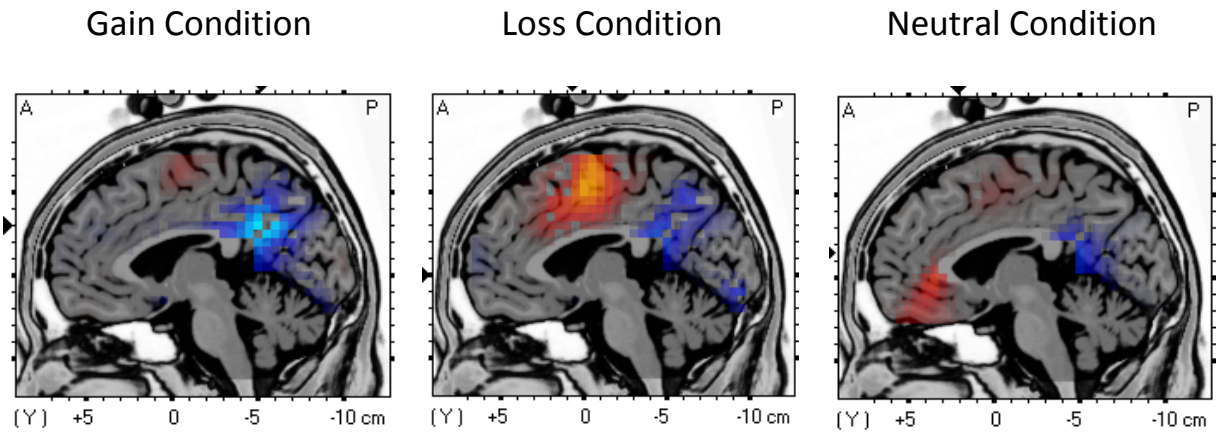

Figure 32: Voxel-by-voxel sLORETA statistical threshold images (*x* = -4) for the error > correct contrast in the three incentive conditions displayed on the MNI templates. Hotter colours indicate relatively higher activity for errors; cooler colours indicate relatively lower activity for errors.

ROI analysis[29]*.* Standardized current density power for dACC and rACC ROI were subjected to an ANOVA with the factors *incentive condition* (gain, loss, and neutral), *correctness* (correct vs. incorrect), and *ROI* (dACC vs. rACC) yielded a reliable main effect of ROI [$F(1,17) = 12.94$, $p < .01$] as well as significant interactions between correctness and ROI [$F(2,34) = 5.72$, $p < .05$, $\varepsilon = .70$] and incentive condition, correctness, and ROI [$F(2,34) = 4.25$, $p < .05$]. Separate ANOVAs for the two ACC subregions revealed a significant interaction between incentive condition and correctness for the dACC ROI only [$F(2,34) = 4.24$, $p < .05$]. Contrasts showed that the error-correct difference in dACC activity was

[29] As the whole-brain analysis identified error-related source activity in areas other than the ACC for the loss condition, additional analyses were conducted that included further areas such as the BA6, BA8, and BA9. These analyses yielded findings that were qualitatively similar to the reported results of for the ACC ROIs.

significantly larger in the loss condition than in gain and neutral condition [F (1,17) = 9.70, p < .05], but did not differ between gain and neutral condition (p = .28) (see Figure 32). Indeed, dACC power was significantly enhanced following errors on loss trials only [t(17) = 3.38, p < .01]. Thus, the pattern of dACC activation largely reflected the modulation of the Ne amplitude across the incentive conditions, whereas no evidence for incentive-related differences in rACC reactivity was found. The latter result appears somewhat discrepant with the whole-brain analysis that revealed phasically increased rACC activation in the neutral condition but not in the gain or loss condition. Given that the significant cluster in the whole-brain contrast contained a much smaller number of voxels than the rACC ROI, it is likely that non-responsive voxels attenuated the differences between the incentive conditions in the ROI analysis.

Incentive-specific relations between ROI activity, Ne amplitude, and behavioural adaptation. To examine the relationship between subregional cingulate activation and Ne amplitude more specifically, multiple regression analyses were conducted for the three incentive conditions with error-related rACC and dACC power as predictors. On gain trials, both error-related dACC and rACC activation reliably predicted larger (i.e., more negative) Ne amplitudes ($ß$ = −.42 and −.68, t = −2.67 and −4.35, p < .05 and .01, respectively; R^2 = .64). Similarly, greater rACC activation was positively related to Ne magnitude in the neutral condition. There was, however, only a marginally significant relation between dACC activity and Ne on neutral trials ($ß$ = −.53 and −.38, t = − 2.88 and −2.11, p < .05 and .054, respectively; R^2 = .56). In the loss condition, only dACC power reliably predicted Ne amplitude ($ß$ = −.71, t = −3.93, p < .01; R^2 = .52). Importantly, when the effect of dACC/rACC activation during correct trials was partialled out, the same pattern of correlations between residual error-related ROI values and Ne amplitude emerged.

In a second step, incorrect-switch performance was regressed on error-related rACC and dACC power. In the gain condition, greater error-related rACC activity predicted more efficient post-error behavioural adjustments ($ß$ = .62, t = 3.12, p < .01, R^2 = .41). Similarly, rACC power scaled with incorrect-switch performance in the neutral condition ($ß$ = .48, t = 2.13, p = .05). Interestingly, there was also a significant negative relation between dACC

activation and incorrect-switch performance (*ß* = −.57, *t* = −2.57, *p* < .05), indicating that reduced dACC power during errors was accompanied by higher error correction rates on neutral trials (R^2 = .36). In contrast, neither rACC nor dACC power were significantly related to error-related behavioral adjustments in the loss condition (*ß* = .25 and .30, *t* = 1.06 and 1.24, *p* = .30 and .23, respectively; R^2 = .16). However, including gain-related dACC power into the equation increased the predictive power of loss-related dACC activation. Greater dACC power predicted better incorrect-switch performance in the loss condition when dACC power on gain trials was controlled for (*ß* = .84, *t* = 2.28, *p* < .05, R^2 = .33). Thus, it appeared that dACC power in the gain condition suppressed criterion-irrelevant variance of dACC power in the loss condition. This "error" variance might reflect a valence-independent effect of motivation on dACC reactivity. No other suppressor effects were obtained in this analysis. The pattern of findings did not change when the effect of dACC/rACC activation during correct trials was accounted for[30].

Summary of findings. To summarize, dACC activation was larger following errors than correct responses and predicted Ne amplitude in the loss condition. Moreover, when accounting for dACC power on gain trials, loss-related dACC reactivity predicted incorrect-switch performance. Although the whole-brain analysis failed to reveal reliable differences in ACC activation between incorrect and correct trials in the gain condition, both error-related rACC and dACC power predicted Ne magnitude. Moreover, greater rACC power was associated with better incorrect-switch performance. For the neutral condition, sLORETA contrasts revealed higher rACC power following errors compared to correct responses. In line with this, error-related rACC activity predicted Ne amplitude as well as incorrect-switch performance. In contrast to the loss condition, dACC activity was inversely related to incorrect-switch performance.

Taken together, these findings indicate that the contribution of different cingulate subregions to error processing and learning-related behavioural adaptation varied as a

[30] No significant relations were found between correct-stay performance and dACC/rACC activity following correct responses.

function of the specific task context. However, there was no evidence to suggest that greater rACC activation underlies the larger Ne amplitude for errors resulting in monetary losses. Instead, differences in dACC power appeared to underlie the increase of the Ne in the loss condition compared to gain and neutral condition. Conversely, for the latter two conditions, rACC power was associated with larger Ne amplitudes and more efficient error-related behavioural adjustments, possibly reflecting a complementary role of this brain region when action monitoring functions subserved by the dACC are less dominant.

9. Discussion of Experiment 3

The main goal of this study was to investigate the impact of appetitive and aversive motivation on performance monitoring and goal-directed behavioural adjustments during feedback-based learning. To this end, the study examined modulations in the amplitude of Ne, FRN, and Pe across different incentive conditions, in which errors led to (1) failure to gain, (2) losses or (3) neither of both. In particular, the present research aimed to determine whether larger Ne amplitudes for motivationally and affectively more salient errors reflect a differential involvement of dorsal and rostral cingulate subregions in error processing and the initiation of subsequent behavioural changes. To this end, an EEG source localization technique known as standardized Low-Resolution Electromagnetic Tomography (sLORETA, Pascual-Marqui, 2002) was employed.

Summary of Main Findings

The present findings show that participants performed better in the loss condition than in the gain condition and worst in the neutral condition. These differences in overall accuracy were largely driven by a higher tendency to adjust responses after errors associated with monetary losses, whereas trial value did not affect correct-stay performance. In the loss condition, errors elicited a larger Ne compared to gain and neutral condition. By contrast, no differences in Ne amplitude were found between gain and neutral condition. Importantly, the incentive-related Ne modulation was not a mere consequence of differences in overall performance and hence improved error detection (cf. Yeung, 2004), as enhanced loss-related Ne amplitudes were also found for a subsample of participants who performed equally well in gain and loss condition. Notably, inter-individual differences in Ne amplitude on loss trials correlated with incorrect-switch performance, suggesting a functional link between the loss-related Ne enhancement and aversively motivated behavioural adjustments. In contrast to previous fMRI findings, source localization revealed greater error-related dACC reactivity on loss trials but greater rACC reactivity on neutral trials. While phasical dACC activation scaled to the size of the Ne in each of the three incentive conditions, greater phasical rACC activation predicted larger Ne amplitudes and improved

error-related behavioral adjustments in the gain and neutral condition only. Rather than a functional distinction between “cognitive” vs. “affective” aspects of error processing, these findings point to a more complex interplay among the cingulate subdivisions in mediating adaptive behaviour control.

Learning-related Changes in Ne, FRN, and Pe

Learning-related changes in the Ne. Importantly, the present study demonstrated that the Ne increased with learning. This finding is line with previous research indicating that learning-related modulations in the Ne can be observed when learning blocks are relatively short and hence include a small number or trials (e.g., Eppinger & Kray, 2011). Hence, in simple two-choice decisions, the Ne appears to grow larger during early stages of learning only. By contrast, the learning-related increase in the correct response-related positivity was much less pronounced than in Experiment 1 and 2. Together, these findings corroborate the notion that the two components reflect dissociable processes, possibly both related to behavioural adaptation but operating at more rapid vs. slower time-scales (Eppinger et al., 2008, 2009).

However, an alternative view on the putative positivity on correct trials has been put forward as well. Ridderinkhof and cowokers (2003) observed that error-*preceding* correct trials are characterized by faster responses and an increased positivity in the time range of the Ne. The authors concluded that these phenomena indicate deficient performance monitoring processes, increasing the error probability on the next trial. Using CSD transformation, Allain and colleagues (2004) showed that the error-preceding positivity actually reflects an attenuation of the CRN, which they assumed is functionally similar to the Ne. Indeed, fMRI studies showed an error-preceding activation increase in default mode regions of the brain and a concurrent decrease of activation in brain regions supporting effortful task-related processing, including pMFC and OFC (Eichele et al., 2008). In the current study, similar to the findings by Allain et al. (2004), response-locked current source density (CSD) ERPs revealed a clear Ne-like wave on correct trials. This negativity decreased with learning in the gain and neutral condition. Thus, the learning-related increase

in the correct response-related positivity in these conditions might also reflect the reduced engagement of effortful monitoring processes, possibly related to a decrease in task demands. By contrast, in the loss condition, the amplitude of the Ne-like wave did not change across bins, but appeared generally somewhat smaller than in gain and neutral condition. In sum, the current findings indicate that it remains a challenging task for future research to disentangle the different influences contributing to learning-related changes in the response-locked ERPs.

The FRN does not change with learning. Unexpectedly, the changes in the Ne over the course of learning were not accompanied by modulations in the FRN. This finding is in sharp contrast with the predictions of the R-L theory (Holroyd & Coles, 2002). Moreover, the present results are inconsistent with previous studies that showed pronounced changes in the FRN to positive feedback as learning progressed (Cohen et al., 2007; Eppinger et al., 2008, 2009). Notably, the lack of learning-related modulations of the FRN in the present study did not appear to reflect influences of reward or punishment motivation on feedback processing, as it was also evident in the neutral condition. One might argue that the learning blocks were too short to obtain reliable effects of learning on the FRN. However, at least two factors render this unlikely. First, there was a marked increase in Ne amplitude in the present experiment. Second, in the Eppinger et al.'s studies (2008, 2009) the most pronounced reduction in the ERPs to positive feedback occurred very early in learning, i.e., from first to second bin. Still, visual inspection of the feedback-locked ERPs in the study of Eppinger and colleagues suggests that the feedback-evoked P300 decreased with learning for positive and, albeit less clearly, for negative feedback. By contrast, in the present study, the P300 decreased with learning only for positive but not for negative feedback. Hence, differential effects in the feedback-evoked P300 might account for the disparate findings. In sum, however, the current data are difficult to reconcile with the literature.

Learning-related changes in the Pe. Consistent with the results of Experiment 1 and 2, the Pe became larger as participants were better able to represent the correctness of their responses. As opposed to the first study, however, the current analyses revealed an increasing positivity for erroneous responses only. This finding parallels the less pronounced

increase in the correct response-related positivity and might indicate a stronger confounding effect of the stimulus-evoked P300 on the response-locked ERPs in Experiment 1 and 2.

Effects of Appetitive and Aversive Motivation on Error Processing and Learning

Monetary incentives improve learning performance. In line with previous findings (Hübner & Schlösser, 2010; Pessoa & Engelmann, 2010; Savine et al., 2010), the accuracy data showed that monetary incentives in gain and loss condition enhanced task performance. Furthermore, mean accuracy rates were higher in the loss condition than in the gain condition, indicating that participants were more strongly motivated to avoid losses than to obtain rewards. This is consistent with the often observed phenomenon that losses have a higher impact on decision making than do gains of equivalent magnitude, commonly referred to as 'loss aversion', (e.g., Ariely, Huber, & Wertenbroch, 2005). Moreover, participants made use of the incentive cues from very early stages of learning, suggesting that higher trial value promoted the mobilization of processing resources, most notably attentional effort (Sarter, Gehring, & Kozak, 2006), thereby enabling a faster establishment of adequate S-R-mappings.

In support of this notion, gain and loss cue elicited larger P300 amplitudes than neutral cue, which is consistent with other reports showing that this ERP component is sensitive to the stimulus-inherent motivational or emotional significance (e.g., Keil et al., 2003; Yeung & Sanfey, 2004; Nieuwenhuis et al., 2005). Given that the P300 has been linked to the encoding and updating of task context (Donchin & Coles, 1988), the amplitude modulation is likely to reflect a more convenient representation of the current task-specific environment on gain and loss trials compared to neutral trials. Alternatively, it has been proposed that the neural processes underlying the P300 optimize goal-directed information processing by amplifying the neural response to motivationally significant events (Nieuwenhuis, Aston-Jones, & Cohen, 2005). However, this increase in selective attention is thought to facilitate encoding and maintenance of the *eliciting* stimulus, i.e., the incentive cue. Therefore, in order to promote learning, a larger P300 should also be elicited by the

imperative stimulus. Yet, no differences between the incentive conditions were found for the target-locked P300[31].

In addition, the beneficial effects of gain and loss cues on learning performance might reflect more deliberative, strategic allocation of processing resources, such as the preferential use of rehearsal strategies for motivationally more significant items. Although the CNV is thought to reflect voluntary mobilization of resources for task-relevant processes (Falkenstein et al., 2003), no amplitude differences were found between the incentive conditions. Still, it should be noted that the study by Falkenstein and coworkers (2003) used a speeded-response task that did not require participants to learn the mapping rules. Thus, in their study, the incentive-related CNV increase was likely to reflect effortful stimulus, cognitive, and motor preparatory processes in the service of fast and accurate responding. By contrast, in the current learning task, incentive cues might have promoted mnemonic processes more strongly[32]. Indeed, it appears from visual inspection of the learning curves that accuracy differences between the loss conditions were most pronounced at the beginning of learning. This would be consistent with the view that the impact of incentives on task performance decreased with a reduction of working memory demands. However, the effect was statistically not reliable, possibly due to the fact that incentive-related effects were generally small compared to the more pronounced learning-related changes in accuracy.

Aversive motivation increases the behavioural impact internal error cues. Notably, a more fine-grained analysis of accuracy data in terms of trial-to-trial behavioural changes revealed influences of incentive condition on incorrect-switch performance only, whereas correct-stay performance was not affected by incentive value. Participants were most likely to change their responses after errors that resulted in monetary losses. No differences in error-related behavioural adjustments were found between gain and neutral condition. Crucially, the effects of incentive value on trial-to-trial behavioural adjustments were mirrored by

[31] Of note, the lack of differences in the target-locked P300 suggests that the differences in Ne amplitude were unlikely to reflect unspecific effects of arousal or orienting.

[32] However, when participants were explicitly asked for their experiences with the learning task during debriefing, they did not report the purposeful use of those strategies. In fact, most of them indicated that they did not pay much attention due the incentive cues but rather focused on learning the mapping rules.

modulations in Ne amplitude. Consistent with previous research (Gehring et al., 1993; Hajcak et al., 2005; Potts, 2011), the Ne was enhanced for motivationally and affectively more salient errors. Crucially, the Ne was sensitive to punishment but not to reward omission, reflected in larger Ne amplitudes on loss trials compared to gain and neutral trials, whereas the Ne did not differ between gain and neutral condition. This pattern of findings is in line with a number of recent reports (Holmes & Pizzagalli; Potts, 2011; Wiswede et al., 2009a).

Similar to the present study, Potts (2011) found larger Ne amplitudes for errors resulting in monetary losses compared to those resulting in reward omission during a flankers task. Thus, by avoiding potential confounding effects inherent in a block design and assuring sufficient motivational/affective relevance of the incentive manipulation, the present study was able to demonstrate robust effects of punishment motivation on error processing and error-related behavioural adjustments. In particular, the current data fit the results of Experiment 1 and 2 and provide strong evidence for the view that appetitive and aversive motivation differentially bias performance monitoring. One of the most striking findings of the present study, however, was the close resemblance of the experimental effects on Ne and incorrect-switch performance. This further substantiates the notion of a specific functional link between the neural processes underlying Ne generation and subsequent strategic behavioural adjustments (Cavanagh et al., 2009; Debener et al. 2005; Frank et al., 2005, Holroyd & Coles, 2002).

Extending these findings, the present data show that aversive motivation specifically enhances the relationship between the neural processes underlying the Ne and behavioural adaptation. This was indicated by a significant correlation between Ne amplitude and incorrect-switch performance in the loss condition. By contrast, no significant relation between Ne and error-related behavioural adjustments was seen in the neutral condition. Interestingly, the Ne also predicted incorrect-switch performance during later stages of learning in the gain condition. On the one hand, these results are consistent previous research that closely links ACC functioning to aversively motivated behaviour control (Frank et al., 2005; Hajcak & Foti, 2008; Shackman et al., 2011; Tops & Boksem, 2010). On the other hand, they support the idea that motivational/affective influences on cognitive performance are not restricted to an unspecific “energizing” function, but are associated with

a sharpening of task-related neural processing (Pessoa & Engelmann, 2010, Savine & Braver, 2010). On this view, the correlation between Ne amplitude and incorrect-switch performance during later stages of learning in the gain condition might indicate that motivational impact of rewards evolves more slowly, possibly not until a certain performance level in the loss condition has been established. However, the Ne is thought to reflect reactive, evaluative control. Hence, the current findings do not preclude the possibility that appetitive motivation may trigger other forms of neural enhancement or optimization. For instance, some researchers proposed an explicit link between reward motivation and proactive control (Braver et al., 2007; Harmon-Jones et al., 2006; Savine et al., 2010). In the present study, the analysis of cue-related ERPs did not appear to provide much support for a reward-related increase in proactive control. Yet, this might reflect insensitivity of the measurements to such processes[33].

In contrast to aversive motivation, reward motivation did not result in an increase of the Ne. This is consistent with a study by Holmes and Pizzagalli (2010) that also failed to obtain reliable Ne differences between reward and non-reward trials in a Stroop task. Moreover, the finding fits demonstrations that short-term induction of negative but not positive affect on a trial-by-trial level is associated with an increase in Ne amplitude (Wiswede et al., 2009a). Yet, van Wouwe and coworkers (2011) found that the induction of positive affect by presenting short movie clips led to a subsequent reduction of the Ne compared to the induction of neutral affect. A similar effect has been reported for embodied positive affect (Wiswede, Münte, Krämer, & Rüsseler, 2009c).

The discrepant findings might reflect differences in the nature of the specific affective manipulations. In the latter two studies, the reduction in Ne amplitude was attributed to positive affect-related increases of tonic DA in the striatum, which is thought to effectively reduce the error-related phasic dips in dopaminergic activity and hence to attenuate the Ne (Frank et al., 2004; Frank & O'Reilly, 2006). By contrast, the successive presentation of motivationally and affectively salient cues is assumed to result in phasic changes of DA (Schultz, 2002; 2006; 2007). These cue-related phasic increases in dopaminergic activity are

[33] Indeed, Harmon-Jones and cowokers (2006) demonstrated that high levels of approach motivation were

likely to exert modulatory effects on prefrontal working memory function such as enhanced maintenance and stabilization of task-relevant information during subsequent processing of the imperative stimulus (Cools & D'Esposito, 2011; Durstewitz & Seamans, 2002). Importantly, the beneficial effects of increased prefrontal DA have been shown to improve error processing as reflected in the Ne (De Bruijn et al., 2004; Tieges, Ridderinkhof, Snel, & Kok, 2004; van Wouwe et al., 2011). Hence, the different susceptibility of the Ne to positive affect-related manipulations might be due to distinct characteristics of dopaminergic activity in the striatum and the PFC.

In any case, the lack of differences in Ne magnitude between gain and neutral condition is noteworthy since theoretical accounts that relate the Ne to the significance of an error would predict a larger amplitude on gain trials (Luu et al., 2003; Olvet & Hajcak, 2008). The observed incentive-related modulations of the Ne are also not readily accommodated by a conceptualization in terms of a "pure" reward prediction error (Holroyd & Holes, 2002) or increased conflict (Botvinick et al., 2001). According to TD RL algorithms, the magnitude of the PE should be the same for (1) a zero outcome when a gain has been expected and (2) a loss when a zero outcome has been expected (e.g. Schultz, 2002). Thus, while a dopaminergic reward prediction error signal might constitute one determinant of the Ne, it is conceivable that interactions between ACC and other neural systems involved in the representation of specific reinforcer value (e.g., the amygdala and the OFC; Frank & Claus, 2006; Pessoa, 2008; Tremblay & Schultz, 1999, 2000) are reflected in the measured scalp potential as well. The conflict monitoring theory also cannot fully account for the present findings, largely for the same reasons as in Experiment 1 and 2. According to the conflict monitoring theory the larger Ne in the loss condition reflects increased post-error conflict due to enhanced processing of the target stimulus after the production of the erroneous response. However, the analyses of performance-matches subgroups showed that the putative strengthening of attentional focus in the loss condition is not necessarily accompanied by better performance. Instead the present findings suggest that aversive

primarily associated with increased relative frontal activity asymmetry (RFA).

motivation led to heightened vigilance to internal performance cues, possibly resulting in an "amplification" of the conflict signal but not higher conflict *per se*.

Interestingly, the incentive-related Ne modulation did not change as a function of learning, that is, errors in the loss condition elicited a larger Ne, irrespective of whether they occurred during earlier or later stages of learning. Thus, even in case of uncertain responses, error processing differed considerably between loss trials on the one hand and gain and neutral trials on the other. By contrast, in Experiment 1 and 2, the effect of failure on the Ne was more pronounced during later phases of learning, that is, as participants were better able to represent the correctness of their responses. However, it is difficult to directly compare these experiments with the current study. First, in Experiment 1 and 2, the learning task involved a greater number of stimulus repetitions. Therefore, the two bins represented other phases of learning than in Experiment 3. Indeed, the Ne did not change across pretest in Experiment 1 and 2, whereas a pronounced learning-related increase was observed in the present study. Hence, it is possible that the amplitude differences would have become larger with prolonged learning in Experiment 3. Second, the results of Experiment 1 and 2 are based on a comparison between two different test phases (pre- and posttest), whereas incentive value was manipulated within the same learning phase in the current study.

However, the current finding of an early reactivity of the medial prefrontal performance monitoring system to punishment cues seems consistent with other research that showed enhanced ACC activation in conjunction with pain avoidance when response uncertainty was high (Diener, Kuehner, & Flor, 2010). Similarly, unpredictable threat has been found to evoke stronger activity in the ACC than predictable threat (Alvarez, Cen, Bodurka, Kaplan, & Grillon, 2011). Thus, the present data are in line with the notion that a core function of the ACC is to enable appropriate behaviour in the face of threat and uncertainty (Shackman et al., 2011).

In sum, the data discussed in this section showed that aversive but not appetitive motivation is associated with a heightened responsivity to internal error cues and more efficient error-related behavioural adjustments. The findings further corroborate the notion of a close link between performance monitoring – as reflected in the Ne – and aversively motivated instrumental control. Importantly, the accuracy data as well as the cue-locked

ERPs showed that the sensitivity of the Ne to losses was unlikely to reflect unspecific motivational factors such as higher effort or arousal. Instead, the pattern of results appeared to indicate a highly specific processing bias towards the avoidance of losses, i.e., the most aversive response outcomes.

Monetary incentives do not clearly affect the FRN. As was the case for the failure manipulation in Experiment 1 and 2, trial-by-trial manipulations of appetitive and aversive motivation did not appear to substantially affect feedback processing as reflected in the FRN. The analysis only revealed that the FRN was larger in the neutral condition compared to gain and loss condition, for both positive and negative feedback. Thus, the present data are in line with previous studies indicating that the processes underlying the FRN encode outcome valence but not magnitude and operate in a context-dependent fashion (Hajcak et al., 2006; Holroyd et al., 2004; Yeung & Sanfey, 2004). Nevertheless, more recent findings suggested that the FRN might also be sensitive to the magnitude of rewards (Goyer et al., 2008; Kreussel et al., 2011). However, in these studies, the feedback stimulus itself provided information about the magnitude of wins or losses, whereas in the present study the feedback indicated only the correctness of the response. Therefore, the “magnitude-aspect” was much less salient. Importantly, it has been shown that the FRN appears to be sensitive to the most salient information the feedback stimulus provides (Nieuwenhuis et al., 2004). Consistent with this notion, only feedback type (correct vs. incorrect) affected the FRN in the present study.

A related but somewhat different explanation for the lack of specific incentive-related modulations in FRN amplitude concerns the participants’ ability to maintain the cue-related information in working memory. Note that participants had to relate the feedback information about the correctness of their response to the representation of item-specific incentive value. This was a quite complex operation that presumably took longer time than the rapid evaluation of feedback information as reflected in the FRN. One might predict that individual differences in working memory capacity would play a decisive role in determining the ability to perform these computations. However, no evidence for such a modulatory influence of working memory capacity (assessed by a modified version of the Digit Ordering Test; Cooper

et al., 1991) was found in the present study. Nonetheless, a closer inspection of individual ERP waveforms revealed a remarkable variability in the FRN effects, suggesting that other variables moderated the effects of appetitive vs. aversive motivation on feedback processing. Given that the FRN has been suggested to reflect the value of an outcome relative to the specific task goal (Holroyd et al., 2006), interindividual differences in the definition of this task goal could account for some variance. While some participants may have primarily focused on the information the feedback provided for learning, others may have focused more strongly on wins and losses. In the latter case, the FRN would be expected to differentiate less clearly between positive feedback in the loss condition and negative feedback in the gain condition.

Though incentive cues did not differentially affect the processing of positive vs. negative feedback, the FRN was generally larger in the neutral condition compared to gain and loss condition. A similar pattern of more negative amplitudes to both positive and negative feedback has been found in highly punishment sensitive individuals (Balconi & Crivelli, 2010) as well as in children compared to young adults (Hämmerer, Li, Müller, & Lindenberger, 2011) and has been proposed to indicate heightened sensitivity to feedback. On this view, the present finding would reflect reduced vigilance to feedback in the motivationally more significant gain and loss condition, which seems counterintuitive. In addition, previous research established a link between a more negative FRN on correct feedback trials and *diminished* processing of positive feedback (Cohen et al., 2007; Eppinger et al., 2008, 2009). Given that the neutral condition was motivationally less salient, it would make sense to assume that participants were less vigilant to positive feedback. However, the same logic would imply that participants were more sensitive to negative feedback in the neutral condition. Hence, the present finding is difficult to integrate with previous data and warrants further investigation.

Taken together, the current results are in line with Experiment 1 and 2 as well as other studies reporting dissociations between FRN and Ne (Eppinger et al., 2008; 2009; Gründler et al., 2009; Hajcak et al., 2003) and provide a further challenge to the assumptions of the R-L theory (Holroyd & Coles, 2002).

Monetary incentives do not clearly affect the Pe. Similar to the FRN, the Pe was largely unaffected by the incentive manipulation. Errors that were more costly in terms of monetary loss or failure to gain did not elicit a larger Pe compared to errors without those consequences. As was discussed with respect to the lack of failure-related effects in the first study, this finding is hard to reconcile with an 'affective-processing' hypothesis (Falkenstein et al., 2000). At the first glance, this finding seems also inconsistent with notion that the Pe reflects the motivational significance of an error and is functionally related to the stimulus-evoked P300 (Overbeek et al., 2005). Yet, it should be mentioned that the analysis revealed a larger positivity after correct and incorrect responses in the loss condition compared to gain and neutral condition. Thus, one might argue that both correct and incorrect responses were more salient on loss trials. Overbeek and colleagues (2005) proposed that Pe might reflect the activity of a more slowly operating deliberate performance evaluation system. Analogous to what has been suggested for the medial prefrontal performance monitoring system and the CRN on correct trials (Allain et al., 2004; Ridderinkhof et al., 2003), the enhanced positivity in the time range of the Pe might indicate a stronger recruitment of this putative parallel system in the loss condition. Interestingly, this would also imply that the two parallel systems are differentially engaged by correct and erroneous responses as only the Ne but not the correct response-related positivity was affected by incentive value.

The Contribution of Cingulate Subregions to Error Processing

A specific aim of the present study was to determine whether larger Ne amplitudes for motivationally and affectively more salient errors reflect a differential involvement of dACC and rACC in error processing and the initiation of subsequent behavioural adjustments. It has been suggested that the rACC evaluates the affective and motivational significance of an error, thereby directly accounting for variations in the scalp-recorded Ne (Luu et al., 2003; van Veen & Carter, 2002). Although this assumption has received some support from fMRI studies (e.g., Taylor et al., 2006, Simões-Franklin et al., 2010), it has not explicitly been tested utilizing the high temporal resolution of ERPs.

Notably, the present data speak against a direct contribution of the rACC to the loss-related Ne enhancement. Only in the neutral condition, the rACC was more active following errors than correct responses, whereas in the loss condition, errors were followed by greater phasical activity in parts of the dACC and the neighboring SMA and pre-SMA. On gain trials, no significant activation differences were obtained between correct and incorrect responses for either of the cingulate subregions. Thus, the Ne enhancement on loss trials appears to reflect a stronger recruitment the dorsal rather than rostral ACC. Further corroborating this notion, only dACC power but not rACC power predicted the magnitude of the Ne in the loss condition. Yet, there was no reliable correlation between error-related dACC activation and incorrect-switch performance. This was somewhat surprising, given the relationship between Ne and error-related behavioural adjustments in the loss condition. Interestingly, when dACC activation on gain trials was included as an additional regressor, the predictive power of dACC activation on loss trials was substantially increased. Thus, it appeared that dACC power in the gain condition suppressed irrelevant variance of dACC power in the loss condition. Given that this effect was not observed for error-related dACC power in the neutral condition, it might be related to non-specific motivational influences on dACC reactivity in gain and loss condition that are not directly reflected in Ne amplitude. In any case, the lack of a direct relationship between error-related phasic dACC activity and incorrect-switch performance in the loss condition suggests that a more complex action-regulation circuitry underlies Ne generation.

By contrast, on gain and neutral trials, both error-related dACC and rACC reactivity were related to Ne amplitude, a finding that has also been reported by studies combining ERP and fMRI measures (e.g., Mathalon, Whitfield, & Ford, 2003). Furthermore, rACC activation predicted incorrect-switch performance in these two conditions. This finding suggests two important conclusions. First, it indicates that not only the dorsal but also the rostral ACC is critically involved in implementing adaptive behaviour control. This view is consistent with previous studies demonstrating increased coupling between rACC and lateral PFC after errors or high conflict (e.g., Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Holmes & Pizzagalli, 2008). Second, the fact that the relationship between error-related rACC activation and incorrect-switch performance was observed in the gain and neutral condition

but not in the loss condition suggests that it was not driven by higher affective or motivational salience. In particular, these results contrast with previous fMRI studies that report increased rACC activation in response to more costly errors (Simões-Franklin et al., 2010; Taylor et al., 2006). Given the comparatively low temporal resolution of the BOLD signal, however, it cannot be ruled out that the activation differences in these investigations did not reflect neural mechanisms underlying the Ne but resulted from later phases in error processing (e.g., conscious appraisal of an error) or even feedback processing. Furthermore, in the current study, participants performed a reinforcement learning task, whereas the above mentioned fMRI studies employed a response competition task (Taylor et al., 2006) and a Go-NoGo task (Simões-Franklin et al., 2010). Given that the nature of task has been shown to play a pivotal role in determining the differential recruitment of cortical systems within the mPFC (Cavanagh et al., 2010b), varying task demands might account to some extent for the divergent pattern of findings.

Taken together, the current findings indicate that the extent to which the Ne amplitude reflects the engagement of dorsal and rostral ACC differs as a function of task context. However, rather than reflecting the “cognitive” vs. “affective” component of error processing, the two cingulate subdivisions may subserve complementary functions in the adaptive regulation of cognition and emotion (Mohanty et al., 2007; Pizzagalli, 2011). In particular, the present data are consistent with the recent suggestion that dorsal parts of ACC and mPFC might also support affective appraisal processes (Etkin, Egner, & Kalisch, 2011) and that the rACC mediates behavioural adaptation (e.g., Etkin et a., 2006; Holmes & Pizzagalli, 2008; Mohanty et al., 2007). Although the rapid affective appraisal of on-going performance certainly plays a key role in error processing and might also be highly relevant for learning (Phelps & Le Doux, 2005; Sander, Grafman, & Zalla, 2003), the present findings do not support the notion that the Ne directly indicates an affective or motivational monitoring function of the ACC. Instead, they emphasize the ACC’s sensitivity to discrepancies between on-going behaviour and current task goals and its involvement in implementing adaptive behavioural change.

10. General Discussion

The aim of the present thesis was to investigate the impact of the motivational and affective context of an action on the neural mechanisms of error and feedback processing during reinforcement learning. This question was addressed in three experiments by utilizing the high temporal resolution of event-related potentials (ERPs). In particular, this thesis focused on three ERP-components thought to reflect neural signals that are used to guide goal-directed behavioural adjustments: the error negativity (Ne), the feedback-related negativity (FRN), and the error positivity (Pe). Experiment 1 and 2 examined how self-relevant failure performance monitoring processes – indexed by Ne, FRN, and Pe – and the ability to use error signals for behavioural adaptation in a subsequent feedback-based learning task. To this end, two phases (pre- and posttest) of a probabilistic learning task were applied. Between pre- and posttest, participants performed a visual search task described as diagnostic of intellectual abilities. In this task, participants were assigned to one of two conditions in which they received either failure feedback (failure-feedback-group) or no feedback (no-feedback-group). In order to disentangle the effects of failure exposure and motivational disengagement over the course of posttest, Experiment 1 and 2 used different posttest instructions. In the first experiment, the posttest was described as indicative of intellectual abilities to ensure high self-relevance. By contrast, in the *second experiment*, the posttest was described in neutral terms. Experiment 3 aimed to determine whether manipulations of appetitive vs. aversive motivation by means of monetary incentives modulate performance monitoring and learning-related behavioural adjustments. A further goal was to explore whether dorsal and rostral subregions of the anterior cingulate cortex (ACC) differentially contribute to error processing, depending on the motivational significance of an error.

The following general discussion is divided into three parts. In the first part, I will discuss implications of the present findings on learning-related changes in Ne, FRN, and Pe. The second part focuses on the most central issues of the present research, namely the impact of failure manipulation and appetitive vs. aversive motivation on the mechanisms of performance monitoring and learning. In the third part, I will summarize the limitations if the

present study, followed by the outline of directions for future research and a general conclusion.

Learning-related Changes in the ERP-correlates of Error- and Feedback Processing

A specific aim of this thesis was to replicate and extend findings by Eppinger and colleagues (2008, 2009) indicating that learning does not only affect the processing of errors and negative feedback, but also the processing of correct responses and positive feedback. These and similar findings gave rise to an on-going debate on the question as to whether response- and feedback-locked positivities are primary determinants of experimental variations in Ne and FRN, respectively (Eppinger et al., 2008; Foti et al., 2011; Holroyd et al., 2008). In particular, Eppinger and coworkers (2008, 2009) showed the correct response-related positivity increased as learning progressed, whereas the feedback-locked positivity to positive feedback decreased with learning. In order to integrate these findings with the R-L theory (Holroyd & Coles, 2002), Eppinger and colleagues suggested that the positive potentials may reflect phasic increases in midbrain dopaminergic activity, i.e., positive PEs, that inhibit the ACC. According to this view, phasic increases rather than dips in midbrain dopaminergic activity drive learning in the ACC.

Does the correct response-related positivity reflect a learning signal? Consistent with the findings by Eppinger and colleagues (2008, 2009), the correct response-related positivity increased over the course of learning in each of the present studies. However, this increase was much more pronounced in Experiment 1 and 2 compared to Experiment 3. Interestingly, the opposite pattern was observed for the Ne. Whereas the Ne did not change with learning in Experiment 1 and 2, a pronounced learning-related enhancement in Ne amplitude was found in Experiment 3. Given that the learning blocks included much more trials in first two experiments compared to the third experiment, these findings indicate that the Ne changes on rapid time-scales at the beginning of learning only, while the correct response-related positivity demonstrates a slower, continuous increase over the course of learning. Thus, the two components are unlikely to reflect neural manifestations of the same dopaminergic PE signal. If this would be the case, continuously increasing reward prediction as reflected in the

correct response-related positivity should be associated with a similarly increasing negative PE indexed by the Ne (e.g., Niv & Montague, 2008; Schultz, 2002). Importantly, the notion that the two components reflect dissociable processes was further corroborated by their different susceptibility to affective/motivational manipulations. Only the Ne – but not the correct response-related positivity – was modulated by the experience of self-relevant failure (Experiment 1 and 2) and the motivational/affective significance of an error in terms of monetary losses (Experiment 3).

Yet, this could simply mean that the Ne does *not* reflect a negative PE. As was also noted by Eppinger et al. (2008, 2009), considerable evidence indicates that the ACC is critically involved not only in monitoring for negative events, but also in reward-motivated behaviour (Amiez et al., 2005; Liu, Hairston, Schrier, & Fan, 2010; Magno, Simões-Franklin, Robertson, & Garavan, 2008; Matsumoto et al., 2007). Indeed, it has been suggested that the ACC's basic function is to signal the need for behavioural change, irrespective of whether things are going better or worse than expected (Magno et al., 2008). Nonetheless, the current data raise some doubt on the assumption that the response-related positivity reflects a dopaminergic learning signal. In Experiment 1 and 2, the correct response-related positivity increased not only in the two learning conditions but also in the chance condition, as well as from pre- to posttest. Critically, the amplitude modulations were not accompanied by performance improvements. Although these changes might have reflected stimulus-evoked P300 activity, they underscore that component overlap constitutes a major obstacle when isolating the learning-related part of the response-locked positivity. In fact, it seems plausible to assume that stimulus-locked components could be responsible for the effects in the response-locked ERPs. For instance, a gradually increasing positivity over midline central sites has been demonstrated for successive stimulus presentations in an extended continuous recognition memory task (e.g., van Strien, Hagenbeek, Stam, Rombouts, & Barkhof, 2005). This repetition effect has been suggested to reflect memory strength. Similar

effects have been attributed to increasing decision confidence (e.g., Finnigan, Humphreys, Dennis, & Geffen, 2002)[34].

Several analysis techniques have been developed to deal with the problem of component overlap. One of these methods is known a current source density (CSD) transformation (Kayser & Tenke, 2006). CSD acts as a spatial filter that amplifies the representation of local neuroelectric activity and attenuates broadly distributed and/or distal activities. Applying this technique on response-locked ERPs in Experiment 3 revealed a Ne-like wave on correct trials that decreased with learning in the gain and neutral condition, whereas in the loss condition, this negativity was already diminished in the first bin. Even more pronounced learning-related decreases in the negativity on correct trials were observed when transforming the ERPs in Experiment 1 and 2. This finding is consistent with the proposal that a decreasing negativity contributes to the learning-related dynamics in the ERPs to correct responses (Allain et al., 204; Ridderinkhof et al., 2003). In the context of the present study, this effect could reflect the reduced need to engage effortful monitoring processes as the representations of the mapping rules become increasingly robust. Consistent with this view, it has been shown that the ACC is more strongly involved in early stages of learning, whereas other regions such as the posterior cingulate contribute to action selection during later stages of learning (Gabriel, Burhans, Talk, & Scalf, 2002). Moreover, the findings by Pietschmann et al. (2008) discussed earlier bear close resemblance to the CSD-transformed data of Experiment 3 and support the idea that higher learning demands require a stronger engagement of the medial prefrontal performance monitoring system.

[34] Another potentially relevant factor directly concerns the impact of response latencies on ERP activity. As Makeig and Onton (2011) pointed out, the P300 is time-locked to the response rather than the stimulus. Crucially, the authors showed that a more distinct (i.e., less temporally "smeared") P300 follows the response by about 100 ms and that this positivity is much stronger with short RTs. A closer inspection of response latencies in Experiment 1 and 2 revealed that the amplitude of the correct response-related positivity was significantly correlated with RT in each of the three learning conditions (r-values $>-.31$, p-values $< .08$). By contrast, no significant correlation was found between Ne amplitude and RT (r-values $<-.22$, p-values $> .22$). Although Eppinger and colleagues (2008, 2009) did not report RT data, it is conceivable that response latencies also decreased as a function of learning in their studies and hence might have contributed to the pronounced response-locked positivity in the deterministic and probabilistic learning condition. Clearly, this issue warrants further investigation.

To summarize, the present data strongly suggest that dissociable neural processes underlie the Ne and the response-locked positivity on correct trials. However, the current studies failed to substantiate the view that the correct response-related positivity reflects a reward-related learning signal. Future studies should combine sophisticated analysis techniques such as principal component analysis (PCA) or independent component analysis (ICA) with computational modelling to provide stronger tests of this proposal.

The FRN does not change with learning. In contrast to the ERP-correlates of internal error processing, the FRN did not vary with learning in any of the three experiments included in this thesis. This is consistent with the findings by Eppinger and colleagues (2008, 2009) for negative feedback trials. Yet, the current studies also failed to obtain learning-related modulations in the ERPs to positive feedback. This was somewhat surprising, given that the learning paradigm in Experiment 1 and 2 was very similar to that used by Eppinger and colleagues (2008, 2009). One explanation of this discrepancy is that the learning bins in Experiment 1 and 2 contained a larger number of trials and might have been insensitive to modulations in the FRN at the beginning of learning. However, the lack of learning-related changes in the FRN in Experiment 3 questions this assumption. Instead, similar to the response-locked ERPs, a direct comparison of the three experiments points to a potentially confounding effect of the stimulus-evoked P300. In Experiment 1 and 2, the ERPs to both positive and negative feedback became less positive as learning progressed, presumably reflecting a reduction of the stimulus-evoked P300 (Walsh & Anderson, 2011; Yeung, Holroyd, & Cohen, 2005; Yeung & Sanfey, 2004). This effect was most pronounced in the deterministic learning condition, supporting the notion that participants paid less attention to the feedback stimulus after they have developed an internal representation of the correct response (see Figures 16 and 22). In Experiment 3, the amplitude reduction was less prominent and limited to positive feedback trials (see Figure 32). Thus, the stimulus-evoked P300 appears to show a different time-course of changes over the course of learning for positive and negative feedback that may have contributed to the learning-related decrease in 'feedback-related positivity' observed in former studies (Cohen et al., 2007; Eppinger et al., 2008, 2009).

However, a number of studies have supported the notion of a reward-related positivity in the feedback-locked ERP (Foti et al., 2011; Potts et al., 2006a). For instance, Foti and colleagues (2011) applied principal component analysis to dissociate a reward-specific positivity from the P300. As for the response-locked ERPs it might be a promising avenue for future research to test whether this positive component changes with learning and other experimental manipulations in a way consistent with a positive PE signal.

Importantly, the current data contradict some of the core predictions of the R-L theory (Holroyd & Coles, 2002) by showing that the FRN does not change with learning, whereas the Ne increases during early stages of learning only. These findings add to a growing number of previous studies indicating that Ne and FRN reflect dissociable neural processes (e.g., Eppinger et al., 2008; Gründler et al., 2009; Hajcak et al., 2003; Pietschmann et al., 2008). Further research is needed to explore more recent ideas according to which reward-related learning signals are the primary determinant of learning-related variations in the two ERP components.

Affective and Motivational Influences on Performance Monitoring and Learning

Of most interest for the present thesis were the effects of the failure manipulation (Experiment 1 and 2) and incentive manipulation (Experiment 3) on the neural mechanisms of error and feedback processing and the ability to use internal and external performance cues for behavioural adaptation. Consistent with previous findings (Brunstein & Gollwitzer, 1996; Hübner & Schlösser, 2010; Krawczyk, et al., 2007; Pessoa & Engelmann, 2010; Savine, et al., 2010), the behavioural data indicated that the motivational/affective manipulations led to an enhancement in task performance in the three experiments. Experiment 1 and 2 showed that the experience of self-relevant failure prevented motivational disengagement at posttest. However, the lack of additional performance improvements in the failure-feedback group in Experiment 1 possibly reflected some detrimental effects of the need to cope with the stressful event. In addition, monetary incentives resulted in higher accuracy rates in gain and loss condition of Experiment 3. Importantly, a closer inspection of the behavioural data in terms of trial-to-trial behavioural

adjustments revealed that rather than inducing unspecific motivational effects such as increased effort, the motivational/affective manipulations gave rise to strategic changes in task performance. Both failure feedback (Experiment 1 and 2) and loss anticipation (Experiment 3) were associated with more efficient error-related adjustments, reflected in higher post-error accuracy (i.e., incorrect-switch performance). Notably, no incentive-related differences were found for correct-stay performance in Experiment 3, demonstrating the specificity of the effects. Thus, self-relevant failure and risk of losing money appeared to promote negatively motivated engagement directed towards the avoidance of threat and punishment.

In case of the failure manipulation, this result is in line with a number of previous findings concerning the influence of social-evaluative stress on learning and memory (Cavanagh et al., 2011a; Petzold et al., 2010; Roozendaal et al., Schwabe & Wolf, 2011). Typically, these studies reported stress-induced alterations in learning strategies rather than overall learning performance. Moreover, the observed strategy shift points to a possible explanation for the inconsistent findings regarding the impact of monetary incentives on cognitive performance (Bonner et al., 2000; Camerer & Hogarth, 1999), if one assumes that the manipulation can trigger both adaptive and maladaptive strategic changes. In support of this notion, Dambacher and coworkers (2011) demonstrated that the efficiency of monetary incentives in speeded perceptual decision tasks depends on the emphasis on speed vs. accuracy, with beneficial effects confined to payoff schemes emphasising speed over accuracy. The authors suggested that this might reflect differential contributions of two parallel processing routes to decision making, supporting rapid but less precise vs. more accurate but slow responses (Trimmer et al., 2008). An interesting possibility following from this view is that failure manipulation and loss anticipation could have promoted the recruitment of the “accurate-but-slow” decision making system. The efficiency of this strategic adjustment, however, was presumably limited by the adaptive response deadline that forced participants to respond quickly. Indeed, one reason for the application of the response deadline algorithm was to prevent influences of a speed-accuracy-tradeoff.

Thus, the behavioural findings strongly suggest that the affective/motivational manipulations employed in the present experiments did not merely result in an enhanced or

reduced recruitment of cognitive resources but also in a differential weighting of the involved processing systems, i.e., a redistribution of available resources according to the prevalent behavioural goals.

Failure experiences and loss aversion promote reactive control. Crucially, the Ne findings from each of the three experiments involved in this thesis strongly supported the notion that self-relevant failure and risk of losing money resulted in a strategic shift towards a reactive-defensive orientation to the environment (Braver et al., 2007; Tops et al., 2010). As was shown by a failure-related increase in Ne amplitude in Experiment 1 and 2 as well as a larger Ne in the loss condition in Experiment 3, aversive motivation was associated with a heightened responsitivity to internal cues indicating performance failures. Most strikingly, these differences in the processing of internal error information predicted subsequent behavioural adjustments, i.e., participants were more likely to learn from past mistakes. Reactive behaviour control has been described as adaptive response to the perception of threat and discrepancies between intended/predicted and actual states (Tops et al., 2010). In particular, this mode of control is thought to strengthen the attentional focus on the current situation, to enhance analytic, self-referential processing, and to facilitate rapid behavioural adaptation to environmental demands (Tops et al., 2010). Such a strategic change in behavioural control appears adequate to overcome the experience of uncontrollable failure that challenged participants' self-definition and presumably induced uncertainty regarding the appropriateness of their own actions. Similarly, loss anticipation was likely to establish a threatening environmental context in which heightened vigilance to internal indictors of errors and discrepancies supported rapid behavioural adjustments in order to avoid harm and punishment.

The results of the present studies are in line with previous research indicating that the monitoring processes underlying the Ne reflect interindividual differences in the sensitivity to negative and threatening events (e.g., Boksem et al., 2006a; 2008; Frank et al., 2005; Hajcak, McDonald, & Simons, 2003; 2004; Tops, Boksem, Wester, Lorist, & Meijman, 2006). However, the present data clearly argue against the view that the Ne is a state-independent marker of an endophenotype for internalizing and externalizing psychopathology (Clayson et

al., 2011; Olvet & Hajcak, 2008). Instead, they corroborate the notion that the medial prefrontal performance monitoring system is flexibly recruited depending on how much emphasis the individual places on avoiding negative response outcomes (Boksem et al., 2008; Falkenstein et al., 2000; Gehring et al., 1993; Hajcak et al., 2005; Potts; 2011; Wiswede et al., 2009b). Extending these latter findings, the current study established a direct link between aversively motivated changes in performance monitoring and the ability to use internal error signals for goal-directed behavioural adaptation.

Interestingly, the present experiments did not yield clear evidence for increased sensitivity to external punishment cues in response to the motivational/affective challenges. Only in Experiment 1, failure-related modulations of the FRN suggested a more differentiated processing of positive and negative feedback. Experiment 2 failed to replicate this finding and rather suggested that the failure experience prevented diminished feedback processing due to motivational disengagement. Although the effect was rather subtle and has to be interpreted with caution, it might underline an important difference between the impact of failure feedback and loss aversion. As was already discussed above, the larger amplitude difference between positive and negative feedback could reflect that participants in the failure-feedback group relied more strongly on external feedback, possibly reflecting a higher need to validate the outcomes of internal monitoring processes. On this view, the failure-related enhancement in Ne and FRN would indicate an attempt to restore the integrity of the performance monitoring system that was challenged through uncontrollable failure. By contrast, in Experiment 3, participants' ability to control aversive consequences was not constrained. Thus, there was no reason to "mistrust" the internal judgements of the action evaluation system. In this case, aversive motivation appeared to trigger a heightened vigilance to internal error information only.

The present findings have important implications for theoretical accounts on performance monitoring (Bernstein et al., 2005; Botvinick et al., 2001; Falkenstein et al., 1991; Gehring et al., 1993; Holroyd & Coles, 2002). In particular, the lack of learning-related changes in FRN amplitude and the different susceptibility of Ne and FRN to the affective/motivational manipulations question the assumption of the R-L theory (Holroyd & Coles, 2002) that both the Ne and the FRN are neural manifestations of dopaminergic PEs.

Yet, the latter result might be explained within the framework of the R-L theory by assuming that aversively motivated reactive-defensive engagement selectively amplifies the magnitude and/or impact of negative PEs arising from mismatch detection on the basis of internal representations of action value. Nonetheless, the pattern of findings in Experiment 1 and 2 suggested that the ACC is not only a passive recipient of dopaminergic learning signals. Rather, the failure-related modulations in Ne amplitude appeared to reflect altered ACC functioning in conjunction with active behavioural coping and systematically changed cost-benefit analyses in response to the stressful experience (Amat et al., 2005; Pascucci et al., 2007; Boksem & Tops, 2008). This is in line with ample evidence for a top-down biasing, model-based influence of the ACC and other prefrontal structures on neural circuits mediating habitual, model-free control (Daw et al., 2005; Doll et al., 2009; Frank et al., 2005; Huys & Dayan, 2009; Miller & Cohen, 2001). Specifically, it has been suggested that the ACC determines the degree to which the outcome of an action guides learning and future behaviour (Behrens & Rushworth, 2008), fitting the specific link between Ne amplitude and behavioural adjustments observed in the current study.

Thus, the current data are consistent with proposals that the ACC and the surrounding mPFC - the proposed source(s) of the Ne - are critically involved in the processing of mismatch and punishment and the initiation of behavioural change (Blair et al., 2006; Cavanagh et al., 2009; Magno et al., 2008; Shackman et al., 2011; Wrase et al., 2007). Indeed, the detection of mismatch or conflict may be a common functional characteristic of all midfrontal negativities thought to originate from mPFC, including FRN, CRN, and N200 (cf. Cavanagh et al., 2010a; Folstein & van Petten, 2008). The idea that the Ne reflects some kind of a mismatch signal is shared by most of the major theoretical accounts on the Ne. Importantly, the present findings emphasize that theories such as the error/mismatch detection hypothesis (Bernstein et al., 1995; Falkenstein et al., 1991; Gehring et al., 1993) and the conflict monitoring theory (Botvinick et al., 2001) have to incorporate the notion that the mismatch signal can be used to adapt future behaviour in a way that is unlikely to reflect an unspecific increase of attentional effort (Sarter et al., 2006). Instead, the present findings indicate a highly specific processing bias towards the avoidance future failure, possibly reflecting a differential weighting of negative response outcomes in the computation of action

values (Rushworth & Behrens, 2008; Cavanagh et al., 2010a; Frank et al., 2005; Jocham et al., 2009).

In contrast to neurocomputational models of performance monitoring such as the R-L theory and the conflict monitoring theory, affective/motivational proposals (Luu et al., 2003; Olvet & Hajcak, 2008) are less specified, making competitive tests more complicated. However, the present data argue against the notion that the Ne merely reflects an affective response in terms of a subjective emotional experience, such as distress or regret. Given that participants showed better performance in the gain condition compared to the neutral condition in Experiment 3, on would expect the higher motivational and affective significance of reward omission to result in larger Ne amplitudes. This was not observed. In fact, a conceptualization of the Ne in terms of a subjective affective experience appears hard to reconcile with findings suggesting that the occurrence of the Ne does not depend on conscious error recognition (Endrass et al., 2007; Nieuwenhuis et al., 2001). In particular, the present study failed to confirm the assumption that the rACC evaluates the affective and motivational significance of an error, whereas the dACC is involved in more "cognitive" aspects of error processing (Luu et al., 2003; van Veen & Carter, 2002). Importantly, the proposal of Luu and colleagues (2003) largely relied on the identification of a rACC source for the Ne. Yet, the source localization results of Experiment 3 did not reveal greater rACC activation for affectively more significant errors. Instead, the findings suggested that the cingulate subdivisions may subserve complementary functions in the adaptive regulation of cognition and emotion (Mohanty et al., 2007; Pizzagalli, 2011).

Potential mechanisms mediating the effects of failure and loss aversion. Although the present findings highlight the need to explore the performance monitoring system in terms of both cognitive and affective/motivational mechanisms, the precise nature of the processes that mediate the observed effects of failure feedback and loss aversion remains to be determined.

Pessoa and Engelmann (2010) proposed three abstract ways of how motivational/affective influences may interact with task-specific cognitive processes. A first scenario suggests that cognition and motivation/affect are segregated both at a functional

and neuroanatomical level and influence task performance independently through parallel routes. Given the ample evidence against a clear-cut segregation of affective and cognitive processes, it may prove difficult to provide evidence for this notion (Pessoa 2008; Pessoa & Engelmann, 2010; Salzman & Fusi, 2010). Nonetheless, some studies suggested that under certain conditions influences of attention and motivation/affect might be dissociable (Bendiksby & Platt, 2006; Vuilleumier, Armony, Driver, & Dolan, 2001). Hence, it remains a challenge for future research to test whether “non-cognitive” mechanisms affect performance monitoring and learning.

Second, it was proposed that the effects of motivational/affective variables are mediated by cognitive processes. On this view, self-relevant failure and loss aversion would have led to an increase in attentional effort, resulting in more efficient target processing (Clayson et al., 2011; Sarter et al., 2006; Yeung, 2004). As outlined above, there were several reasons to suggest that the present findings did not exclusively reflect higher effort or focused attention. Most importantly, the observed effects pointed to aversively motivated strategic changes in task performance rather than an unspecific performance improvement. By contrast, appetitive motivation was not associated with altered functioning of the medial prefrontal performance monitoring system as reflected in Ne and FRN, emphasising the specificity of the effects. However, non-specific motivational effects clearly played a role in mediating the effects of failure and incentive manipulation. This was indicated by the findings that (1) self-relevant failure effectively preserved task engagement (Experiment 2) and (2) monetary incentives improved overall accuracy (Experiment 3).

The third proposal on the nature of the relationship between cognition and affect/motivation assumes that they constitute highly integrated processes that are not clearly decomposable (Pessoa & Engelmann, 2010). Indeed, the ACC is considered a central hub in which affectively and motivationally relevant information is linked to motor areas (Bush et al., 2000; Shackman et al., 2011), fitting the notion that this structure was critically involved in mediating the effects of failure and loss aversion on task performance. Consistent with recent suggestions (Shackman et al., 2011), the present work did not yield evidence in support of a segregationist view of ACC functioning, with the dorsal and rostral ACC involved in cognitive and affective aspects of error processing, respectively. Instead, greater error-related dACC

activation was observed in the loss condition, whereas the rACC was more active following errors compared to correct responses in the neutral condition only. However, given the limited number of studies that analysed source activity underlying the Ne, it is difficult to fully interpret the pattern of subregional activation differences.

Further candidate regions for the integration of motivational and affective influences with task-specific processing include the OFC and the amygdala. In support of this notion, Wrase and coworkers (2007) showed that not only ACC activation but also activity in lateral OFC predicted behavioural adaptation following punishing outcomes in a monetary incentive delay task. This is in line with the suggestion that the OFC contributes to rapid learning-related adaptations on a trial-to-trial basis, possibly reflecting the active maintenance of negative reinforcement values in working memory (Frank & Claus, 2006; Frank et al., 2007a). Note, the OFC is functionally and anatomically closely connected to the amygdala (Murray & Izquierdo, 2007) and has been strongly implicated in the representation of the reward value of stimuli (Kringelbach et al., 2003; O'Doherty et al., 2001; Plassmann et al., 2007; Rushworth et al., 2007). Furthermore, both amygdala and OFC are closely connected to both the ventral striatum and the midbrain DA system, which led some researchers to suggest that the two structures are critically involved in the computation of the PE (Maia, 2009). Specifically, the amygdala has been shown to encode a PE during avoidance learning (Prevost et al., 2011; Yacubian et al.; 2006). Together with the current finding that aversive motivation was specifically associated with more efficient trial-to-trial behavioural adjustments following errors, the above evidence suggests that structures like the OFC and the amygdala might have contributed to the observed effects of failure and loss aversion.

Moreover, given that the present effects were specifically linked to aversive motivation, it seems worth noting that the amygdala has been hypothesized regulate the engagement of appetitive and aversive behavioural systems (Pessoa, 2008; Prevost et al., 2011; Salzman & Fusi, 2010). In fact, accumulating evidence suggested that dissociable albeit overlapping neural circuits might be involved in the control of behaviour by appetitive and aversive cues (Gray et al., 2002; Small et al., 2005; Tops et al., 2010; Wrase et al., 2007; Yacubian et al., 2006). The present research suggests a close link between aversive motivation and the activity of the medial prefrontal performance monitoring system as

reflected in the Ne. However, there was no evidence for influences of the motivational/affective manipulations on the response- and feedback-locked positivities on correct trials. Future studies may probe the relationship between these putatively reward-related components and appetitive motivation using more sophisticated EEG analysis techniques.

Finally, cognitive and motivational/affective processes might be integrated by the activity of neuromodulator systems (Pessoa, 2008; Pessoa & Engelmann, 2010; Tops et al., 2010). In particular, it has been suggested that affect- and motivation related DA dynamics lead to sharpening of task-specific processing by improving the neuronal signal-to-noise ratio in target structures (Durstewitz & Seamans, 2002; Pessoa & Engelmann, 2010). On this view, gain and loss cues in Experiment 3 might have triggered heightened dopaminergic activity, possibly mediated by valuation regions such as the amygdala and the OFC. Enhanced prefrontal DA levels could in turn support working memory functions of OFC and dlPFC, whereas enhanced striatal DA levels could support flexible updating of task-relevant contents (Cools & D'Esposito, 2011; Frank et al., 2007a). Moreover, DA has been shown to promote rapid memory formation in hippocampus and surrounding MTL regions (Shohamy & Adcock, 2010). Still, one could argue that the DA system responds to reward but not punishment cues (Schultz, 2000, 2006, 2007), making dopaminergic mechanisms unlikely to facilitate task-related processing in the loss condition. However, recent studies demonstrated even greater activation in valuation regions, including VTA and ventral striatum, when participants learned to avoid losses than when they learned to attain gains, supporting the notion that motivational/affective relevance of an event might determine activity in reward-related regions (McKell Carter, MacInnes, Huettel, & Adcock, 2009; Niznikiewicz & Delgado, 2011). In addition, down-regulation of mesolimbic and mesocortical DA transmission is thought to play a central role in mediating the effects of mental fatigue (Chaudhuri & Behan, 2000, 2004; Sarter, et al., 2006). Specifically, the ACC it has been assigned a pivotal role in determining the changes in dopaminergic activity based on cost-benefit analyses (Boksem & Tops, 2008). Thus, dopaminergic mechanisms were likely to contribute to the differential effects of prolonged task performance in the failure-feedback and no-failure-feedback groups in Experiment 1 and 2.

Of note, there is evidence indicating that the activity of the midbrain DA system is strongly affected by modulations in cholinergic activity (Zhou, Wilson, & Dani, 2003). Indeed, a bias towards reactive avoidance has been primarily associated with cholinergic mechanisms (Tops et al., 2010). In particular, acetylcholine has been implicated in the expectancy of uncontrollability and uncertainty, suggesting that cholinergic mechanisms – possibly working in tandem with DA - might have mediated the failure-induced shift towards reactive control.

A parallel error evaluation mechanism underlying the Pe? One of the most robust findings of the present research was that the Pe increased with learning. Thus, in contrast to the Ne and the FRN, the Pe appeared to be sensitive to gradual changes in the strength of response representations. Nonetheless, the Pe was largely unaffected both by the failure induction and the incentive manipulation employed in the present thesis. Moreover, the Pe did not predict learning-related trial-to-trial behavioural adjustments. The current findings thus corroborate the view that distinct aspects of error processing are reflected in Ne and Pe. Thus far, however, a detailed theory on the functional significance of the Pe is lacking. Most notably, Overbeek and coworkers (2005) proposed that the Pe might signify the activity of a slower more deliberate system that evaluates the motivational significance of salient errors and operates in parallel to the rapid preconscious system underlying the Ne (see also Ridderinkhof et al., 2009). Although this evaluative system is also thought to mediate post-error adaptation, these effects might differ from the learning-related behavioural adjustments the present study focused on. First, Ridderinkhof and colleagues (2009) stated that errors have to be sufficiently salient to trigger the adaptation mechanisms associated with the Pe. Thus, it is conceivable that errors in the feedback-based learning task were simply not salient enough. In support of this view, Pe amplitudes were considerably smaller in the current experiments than those observed in other studies using response conflict tasks. Second, the proposed functional similarity between Pe and P300 suggests a relationship to the mobilization of processing resources for immediate error correction rather than strategic changes in post-error behaviour (cf. Overbeek et al, 2005). Nonetheless, the present data are consistent with previous studies that also failed to obtain modulations in Pe amplitude

due to motivational/affective manipulations (Clayson et al., 2011; Wiswede et al., 2009b). In sum, available evidence thus seems somewhat inconsistent with the idea that the Pe is related the evaluation of the motivational significance of an error. Indeed, it has been demonstrated that the Pe might reflect a delayed stimulus-related P300 rather than response monitoring processes per se (Shalgi, Barkan, & Deouell, 2009). As was the case for the correct response-related positivity, the latter finding could also explain why the Pe increased with learning and further highlights the problems arising from component overlap in interpreting response-locked ERPs.

Limitations of the Present Study and Directions for Future Research

Though the present thesis provided important new insights in the neural mechanisms of performance monitoring and learning, some limitations should be addressed that have to be taken into account when interpreting the results. A basic shortcoming inherent to the ERP approach is the need to average across a relatively large number of trials to obtain reliable measures of the ERP components. This results in very long experimental sessions and makes the data susceptible to effects of fatigue. This effect is even more problematic when a pre-post-design is applied - as was clearly evident from the data of the no-failure-feedback group in Experiment 2. However, as was discussed above, the additional motivational manipulation proved extremely helpful in disentangling the effects of the failure manipulation and fatigue in the two experiments.

Moreover, the pre-post design limited the absolute number of trials in pre- and posttest. Therefore, the bins might have been too large to adequately represent learning-related dynamics of Ne and FRN in Experiment 1 and 2. Indeed, the findings of Experiment 3 showed that the Ne exhibited learning-related modulations when the learning blocks included a reduced number of trials. Yet, the FRN did not increase over the course of learning in either experiment, indicating that methodological problems were not the primary reason for the lack of findings. In addition, it should be noted that the core findings of the present study did not critically depend on the modulation of Ne and FRN over the course of learning. In

fact, the findings of Experiment 1 and 2 mainly reflected pre-post amplitude differences. Similarly, in Experiment 3, no interaction of incentive condition and learning was obtained.

Another potentially critical point was the use of a between-subjects-design in Experiment 1 and 2. Therefore, one cannot definitely preclude the possibility that the present findings were attributable to pre-experimentally existent group differences, particularly in view of the fact that the sample sizes were relatively small. To control for confounding influences of baseline differences in learning performance, participants in the experimental groups were matched for overall accuracy at pretest. Moreover, the experimental groups did not differ with respect to the personality measures. Nonetheless, the analysis of the feedback-locked ERPs in Experiment 2 suggested that some group differences were present already at pretest. It was all the more important that Experiment 2 largely replicated the findings of Experiment 1, thereby considerably increasing the trustworthiness of the experimental effects.

However, the small sample sizes might have been particularly problematic for the analysis of interindividual differences. Though previous studies also included relatively small samples (e.g., Boksem, 2006a; 2008), the present design was complex and involved additional sources of variance such as learning performance that were likely to disguise the effects of trait differences. Thus, an increase in statistical power would be desirable. Nevertheless, it should be mentioned that in the present study the relationship between personality measures and ERP correlates of error and feedback processing was also weak when the pretest data of all participants were collapsed. Yet, one may criticize that the present study used a very homogeneous sample. Indeed, the sample included a high proportion of first-year psychology students. Nonetheless, the descriptive statistics of the covariates for the total sample did not reveal deviations from the norms (means and standard deviations). Therefore, limited variance was unlikely to account for the lack of significant findings.

A further limitation of the present study was the use of between-subject analyses to test for relationships between the ERP components and behavioural adjustments. Although it would be highly desirable to analyse the effects of motivational/affective manipulations, ERP measures and behavioural adjustments in an intra-subject design, the current experiments

did not include a sufficient number of trials to perform those contrasts. Future studies may include multiple sessions to ensure larger numbers of error trials.

Furthermore, the comparatively low spatial resolution (~1-2 cm) of the sLORETA source localization technique potentially limits the conclusions that can be drawn from the analysis of subregional ACC activity in Experiment 3 (Pizzagalli, 2007). It should be noted, however, that the validity of the algorithm has been confirmed by a number of studies using fMRI (e.g., Mulert et al., 2004), PET (Zumsteg, Friedman, Wennberg, & Wiesner, 2005), and intracranial recordings (Zumsteg, Wennberg, Treyer, Buck, & Wiesner, 2006). Specifically, the ACC has been shown to be localized correctly by this method (Pizzagalli et al., 2004).

In addition, the Ne has been characterized as part of an ongoing theta oscillation underlying the joint functioning of a distributed action monitoring network (Luu et al. 2004, Trujillo & Allen, 2007). There is considerable evidence indicating that non-phase-locked spectral perturbations rather than the phase-locked ERP might correspond to the blood-oxygen-level-dependent (BOLD) signal measured by fMRI (Engell, Huettel, & McCarthy, 2011). Hence, the discrepancies between the current localization results and previous fMRI findings regarding the sensitivity of the rACC to the motivational and affective salience of an error may be reflective of non-phase locked dynamics in the activity of the medial prefrontal action monitoring circuitry. Future studies using functional connectivity analyses of fMRI data or combing blind source separation (independent component analysis) and time-frequency analyses of EEG data should be helpful tools in elucidating the functioning of this network.

Finally, it should be noted that the present study did not fit the behavioural data to a computational RL model. On the one hand, the estimation of parameters such as learning rate or PE would allow more stringent tests of theoretical accounts like the R-L theory (Holryod & Coles, 2002). On the other hand, computational modelling is a promising tool to shed light on the specific mechanism of motivational/affective influences on cognitive performance.

Conclusions

Taken together, the present experiments provided the first evidence that modulations in error processing due to motivational/affective manipulations are associated with corresponding changes in the ability to use these error signals for learning-related behavioural adjustments. Consistent with previous research that has established a link between the Ne and the evaluation of the affective and motivational significance of an error (e.g., Gehring et al., 1993; Hajcak et al., 2005; Luu et al., 2003), exposure to uncontrollable failure (Experiment 1 and 2) and risk of losing money (Experiment 3) led to an increase of the Ne during a feedback-based learning task. These findings were extended by the observation that the Ne amplitude enhancement was accompanied by more efficient error-related behavioural adaptation. Crucially, the increase in Ne amplitude was not a mere consequence of better overall performance but was specifically accompanied by higher post-error accuracy. Therefore, the effect is unlikely to reflect an unspecific increase of attention or arousal. Instead, it indicated a shift towards reactive-defensive control, denoting the tendency to recruit control processes when a negative event has already occurred (as opposed to 'proactive control'; Braver, Gray, & Burgess, 2007; Tops, Boksem, Luu, & Tucker, 2010). Failure feedback and loss anticipation thus appear to induce an environmental context in which participants are particularly vigilant to potential threats and negative response outcomes, presumably facilitating rapid behavioural adaptation to emergent demands (Tops et al., 2010). This is line with previous research that has established a relation between the activity of the medial prefrontal action monitoring system and sensitivity to negative stimuli and events (Boksem et al., 2006b; Cavanagh et al., 2011a; Frank et al., 2005; Hajcak & Foti, 2008).

In summary, the present findings offer support for the notion that the Ne constitutes an aversive teaching signal mediating adaptive behavioural changes after maladaptive decisions. Although the engagement of the underlying medial prefrontal system is dependent on the motivational and affective context of an action, the Ne does not appear to directly reflect the affective appraisal of an error. Instead, a threatening context in terms of monetary losses specifically strengthened the relationship between Ne amplitude and error-related

behavioural adjustments. In particular, source localization of the Ne suggested that the extent to which rostral parts of the ACC contribute to Ne generation is not determined by the motivational and affective salience of an error. Rather than reflecting the “cognitive” vs. “affective” component of error processing, dorsal and rostral cingulate subregions might subserve complementary and partially overlapping control functions in the service of adaptive regulation of cognition and emotion. The results of the current investigation are consistent with a more recent view according to which the common denominator of ACC activation in studies of cognitive control, negative affect, and pain is the need to guide action selection in the face of uncertainty and threat (Shackman et al., 2011).

11. References

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12. Appendix

RT Data Analyses

Experiment 1

RT data (see Table 4) were subjected to an ANOVA with the between-subject factor *feedback group* (no-failure feedback vs. failure feedback), and the within-subjects factors *test phase* (pre- vs. posttest), *learning condition* (deterministic, probabilistic, and chance condition), and *correctness* (correct vs. incorrect). The analysis yielded a significant main effect of test phase [$F(1,33) = 60.29$, $p < .001$], indicating that response latencies decreased from pre- to posttest. Moreover, we found reliable main effects of learning condition [$F(2,66) = 23.51$, $p < .001$] and correctness [$F(1,33) = 23.68$, $p < .001$] that were qualified by interactions between learning condition and correctness [$F(2,66) = 7.19$, $p < .002$], and test phase, learning condition, and correctness [$F(2,66) = 7.28$, $p < .002$]. Follow-up ANOVAs yielded a significant interaction between learning condition and correctness for posttest [$F(2,66) = 23.13$, $p < .001$, $\varepsilon = .76$] but not for pretest ($F < 1$). At posttest, responses were faster on incorrect than correct trials ($p < .001$). Contrasts revealed that this difference increased with feedback validity (p-values < .001). No main effect or interaction involving the factor feedback group approached significance (p-values > .10).

Tabelle 4. Mean RT (Standard deviation) in the three learning conditions for the no-failure feedback group and the failure feedback at group pre- and posttest (Experiment 1).

RT	No-failure feedback group		Failure feedback group	
	Correct	Incorrect	Correct	Incorrect
Pretest				
Deterministic learning	432 (28)	425 (45)	442 (27)	440 (39)
Probabilistic learning	441 (29)	441 (49)	449 (33)	448 (38)
Chance condition	439 (27)	436 (39)	449 (29)	445 (35)
Posttest				
Deterministic learning	406 (28)	382 (33)	395 (33)	377 (31)
Probabilistic learning	406 (31)	394 (37)	400 (33)	390 (29)
Chance condition	406 (33)	406 (30)	395 (33)	396 (33)

Post-error RT data analyses. Post-error RT data (see Table 5) were analyzed using an ANOVA with the factors *feedback group*, *test phase*, *learning condition*, and *correctness*. The analysis yielded a reliable main effect of test phase [$F(1,33) = 60.96$, $p < .001$], indicating that post-error RT decreased from pre- to posttest. Moreover, we found significant interactions between test phase and learning condition [$F(2,66) = 5.22$, $p < .019$, $\varepsilon = .76$] and test phase, learning condition, and correctness [$F(2,66) = 3.81$, $p < .028$]. Follow-up analyses showed that the decrease in post-error RT was more pronounced for the deterministic and probabilistic learning condition compared to the chance condition on incorrect trials [$F(1,33) = 20.13$, $p < .001$] but not correct trials ($F < 1$). In addition, a reliable interaction between feedback group, correctness, and learning condition was obtained [$F(2,66) = 4.54$, $p < .015$]. Separate ANOVAs revealed a significant interaction between feedback group and correctness only for the deterministic learning condition [$F(1,33) = 6.52$, $p < .017$], reflecting that response latencies were relatively shorter on incorrect trials in the no-failure feedback group. No further effect of feedback group was found to be reliable (p-values > .12).

Tabelle 5. Mean post-error RT (Standard deviation) in the three learning conditions for the no-failure feedback group and the failure feedback at group pre- and posttest (Experiment 1).

Post-error RT[1]	No-failure feedback group		Failure feedback group	
	Correct	Incorrect	Correct	Incorrect
Pretest				
Deterministic learning	446 (42)	437 (58)	451 (37)	468 (68)
Probabilistic learning	448 (41)	449 (52)	455 (34)	456 (47)
Chance condition	438 (40)	434 (40)	449 (30)	446 (34)
Posttest				
Deterministic learning	403 (30)	385 (44)	394 (38)	387 (37)
Probabilistic learning	408 (32)	397 (41)	399 (35)	385 (40)
Chance condition	405 (33)	410 (30)	394 (36)	396 (32)

[1]Note that post-error RT refers to the next repetition of a given stimulus after a variable number of intervening items.

Experiment 2

Consistent with Experiment 1, RT decreased from pre- to posttest [$F(1,31) = 76.30$, $p < .001$] (see Table 6). As was indicated by a significant interactions between feedback group and test phase, [$F(1,31) = 12.46$, $p < .002$] and feedback group, test phase and learning condition [$F(2,62) = 3.28$, $p < .045$], this decrease was more pronounced for the no-failure feedback group, particularly in the probabilistic and chance condition compared to the deterministic learning condition [$F(1,31) = 5.90$, $p < .022$]. Furthermore, the analysis yielded significant main effects of learning condition [$F(2,62) = 51.05$, $p < .001$, $\varepsilon = .87$] and correctness [$F(1,31) = 42.26$, $p < .001$] that were qualified by an interaction between learning condition and correctness [$F(2,62) = 38.11$, $p < .001$, $\varepsilon = .87$]. Follow-up analyses revealed that erroneous responses were faster than correct responses in the deterministic and probabilistic learning condition [$F(1,31) = 53.50$ and 18.04, respectively, p-values $< .001$] but not in the chance condition [$F < 1$].

Tabelle 6. Mean RT (Standard deviation) in the three learning conditions for the no-failure feedback group and the failure feedback at group pre - and posttest (Experiment 2).

RT	No-failure feedback group		Failure feedback group	
	Correct	Incorrect	Correct	Incorrect
Pretest				
Deterministic learning	442 (31)	411 (30)	424 (25)	402 (21)
Probabilistic learning	449 (36)	439 (31)	430 (20)	423 (29)
Chance condition	452 (37)	451 (37)	429 (25)	430 (22)
Posttest				
Deterministic learning	407 (34)	378 (26)	408 (28)	380 (30)
Probabilistic learning	404 (29)	394 (25)	414 (25)	401 (24)
Chance condition	403 (35)	399 (30)	411 (31)	412 (28)

Post-error RT data analyses. Post-error RT (see Table 7) decreased from pre- to posttest [$F(1,31) = 62.61$, $p < .001$]. A significant interaction between feedback group and test phase [$F(1,31) = 9.46$, $p < .005$], reflected a greater pre-post difference in post-error RT for the no-failure feedback group. Furthermore, we found significant main effects of learning condition [$F(2,62) = 8.04$, $p < .002$] and correctness [$F(1,31) = 26.72$, $p < .001$], as well as an interaction between learning condition and correctness [$F(2,62) = 51.05$, $p < .001$, $\varepsilon = .87$]. Follow-up analyses showed that response latencies were shorter on incorrect than correct trials in the deterministic and probabilistic learning condition [$F(1,31) = 19.50$ and 15.56, respectively, p-values < .001] but not in the chance condition [$F < 1$].

Tabelle 7. Mean post-error RT (Standard deviation) in the three learning conditions for the no-failure-feedback group and the failure-feedback group at pre- and posttest (Experiment 2)

RT	No-failure feedback group		Failure feedback group	
	Correct	Incorrect	Correct	Incorrect
Pretest				
Deterministic learning	439 (33)	419 (43)	429 (25)	412 (33)
Probabilistic learning	454 (41)	445 (38)	431 (23)	431 (33)
Chance condition	453 (41)	450 (36)	428 (24)	430 (22)
Posttest				
Deterministic learning	405 (31)	390 (38)	415 (34)	391 (31)
Probabilistic learning	402 (30)	384 (25)	419 (27)	391 (27)
Chance condition	401 (33)	399 (29)	411 (33)	414 (31)

13. Abbreviations

A-O	action-outcome
ACC	anterior cingulate cortex
ACS-90	Action Control Scale
ANCOVA	analysis of covariance
ANOVA	analysis of variance
BAS	behavioural approach system
BfS	Befindlichkeitsskala
BG	basal ganglia
BIS	behavioural inhibition system
COMT	catechol-*O*-methyltransferase
CR	conditioned response
CS	conditioned stimulus
CSD	current source density
DA	dopamine
DA-RPE	dopamine reward prediction error
dACC	dorsal anterior cingulate cortex
DC	direct current
ERP	event-related potential
dlPFC	dorsolateral prefrontal cortex
FFS	fight-flight system
fMRI	functional magnetic resonance imaging
FRN	feedback-related negativity
HPA	hypothalamus-pituitary adrenal
MANOVA	multivariate analysis of variance
MDD	Major depressive disorder
MFC	medial frontal cortex

MNI	Montreal Neurological Institute
mPFC	medial prefrontal cortex
ms	millisecond
MTL	medial temporal lobe
NA	negative affect
Ne	error negativity
OCD	obsessive compulsive disorder
OFC	orbitofrontal cortex
PA	positive affect
PANAS	Positive Negative Affect Scale
Pe	error positivity
PE	prediction error
PFC	prefrontal cortex
PRO	prediction of response-outcome
R-O	response-outcome
rACC	rostral anterior cingulate cortex
RCZ	rostral cingulate zone
RL	reinforcement learning
RST	reinforcement sensitivity theory
RT	reaction time
S-O	stimulus-outcome
S-R	stimulus-response
sLORETA	standardized low-resolution electromagnetic tomography
SMA	supplementary motor area
SNc	zona compacta of the substantia nigra
(t)CNV	(terminal) contingent negative variation
TD	temporal difference
UR	unconditioned response

US	unconditioned stimulus
vmPFC	ventromedial prefrontal cortex
VTA	ventral tegmental area

14. Annotation

Some oft the data reported in this thesis are also included in the following manuscripts:

Unger, K., Kray, J., & Mecklinger, A. (2012). Worse than feared? Failure induction modulates the electrophysiolgical signature of error monitoring during subsequent learning. *Cognitive, Affective, and Behavioral Neuroscience, 12*, 34-51.

Unger, K., Heintz, S., & Kray, J. (2012). Punishment sensitivity modulates feedback processing but not error-induced learning. *Frontiers in Human Neuroscience, 6:*186.

Unger, K. & Kray, J. (submitted). Differential effects of gain vs. loss anticipation on performance monitoring and error-induced learning.