Negative affect-related changes of action monitoring brain processes: An experimental analysis

Kristien Aarts

Promotor: Prof. Dr. Gilles Pourtois

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CONTENTS

CHAPTER 1: GENERAL INTRODUCTION

"Errare humanum est, perseverare diabolicum"

"To err is human, to persist is of the devil"

(Seneca the younger)

This famous quote emphasizes the importance of action-monitoring and error-detection brain processes for adaptive behavior. To not persist in making errors necessarily implies that these errors are swiftly detected as distinctive motivational events, and they eventually guide or promote learning, thanks to dedicated action-monitoring systems in the human brain (Holroyd & Coles, 2002). Usually, error monitoring does not operate in isolation, but is part of a larger executive control system that enables to exert control over behavior, and is composed of several key cognitive components: inhibition, updating and shifting (Miyake et al., 2000). While inhibition refers to the ability to suppress dominant pre-potent responses, irrelevant information in the external environment or distracting thoughts, updating is defined as the active manipulation of relevant information in working memory. Shifting can be seen as the capacity to quickly alternate between different tasks or mental states. This prefrontal-based executive control system has generally been conceived in the past as an efficient cognitive monitoring system operating on specific mental processes, independently of changes in affect or emotion (Bush, Luu, & Posner, 2000; Duncan & Owen, 2000). However, more recently, this classical view has somewhat been challenged and systematic influences of affective dimensions on this prefrontal executive control system have been taken into account, and eventually modeled to explain how control over behavior may be exerted to cope with fast changing contingencies in the environment (Rushworth, Mars, & Summerfield, 2009). In this perspective, executive control systems and affective control systems are no longer seen as fully separate or non-overlapping, but instead reciprocal and dynamic interactions between these systems are thought

to govern the implementation of adaptive behavior (Etkin, Egner, & Kalisch, 2011; Mansouri, Tanaka, & Buckley, 2009). According to Pessoa (2009) and Bishop (2007), executive control may be influenced by emotion, either in a 'stimulus-driven' or in a 'state dependent' manner. In the former case, emotional stimuli (e.g. emotional faces or affective scenes), disrupt goal-directed processing and attention allocation (Corbetta & Shulman, 2002; Vuilleumier, 2005). By contrast, state-dependent effects can be induced by reward-related manipulations for example (Hickey, Chelazzi, & Theeuwes, 2010; Kennerley & Wallis, 2009) or may be related to specific pre-existing traits or affective dimensions that shape online executive control as well. In particular, negative affect like anxiety and depression has often been linked to alterations in executive control processes (Bishop, 2007; Elliott, 1998; Eysenck, Derakshan, Santos, & Calvo, 2007; Visu-Petra, Miclea, & Visu-Petra, 2012). In this work, I focus on these latter effects and how they influence error monitoring brain processes.

In this context, error monitoring is no exception to the rule, and a growing number of studies and models have confirmed how deeply intricate error-related affective and cognitive control processes are (Frank, Woroch, & Curran, 2005; Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010). Errors (committed with neutral stimuli using well-controlled laboratory tasks, such as the Stroop or Flanker task) are not simply noisy events inadvertently promoting learning (Holroyd & Coles, 2002), they are also able to trigger a cascade of affective or defensive reactions (Hajcak & Foti, 2008; Pourtois et al., 2010), as well as specific attentional orienting effects (Notebaert et al., 2009; Ridderinkhof, Ramautar, & Wijnen, 2009), suggesting that they are swiftly processed as distinctive motivational events, and hence they are not devoid of emotion (Luu, Collins, & Tucker, 2000). For example, a recurrent and robust finding in the affective neuroscience literature is the overactive or hyper sensitive error-monitoring system found in individuals with enhanced negative affect, including trait anxiety or depression (Endrass et al., 2010; Olvet & Hajcak, 2008; Vaidyanathan, Nelson, & Patrick, 2012). However, the exact functional meaning of these overactive

error-monitoring effects remains unclear. Do they reflect a general break-down in executive control in these participants, or rather a differential motivational or affective reaction during the processing of these adverse events? The goal of my PhD dissertation was to address these questions using standard experimental methods, and more specifically to better characterize possible changes in error-monitoring brain processes [when they are explored primarily using advanced Electroencephalogram (EEG)/Event-related potentials (ERP) methods] induced by anxiety or depression, and eventually clarify their functional meaning. To explore error monitoring in (sub clinical) high anxious or clinically depressed patients, I used a common Go/noGo task and methodology. In this task, the differential processing of a specific type of error is investigated, namely the failure to inhibit a pre-potent response tendency (Miyake et al., 2000). In these conditions, participants have to rely primarily on an internal (pre-existing or stored) representation of their action to infer whether a response error has been made or not. These conditions are therefore optimal to explore changes in early error-monitoring processes as a function of anxiety or depression, given that these two traits have usually been seen as resulting from "internalizing" troubles (Krueger, 1999). By contrast, in some other daily-life situations, performance monitoring cannot be achieved solely based on an internal (motor) representation of the action, but the additional processing of external cues in the environment (i.e. feedback) is needed to gauge whether the action is correct or not (Holroyd, Hajcak, & Larsen, 2006). This aspect was also addressed in my dissertation in such a way to assess whether negative affect (trait anxiety) influences internal and external action-monitoring processes equally. Finally, I also devised a new method enabling to decipher "online" the actual affective value of actions generated by participants during the Go/noGo task in order to gain insight into the extent and nature of the substantial changes induced by anxiety or depression during early error (and action) monitoring in this task.

This introductory section is organized as follows. First, I review neuroscientific evidence from ERP and activation studies in the literature, that have focused on delineating the electrophysiological properties and time-course

of error detection, and action-monitoring brain mechanisms more generally. Next, I focus on the phenomenology of "negative affect" in psychology and experimental psychopathology, and more specifically trait anxiety and depression, before clarifying the nature and extent of changes in performance-monitoring and error-detection brain processes accounted for by each of these two psychopathological conditions. Finally, I outline the content of the five empirical chapters and three main research questions included in this dissertation.

ERROR MONITORING

In 1966, Rabbitt reported that individuals systematically slowed down for trials immediately following an error in a continuous performance choice-response task, while no similar slowing effect was found for trials following correct responses. This post-error slowing effect (Laming, 1979) provides evidence for the existence of remedial or adaptive (perhaps attentional orienting) processes following error detection (Danielmeier & Ullsperger, 2011; Notebaert et al., 2009), and hence, it lends support to the idea of core automatic error-detection systems triggering this (secondary) regulative effect. Based on these behavioral results, one may thus assume that cognitive control likely operates thanks to an early error-detection mechanism that in turn leads to a second regulation or adaptive effect, preventing errors to repeat because more control or attention is suddenly exerted once these adverse events have properly been detected (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Significant insights into error-monitoring processes (and more specifically the existence of early, automatic and generic error-detection processes in the human brain) have more recently been obtained using ERPs. A large number of ERP studies have confirmed that error detection is associated with specific error-related ERP components, early on following the onset of the incorrect key press (or motor command), and presumably based on an internal representation of (motor) actions (e.g. corollary discharge originating from the motor cortex; see Sommer & Wurtz, 2008), given this ultra-fast time-course (Mathalon, Whitfield, & Ford,

2003). These deflections include the error-related negativity (ERN/Ne; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring, Goss, Coles, Meyer, & Donchin, 1993) and the error positivity (Pe; Falkenstein et al., 2000; Nieuwenhuis, Ridderinkhof, Blow, Band, & Kok, 2001). Moreover, an ERN/Ne-like component has also been reported and coined the feedback-related negativity (FRN; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Miltner, Braun, & Coles, 1997) in situations where action monitoring is necessarily based on the processing of external (evaluative) feedback, as opposed to internal motor representations in the case of the ERN/Ne and Pe components.

Before I review the exact neurophysiological properties and likely functional meaning of these error-related ERP components, I first introduce the ERP technique in general, as well as the set of brain regions involved in error detection, or more broadly action monitoring.

Event-related potentials (ERPs)

The ERP technique

ERPs provide a non-invasive and direct measure of brain activity related to the processing of specific events in the external environment (e.g. a visual stimulus), or in the participant (e.g. a response) (see Figure 1AB). ERPs are time-resolved, because they provide a temporal resolution at a millisecond time scale, even though these recordings take place at distance (i.e. at the scalp level using specific electrodes or sensors) from the putative (cortical) dipolar brain generators. ERPs are not visible directly, but they are embedded in the electroencephalogram (EEG), which is the raw electrical signal of the brain being 10 to 50 times larger in amplitude than the ERPs. Therefore, a standard averaging procedure is needed to extract ERPs from the continuous EEG (Rugg & Coles, 1995). This requirement has important implications regarding the interpretation of ERPs, which always represent a compound brain activity across many trials (Makeig, Debener, Onton, & Delorme, 2004). This is especially true in the case of response errors, which are usually rare and deviant events (Olvet & Hajcak, 2009b). The underlying assumption is that the neuronal activity generated for all individual events is stable and reproducible, such that the use

of an averaging procedure allows to remove the noise (which is by definition random across events) and to reliably identify the signal (which is systematic across the different individual events or presentations). This neuronal activity in response to an event and giving rise (after averaging) to a given ERP component (or multiple) actually corresponds to the synchronous activation of the post-synaptic dendritic potentials of a large population of neurons. These (pyramidal) neurons are arranged in a geometrical configuration such as to yield a dipolar field. EEG is sensitive to both tangential and radial components of a current source. ERPs provide a direct estimate of these dipolar fields. Because the recording of the EEG usually occurs using scalp electrodes, the spatial resolution of the resulting ERPs is by definition limited, compared to other neuroimaging techniques, like functional Magnetic Resonance Imaging (fMRI) or Positron Emission Tomography (PET). However, a way to partly overcome this limitation is to use a large array of electrodes (up to 128, such as used in this dissertation) covering evenly and densely all scalp locations, and apply additional data analyses (including topographical mapping analyses; see Murray, Brunet, & Michel, 2008; Pourtois, Delplanque, Michel, & Vuilleumier, 2008) and mathematical transformations to gain insight into the underlying configuration of brain generators giving rise to the ERPs. For example, standardized low resolution brain electromagnetic tomography (sLORETA) (Pascual-Marqui, 2002) can be used as a powerful tool to source-localize the recorded ERP components and hence obtain additional critical information about the cortical regions involved in the generation of a specific ERP component (besides its actual amplitude and latency at the scalp level). I adopted this logic in my dissertation when using this specific technique and explored the malleability of error-related brain processes in anxiety or depression. Hence, ERPs provide a neurophysiological signal to timely study brain functions (Michel & Murray, 2011), including performance monitoring and error detection. Each ERP component is usually characterized by specific and precise neurophysiological properties, including the latency (following the onset of the event), the amplitude, the polarity, the topography (i.e. distribution of the electric field over the scalp surface), as well as the underlying brain generators (Luck, 2005). In the

case of error detection and performance monitoring, many studies have already identified in the past several robust error-related or performance-monitoring-related ERP components, and have clarified their specific neurophysiological properties, as reviewed in the next section.

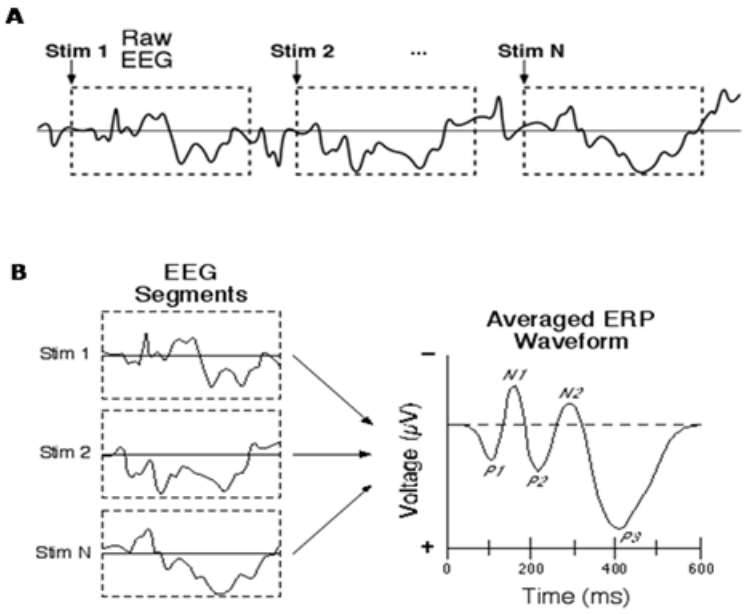

Figure 1. (A) Raw EEG. The rectangles show a 600 ms interval locked to the stimulus (B) EEG segments are averaged.

The error-related negativity (ERN/Ne)

The ERN/Ne is a negative component in the ERP (see Figure 2A), time-locked and phase-locked to the onset of a response error, and is thought to reflect the neural activity of a larger dopaminergic-dependent system that is involved in the rapid monitoring of actions, and the detection of response errors (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein et al., 2000; Frank et al., 2005; Gehring, Coles, Meyer, & Donchin, 1990; Gehring et al., 1993; Gehring & Willougby, 2002; Holroyd & Coles, 2002). The ERN/Ne is usually elicited between 0 and 100 ms post-error onset, is maximal at frontocentral electrodes, consistent with underlying brain sources located in the dorsal anterior cingulate cortex (dACC; Bediou, Koban, Rossett, Pourtois, & Sander, 2012; Carter et al., 1998; Debener, Ullsperger, Fiehler, von Cramon, & Engel, 2005; Dehaene, Posner, & Tucker, 1994; Herrmann, Rommler, Ehlis, Heidrich, & Fallgater, 2004; Kiehl, Liddle, & Hopfinger, 2000; O'Connell et al., 2007; Vocat,

Pourtois, & Vuilleumier, 2008). Given this ultra-fast unfolding in dACC following error detection, it has been advocated that the ERN/Ne reflects an early, generic and automatic reaction to errors, based on the rapid detection of a mismatch between the actual and intended, or desired, motor action, before any sensori-motor or proprioceptive feedback comes into play (Bush et al., 2000). This early error-related component is generated regardless of the response modality or effector with which response errors are made (e.g. hand, finger and foot; Falkenstein et al., 2000; Holroyd, Dien, & Coles, 1998). Strikingly, error awareness is not necessary for the generation of the ERN/Ne (Nieuwenhuis et al., 2001; O'Connell et al., 2007; Shalgi, Barkan, & Deouell, 2009; but see Wessel, Danielmeier, & Ullsperger, 2011). The ERN/Ne is however larger when accuracy, as opposed to speed, is stressed in task instructions (Falkenstein et al. 2000; Gehring et al., 1993), as well as when errors become motivationally significant events (Bernstein, Scheffers, & Coles, 1995; Falkenstein et al., 2000; Hajcak & Foti, 2008; Hajcak, Moser, Yeung, & Simons, 2005). These two findings unambiguously link this early medial frontal ERP component to action-monitoring and error-detection processes.

Several theories have been put forward to account for these remarkable electrophysiological properties. Initially, this component was interpreted as reflecting a rough "cognitive" mismatch signal between the intended and actual motor action (Bernstein et al., 1995; Coles, Scheffers, & Holroyd, 2001; Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1993; Mathalon et al., 2003). Alternatively, Holroyd and Coles (2002) posited in their influential model that the ERN/Ne is a reward-prediction error signal. Using the reinforcement learning framework (Sutton & Barto, 1998), these authors stated that the ERN/Ne is generated when the current action is worse than the expected or desired one (see also Frank et al., 2005). This negative prediction error is reflected by a phasic decrease of dopamine in deep midbrain regions (Fiorillo, Tobler, & Schultz, 2003), which releases the dACC via specific fronto-striatal loops (Seifert, von Cramon, Imperati, Tittgemeyer, & Ullsperger, 2011), and in turn yields the ERN/Ne component. Yet, another account for the ERN/Ne

was proposed by Botvinick et al. (2001). These authors underscored that the ERN/Ne is not specifically elicited following response errors, but rather signals a conflict among competing responses, like a tendency to respond vs. to withhold a response (see also Yeung, Botvinick, & Cohen, 2004). In this framework, response errors and conflicts are somehow lumped together, and therefore the ERN/Ne is not seen as an error-selective signal, but instead as a conflict-related component. Some authors have also found a link between the magnitude of the ERN/Ne and post-error adjustments in behavior, including the post-error slowing effect (Debener et al., 2005; Gehring et al., 1993). Finally, some authors have also advocated that the ERN/Ne corresponds to an affective evaluative signal related to the emotional or motivational significance of errors (Hajcak & Foti, 2008; Luu et al., 2000; Luu et al., 2003; Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003). Consistent with this view, these authors have reported changes in the amplitude of the ERN/Ne as a function of the negative affective state or trait of the participants. I present and discuss these findings more thoroughly later.

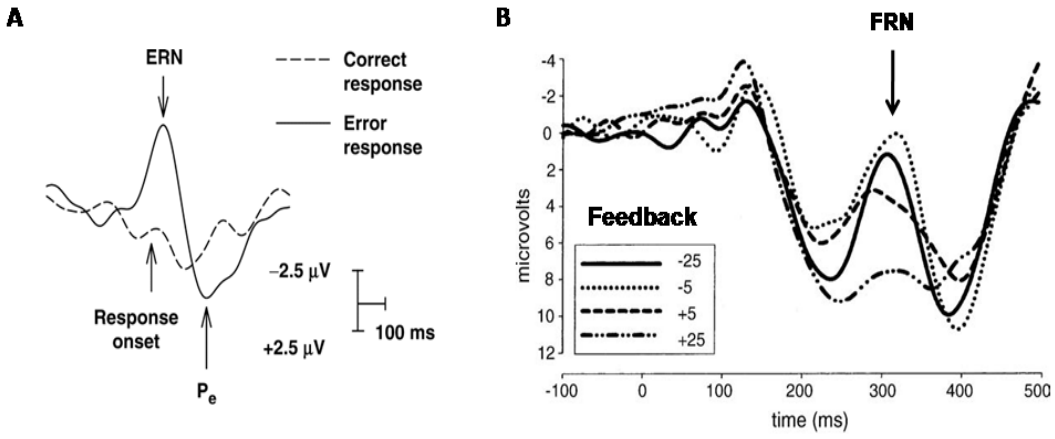

Figure 2. (A) Illustration of the ERN/Ne and Pe components elicited following errors during a standard behavioral task (data from Amodio, Kubota, Harmon-Jones and Devine, 2006). (B) Illustration of the FRN component elicited following negative feedback during a gambling task (data from Hajcak, Moser, Holroyd, & Simons, 2006)

The correct-related negativity (CRN)

A negative component similar to the ERN/Ne but associated to correct responses, is the correct-related negativity (CRN). The CRN is typically smaller in amplitude than the ERN/Ne (see Figure 2A), but these two components share the

same early time-course following action execution, as well as the same medial frontal scalp distribution, and likely similar neural sources within the dACC (Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). The observation of an ERN/Ne-like response in the absence of an error led some authors to argue that the ERN/Ne and CRN are actually not distinct, but they reflect the same generic early cognitive control process (Falkenstein et al., 2000; Roger, Bénar, Vidal, Hasbroucq, & Burle, 2010; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal et al., 2000). An increased CRN has also been related to enhanced response uncertainty (Coles et al., 2001; Pailing & Segalowitz, 2004a). At any rate, it remains important to establish whether motivational components or negative affect influences both the ERN/Ne and CRN components (Hajcak, McDonald, & Simons, 2004), or the ERN/Ne component alone, and therefore the early error-monitoring processes selectively. This question too, was addressed in my PhD dissertation.

The error-positivity (Pe)

This ERN/Ne component is usually followed by a large positive component, the Pe (see Figure 2A). The Pe is a broad deflection resembling the P3 component, peaking over the vertex (or more posterior parietal scalp positions along the midline, such as PZ) roughly 150-300 ms after response error onset, with neural generators involving possibly more rostral ACC as well as posterior cingulate and insular cortex regions, compared to the earlier ERN/Ne component (Dhar, Wiersema, & Pourtois, 2011; Falkenstein et al., 1991; Falkenstein et al., 2000; Herrmann et al., 2004; Nieuwenhuis et al., 2001; O'Connell et al., 2007; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). This error-related component can be dissociated at the functional level from the preceding ERN/Ne component (Nieuwenhuis et al., 2001; Overbeek et al., 2005). Although the Pe is typically less investigated and hence less well understood than the ERN/Ne component in the literature, some authors have nevertheless linked this component to the conscious registration of response errors (Dhar et al., 2011; Endrass, Franke, & Kathmann, 2005; Leuthold & Sommer, 1999; Nieuwenhuis et al., 2001). Alternatively, it could also reflect an affective appraisal of errors (Falkenstein et

al., 2000; van Veen & Carter, 2002), a P300-like attention orienting response (Davies, Segalowitz, Dywan, & Pailing, 2001; Hajcak, McDonald, & Simons, 2003b; Ridderinkhof et al., 2009), or an accumulation of evidence process that timely informs about error commission (Steinhauser & Yeung, 2010).

The feedback-related negativity (FRN)

Whereas the ERN/Ne or CRN, and Pe components reflect error detection based on internal monitoring processes, the feedback-related negativity (FRN, see Figure 2B) likely reflects the same process than the ERN/Ne, when it is achieved based on external stimuli (feedback; see Figure 2B). The FRN shares many electrophysiological properties with the response related ERN/Ne component: It is a negative component peaking at fronto-central electrodes roughly 250-300 ms post-negative (visual) feedback onset and that is likely generated within the dACC as the ERN/Ne (Gehring & Willoughby, 2002; Miltner et al., 1997). According to Holroyd and Coles (2002), the same dopaminergic-dependent reinforcement learning brain system is at stake for the FRN and ERN/Ne, based on internal and external monitoring cues, respectively (see also Frank et al., 2005; Chase, Swainson, Durham, & Benham, 2011). Usually, the FRN is larger for negative compared to positive feedback and for unexpected compared to predictable outcomes (Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003). These findings point to the involvement of the FRN in the processing of the valence or reward value of the feedback. Hence, the FRN, when contrasted to the ERN/Ne, provides a powerful means to assess the efficiency of performance-monitoring brain processes when based on external, as opposed to internal, evaluative cues. Accordingly, possible differential effects of trait anxiety on internal (ERN/Ne) vs. external (FRN) monitoring processes were also investigated in this work by means of these two performance-monitoring ERP components.

Brain areas associated with performance monitoring

In addition to source localization analyses of ERP data that have shown the ubiquitous involvement of the dACC in the generation of the ERN/Ne, CRN, Pe

and FRN, fMRI studies have largely corroborated the importance of this region for performance monitoring and error detection (Bush et al., 2000). These brain mapping studies based on fMRI (or PET) have also clearly shown that this dACC region (sometimes called rostral cingulate zone, RCZ; see Klein et al., 2007) is not operating in isolation, but is actually part of a larger brain circuit (see Figure 3), encompassing fronto-striato-thalamo-cortical loops involved in performance monitoring (Danielmeier, Eichele, Forstmann, Tittgemeyer, & Ullsperger, 2011; Seifert et al., 2011; Ullsperger & von Cramon, 2006).

Anterior cingulate cortex (ACC)

Several ERP studies have identified the dACC as the main neural source of the ERN/Ne (Carter et al., 1998; Debener et al., 2005; Dehaene et al., 1994; Herrmann et al., 2004; Kiehl et al., 2000; O'Connell et al., 2007; Vocat et al., 2008), Pe (Falkenstein et al., 1991; Falkenstein et al., 2000; Herrmann et al., 2004; Nieuwenhuis et al., 2001; O'Connell et al., 2007; Overbeek et al., 2005), CRN (Vidal et al., 2000) or FRN component (Gehring & Willoughby, 2002; Holroyd & Coles, 2002). Although these neurophysiological studies have clearly confirmed the involvement of the dACC (or RCZ) in error monitoring (Bush et al., 2000; Devinsky, Morrell, & Vogt, 1995; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004), lesions of the RCZ (mainly resulting from corpus callosotomy or tumors) were nonetheless not always associated with clear cut error-monitoring or -detection impairments in humans, suggesting that this medial frontal cortex region is probably sufficient, but not necessary for normal error monitoring (Fellows & Farah, 2005). Neuro-anatomically, the ACC is part of the limbic system and more specifically forms the frontal/anterior part of the cingulate cortex (Brodmann Areas – BAs, 24, 32 and 33). The ACC classically consists of two major subdivisions that subserve distinct functions, an emotional rostral part (rACC; including BAs 24, 25, 32 and 33), and a cognitive dorsal part (dACC that includes BA 24 and 32) (Bush et al., 2000; Devinsky et al., 1995). The dACC has been shown to be activated during cognitively demanding tasks and is part of a larger attention network (Corbetta & Shulman, 2002) that includes the lateral prefrontal cortex (lPFC; BA 46/9), parietal cortex (BA7), premotor and

supplementary motor areas (BA6). By contrast, the affective rostral subdivision of the ACC is activated during affect-related tasks and is connected to the amygdala, periaqueductal gray, nucleus accumbens, hypothalamus, insula, hippocampus and orbitofrontal cortex (see Figure 3). Whereas the affective vs. cognitive subdivision of the ACC has proven to be particularly useful to account for a variety of activation patterns (Bush et al., 2000) or behavioral impairments following brain damage (Devinsky et al., 1995), more recently, alternative neuro-anatomical models of the ACC have been put forward that somehow provide a more integrated (and less segregated) view of ACC functioning (Etkin et al., 2011).

FMRI or PET studies have found an increased ACC activity during error detection (Carter et al., 1998; Kiehl et al., 2000; Menon, Adleman, White, Glover, & Reiss, 2001; van Veen & Carter, 2002) and negative feedback processing (Holroyd & Coles, 2002). However, as was the case for the ERN/Ne, it is unclear from these activation studies whether this increased ACC activity reflects error detection per se, or more generally conflict detection (see Carter et al., 1998; Kerns et al., 2004). According to these theories, the dACC activation during error detection might actually reflect conflict monitoring, and in turn an enhanced engagement of cognitive control. Alternatively, an enhanced ACC activation to errors or even conflicts might reflect an appraisal of the distinctive affective or motivational value of these events, not because they are errors or conflicts by themselves, but because these specific events are negatively marked (Dreisbach & Fischer, 2012) and therefore, they usually readily signal a need to exert enhanced control on behavior (Hajcak & Foti, 2008; Hajcak et al., 2005; Luu et al., 2003; Pizzagalli, Peccoralo, Davidson, & Cohen, 2006; Polli et al., 2009).

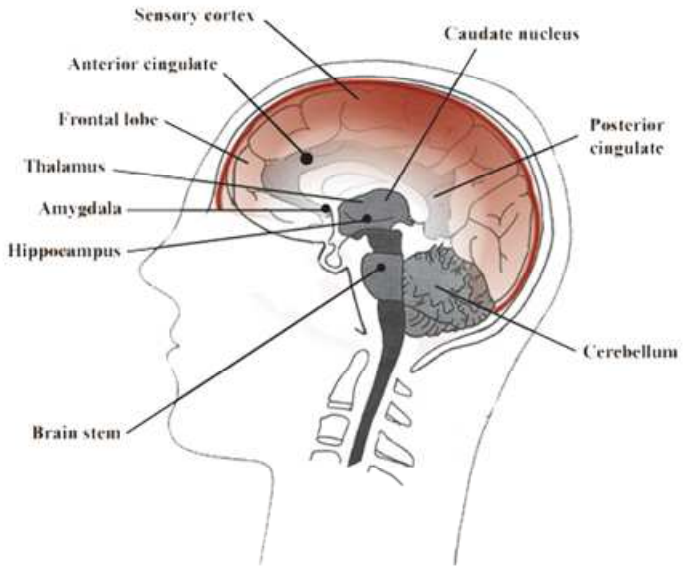

Figure 3. Key brain regions typically associated with performance monitoring. Image taken from http://brain.mcmaster.ca/cp720/

Lateral Prefrontal Cortex (lPFC)

The lPFC is a key structure involved in executive functions and cognitive control (Botvinick et al., 2001; Duncan & Owen, 2000; Rushworth, Buckley, Behrens, Walton, & Bannerman, 2007). The lPFC has been related to the maintenance and updating of task sets (Barber & Carter, 2005; Brass & von Cramon, 2004; Bunge, Klingberg, Jacobsen, & Gabrieli, 2000; Derfuss, Brass, & Von Cramon, 2004; MacDonald, Cohen, Stenger, & Carter, 2000). With respect to error monitoring, activity in the lPFC has been linked not so much to the quick evaluative component, but more to the subsequent regulation component following "automatic" error detection. More specifically, when an error is committed and an ERN/Ne is rapidly generated in the dACC, an increase in attentional control is subsequently needed and this remedial process would be reflected in an increased activity in the dorso lPFC following errors (Carter et al., 1998; Debener et al., 2005; Garavan, Ross, Murphy, Roche, & Stein, 2002; Ridderinkhof et al., 2004; Swick & Turken, 2002). Lesion studies also showed that unilateral damage to the lPFC resulted in an impaired generation of the

electrophysiological markers of error processing (Ullsperger, von Cramon, & Muller, 2002; Gehring & Knight, 2000). Both studies showed a blunted difference between correct and incorrect trials at the level of the ERN/Ne-CRN following lPFC damage. Moreover, Ullsperger and von Cramon (2006) additionally showed that patients with lesions circumscribed to the lPFC had a smaller ERN/Ne, and no Pe or CRN component. Based on these results, one can conclude that lPFC seems to play an important role in the early evaluation of actions and errors, as early as when the ERN/Ne reaches its maximum amplitude.

Basal Ganglia

The basal ganglia consisting of a group of nuclei at the base of the forebrain play an important role in error monitoring as well, especially its constitutive mesencephalic dopamine system. Phasic changes in spiking activity of dopaminergic neurons within this system are thought to signal errors in reward prediction to the striatum, as well as to the cortex (Fiorillo et al., 2003; Jocham, Klein, & Ullsperger, 2011). This phasic change in levels of dopamine in the forebrain remotely influences the ACC region, given the fronto-striatal loops connecting these non-overlapping brain regions, especially during performance monitoring (Seifert et al., 2011). The reinforcement learning theory of the ERN/Ne component is actually based on this specific brain circuitry, and hence indirectly to dopaminergic changes in the forebrain and basal ganglia during early error detection (Frank et al., 2005; Holroyd & Coles, 2002). More specifically, phasic decreases in dopamine activity, indicating a negative reward prediction error (i.e. when the action is worse than expected), are associated with a larger ERN/Ne component and phasic increases, indicating a positive reward prediction error (i.e. when the action is better than expected), with a smaller ERN/Ne component. Accordingly, lesions of the basal ganglia have been related to an impaired ERN/Ne component, as well as to the absence of a Pe and CRN components during error monitoring (Ullsperger & von Cramon, 2006). Moreover, after the administration of a dopaminergic agonist, the early ERN/Ne was found to be larger (De Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004), while it was smaller after the administration of a dopaminergic antagonist (Zirnheld et

al., 2004), linking this specific neurotransmitter to early error-detection brain processes reflected by the ERN/Ne ERP component. Also patients with selective dopaminergic deficits in the basal ganglia, like Parkinson patients, show reduced ERN/Ne amplitudes during error commission (Falkenstein et al., 2001; Holroyd & Coles, 2002). Lesions in the striatum have also been shown to affect the amplitude of the ERN/Ne component (Ullsperger & von Cramon, 2006).

Other brain regions

As clearly outlined here above, error detection is not circumscribed to the ACC, but accumulating data from imaging, neuropsychology and EEG point to the involvement of a large brain network in this process (see Figure 3). Hence, whereas the involvement of the ACC in error detection is ubiquitous, this function is likely to be sub served by multiple brain regions, besides this medial frontal cortex area.

Several source localization studies found that the ERN/Ne is not only generated in the ACC but that the surrounding medial prefrontal cortex (mPFC) and more specifically the pre Supplementary Motor Area (SMA;BA6) is also involved early on following response onset in error monitoring (Kiehl et al., 2000; Menon et al., 2001). This region is known to play an important role in motor preparation (Picard & Strick, 1996; Rizzolatti, Luppino, & Matelli, 1996; Tanji, 1994), as well as in higher level motor control (Picard & Strick, 1996; Schubotz & von Cramon, 2001; Tanji, 1994). According to Rizzolatti et al. (1996) and Vorobiev, Govoni, Rizzolatti, Matelli and Luppino (1998), the pre-SMA is involved in complex cognitive and motivational aspects related to motor control.

Moreover, an association between the rACC and the amygdala during action monitoring has been evidenced by Polli et al. (2009). These authors showed that the activation in the right amygdala and right rostral ACC predicted greater accuracy, while the left amygdala activation predicted a higher error rate. An early role of the amygdala during error monitoring has been confirmed by Pourtois et al. (2010). These authors found, using direct intracranial recordings in two pharmaco-resistant epileptic patients, that response errors

(during a Go/noGo task) led to a delayed neural response in the amygdala, compared to correct responses. Moreover, these authors found that this effect was distinct from the typical early error detection effect taking place in a non-overlapping dACC region, while the two regions showed an enhanced connectivity (in the theta band) early on following response onset. These results have been interpreted in terms of a rapid encoding of the behavioral relevance of (self-generated) motor actions in the amygdala (see also Sander, Grafman, & Zalla, 2003).

Another structure that plays a role in error monitoring, and more specifically in error awareness (and hence the generation of the Pe ERP component), is the anterior insula (Dhar et al., 2011; Hester, Nestor, & Garavan, 2009; Klein et al., 2007; Ullsperger et al., 2010; Wessel et al., 2011). Although speculative at this stage, the anterior insula, which has important reciprocal anatomical connections with the mPFC and the ACC, would participate to error awareness since this same region is involved in interoceptive awareness and the regulation of the body's homeostasis (Craig, 2002; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Singer, Critchley, & Preuschoff, 2009). These "internalizing" processes would therefore play a role in the conscious detection of self-generated response errors (and by extension the Pe component; see Nieuwenhuis et al., 2001; Ridderinkhof et al., 2009), especially in situations when this process is mostly based on the rapid monitoring of internal motor representations (see Dhar et al., 2011; Wessel et al., 2011). Along the same lines, another important region that has been overlooked regarding error-detection brain processes but seems however to participate to them is the orbitofrontal cortex (OFC). Lesions of the OFC lead to a blunted ERN/Ne (Swick & Turken, 2002), and more recently the OFC was linked to the evaluative encoding of self-generated actions, as being either correct or incorrect (see Dhar et al., 2011).

Finally, the thalamus also seems to modulate ACC activity during performance monitoring and error detection. The thalamus is closely connected to the ACC and integrates inputs from the striatum, the lPFC and the cerebellum (Seifert et al., 2011). Recently, Seifert et al. (2011) showed that patients with

focal thalamic lesions had a smaller ERN/Ne amplitude, a reduced error awareness and decreased post-error adjustments. Peterburs et al. (2011) also found altered error processing following thalamic lesions.

Summary

Error detection and performance monitoring are thus characterized by well-defined ERP components (ERN/Ne and Pe), and these processes seem to depend on the integrity of a large scale brain network, wherein the ACC probably plays a central or pivotal role, given its widespread anatomical (reciprocal) connections with a large number of key brain regions, both at the cortical and subcortical levels. This review of the existing neuroscientific data also highlights that these processes are probably more complex than it may appear at first sight. In particular, error monitoring appears to involve core cognitive functions or mechanisms (e.g. reinforcement learning signals), but this process is not immune to changes in motivation or affect, nor is it encapsulated from an anatomical point of view. As I review in the following sections, negative affect (anxiety or depression) actually exert strong modulatory effects on this efficient error-detection brain machinery. First, I outline the basic characteristics of trait anxiety and depression in experimental psychopathology, before reviewing and clarifying what are their respective influences on performance-monitoring and error-detection brain processes.

NEGATIVE AFFECT

Negative affect in internalizing psychopathology

Phenomenology

Negative affect is one of the two dimensions that has consistently been observed as an important constituting factor of the affective structure (Watson & Tellegen, 1985). In contrast to the other affective dimension (i.e. positive affect) that reflects the extent to which a person feels enthusiastic, active and alert, negative affect has been characterized by subjective distress and includes a variety of aversive mood states, including anger, contempt, disgust, guilt, fear

and nervousness (Watson, Clark, & Tellegen, 1988). These negative affect characteristics have been shown to play a key role in the genesis and maintenance of internalizing disorders (Brown, 2007; Clark & Watson, 1991; Tellegen, 1985), which is a class of disorders that is best characterized by a tendency to internalize psychological distress and that has typically been opposed to externalizing disorders in which individuals rather tend to externalize their distress, as reflected for example in enhanced impulsivity, sensation seeking or behaviors that may go against the social norms (Krueger, 1999). Negative affect as a separate entity has more specifically been shown to predict the development of internalizing disorders. Several studies reported that individuals with high levels of negative affect were experiencing more anxiety and depressive symptoms (Jylha & Isometsa, 2006), and were also suffering more from anxiety disorders (Hettema, Prescott, & Kendler, 2004) and major depressive disorders (Brown, Chorpita, & Barlow, 1998; Schmitz, Kugler, & Rollnik, 2003). Moreover, negative affect has been closely linked or shares many characteristics with personality traits such as harm avoidance (Cloninger, 1986), neuroticism (Costa & McCrae, 1988), or behavioral inhibition sensitivity (BIS; Carver & White, 1994), which involves a hypersensitivity to threat (or punishment) cues and subsequent withdrawal. However, despite the fact that anxiety and depression belong to the same class of internalizing disorders that is primarily characterized by negative affect, these two psychopathology conditions are obviously not fully overlapping in their phenomenology and/or neurobiological substrates, and they each have specific attributes (Brown, 2007; Clark & Watson, 1991), as reviewed in the next sections.

Error monitoring

Both anxiety and depression, or internalizing disorders in general, are thought to be characterized by an increased sensitivity towards errors and negative feedback, especially when they are perceived as challenging self-efficacy, or seen as potential social threats (Abela & D'Alessandro, 2002; Beats, Sahakian, & Levy, 1996; Conway, Howell, & Giannopoulos, 1991; Elliott, Sahakian, Herrod, Robbins, & Paykel, 1997; Elliott et al., 1996; Enns & Cox, 1999;

Henriques & Leitenberg, 2002; Holmes & Pizzagalli, 2008; Pizzagalli et al., 2006; Shafran & Mansel, 2001). This enhanced sensitivity to errors in individuals with internalizing disorders has also been confirmed by previous ERP studies showing generally larger ERN/Ne and/or CRN amplitudes in these individuals (Hajcak, McDonald, & Simons, 2003a; Simons, 2010; Vaidyanathan et al., 2012; Vocat et al., 2008). Based on this evidence, some authors have put forward the notion that the ERN/Ne could even be considered as a reliable endophenotype (or stable biomarker) for internalizing disorders (Olvet & Hajcak, 2008). Before I review in more details the existing neuroscientific evidence linking the ERN/Ne-CRN ERP component to internalized disorders, I first outline the main characteristics of anxiety and depression, separately, both in terms of psychological constructs and putative neurobiological substrates.

Anxiety

Phenomenology

Anxiety is an adaptive and normal warning reaction of our biological system that prepares the body to react appropriately in front of potentially dangerous or harmful situations in the environment (e.g. stressors). When these dangerous situations concern the actual or anticipated occurrence of an explicit threatening stimulus, the anxious reaction is referred to as “fear”. By contrast, “anxiety” is triggered by less explicit or more generalized cues (Lang, Davis, & Öhman, 2000). Anxious reactions consist of loose changes concurrently at emotional, cognitive, physiologic and behavioral levels. Anxious individuals experience for example high levels of negative affect (Brown et al., 1998; Clark & Watson, 1991); they are hypervigilant (Eysenck, 1992), showing a specific attention bias towards threat (Mathews & Macleod, 1994), and they show a tendency to worry or ruminate (Muris, Roelofs, Rassin, Franken, & Mayer, 2005). Physiologically, hyper arousal like pounding heartbeat, sweating, dizziness, feeling of choking, and shortness of breath is observed (Brown et al., 1998; Nitschke, Heller, Palmieri, & Miller, 1999); at the behavioral level, anxious individuals are characterized by avoidance (Gray, 1982) and sometimes inhibition (Carver & White, 1994; Jorm et al., 1999). These characteristics can fluctuate

over time or occur in specific situations in which the level of perceived threat is high, and in these situations high levels of state anxiety will be observed and measured (Rossi & Pourtois, 2011). Usually, these state-dependent reactions are adaptive and they serve the function to protect us from harm and hazard (Lang et al., 2000). However, when individuals are characterized by a sustained increased sensitivity to stressors (i.e. high trait anxiety) or when anxious reactions become stable or chronic, anxiety can become maladaptive, eventually result in a disorder and strongly interfere with the daily life activities or occupations of a person (Barlow, 1988; Rosen & Schulkin, 1998).

Anxiety is not a monolithic construct though. Two main classes of anxiety have typically been identified in the literature (i.e. anxious apprehension vs. anxious arousal; Engels et al., 2007; Watson et al., 1995). Anxious apprehension is mainly characterized by worry about the future, by verbal rumination and usually encompasses anxiety disorders or trait-related anxiety, generalized anxiety disorders and obsessive compulsive disorders (Barlow, 1991; Heller, Nitschke, Etienne, & Miller, 1997). By contrast, anxious arousal is characterized by somatic tension and physiological hyper arousal, and includes panic disorders, phobia or post traumatic stress disorders (Watson et al., 1995). Distinctive and common characteristics across these various types of anxiety have also been evidenced at the neurobiological level, as discussed here below.

Neurobiology

Because a common characteristic among anxiety disorders is excessive distress (Brown et al., 1998; Clark & Watson, 1991), either in response to explicit stimuli (i.e. fear) or to more general stimuli (i.e. anxiety), neurobiological effects of anxiety have mostly been established based on animal models of fear (Davis, 2006; Indovina, Robbins, Nunez-Elizalde, Dunn, & Bishop, 2011; LeDoux, 1996; Maren, 2008; Phelps & LeDoux, 2005). What these different models share in common is the predominant role of the amygdala (here reflected by an overactive amygdala activation) in the pathogenesis and maintenance of the disease, including posttraumatic stress disorders (PTSD), social anxiety disorders (SD), specific phobias (SP) and panic disorders (PD), but not in obsessive

compulsive disorders (OCD) (Etkin & Wager, 2007; Shin & Liberzon, 2010). Also the (anterior) insular cortex, a region involved in the regulation of the autonomic nervous system (Oppenheimer, Gelb, Girvin, & Hachinski, 1992), proprioception and interoception (Craig, 2002) is typically more active during the processing of a variety of negative emotions (Phan, Wager, Taylor, & Liberzon, 2002), and is consistently found to be hyperactive in a wide range of anxiety disorders (Etkin & Wager, 2007; Shin & Liberzon, 2010). However, in accordance with the variability in the phenomenology and symptomatology across anxiety disorders, this increased amygdala and insula activity are also variable depending on the type of anxiety disorders (Etkin & Wager, 2007). Increased amygdala function appears for example to be more diagnostic of phobic disorders. In their meta-analysis, Etkin and Wager (2007) also reported altered brain activity in other regions that could possibly be specific for certain anxiety disorders. For example, a smaller hippocampus volume tends to be systematically associated with PTSD. A blunted activation of the hippocampus accompanied by an increased rACC activation is a rather consistent observation in panic disorders, whereas OCD would mainly be associated with dysfunctions in striatal-orbitofrontal circuits (Graybiel & Rauch, 2000).

Interestingly, also the ACC, this large pMFC area that is typically involved in the generation of several error-related ERP components and performance monitoring more generally, has consistently been found to be dysfunctional across several anxiety disorders (Bishop, 2009; Bush et al., 2000; Devinsky et al., 1995; Etkin & Wager, 2007; Shin & Liberzon, 2010). Abnormal ACC activity was more specifically observed in patients with panic disorders (Bystritsky et al., 2001), in veterans with PTSD (Rauch et al., 1996; Shin et al., 2001), or in simple phobia (Rauch et al., 1995). Also anxiety induction in healthy adult participants was associated with increased blood flow in the ACC (Bishop, 2007; Kimbrell et al., 1999). This strong relationship between the ACC and multiple anxiety disorders has led some researchers to argue that ACC hyperactivity may actually be related to the experience of symptoms that are common to all anxiety disorders, including worry and distress (Kimbrell et al., 1999; Malizia, 1999). In

fact, these findings showing that the ACC is probably involved in the pathophysiology of many anxiety disorders, combined with the evidence showing that the ACC is directly implicated in performance monitoring and in error detection, have fostered the idea that anxiety disorders (or trait anxiety more generally) may actually be associated with specific performance-monitoring or error-detection impairments, as outlined in the next section.

Error monitoring

At the behavioral level, trait anxiety has often been related to altered performance in cognitive or attentional control tasks (Bishop, 2009; Derryberry & Reed, 2002; Eysenck et al., 2007). In the attentional control theory (Eysenck et al., 2007), it has been proposed that anxiety does not primarily affect the effectiveness or the accuracy of attentional control (i.e. the number of errors), but more the efficiency (i.e. the speed). Processing efficiency is here defined as the latent relationship between performance's effectiveness and the amount of efforts spent in the task to reach a certain level of performance (Berggren, Hutton, & Derakshan, 2011). However, no study to date has attempted to use this specific framework to account for anxiety-related changes during early error-monitoring brain processes, as revealed using ERP measurements. Many ERP studies have already reported enhanced or overactive ERN/Ne (and often CRN) components in patients with anxiety disorders during early error monitoring in standard interference tasks, including OCD (Endrass, Klawohn, Schuster, & Kathmann, 2008, Endrass et al., 2010; Gehring, Himle, & Nisenson, 2000; Hajcak & Simons, 2002; Johannes et al., 2001; Stern et al., 2010) or generalized anxiety disorders (Weinberg, Olvet, & Hajcak, 2010). However, a larger ERN/Ne is usually not only observed in individuals with clinical levels of anxiety or clear cut anxiety disorders, but also in healthy adult participants showing high levels of sub clinical trait anxiety (but less systematically for changes in state anxiety), as estimated using standard questionnaires or inventories available in the literature (Rossi & Pourtois, 2011; Spielberger, 1983). Hence, participants with higher levels of trait anxiety were shown to have increased ERN/Ne and CRN components (Hajcak et al., 2003a, 2003b) during early action monitoring, as was the case for healthy

participants characterized by an enhanced sensitivity to punishment (Boksem, Tops, Wester, Meijman, & Lorist, 2006). When considering the existing ERP studies, this increased ERN/Ne-CRN during early action monitoring in high anxious individuals seems to be related to stable trait characteristics, but to be not selective for error processing (i.e. the CRN component is also usually typically increased in these participants or patients, compared to low anxious individuals). Mixed results were obtained for a similar increased ERN/Ne–CRN component in individuals with enhanced levels of state anxiety. While Moser, Hajcak and Simons (2005) observed a similar ERN/Ne-CRN in phobic individuals directly exposed to their phobia-related objects vs. controls, Hajcak, Franklin, Foa and Simons (2008) observed a similar ERN/Ne-CRN before and after treatment of OCD symptoms. Moreover, this modulatory (boosting) effect of trait anxiety on action-monitoring processes appears to be specific and primarily concerns the early "automatic" stages of action monitoring (not restricted to error processing thereof), as reflected by the amplitude changes at the level of the ERN/Ne-CRN component. However, typically, trait anxiety does not alter the following stage or error or action monitoring, as reflected by the error-related Pe component, whose amplitude does not vary with trait anxiety or anxiety disorders, unlike the preceding ERN/Ne-CRN component (Endrass et al., 2008; Hajcak et al., 2003a; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006; McDermott et al., 2009; Weinberg et al., 2010). In accordance with these neurophysiological findings, a hyperactive ACC has also been found during error monitoring in anxious individuals (Fitzgerald et al., 2005; Paulus, Feinstein, Simmons, & Stein, 2004; Ursu, Stenger, Shear, Jones, & Carter, 2003).

As already said above, whether this early increased action monitoring seen in anxious individuals is truly error specific or reflects instead a general response monitoring or attention deficit (Bishop, 2007), remains an open question. Indeed, whereas some earlier ERP studies showed an enhancement of both the ERN/Ne and CRN components in individuals with anxiety (Endrass et al., 2008; Endrass et al., 2010; Hajcak et al., 2003a, 2003b; Hajcak & Simons, 2002), other studies did not report systematic amplitude changes at the level of the CRN in

anxiety (Gehring et al., 2000; Hajcak et al., 2008; Stern et al., 2010; Weinberg et al., 2010). Moreover, this uncertainty is also probably explained by the fact that the CRN is usually much smaller in size than the ERN/Ne component (Luu & Tucker, 2001; Luu, Tucker, & Makeig, 2004; Vidal et al., 2003; Vidal et al., 2000) hence leading potentially to a floor effect for this ERP component. Moreover, all the ERP studies reviewed here, have used amplitude measurements at a few electrode positions (Picton et al., 2000), a method that may not be sensitive enough to capture more global (sometimes subtle) differences in the expression (rather than the strength or amplitude) of the ERN/Ne and CRN components as a function of trait anxiety (Pourtois, 2011; Pourtois et al., 2008). **I directly addressed this specific issue in the present dissertation (see Chapter 2)**.

With respect to the neural processing of external evaluation cues (i.e. feedback and the FRN component), the existing literature exploring possible effects of anxiety on this performance-monitoring process is scant. De Pascalis et al. (2010) found that individuals who were more sensitive to punishment (as evidenced using the BIS-BAS scale, see Carver & White, 1994) had a larger FRN to monetary loss on noGo trials during a Go/noGo task. By contrast, two other ERP studies reported a larger FRN amplitude for low, compared to high anxious individuals (Gu et al., 2010; Simons, 2010). Accordingly, **another goal of the present doctoral dissertation was to compare, using advanced ERP methods, effects of sub clinical trait anxiety on the processing of internal (ERN/Ne) vs. external (FRN) cues during action monitoring (see Chapters 2 and 4)**.

Depression

Phenomenology

Although anxiety and depression may be seen as belonging to a shared continuum (with anxiety disorders sometimes evolving to depression) and strongly co-occur and covary with each other (Clark & Watson, 1991; Mineka, Watson, & Clark, 1998; Sufka et al., 2006), major depressive disorder (MDD) has a different phenomenology than anxiety. MDD is a syndrome that is characterized by persistent negative mood states, like fear, sadness and guilt, and also by anhedonia or a decrease in the ability to experience positive affect,

like feelings of joy, energy, enthusiasm, interest, alertness and self-confidence (Brown et al., 1998; Kring & Bachorowski, 1999; Watson et al., 1995). These strong emotional disturbances are accompanied by executive function deficits like difficulties in short-term and working memory (Rose & Ebmeier, 2006; Watts, 1985), attention and concentration (Beblo, Baumann, Wallesch, & Hermann, 1999; Fox, Russo, & Dutton, 2002) and cognitive control (Elliott et al., 1997). Moreover, research has also confirmed that these profound and pervasive emotional disturbances in depression are actually backed up by severe deficits regarding information processing in general. More specifically, several studies showed that depression is associated with cognitive biases towards negative information (De Raedt, Koster, & Joormann, 2010), mainly related to memory (Bradley, Mogg, & Williams, 1995) but less to attentional processes that are more selectively influenced by levels of anxiety (Mineka, Rafaeli, & Jovel, 2003; Mineka & Sutton, 1992; Mineka et al., 1998). Some authors have suggested that these cognitive impairments are actually related to core executive functioning problems, and more specifically to basic inhibitory deficits or a failure to disengage from negative stimuli (Fox et al., 2002; Koster, DeRaedt, Goeleven, Franck, & Crombez, 2005; Koster, Leyman, DeRaedt, & Crombez, 2006), which can therefore form the base of prolonged self-focused rumination (Gotlib & Joormann, 2010; Joormann, 2006), which is the tendency or style to think repetitively about the causes and consequences of negative or adverse life events (Nolen-Hoeksema, 2000; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). These profound cognitive and emotional disturbances that are usually running together with physical or more somatic disturbances, including sleep, appetite and libido, are diagnostic of depression (American Psychiatric Association - DSM-IV, 2000).

Neurobiology

Like it is often the case with brain diseases, depression cannot be related to a single or circumscribed dysfunctional brain area, but effects of depression on brain activity are probably diffuse and widespread, and best explained by a system-level or network account (Damasio, 1989; De Raedt & Koster, 2010;

Drevets & Raichle, 1998). Regions that are found to be most affected by depression are the frontal cortex, the hippocampus, the striatum, the ACC, limbic and paralimbic areas like the amygdala, thalamus, hippocampus, basal ganglia and anterior temporal lobes. Some of these regions (i.e. frontal cortex, the hippocampus, the striatum and limbic areas as the subgenual cingulate cortex) were found to be smaller (i.e. reduced volume) in depressed patients compared to healthy controls (Anand & Shekhar, 2003). The functional activation in some of these regions, together with other regions, was also found to be influenced by depression. Depression-related decreased activations were observed in "cognitive" control regions, such as the dlPFC and ACC (Anand & Shekhar, 2003; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Mayberg, 1997; Seminowicz et al., 2004), while increased activations were evidenced in other limbic and paralimbic regions (i.e. hippocampus, amygdala, thalamus, basal ganglia and anterior temporal lobes; Anand & Shekhar, 2003). Mayberg (1997) put forward the idea that a dysregulation between limbic and cortical areas might play a key role in depression (see also Drevets & Raichle, 1998). More specifically, she proposed that the "dorsal compartment", which encompasses in her view brain regions that are involved in attentional and cognitive processes affected by this illness (i.e. dlPFC, dACC, inferior parietal cortex and basal ganglia), does not interact properly anymore with the "ventral compartment", which includes paralimbic cortical, subcortical regions like the insula and subgenual cingulate, and brainstem, all of which are hypothesized to mediate somatic symptoms related to sleep, appetite or libido. According to Mayberg (1997), the reciprocal links between this dorsal and ventral compartment are assumed to be regulated or controlled by yet another region, namely the rACC which is found to be overactive in depressed individuals. Accordingly, various therapies targeting selectively this rACC for the treatment of depression have been proposed in the literature (Mayberg, 2009; Mayberg et al., 1997; Pizzagalli, 2011).

Error monitoring

At the behavioral level, depressed individuals show exaggerated reactions to errors or negative feedback on task performance (Beats et al., 1996; Elliott et

al., 1997; Elliott et al., 1996), an excessive concern or worry related to error commission (Enns & Cox, 1999; Shafran & Mansell, 2001), an increase in negative mood after perceived failures (Abela & D'Alessandro, 2002; Henriques & Leitenberg, 2002), difficulties in regulating failure-related thoughts following negative feedback (Conway et al., 1991), as well as a decreased accuracy following error commission (Holmes & Pizzagalli, 2008; Pizzagali et al., 2006). Thus, these behavioral results suggest an over sensitivity in depression (very much like in anxiety) to self-generated errors or negative outcome (feedback) regarding performance.

ERP studies corroborated these findings, even though mixed results were obtained. Some earlier ERP studies reported larger ERN/Ne amplitudes in MDD patients compared to healthy controls (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008; 2010), while other studies reported similar (Compton et al., 2008; Schrijvers et al., 2008; Schrijvers et al., 2009) or even smaller ERN/Ne amplitudes in MDD patients (Ruchsow et al., 2006; Ruchsow et al., 2004). Effects of depression on the CRN component are not always consistent either. Olvet et al. (2010) and Schrijvers et al. (2009) reported larger CRN amplitudes in depressed patients, but Holmes and Pizzagalli (2008; 2010) found comparable CRN amplitudes between depressed and control individuals. Likewise, discrepant findings have also been reported regarding possible amplitude variations of the Pe component as function of depression. While Chiu and Deldin (2007), Compton et al. (2008) and Holmes and Pizzagalli (2008) observed similar Pe amplitudes for controls and MDD patients, Schrijvers et al. (2008) and Schrijvers et al. (2009) reported smaller Pe amplitudes in depressed individuals.

With respect to the processing of external evaluation cues or feedback and its possible impairment in depression, no clear picture currently emerges in light of the existing literature. Tucker et al. (2003) reported a small differentiation at the level of the FRN component between positive and negative feedback (hence suggesting some performance monitoring based on the processing of external evaluative cues) in individuals who scored either low or high on a depression scale, whereas individuals who scored in the middle were characterized by a

larger FRN. Foti and Hajcak (2009) found smaller FRN amplitudes in depressed compared to control participants. By contrast, Santesso et al. (2008) investigated feedback processing in remitted depressed individuals and showed a larger differentiation between positive and negative feedback, compared to controls. However, Ruchsow et al. (2004) did not report any difference at the level of the FRN between positive and negative feedback processing in depressed patients.

To sum up, the picture emerges that possible alterations (in terms of neurophysiological or ERP effects) induced by depression during early action monitoring and error detection appear much less clear or consistent, than what has already been found by comparison for trait anxiety. Usually, high anxious individuals show increased ERN/Ne (and often CRN as well) components during the early monitoring of errors, while the subsequent Pe component is unchanged. No such (or any other consistent) pattern is seen in depression (Vaidyanathan et al., 2012). Accordingly, **another goal of the present doctoral dissertation was to address this question, and better characterize and systematically compare possible error-monitoring deficits at the level of the ERN/Ne, CRN and Pe components between sub clinical high anxious (see Chapters 2 and 4) vs. clinically depressed patients (Chapter 3).**

RESEARCH GOALS AND OVERVIEW OF THE FIVE EMPIRICAL CHAPTERS

The main goal of my dissertation was to gain insight into the possible links between negative affect and early error-monitoring brain processes, and more specifically, try to better understand the functional meaning of alterations seen during early error-monitoring processes in either anxiety or depression. Error monitoring is usually not trivial to explore in standard laboratory conditions, because response errors are by definition rare and deviant events, but also because human beings try hard to avoid making errors during standard interference or perceptual-decision making tasks, which somehow challenge their self-efficacy. To overcome this major problem, I adopted a common methodology and task setting across the different studies performed and reported in this dissertation.

The asset of this task is that many unwanted response errors (perceived as such) could be recorded in each and every participant, within a relatively short period of time. The experimental paradigm that I selected for this purpose included a speeded Go/noGo task that was previously validated in several groups of adult participants (Dhar & Pourtois, 2011; Koban, Pourtois, Vocat, & Vuilleumier, 2010; Pourtois, 2011; Vocat et al., 2008). This task requires participants to respond to a pre-defined target (i.e. Go stimulus) which is presented frequently throughout the experimental session (2/3 of trials) but to withhold responding when encountering a non-target (i.e. noGo stimulus), which is presented less frequently (1/3 of trials). Hence, using this task, one can measure the ability to exert inhibitory control on noGo stimuli. In other words, the inhibition of a pre-potent response tendency is investigated in this standard task (Miyake et al., 2000). In the speeded Go/noGo task selected in my empirical studies, visual stimuli consisted of simple geometric symbols, namely arrows, devoid of any affective or motivational value, enabling to explore "pure" effects of anxiety or depression on action- or error-monitoring brain processes. Each trial had always a similar temporal structure, as follows. First, a black arrow pointing either up or down was presented. Then, after a variable delay, this up- or down-arrow changed color, and became green most of the time. If the initial black arrow became green (i.e. Go stimulus), the participant was instructed to respond as fast as possible by pressing a pre-defined response key. However, occasionally, the initial arrow could turn blue (instead of green) or the in-plane orientation between the initial black arrow and subsequent green arrow was swapped. These two cases corresponded to noGo stimuli and participants were explicitly asked to not respond when encountering these specific instances. Hence, response errors occurred in this task when participants made key presses on these noGo stimuli, corresponding to False Alarms (FAs). Task instructions are therefore quite simple and swiftly understood by all participants, including anxious (see Chapter 2 and 4) and clinically depressed individuals (see Chapter 3).

To promote the occurrence of (many) FAs, I adapted a specific experimental procedure, based on the use of a response deadline. On each and every trial, the actual speed (Reaction Time – RT) for Go stimuli was calculated and compared against an arbitrary limit. This limit was determined individually, for each subject separately, and adjusted online throughout the course of the experiment to deal with unspecific effects of time or fatigue. Accordingly, besides the actual accuracy for each and every trial, the speed was assessed such that if the current RT was slower than this arbitrary limit (i.e. Slow Hits), participants received negative feedback, whereas if the current RT was faster than this limit (i.e. Fast Hits), a positive feedback was given. By emphasizing speed like that, participants adopt a specific response mode and respond fast or in a rather impulsive way, and therefore they are more likely to make FAs on noGo stimuli. However, any violation of task rules (i.e. reacting in front of a noGo stimulus), when occurring, is immediately obvious to participants, given the simplicity of these rules, enabling to study error-monitoring or -detection processes (see Chapter 2). Thus, this procedure allows to collect a high number of commission errors (consisting of FAs on noGo trials) in each and every participant, despite inter-individual differences in RTs, within a short period of time (~ 30 min), without inducing excessive frustration. This was an important pre-requisite to compute reliable error-related ERP waveforms, based on a substantial number of response errors, and eventually compare these error-related ERP waveforms to waveforms obtained for correct responses. Another added value of this procedure is that on each trial performance is evaluated by external evaluative feedback, hence making it possible to study not only internal action-monitoring processes (presumably occurring rapidly after the key press; see Chapter 2), but also external action-monitoring processes (occurring at the time of evaluative feedback delivery; see Chapter 4) using a uniform task. Given the updated speed limit used, participants were uncertain about their actual speed for a given trial, and hence they had to rely on and attend to these feedback stimuli in such a way to infer whether their actions were timely (fast hits/positive feedback) or not (slow hits/negative feedback).

Possible effects of negative affect (either trait anxiety or depression) on error monitoring were explored at the behavioral level (i.e. accuracy and RT data collected during the task), but also, and more importantly, using concurrent high-density EEG measurements. More specifically, in Chapters 2, 3, 4 and 6, continuous EEG was acquired at 512 Hz using 128 channels distributed evenly over the scalp surface, while participants performed the Go/noGo task outlined here above. I performed standard peak analyses following standard practice (Picton et al., 2000), looking at amplitude changes of specific action or error-monitoring ERP components (including the ERN/Ne, CRN, and Pe components for internal monitoring effects; but the FRN component for external monitoring effects). I also carried out additional topographical analyses to gain insight into the configuration of the putative brain generators underlying these specific ERP components (Pourtois et al., 2008).

Levels of (sub clinical) trait anxiety were established in psychology students at Ghent University by means of the validated Dutch version of the Spielberger State-Trait Anxiety Inventory – Trait Version (STAI-T; Spielberger, 1983 translated by Defares, van der Ploeg, & Spielberger, 1979). This questionnaire consists of 20 items referring to symptoms of anxiety like tension, nervousness, worry and apprehension, and participants have to rate the level to which they experience these symptoms on a scale from 1 to 4. I also verified, using the same inventory (State Version), whether levels of state anxiety accounted for changes observed at the level of the error-related ERP components recorded in my studies. Ambulatory clinically depressed patients (Chapter 3) were recruited from a local psychiatric clinic in Ghent and the severity of their current MDD episode was assessed using the Hamilton Rating Scale for Depression (HAM-D; Hamilton, 1960), which is a multiple choice questionnaire consisting of 17 items in which the severity of symptoms observed in depression such as negative mood, insomnia, agitation, anxiety and weight loss, is rated. The Mini-International Neuropsychiatric Interview (MINI), a structured clinical interview (Sheehan et al., 1998), was also used to corroborate the inclusion of clinically depressed patients in my study (see Chapter 3).

In this doctoral dissertation, I mainly addressed three different research questions, informing about the existing links between negative affect and error-monitoring brain processes. First, using the speeded Go/noGo task described here above, I compared the behavior and ERP components to error commission of a group of low vs. high (sub clinical) anxious psychology students (Chapter 2). I focused on early error-related brain activities, namely the ERN/Ne and Pe components. The goal of this ERP study was to better characterize possible changes induced by trait anxiety on these early error-monitoring brain functions, using not only standard peak analyses (Picton et al., 2000), but also complementing methods informing about the putative neural sources giving rise to these ERP components, and their possible sensitivity to changes in levels of trait anxiety (Pourtois et al., 2008). The exact same method and logic was used in Chapter 3, where I compared error-monitoring brain processes in non-depressed healthy participants vs. clinically depressed patients. Accordingly, **these two studies (chapters 2 and 3) enabled to investigate whether sub clinical trait anxiety and MDD, which share many characteristics but are also dissociable, influenced early error-monitoring processes in a similar way or not. This was the first main research question investigated in my doctoral research, addressed in Chapters 2 and 3.** Next, I investigated, still using ERP methods, effects of trait anxiety on the processing of external evaluative feedback, focusing therefore on the FRN component (Chapter 4). **The main aim of this study was to examine whether sub clinical trait anxiety would alter performance-monitoring processes when these generative processes are no longer based on internal cues (see Chapter 2), but rather on external evaluative feedback provided to participants. This specific question was the second main issue addressed in my doctoral dissertation (Chapters 2 and 4)**. Whereas studies reported in Chapters 2 to 4 informed about the possible locus and the expression of anxiety- or depression-dependent alterations during early error monitoring (or feedback processing), they do not shed light on the actual functional meaning of these changes. **What does an enhanced ERN/Ne component in anxiety truly reflect?** Therefore, I designed and validated a new method (Chapter 5) to infer, indirectly at the behavioral level, the actual affective meaning of self-generated

actions performed during the Go/noGo task. The rationale (Chapter 5) was to test, using a novel action-word evaluative priming sequence, whether unwanted response errors were indeed "automatically" marked as negative events, and moreover assess whether this automatic appraisal of self-generated actions may be impaired in sub clinical trait anxiety. By doing so, I aimed at providing a plausible theoretical account for the modulation of early error-monitoring brain processes by trait anxiety (and depression to a lesser extent). Finally, I also delineated the actual electrophysiological time-course of the evaluative priming effects reported in chapter 5, using ERP methods (Chapter 6). **Hence, the third main research question explored in this work (Chapters 5 and 6) concerned the actual affective or motivational value of self-generated actions, including response errors, with the aim to eventually better understand possible disturbances observed at the neurophysiological level during early error monitoring in anxious or depressed individuals (see Chapters 2 to 4).**

CHAPTER 2: ANXIETY DOES NOT ONLY INCREASE, BUT ALSO ALTERS EARLY ERROR-MONITORING FUNCTIONS[1]

"Anxiety has profound influences on a wide range of cognitive processes, including action monitoring. Event-Related brain Potential (ERP) studies have shown that anxiety can boost early error-detection mechanisms, as reflected by an enhanced Error-Related Negativity (ERN/Ne) following errors in high anxious, compared to low anxious participants. This observation is consistent with the assumption of a gain control mechanism exerted by anxiety onto error-related brain responses within the dorsal Anterior Cingulate Cortex (dACC). However, whether anxiety simply enhances or rather alters early error-detection mechanisms remain unsolved. In this study, we compared the performance of low vs. high trait anxious participants during a Go/noGo task while high-density EEG was recorded. The two groups showed comparable behavioral performance, although levels of state anxiety increased following the task for high anxious participants only. ERP results confirmed that the ERN/Ne to errors was enhanced for high, relative to low anxious participants. However, complementary topographic analyses revealed that the scalp map of the ERN/Ne was not identical between the two groups, suggesting that anxiety did not merely increase early error-detection mechanisms, but also led to a qualitative change in the early appraisal of errors. Inverse solution results confirmed a shift within the ACC for the localization of neural generators underlying the ERN/Ne scalp map in high anxious participants, corroborating the assumption of an early effect of anxiety on early error-monitoring functions. These results shed new light on the dynamic interplay between anxiety and error-monitoring functions in the human brain."

[1] Aarts, K., & Pourtois, G. (2010). Anxiety not only increases, but also alters early error-monitoring functions. Cognitive, Affective, and Behavioral Neuroscience, 10(4), 479-492.

INTRODUCTION

The early and efficient detection of a mismatch between the actual and expected or desired motor action provides human organisms with adaptive and flexible behaviors, since error detection typically leads to learning and the implementation of remedial action (Holroyd & Coles, 2002; Rabbitt, 1966). Converging neuroscience evidence has revealed that the medial frontal cortex, and the dorsal Anterior Cingulate Cortex (dACC) is primarily involved in the early detection of errors, or more generally conflicts, whereas lateral frontal or prefrontal regions are implicated in behavioral adjustments following errors (Carter et al., 1998; MacDonald, Cohen, Stenger, & Carter, 2000; Ridderinkhof, Nieuwenhuis, & Braver, 2007; van Veen & Carter, 2006) . In this view, the medial frontal cortex (and dACC) provides important cognitive control mechanisms, including early error detection. However, errors are also typically rare, deviant and negative events. Hence, errors also call for affective control processes (Hajcak & Foti, 2008; Ochsner & Gross, 2005), beyond their ubiquitous effects on cognitive control processes. Nonetheless, much less is known about the nature and extent of affective influences onto early error-detection processes, in comparison to a wealth of studies that have primarily focused on cognitive control effects (Ridderinkhof et al., 2007).

Event-Related brain Potential (ERP) studies have largely contributed to gain new insight into the time-course and neural bases of cognitive control mechanisms, including error detection (Taylor, Stern, & Gehring, 2007). The commission of errors is typically associated with the generation of a reliable negative ERP component early on following the onset of incorrect motor responses, the Error-Related Negativity (ERN/Ne) (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring, Coles, Meyer, & Donchin, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN/Ne component peaks at fronto-central electrodes along the midline (FCz or Fz electrode position), roughly 0 - 100 ms after (incorrect) response onset, and is thought to be primarily generated within the dACC

(Debener, Ullsperger, Fiehler, von Cramon, & Engel, 2005; Dehaene, Posner, & Tucker, 1994; Herrmann, Rommler, Ehlis, Heidrich, & Fallgatter, 2004; O'Connell et al., 2007; Vocat, Pourtois, & Vuilleumier, 2008). Hence, the ERN/Ne occurs too early to reflect sensorimotor or proprioceptive feedback, but instead, it is assumed to reflect the automatic and rapid detection of a mismatch between the actual and expected or desired motor action (Falkenstein et al., 2000; Nieuwenhuis et al., 2001). Following the ERN/Ne, the Error Positivity (Pe) is usually elicited in response to incorrect responses (Falkenstein et al., 2000; Nieuwenhuis et al., 2001). The Pe component is a broad positive deflection resembling the P3 component, peaking over the vertex (or more posterior parietal scalp positions along the midline, such as PZ) roughly 150 - 300 ms after (incorrect) response onset, with neural generators involving more posterior cingulate regions (Herrmann et al., 2004; O'Connell et al., 2007).

Although the ERN/Ne is usually described as reflecting cognitive or learning processes (Bernstein, Scheffers, & Coles, 1995; Coles, Scheffers, & Holroyd, 2001; Falkenstein et al., 1991), several ERP studies showed that the ERN/Ne also captures variations in affect or motivation. This observation is consistent with the assumption that errors do not only provide important learning or cognitive signals, but they also convey an important emotional significance (Bush, Luu, & Posner, 2000; Gehring & Willougby, 2002; Pailing & Segalowitz, 2004; Pourtois et al., 2010). For example, Hajcak, Moser, Yeung and Simons (2005) suggested that an error is primarily a motivationally salient event, as the ERN/Ne was significantly larger for errors related to high monetary value. More evidence on the relationship between affect and the ERN/Ne comes from studies looking at variations in trait affect. Several researchers found that individuals scoring high on trait anxiety and negative affect are characterized by an increased ERN/Ne (Boksem, Tops, Wester, Meijman, & Lorist, 2006; Hajcak, McDonald, & Simons, 2003a, 2004; Olvet & Hajcak, 2008). This increased sensitivity for errors in individuals with anxiety characteristics suggests that the ERN/Ne also somehow reflects an affective evaluation during error detection (Bush et al., 2000; Olvet & Hajcak, 2008).

Interestingly, research on anxiety-related differences in ERN/Ne has not been linked to the broader literature on cognitive control in anxiety. The cognitive literature in anxiety allows to deriving relatively specific predictions in this context. According to the processing efficiency theory (Eysenck & Calvo, 1992), trait anxiety influences the efficiency (rather than the amount or effectiveness) of cognitive performance. They claim that anxious individuals will not show performance decrements on most task as they recruit extra processing resources, which eventually hampers the amount of resources available for concurrent task processing. In this model, attentional control is the key mediating factor between anxiety and cognitive performance (Eysenck, Derakshan, Santos, & Calvo, 2007). This theory predicts that attention is more readily allocated to internal threatening stimuli (i.e. worrying thoughts) in high anxious subjects, reducing therefore the attentional focus on the current task demands. However, to maintain a standard level of performance, anxious subjects compensate for this reduced efficiency by increasing cognitive effort. This mechanism could potentially account for the fact that while an altered ERN/Ne is generally observed in high compared to low anxious subjects, no direct effect of anxiety on behavior (e.g. the number of errors), is usually evidenced (Hajcak et al., 2003a; Vocat et al., 2008). As our discussion of error detection suggests that errors are associated with cognitive as well as affective correlates, attentional control theory would predict that errors in high anxious individuals are not only associated with an increased ERN/Ne related to ACC activity but will also be related to a different pattern of neural activity in areas involved in emotion processing and cognitive control.

Brain imaging studies have confirmed that increased effort translates as enhanced activation in brain regions associated with cognitive control, including the dorsolateral or ventrolateral prefrontal cortex (DLPFC or VLPFC), and dACC (Cazalis et al., 2003; Donohue, Wendelken, & Bunge, 2008; Wagner, Maril, Bjork, & Schacter, 2001). Interestingly, anxiety was found to reduce activation in these cognitive control areas (Bishop, 2007; Bishop, Duncan, Brett, & Lawrence, 2004). Moreover, a reduced efficiency might actually result from a change in the

temporal recruitment of these cognitive control areas, as recently shown (Braver, Gray, & Burgess, 2007; Fales et al., 2008). These findings therefore suggest that anxiety may alter the recruitment of cognitive control areas during task performance, and as a result, lead to a reduced processing efficiency. However, with respect to error-detection mechanisms (which is a crucial component of cognitive control), to our knowledge, no study has examined whether low and high anxious participants differ only in their reaction to errors (as primarily reflected by the size of the ERN/Ne component), or alternatively, also make use of different cognitive control, and more specific, error-detection brain networks during the early processing of these negative events.

The goal of this study was to address this question using a modern ERP topographic mapping technique (Murray, Brunet, & Michel, 2008; Pourtois, Delplanque, Michel, & Vuilleumier, 2008). More specifically, we aimed to test whether trait anxiety merely enhances early error-related brain activities, or alternatively, it may also alter the expression (and not only the strength) of these brain responses (as revealed by a topographic change of the ERN/Ne scalp map with anxiety), in keeping with the main prediction of the attentional control theory (Eysenck et al., 2007). We therefore compared, using high-density EEG, the electrophysiological responses to commission errors in two groups of healthy participants, differing only with respect to their subclinical level of trait anxiety. We used a speeded Go/noGo task, previously validated in a group of adult participants (Vocat et al., 2008). The added value of this task is that it enables to collect a high number of commission errors [consisting of False Alarms (FAs) on noGo trials] in each participant, despite inter-individual differences in reaction times (RTs), within a short period of time (~30 min), and without inducing excessive frustration. This was an important pre-requisite to compute reliable ERP waveforms based on a substantial number of trials, including for errors. Furthermore, neutral stimuli (i.e. colored arrows) were used during this task, in such a way that electrophysiological responses to errors committed with neutral stimuli could be compared between the two groups, and a relatively pure modulation of trait anxiety on these brain responses could be eventually

assessed. Based on the evidence reviewed above, we predicted that behavior would not differ between low and high anxious subjects and that high anxious participants would show larger ERN/Ne for errors than low anxious participants (Olvet & Hajcak, 2008). We also surmise a substantial change in the configuration of the electric field of the ERN/Ne for high anxious relative to low anxious individuals, suggesting the involvement of partly distinct neural generators, in agreement with the processing efficiency theory (see Eysenck et al., 2007; Fales et al., 2008). This would indicate that high anxious participants do not only respond stronger to their own response errors, but that they likely recruit a different network of cognitive control brain regions during this process, relative to low anxious individuals.

METHOD

Participants

Four hundred and seventy nine first year University psychology students were asked to fill out several questionnaires, including measures of anxiety, in exchange of course credits. Within this large sample of psychology students, individuals scoring within the lowest quartile (low anxious) or the highest quartile (high anxious) of the distribution of trait anxiety scores, were invited to participate in the ERP experiment, in such a way to obtain two homogenous groups differing with respect to their levels of trait anxiety. A total of 32 undergraduate psychology students eventually participated in this experiment in exchange of 20 Euro payment. Participants had normal or corrected-to-normal vision. Trait anxiety levels of participants were primarily screened using a validated Dutch version of the Spielberger State-Trait Inventory – Trait Version (STAI-T; Spielberger, 1983 translated by Defares, van der Ploeg, & Spielberger, 1979). Based on these trait anxiety scores, two groups of equal size were formed. Sixteen participants (2 male; 3 left-handed) with a mean age of 19.06 years (*SEM* = 0.39) were assigned to the high trait anxious group (*M* = 51.50, *SEM.* = 0.99, *Range*: 45 - 58), the 16 remaining participants (2 male; 2 left-handed) with a mean age of 18.56 (*SEM* = 0.26) to the low trait anxious group (*M* = 29.69, *SEM* =

0.80, *Range*: 25 - 36). The study was approved by the local university ethical committee.

Speeded Go/noGo task

We used a modified version of a speeded Go/noGo task previously validated in a group of healthy participants (Figure 1; Vocat et al., 2008). Visual stimuli were shown on a 17-inch LCD screen. They consisted of an arrow (11.4° x 0.05° of visual angle at a 60 cm viewing distance) that was presented in the center of the screen on a white background. Each trial started with a blank screen that lasted for 1000 ms. Then, a black arrow (i.e. cue), either oriented up or down, was presented. After a variable interval ranging from 1000 up to 2000 ms, the black arrow became either green (i.e. target) or turquoise while its orientation could either remain identical or shift in the opposite direction. Participants were asked to perform a speeded color plus orientation discrimination task. When the black arrow turned green and the orientation remained unchanged, participants were instructed to press the space bar as fast as possible with a pre-defined finger of their dominant hand (Go trials; see Figure 1A). However, participants had to withhold responding when either the arrow became green but changed orientation, or when the arrow became turquoise and kept its initial orientation, enabling two types of noGo trials (based either on the orientation or color; see Figure 1B). For noGo trials, this color arrow remained on the screen for a maximum duration of 1000 ms. Instructions emphasized both speed and accuracy.

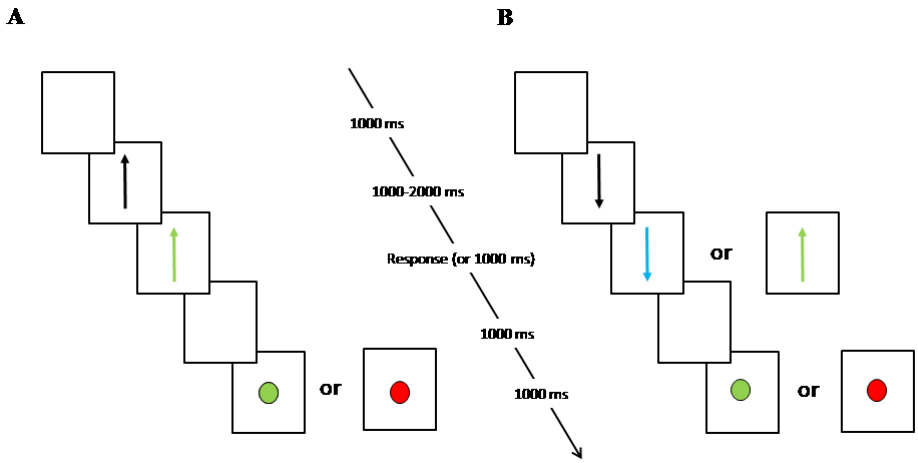

Figure 1. Stimuli and task. Participants had to respond by pressing the spacebar as fast as possible only when the arrow became green and kept its initial orientation (A), but not otherwise (B).

We used an online adaptive algorithm to set up a limit for "correct"/fast RT (i.e. deadline procedure). The rationale of this procedure was to facilitate the occurrence of fast decisions, and hence the occasional making of errors on noGo trials. Participants had to respond fast on Go trials, but their performance actually depended on this strict time limit, updated on a trial-by-trial basis. At the beginning of the experiment, the RT limit was set to 300 ms (this cutoff was determined based on previous pilot testing). This limit was adjusted online as a function of the immediately preceding trial history, more specifically as the sum of current and previous RT divided by two. This procedure was found to be particularly efficient to produce a high number of commission errors within a short period of time. For any given Go trial, the actual RT was always compared with the RT on the previous Go trial. If the current RT was slower than the previous RT, the participant received a negative feedback (red dot). If the RT was faster than the previous one, a positive feedback (green dot) was presented. This procedure ensured obtaining many FAs on noGo trials despite fluctuations in speed on a trial-by-trial basis, because this arbitrary cut-off for correct responses was updated and adjusted online after each trial, separately for each participant.

The experiment consisted of 20 practice trials and 360 test trials. The test trials were divided into 6 blocks of 60 trials each (40 Go trials and 10 noGo trials

of each type). Trial presentation was randomized within blocks. After every block, the experimenter emphasized the importance of speed as well as accuracy in this task. Stimulus presentation and response recording were controlled using E-prime software (V2.0., http://www.pstnet.com/products/e-prime/).

Questionnaires

The 32 participants filled out the Dutch version of the STAI-T (Defares et al., 1979; Spielberger, 1983) and their scores served as a basis to form the low and high anxious group. Because trait anxiety is typically related to punishment sensitivity, participants also completed the Dutch version of the Behavioral Inhibition Sensitivity (BIS)/Behavioral Activation Sensitivity (BAS) scales (Carver & White, 1994; Franken, Muris, & Rassin, 2005). Importantly, we also measured levels of state anxiety of these 32 participants, both before and after the Go/noGo task, using the STAI-S.

EEG acquisition and analysis

Continuous EEG was acquired at 512 Hz using a 128-channel (pin-type) Biosemi Active Two system (http://www.biosemi.com) referenced to the CMS-DRL ground. ERPs of interest were computed offline following a standard sequence of data transformations (Picton et al., 2000): (1) -500/+1000 segmentation around the motor response, (2) pre-response interval baseline correction (from -500 ms to motor response), (3) vertical ocular correction for blinks (Gratton, Coles, & Donchin, 1983) using the difference amplitude of two electrodes attached above and below the left eye, (4) artifact rejection (*M* = -75/+75, *SEM* = 2.0 amplitude scale (µV) across participants; no significant difference between low anxious (*M* = 76.56, *SEM* = 2.84) and high anxious participants (*M* = 72.5, *SEM* = 2.81) was observed, $t(30) = 1.02$, $p > .10$), (5) averaging of trials for each of the four experimental conditions (Fast Hits, Slow Hits, Color FAs and Orientation FAs), and (6) low pass digital filtering of the individual average data (30 Hz).

We primarily focused on two well-documented error-related ERP components following motor execution (Falkenstein et al., 2000), on the ERN/Ne,

with a maximum negative amplitude over fronto-central electrodes along the midline (electrode FCz) early on following motor execution (0 - 100 ms post-response onset), immediately followed by the Pe component (150 - 300 ms post-response onset), with a maximum positive amplitude over more posterior and central locations along the midline (electrode Cz). Hence, we performed a conventional peak analysis for each of these two error-related ERP deflections (Picton et al., 2000). For each ERP component and each condition separately, we calculated the area under the curve, during the 30 - 60 ms interval post-response onset at electrode FCz for the ERN/Ne amplitude, and during the 180 - 270 ms interval post-response onset at electrode Cz for the Pe component. The selection of these two specific scalp locations (and time window) was based on the topographic properties of the present dataset, as well as based on converging results obtained in previous ERP studies for these two electrode positions (Dehaene et al., 1994; Gehring et al., 1990; Hajcak et al., 2003a). Statistical analyses were performed on the mean amplitude of each area using a 2 (accuracy) x 2 (anxiety) repeated measures ANOVA, with a significance alpha cutoff set to $p < .05$.

Topography

In order to capture more global ERP differences between low and high anxious individuals during the detection of errors, a detailed topographic mapping analysis of the ERP data was next performed, following a conventional data-analysis scheme (Michel, Seeck, & Landis, 1999; Michel et al., 2001; Murray et al., 2008; Pourtois, Dan, Grandjean, Sander, & Vuilleumier, 2005; Pourtois et al., 2008; Pourtois, Thut, Grave de Peralta, Michel, & Vuilleumier, 2005).

To precisely characterize topographic modulations over time and conditions, we applied a pattern or spatial cluster analysis procedure. The pattern analysis efficiently summarizes ERP data by a limited number of field configurations, previously referred to as functional microstates (Lehmann & Skrandies, 1980; Michel et al., 1999). Here, we performed a topographic pattern analysis on group-averaged data from -150 ms until 450 ms after response onset

(300 consecutive time frames at 512 Hz sampling rate), using a standard cluster (or spatio-temporal segmentation) method (K-means; Pascual-Marqui et al., 1995) and then fitted the segmentation results back to individual data for subsequent statistical testing. The rationale and basic principles of this temporal segmentation method have been extensively described elsewhere (Michel et al., 1999; Murray et al., 2008). The spatio-temporal segmentation algorithm is derived from spatial cluster analysis (Pascual-Marqui, Michel, & Lehmann, 1995) and allows the identification of the most dominant scalp topographies appearing in the group-averaged ERPs of each condition and over time, while minimizing the biases for the selection of time-frames or electrodes of interest. Importantly, this procedure allows identifying dominant scalp topographies, irrespective of (local or global) changes in amplitude (Michel et al., 1999; Murray et al., 2008). The optimal number of topographic maps explaining the whole data set is determined objectively using both cross validation (Pascual-Marqui et al., 1995) and Krzanowski-Lai (Tibshirani, Walther, & Hastie, 2001) criteria. The dominant scalp topographies (identified in the group-averaged data) are then fitted to the ERPs of each individual subject using spatial fitting procedures to quantitatively determine their representation across subjects and conditions. This procedure thus provides fine-grained quantitative values, such as the duration of a specific topographic map or its Global Explained Variance (GEV, or goodness of fit), which are critical indices of the significance of a given topography, not available otherwise in a classical component analysis (Picton et al., 2000). GEV represents the sum of the explained variance weighted by the Global Field Power (GFP) at each moment in time. GEV was entered in repeated-measure analyses of variance (ANOVA) with two within-subject factors, accuracy (hits vs. errors) and map configuration (i.e. the electric field distributions previously identified by the spatial cluster analysis), and anxiety (high vs. low anxious participants) as between-subject factor. These analyses were carried out using CARTOOL software (Version 3.34; developed by D. Brunet, Functional Brain Mapping Laboratory, Geneva, Switzerland).

Source localization

Finally, to estimate the likely neural sources underlying the electrical field configurations identified by the previous analyses, we used a specific distributed linear inverse solution, namely standardized low-resolution brain electromagnetic tomography (sLORETA, Pascual-Marqui, 2002). sLORETA is based on the neurophysiological assumption of coherent co activation of neighboring cortical areas (known to have highly synchronized activity, see Silva, Amitai, & Connors, 1991) and, accordingly, it computes the "smoothest" of all possible activity distributions (i.e. no a-priori assumption is made on the number and locations of the sources). Mathematical validation of this distributed source localization technique has been recently demonstrated (Sekihara, Sahani, & Nagarajan, 2005). sLORETA solutions are computed within a three-shell spherical head model co-registered to the MNI152 template (Mazziotta et al., 2001). The source locations were therefore given as (x, y, z) coordinates (x from left to right; y from posterior to anterior; z from inferior to superior). sLORETA estimates the 3-dimensional intracerebral current density distribution in 6239 voxels (5 mm resolution), each voxel containing an equivalent current dipole. This 3-dimensional solution space, in which the inverse problem is solved, is restricted to the cortical gray matter (and hippocampus). The head model for the inverse solution uses the electric potential lead field computed with a boundary element method applied to the MNI152 template (Fuchs, Kastner, Wagner, Hawes, & Ebersole, 2002). Scalp electrode coordinates on the MNI brain are derived from the international 5% system (Jurcak, Tsuzuki, & Dan, 2007). The calculation of all reconstruction parameters was based on the computed common average reference. sLORETA units were scaled to ampere per square meter (A/m^2).

RESULTS

Behavioral results

RTs faster than 150 ms (*M* = 0.83%) and slower than 500 ms (*M* = 1.18%) were removed from the subsequent analyses. The number of excluded trials did

not differ between groups, $t(30) = 0.52$, $p > .10$. Color and orientation FAs were collapsed together (error condition), as there was no significant difference between the two error types. Likewise, fast hits and slow hits were collapsed and treated as a single condition (hit condition). Accuracy and RT data are presented in Table 1. The selected task turned out to be efficient in inducing a high number of unavoidable errors, consisting of FAs on noGo trials. Error rates did not differ between the low anxious ($M = 38.81$, $SEM = 3.84$) and the high anxious group ($M = 46.56$, $SEM = 3.98$), $t(30) = 1.40$, $p > .10$. As expected, participants were quicker for FAs than for hits, $F(1,30) = 296.58$, $p < .001$. However, the speed did not differ between low and high anxious individuals, $F < 1$, and the interaction between accuracy and anxiety did not reach statistical significance, $F < 1$. Moreover, no group difference in efficiency (computed as the ratio between accuracy and speed; see Stoeber & Eysenck, 2008 for exact formula), was observed, $t(30) = 0.84$, $p > .10$. These results suggest a comparable behavioral performance in these two groups.

Table 1

Behavioural results during the Go/noGo task, separately for low and high anxious participants

	RT (ms)		Accuracy (number)	
	Errors	Hits	Errors	Hits
Anxiety	*M*	*M*	*M*	*M*
	SEM	*SEM*	*SEM*	*SEM*
Low	264.99	298.67	38.81	237.81
	4.61	4.04	3.84	0.74
High	261.08	292.44	46.56	238.50
	4.93	4.68	3.98	0.62

During the Go/noGo task, a classical post-error slowing effect was observed (Laming, 1979; Rabbitt, 1966). Consistent with a systematic adaptation following errors, RTs were reliably slower for hits immediately following errors ($M = 301.31$, $SEM = 4.18$), as compared with hits following another hit ($M =$

292.52, *SEM* = 3.17), $F(1,30) = 7.46$, $p < .05$. The magnitude of the post-error slowing effect did not differ between groups, $F < 1$.

Questionnaires

As expected, the level of state anxiety before the task differed significantly between the two groups (low trait anxiety: *M* = 31.62, *SEM* = 1.11, *Range*: 25 - 42; high trait anxiety: *M* = 38.12, *SEM* = 1.56, *Range*: 31 – 51), $t(30) = -3.39$, $p < .01$. After the Go/noGo task, this level of state anxiety reliably increased, $F(1, 30) = 5.20$, $p < .05$, although low trait anxious individuals had still a lower level of state anxiety (*M* = 33.50, *SEM* = 2.17) than high trait anxious individuals (*M* = 41.69, *SEM* = 1.56), $t(30) = 3.06$, $p < .01$. Post-hoc comparisons revealed that this increase of state anxiety after, relative to before, the Go/noGo task, was only significant in high anxious participants, $t(15) = 2.11$, $p = .05$, but not in low anxious participants, $t(15) = 1.11$, $p > .10$ (Table 2). This result suggests a differential influence of the speeded Go/noGo task on subjective levels of state anxiety in low vs. high anxious participants.

Table 2

State anxiety scores (STAI-S) before and after performing the speeded Go/noGo task

	State anxiety	
Trait anxiety	Before *M (SEM)*	After *M (SEM)*
Low	31.62 (1.11)	33.50 (2.17)
High	36.56 (1.00)	41.44 (1.48)

Moreover, the scores on the BIS/BAS further confirmed that the two groups differed significantly with respect to the trait related anxiety characteristic, punishment sensitivity. BIS scores were significantly higher in high trait anxious (*M* = 22.00, *SEM* = 0.67, *Range*: 18 - 26) than in low trait anxious subjects (*M* = 19.13, *SEM* = 0.49, *Range:* 16 - 22), $t(30) = -3.48$, $p < .01$. No significant difference between the low and high trait anxious group was evidenced for the BAS-scores (BAS-drive: low trait anxious: *M* = 11.69, *SEM* =

0.27, high trait anxious: *M* = 11.63, *SEM* = 0.43, *t*(30) = 0.12, *p* > .10; BAS-fun: low trait anxious: *M* = 11.63, *SEM* = 0.24, high trait anxious: *M* = 11.81, *SEM* = 0.46, *t*(30) = -0.36, *p* > .10; BAS-reward: low trait anxious: *M* = 15.19, *SEM* = 0.34, high trait anxious: *M* = 15.56, *SEM* = .44, *t*(30) = -0.67, *p* > .10).

ERP results

Consistent with many previous ERP studies (Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1993; Hajcak et al., 2004; Nieuwenhuis et al., 2001), we recorded two distinct and conspicuous error-related ERP components following motor execution, which have been previously associated with error-detection brain mechanisms (i.e. the ERN/Ne and the Pe). During the speeded Go/noGo task, the commission of errors was unambiguously associated with the generation of these two well-characterized error-related ERP components (Figure 2).

ERN/Ne

When participants made errors, there was a clear sharp negative deflection that peaked roughly 40-50 ms post-response onset, with a maximum amplitude at fronto-central electrodes along the midline, including FCz (Figure 2). These electrophysiological properties are consistent with the ERN/Ne. Consistent with previous ERP studies (Falkenstein et al., 1991; Gehring et al., 1993), the amplitude of the ERN/Ne was reliably larger for errors (*M* = -3.11, *SEM* = 0.59), relative to hits (*M* = -1.62, *SEM* = 0.52), *F*(1,30) = 22.02, *p* < .001. An ANOVA performed on the amplitude values of the ERN/Ne, as measured at the standard electrode FCz, disclosed a near-significant interaction between anxiety and accuracy, *F*(1,30) = 3.53, *p* = .07. Compared to hits, errors elicited a larger ERN/Ne component in high anxious participants, *t*(15) = 4.61, *p* < .001 (Figure 2E), than in low anxious participants, *t*(15) = 2.00, *p* = .06 (Figure 2B). However, a direct comparison of the ERN/Ne between high anxious (*M* = -3.95, *SEM* = 0.65) and low anxious participants (*M* = -2.27, *SEM* = 0.97) did not reach statistical significance, *t*(30) = 1.46, *p* > .10. Likewise, for hits, the early negativity (i.e. the Correct Related Negativity - CRN; Burle, Roger, Allain, Vidal, & Hasbrouckq, 2008; Coles et al., 2001; Vidal, Hasbrouckq, Grapperon, & Bonnet, 2000), was

comparable across the two anxiety groups (low anxious: *M* = -1.37, *SEM* = 0.83; high anxious: *M* = -1.86, *SEM* = 0.63), $t(30) = -0.54$, $p > .10$. Note that because our speeded Go/noGo task was quite demanding and uncertainty about accuracy (at the time of motor execution) was presumably equally high for errors and hits, it was not surprising to find a large CRN component for correct hits in this study (see also Pailing & Segalowitz, 2004a). Importantly, the CRN component was still significantly smaller in amplitude than the ERN/Ne in both low anxious ($p = .06$) and high anxious ($p < .001$) participants. Several authors already pointed out the electrophysiological similarities between the ERN/Ne and the CRN (Allain, Carbonnell, Falkenstein, Burle, & Vidal, 2004; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal et al., 2000). These authors argued that the ERN/Ne (and CRN) might reflect either a more general comparison process (active after both errors and correct responses) or an emotional/arousal reaction (instead of an error-detection process per se).

Pe

For errors, the ERN/Ne was immediately followed by a large positive potential, with maximum amplitude over more posterior scalp positions, including Cz. This positive component was strongly attenuated for correct hits (Figure 2). These electrophysiological properties are compatible with the error-related Pe component (Falkenstein et al., 2000; Ridderinkhof, Ramautar, & Wijnen, 2009).

FAs on noGo trials elicited a large Pe, relative to correct hits (Figure 2). However, this accuracy effect at the level of the Pe component was similar for low and high anxious participants, unlike what was found for the ERN/Ne. Statistical analyses confirmed these observations. An ANOVA performed on the mean amplitude of the Pe recorded at electrode Cz revealed a main effect of accuracy, $F(1,30) = 146.29$, $p < .001$, indicating a much larger Pe component for errors (*M* = 13.95, *SEM* = 1.31) than correct hits (*M* = 4.90, *SEM* = 1.24). This significant accuracy effect was not influenced by trait anxiety (Figure 2CF), $F < 1$.

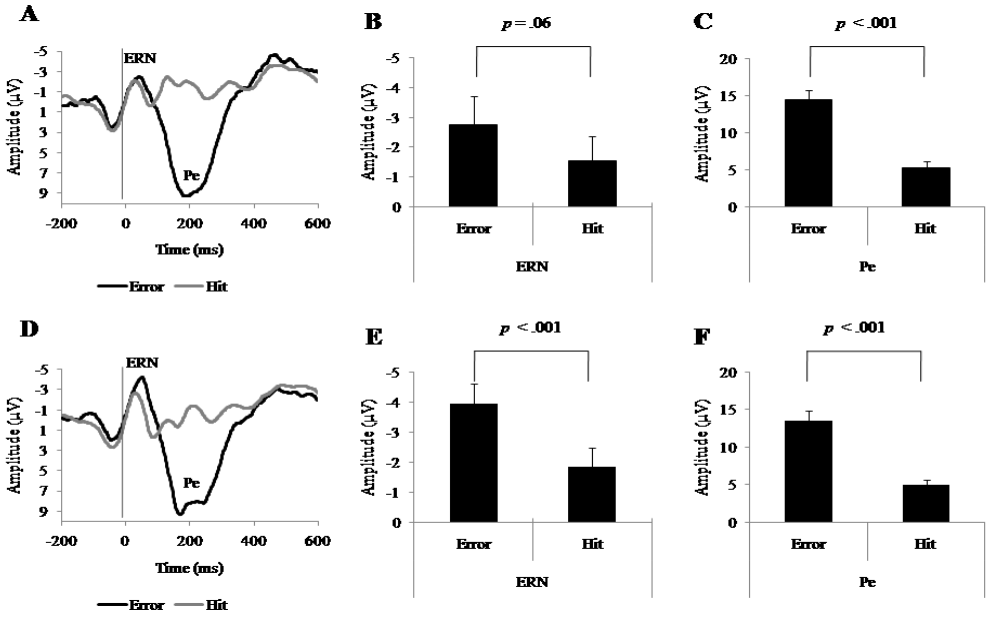

Figure 2. ERP results. (A) Grand average ERP waveforms (electrode FCz) for low anxious participants. (B) Mean amplitude (μV) ± 1 standard error of the mean of the ERN/Ne for errors and hits in low anxious participants. (C) Mean amplitude (μV) ± 1 standard error of the mean of the Pe for errors and hits in low anxious participants. (D) Grand average ERP waveforms (electrode FCz) for high anxious participants. (E) Mean amplitude (μV) ± 1 standard error of the mean of the ERN/Ne component for hits and errors in high anxious participants. (F) Mean amplitude (μV) ± 1 standard error of the mean of the Pe component for hits and errors in high anxious participants.

Results of topographic analyses

Following standard practice (Michel et al., 1999; Murray et al., 2008; Pourtois et al., 2008), the topographic segmentation analysis was first performed using a broad temporal window, starting 150 ms before response onset and ending 450 ms after response onset (i.e. 300 consecutive time frames, corresponding to 600 ms), encompassing the two main error-related ERP components (ERN/Ne and Pe). A solution with 10 maps explained 97% of the variance. Remarkably, during the time interval corresponding the ERN/Ne and CRN component (~20-50 ms post-response onset), we found that the scalp distribution for errors had a different configuration for high anxious relative to low anxious participants, whereas the scalp distribution for correct hits was similar between these two groups (Figure 3). Hence, the scalp map corresponding the CRN, was similar between the two groups (Figure 3A; map#1),

while a differential distribution of the negative activity over fronto-central electrodes was evidenced between low and high anxious participants for errors (ERN/Ne). Clearly, the fronto-central negative activity associated with errors (ERN/Ne) showed a broader and more extended (pre)frontal distribution for high anxious participants (Figure 3C; map#3), relative to low anxious participants (Figure 3B; map#2), where this negative activity was clearly circumscribed to a few electrode positions, including FCz. This result showed therefore a change in the configuration of the electric field (topography), regardless of (local) variations in amplitude (ERPs, see Methods).

These observations were further verified by statistical analyses performed on the topographic data (i.e. the Global Explained Variance - GEV), obtained from the fitting procedure (Figure 3DEF). These three dominant scalp topographies (identified in the group-averaged data) were fitted to the ERPs of each individual subject during the time interval corresponding to the ERN/Ne and CRN to quantitatively determine their representation across subjects and conditions. Finally, we submitted these GEV values to a 3 (map) x 2 (anxiety) x 2 (accuracy) repeated measures ANOVA. This analysis revealed a significant three-way interaction, $F(2,60) = 3.05$, $p = .05$. An additional 2 (anxiety) x 2 (accuracy) ANOVA run for each map separately confirmed that map#1 (Figure 3D) was specific to correct hits but shared across the two groups (as revealed by a significant main effect of accuracy, $F(1,30) = 40.85$, $p < .001$, but no interaction with anxiety, $F < 1$), whereas maps#2 and 3 were specific to errors (Figure 3EF), though with a clear cut dissociation between the two groups for these two error-related scalp topographies. This first result is in line with a previous topographic mapping study showing that the ERN/Ne and CRN led to different scalp distributions (and not only a change in the electric field strength, see Vocat et al., 2008). More importantly, both for map#2 and map#3, the ANOVA disclosed a significant interaction between accuracy and anxiety, $F(1,30) = 6.70$, $p < .05$ and $F(1,30) = 5.18$, $p < .05$, for map #2 and map#3, respectively. For low anxious participants (Figure 3E), post-hoc paired t-tests showed that map#2 had a larger GEV for errors, relative to hits, $t(15) = 2.88$, $p = .01$, whereas such an effect was

not observed with map#3 in this group, $t(15) = -0.59$, $p > .10$. Symmetrically, for high anxious participants (Figure 3F), map#3 had a larger GEV for errors relative to hits, $t(15) = 2.89$, $p = .01$, whereas such an effect was absent with map#2 in this group, $t(15) = -0.35$, $p > .10$. These topographic mapping results therefore suggested a clear dissociation in the configuration of the electric field associated with errors (ERN/Ne scalp map) between low and high anxious participants. Note that this difference concerned the topography, but not the amplitude of the ERP signal. Because changes in the distribution of the electric field over the scalp surface (topography) necessarily denote alterations in the underlying configuration of intracranial generators (Lehmann & Skrandies, 1980; Michel et al., 2001), these results indicated that high anxious individuals may recruit a different network of brain regions early on following the occurrence of errors, compared to low anxious participants. This assumption was next verified, using a distributed source localization technique (sLORETA).

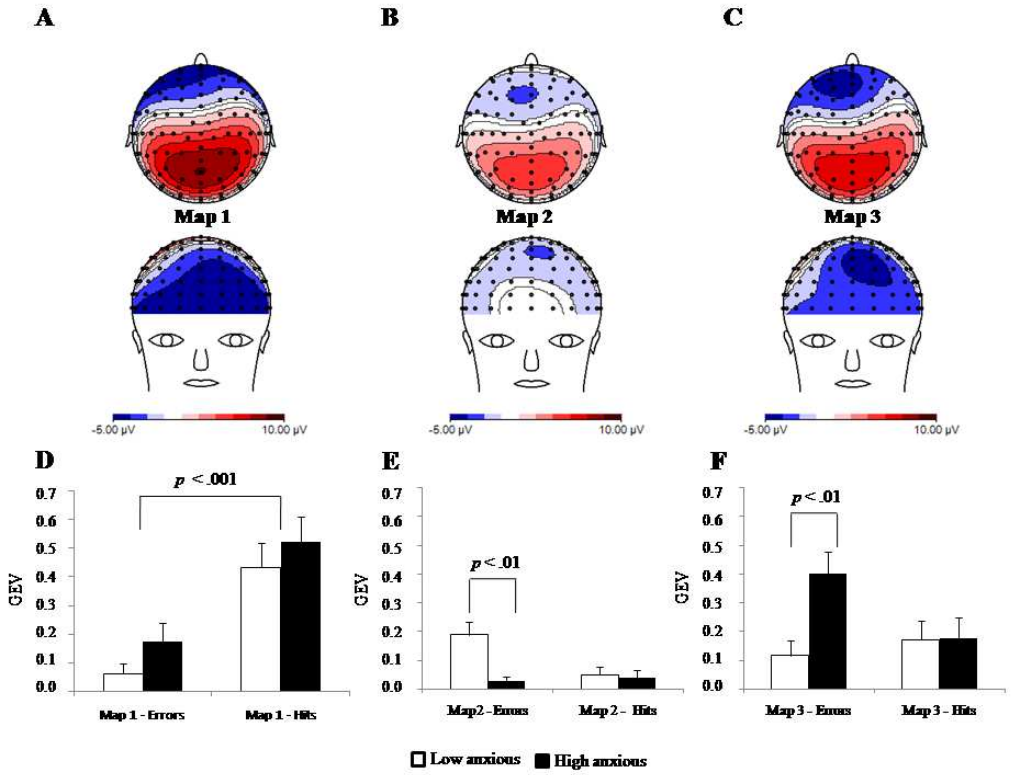

Figure 3. Dominant topographic maps (horizontal and frontal views) during the time interval corresponding either to the ERN/Ne or CRN (20 - 50 ms post response onset). (A) The scalp map of the CRN (map#1) was shared between the two groups. (B) The scalp map of the ERN/Ne for low anxious participants (map#2), showing a circumscribed negative activity around FCz electrode position. (C) The scalp map of the ERN/Ne for high anxious participants (map#3), showing a broader negative activity over frontal and prefrontal electrodes, compared to low anxious participants. (D) The CRN scalp map (map#1) showed a significant main effect of condition (explaining more variance for hits than errors, regardless of the experimental group). (E) The ERN/Ne scalp map for low anxious participants (map#2) was found to be specific for errors in this group. (F) Likewise, the ERN/Ne scalp map for high anxious participants (map#3) was found to be specific for errors in this group Error bars represent ± 1 standard error of the mean.

Source localization results

To gain insight into the putative configuration of the intracranial generators of these different topographic maps during the time interval corresponding to the ERN/Ne and CRN, we performed a source localization analysis, using sLORETA (Pascual-Marqui, 2002). For map#1 (corresponding to the CRN scalp map, which was clearly shared across the two groups and specific to correct hits), sLORETA disclosed a main generator/cluster within the posterior

parietal cortex, extending ventrally towards the posterior cingulate gyrus (Figure 4). A maximum activation was found in the precuneus (Brodmann area 7, with an extended activation toward Brodmann area 31) for this CRN scalp map (MNI coordinates: -10x, -80y, +50z). More importantly, sLORETA confirmed that the configuration of the intracranial generators underlying the ERN/Ne scalp map (errors) were roughly similar between low and high anxious participants and primarily involved medial frontal/dorsal anterior cingulate cortex (dACC) regions (Dehaene et al., 1994; Herrmann et al., 2004; O'Connell et al., 2007; Vocat et al., 2008), though with some substantial differences in the exact localization of these intracerebral generators within the dorsal ACC, as suggested by the topographic mapping analyses. While Vidal et al. (2000) found that the CRN and the ERN/Ne had the same neural generators (i.e. dACC), here we found in contrast that the neural generators of the CRN were different from those of the ERN/Ne, and they primarily involved more posterior cingulate regions, whereas the ERN/Ne was associated with neural activity originating from the dorsal ACC (Dehaene et al., 1994; Herrmann et al., 2004; O'Connell et al., 2007; Vocat et al., 2008). Therefore, our results support the hypothesis that errors did not simply amplify the activity of a generic action-monitoring system that would be equally engaged by correct and incorrect actions (Vidal et al., 2000), but rather, they rely on a specialized brain system localized within the dorsal ACC, with a significant modulation in this latter brain network as a function of levels of trait anxiety.

For low anxious participants (Figure 4B), the neural generators of the ERN/Ne were mainly localized within superior frontal gyrus/dorsal ACC (maximum: 5x, 10y, 60z; Brodmann areas 6 and 32), whereas for high anxious participants (Figure 4C), they also involved the superior frontal gyrus/dorsal ACC (maximum: -5x, 5y, 60z; Brodmann areas 6 and 24), but with a shift towards the front, compared to low anxious participants (Figure 4B). Importantly, a direct comparison between the two groups confirmed a different configuration of intracranial generators for the ERN/Ne (Figure 4D). Whereas the main generators of the ERN/Ne primarily involved the dorsal ACC for low anxious participants (Brodmann area 24), they were localized in a more anterior region for high

anxious participants, corresponding to the most anterior part of the ACC and medial frontal gyrus (Brodmann areas 32 and 10). In addition, another generator was found within the posterior cingulate gyrus/paracentral lobule for high anxious participants (Brodmann areas 31 and 5).

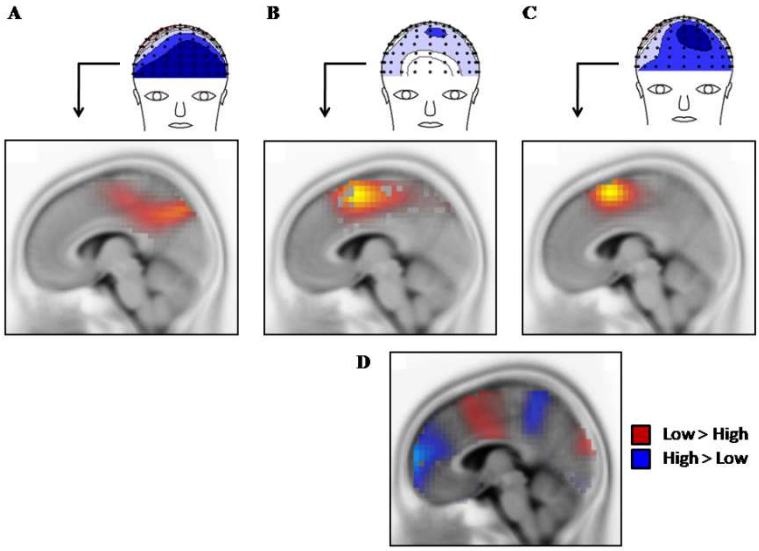

Figure 4. Source localization results, based on sLORETA. (A) Inverse solution for the CRN scalp map (map#1) (B) Inverse solution for the ERN/Ne scalp map for low anxious participants (map#2) (C) Inverse solution for the ERN/Ne scalp map for high anxious participants (map#3).

DISCUSSION

The goal of this study was to investigate the links between error-monitoring functions and individual differences in trait anxiety, primarily using ERP measurements. More specifically, we tested the prediction that the ERN/Ne, an early electrophysiological marker of error detection (Falkenstein et al., 2000), was not only relatively enhanced in high compared to low anxious individuals (Olvet & Hajcak, 2008) (quantitative account), but that trait anxiety could also alter the expression of this ERP component (qualitative account), consistent with the assumption of abnormal or altered early error-related brain reactions in anxious individuals. A number of important new results emerge from this study.

First, we found a comparable behavioral performance (and efficiency) for the two groups during the speeded and demanding Go/noGo task. High anxious participants did not commit more errors (nor were they slower or faster with

errors) than low anxious participants. This rules out the possibility that ERP differences observed between these two groups actually resulted from different behavioral effects during this Go/noGo task. In addition, the two groups showed a comparable classical post-error slowing effect (Laming, 1979; Rabbitt, 1966), suggesting preserved error monitoring and adaptation effects in these two groups. These results corroborate previous findings showing that behavioral measures of cognitive control abilities do not differ between low and high anxious subjects (Gehring et al., 2000; Hajcak et al., 2003a; Hajcak & Simons, 2002; Luu, Collins, & Tucker, 2000). However, we found that the speeded Go/noGo task had a differential influence on subjective levels of state anxiety in low and high anxious subjects. Only high anxious participants showed increased levels of state anxiety following the task (relative to a baseline state anxiety measure obtained before the task), compared to low anxious participants. This result suggests that the speeded Go/noGo task had a differential influence on the experience of negative affect in high vs. low anxious participants.

Importantly, ERP results confirmed a dissociation between the two groups. High anxious participants showed a larger difference between the ERN/Ne and the CRN, compared to low anxious participants, suggesting an increased sensitivity to errors in the former group. Noteworthy, complementary topographic analyses actually indicated that the ERN/Ne scalp map underwent a reliable configuration change for high anxious, relative to low anxious participants, although the CRN scalp map was shared across these two groups, suggesting that errors, but not correct hits, were differentially processed in these two groups. Clearly, the ERN/Ne scalp map had a different configuration for high anxious, relative to low anxious individuals, and concerned more anterior and (pre)frontal electrodes. This result suggests that these two groups used partly non-overlapping brain networks, early on following the onset of an incorrect response during performance monitoring. This conjecture was formally verified by the subsequent source localization analysis, which disclosed a shift of neural generators within the ACC for high, relative to low, anxious participants during

the time interval corresponding to the ERN/Ne. We discuss the implications of these new results in greater detail below.

Augmented ERN/Ne to errors in high anxious participants

The results of the conventional peak analysis were in line with previous ERP findings, that showed links between trait anxiety and the magnitude of the ERN/Ne. Earlier ERP studies already reported that the ERN/Ne to errors was increased during speeded RT tasks in participants with anxiety characteristics (Boksem et al., 2006; Endrass, Klawohn, Schuster, & Kathmann, 2008; Gehring et al., 2000; Hajcak et al., 2003a, 2004; Hajcak & Simons, 2002; Luu et al., 2000). While some previous studies also found an effect of trait anxiety on ERN/Ne amplitudes both for errors and hits (Hajcak et al., 2003a), here we found an interaction effect between accuracy (errors vs. hits) and anxiety (low vs. high anxious), precluding the possibility that trait anxiety affected equally the early processing of errors and hits during the speeded Go/noGo task. The results for the ERN/Ne (peak analysis) showed that the amplitude difference between errors and hits was larger in high, compared to low, anxious participants, suggesting a higher sensitivity to errors in high anxious participants, despite similar behavioral performances in these two groups. These new results are therefore consistent with the motivational significance theory of the ERN/Ne (Hajcak & Foti, 2008; Hajcak et al., 2003a; Luu et al., 2000), which predicts that this specific error-related ERP component mainly indexes the motivational significance of errors. Hence, participants like high anxious individuals, who are more sensitive to negative events and punishment, should also react more strongly to errors and as a corollary, present a (relatively) larger ERN/Ne to errors. Our new results for the ERN/Ne component support this assumption.

Alteration of early error-detection brain mechanisms in high anxious participants

While our new ERP results are overall compatible with the motivational significance theory of the ERN/Ne (Hajcak & Foti, 2008; Hajcak et al., 2003a; Luu et al., 2000), they also provide important new information as they show a

dissociation in the expression of the ERN/Ne between the two groups. This global difference concerning the distribution of the electric field (rather than its strength) could not be captured using a conventional peak analysis (Picton et al., 2000). Hence, not only the magnitude of the ERN/Ne was larger in high anxious participants compared to low anxious participants, but its scalp distribution was also altered in the former compared to the latter group. Whereas the CRN scalp map was shared across the two groups (and mainly involved posterior parietal regions – Brodmann area 7, with an extended activation toward Brodmann area 31), the ERN/Ne scalp map had a different configuration in high vs. low anxious participants. The fronto-central negative activity associated with errors (ERN/Ne) showed a broader and more extended (pre)frontal distribution for high anxious participants, relative to low anxious participants, where this early negative activity was clearly circumscribed to a few electrode positions, including FCz.

For each group, we found that the ERN/Ne scalp map could be reliably modeled by a solution with distributed generators within the dorsal ACC, consistent with many previous ERP studies that primarily ascribed the ERN/Ne either to the activity of the Premotor/Supplemental motor area or the dorsal ACC, or sometimes both (Dehaene et al., 1994; Herrmann et al., 2004; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Miltner, Braun, & Coles, 1997; O'Connell et al., 2007) . However, we found that the differential scalp map for the ERN/Ne between the two groups could be explained by a slight shift within the dorsal ACC for the exact location of the intracranial generators. For low anxious participants, the ERN/Ne was primarily generated in the Premotor/Supplemental motor area and in the dorsal ACC (Brodmann areas 6 and 32), while for high anxious participants, the maximum within the dorsal ACC shifted towards the front, and involved more frontal and dorsal parts of the medial frontal cortex (Brodmann areas 6 and 24). Furthermore, a direct comparison between groups (Figure 4D) revealed that high anxious participants recruited more anterior as well as posterior medial frontal regions during the time interval of the ERN/Ne, relative to low anxious participants. Noteworthy, this contrast disclosed that anterior medial frontal and rostral ACC regions were

more activated in high anxious participants, as opposed to more dorsal ACC regions in low anxious participants (Figure 4D). This finding may therefore indicate that not only cognitive but also emotional monitoring effects were temporarily active in high anxious participants during the early detection of response errors (see Bush et al., 2000).

Interestingly, this substantial alteration of the electric field configuration underlying the ERN/Ne as a function of trait anxiety could be explained by the attentional control theory (Eysenck & Calvo, 1992; Eysenck et al., 2007). This model predicts that with similar task demands high anxious participants recruit more cognitive resources (i.e. they are less efficient) than low anxious participants to reach the same level of performance. Our behavioral results are consistent with this theory, as trait anxiety did not influence performance (see also Compton et al., 2007; Hajcak et al., 2003a). To compensate for this reduced efficiency, the use of more cognitive (or emotional) resources in the high anxious group could translate as a different recruitment of cognitive control areas in anxiety (see Braver et al., 2007; Fales et al., 2008 for converging evidence). Our observation of a qualitative difference in the neurophysiological expression and intracranial generators of the ERN/Ne with trait anxiety therefore corroborates this view. This effect might reflect the activation of distinct cognitive control processes in high anxious participants, a self-generated compensatory strategy used by these participants to deal with the immediate need of behavioral adjustments imposed by the early detection of unforced errors during this Go/noGo task (Eysenck & Calvo, 1992).

More specifically, we suggest that trait anxiety alters early error-detection mechanisms (an important component of cognitive control) within the dorsal division of the ACC (Brodmann areas 24 and 32). Previous studies already demonstrated that different areas in the rostral division of the ACC contribute differentially to action monitoring and cognitive control. For example, while the anterior part of the rostral ACC was assumed to exhibit conflict specific effects, the posterior part of the rostral ACC was found to be less sensitive to conflict and showed more general action-monitoring effects (Milham & Banich, 2005).

Moreover, different subdivisions of ACC may serve different functions, with a shift between emotional and cognitive operations during behavioral control along an anterior-posterior axis (Bush et al., 2000). Hence, our results suggest that high anxious participants may call extra emotional control regions within the rostral ACC during the early detection of errors, relative to low-anxious participants, who showed a more typical dorsal ACC contribution during this process (see Dehaene et al., 1994; Bush et al., 2000). The observed shift of the neural generators for the ERN/Ne within the dorsal ACC as a function of trait anxiety suggests that different cognitive control areas may also exist within the dorsal ACC. Moreover, low and high anxious participants seem to differentially recruit these areas, indicating that errors may acquire a different cognitive or motivational significance in high, as opposed to low anxious participants. Thus, high anxious participants not only respond stronger to self-generated errors, but they also react in a different way, relative to non anxious participants.

To conclude, the results of this study show that trait anxiety can lead to qualitative (and not only quantitative) changes during the earliest stage of error monitoring. As such, these findings are consistent with the attentional control theory (Eysenck et al., 2007) and they may help better understand effects of trait anxiety on cognitive control brain mechanisms. Future ERP studies should further investigate what may be the influence of these qualitative changes during early error monitoring onto the regulatory component of this process, which presumably takes place later after error commission and involves other brain structures, including the dorsolateral prefrontal cortex.

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CHAPTER 3: ELECTRICAL BRAIN IMAGING REVEALS THE EXPRESSION AND TIMING OF ALTERED ERROR-MONITORING FUNCTIONS IN MAJOR DEPRESSION

"Major depressive disorder (MDD) is characterized by disturbances not only in affect or motivation, but also in cognitive control. These latter impairments sometimes include error-detection brain processes, although their actual expression at the electrophysiological level remains unclear. In this study, we compared 17 MDD patients and 17 healthy controls (HCs), while they performed a speeded Go/noGo task designed to explore error-monitoring functions. MDD patients had overall slower reaction times (RTs) than HCs for correct Go and incorrect noGo trials, however accuracy for Go and noGo trials did not differ between groups. Unwanted response errors committed by participants during the task were associated with two well-described error-related ERP components, the error-related negativity (ERN/Ne) and error positivity (Pe). Using electrical brain imaging, we found that whereas the ERN/Ne had the same magnitude in both groups at the scalp level, MDD patients showed however overactive medial frontal cortex (MFC; Brodmann Area - BAs 8 and 9) activation during this early time interval following error commission. By contrast, the subsequent Pe component was substantially blunted in MDD patients compared to HCs, and this effect was accompanied by a reduced activation of ventral anterior cingulate cortex (ACC; BAs 24 and 32) regions. Additional analyses showed that this Pe effect was related to excessive ruminative thinking in MDD patients. These results suggest that MDD has multiple cascade effects on early error-monitoring brain mechanisms. An overactive early error-detection process in MFC (ERN/Ne) could inadvertently unlock extra self-reflection or internal monitoring processes in these patients, an interference effect that would somehow prevent the rapid conscious appraisal of errors in ventral ACC, and in turn decrease the amplitude of the Pe component."

INTRODUCTION

The ability to continuously assess whether our actions are goal conducive or not, is an important feature of cognitive control and is crucial for the flexible adjustment and optimization of behavior (Holroyd & Coles, 2002; Rabbitt, 1966). Selective impairments in this action-monitoring process can result in serious daily life problems or maladaptive behavior, and have often been reported in specific psychiatric populations (Olvet & Hajcak, 2008; Ullsperger, 2006). For example, prior studies in individuals with major depressive disorder (MDD) have demonstrated exaggerated reactions to errors or negative feedback on task performance (Beats, Sahakian, & Levy, 1996; Elliott, Sahakian, Herrod, Robbins, & Paykel, 1997; Elliott et al., 1996), an excessive concern or worry related to error commission (Enns & Cox, 1999; Shafran & Mansell, 2001), an increase in negative mood after perceived failures (Abela & D'Alessandro, 2002; Henriques & Leitenberg, 2002), difficulties in regulating failure-related thoughts following negative feedback (Conway, Howell, & Giannopoulos, 1991), as well as a decreased accuracy following error commission (Holmes & Pizzagalli, 2008; Pizzagali, Peccoralo, Davidson, & Cohen, 2006). This overactive monitoring of errors or negative outcome might be accounted for by selective deficits in executive functions (Mayberg, 1997) and eventually bolster ruminative response styles in these depressed participants (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008).

These selective impairments in action monitoring typically observed in depressed patients have also been revealed by earlier neurophysiological work. More specifically, given the well-known prefrontal-limbic dysregulation underlying the onset and maintenance of MDD (Mayberg, 1997), and the involvement of this specific brain network in error monitoring (Bush, Luu, & Posner, 2000; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Seifert, von Cramon, Imperati, Tittgemeyer, & Ullsperger, 2011; Ullsperger & von Cramon, 2006), it is not surprising that executive functions deficits in MDD also concern this specific cognitive function. Previous studies established that error

monitoring subsumes not only deep midbrain dopaminergic structures (Frank, Worroch, & Curran, 2005; Holroyd & Coles, 2002), but also paralimbic emotion-related brain regions, including the rostral Anterior Cingulate Cortex (rACC) (van Veen & Carter, 2002), the amygdala (Pourtois et al., 2010) and the orbito-frontal cortex (Dhar, Wiersema, & Pourtois, 2011). Likewise, error monitoring critically depends upon cognitive control cortical regions, including the dorsal Anterior Cingulate Cortex (dACC; Dehaene, Posner, & Tucker, 1994; MacDonald, Cohen, Stenger, & Carter, 2000) and the Prefrontal Cortex (PFC; Kiehl, Liddle, & Hopfinger, 2000; Menon, Adleman, White, Glover, & Reiss, 2001), all being selectively affected in MDD (Mayberg, 1997). In line with this framework, Holmes and Pizzagali (2008) showed, using scalp electroencephalogram (EEG) measurements, that depression is associated with an increased activation within midline regions that encompass the rACC and the medial PFC ~80 ms after error commission, as well as a disrupted connectivity between the rACC and the left dorsolateral PFC. While in healthy controls (HCs) an increased ACC activity predicted the activity in the left dlPFC ~472 ms after the commission of an error, this relationship was not found in MDD patients.

As outlined in this last study (Holmes & Pizzagali, 2008), Event-Related Potential (ERP) experiments looking at possible alterations of error-monitoring functions in MDD have usually focused on two well-characterized error-related components, namely the error-related negativity (ERN/Ne), or error negativity (Ne), and the error positivity (Pe). The ERN/Ne is a negative deflection peaking ~ 0-100 ms following an incorrect response, with a maximum amplitude over fronto-central midline recording sites (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring, Coles, Meyer, & Donchin, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The neural generators of this component have consistently been found in medial frontal cortex (MFC) and dACC, with sometimes a contribution of more rACC regions (Dehaene et al., 1994; Holroyd, Dien, & Coles, 1998; van Veen & Carter, 2002). These results are compatible with the assumption that the dACC is an important hub for cognitive control and executive functions, including early error detection (Bush et al., 2000; Carter et

al., 1998; Devinsky, Morrell, & Vogt, 1995). A similar but smaller negativity is also observed following correct actions, the correct-related negativity (CRN; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). This observation of a reduced early ERN/Ne-like response for correct responses has challenged the notion that the ERN/Ne is selectively involved in error monitoring, and led some authors conclude that the ERN/Ne and CRN actually reflect activity of a generic action-monitoring system in dACC (Roger, Bénar, Vidal, Hasbroucq, & Burle, 2010; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal, Hasbrouckq, Grapperon, & Bonnet, 2000. This early negative ERN/Ne component is followed by a large positive deflection during error monitoring, the Pe. The Pe usually reaches its maximum amplitude over centro-parietal scalp recordings along the midline ~200-400 ms post-response onset (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Nieuwenhuis, Ridderinkhof, Blow, Band, & Kok, 2001; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). Previous studies have ascribed either the rACC as the main generator of the Pe (Herrmann, Rommler, Ehlis, Heidrich, & Fallgater, 2004; van Veen & Carter, 2002) or a network comprising MFC regions and the insula (Dhar et al., 2011). Unlike the ERN/Ne, the Pe is thought to reflect a more elaborate, perhaps conscious stage of error detection (Nieuwenhuis et al., 2001). Alternatively, it could reflect an affective appraisal of an error (Falkenstein et al., 2000; van Veen & Carter, 2002), a P300 like orienting response (Ridderinkhof, Ramautar, & Wijnen, 2009), or the accumulation of evidence that an error has been committed (Steinhauser & Yeung, 2010).

Each of these two error-related ERP components was previously shown to vary with MDD, even though mixed results were obtained regarding the exact nature and direction of these changes during early error-monitoring brain processes. While some studies found a larger ERN/Ne in MDD patients compared to Healthy Controls (HCs) (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008; Holmes & Pizzagalli, 2010), other studies reported similar (Compton et al., 2008; Schrijvers et al., 2008; Schrijver et al., 2009) or even smaller ERN/Ne amplitudes in MDD patients (Ruchsow et al., 2006; Ruchsow et al., 2004). Likewise,

discrepant findings have also been reported in these studies for amplitude variation of the Pe component as function of depression. While Chiu and Deldin (2007), Compton et al. (2008) and Holmes and Pizzagalli (2008) observed similar Pe amplitudes for HCs and MDD patients, Schrijvers et al. (2008) and Schrijvers et al. (2009) reported smaller Pe amplitudes in MDD patients compared to HCs. These mixed results may be explained by the fact that MDD is not a single or unified construct (i.e. there is a substantial heterogeneity in the form and expression of this disorder across patients, see Mayberg, 1997), as well as possible differences in depression severity (Schrijvers et al., 2009). More generally, this heterogeneity somehow challenges the assumption that the ERN/Ne component represents a stable endophenotype of internalizing disorders (see Olvet & Hajcak, 2008). Both depression and anxiety are typically described as belonging to the internalizing dimension of psychopathology (Vaidyanathan, Nelson, & Patrick, 2012) and accordingly, are characterized by an increased sensitivity to errors by higher negative affect (Clark & Watson, 1991). However, only high levels of trait anxiety (even at the sub clinical level), but not depression, have consistently been related to an augmented ERN/Ne during error commission (Aarts & Pourtois, 2010; Hajcak et al., 2003a). Hence, the picture emerges that whereas the link between an increased ERN/Ne and elevated levels of trait anxiety is relatively clear and consistently found across many studies (Olvet & Hajcak, 2008), much less is known about the actual effects of MDD on early error-monitoring brain processes (i.e. ERN/Ne and Pe components).

Another likely source of variability across the existing studies and the sometimes discrepant findings might be related to the fact that effects of depression (or anxiety) on error-monitoring brain processes do not necessarily change the strength or the raw amplitude of the recorded ERP signal (especially when the ERP signal concerns a limited number of sites), but rather its overall expression at the scalp level, and hence the underlying configuration of brain generators that might undergo some anxiety or depression-related changes as well. However, these subtle changes are usually more difficult to pick up using

standard peak measurements (Picton et al., 2010; Pourtois, Delplanque, Michel, & Vuilleumier, 2008). Consistent with this interpretation, we recently found out that trait anxiety essentially altered the topography (more than the actual strength) of the ERN/Ne component, suggesting a change in the underlying configuration of brain networks recruited for early error detection in anxious vs. non-anxious individuals (see Aarts & Pourtois, 2010). Accordingly, it remains to be established whether in the absence of any significant change for the strength of the ERN/Ne or Pe component between MDD patients and HCs at the scalp level, a concurrent change of the topography for these two ERP components could be detected or not.

The goal of our study was to address this question, and better characterize at the electrophysiological level possible changes of early error-monitoring brain processes (with a focus on the ERN/Ne and Pe components) in MDD patients. Using 128-channels high-density EEG, we compared in MDD patients vs. HCs the electrophysiological responses to commission errors performed during a standard and previously validated Go/noGo task (Aarts & Pourtois, 2010, 2012; Vocat, Pourtois, & Vuilleumier, 2008) The added value of this task is that it enables to collect within a short period of time (~30 min) a high number of (unwanted) commission errors (i.e. False Alarms - FAs) on noGo trials, in each and every participant, despite inter-individual differences in reaction times (RTs) and without inducing excessive frustration. Because depression is commonly associated with enhanced levels of trait anxiety and they both encompass internalizing disorders (Jorm et al., 1999; Mineka, Watson, & Clark, 1998; Nolen-Hoeksema et al., 2008), we surmised that the ERN/Ne of MDD patients may be larger than the ERN/Ne of HCs (Olvet & Hajcak, 2008). Moreover, we predicted that the latter effect might be associated with overactive or abnormal activities in MFC regions, including dACC (see also Holmes & Pizzagalli, 2008). Regarding possible effects of MDD on the subsequent Pe component, we did not formulate clear predictions because of the mixed results previously reported in the literature (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008; Holmes & Pizzagalli, 2010; Schrijvers et al., 2008; Schrijvers et al., 2009). Using standard component

(Picton et al., 2000) as well as alternative topographical analyses (Michel et al., 2001; Pourtois et al., 2008), we therefore aimed at better characterizing the possible changes of early error-monitoring brain processes associated with MDD, with a focus not only on the ERN/Ne component, but also on the subsequent Pe component, bearing in mind that these two ERP components likely reflect different cognitive or affective processes during error monitoring (Overbeek et al., 2005; van Veen & Carter, 2006). Whereas the ERN/Ne may be related to an early error-detection stage operating on an internal motor representation of action and relying primarily on midbrain-cingulate dopaminergic loops (Frank et al., 2005; Holroyd & Coles, 2002), the Pe would instead index a more strategic and elaborate process, possibly involving specific attentional (Ridderinkhof et al., 2009) or even interoceptive (Dhar et al., 2011) components. An unanswered question is whether MDD, unlike (sub clinical) trait anxiety, may influence both processes during error monitoring or not. Finally, because previous studies reported that rumination accounted for executive functions deficits in MDD (Watkins & Brown, 2002) and a lack of attentional flexibility (Davis & Nolen-Hoeksema, 2000), we also assessed whether ruminative thinking as a distinctive cognitive style (Nolen-Hoeksema, 2000; Nolen-Hoeksema et al., 2008), besides levels of trait anxiety (Clark & Watson, 1991; Olvet & Hajcak, 2008), might be related to changes observed in MDD patients at the electrophysiological level (ERN/Ne and Pe components) during the early monitoring of response errors in our speeded Go/noGo task.

METHODS

Participants

Twenty non-depressed HCs (15 females; mean age: 41, *SEM* = 3.27) and 22 individuals meeting the DSM-IV criteria for MDD (15 females; mean age: 36, *SEM* = 2.66) participated in this study. The data of eight participants had to be excluded from the analyses because they did not commit enough errors (i.e. < 6; 2 HCs and 5 MDD patients) or the recorded EEG data were contaminated by too many artifacts (i.e. more than 20%) precluding the possibility to compute reliable

ERP waveforms (1 HC). In total, the data of 17 HCs (14 females; mean age: 41, *SEM* = 3.77) and 17 MDD patients (10 females; mean age: 36, *SEM* = 3.04) were included in the analyses. Demographic and clinical characteristics of these participants are outlined in Table 1.

Table 1

Descriptive statistics for healthy controls (HC) and depressed patients (MDD)

	HC	MDD	
	M (SEM)	*M (SEM)*	*p*
N	17	17	
Age	41.24 (3.77)	35.76 (3.04)	.27
Sex	3M/14F	7M/10F	.14
Education			
STAI_T	29.63 (1.32)	63.81 (1.89)	< .001
HAM_D	0.24 (0.14)	28.12 (1.33)	< .001
BDI_II	1.59 (0.97)	33.24 (2.87)	<.001
RRS_TOT	31.24 (1.32)	77.00 (2.90)	< .001
Age at onset		30.76 (3.20)	
Length of episode (months)		7.35 (1.38)	
Number of episodes		2.76 (.35)	

These MDD ambulatory patients were recruited from a local Belgian psychiatric clinic and were diagnosed with MDD and/or anxiety disorders. Prior to testing, the Hamilton Rating Scale for Depression (HAM-D; Hamilton, 1960) and the Mini-International Neuropsychiatric Interview (MINI), a structured clinical interview (Sheehan et al., 1998), were administered to examine the severity of the current MDD episode (HAM-D: *M* = 28.12; *SEM* = 1.33). Exclusion criteria were 1) other mood disorders than MDD; 2) the intake of anti-psychotics, tricyclic anti-depressants and/or long lasting benzodiazepines; 3) a history of neurological disorder, including epilepsy, head injury and loss of consciousness; 4) a history of electroconvulsive therapy; 5) alcohol abuse during the past year; 6) a past or present substance dependence; 7) past or present experience of

psychotic episodes; or 8) learning disorders. All MDD participants had a normal anti-depressant medication during the time of testing (i.e. either based on Selective Serotonin Reuptake Inhibitors – SSRI, or Selective Noradrenalin Reuptake Inhibitors - SNRI). HCs were recruited using advertisements in local newsletters or newspapers and were free of any mediaction at the time of testing.

All participants were native Dutch speakers, gave their written informed consent and were payed 20 Euro. The study was approved by the medical ethics committee of the Ghent University hospital.

Stimuli and task

We used a speeded Go/noGo task previously used and validated in another group of HCs (Figure 1; Vocat et al., 2008), as well as in a group of (sub clinical) high anxious individuals (Aarts & Pourtois, 2010). Visual stimuli were shown on a 17-inch LCD screen. They consisted of an arrow (11.4° x 0.05° of visual angle at a 60 cm viewing distance) that was presented in the center of the screen on a white background. Each trial started with a fixation cross that lasted for 1000 ms. Then, a black arrow (i.e. cue), either oriented up or down, was presented. After a variable interval ranging from 1000 up to 2000 ms, the black arrow became either green (i.e. target) or turquoise while its in-plane orientation could either remain identical or shift in the opposite direction. Participants were asked to perform a speeded color plus orientation discrimination task. When the black arrow turned green and the orientation remained unchanged (2/3 of the trials), participants were instructed to press a predefined key on the response box as fast as possible with a predefined finger of their dominant hand (Go trials). However, participants had to withhold responding when either the arrow became green but changed orientation (1/3 of the trials), or when the arrow became turquoise and kept its initial orientation (1/3 of the trials), enabling two types of noGo trials (based either on the orientation or color). For noGo trials, this color arrow remained on the screen for a maximum duration of 1000 ms. Instructions emphasized both speed and accuracy.

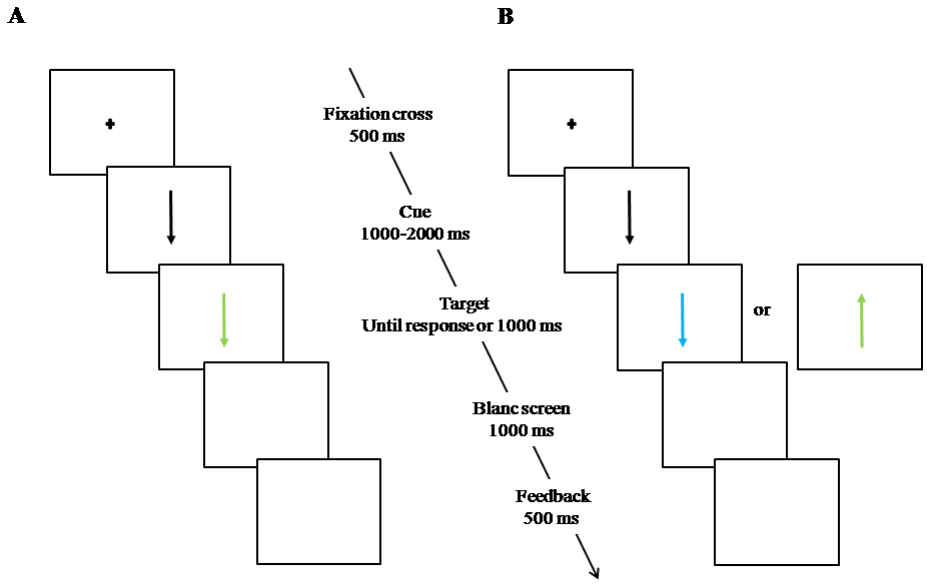

Figure 1. Stimuli and task. (A) On each trial, a black arrow was first presented. After a variable interval (1000-2000 ms), the black arrow usually (2/3 -Go trials) became green and kept its initial orientation (either up or down). (B) On the remaining 1/3 of the trials, it became either turquoise and/or green but with a change in orientation (noGo trials).

We used a specific procedure to ensure that the number of errors was roughly balanced between MDD patients and HCs. This pre-requisite was important, given that the ERN/Ne amplitude varies according to the number of errors (i.e. the ERN/Ne is larger when response errors are less frequent; see e.g. Gehring et al, 1993). To achieve this, we used a response deadline that was calibrated beforehand, adjusted and updated online at the single subject level (see also Aarts & Pourtois, 2010 for a similar procedure). For each and every Go trial, the RT was compared against an arbitrary limit. If the RT was slower than this arbitrary limit ("slow hit"), then the participant received a negative feedback on task performance whereas if the RT was faster than this limit ("fast hit"), he/she received no feedback (see Figure 1). This procedure promotes the occurrence of fast RTs, and accordingly the commission of errors on deviant/infrequent noGo trials. Unbeknown to participants, specific short calibration blocks were used and interleaved throughout the experimental session to define and update the RT limit that was subsequently used during the following experimental/test blocks. For the first two test blocks, the limit was set to 80% of the mean RT from the first calibration block. After these 2 blocks, this

limit was updated and set to 90% of the mean RT to account for effects of fatigue and learning, before the participant completed two other test blocks. The limit was computed and updated a third final time, before the participant completed the last two test blocks (see also Vocat et al., 2008; Pourtois et al., 2010). A thousand ms after "slow hits", the participant received as feedback the words "too slow" in Dutch printed in a red frame for 500 ms. For all the other conditions (Fast Hit, Error, Omission and Correct Inhibition), no feedback on task performance was presented in such a way to increase the monitoring of self-generated actions based on internal motor representations. Finally, to maintain a constant level of attention and involvement in the task throughout the experimental session, the cumulative accuracy (in %) was continuously updated and displayed in the upper part of the screen during each inter-trial interval (with a central fixation cross; see also Vocat et al. 2008 for a similar procedure).

The experiment consisted of a single practice block of 12 trials (4 Go, 4 noGo of each condition), 3 calibration blocks of 14 trials each (10 Go and 2 noGo of each type) and 6 test blocks of 60 trials (40 Go trials and 20 noGo trials). Each calibration block was followed by two consecutive test blocks. Trial presentation was randomized within blocks. Stimulus presentation and response recording were controlled using E-prime software (V2.0., http://www.pstnet.com/products/e-prime/).

Questionnaires

Levels of depression, trait anxiety and ruminative thinking were verified, prior to testing, using the trait version of the State Trait Anxiety Inventory (STAI – Defares, van der Ploeg, & Spielberger, 1979; Spielberger, 1983), the Beck Depression Inventory (BDI-II; Beck, Ward, Mendelson, Mock & Erbaugh, 1961), the HAM-D (Hamilton, 1960) and the Rumination Response Scales (RRS; Nolen-Hoeksema & Morrow, 1991).

Analysis of behavioral data

RTs faster than 150 ms (Error: *M* = .93, *SEM* = .38; Hit: *M* = .36, *SEM* = .18) and longer than 800 ms (Error: *M* = 2.41, *SEM* = 1.06; Hit: *M* = 1.58, *SEM* = .41) were removed from the subsequent analyses. Next, RTs faster than *M* – 2.5 *SD* (Error: *M* = .00, *SEM* = .00; Hit: *M* = .01, *SEM* = .01) or slower than *M* + 2.5 *SD* (Error: *M* = 2.31, *SEM* = .48; Hit: *M* = 2.72, *SEM* = .18) were also excluded. The number of outliers was not significantly different between HCs and MDD patients, all $p > .10$. Color and orientation errors were collapsed together (error condition) since there was no significant difference or group difference between these two error types (see also Aarts & Pourtois, 2010; Pourtois et al., 2010 for similar results). Likewise, fast and slow hits were collapsed and treated as a single condition (hit condition). Mean RTs for errors and Hits as well as the number of errors and Hits were then computed and compared by means of 2 x 2 mixed analysis of variances (ANOVAs), with group (HC vs. MDD) as between-subjects factor and accuracy (Error vs. Hit) as within subjects variable.

EEG recording

Continuous EEG was acquired at 512 Hz using a 128-channel (pin-type) Biosemi Active Two system (http://www.biosemi.com) referenced to the CMS-DRL ground. ERPs of interest were computed offline following a standard sequence of data transformations (Picton et al., 2000): (1) -500/+1000 segmentation around the onset of the response, (2) pre-response interval baseline correction (from -500 ms to response onset), (3) vertical ocular correction for blinks (Gratton, Coles, & Donchin, 1983) using the difference amplitude of two electrodes attached above and below the left eye, (4) artifact rejection [*M* = -88.53/+88.53, *SEM* = 2.36 amplitude scale (µV) across participants; no significant difference between HCs (*M* = 92.35, *SEM* = 3.25) and MDD patients (*M* = 84.71, *SEM* = 3.25) was evidenced, $t(30) = 1.67$, $p > .10$], (5) averaging of trials, separately for each group (HC vs. MDD) and experimental condition (errors vs. hits), and (6) 30 Hz low pass digital filtering of the individual average data.

We primarily focused on two well-documented error-related ERP components following motor execution (Falkenstein et al., 2000), on the ERN/Ne, with a maximum negative amplitude over fronto-central electrodes along the midline early on following motor execution (0 - 100 ms post-response onset), immediately followed by the Pe component (150 - 300 ms post-response onset), with a maximum positive amplitude over more posterior and central locations along the midline.

Standard peak analyses

We performed a conventional area under the curve analysis for each of these two error-related ERP deflections (Picton et al., 2000). For each ERP component and each condition separately, we calculated the area under the curve, during the 25 - 55 ms interval post-response onset at electrode FCz for the ERN/Ne amplitude, and during the 150 - 210 ms interval post-response onset at electrode Cz for the Pe component. The selection of these two specific scalp locations (and time windows) was based on the topographic properties of the present dataset, as well as previous ERP studies focused on the same error-related ERP activities (Dehaene et al., 1994; Gehring et al., 1990; Hajcak et al., 2003a). Statistical analyses were performed on the mean amplitude of each area using a 2 (accuracy) x 2 (group) repeated measures ANOVA, with a significance alpha cutoff set to $p < .05$. Simple bivariate Pearson correlations as well as multiple linear regression analyses were also conducted to explore whether inter-individual variations along levels of depression (BDI-II and/or HAM-D), trait anxiety (STAI-T) and/or rumination (RRS) might account for amplitude changes at the level of the ERN/Ne or Pe component, and eventually determine which of these stable personality traits best predicted the magnitude of the ERN/Ne or Pe ERP component.

Topographical analyses

Although classical area under the curve analyses are already informative regarding local amplitude changes at a few pre-defined electrode locations during action monitoring, they do not inform about more global and concurrent

changes in the distribution of the entire ERP electric field (i.e. topography) that may sometimes take place regardless of these local amplitude changes (Lehmann & Skrandies, 1980). Therefore, in order to capture more global ERP differences between HCs and MDD patients during the early detection and monitoring of response errors, a complementing detailed topographic mapping analysis of the ERP data was performed, following a conventional data-analysis scheme (see Figure 2; Michel, Seeck, & Landis, 1999; Michel et al., 2001; Murray, Brunet, & Michel, 2008; Pourtois, Dan, Grandjean, Sander, & Vuilleumier, 2005; Pourtois et al., 2008; Pourtois, Thut, Grave de Peralta, Michel, & Vuilleumier, 2005). To precisely characterize topographic modulations over time and across conditions, we used a standard spatial cluster analysis. This pattern analysis efficiently summarizes complex ERP data set into a smaller number of dominant field configurations, previously referred to as functional microstates (Lehmann & Skrandies, 1980; Michel et al., 1999). The rationale and basic principles of this temporal segmentation method have already been extensively described elsewhere (Michel et al., 1999; Murray et al., 2008). Following standard practice, we first performed a topographic pattern analysis on the grand-average ERP data from -55 ms until 379 ms after response onset (222 consecutive time frames at 512 Hz sampling rate, encompassing the ERN/Ne and Pe components), using a standard K-means cluster method (Pascual-Marqui et al., 1995). The optimal number of topographic maps explaining the whole data set was determined objectively using both cross validation (Pascual-Marqui et al., 1995) and Krzanowski-Lai (Tibshirani, Walther, & Hastie, 2001) criteria. The dominant scalp topographies (identified by the previous analysis) were then fitted back to the ERP data of each individual subject using spatial fitting procedures to quantitatively determine their representation across subjects and conditions. This procedure thus provides fine-grained quantitative values, such as the Global Explained Variance (GEV, or goodness of fit), which is a critical estimate of the significance of a given topography, not available otherwise in a classical component analysis (Picton et al., 2000). GEV represents the sum of the explained variance weighted by the Global Field Power (GFP) at each moment in time. The resulting GEV values were entered in ANOVAs with two within-subject

factors, accuracy (errors vs. hits) and map configuration (i.e. the dominant electric field distributions identified by the spatial cluster analysis), as well as group (HC vs. MDD) as between-subject factor. These analyses were carried out using CARTOOL software (Version 3.34; developed by D. Brunet, Functional Brain Mapping Laboratory, Geneva, Switzerland).

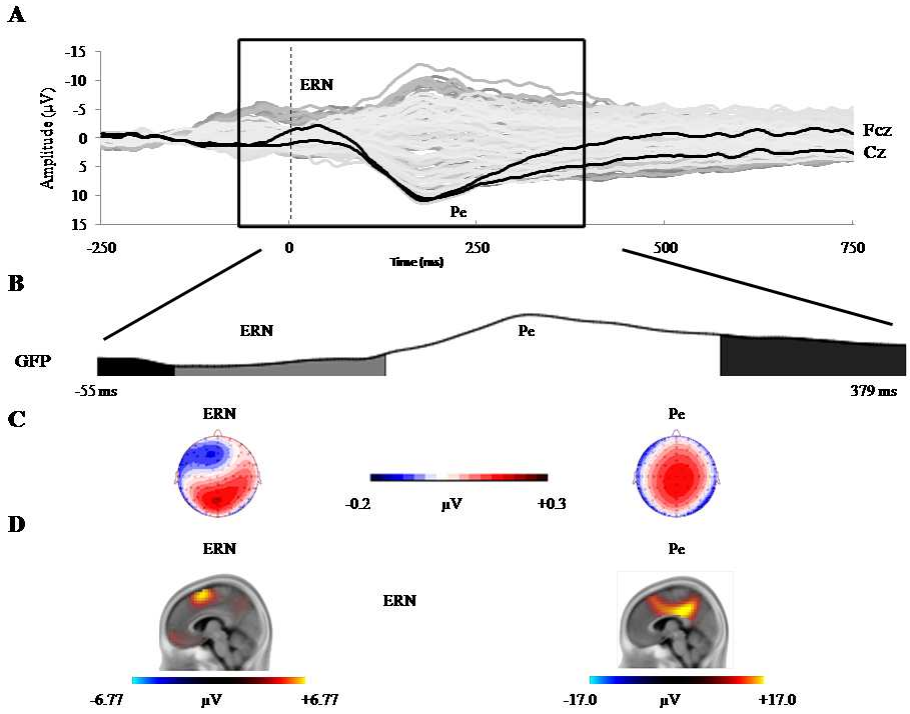

Figure 2. Illustration of the advanced cluster analysis used to identify the dominant error-related ERN/Ne and Pe topographical components. (A) This analysis used all (128) electrodes and time-frames spanning from -55 ms to 379 ms after response onset, encompassing these two error-related ERP components. A butterfly view of the grand-average ERP data of HCs (errors) from -500 to +1000 ms around the response is shown, as well as the corresponding time interval selected for the segmentation in topographical components. (B) Two stable maps, the ERN/Ne and Pe, were clearly isolated during this specific time interval. (C) Horizontal voltage maps confirmed that these two maps unambiguously corresponded to the ERN/Ne (first) and Pe (second) component during early error monitoring. (D) SLORETA was finally used to gain insight into the putative neural generators underlying these dominant scalp configurations.

Source localization analyses

Finally, to estimate the likely neural sources underlying the dominant error-related electrical field configurations identified by the previous analyses, we used a specific distributed linear inverse solution, namely standardized low-

resolution brain electromagnetic tomography (sLORETA, Pascual-Marqui, 2002). sLORETA is based on the neurophysiological assumption of coherent co activation of neighboring cortical areas (known to have highly synchronized activity, see Silva, Amitai, & Connors, 1991) and, accordingly, it computes the "smoothest" of all possible activity distributions (i.e. no a-priori assumption is made regarding the number and locations of the sources). Mathematical validation of this distributed source localization technique has been demonstrated (Sekihara, Sahani, & Nagarajan, 2005). sLORETA solutions are computed within a three-shell spherical head model co-registered to the MNI152 template (Mazziotta et al., 2001). The source locations were therefore given as (x, y, z) coordinates (x from left to right; y from posterior to anterior; z from inferior to superior). sLORETA estimates the 3-dimensional intracerebral current density distribution in 6239 voxels (5 mm resolution), each voxel containing an equivalent current dipole. This 3-dimensional solution space, in which the inverse problem is solved, is restricted to the cortical gray matter (and hippocampus). The head model for the inverse solution uses the electric potential lead field computed with a boundary element method applied to the MNI152 template (Fuchs, Kastner, Wagner, Hawes, & Ebersole, 2002). Scalp electrode coordinates on the MNI brain are derived from the international 5% system (Jurcak et al., 2007). The calculation of all reconstruction parameters was based on the computed common average reference. sLORETA units were scaled to ampere per square meter (A/m^2). We eventually directly compared inverse solution results between MDD patients and HCs, separately for the ERN/Ne and Pe component using unpaired *t*-tests.

RESULTS

Behavior

Accuracy and RT data are presented in Table 2. The number of errors was similar between MDD patients and HCs, $t(32) = 1.34$, $p > .10$. All participants were faster for errors compared to Hits, $F(1, 32) = 37.61$, $p < .001$, but overall, MDD patients reacted slower than HCs, $F(1, 32) = 4.73$, $p < .05$, but importantly

this latter RT slowing did not interact with accuracy, $F(1, 32) = 1.29$, $p > .10$. Finally, a classical post-error slowing effect (Laming, 1979; Rabbitt, 1966), indicated by slower RTs to Hits following errors compared to Hits following Hits, was evidenced, $F(1, 32) = 4.99$, $p < .05$, and this adaptation effect was similar between the two groups ($F(1, 32) = 1.47$, $p > .10$).

Table 2

Accuracy and RTs in the speeded Go/noGo task, separately for healthy controls (HC) and depressed patients (MDD)

		HC		MDD		
		M	*SEM*	*M*	*SEM*	*p*
Number	Error	29.00	4.43	21.71	3.18	.19
	Hit	232.76	3.65	223.18	5.56	.16
	Post-error	65.07	2.16	62.47	3.65	.16
	Post-hit	65.27	0.58	65.13	0.47	.19
Speed	Error	263.79	9.49	316.07	20.21	.03
	Hit	325.31	12.15	358.35	16.32	.11
	Post-error	345.95	12.80	363.67	18.73	.44
	Post-hit	321.86	12.74	356.53	16.45	.11

ERP components

A clear negative deflection was observed ~40 ms after error commission, with a maximum amplitude over fronto central electrodes (e.g. FCz). These electrophysiological properties were consistent with the ERN/Ne (Figure 3AB). Consistent with previous ERP studies (Falkenstein et al., 1991; Gehring et al., 1993), this early negative component was larger following errors compared to hits, $F(1, 32) = 5.86$, $p < .05$. Although this difference appeared to be larger for MDD patients [$M = 2.37$; $SEM = .92$; $t(16) = -2.57$, $p < .05$] compared to HCs ($M = 1.07$; $SEM = 1.08$; $t < 1$), there was no significant effect of group, $F(1, 32) = 2.37$, $p > .10$, nor a significant interaction between accuracy (error vs. hit) and group (HC vs. MDD), $F < 1$ (Figure 3CD). Consistent with previous ERP studies using this specific Go/noGo task (Aarts & Pourtois, 2010; Dhar & Pourtois, 2011), given the

speed pressure imposed to participants and the relatively high number of errors committed within a short period of time, the ERN/Ne (errors) – CRN (hits) amplitude difference was actually modest at this specific electrode position (FCZ), though being well significant, suggesting that response errors were discriminated from correct responses (hits) early on following response onset.

The ERN/Ne was followed by a large positive component that was the largest at central electrodes along the midline (i.e. Cz) and that was clearly sensitive to accuracy, being reliably larger for errors relative to hits, $F(1, 32) = 117.80$, $p < .001$. These properties (latency, polarity, topography) were compatible with the generation of a Pe component during early action monitoring and error detection. This positive component was overall larger in HCs compared to MDD patients, $F(1, 32) = 8.76$, $p < .01$, but importantly, this effect significantly interacted with group, $F(1, 32) = 5.22$, $p < .05$. This significant interaction showed that the difference in Pe amplitude between errors and hits was larger for HCs ($M = 5.77$, $SEM = .61$, $t(16) = 9.43$, $p < .001$), compared to MDD patients ($M = 3.76$, $SEM = .63$, $t(16) = 5.98$, $p < .001$) (see Figure 3EF).[1]

[1] Since MDD patients were overall slower than HCs, we performed an additional control analysis on the mean amplitude of the Pe component taking into account this speed/RT difference. We more specifically included speed (RT for either Hits or FAs) as a regressor in an ANCOVA with accuracy (FA vs. Hit) as within-subjects factor and group (MDD vs. HC) as between-subjects factor. This analysis revealed significant main effects of accuracy, $F(1, 32) = 9.86$, $p < .01$, and group, $F(1, 32) = 6.37$, $p < .01$, whereas the interaction between group and accuracy was still marginally significant, $F(1, 32) = 3.44$, $p = .07$ in this control analysis.

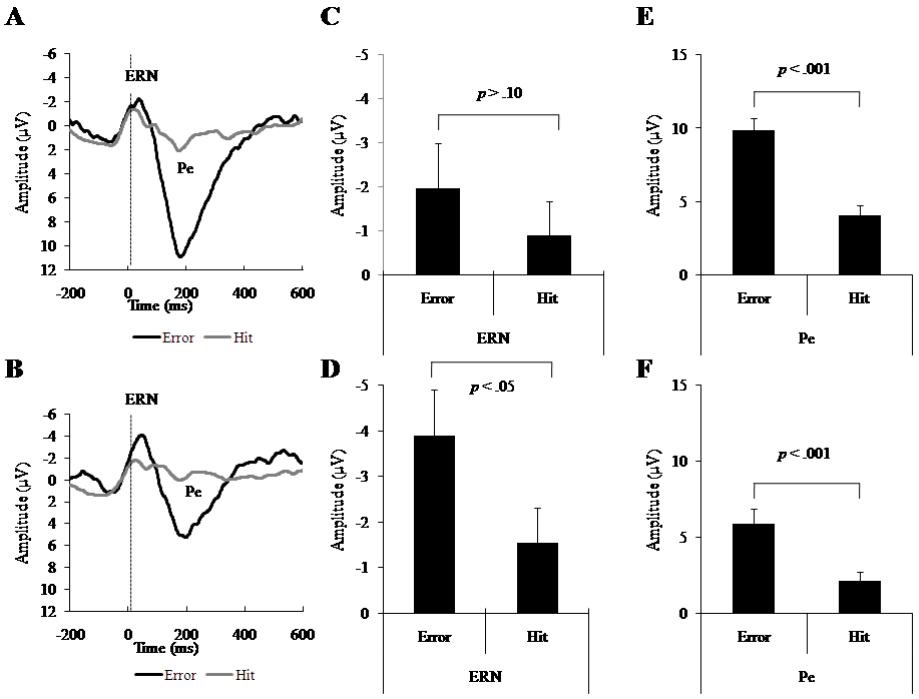

Figure 3. ERP results. (A) Grand average ERP waveforms (electrode FCz) for the HCs and (B) for the MDD patients. (C) Mean amplitude (μV) ± 1 standard error of the mean (S.E.M) of the ERN/Ne for errors vs. hits in HCs and (D) in MDD patients. (E) Mean amplitude (μV) ± 1 S.E.M of the Pe for errors vs. hits in HCs and (F) in MDD patients.

ERP-behavior correlations

We did not find evidence for a reliable link between the amplitude of the ERN/Ne and depression-related characteristics for all participants (i.e. HCs and MDD patients together) (BDI-II: r = -.22, p > .10; HAM-D: r = -.25, p > .10; STAI-T: r = -.29, p > .10; RRS: r = -.24, p > .10). By contrast, correlation analyses showed that the amplitude of the error-related Pe component was related to these depression characteristics (BDI-II: r = -.46, p < .01; HAM-D: r = -.44, p < .01; STAI-T: r = -.43, p < .05; RRS: r = -.41, p < .01); this positive deflection being systematically smaller when depression-related characteristics were higher. We next carried out a regression analysis to assess which predictor (i.e. BDI-II, HAM-D, STAI-T or RRS) best accounted for these amplitude changes at the level of the Pe component. This analysis revealed that despite the obvious multi collinearity between these variables, rumination (RRS) was actually the best significant predictor of these amplitude variations at the level of the error-related Pe component, p < .05. 19% of the Pe amplitude variance was explained by

ruminative thinking alone. When RRS was included in the statistical model, none of the other predictors was longer significant, all $p > .10$.

When we split the ERP data according to groups (HCs vs. MDD patients), we additionally found a marginally significant negative correlation between the amplitude of the ERN/Ne and trait anxiety (STAI-T; $r = -.45$, $p = .08$) as well as between the amplitude of the ERN/Ne and depression (BDI-II; $r = -.48$, $p = .05$) in HCs. In line with previous studies and models (Olvet & Hajcak, 2008), higher levels of sub clinical anxiety or depression were associated with larger ERN/Ne amplitudes. By contrast, no significant correlations between the ERN/Ne amplitude and these depression-related characteristics were found in the MDD group, all $p > .10$.

Topographical components

A spatio-temporal cluster analysis was performed on a large time-window, encompassing the error-related ERN/Ne and Pe components (i.e. starting 55 ms before response onset and ending 379 ms after response onset, corresponding to 222 consecutive time frames or 434 ms). A solution with 8 dominant maps/topographies explained 94% of the variance. Note that these dominant maps were identified regardless of local or global (i.e. global field power) changes in the amplitude or strength of the ERP signal, following standard practice. Next, we analyzed in greater detail the dominant maps generated during the time interval corresponding to the ERN/Ne and Pe, and their likely variations as a function of accuracy and/or group.

During the time interval corresponding the ERN/Ne vs. CRN component (starting ~10 ms – before response onset and ending ~90 ms post-response onset), a main change in the topography between errors and Hits was evidenced (see also Aarts & Pourtois, 2010). Whereas the topography for hits was characterized by a broad negative activity extending towards prefrontal sites (CRN map), the scalp distribution for response errors was qualified by a negative activity circumscribed to a few precentral electrode positions, including FCz (Figure 4A; ERN/Ne map), in line with previous results obtained with the same

task (Aarts & Pourtois, 2010; Vocat et al., 2008). Hence, early on following response onset, a reliable change in the electric field configuration occurred between response errors and hits. This result was important because it suggested that beyond local amplitude variations found for the peak of the ERN/Ne component (FCZ electrode, see results for classical area under the curve analyses above), errors were unambiguously associated with a change in the underlying configuration of generators, relative to hits (Lehmann & Skrandies, 1980; Pourtois et al., 2008). Following standard practice, we next performed a fitting of these two dominant maps back to the individual ERP data to verify, at the statistical level, whether this topography-related change was significant (and different across the two groups) or not. We therefore submitted the GEV values obtained for these two dominant maps after fitting to a 2 (map) x 2 (group) x 2 (accuracy) repeated measures ANOVA. This analysis revealed a significant interaction between accuracy and map/scalp configuration, $F(1, 32) = 60.40$, $p < .001$. While the CRN map explained more variance for hits than errors, $t(33) = -9.22$, $p < .001$, the ERN/Ne map had a symmetric profile, explaining more variance for errors than hits, $t(33) = 3.61$, $p = .001$. However, this interaction effect was similar for MDD patients and HCs, $F < 1$ (Figure 4B).

Regarding the time interval corresponding to the Pe component (~145 – 281 ms post response onset), a specific error-related topography (Pe map, with a maximum amplitude at electrode CZ) was identified alike, compared to hits that elicited a broad and distinct posterior positivity during the same time interval (see Figure 4C), in agreement with previous studies (Aarts & Pourtois, 2010; Vocat et al., 2008). Further analyses computed on the mean GEV values obtained for these two dominant maps confirmed a significant interaction between accuracy and map, $F(1, 32) = 28.30$, $p < .001$. Whereas the Pe map explained more variance for errors than hits, $F(1, 32) = 30.73$, $p < .001$, the other concurrent map (posterior positivity map) showed a symmetric effect, explaining more variance for hits than errors, $F(1, 32) = 18.53$, $p < .001$. Interestingly, this analysis also showed a significant interaction between map and group, $F(1, 32) = 7.96$, $p < .01$ (Figure 4D). This interaction was explained by the fact that the Pe

map (being diagnostic of error processing) explained more variance for errors committed by HCs than MDD patients, $t(32) = 3.67$, $p < .001$. The same effect was evidenced, though much weaker, for hits, $t(32) = 2.32$, $p < .05$. However, the concurrent posterior positivity map associated with hits was not significantly influenced by group, both for errors [$t(32) = -1.27$, $p > .10$] and hits ($t < 1$), suggesting that MDD influenced primarily the neural processing of errors, but not hits.

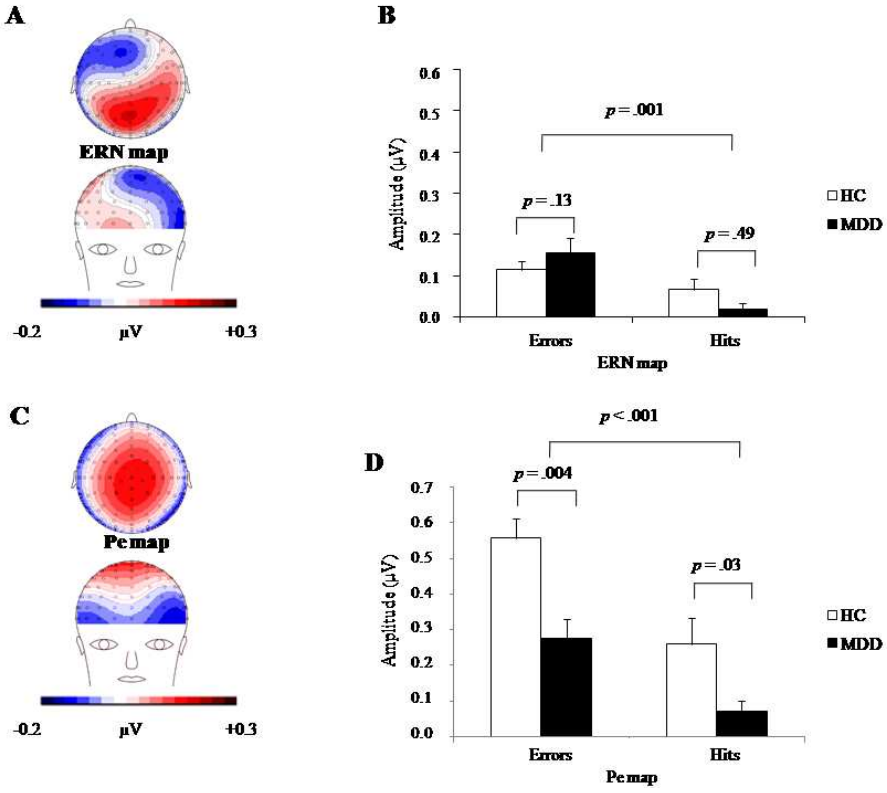

Figure 4. Dominant topographical components (horizontal and frontal views) during the time interval (-10 - 90 ms around response onset) corresponding either to the ERN/Ne (top) or Pe (bottom). (A) The scalp map of the ERN/Ne showed a negative activity reaching its maximum at FCz electrode position, and extending towards left lateral frontal electrodes. (B) The ERN/Ne topographical component explained more variance for errors compared to hits. (C) The scalp map of the Pe was characterized by a broad positive activity over central electrode positions. (D) The Pe topographical component explained more variance for errors compared to hits.

Inverse solutions

To gain insight into the putative configuration of the intracranial generators underlying these global topographic-dependent changes, we source-

localized the ERN/Ne and Pe maps, separately, using sLORETA (Pascual-Marqui, 2002).

This analysis confirmed that the configuration of the intracranial generators underlying the ERN/Ne scalp map (errors) were similar between HCs and MDD patients. These generators primarily involved MFC/dACC regions, consistent with several earlier studies (Debener, Ullsperger, Fiehler, von Cramon, & Engel, 2005; Dehaene et al., 1994; Herrmann et al., 2004; O'Connell et al., 2007; Vocat et al., 2008). For HCs, the neural generators of the ERN/Ne were mainly localized within superior frontal gyrus/dACC (maximum: 6x, 6y, 44z; BAs 32, 24 and 6), whereas for MDD patients, they also involved the superior frontal gyrus/dACC (maximum: 6x, 6y, 44z; BAs 6, 8, 32 and 24), with a slight shift towards the front for the maxima, compared to HCs (Figure 5A). Importantly, a direct statistical comparison in the inverse solution space (see Table 3) between the two groups showed that MDD patients had a significantly stronger MFC/dLPFC (BA6, BA8 and BA9) activation compared to HCs, while the ERN/Ne of HCs was associated with an additional activation in the posterior cingulate cortex (BAs 29 and 30) (Figure 5B). By contrast, the CRN map was associated with a main generator/cluster within medial frontal/dACC regions, equally in both groups. The maximum was localized within the superior frontal gyrus (BA6; MNI coordinates: 5x, -0y, +70z) (see Table 3).

Regarding the Pe component, sLORETA showed that its underlying brain generators primarily involved the insula (BA 13) and a widespread cluster encompassing different cingulate areas, namely BAs 23, 24 and 31 (see Figure 5C). This network was not evidenced for the posterior positivity map associated with hits during the same time interval. A direct comparison between the two groups revealed a significantly stronger recruitment of deep/ventral cingulate areas (BAs 23, 24, 31 and 32; see Figure 5D) for HCs compared to MDD patients during the processing of errors (see Table 3).

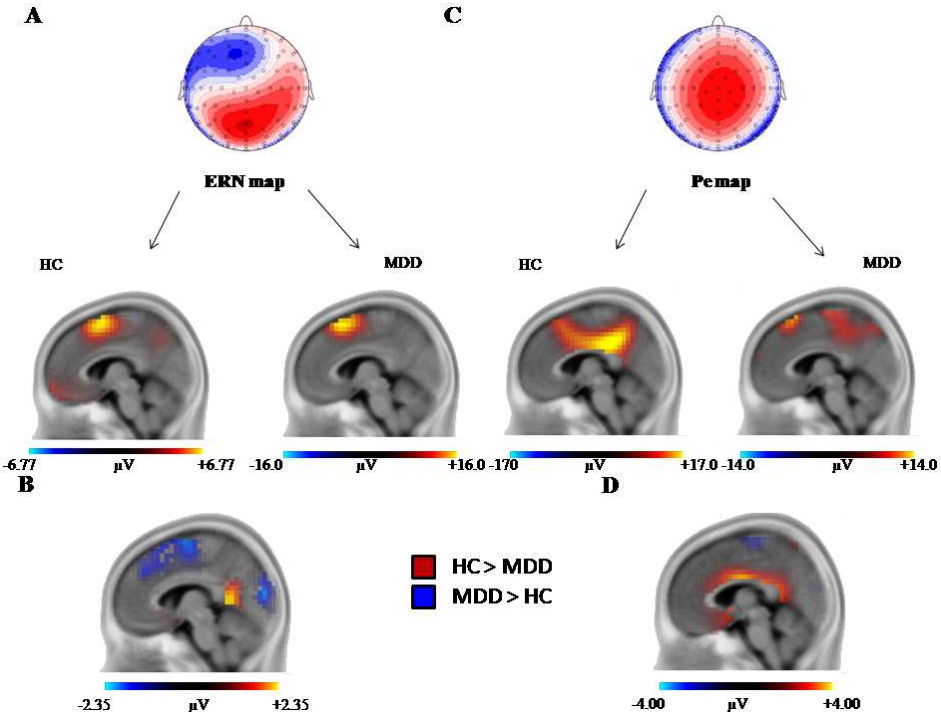

Figure 5. Source localization results, based on sLORETA. (A) Inverse solution for the ERN/Ne topographical component, separately for HCs and MDD patients, revealing a main cluster in the dACC (BAs 32 and 24) and MFC (superior frontal gyrus – BA 6). (B) A direct statistical comparison between the two groups showed that MDD patients had a significantly stronger MFC/dLPFC (BAs 6, 8 and 9) activation compared to HCs, while the ERN/Ne of HCs was associated with an additional activation in the posterior cingulate cortex (BAs 29 and 30) (C) Inverse solution for the Pe topographical component, separately for HCs and MDD patients, revealing a main extended cluster in the cingulate areas (BAs 23, 24 and 31) in the former participants, but not the patients. (D) A direct comparison between the two groups showed that HCs recruited more ventral cingulate areas (BAs 23, 24, 31 and 32) as well as insula regions (BA13, not shown on this view) compared to MDD patients.

Tabel 3

MNI coordinates of the differential error-related peak activations between HCs and MDD patients, separately for the ERN/Ne and Pe component

Component	Regions of Interest (ROI)	MNI Coordinates				sLORETA
		BA	x	y	z	p-values
ERN/Ne	Superior frontal gyrus	6	-5	-5	70	*
		8	-25	25	45	*
		9	-30	25	40	.09
	Posterior cingulate	30	15	-55	5	*
		29	10	-45	5	*
Pe	Insula	13	35	-10	15	**
	Cingulate gyrus	23	5	-15	30	**
		24	5	-5	30	**
		31	20	-45	25	**

Note. * p < .05; ** p < .01

DISCUSSION

In this study, HCs vs. MDD patients performed a speeded Go/noGo task and occasionally made unwanted errors on noGo stimuli while their error-related brain activities were tracked and compared using high density EEG. HCs and MDD patients were similar regarding laterality and age. Standard clinical interviews and results obtained at several additional questionnaires or inventories confirmed that the patients had clinical levels of MDD, but not the HCs. Importantly, we used a speeded Go/noGo task previously validated in the literature (Aarts & Pourtois, 2010; Dhar & Pourtois, 2011; Vocat et al., 2008) that offered the added value to characterize functional alterations in early error-detection brain processes as a function of depression, when these modifications were not confounded by obvious changes in the behavior or task performance (e.g. the error rate) of MDD patients vs. HCs. Whereas MDD patients responded

overall slower compared to HCs, they did not commit more or less response errors than these healthy participants, enabling a neat comparison of error-monitoring brain processes across these two groups, using ERP measurements. Despite a balanced accuracy and similar standard post-error adjustment, our ERP results show a statistically similar early ERN/Ne component in MDD patients and HCs. However, the ERN/Ne of MDD patients was associated with enhanced MFC activation, relative to HCs. In addition, a large difference was found for the subsequent Pe component during early error monitoring. This component was substantially blunted in MDD patients compared to HCs. Moreover, we found that this effect was likely explained by a reduction in the activation of ventral medial cingulate areas during the generation of the Pe component. Of note, this outcome was obtained not only based on standard peak measurements carried out at a few isolated standard and representative electrode positions (Picton et al., 2000), but also based on a thorough and experimenter-free evaluation of the global topography (all 128 channels taken into account) of these two specific error-related ERP components, using an alternative spatio-temporal cluster analysis (Lehmann & Skrandies, 1980; Pourtois et al., 2008). We discuss the implication of these new results in greater detail here below.

Balanced behavioral performance between MDD patients and HCs

MDD patients and HCs committed ~25 response errors. This number, which was balanced across groups, enabled us to compute reliable error-related ERP waveforms for each individual. Because of the imposed time pressure and specificities of our speeded Go/noGo task, we were therefore able to compare error-related brain activities, using scalp EEG measurements, between MDD patients and HCs when these brain effects were not simply accounted for by systematic changes in the behavior, including the number of response errors (Gehring et al., 1993). Although MDD patients were overall slower compared to

HCs, the blunted Pe component for MDD patients compared to HCs was not merely explained by this speed difference across groups[2].

Although this outcome suggests that MDD patients may have preserved error-monitoring functions when investigated at the behavioral level, we have to acknowledge that this result might be explained by the use of a speeded Go/noGo task and an individually calibrated response deadline. The added value of this procedure is that the likelihood of error commission is reliably increased within a relatively short period of time despite simple task rules and demands (that can easily be understood by patient populations), but inter-individual variability in error making is by definition somehow neutralized in these circumstances. Accordingly, it is likely that MDD patients would show a different pattern of behavioral results relative to HCs, if no such calibration procedure would be implemented or another interference task (e.g. flanker or Stroop), would be used (Degl'Innocenti, Agren, & Backman, 1998).

Preserved ERN/Ne in MDD

Our results for the ERN/Ne do not point to obvious error-monitoring impairments at the level of this early dopaminergic-dependent ERP component in MDD. Although the ERN/Ne was slightly larger at the scalp level in MDD patients compared to HCs, this difference did not reach significance. In our study, we included in our MDD sample severely depressed individuals (as reflected by their HAM-D and BDI-II scores) and this severity may potentially account for the lack of differential effect at the level of the ERN/Ne between MDD patients and HCs. Previous ERP studies already reported unchanged or even diminished ERN/Ne amplitudes in severely depressed individuals that are characterized by

[2] Although MDD patients were taking medication at the time of testing while HCs did not, it appears unlikely that this factor alone would account for the observed ERP differences (mostly concerning the Pe component) between groups, even though we cannot formally rule out this alternative account. Regular anti-depressant drugs (e.g. SSRI or SNRI), as used by the MDD patients included in our study, have not been linked to systematic alterations of the amplitude or morphology of error-related ERP components in previous studies (De Bruijn et al., 2004; De Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006; Stern et al., 2010).

apathy, anhedonia and psychomotor retardation (Schrijvers et al., 2008; Schrijvers et al., 2009; Schrijvers, Hulstijn, & Sabbe, 2008). On the other hand, our new results showing a trend towards an increase in the amplitude of the ERN/Ne component with increasing levels of trait anxiety in HCs (r = -.45, p = .08) as well as a slightly larger (though non-significant) ERN/Ne in MDD patients compared to HCs, both confirm that this early error-monitoring activity is influenced by affective or motivational factors, besides accuracy. Moreover, these data also provide support for the observation that an enhanced ERN/Ne component is usually observed in high compared to low anxious participants (Aarts & Pourtois, 2010; Endrass, Klawohn, Schuster, & Kathmann, 2008; Gehring et al., 2000; Hajcak et al., 2003a, 2004; Hajcak & Simons, 2002; Johannes et al., 2001; Pailing & Segalowitz, 2004). Using a complementary topographical and source localization analysis, we also confirmed that the ERN/Ne component was related to brain generators within the medial frontal gyrus (BA6) and dACC (BA24), whereas during the same early time interval following response onset, the monitoring of hits was associated with activity in medial frontal gyrus (BA6), as well as in non-overlapping cortical regions, including posterior parietal regions (BA7, with an activation extending towards BA 31) (see also Aarts & Pourtois, 2010). The contribution of Premotor/Supplementary motor area and/or the dACC in early error-monitoring processes is consistent with previous ERP and fMRI studies (Dehaene et al., 1994; Herrmann et al., 2004; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Miltner, Braun, & Coles, 1997; O'Connell et al., 2007; Ullsperger & von Cramon, 2004). Interestingly, we also found that the ERN/Ne of MDD patients was accounted for by an enhanced MFC/dLPFC (BA8 and BA9) activity, relative to HCs. A direct comparison between groups confirmed that MDD recruited extra dLPFC areas (BA6, BA8 and BA9), which have generally been implicated in cognitive control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Duncan & Owen, 2000; Miller & Cohen, 2001; Ridderinkhof et al., 2004). Other studies (Hoehn-Saric, Lee, McLeod, & Wong, 2005; Sinha, Mohlman, & Gorman, 2004; Thayer & Lane, 2000; Wu et al., 1991) have also related an increased dlPFC activity to augmented ruminative thinking or worry, which is a landmark of MDD (Nolen-Hoeksema, 2000; Nolen-Hoeksema et al., 2008).

Accordingly, the observed enhanced dlPFC activity found in MDD patients during the early monitoring/detection of response errors (besides the normal dACC activation, shared with HCs) might be related to intrusive ruminative processes, that would affect the interplay between dlPFC and ACC during error-monitoring and accordingly the size of this error-related activity, as measured using scalp recordings (see Pizzagalli, 2011). However, we note that we did not find evidence for a clear link between levels of trait-like ruminative thinking (as estimated using the RRS, see Nolen-Hoeksema & Morrow; Raes, Hermans, & Eelen, 2003) and amplitude variation at the level of the ERN/Ne component. Such a relationship was however revealed with the subsequent error-related ERP component, the Pe that was markedly blunted in MDD patients relative to HCs.

MDD alters error-monitoring process reflected by the Pe component

The results of our study unambiguously show that MDD patients have a substantially smaller Pe component, relative to HCs during early error monitoring, in the absence of obvious difference at the behavioral level between these two groups. At first sight, this result is consistent with earlier ERP studies examining error-monitoring brain functions in severely depressed patients (Olvet, Klein, & Hajcak, 2010; Schrijvers et al., 2008; Schrijvers et al., 2009). This finding is however at variance with other studies performed in mildly to moderately depressed individuals (Chiu & Deldin, 2007; Compton et al., 2008; Holmes & Pizzagalli, 2008) and where no systematic alteration or reduction of the Pe component was found. A decreased Pe component during error monitoring in MDD patients might potentially be explained either by symptom severity (which is stronger in MDD patients, as tested in our study, compared to moderately depressed individuals in some of these earlier studies), or by alterations of specific cognitive control processes involved in the early monitoring of response errors. First, since MDD is characterized by general apathy or blunted affect (DSM-IV-TR, APA, 2000), it might be that these patients also have overall blunted emotional reactions, including to their own/self-generated errors, which are nonetheless usually distinctive or salient events from a motivational point of view (Aarts, De Houwer, & Pourtois, in revision; Luu,

Collins, & Tucker, 2000). Consistent with this view, previous studies have linked the error-related Pe component to an emotional reaction to these adverse events (van Veen & Carter, 2002). Alternatively, given the generally impaired motivation in MDD patients (DSM-IV-TR, APA, 2000) and the link between the Pe component and the motivational significance of an error (Overbeek et al., 2005), this effect might translate a change in the detection of an otherwise salient or behaviorally relevant event (i.e. unwanted response error). We note however that the post-error adjustment following errors (Danielmeier & Ullsperger, 2011; Notebaert et al., 2009; Rabbitt, 1966) was spared, though reduced in MDD patients relative to HCs in our study. Moreover, because MDD patients made as many fast hits as HCs (indicating indirectly that they were equally able to comply with task demands than HCs), a mere change in levels of "intrinsic" motivation during the task cannot easily account for the present ERP results. Hence, a decreased motivational saliency account seems unlikely given the normal, spared attention or cognitive control reaction following the detection of these (rare) negative events in MDD, as well as their preserved ERN/Ne component (Hajcak, Moser, Yeung, & Simons, 2005). Such an impairment could perhaps be revealed if more complex discrimination or interference tasks would be used to probe changes in early error-monitoring brain functions in MDD patients, unlike the more simple inhibition of a pre-potent response tendency, as explored using the present speeded Go/noGo task (Miyake et al., 2000). In these circumstances, it remains to be established whether a decreased error-related Pe component could be associated or even predict mal-adaptive behavioral changes following the detection of these events (e.g. a blunted post-error slowing effect). Finally, it might be the case that a blunted Pe component in MDD patients could indirectly result from ruminative thinking, or the consequence of this specific cognitive style. Consistent with this view, we found that ruminative thinking was actually the best predictor of (a reduction of) the Pe component recorded in our study. Because MDD patients automatically and repetitively focus on and orient to their distress, negative feelings and negative thoughts, it might be that these general intrusive thoughts, and maybe also negative thoughts selectively triggered by unwanted error commission, would consume specific attentional or cognitive

control resources that are normally used by Pe brain systems to timely and efficiently monitor and register these incorrect actions. In this view, the accumulation of evidence process leading to the conscious detection of a response error, as reflected by the Pe component (Steinhauser & Yeung, 2010), would be impaired since other intrusive thoughts may prevent its normal unfolding. This limited resource account is also consistent with the idea that the Pe somehow reflects a "bottom-up" attentional orienting process, similarly to the P300 component (Ridderinkhof et al., 2009). Presumably, if less "bottom-up" attention is allocated to the (internal) monitoring of actions and errors (because attention resources are used by a concurrent mental process, for example rumination), this monitoring and the conscious registration of these errors are by definition less effective or sharp. Interestingly, previous studies already reported a decreased noGo P300 in depressed individuals (Ruchsow et al, 2008). Hence, in this framework, the blunted Pe component in MDD patients would correspond to a more general deficit of bottom-up attention control, when this control has to be exerted on internal/motor representations.

At any rate, future studies are needed to validate this conjecture and assess whether (i) the automatic detection of unwanted response errors in "high ruminators" (who are usually depressed) may more easily trigger an extra burst of rumination or negative intrusive thoughts (Conway et al., 1991), and (ii) this early effect predicts, or is causally related to a reduction of the Pe component during early error monitoring. In this regard, it might be valuable to assess possible changes in early error-related brain processes (with a focus on the ERN/Ne and Pe ERP components) of MDD patients after they completed a treatment or followed a cognitive behavioral therapy aimed at reducing adverse effects of ruminative thinking (Moore & Malinowski, 2009; Siegle, Ghinassi, & Thase, 2007). Likewise, since adverse effects of rumination may transiently be suppressed or downplayed (by using for instance specific verbal working memory strategies/manipulations, see McEvoy, Smith, & Gevins, 1998; McNamara & Scott, 2001), it may turn out to be valuable to assess whether the use of these specific cognitive strategies may help restore a normal Pe component during

error monitoring in MDD patients. Such a positive outcome would strengthen the link between this specific ERP component and its selective impairment in MDD due to interfering accessory ruminative thinking processes.

ERN/Ne and Pe reflect different stages of error monitoring

More generally, our new ERP results confirm that the ERN/Ne and Pe component likely reflect two different functional stages during early error-monitoring (Overbeek et al., 2005). First, we confirmed that they were each associated with different brain networks and they were each differently influenced by MDD. While in our previous study using the exact same task and ERP methodology we showed that sub clinical trait anxiety influenced primarily the ERN/Ne component and its underlying brain generators, with no change at the level of the Pe component (Aarts & Pourtois, 2010), we found in this study a complementary picture. MDD primarily influenced the Pe component, while leaving almost untouched the ERN/Ne, though this latter component was slightly increased, compared to HCs. Note that this latter result rules out the possibility that MDD would simply be associated with a general reduction of brain activations (and hence the resulting ERP signal) during error monitoring. This dissociation between the ERN/Ne and Pe revealed by different psychopathology conditions (sub clinical trait anxiety vs. MDD; two internalizing disorders; see Krueger, 1999) indirectly suggests that these two components likely reflect different monitoring processes during early stages of error detection. In light of this dissociation, we advocate that effects of anxiety at the level of the ERN/Ne (i.e. augmented ERN/Ne, but not Pe) might actually reflect an automatic compensatory "attention control"/effort mechanism used by high anxious individuals to cope with their worrisome thoughts probably concerning the avoidance of errors/failures/adverse events in the future that are perceived as challenging self-efficacy (see Eysenck, Derakshan, Santos, & Calvo, 2007 for a similar view). Alternatively, an enhanced ERN/Ne in high anxious individuals could translate the activation of additional cognitive or emotional control brain regions (possibly involving a more rostral ACC regions), as indirectly confirmed in our previous ERP study (Aarts & Pourtois, 2010). By contrast, MDD patients, who

are characterized by an inability to experience positive affect (anhedonia), impaired motivation and by a perseverative focus on negative thoughts or feelings (i.e. rumination), are no more able to automatically regulate error detection alike (and hence they show a normal ERN/Ne component), while this excessive ruminative thinking style inadvertently consumes resources away from the main error-monitoring function, which in turn leads to a blunted Pe component in these patients. Future studies (possibly crossing data from psychology, biology and epidemiology) are needed to establish whether changes in the expression of these two early error-related brain components are related to different psychopathology conditions (sub clinical trait anxiety vs. MDD) and may eventually provide stable endophenotypes, as recently put forward in the literature (Olvet & Hajcak, 2008).

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CHAPTER 4: ANXIETY DISRUPTS THE EVALUATIVE COMPONENT OF PERFORMANCE MONITORING: AN ERP STUDY [1]

"Thirty low and 30 high anxious participants performed a speeded Go/noGo task during which they had to rely on evaluative feedback to infer whether their actions were timely (correct) or not. We focused on FRN, an ERP component that is sensitive to the valence of feedback. Depending on the context, neutral faces served either as positive or negative feedback. Whereas the FRN of low anxious individuals did discriminate between neutral faces when used either as positive or negative feedback, the FRN of high anxious individuals did not. However, before the FRN, we also found evidence for a differential perceptual effect at the level of the N170 face-specific component between the two feedback conditions, equally so in low and high anxious individuals. These results suggest that anxiety disrupts selectively the evaluative component of performance monitoring, which presumably allows to ascribe a given value (either positive or negative) to actions."

[1] Aarts & Pourtois (2012). Anxiety disrupts the evaluative component of performance monitoring: An ERP study. Neuropsychologia.

INTRODUCTION

Depending on the situation and circumstances, the control of behavior is based on the monitoring of either internal or external signals, or sometimes a combination of both. For example, the adequacy of a given action in response to a familiar stimulus may be determined based on an internal representation allowing to compare the discrepancy between the actual and expected or desired action, with a swift detection of any divergence between the two (Gehring, Goss, Coles, Meyer, & Donchin, 1993). However, in many situations, performance monitoring cannot be achieved solely based on the processing of internal signals, but the processing of new external feedback information in the environment is required to establish whether the current action is appropriate (e.g. timely or correct), or not. Hence, the processing of feedback information available in the environment often indicates the appropriateness of certain actions and in turn allows to correct or adjust behavior if required, eventually leading to learning and preventing errors from recurring in the future (Holroyd & Coles, 2002; Rabbitt, 1966).

Several ERP studies looking at outcome evaluation processes based on external feedback have described an ERP component, the feedback-related negativity (FRN) that is selectively associated with the processing of the valence or motivational significance of the feedback (Gehring & Willoughby, 2002; Holroyd & Coles, 2002; Miltner, Braun, & Coles, 1997). The FRN is a negative component peaking at fronto-central electrodes roughly 250-300 ms after presentation of relevant feedback information. Usually, the FRN was found to be larger after negative feedback on task performance (e.g. the presentation of an evaluation signal indicating error commission or monetary loss) compared to positive feedback (e.g. the presentation of an evaluation signal indicating correct performance or monetary reward; Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003). These findings point to the involvement of the FRN in the processing of the valence or reward value of the feedback. Interestingly, the FRN component shares many electrophysiological properties with another ERP component, the

error-related negativity (ERN/Ne; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring et al., 1993), which is also involved in performance monitoring, though based on the processing of internal error signals. The ERN/Ne is a negative component generated roughly 50-100 ms following error commission over fronto-central scalp electrodes. In both cases, this negative ERP component would reflect the activation of a reinforcement learning system within the dorsal Anterior Cingulate Cortex (dACC) that enables a rapid evaluation of outcomes or actions (Frank, Woroch, & Curran, 2005; Holroyd & Coles, 2002).

Noteworthy, although the FRN primarily reflects an evaluative component, this ERP component is also permeable to individual differences in affect. Because the hypersensitivity to negative events and the tendency to worry about negative outcomes are hallmarks of several affective personality traits or disorders like anxiety and depression (Maner & Schmidt, 2006; Mineka, Rafaeli, & Jovel, 2003; Wray & Stone, 2005), one may assume that performance monitoring may vary with these affective personality traits. Consistent with this hypothesis, several studies have reported an effect of anxiety or depression on the ERN/Ne (e.g. Aarts & Pourtois, 2010; Holmes & Pizzagalli, 2008; Olvet & Hajcak, 2008). By contrast, the evidence supporting a systematic modulation of the FRN (and hence the processing of external evaluative feedback) as a function of negative affect is mixed. In a recent study, De Pascalis, Varriale, & D'Antuono (2010) found that individuals who were more sensitive to punishment (as measured using the BIS/BAS; see Carver & White, 1994) had a larger FRN to monetary loss following incorrect noGo trials during a Go/noGo task. In an earlier ERP study, Tucker, Luu, Frishkoff, Quiring, & Poulsen (2003) found that (clinically) depressed patients had increased FRN following all feedback (i.e. feedback following fast, medium as well as slow responses). Surprisingly, moderately depressed individuals showed larger FRN following feedback evaluating slow responses compared to the FRN amplitude in severely depressed patients. In contrast to these results, Foti and Hajcak (2009) reported a blunted difference in FRN amplitude between negative (non-reward) and positive (reward) feedback in depressed individuals. When turning to anxiety, which is usually related to

depression (Beck, Epstein, Brown, & Steer, 1988; Mendels, Weinstein, & Cochrane, 1972) and punishment sensitivity (Bijttebier, Beck, Claes, & Vandereycken, 2009), but which is also mainly characterized by an extreme worry about the expectancy of possible failures in the future (Eisenberg, Baron, & Seligman, 1998; Mitte, 2007; Shepperd, Grace, Cole, & Klein, 2005), the results of two studies converged and showed a larger FRN amplitude for low, compared to high anxious individuals (Gu, Huang, & Luo, 2010; Simons, 2010). According to Yeung, Holroyd, & Cohen (2005), the FRN also reflects an evaluation process that is influenced by the motivational significance of ongoing actions. These authors reported a correlation between the amplitude of the FRN and the subjective involvement in the task. Consistent with this notion, two recent ERP studies confirmed that evaluative feedback processing (and hence the FRN component) is also influenced by higher-level cognitive or motivational factors (i.e. responsibility; see Li et al., 2010, and empathy; see Fukushima & Hiraki, 2009), which may, depending on the context or situation, make the evaluative feedback stimulus more or less salient. Hence, depending on the specific goals and needs, the FRN may vary in magnitude in response to evaluative performance feedback. These studies therefore confirm that motivational significance (besides valence) may be an important determinant of the amplitude modulations of the FRN found during standard performance-monitoring tasks. More generally, these results suggest that the FRN component is not encapsulated or immune to higher-level motivational or emotional factors, such that the affective predispositions of the participant may in principle modulate the size and expression of this performance-monitoring ERP component. In this study, we tested this prediction and compared the FRN of low vs. high trait anxious individuals during a standard speeded Go/noGo task.

The goal of our study was to investigate effects of sub clinical trait anxiety on performance monitoring, when this process primarily relies on the processing of external evaluative feedback (with a focus on the FRN component therefore). Notably, these external feedback consisted of neutral and emotional faces in our study, because these visual stimuli usually provide important social and

ecologically-valid signals used to gauge the actions and intentions of our conspecifics in daily life situations. Moreover, because emotional faces are complex stimuli that carry an intrinsic emotional value (when compared to abstract symbolic cues) and because negative emotional faces might be perceived or attended differentially in high compared to low anxious individuals (Fox, Russo, & Dutton, 2002; Knyazev, Bocharov, Slobodskaya, & Ryabichenko, 2008), we used an experimental procedure enabling to explore performance-monitoring brain effects when the intrinsic valence/pleasantness of the feedback stimulus was controlled for and eventually neutralized. More specifically, we compared performance monitoring (i.e. FRN) of low vs. high trait anxious participants when the feedback information used was kept constant (i.e. the same neutral visual stimuli served as performance feedback), but the perceived experimental situation could be either "positive" or "negative". This manipulation allowed us to compare the exact same physical stimuli (i.e. neutral faces) used as performance feedback for positive outcomes in one context and for negative outcomes in the other, and test if performance-monitoring brain processes (with a focus on the FRN component) differed between low vs. high trait anxious individuals.

We tested the hypothesis that performance-monitoring processes of high anxious participants based on the processing of external evaluative feedback may be impaired, reflected by a blunted FRN to negative feedback in these participants. More specifically, we surmised that the impairment in high anxious individuals does not translate a relative insensitivity to outcome evaluation in general, but reflects instead a failure to readily compare the perceived valence of the feedback with the inferred (internalized) value of the action (just performed). In this framework, a blunted FRN component may reflect an inability to relate the valence of the feedback (either positive or negative) to the internalized value of the action (that has been made prior to feedback delivery and therefore awaits evaluation; see Holroyd & Coles, 2002). To indirectly validate this assumption, we also explored the possible relationship between "locus of control" (LOC; Rotter, 1966) and the FRN component. The LOC provides an

estimate of attribution style, defined as the disposition to ascribe the cause of actions or events to either internal or external drives or forces. We reasoned that participants with an internal (as opposed to external) LOC may probably more easily relate or integrate the value of the (external) evaluative feedback with the (internally-generated) action (i.e. cause) they have just made and which is evaluated by the feedback. Accordingly, if the FRN reflects the integration process linking the perceived valence of the feedback with the internalized value of the action (just performed) during performance monitoring, we may thus predict a larger FRN for individuals characterized by a more internal (as opposed to external) LOC. Moreover, because earlier studies found a relationship between LOC and trait anxiety (i.e. high anxious individuals have a more external LOC; see Archer, 1979), we sought to assess whether higher levels of trait anxiety may somehow downplay the possible link between LOC and the FRN (see also Gu et al., 2010; Hajcak, McDonald, & Simons, 2003a).

Although we mainly focused on the FRN component in this study, given the strong link between this specific ERP deflection and performance-monitoring processes (Holroyd & Coles, 2002), we could also explore whether trait anxiety and/or the perceived valence of the feedback not only influenced the FRN component, but also an earlier structural encoding stage during evaluative feedback processing. Faces elicit a well-described category-selective ERP component (i.e. the N170), which reflects structural encoding (Bentin, Allison, Puce, Perez, & McCarthy, 1996; George et al., 1996). This component peaks 150-170 ms after face stimulus onset with a maximum amplitude over right lateral occipital-temporal and hence it can easily be dissociated in time and space from the FRN deflection. Although some previous ERP studies have failed to reveal any change of the N170 amplitude with the emotional facial expression content of the faces (Eimer & Holmes, 2002), other studies have reported systematic modulations of this category-selective ERP component with emotional facial expressions, especially so for negative expressions such as fear and anger for which the amplitude of the N170 was augmented, compared to a neutral facial expression (Batty & Taylor, 2003; Campanella, Quinet, Bruyer, Crommelinck, &

Guerit, 2002; Righart & de Gelder, 2006; Vuilleumier & Pourtois, 2007). Based on these previous ERP results (Vuilleumier & Pourtois, 2007), we surmised that the N170 would be larger for neutral faces used as negative feedback, compared to positive feedback. By contrast, since previous ERP studies mainly failed to provide evidence for a clear effect of anxiety at this early stage of face processing (Kolassa, Kolassa, Musial, & Miltner, 2007; Kolassa & Miltner, 2006; Muhlberger et al., 2009; Rossignol, Philippot, Douillez, Crommelinck, & Campanella, 2005), we did not predict any strong effect of trait anxiety on the amplitude of the N170.

METHODS

Participants

A total of 73 undergraduate students participated in this experiment in exchange of 20 Euro payment. Ten individuals had later to be excluded from the analysis due to an obvious discrepancy between the level of trait anxiety measured by the STAI-T during the pre-screening phase (at the beginning of the academic year) and their actual level of trait anxiety measured a second time at the day of testing (2–6 months later). Moreover, the data of 3 other participants had to be disregarded due to excessive noise and artifacts during the EEG recording. Hence, the final sample consisted of 60 participants. Using a standard median-split (*Me* = 37), we created a group of sub clinical high trait anxious participants and a group of low trait anxious participants. These two groups did not differ with respect to age and gender (see Table 1). They were all right handed, had no history of psychiatry or neurological disease, were free of any psychoactive medication and had normal or corrected-to-normal vision. They gave written informed consent prior to the experiment, and the study was approved by the local ethical committee (Faculty of Psychology & Educational Sciences, Ghent University).

Table 1.

Descriptive statistics for the low and high anxious group

	Low anxiety				High anxiety			
	Negative context		Positive context		Negative context		Positive context	
	M	*SEM*	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
SEX	3M		2M		2M		2M	
Age	20.00	0.54	20.40	0.72	19.60	0.24	19.40	0.50
STAI-T	28.73	0.93	28.47	0.82	44.40	1.93	45.07	1.88
STAI-S1	29.67	1.51	31.13	1.37	36.40	1.67	38.53	1.78
STAI-S2	33.47	2.07	37.27	2.19	40.73	1.58	43.33	2.49
LOC	12.53	0.87	11.13	0.92	12.60	0.99	12.93	0.95

Speeded Go/noGo task

We used a modified version of a speeded Go/noGo task previously used and validated in a group of low and high (sub clinical) anxious participants (Figure 1; Vocat, Pourtois, & Vuilleumier, 2008; Aarts & Pourtois, 2010). Visual stimuli were shown on a 19-inch LCD screen. They consisted of an arrow (11.4° x 0.05° of visual angle at a 60 cm viewing distance) that was presented in the center of the screen on a white background. Each trial started with a black fixation cross that lasted for 1000 ms. Then, a black arrow (i.e. cue) either oriented up or down, was presented. After a variable interval ranging from 1000 ms up to 2000 ms, the black arrow became either green or turquoise while its orientation could either remain identical or shift in the opposite direction. When the black arrow turned green and the orientation remained unchanged, participants were instructed to press a predefined key on the response box as fast as possible with the index finger of their right hand (Go trials). However, participants had to withhold responding when either the arrow became green but changed orientation, or when the arrow became turquoise and kept its initial orientation. For noGo trials, this color arrow remained on the screen for a maximum duration of 1000 ms. Instructions emphasized both speed and accuracy. After the response, feedback was presented for 1000 ms (a 1000 ms blank screen preceded this feedback).

We used an online adaptive algorithm to set up a limit for "correct"/fast reaction times (RTs) (i.e. deadline procedure). The rationale of this procedure was to facilitate the occurrence of fast decisions and in turn increase uncertainty regarding the actual speed. At the beginning of the experiment, the RT limit was set to 300 ms (this cutoff was determined based on previous pilot testing; Vocat et al., 2008). This limit was adjusted online as a function of the immediately preceding trial history, more specifically as the mean of current and previous RT. If the current RT was slower than this limit (arbitrarily classified as "slow hit"), the participant received negative feedback. If the RT was faster than the limit, positive feedback was presented (arbitrarily classified as "fast hit"). Hence, feedback was used to stress both speed and accuracy. When the response was incorrect (i.e. either a false alarm - response on noGo trial, or an omission - absence of response on Go trial), negative feedback was presented alike. By contrast, participants received positive feedback when they correctly withheld responding on noGo trials. The added value of this adaptive algorithm is that uncertainty about speed RT is actually high throughout the task, which motivates participants to actively attend to the feedback information displayed systematically after each response in such a way to infer whether their actions are timely (fast hits/positive feedback) or not (slow hits/negative feedback). By contrast, feedback following actions on noGo trials, either correct inhibitions or false alarms, was not informative as participants could readily evaluate the accuracy of their actions on noGo trials using internal monitoring systems. Therefore, we primarily focused on the ERP responses to evaluative feedback following correct Go trials, corresponding either to fast hits (positive feedback) or slow hits (negative feedback).

Feedback on task performance consisted of emotional or neutral faces. However, in order to control for the intrinsic emotional value of these faces (and focus on performance-monitoring processes), we created two different emotional contexts such that we could compare the exact same neutral face stimuli used in two opposite situations (either a positive outcome/fast hit or a negative outcome/slow hit). More specifically, in the positive context, neutral

faces served as negative feedback (slow hits) and were presented together with happy faces that served as positive feedback (fast hits, see Figure 1A). By contrast, in the negative context, neutral faces served as positive feedback (fast hits), and were presented together with angry faces that were used as negative feedback (slow hits, see Figure 1B). Each participant (*n* = 60; 30 low and 30 high anxious) was randomly assigned to one of these two emotional contexts (hence this variable was a between-subject factor). As a result, 4 experimental groups of equal sizes (*n* = 15) were created by crossing trait anxiety level (low vs. high) and emotional context (negative vs. positive).

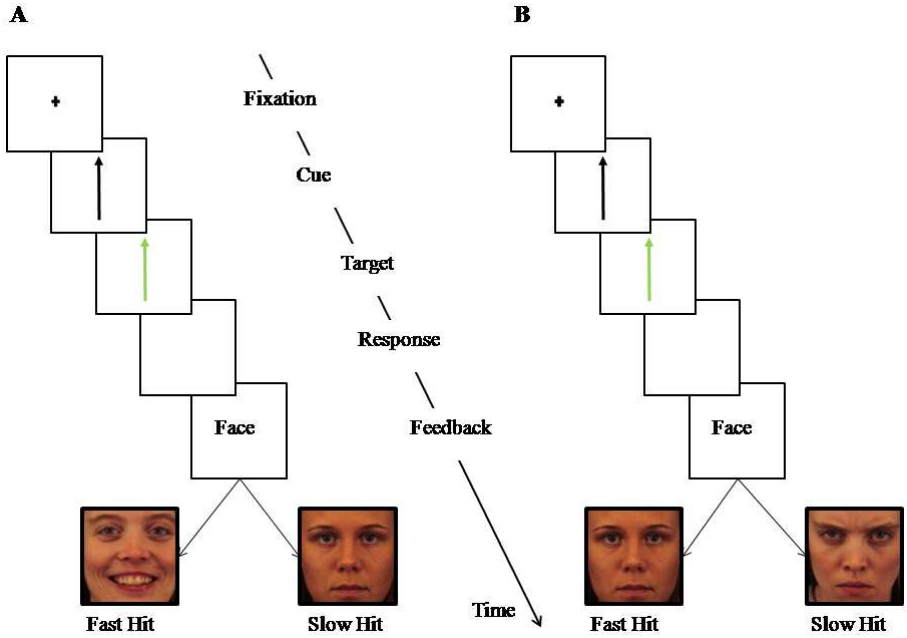


Figure 1. Stimuli and task. (A) In the positive context, neutral faces were used as negative feedback and happy faces as positive feedback. (B) By contrast, in the negative context, angry faces were used as negative feedback whereas the exact same neutral faces were used as positive feedback.

The experiment consisted of 60 practice trials and 360 test trials. The test trials were divided into 6 blocks of 60 trials each (40 Go and 20 noGo trials, 10 of each type). Trial presentation was randomized within blocks. After the first block, the experimenter emphasized again the importance of speed as well as accuracy in this task. Between blocks, a brief self-paced pause (always shorter than 5 min) was implemented. Stimulus presentation and response recording were

controlled using E-prime software (V2.0., http://www.pstnet.com/products/e-prime/).

Face stimuli

Ten different face identities (5 per gender) displaying a neutral, happy or angry emotional expression were selected from the Karolinska Directed Emotional Faces database (KDEF; Lundqvist, Flykt, & Öhman, 1998). Within each emotional expression category (i.e. angry, happy and neutral) faces were selected randomly in order to control for differences in identity and gender between negative and positive feedback. Based on independent ratings obtained for these 10 faces (Goeleven, De Raedt, Leyman, & Verschuere, 2008), we could establish that the arousal and intensity level of these faces did not differ significantly between angry and happy faces, $t < 1$. The neutral faces were rated as less arousing and intense compared either to the angry faces (intensity: $t(18) = 3.70$, $p < .005$; arousal: $t(18) = 6.90$, $p < .001$) or the happy faces (intensity: $t(18) = 6.15$, $p < .001$; arousal: $t(18) = 11.30$, $p < .001$). After completing the task, every face used during the experiment was presented again one by one to each participant and he/she was asked to rate the valence of the face using a visual analog scale ranging from -50 (very negative) to +50 (very positive). The face remained on the screen until response. These subjective ratings of the faces allowed us (i) to check that the emotion (or lack of) displayed by the face was properly recognized as such by participants, and (ii) more importantly, to assess whether the valence of neutral faces would reliably vary across the two emotional contexts, in a predictive way (i.e. neutral faces in the positive context would be judged as relatively more negative, whereas neutral faces in the negative context would be judged as relatively more positive). Hence, these subjective ratings of the faces also provided an indirect check of the manipulation of the emotional context performed in our study.

Questionnaires

We measured levels of state anxiety both before and after the Go/noGo task, using the state version of the STAI. Importantly, we also measured the

attribution style and more specifically the LOC of each participant, using a standard questionnaire (Rotter, 1966). This questionnaire may be useful, as it provides an estimate of the inclination of participants to attribute outcomes in daily life situations to either internal as opposed to more external causes. Higher LOC scores correspond to a tendency to attribute the cause of events or situations to external drives or forces. Previous studies generally showed a positive relationship between externality and trait anxiety (Archer, 1979) and such positive correlation was also confirmed in our study in the low (r = .50, p < .005) but not in the high anxious group (r = .11, p >.10).

EEG acquisition

Continuous EEG was acquired at 512 Hz using a 128-channel (pin-type) Biosemi Active Two system (http://www.biosemi.com) referenced to the Common Mode Sense (CMS)-Driven Right Leg (DRL) ground. ERPs of interest were computed offline following a standard sequence of data transformations (Picton et al., 2000): (1) Re-referencing of the EEG signal using a common average reference; (2) -500/+1000 ms segmentation around the onset of the feedback stimulus; (3) pre-stimulus interval baseline correction (from -500 ms to feedback onset); (4) vertical ocular correction for blinks (Gratton, Coles, & Donchin, 1983) using the difference amplitude of two electrodes attached approximately 1 cm above and below the left eye; (5) a second pre-stimulus interval baseline correction (from -500 ms to feedback onset); (6) semi-automatic artifact rejection [electrodes with 20% or more noise at an amplitude level of 100 μV were excluded, M = 6 electrodes, SEM = 1; no significant difference between groups (low vs. high anxiety) and contexts (negative vs. positive), F(1, 56) = 1.61, p > .10; amplitude (μ) scale across participants, M = -85/+85, SEM = 2; no significant difference between groups and contexts, F(1, 56) = .12, p > .10; % of rejected artifacts: M = 14, SEM = 1; no significant difference between groups and contexts, F(1, 56) = .97, p > .10]; (7) averaging of the stimulus-locked ERPs for each type of feedback separately (i.e. negative feedback following a slow hit and positive feedback following a fast hit), and (8) low pass digital filtering of the individual average data (30 Hz).

We primarily focused on two well-documented ERP components, the FRN and the N170. Because peak or area measures of the FRN may confound variation in the FRN with differences in other adjacent ERP components, such as the P300, the FRN was measured base-to-peak over a fronto-central electrode along the midline (i.e. electrode FCz) where the FRN reaches its maximum amplitude (see Holroyd et al., 2004) 150-350 ms after feedback onset. More specifically and following standard practice (see Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003), the FRN amplitude was quantified as the difference between (i) the maximum amplitude value between 150 ms and 250 ms following feedback onset at electrode FCz and (ii) the most negative amplitude value occurring between this first maximum and up to 350 ms after feedback onset at the same electrode location. The N170 amplitude was measured at occipito-temporal sites (left electrodes: D30, D31, D32, A9, A10 and A11; right: B6, B7, B8, B10, B11 and B12) as the maximal negative peak amplitude occurring during a restricted time-window spanning from 150 to 200 ms post-face stimulus (feedback) onset (see Bentin et al., 1996).

Data analyses

RTs faster than 150 ms and slower than 500 ms were removed from the analyses (see also Aarts & Pourtois, 2010). Using these criteria, 0.42 % (*SEM* = 0.13) of the RT data were found to be faster than 150 ms while 2.69 % (*SEM* = 0.40) were slower than 500 ms. In total, 3.11 % of the RT data were eventually removed. The percentage of outliers was similar between groups (RTs faster than 150 ms: $F(1, 56) = .61$, $p > .10$; RTs slower than 500 ms: $F(1,56) = .06$, $p > .10$) and contexts (RTs faster than 150 ms: $F(1, 56) = .34$, $p > .10$; RTs slower than 500 ms: $F(1,56) = 1.63$, $p > .10$), and no significant interaction was found between those two factors (RTs faster than 150 ms: $F(1, 56) = 1.64$, $p > .10$; RTs slower than 500 ms: $F(1,56) = 1.87$, $p > .10$).

Because the presentation of feedback information following correct inhibitions (on noGo trials) or response errors (i.e. False Alarms on noGo trials), was not informative, only ERP components in response to feedback following fast (positive feedback) and slow hits (negative feedback) were included in the

analyses. Unlike response errors or correct inhibitions, in these two conditions, participants had actually to rely on external feedback information to determine, given the speed pressure imposed, whether their responses were “correct” (fast) or not (slow), relative to the arbitrary limit updated on a trial-by-trial basis. We first performed statistical analyses in which we directly compared the exact same feedback stimuli (neutral faces) used either as positive (fast hits) or negative (slow hits) outcome. These analyses enabled to exclude low-level differences (as well as intrinsic pleasantness) between these two opposite evaluative outcomes.

N170 peak amplitudes were analyzed using a mixed model ANOVA including the between-subject factors group (low vs. high anxiety) and context (negative vs. positive), and the within-subject factor electrode position (6), as well as hemisphere (right vs. left). The last within-subject factor was included in the analysis to verify if the N170 component recorded in this study was larger in the right compared to the left hemisphere (Bentin, et al., 1996; Itier & Taylor, 2004). We also ran an auxiliary analysis in which we examined amplitude modulations of the N170 for emotional as well as neutral faces. In this more complex model, N170 peak amplitudes were analyzed using a mixed model ANOVA including the between-subject factors group (low vs. high anxiety) and context (negative vs. positive), and the within-subject factor electrode position (6), valence of feedback (negative vs. positive) and hemisphere (right vs. left).

FRN base-to-peak amplitudes were first analyzed for neutral faces only using a mixed model ANOVA including the between-subject factors group (low vs. high anxiety) and context (negative vs. positive). Next, FRN base-to-peak amplitudes were analyzed for emotional and neutral faces using an ANOVA including the factors group (low vs. high anxiety) and context (negative vs. positive) and the within-subject factor valence of feedback (negative vs. positive).

RESULTS

Trait anxiety

Participants of each group (low vs. high anxiety) were randomly assigned to one of the two contexts (negative vs. positive). As expected, trait anxiety differed significantly between groups, $F(1, 56) = 118.49$, $p < .001$, while no main effect of context, $F(1, 56) = .02$, $p > .10$, and no interaction between group and context was observed, $F(1, 56) = .10$, $p > .10$.

Subjective ratings of the faces

At the end of the experimental session, participants were asked to rate the valence of every face used as performance feedback using a visual analog scale ranging from negative (-50) to positive (+50) values. Due to technical problems, the rating data of two low anxious individuals who were assigned to the positive context could not be saved properly and were lost. Critically, neutral faces in the positive context were evaluated as more negative ($M = -18.30$, $SEM = 1.53$) compared to the same neutral faces presented in the negative context ($M = 12.00$, $SEM = 1.92$), $F(1, 54) = 151.28$, $p < .001$, confirming that these neutral faces used as feedback had acquired a differential valence depending on the emotional context. This effect was not different for low vs. high anxious participants, $F(1, 54) = .77$, $p > .10$. No significant main effect of trait anxiety was evidenced on these ratings, $F(1, 54) = 2.14$, $p > .10$. Happy and angry faces were, as expected, clearly rated as positive ($M = 33.85$, $SEM = 1.37$) and negative ($M = -33.83$; $SEM = 1.12$), respectively, but these ratings did not differ between low and high anxious participants, $F(1, 54) = .004$, $p > .10$.

State anxiety

As expected, the level of state anxiety before the task differed significantly between the two groups, $F(1, 56) = 19.73$, $p < .001$ (see Table 1). After the Go/noGo task, this level of state anxiety reliably increased (see also Aarts & Pourtois, 2010, for similar finding), $F(1, 56) = 23.88$, $p < .001$, but low trait anxious individuals still had a lower level of state anxiety than high trait anxious

individuals, $F(1, 56) = 10.00$, $p < .005$. This increase in state anxiety level was not influenced by context, $F(1, 56) = .51$, $p > .10$, neither did context interact significantly with group, $F(1, 56) = .23$, $p > .10$. These results confirmed that the Go/noGo task was demanding, and that the constant and updated speed pressure imposed likely led to an increased experience of negative affect (equally so in both groups and contexts), given the intrinsic difficulty to keep producing fast correct responses throughout the experimental session in these conditions (see Aarts & Pourtois, 2010).

Behavioral results

After each trial, feedback on task performance was presented. Negative feedback (either a neutral face in the positive context or an angry face in the negative context) was presented following response errors (i.e. False Alarms or Slow Hits), while positive feedback (either a neutral face in the negative context or a happy face in the positive context) was presented following correct inhibitions (on noGo trials) or fast hits. Performance during the Go/noGo task was comparable between groups (low vs. high anxiety) and contexts and no significant interaction between group and context was evidenced (see Table 2a and 2b). Participants committed on average 24% or 29 errors in the speeded Go/noGo task and this percentage/number did not differ between groups, $F(1, 56) = 2.12$, $p > .10$, and contexts, $F(1, 56) = 1,30$, $p > .10$. Similarly, no significant differences in the number of fast or slow hits were observed between groups (fast hits: $F(1, 56) = .44$, $p > .10$; slow hits: $F(1, 56) = .74$, $p > .10$) and contexts (fast hits: $F(1, 56) = 2.63$, $p > .10$; slow hits: $F(1, 56) = 1.80$, $p > .10$), and the interaction between group and context did not reach significance (fast hits: $F(1, 56) = .00$, $p > .10$; slow hits: $F(1, 56) = .70$, $p > .10$) (see Table 2a). As expected (see Aarts & Pourtois, 2010), participants reacted faster on incorrect noGo trials ($M = 248.67$, $SEM = 3.57$) than on slow hits ($M = 310.47$, $SEM = 2.96$), $F(1, 56) = 677.00$, $p < .001$, but faster on fast hits ($M = 233.14$, $SEM = 2.31$), $F(1, 56) = 7235.83$, $p < .001$. These RTs were comparable for both groups and contexts (all p's $> .10$). Moreover, a typical post-error slowing effect was observed indicated by slower decisions to hits following an error compared to hits following another

hit, $F(1, 56) = 50.03$, $p < .001$. This effect was not different between contexts, $F(1, 56) = .01$, $p > .10$, and groups, $F(1, 56) = 2.62$, $p > .10$, nor did the interaction between group and context reach significance, $F(1, 56) = .53$, $p > .10$ (see Table 2b), suggesting preserved behavioral performance and cognitive control abilities in the two groups and two contexts. Altogether, these behavioral results showed comparable performance (accuracy and speed) for low and high anxious participants, and for the two emotional contexts. This allowed us to compare the feedback-related ERP effects between groups and contexts, while the number of positive and negative feedback was balanced across groups and conditions.

Table 2a.

Accuracy results in the speeded Go/noGo task

		Accuracy (Number)					
		Fast Hits		Slow Hits		Errors	
Anxiety	Context	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
Low	Negative	73	7	158	6	28	5
	Positive	82	5	147	5	36	5
High	Negative	69	5	158	5	24	4
	Positive	79	6	156	5	26	4

Note: None of the group differences were significant ($p > .05$)

Table 2b.

RT results in the speeded Go/noGo task

		Speed (ms)				
		Fast Hits	Slow Hits	Errors	Post-error Hit	Post-hit Hit
Anxiety	Context	*M (SEM)*	*M (SEM)*	*M(SEM)*	*M(SEM)*	*M(SEM)*
Low	Negative	237.86 (4.56)	314.68 (6.15)	256.81 (7.22)	302.91 (8.89)	287.10 (7.76)
	Positive	226.58 (5.69)	304.45 (5.55)	242.14 (8.92)	285.30 (7.76)	273.62 (7.41)
High	Negative	234.61 (4.82)	233.50 (6.47)	247.21 (5.96)	306.78 (9.92)	286.49 (6.66)
	Positive	233.50 (2.97)	308.77 (5.72)	248.51 (6.26)	303.89 (10.07)	280.40 (6.17)

ERP results

N170 component

Visual ERPs time-locked to the onset of the face feedback clearly showed a conspicuous negative deflection around 178 ms following stimulus onset (see Figure 2AB), with a maximum amplitude over lateral occipito-temporal electrodes on both sides, with a clear right hemispheric dominance (see Figure 2CD). These properties were compatible with the face-specific N170 component (Bentin et al., 1996). We first carried out a statistical analysis in which we compared the amplitude of the N170 generated in response to the exact same physical stimuli (i.e. neutral faces), but in two different contexts (negative context where neutral faces were used as positive feedback; and positive context where neutral faces were used as negative feedback).Results of this analysis showed that the N170 was significantly larger in the right (M = -7.78) compared to the left hemisphere (M = -6.15), $F(1, 56) = 8.20$, $p = .006$, but more importantly, that this face-specific component was larger in the positive context (M = -8.18), compared to the negative context (M = -5.74), $F(1, 56) = 5.54$, $p < .05$. This result indicated a larger N170 component for neutral faces when used as negative feedback (i.e. positive context) relative to the same neutral faces when used as positive feedback (i.e. negative context). This effect did not differ between low and high anxious participants, $F(1, 56) = .64$, $p > .10$, nor was there a main effect of group, $F(1, 56) = .03$, $p > .10$. (see Figure 2EF). This result was important as it suggested that when carefully controlling for low-level differences (and intrinsic pleasantness), the valence of the feedback was processed differentially as a function of the emotional context, as early as 170-180 ms post-stimulus onset, equally so for low and high anxious participants.

Next, we performed a more complex data analysis where we included emotional faces as well. This analysis showed that the amplitude of the N170 was concurrently influenced by the valence of the feedback and the context, $F(1, 56) = 33.72$, $p < .001$. While in the negative context, the N170 was slightly larger for negative feedback (i.e., angry face; M = -6.13) than positive feedback (i.e. neutral face; M = -5.74, $F(1, 56) = 3.07$, $p < .10$), in the positive context, the N170

was clearly larger for positive feedback (i.e. happy face; *M* = -9.46), compared to negative feedback (i.e. neutral face; *M* = - 8.18, *F*(1, 56) = 48.00, *p* < .001). This effect was not modulated by the level of trait anxiety, *F*(1, 56) = 1.45, *p* = .23 (Figure 2AB). These results suggest that probably not the valence of the feedback per se, but instead the perceived emotionality (e.g. arousal) of the faces increased the amplitude of the N170 (Batty & Taylor, 2003; Vuilleumier & Pourtois, 2007).

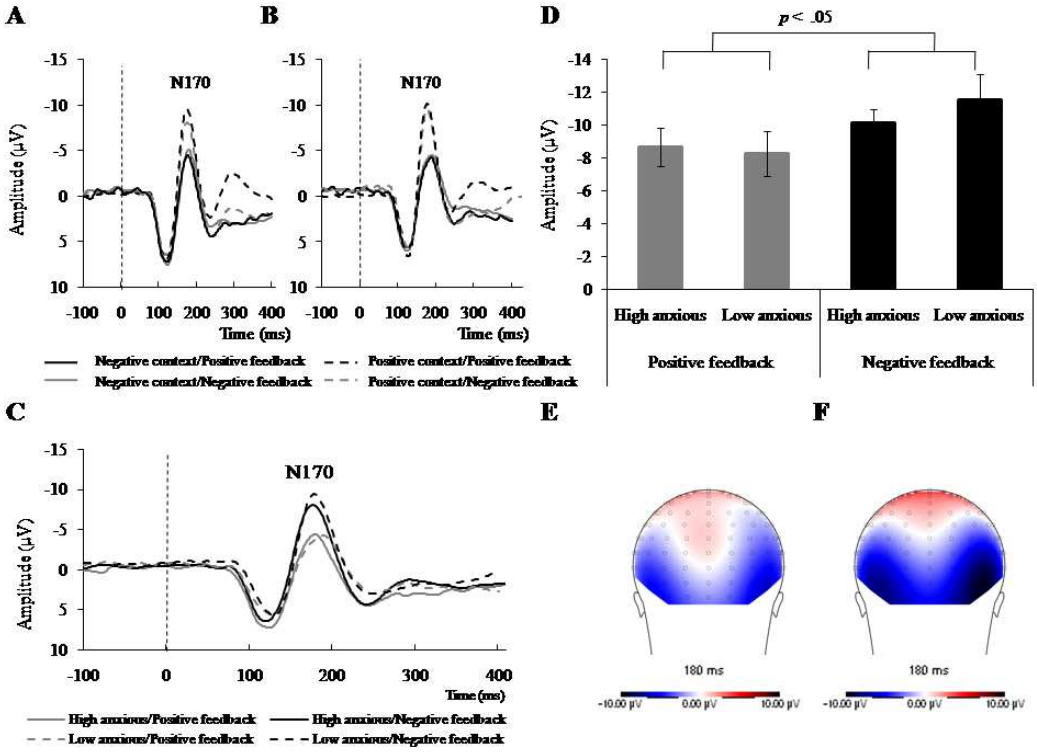


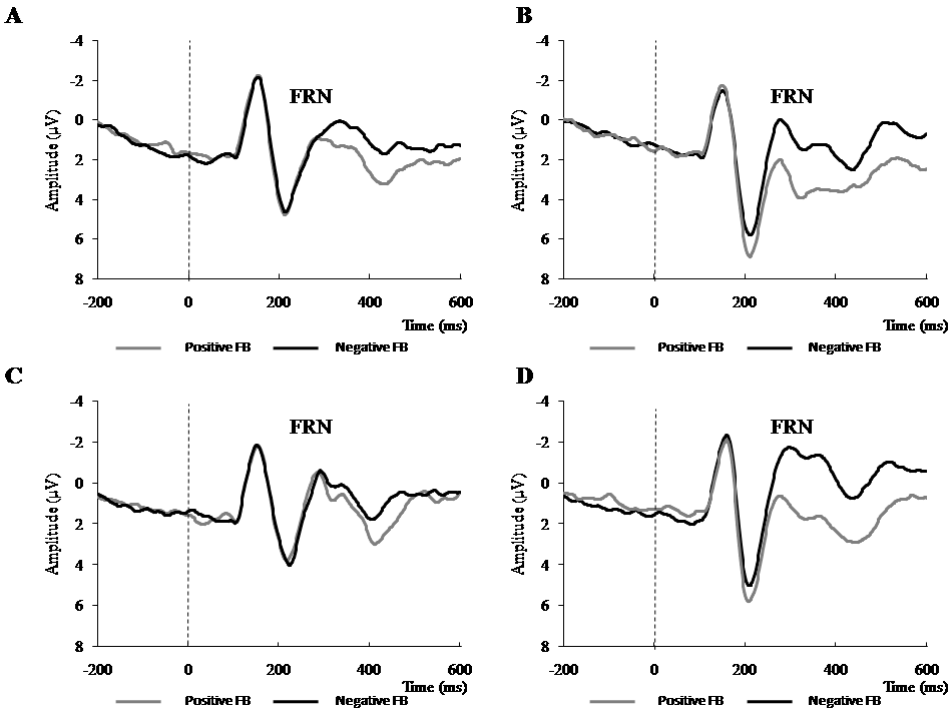
Figure 2. N170 results for emotional and neutral face feedback, separately. Grand average ERP waveforms (at occipito-temporal electrode B7, right hemisphere) for high (A) and low (B) anxious participants in the negative (neutral and angry faces) and the positive context (neutral and happy faces). (C) N170 occipital scalp map for neutral faces in the negative context (i.e. positive feedback). (D) N170 occipital scalp map for neutral faces in the positive context (i.e. negative feedback). (E) Grand average ERP waveforms (occipito-temporal electrode B7, right hemisphere) for low and high anxious participants for neutral faces serving as positive feedback (in the negative context) and negative feedback (in the positive context). (F) Mean amplitude (μV; electrode B7) ± 1 standard error of the mean of the N170 for neutral faces serving as positive feedback (in the negative context) and negative feedback (in the positive emotional context) in low and high anxious participants.

FRN component

Following the N170, another negative deflection was observed ~250 ms over fronto-central electrodes (e.g. FCz) consistent with the electrophysiological properties of the FRN (Holroyd & Coles, 2002). As expected, when computing the difference wave (negative feedback - positive feedback), the obtained negative activity reached its maximum amplitude at electrode FCz ~250 ms post-feedback onset. Results of the univariate ANOVA performed on the amplitude of the FRN in response to neutral faces, with context and group as between-subject factors revealed a significant effect of context, $F(1, 56) = 9.51$, $p = .003$, indicating that neutral faces in the positive context (which corresponded to negative feedback) elicited a larger FRN ($M = 8.18$, $SEM = 0.54$) than the exact same neutral faces in the negative context (which corresponded to positive feedback) ($M = 6.13$, $SEM = 0.41$). However, this differential effect of context (i.e. valence of feedback) was different for low vs. high anxious individuals, $F(1, 56) = 3.04$, $p = .09$. Planned comparisons revealed that neutral faces presented in the positive context led to a significantly larger FRN than the same neutral faces used in the negative context, but only for low anxious participants, $F(1, 28) = 12.06$, $p < .005$ (see Figure 3ABC). No such differential effect of context was observed for the amplitude of the FRN for high anxious individuals, $F(1, 28) = .87$, $p > .10$ (see Figure 3DEF). This result suggests that, unlike low anxious participants, high anxious participants failed to differentiate the acquired valence of the feedback on task performance conveyed by these neutral faces. This finding corroborated the assumption of a selective performance-monitoring deficit, as evidenced here for the FRN amplitude, in high anxious participants.

Next, FRN amplitudes were analyzed for neutral and emotional faces concurrently in an auxiliary analysis. This ANOVA revealed a significant three way interaction between valence, context and anxiety, $F(1, 56) = 4.75$, $p < .05$. While both low and high anxious individuals did not differentiate positive from negative feedback in the negative context, $F(1, 28) = 0.22$, $p = .64$ (Figure 3GH), a clear effect of feedback valence was observed in the positive context, $F(1, 28) = 32.49$, $p < .001$. This effect was larger in low anxious ($M = 1.83$, $SEM = 0.33$), $t(14) =$

5.47, $p < .001$, compared to high anxious individuals, ($M = 0.60$, $SEM = 0.26$), $t(14) = 2.27$, $p < .05$ (Figure 3IJ). Hence, this result confirmed that the amplitude of the FRN component varied with the valence of the feedback, depending on levels of trait anxiety[1].



[1] Similar results were obtained when the STAI-T scores (after log transformation because they were not normally distributed) were included in the analyses as a covariate, i.e., significant three way interaction (valence x anxiety x context): $F(1, 56) = 4.10$, $p < .05$; positive context: significant main effect of valence: $F(1, 28) = 5.20$, $p < .05$, interaction between anxiety and valence: $F(1, 28) = 3.59$, $p = .07$; negative context: no significant main effect of valence: $F(1, 28) = 1.16$, $p = .29$, no significant interaction between anxiety and valence: $F(1, 28) = 1.07$, $p = .30$.

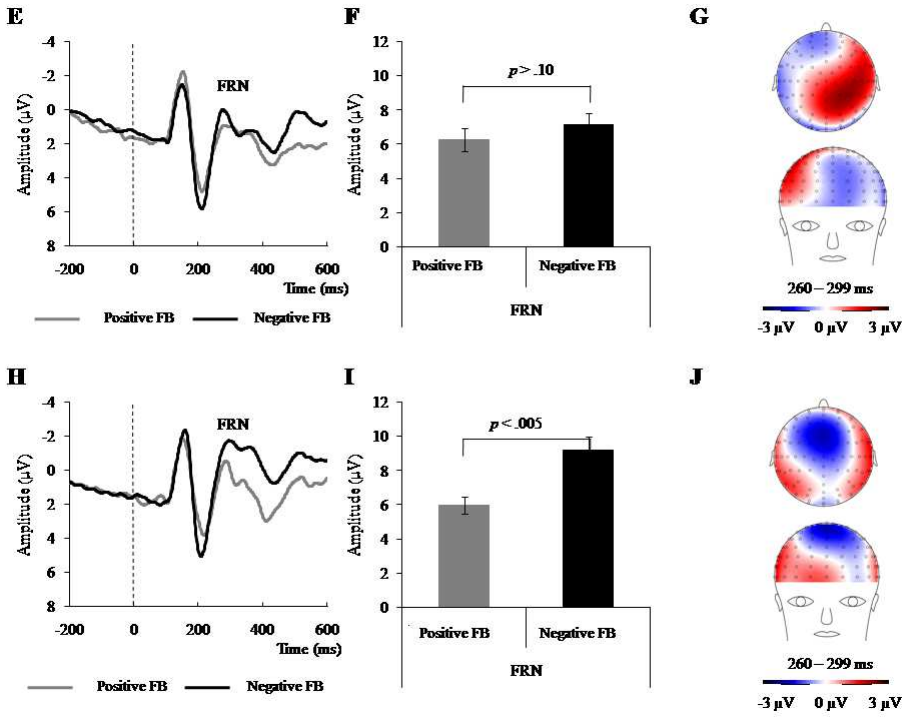


Figure 3. FRN results for emotional and neutral faces, separately. (A) Grand average ERP waveforms (electrode FCz) for low anxious participants for neutral faces serving as positive feedback (in the negative context) and negative feedback (in the positive emotional context). (B) Mean amplitude (μV) ± 1 standard error of the mean of the FRN (base-to-peak measure) for neutral faces serving as positive feedback and negative feedback in low anxious participants. (C) Horizontal and frontal scalp topography of the FRN (260-300 ms post-stimulus onset) for low anxious individuals, obtained after subtracting the negative feedback from positive feedback, showing a typical FRN voltage map distribution (i.e. circumscribed negative activity around FCz electrode position) in this group, relative to high anxious participants (compare with B). (D) Grand average ERP waveforms (electrode FCz) for high anxious participants for neutral faces serving as positive feedback and negative feedback. (E) Mean amplitude (μV) ± 1 standard error of the mean of the FRN for neutral faces serving as positive feedback and negative feedback in high anxious participants. (F) Horizontal and frontal scalp topography of the FRN (260-300 ms post-stimulus onset) for high anxious individuals, obtained after subtracting the positive context from the negative context condition. Grand average ERP waveforms (electrode FCz) for high (G) and low (H) anxious participants when neutral faces were used as positive feedback and angry faces as negative feedback (negative context). Grand average ERP waveforms (electrode FCz) for high (I) and low (L) anxious participants when neutral faces were used as negative feedback and happy faces as positive feedback (positive context).

Interestingly, additional correlation analyses confirmed that low vs. high anxious individuals reliably differed at the level of the FRN, and hence during the rapid monitoring of performance feedback. We found a significant negative

correlation between the LOC and the amplitude of the FRN to neutral faces in low anxious individuals irrespective of the emotional context (r = -.49, p < .01; see Figure 4A), while no such association was evidenced in high anxious individuals (r = -.03, p > .10; see Figure 4B). This significant correlation found in low anxious participants indicated that the larger the FRN component, the more the behavior was (usually) attributed to internal causes in these individuals[2].

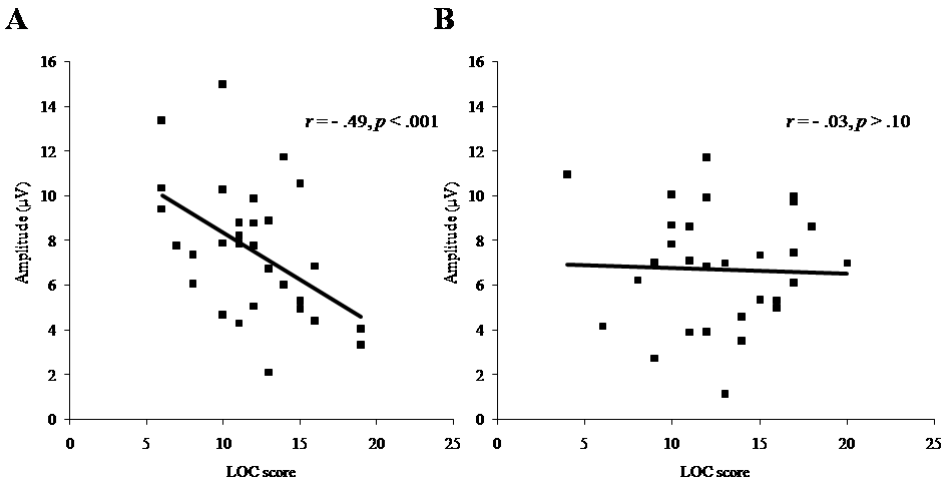


Figure 4. Correlation between FRN amplitude and subjective estimate of LOC for low (A) vs. high anxious (B) individuals.

Finally, we performed additional control analyses to ascertain that these FRN results were not confounded by an overlapping P300 or Late Positive Potential (LPP) effect, given that previous ERP studies showed a blunted LPP in high compared to low anxious individuals (Foti, Olvet, Klein, & Hajcak, 2010;

[2] We also computed and analyzed response-locked ERPs, with a focus on the ERN component that was previously shown to vary with trait anxiety (e.g., Aarts & Pourtois, 2010), especially in situations where action monitoring did not rely exclusively on the processing of external feedbacks on task performance, but internal action monitoring (i.e., no feedback) was required (Olvet & Hajcak, 2009a). Response-locked ERPs revealed a clear negative component peaking ~30 ms post response onset, with a maximum amplitude at fronto-central electrodes along the midline (including FCz), and which was substantially larger for response errors relative to correct hits, $F(1, 55) = 23.14$, $p < .001$. These electrophysiological properties were compatible with the ERN/Ne (Falkenstein et al., 2000; Gehring et al., 1993). However, the ERN amplitude did not vary between low vs. high anxious participants, $F < 1$, nor between the negative vs. positive context, $F < 1$, consistent with previous findings (Olvet & Hajcak, 2009a).

Weinberg & Hajcak, 2010). At posterior parietal leads along the midline (electrode Pz), we isolated a positive component time-locked to the onset of the feedback, sharing similarities with the LPP. This component peaked 350 ms post-feedback onset and lasted ~650 ms, hence showing a sustained activity. Results showed that the mean amplitude of this LPP component (as computed during this time interval at electrode Pz) was larger for positive compared to negative feedback, $F(1, 56) = 19.29$, $p < .001$, but this valence effect was not modulated by anxiety, $F(1, 56) = .00$, $p > .10$, or context, $F(1, 56) = 1.33$, $p = .25$. The interaction between context and anxiety did not reach significance either, $F(1, 56) = .12$, $p > .10$. This analysis also disclosed that the LPP was smaller in high compared to low anxious individuals, $F(1, 56) = 4.71$, $p < .05$, in agreement with these previous studies (Foti et al., 2010; Weinberg & Hajcak, 2010). These control analyses confirmed that the reported FRN effect (and its modulation by levels of trait anxiety and emotional context) did not overlap (in time and electrode locations) with a later LPP effect taking place during feedback processing.

DISCUSSION

The goal of this study was to test the assumption that high anxious participants may exhibit action-monitoring deficits, as reflected by an invariance of the FRN to opposite performance feedback. Given that low and high anxious individuals might already differ in the way they actually perceive the intrinsic pleasantness of the feedback (regardless of any influence of higher-order performance-monitoring brain mechanisms), we also looked at an earlier perceptual ERP component, namely the face-specific N170 (Bentin et al., 1996), and verify whether this earlier brain response could vary with the valence of the feedback (as implemented with a contextual modulation, see also Righart & de Gelder, 2006). A number of new results emerge from this ERP study.

First, we found a comparable behavioral performance (i.e. accuracy and speed) between low and high anxious individuals during the speeded Go/noGo task, and between the positive and negative emotional context. This result confirmed that trait anxiety did not simply alter behavioral performance during

our speeded Go/noGo task (Aarts & Pourtois, 2010; Hajcak et al., 2003a) and that the ERP difference found at the level of the FRN between high vs. low anxious participants could not be related to obvious changes in the behavior across these two groups. Moreover, we did find evidence for an increase in levels of state anxiety induced by the Go/noGo task (pre-post comparison; see also Aarts & Pourtois, 2010), but this change was actually the same in both groups and contexts. Importantly, emotional ratings of the faces also confirmed that neutral faces acquired a different valence depending on the emotional context they were embedded in (i.e. they were perceived as relatively more negative when used as negative, compared to positive feedback), but this contextual modulation effect was similar in both groups, confirming preserved perceptual functions in high anxious participants.

Secondly, our new ERP results show that, when controlling for the intrinsic pleasantness of the feedback stimuli, the face specific N170 component (Bentin et al., 1996) was reliably increased for neutral faces used as negative feedback, relative to the same neutral faces used a positive feedback (see also Vuilleumier & Pourtois, 2007). Importantly, this differential structural encoding of the face as a function of the acquired valence of the evaluative feedback was similar for low vs. high anxious participants. Moreover, following the N170, a larger FRN component was found for neutral faces serving as negative feedback compared to the same neutral faces serving as positive feedback, but only in low anxious participants. These new electrophysiological findings therefore confirm that performance monitoring was modulated by levels of trait anxiety, as only low, but not high anxious individuals, showed a systematic variation of the FRN amplitude as a function of the valence of the feedback. However, our ERP results also showed that this effect of anxiety on feedback processing was component specific and concerned mainly the FRN component. The dissociation found between the N170 and FRN component during feedback processing in high anxious individuals suggests that trait anxiety does not simply alter evaluative feedback processing in general. Instead, it specifically influences a stage of performance monitoring (reflected by the FRN component) during which the

perceived valence of the feedback is presumably compared to the internalized value of the action (Holroyd & Coles, 2002). However, our additional results obtained for the N170 component also show that the positive vs. negative valence of the feedback is correctly perceived as such by these high anxious participants, ruling out the possibility of a low-level perceptual deficit accounting for our FRN findings. Interestingly, we also found that across low anxious participants, the amplitude of the FRN was related to the attribution style (as measured using a standard questionnaire, see Rotter, 1966), whereas no such relationship could be evidenced in high anxious participants. The amplitude of the FRN was larger for low anxious individuals who were more inclined to attribute the cause or origin of their actions or behavior to internal (as opposed to external) drives or forces. Altogether, these new ERP results inform about the stage of processing following evaluative feedback onset during which trait anxiety may reliably influence performance monitoring. We discuss the implication of these new results in more detail here below.

Spared encoding of the emotional value of the feedback in anxiety

Our ERP results for the N170 component showed that high anxious individuals could actually reliably and correctly decode the intrinsic emotional value of the feedback information, despite an apparent deficit in linking this emotional value to a correct error prediction signal (as shown by the FRN). Hence, effects of trait anxiety on performance monitoring appear to be rather selective, since they mainly concern a specific stage of processing (the mid-latency FRN component), while leaving unaffected earlier perceptual stages (N170 component) during evaluative feedback processing. Previous ERP studies already showed that context influences the early structural encoding of faces, as shown by enhanced N170 components for faces embedded in negative context/background information (Righart & de Gelder, 2006, 2008). Here, we found an enhanced N170 component for neutral faces associated with a negative outcome, relative to the exact same faces used as positive feedback. However, because we found that the N170 amplitude was in both contexts increased for emotional compared to neutral faces, it appears that the emotional significance

or level of arousal (instead of the valence per se) of the face may be the critical dimension influencing this early visual component (see also Batty & Taylor, 2003). Importantly, when neutral faces were used as negative feedback and directly compared to the exact same neutral faces used as positive feedback, a larger N170 was observed for negative compared to positive feedback. This might indicate an augmented emotional significance of neutral faces in the positive emotional context. Crucially, our results for the N170 showed that this effect of emotional significance was similar in low and high anxious individuals, suggesting preserved perceptual emotional processes (i.e. structural encoding of the face) in high anxious participants during evaluative feedback processing. Behavioral results obtained for the ratings of the faces also corroborated this conclusion.

Selective alteration of performance monitoring in anxiety

By contrast, a modulatory effect of trait anxiety during evaluative feedback processing was evidenced when looking at the fronto-central FRN component. While this performance-monitoring component reliably discriminated between negative and positive feedback in low anxious participants, it did not in high anxious participants. Strikingly, the amplitude of the FRN for positive and negative feedback in high anxious individuals was similar (i.e. no larger FRN for negative compared to positive feedback), and comparable in both cases to the FRN following positive feedback in low anxious individuals. This suggests impaired performance-monitoring functions in anxiety. Although the morphology of the FRN component found in this study was slightly different compared to previous studies (Hajcak, Holroyd, Moser, & Simons, 2004; Holroyd, Larsen, & Cohen, 2004), this difference may be due to the use of complex facial stimuli as performance feedback, relative to simple symbolic cues in these earlier studies. Likewise, here outcome evaluation at the level of the FRN was actually based on speed (fast vs. slow hits), but not accuracy, a factor that might potentially account for changes in the morphology of this performance-monitoring ERP component across studies. At any rate, future studies are needed to corroborate this statement. Importantly, control analyses showed that the

reported FRN results did not overlap with a later LPP effect (Foti, Hajcak, & Dien, 2009; Schupp et al., 2004), the latter being indicated by a blunted LPP component for high compared to low anxious participants, consistent with previous ERP studies (Foti et al., 2010; Weinberg & Hajcak, 2010). Our ERP results further show that the effect of feedback valence was only observed in the positive context, where happy faces and "neutral" faces were presented, and that this difference was larger for low, compared to high anxious participants. In the negative context, the amplitude of the FRN did not differentiate between angry and "neutral" faces. These FRN results are in line with previous studies that did already report a comparable asymmetry, with a larger differentiation at the level of the FRN between neutral and positive feedback than between negative and neutral feedback (Hajcak, Holroyd, Moser, & Simons, 2004; Holroyd & Coles, 2002).

The main ERP result showing a modulatory effect of trait anxiety on the FRN component is in accordance with previous studies (Gu et al., 2010; Simons, 2010) and more generally, the reinforcement learning theory (Holroyd & Coles, 2002). This model proposes that that the FRN component reflects the perceived discrepancy between the expected and the actual outcome (i.e. prediction error), here based on the processing of an external evaluative feedback (as opposed to an internal motor representation for the ERN/Ne component). A larger FRN in low compared to high anxious individuals suggests that trait anxiety likely influences the encoding of the prediction error signal during the processing of simple action-outcome sequences. Presumably, high anxious individuals might show a tendency to expect more negative external feedback/evaluations compared to low anxious individuals, and as a result these former participants would show blunted reactions to negative feedback, because the discrepancy between the actual and expected outcome is, by definition, smaller. Consistent with this notion, Maner and Schmidt (2006) showed a link between anxiety and pessimistic outcome expectancy. By contrast, here we did not find any modulation of the ERN/Ne component (and hence internal monitoring processes) as a function of trait anxiety, unlike previous ERP studies (Aarts &

Pourtois, 2010; Hajcak et al., 2003a). This discrepancy could be explained by the use of salient evaluative feedback in this study (i.e. emotional faces), which may have introduced a strong bias towards the monitoring of these external evaluative feedback at the cost of more internally-oriented monitoring processes. Interestingly, in this condition, effects of trait anxiety on internal monitoring brain processes (i.e. ERN/Ne component) seem to disappear, in line with previous ERP results (Olvet & Hajcak, 2009a).

The assumption that trait anxiety may selectively influence a performance-monitoring process through which the perceived valence of the feedback is readily integrated with the internalized value of the action is indirectly supported by our additional correlation analysis between LOC and the amplitude of the FRN. Our results show that low anxious individuals characterized by an internal LOC had a larger FRN, relative to low anxious individuals with a more external LOC. This result indirectly confirms that the FRN is not only sensitive to the valence of the feedback per se, but also to higher-level motivational or emotional factors, including the motivational significance of our actions (Gehring and Willoughby, 2002; Yeung et al., 2005). Noteworthy was the absence of this relationship in high trait anxious participants, confirming that this psychopathological condition (here at the subclinical level) may reliably alter performance-monitoring brain systems. Hence, this anxiety-related deficit during performance monitoring may concern a specific generative process enabling to readily bind the (internalized) value of the action with the perceived valence of the feedback. However, we have to acknowledge that because our trait anxiety estimate (based on a standard questionnaire in the literature) likely measures negative affect (or even depression) (e.g. Nitschke, Heller, Imig, McDonald & Miller, 2001; Rossi & Pourtois, 2011), enhanced levels of negative affect or internalized personality traits in general, rather than trait anxiety per se (see also Olvet & Hajcak, 2008), may account for the amplitude variations observed at the level of the FRN component in our study.

Conclusion

Results of this ERP study reveal a specific performance-monitoring deficit associated with subclinical trait anxiety, although low and high anxious participants showed comparable behavioral performance during this speeded Go/noGo task. Our FRN results suggest that high anxious individuals have a selective impairment in integrating the emotional value or motivational significance of the feedback with the internalized value of the action executed 1000 ms prior to feedback delivery. This effect might be imputed to a selective change produced by trait anxiety in the normal reinforcement learning signal generated during action monitoring. However, our ERP results also show that the rapid decoding of the emotional significance of the facial feedback information (as reflected by the N170 component) is not altered in high compared to low anxious individuals, suggesting a component specific effect of anxiety during evaluative feedback processing. As such, our new ERP findings help better characterize the precise temporal locus during which trait anxiety reliably changes and influences performance-monitoring brain functions.

ACKNOWLEDGMENTS

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CHAPTER 5: EVIDENCE FOR THE AUTOMATIC EVALUATION OF SELF-GENERATED ACTIONS[1]

"The accuracy of simple actions is swiftly determined through specific monitoring brain systems. However, it remains unclear whether this evaluation is accompanied by a rapid and compatible emotional appraisal of the action that allows to mark incorrect actions as negative/bad and conversely correct actions as positive/good. In this study, we used a new method to decode the affective value of simple actions generated by participants during a standard Go/noGo task. Immediately after each Go/noGo action, participants responded to the valence of either a positive or a negative word. Results showed that False Alarms performed during the Go/noGo task led to a faster evaluative categorization of negative words relative to positive words. This action - word evaluative priming effect occurred when the interval between these two events was set to either 300 or 600 ms, but not 1000 ms. Finally, higher levels of trait anxiety were associated with a reduction of the evaluative priming effect. Our results suggest that simple actions are rapidly evaluated as positive or negative depending on the automatic monitoring of their perceived accuracy."

[1] Aarts, K., De Houwer, J., & Pourtois, G. (under revision). Evidence for the automatic evaluation of self-generated actions. Cognition.

INTRODUCTION

Human beings constantly and effortlessly categorize external stimuli in their environment as good or bad. This function is adaptive because it enables us to unlock rapidly appropriate behavioral responses, for example to approach a positive stimulus or avoid a negative stimulus (Cacioppo, Priester, & Berntson, 1993; Chen & Bargh, 1999; Neumann, Förster, & Strack, 2003). Evidence for automatic evaluative processing has been obtained in evaluative priming studies (De Houwer, Hermans, & Eelen, 1998; Fazio, Sanbonmatsu, Powell, & Kardes, 1986; Hermans, De Houwer, & Eelen, 1994). Evaluative priming refers to the fact that reaction times (RTs) for categorizing the valence of a target word (e.g. cold), are shorter when it is preceded by a prime with the same valence (e.g. cancer), than when it is preceded by a prime with a different valence (e.g. happy). Given that there is very little time between the onset of the prime and the onset of the target (typically less than 300 ms) and participants are asked to ignore the primes, these results suggest that the valence of the prime is processed automatically in the sense of rapidly and unintentionally. Evaluative priming has already been observed for a wide range of external stimuli in the environment, including words (Fazio et al., 1986), pictures (Hermans et al., 1994), black and white line drawings (Giner-Sorolla, Garcia, & Bargh, 1999), motivationally-relevant stimuli (i.e. rewarded and unrewarded colors; see Moors & De Houwer, 2001), odors (Hermans, Baeyens, & Eelen, 1998) and tones (Reber, Haerter, & Sollberger, 1999).

Presumably, automatic evaluation is a generic function and does not only concern external stimuli in the environment, but also self-generated actions. Actions in response to stimuli are usually deemed conducive or obstructive depending on their actual match with goals stored in long term memory (Scherer, 1984, 1988). Indirect evidence supporting this view comes from recent psychophysiology studies showing that unwanted response errors (i.e. goal obstructive events) yield larger skin conductance responses and greater heart rate deceleration than correct decisions (Hajcak et al., 2003b), as well as a larger

startle potentiation (Hajcak & Foti, 2008) and differential early activation in the amygdala (Pourtois et al., 2010). These results suggest that errors may be perceived as aversive events, and accordingly be associated with enhanced arousal within the autonomic nervous system. It has also been shown that through conditioning, a specific action (e.g. a key press), can become aversive as evidenced by the fact that the selection of the action is faster by the presence of an irrelevant negative word (Beckers, De Houwer, & Eelen, 2002). Although these studies give first hints on the acquired emotional value of specific actions, they do not inform us about whether valence specific effects can be obtained as a function of the perceived goal conduciveness of simple self-generated actions. More specifically, it is likely that the post-error detection changes in autonomic or brain activity that were observed in previous studies merely reflect enhanced arousal (Hajcak & Foti, 2008) or attention orienting (Notebaert et al., 2009) rather than a genuine affective marking of these actions as negative events. Moreover, whereas the focus is mainly on response errors in these earlier studies, much less is known about a possible symmetric affective tagging of correct actions as positive events by generic internal action-monitoring brain systems. In other words, it still needs to be determined whether incorrect actions are automatically categorized as negative events relative to correct actions, while conversely correct actions would implicitly be associated with positive emotions, relative to response errors.

We addressed this question using a novel experimental paradigm suited to decode online the emotional value of simple self-generated actions performed during a standard Go/noGo task by healthy adult participants. Participants performed a speeded Go/noGo task (Vocat, Pourtois, Vuilleumier, 2008), which was combined with an evaluative word categorization task. Unbeknown to participants, actions performed during the Go/noGo task (either correct or incorrect responses) served as primes whereas the words (positive or negative) were used as targets. In line with the logic underlying evaluative priming effects, we predicted that the time needed to categorize a target word would be systematically influenced by the putative valence of the preceding action, the

latter being presumably decoded rapidly following or even during action execution in specific cognitive and emotion control systems (De Bruijn, de Lange, von Cramon, & Ullsperger, 2009). More precisely, we expected participants to be faster to categorize a target word as negative if the preceding action was incorrect and to categorize a target word as positive if the preceding action was correct.

We also examined some of the functional properties of automatic evaluation of correct and incorrect actions. More specifically, we tested whether the effect was moderated by the time between the action and the target word and by the affective disposition of the participants. Previous studies with word primes and word targets reported reliable evaluative priming effects with a short stimulus onset asynchronies (SOAs) between prime and target (i.e. 300 ms or less) but not with long SOAs (e.g. 1000 ms; see Fazio et al., 1986; De Houwer et al., 1998; Hermans et al., 1994). Based on these results, it was concluded that the automatic evaluation of words is a fast acting automatic process. In our first two experiments, the length of the SOA between the self-generated action and the presentation of the target word was constant and set to 300 ms. It was increased to 600 ms in Experiment 3 and to 1000 ms in Experiment 4. If the evaluation of correct and incorrect actions is also a fast acting automatic process (see Pourtois et al., 2010, for converging neuroscientific evidence) then evaluative priming should be observed at short (300 ms) but not long (1000 ms) SOAs. With regard to the moderating impact of the affective disposition of participants, we focused on trait anxiety. Given that anxiety and the prolonged experience of negative affect usually lead to altered action-monitoring effects (see Aarts & Pourtois, 2010; Hajcak, McDonald, & Simons, 2003a), we tested whether (subclinical) high trait anxious individuals might show a different evaluative priming effect compared to low anxious participants, consistent with an impaired ability of the former participants to readily assign an affective value to a self-generated action (see Aarts & Pourtois, 2012).

METHOD

Participants

Twenty-one first-year female undergraduate psychology students (Age: *M* = 18.52; *SEM* = 0.40; Range = 17 - 25) participated in Experiment 1. Fifteen undergraduate students (14 women; Age: *M* = 21.4, *SEM* = .38, Range = 18 - 23) took part in Experiment 2. Twenty-two undergraduate students participated in Experiment 3 (19 women; Age: *M* = 21.73; *SEM* = .50, *Range* = 19 - 28). Finally, 20 undergraduate students took part in Experiment 4 (18 women; Age: *M* = 23.05, *SEM* = .86, Range = 18 – 26). All participants were right-handed, native Dutch speakers who did not have a history of neurological or psychiatric disease and had normal or corrected-to-normal vision. The study was approved by the local ethics committee. All students participated in exchange for course credits or for money (10 Euro).

Stimuli

In the Go/noGo task, visual stimuli consisted of an arrow (subtending 11.4◦ ×0.05◦ of visual angle at a 60 cm viewing distance), that was presented in the center of a white homogenous background, and oriented either upward or downward (see Figure 1). The arrow was first black, and could then turn either green or turquoise. These two colors were matched for luminance. These different combinations of color and orientation were used as cues in the Go/noGo task.

In the evaluative categorization task, targets were 30 positive and 30 negative words, either nouns or adjectives (see Table 1), and were selected from the Dutch affective rating list of Hermans and De Houwer (1994). *T*-tests showed that these positive and negative words differed significantly on the affective dimension, $t(58) = 36.57$, $p < .001$, $\eta_p^2 = .95$, but not on the familiarity dimension, $t < 1$, nor with respect to the number of letters, $t < 1$.

Table 1.

Target words selected from the Dutch affective rating list of Hermans and De Houwer (1994)

Positive targets		*Negative targets*	
Hawaii (Hawaii)	trouw (fidelity)	ruw (rude)	stank (stench)
engel (angel)	lente (spring)	haat (hate)	drugs (drugs)
goud (gold)	baby (baby)	moord (murder)	virus (virus)
regenboog (rainbow)	parfum (parfume)	aids (aids)	puist (pustule)
bruid (bride)	knuffel (hug)	vals (false)	zweer (sore)
applaus (applause)	feest (part)	pijn (pain)	oorlog (war)
hemel (heaven)	oprecht (sincere)	dief (thief)	kanker (cancer)
geboorte (birth)	zomer (summer)	dood (dead)	hitler (hitler)
vrede (peace)	humor (humor)	graf (tomb)	geweren (guns)
spel (game)	bloemen (flowers)	sluw (sly)	ongeval (accident)
geschenk (gift)	omhelzing (embrace)	hoer (hore)	brutaal (impudent)
cadeau (present)	vakantie (holiday)	koud (cold)	vulgair (vulgar)
trots (proud)	droom (dream)	zwak (weak)	ongezond (unhealthy)
melodie (melody)	leven (life)	spin (spider)	hatelijk (hasty)
romantiek (romanticism)	liefde (love)	vuil (dirty)	vijandig (hostile)

Procedure

Participants performed a standard speeded Go/noGo task (Vocat et al., 2008) interleaved with a visual word categorization task (see Figure 1). Actions performed during the speeded Go/noGo task actually served as primes whereas words were deemed targets in analogy with a conventional prime-target sequence during evaluative priming. Each trial started with a fixation cross that lasted for 500 ms. Afterwards, a black arrow, either oriented up or down, was presented at the position previously occupied by the fixation cross. After a variable interval ranging from 1000 ms to 2000 ms, the black arrow became either green or turquoise while its orientation could either remain identical or shift in the opposite direction compared to the initial black arrow. When the black arrow turned green and the orientation remained unchanged, participants were instructed to press a pre-defined button of the response box as fast as possible with the index finger of their left hand (Go trials). However, participants

had to withhold responding when either the arrow became green but changed orientation, or when the arrow became turquoise and kept its initial orientation, enabling two noGo trial types. Instructions emphasized both speed and accuracy, such that not only accuracy, but also the perceived speed was later evaluated as being correct or incorrect. For each trial, speed was evaluated using an individually calibrated RT limit computed during a training block that preceded each session of two test blocks. This limit was thus calculated and updated three times in total (before Blocks 1 and 2 – Session 1, before Blocks 3 and 4 – Session 2, and before Blocks 5 and 6 – Session 3). This allowed us to deal with unspecific learning effects over time and maintain a similar number of correct and incorrect responses throughout the experiment. For the first session, the upper limit was set to 70% of the mean RT from the first training block. For the two subsequent sessions, this upper limit was updated and set to 80% of the mean RT during the respective training block. Hence, this procedure required participants to respond at least 30% faster (first session) or 20% faster (second and third sessions) on Go trials than their average speed during the respective training block. This procedure ensured a sufficient number of response errors on noGo trials and allowed us to distinguish between Fast Hits (i.e. responses on Go trials that were emitted more quickly than the individually-titrated RT limit) or Slow Hits (i.e. responses on Go trials that took longer than the RT limit). Errors were formally defined as overt responses on noGo trials (i.e. FAs), while correct inhibitions corresponded to withheld responses on the same noGo trials.

Three hundred milliseconds after an action was executed, a target word was presented. The same 300 ms SOA was used in Experiment 2 because this experiment was mainly run to provide a replication of the results obtained in Experiment 1. The SOA was set to 600 ms in Experiment 3 and to 1000 ms in Experiment 4 in order to assess whether an evaluative priming effect was sensitive to the time elapsed between prime (action) and target (word). For correct inhibitions, the target word was presented 1500 ms after the presentation of the colored arrow plus the length of the SOA. Participants were instructed to categorize the valence of the target word (positive or negative) as

fast and as accurately as possible by pressing one of two predefined keys of the response box using their dominant hand. Hence, the evaluative word categorization task was executed with a different effector than the Go/noGo task. The target word remained on the screen until the participant responded or 3000 ms elapsed. In order to balance the presentation of positive vs. negative words following Fast Hits, Slow Hits, Correct Inhibitions, and FAs, the target word that was presented following an action was selected randomly on each trial. After the word categorization, participants received feedback informing them about their accuracy for the two consecutive tasks. The feedback for the Go/noGo task indicated whether the performed action was correct (and fast enough), incorrect or too slow, while the feedback for the word categorization could be either correct or incorrect. Both feedback signals remained on the screen for 2000 ms.

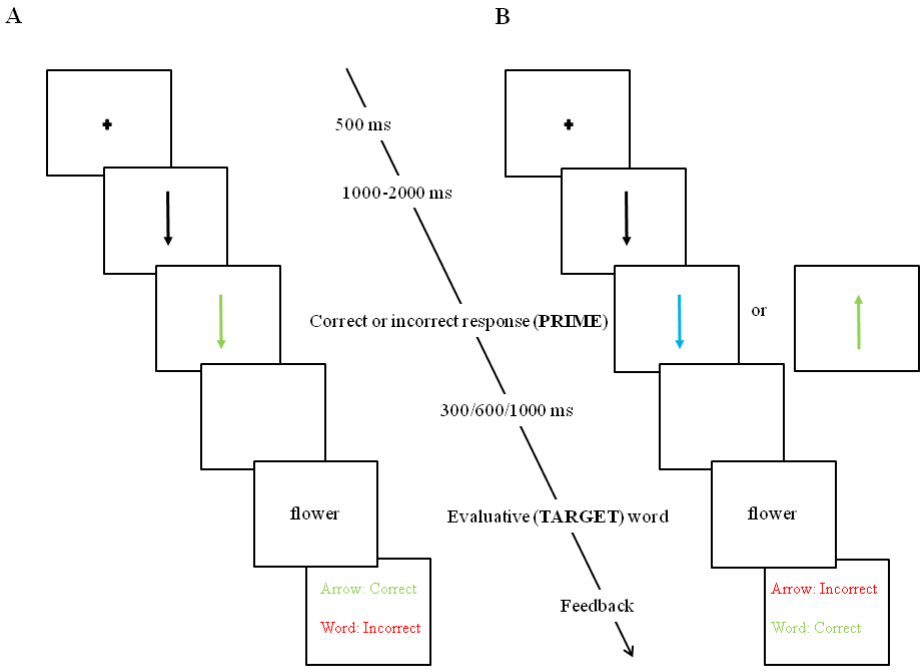

Figure 1. Stimuli and task. Participants had to respond by pressing a button of the response box as fast as possible with their non-dominant hand only when the arrow became green and kept its initial orientation (A), but not otherwise (B).

After a practice phase including 24 trials, the experiment was divided into 3 sessions, each starting with a training block (containing 28 trials: 20 Go and 8

noGo trials), followed by two test blocks (each containing 72 trials: 48 Go and 24 noGo trials). Note that participants were unaware that training blocks were actually used as calibration blocks to compute the RT limit used during the two following test blocks. Trial presentation was randomized within blocks. Between blocks, a small break (no longer than 5 min) was introduced. The whole experiment included 540 trials and lasted on average 50 min. Stimulus presentation and response recording were controlled using E-prime software (V2.0., http://www.pstnet.com/products/e-prime/).

Accuracy and RTs (correct responses) for the evaluative word categorization task were analyzed using repeated measures analyses of variance (ANOVAs) as a function of (i) the valence of the target word (either positive or negative) and (ii) the type of action (FA, Fast Hit or Slow Hit) preceding word presentation. We did not include in these analyses RTs and errors for target categorization when a response on noGo trials was correctly inhibited because no overt (Go) action was performed in this condition. Additional statistical analyses confirmed that the evaluative categorization was not influenced by these preceding correct inhibitions as the speed to categorize negative words did not differ significantly from the speed to categorize positive words (all *T*s < 1 in Experiments 1-4).

After completion of the three experimental sessions, the Dutch version of the trait version of the Spielberger State-Trait Anxiety Inventory (STAI) (Defares, van der Ploeg, & Spielberger, 1979; Spielberger, 1983) was filled out by the participants.

RESULTS

In all four experiments, trials with RTs shorter than 150 ms or longer than 500 ms in the Go/noGo task were discarded, as were trials in which the RT on the evaluative categorization task exceeded 2.5 *SD* from the mean RT per condition (see Table 2). Two participants (female) were not included in the statistical analyses of the data of Experiment 4 because they did not commit sufficient (i.e. minimum 10) FAs to compute reliable accuracy or RT estimates for each

condition separately (i.e. positive words following FAs vs. negative words following FAs). Hence, in Experiment 4, only the data of the remaining 18 participants were included in the analysis.

Table 2.

Percentages outlier trials in the Go/noGo task (< 150 ms or > 500 ms) and in the evaluative categorization task (< or > than RTs ± 2.5 SD)

		Exp 1		Exp 2		Exp 3		Exp 4	
Criterium	Condition	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
< 150 ms	FAs	4.25	1.27	3.02	0.87	5.78	1.33	6.63	2.21
	Fast	6.69	2.02	4.47	1.76	9.34	2.40	10.44	4.33
> 500 ms	FAs	3.04	1.71	0.90	0.32	2.37	1.36	2.81	0.97
	Slow	6.45	1.11	3.73	1.23	7.90	1.92	7.16	1.08
> or <than	Negative	2.89	0.21	2.91	0.22	5.60	1.13	3.30	0.21
RTs ± 2.5 SD	Positive	2.82	0.15	3.10	0.28	3.30	0.76	3.38	0.19

Evaluative Categorization Task

Speed

In Experiment 1, the ANOVA performed on the mean RTs for correct responses revealed a significant interaction effect between action type and word type, $F(2, 40) = 13.51$, $p < .001$, $\eta_p^2 = .40$. This interaction resulted from faster evaluative categorizations when the valence of the word was congruent with the putative affective value of the action. More specifically, RTs for negative words following FAs were shorter compared to RTs for positive words following FAs, $t(20) = -2.57$, $p < .05$, $\eta_p^2 = .25$, while symmetrically, participants tended to categorize positive words faster compared to negative words when they followed Fast Hits, $t(20) = 1.81$, $p = .08$, $\eta_p^2 = .14$. Following Slow Hits, no significant RT difference emerged between negative and positive words, $t < 1$. The main effect of word type was not significant, $F < 1$. By contrast, the ANOVA revealed a significant main effect of action type, $F(2, 40) = 26.04$, $p < .001$, $\eta_p^2 =$

.57, reflecting longer RTs for words following FAs[1] compared to words following either Fast Hits, $F(1, 20) = 30.00$, $p < .001$, $\eta_p^2 = .60$, or Slow Hits, $F(1, 20) = 30.25$, $p < .001$, $\eta_p^2 = .60$, an effect in line with a systematic post-error slowing (Danielmeier & Ullsperger, 2011; Rabbitt, 1966). Shorter RTs were also observed for words following Fast Hits compared to Slow Hits, $F(1, 20) = 6.10$, $p < .05$, $\eta_p^2 = .23$ (see Figure 2A).

An almost identical interaction effect between action type and word type was found in Experiment 2, $F(1, 28) = 13.60$, $p < .001$, $\eta_p^2 = .49$, and Experiment 3, $F(2, 42) = 14.62$, $p < .001$, $\eta_p^2 = .41$, but not in Experiment 4, $F < 1$[2]. Also no effect of word type was observed for Experiments 2-4 (Experiment 2: $F(1, 28) = 2.02$, $p > .10$, $\eta_p^2 = .13$; Experiment 3: $F(1, 42) = 1.56$, $p > .10$, $\eta_p^2 = .07$; Experiment 4: $F < 1$, $\eta_p^2 = .02$) while the post-error slowing effect was observed in all experiments (Experiment 2: $F(2, 40) = 26.80$, $p < .001$, $\eta_p^2 = .66$; Experiment 3: $F(2, 42) = 5.13$, $p < .05$, $\eta_p^2 = .20$; Experiment 4: $F(2, 34) = 15.49$, $p < .001$, $\eta_p^2 = .48$; see Figure 2BCD).

[1]Because this general RT slowing following FAs compared to Fast Hits might lead to an artificial increase in evaluative priming for FAs compared to Fast Hits, we also analyzed log transformed RTs. This analysis confirmed a significant interaction effect between action type and word type, $F(2, 40) = 16.41$, $p < .001$, $\eta_p^2 = .45$. RTs for negative words following FAs were shorter compared to RTs for positive words following FAs, $t(20) = -2.23$, $p = .04$, $\eta_p^2 = .20$, while participants categorized positive words faster compared to negative words when they followed Fast Hits, $t(20) = 3.71$, $p < .001$, $\eta_p^2 = .41$.

[2]We performed an omnibus ANOVA on the RT data collected across the four experiments to verify that the categorization of the target word was reliably influenced by the putative affective value of the preceding action, only when a short (Experiments 1 and 2) but not long SOA (Experiment 4) was used between these two events, consistent with our prediction. This analysis confirmed a significant three-way interaction between action type, word type and SOA, $F(4, 146) = 3.96$, $p < .01$, $\eta_p^2 = .10$.

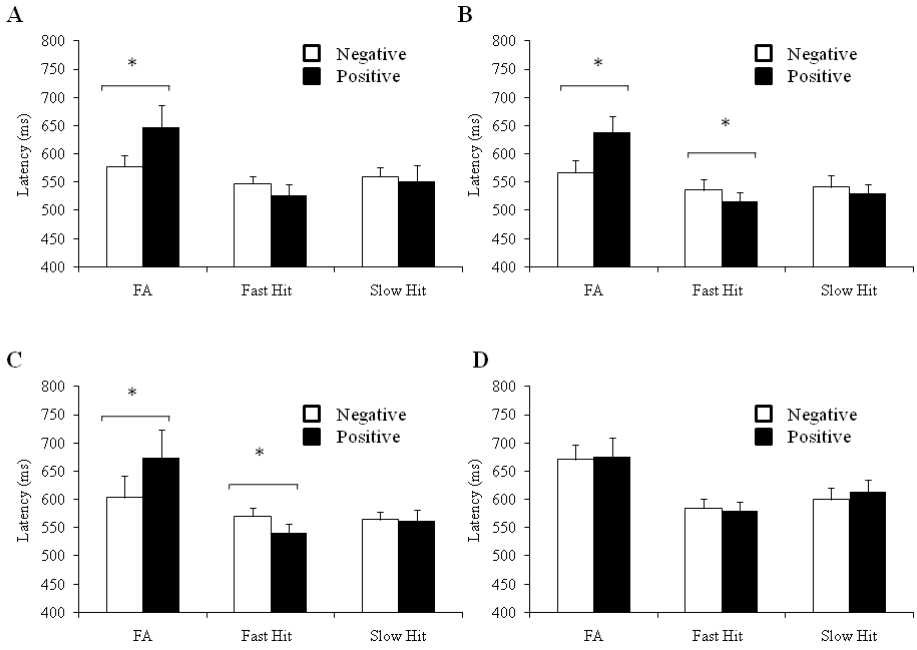

Figure 2. Mean RTs (+ 1standard error of the mean (SEM)for bars) for correct evaluative categorizations as a function of prime type (FA, Fast Hit or Slow Hit) and word type (Negative or Positive Words) in (A) Experiment 1 (SOA = 300ms), (B) Experiment 2 (SOA = 300 ms), (C) Experiment 3 (SOA = 600 ms) and (D) Experiment 4 (SOA = 1000ms). * p< .05.

Accuracy

In Experiment 1, the ANOVA performed on accuracy data (i.e. % correct responses) revealed a significant interaction between action type (FA, Fast Hit, Slow Hit) and word type (Negative Word, Positive Word), $F(2, 40) = 6.05$. $p < .01$, $\eta_p^2 = .23$. This interaction indicated that participants were less accurate to categorize words as positive following FAs, compared to negative words following FAs, $t(20) = 2.81$, $p < .05$, $\eta_p^2 = .28$. Accuracy was similar for categorizing positive vs. negative words following either Fast Hits, $t(20) = -1.38$, $p > .10$, $\eta_p^2 = .09$, or Slow Hits, $t(20) = -1.01$, $p > .10$, $\eta_p^2 = .05$. Furthermore, the main effect of action type approached significance, $F(2, 40) = 3.00$, $p = .06$, $\eta_p^2 = .13$, indicating higher accuracy following Fast Hits compared to FAs, $F(1, 40) = 7.58$, $p < .05$, $\eta_p^2 = .28$ (see Table 3). Finally, the main effect of word type was not significant, $F < 1$.

A similar interaction between action type and word type was observed in Experiment 2, $F(1, 28) = 14.39$, $p < .001$, $\eta_p^2 = .51$, and Experiment 3, $F(2, 42) = 14.62$, $p < .001$, $\eta_p^2 = .41$, but not in Experiment 4, $F(1, 34) = 1.63$, $p > .10$, $\eta_p^2 =$

.09. Also, a similar effect of action type was observed in Experiments 2-4 (Experiment 2: $F(2, 28) = 4.17$, $p < .05$, $\eta_p^2 = .23$; Experiment 3, $F(2, 42) = 9.28$, $p < .001$, $\eta_p^2 = .30$; Experiment 4, $F(2, 34) = 8.17$, $p = .001$, $\eta_p^2 = .33$). A significant effect of word type was observed in Experiment 2, $F(1, 28) = 6.11$, $p < .05$, $\eta_p^2 = .30$ and Experiment 3, $F(1, 42) = 6.56$, $p < .05$, $\eta_p^2 = .24$, with less accurate categorizing for negative compared to positive words. This effect was not observed in Experiment 4, $F(1, 34) = 1.63$, $p > .10$, $\eta_p^2 = .09$ (see Table 3).

Table 3.

Mean accuracy (% correct responses) in the evaluative categorization task.

		Exp 1		Exp 2		Exp 3		Exp 4	
		Neg	Pos	Neg	Pos	Neg	Pos	Neg	Pos
FAs	*M*	94.72	89.50	98.06	90.36	97.34	86.01	96.17	92.39
	SEM	1.83	1.66	0.92	1.43	0.72	3.07	1.69	2.31
Fast Hits	*M*	94.27	95.92	98.14	97.39	96.07	97.27	98.56	98.28
	SEM	1.25	1.18	0.80	1.31	0.95	0.71	0.40	0.43
Slow Hits	*M*	92.48	94.11	95.96	96.58	95.52	97.62	97.67	97.39
	SEM	1.75	1.29	1.66	0.64	0.62	0.52	0.58	0.51

Note. Exp = Experiment, Neg = Negative,Pos = Positive

Go/noGo task

To explore performance during the Go/noGo task, we analyzed the number and speed of executed actions during that task as a function of action type (FA, Fast Hit, Slow Hit). The analysis of the number of actions revealed a significant main effect, $F(2, 40) = 50.57$, $p < .001$, $\eta_p^2 = .72$. The number of FAs was significantly smaller than the number of Fast Hits, $t(20) = -5.50$, $p < .001$, $\eta_p^2 = .60$. Moreover, participants made significantly less Fast Hits compared to Slow Hits, $t(20) = -4.501$, $p < .001$, $\eta_p^2 = .50$. As expected, a significant main effect of action type was also observed for speed, $F(2, 40) = 275.36$, $p < .001$, $\eta_p^2 = .93$. RTs for Slow Hits were longer than RTs for Fast Hits, $t(20) = -24.37$, $p < .001$, $\eta_p^2 = .97$, while RTs for FAs were shorter than RTs for Slow Hits, $t(20) = 3.14$, $p = .005$, $\eta_p^2 = .94$, but longer than RTs for Fast Hits, $t(20) = -17.38$, $p < .001$, $\eta_p^2 = .33$ (see Table

4). A similar main effect of action type on the number of actions and speed was also observed in Experiments 2-4 (see Table 4).

Table 4.

Mean accuracy (number),latencies (ms) and effect sizes during the Go/noGo task.

			Exp 1	Exp 2	Exp 3	Exp 4
Accuracy	FAs	*M*	58.10	60.47	61.73	45.89
		SEM	6.33	5.73	6.10	5.20
	Fast Hits	*M*	106.00	78.87	102.45	98.67
		SEM	6.62	10.49	8.33	9.31
	Slow Hits	*M*	162.00	198.20	162.55	165.39
		SEM	6.27	11.02	8.45	8.97
	Effect of action	η_p^2	.72***	.76***	.60***	.70***
	FA vs. Fast	η_p^2	.60***	.14	.33**	.56***
	FA vs. Slow	η_p^2	.03	.69***	.90***	.30*
	Fast vs. Slow	η_p^2	.50	.90***	.39**	.45**
Speed	FAs	*M*	234.7	222.40	223.78	229.00
		SEM	5.33	2.66	4.62	5.69
	Fast Hits	*M*	221.77	204.93	209.25	223.50
		SEM	6.34	5.76	7.23	8.32
	Slow Hits	*M*	307.50	276.60	286.51	301.61
		SEM	7.02	3.80	8.32	8.12
	Effect of action	η_p^2	.93***	.91***	.60***	.88***
	FA vs. Fast	η_p^2	.33***	.44**	.36**	.06
	FA vs. Slow	η_p^2	.94**	.96***	.85***	.90***

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Relation between trait anxiety levels and the magnitude of the evaluative priming effect

To put to the test our third prediction, we assessed whether levels of trait anxiety of our participants were related to the size of the evaluative priming effect. To address this, for each participant of Experiments 1-3 (i.e. all

experiments in which a significant evaluative priming effect was found), the magnitude of evaluative priming was calculated as the difference in RT between incongruent action-word pairs (i.e. FA–positive and Fast Hit–negative) and congruent action-word pairs (i.e. FA–negative and Fast Hit–positive). The larger this difference score, the higher the influence of the preceding affective value of the action on the current evaluative categorization. Using a standard Pearson coefficient correlation analysis, we found across participants of Experiments 1-3 a significant negative correlation between levels of trait anxiety and these evaluative priming scores, , r = -.28, p < .05 (see Figure 3). This correlation showed that participants with higher levels of trait anxiety had a smaller evaluative priming effect. When including the non-significant evaluative priming results of Experiment 4 in this analysis, the correlation was no longer significant, r = -.19, p > .10.

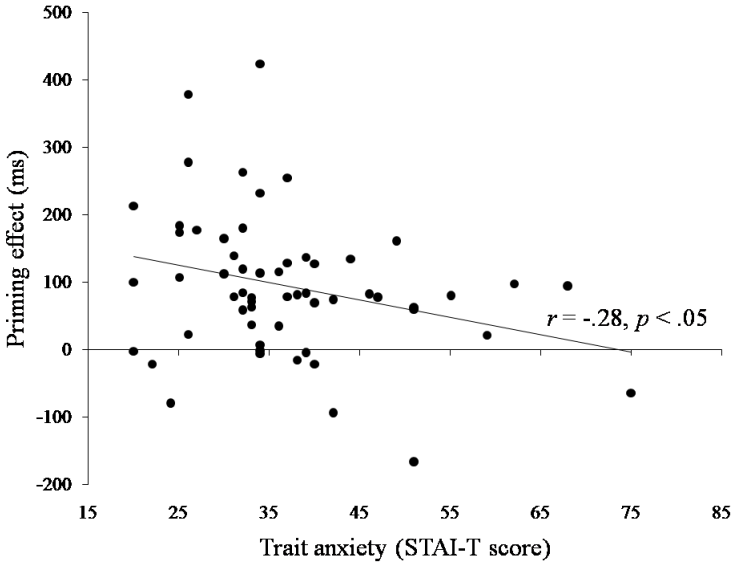

Figure 3. Significant negative correlation between evaluative priming effect [measured as the difference in RT latency between incongruent trials (i.e. FAs-Positive and Fast Hits-Negative) and congruent trials (i.e. FAs-Negative and Fast Hits-Positive)] and levels of trait anxiety (measured using a standard questionnaire, see Methods). This correlation was calculated for participants of Experiments 1-3 together where a significant evaluative priming effect was evidenced.

DISCUSSION

The results of our experiments reveal that simple self-generated actions during a speeded Go/noGo task are swiftly evaluated along a negative-positive dimension. This internal appraisal influences the valence categorization of an immediately following target word, even though these two different and non-overlapping events (i.e. action and word) belong to two clearly separated tasks performed with two different effectors. Our findings have several important implications that we address below.

Affective value of the action primes evaluative categorization

We are the first to report evaluative priming effects that are triggered by the putative affective value which is rapidly and in an online manner assigned to self-generated actions (correct vs. incorrect) via an internal meta cognitive feedback mechanism (Fernandez-Duque, Baird, & Posner, 2000; Winkielman, Schwarz, Fazendeiro, & Reber, 2003). These evaluative priming effects suggest that FAs were evaluated as more negative compared to Hits (either Fast or Slow, see results of Experiments 1-3) while Fast Hits were evaluated as more positive compared to FAs (see results of Experiments 2-3). Therefore, our behavioral results go beyond earlier studies showing that different psycho physiological reactions, like larger skin conductance responses, greater heart rate deceleration and larger startle potentiation usually follow incorrect compared to correct actions (Hajcak & Foti, 2008; Hajcak et al., 2003b). The novel contribution of our study is to show that beyond these enhanced arousal or attention orienting effects following the detection of these adverse events, dedicated internal monitoring systems enable organisms to rapidly map specific affective values (either negative or positive) onto self-generated actions (either incorrect or correct). This mechanism appears to operate along a genuine valence dimension, which is not restricted to errors or a specific class of deviant outcomes (De Bruijn et al., 2009). In addition to showing that actions performed during a simple Go/noGo task are actually quickly evaluated as negative or positive, our results suggest that this affective marking of the action functions at an abstract level of

action representation, as opposed to being bound to a specific motor output or command. The latter conclusion is supported by the fact that the exact same key presses were performed for correct (either Fast or Slow Hits) and incorrect actions (FAs) during our Go/noGo task. This abstract online affective appraisal of the action may in fact concern goal conduciveness (Frijda, 1987; Scherer, 1984, 1988), that is, an evaluation of whether an action is conducive (positive/Fast Hits) or obstructive (negative/FAs) for reaching the goals set out by the Go/NoGo task.

For FAs, we not only observed evaluative priming but also post-error slowing (Laming, 1979; Rabbitt, 1966) as indicated by slower evaluative categorizations following FAs than hits (either fast or slow). However, this general slowing effect did not interact with the evaluative priming effect. This lack of interaction suggests that the perceived accuracy of actions is quickly evaluated and used to guide future emotional as well as cognitive processing. Whereas post-error slowing likely deals with enhanced cognitive or attention control aimed at preventing errors to repeat over time (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Notebaert et al., 2009), evaluative priming seems to reflect the online and internal tagging of a specific affective value (negative vs. positive) to a particular action (incorrect vs. correct). This idea is supported by the observation that the size of the evaluative priming effect did reliably correlate (negatively) with levels of trait anxiety (r = -.28, p< .05) while no such relationship was found between anxiety and post-error slowing (r = -.004, p = .98) even though evaluative priming and post-error slowing were clearly related to one another (r = .42, p = .001). This relationship between evaluative priming and post-error slowing suggests that the emotional tagging of the action may be boosted if more efforts are exerted to prevent errors to reoccur, consistent with recent theoretical accounts (see Verguts, Notebaert, Kunde, & Wuhr, 2011). More generally, our new results are compatible with recent neuroscience findings showing that not only cognitive control systems are involved in action monitoring (and they likely include regions of the dorsal ACC besides deeper dopaminergic midbrain structures; see Klein et al., 2007), but also emotion

control systems (including the amygdala) play an important role in this process, at a similar early latency following action execution (see Pourtois et al., 2010).

Functional properties of automatic evaluation of actions

Another important new result of our study concerns the actual time-course of the action-word evaluative priming effect. It is well established that especially at short intervals (SOA) between the prime and the target, a substantial priming effect is observed (Fazio et al., 1986; Hermans, De Houwer, & Eelen, 2001). Here we found that the putative affective value of action influenced the subsequent evaluative categorization process only if the SOA was either 300 ms or 600 ms. However, we did not find a similar evaluative priming effect with an SOA of 1000 ms, whereas previous studies with word primes failed to find an effect with SOAs longer than 300 ms. This discrepancy might be due to the task-relevance of the (action) prime in our experiments. More specifically, whereas the action primes in our experiments were self-generated and informative regarding performance on the Go/NoGo task, the prime words in previous studies were provided by the experimenter and essentially irrelevant for any task.

Finally, we observed that the evaluative priming effect was clearly related to the level of trait anxiety of our participants, as the evaluative priming effect became smaller with increasing levels of trait anxiety. This observation is in accordance with results from evaluative priming studies using external stimuli as primes that already reported blunted priming effects in high anxious participants (Berner & Maier, 2004; Glaser & Banaji, 1999; Maier, Berner, & Pekrun, 2003). More generally, this significant correlation is consistent with previous action-monitoring studies which have shown that high trait anxious participants usually exhibit action-monitoring deficits, indicated by impairments to decode or read out the actual value of their actions using internal monitoring processes and swiftly relate it to (positive or negative) external performance feedback information presented in the environment (Aarts & Pourtois, 2012; Hajcak, McDonald, & Simons, 2003a). Consistent with this view, a correlational analysis showed that participants with higher levels of trait anxiety exhibited a blunted evaluative priming effect. Hence, these results suggest that in these individuals,

the rapid attribution process linking a specific value (either positive or negative) to an action (either correct or not) may somewhat be impaired, such that their online and internal action-monitoring processes can in turn only weakly prime the immediately following evaluative categorization process. Alternatively, high anxious participants may show less priming than low anxious participants if they have a bias to focus exaggeratedly their attention on internal representations (Eysenck, 1992; Muris et al., 2005). However, this alternative account appears unlikely because high anxious participants were as fast (non-significant action type x word type x anxiety level interaction: $F(2, 148) = 1.14$, $p > .10$) and accurate (non-significant action type x word type x anxiety level interaction: $F < 1$) as low anxious participants in orienting towards the target word following the action. Likewise, high and low anxious participants emitted a similar number of FAs, $t(74) = -1.36$, $p > .10$, nor did they differ in response speed, $t < 1$, during the speeded Go/noGo task. Hence, the present results also suggest that our new action-word evaluative priming paradigm may be suited to reveal specific impairments in action-monitoring processes, such as observed in psychopathological conditions or in individuals with certain personality traits (e.g. enhanced levels of trait anxiety).

Conclusions

The results of this study show, for the first time, that the valence of simple self-generated actions is swiftly appraised. Unwanted FAs made during a simple Go/noGo task are actually perceived as more negative events compared to Fast Hits, while the latter events are perceived as more positive than the former outcomes. This affective appraisal of the action is in all likelihood based on the actual perceived goal conduciveness of the action, as achieved through a rapid and efficient internal action-monitoring process. If a target word is presented within 600 ms following one of these two actions and shares the same intrinsic valence as the goal conducive or obstructive action, then participants are quicker and better at categorizing this word as either positive or negative, revealing a genuine action-word evaluative priming effect. No such effect is seen if 1000 ms elapses between the action and the onset of the target word, suggesting that

this effect is short-lived and automatic. Finally, this effect is blunted in participants showing enhanced levels of trait anxiety, suggesting that (i) it is most likely the affective value of the action used as prime which is driving this strong evaluative priming effect; (ii) these participants have action-monitoring difficulties in linking specific affective values (either positive or negative) to their self-generated actions (either correct or incorrect).

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CHAPTER 6: DIFFERENTIAL AFFECTIVE COLORING OF SELF-GENERATED ERRORS VS. CORRECT RESPONSES: EVIDENCE FROM ERPS

"The accuracy of simple actions is swiftly determined through specific monitoring brain systems. Event-related brain potential (ERP) studies have shown that error commission is associated with the generation of the error-related negativity (ERN/Ne), an early action-monitoring component following response onset. However, the exact functional meaning of this automatic evaluation of actions remains unclear. Whereas some studies have emphasized that it primarily reflects a basic reinforcement learning signal, other authors have suggested that it also indexes a motivational-significance effect, given that response errors usually call for rapid changes in the behavior, and have a negative connotation. In this study, we used a new method to decode indirectly the affective value of simple actions generated by participants during a standard Go/noGo task. Immediately after each response on the Go/noGo stimulus, participants categorized the valence of either a positive or a negative word. EEG was recorded concurrently. Behavioral results showed that response errors performed during the Go/noGo task led to a faster evaluative categorization of negative compared to positive words, with a symmetric result obtained following correct actions. Remarkably, this RT facilitation for the word (i.e. evaluative priming effect) was positively correlated to the magnitude of the early negative ERP component generated 300 ms earlier in response to the Go/noGo stimulus. Moreover, we found that whereas response errors influenced early perceptual stages of emotion word processing (EPN effect), correct responses mainly influenced a later process during emotion word processing (LPP effect). These results suggest that response errors are automatically assessed as more negative events compared to correct actions, an affective effect that can be captured by amplitude variations at the level of the ERN/Ne component.

INTRODUCTION

In daily life situations, we have to rapidly evaluate the outcome of our self-generated actions, and adapt our behavior appropriately when a potential mismatch is detected between the actual and the intended or desired action. Although this evaluative process seems to be largely automatic, few studies have actually corroborated this assumption and focused on a better characterization of the rapid and effortless decoding of the affective value of self-generated actions. According to the reinforcement learning framework (Frank, Woroch, & Curran, 2005; Holroyd & Coles, 2002), the accuracy of self-generated actions is swiftly determined via dedicated fronto-striatal loops in the brain. These monitoring systems quickly detect any deviance between the actual and intended or desired action, and in turn trigger a cascade of alerting reactions and remedial processes, when such a discrepancy is noticed (Rabbitt, 1966). Previous studies have shown that these alerting reactions concern not only changes in cognitive control, but also in emotion control brain processes (Carter et al., 1998; Hajcak & Foti, 2008; MacDonald, Cohen, Stenger, & Carter, 2000; Ochsner & Gross, 2005; Ridderinkhof, Nieuwenhuis, & Braver, 2007; van Veen & Carter, 2006) . For example, self-generated response errors committed during standard laboratory interference tasks have been associated with larger skin conductance reactions and a greater heart rate deceleration than correct actions (Hajcak et al., 2003b), as well as a larger startle potentiation (Hajcak & Foti, 2008), and differential early activation in the amygdala (Pourtois et al., 2010).

In a recent study (Aarts, De Houwer & Pourtois, in revision), we sought to assess whether self-generated actions were indeed not only swiftly marked as being correct or not by these putative reinforcement learning systems, but also as being good or bad, and hence quickly appraised along an affective dimension. To address this question, we developed a new paradigm in which actions performed by participants during a standard Go/noGo task were immediately followed by evaluative words (either positive or negative) requiring overt discrimination. We conjectured that if actions (serving as primes) were automatically appraised along an affective dimension, then the processing of the

valence of the immediately following word (serving as targets) should systematically be influenced at the behavioral level, as reflected in an evaluative priming effect (Fazio, Sanbonmatsu, Powell, & Kardes, 1986; Hermans, De Houwer, & Eelen, 1994): participants should be faster at categorizing negative words following errors compared to positive words, but faster for positive than for negative words following correct actions. The results of our behavioral study confirmed these predictions (see Aarts et al., in revision). Therefore, these results suggest that self-generated actions are quickly tagged by meta-cognitive systems (Fernandez-Duque, Baird, & Posner, 2000; Winkielman, Schwarz, Fazendeiro, & Reber, 2003) not only as being correct or not, but also as being good or bad. In the present study, we used event-related potentials (ERP) methods to gain insight into the electrophysiological time-course and possible manifestations of this evaluative priming effect.

Previous ERP studies have already shed light on the electrophysiological markers of action evaluation or performance monitoring. More specifically, several converging ERP studies described a specific ERP component associated with the early detection of response errors within the anterior cingulate cortex (ACC) (i.e. the error-related negativity – ERN, or negativity error - Ne; Dehaene, Posner, & Tucker; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Coles, Meyer, & Donchin, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN/Ne corresponds to a negative deflection peaking ~ 50 ms following the (motor) onset of an error, with a maximum amplitude over fronto-central midline recording sites, consistent with underlying brain generators likely located in the ACC (Dehaene et al., 1994; Holroyd, Dien, & Coles, 1998; van Veen & Carter, 2002). Correct actions performed under speed pressure are also associated with the generation of a similar but smaller, negative component at the same fronto-central recording sites and early latency following response onset (i.e. the correct-related negativity (CRN); Allain, Carbonnell, Falkenstein, Burle, & Vidal, 2004; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). The CRN is probably sharing generic brain generators in the ACC with the ERN/Ne (Roger, Bénar, Vidal, Hasbroucq, &

Burle, 2010). This early action-monitoring deflection (ERN/Ne-errors; CRN-correct responses) is usually followed by a large error-related component, the error-positivity (Pe), which peaks ~200 - 400 ms post-response onset over centro-parietal recording sites along the midline (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Nieuwenhuis, Ridderinkhof, Blow, Band, & Kok, 2001; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). Unlike the ERN/Ne that is reflecting an automatic (in the sense of preconscious) stage of error detection, the Pe is thought to reflect a more elaborate, perhaps conscious stage of error detection, likely reflecting the accumulation of evidence that an error has been committed (Nieuwenhuis et al., 2001; Steinhauser & Yeung, 2010)

Although the ERN/Ne is thought to reflect primarily a reinforcement learning "mismatch" signal that rapidly informs about a discrepancy between the actual and the expected motor outcome (Frank et al., 2005; Holroyd & Coles, 2002) or perhaps about the occurrence of a response conflict between an erroneous and error-correcting response Botvinick, Braver, Barch, Carter, & Cohen, 2001, other studies have emphasized the link between the (size of the) ERN/Ne and concurrent changes in motivation or affect. Overactive error-monitoring processes and increased ERN/Ne (but not Pe) amplitudes have for example been observed in patients with obsessive compulsive disorders (Endrass, Klawohn, Schuster, & Kathmann, 2008; Gehring, Himle, & Nisenson, 2000; Hajcak & Simons, 2002; Johannes et al., 2001; Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005), in healthy students showing higher levels of sub clinical trait anxiety (Aarts & Pourtois, 2010; Hajcak, McDonald, & Simons, 2003a; Moser, Moran, & Jendrusina, 2012) or negative affect (Hajcak, McDonald, & Simons, 2004). In line with these studies, Luu, Collins, & Tucker (2000) initially suggested that the ERN/Ne component may reflect a negative motivational or affective reaction to errors, even though, no study to date has formally linked amplitude-changes at the level of the ERN/Ne to the (implicit) differential emotional or motivational processing of these adverse or negative events. A first goal of our study was to assess whether such a brain-behavior relationship could

be found, given that our previous study confirmed that self-generated response errors were "automatically" marked as more negative compared to correct actions (see Aarts et al., in revision). More specifically, we sought to demonstrate that if the ERN/Ne reflects the automatic affective evaluation of self-generated actions, then its amplitude might be related to the evaluative priming effect or to how fast participants categorized the emotional words following actions are either positive or negative. Such an outcome would provide more direct (possibly causal) evidence for the involvement of this early action-monitoring ERP component in the automatic affective marking of self-generated actions (Luu et al., 2000; Pourtois et al., 2010).

Moreover, the use of ERP methods enabled us to track the time-course of the action-word evaluative priming effect. Hence, the second main goal of our study what to use this time-resolved neurophysiological technique to better characterize when precisely following emotional word onset the processing of the valence of the word was substantially influenced by the affective value of the just preceding self-generated action. To address this question, we primarily focused on two specific time intervals/components following emotional word onset during which reliable differential effects of the emotional valence and/or arousal value of the word were systematically found across several earlier ERP studies, namely the EPN and LPP (Kissler, Assadollahi, & Herbert, 2006). Depending on the task demands and specific verbal stimulus sets used, early, late or a combination of both effects can be seen following (written) emotional word onset. Usually an enhanced early posterior negativity (EPN) has been found ~ 200-250 ms post-stimulus onset for emotional in comparison to neutral words (Herbert, Junghöfer, & Kissler, 2008; Kissler, Herbert, Peyk, & Junghöfer, 2007; Kissler, Herbert, Winkler, & Junghöfer, 2009; Schacht & Sommer, 2009a). Emotional words also lead to a larger ERP signal than neutral words at the level of the P300 component (Naumann, Bartussek, Diedrich, & Laufer, 1992) or the Late Positive Potential - LPP (Naumann et al., 1992). These two differential ERP effects (EPN and LPP) for emotional relative to neutral words are thought to be

related primarily to the processing of the arousal value of the words (Kissler et al., 2006).

As it turned out, the action-word sequence led to a substantial distortion of the ERP signal time-locked to the onset of the emotional words in our study. This distortion was primarily accounted for by large residual effects (occurring in the pre-stimulus baseline) of the preceding actions (especially in the case of response errors eliciting prominent ERN/Ne and Pe components) onto the visual ERP generated in response to the emotional words. These words were always presented 300 ms (fixed interval) following action execution, in accordance with our previous behavioral study (Aarts et al., in revision) where we found that this specific interval between the offset of the action and the onset of the word was optimal to obtain a reliable evaluative priming effect. However, this specific setting was apparently not compatible with the recording of artifact-free ERP components generated in response to the visual emotional words. To overcome this limitation and to be able to identify nonetheless reliable EPN and LPP-like effects with high confidence, participants performed an additional "localizer" experiment. In this auxiliary experiment, the negative and positive words presented during the main evaluative categorization task were presented now in isolation (without any interfering actions embedded between these visual stimuli) and in random order, in addition to neutral words used as a control condition. The ERP data recorded during this localizer allowed us to carefully characterize, using an independent ERP data set, the emotion-related EPN and LPP effects related to the differential processing of these emotional written words, compared to neutral words. During this localizer experiment, participants performed a standard one-back task (requiring the detection of rare, immediate repetitions of the same words) in order to balance task demands and attention across these three conditions (i.e. neutral, positive and negative words). The information gathered from this independent "localizer" run regarding the latency and morphology of visual emotional word processing ERP effects allowed us to establish whether the distorted EPN and/or LPP components recorded during the main evaluative priming experiment were reliably modulated by the putative

valence of the preceding action. Accordingly, we assessed whether the processing of positive vs. negative words was different at the level of the EPN and/or LPP when the preceding action was a response error. We also assessed whether positive vs. negative words were associated with different EPN and/or LPP effects when the preceding action was a correct response.

METHODS

Participants

Twenty undergraduate students (18 female; Age: *M* = 21.65, *SEM* = .39) took part in the present study. The data of five participants had to be excluded from the analyses because the number of EEG epochs per condition was too limited in order to calculate reliable ERP waveforms (i.e. < 10; *n* = 4) or because of excessive noise in the continuous EEG data (*n* = 1). The final sample contained 15 participants (14 female; Age: *M* = 21.4, *SEM* = .38). They were all right-handed, native Dutch speakers who did not have a history of neurological or psychiatric disease, and had normal or corrected-to-normal vision. The study was approved by the local ethics committee. All participants were paid 20 Euro.

Stimuli

Go/noGo task

Visual stimuli consisted of an arrow (subtending 11.4◦ ×0.05◦ of visual angle at a 60 cm viewing distance) that was presented in the center of a white homogenous background, and oriented either upward or downward (see Figure 1). The arrow was first black, and could then turn either green or turquoise. These two colors were matched for luminance. These different combinations of color and orientation were used as cues in the Go/noGo task.

Evaluative categorization task

Targets were 30 positive and 30 negative words, either nouns or adjectives (see Table 1), and were selected from the Dutch affective rating list of Hermans and De Houwer (1994). *T*-tests showed that these positive and negative words differed significantly on the affective dimension, $t(58) = 36.57$, $p < .001$, $\eta_p^2 = .95$,

but not on the familiarity dimension, $t < 1$, nor with respect to the number of letters, $t < 1$.

Word repetition detection task

Thirty positive, 30 negative and 30 neutral words, either nouns or adjectives (see Table 1), were selected from the Dutch affective rating list of Hermans and De Houwer (1994). The positive and negative words were the same as those used for the evaluative categorization task. *T*-tests showed that neutral words were significantly different from negative and positive words on the affective dimension, $F(2, 89) = 620.72$, $p < .001$, but not on the familiarity dimension, $F(2, 89) = 1.48$, $p > .10$, nor with respect to the number of letters, $F < 1$.

Table 1.

Words selected from the Dutch affective rating list of Hermans and De Houwer (1994)

Positive	*Negative*	*Neutral*
applaus (applause)	aids (aids)	autobus (bus)
baby (baby)	brutaal (impudent)	beton (concrete)
bloemen (flowers)	dief (thief)	boog (arc)
bruid (bride)	dood (dead)	bord (plate)
cadeau (present)	drugs (drugs)	broek (pant)
droom (dream)	geweren (guns)	buik (belly)
engel (angel)	graf (tomb)	doos (box)
feest (part)	haat (hate)	eend (duck)
geboorte (birth)	hatelijk (hasty)	gemiddeld (average)
geschenk (gift)	hitler (hitler)	geur (smell)
goud (gold)	hoer (hore)	golf (wave)
Hawaii (Hawaii)	kanker (cancer)	hek (fence)
hemel (heaven)	koud (cold)	hoed (hat)
humor (humor)	moord (murder)	hoofdsteun (head support)
knuffel (hug)	ongeval (accident)	inkt (ink)
lente (spring)	ongezond (unhealthy)	klei (clay)
leven (life)	oorlog (war)	krant (newspaper)
liefde (love)	pijn (pain)	kruid (herb)
melodie (melody)	puist (pustule)	mand (basket)
omhelzing (embrace)	ruw (rude)	muren (walls)
oprecht (sincere)	sluw (sly)	normaal (normal)
parfum (parfume)	spin (spider)	olifant (elephant)
regenboog (rainbow)	stank (stench)	schaar (scissors)
romantiek (romanticism)	vals (false)	slager (butcher)
spel (game)	vijandig (hostile)	takken (branches)
trots (proud)	virus (virus)	tas (bag)
trouw (fidelity)	vuil (dirty)	venster (window)
vakantie (holiday)	vulgair (vulgar)	vierkant (square)
vrede (peace)	zwak (weak)	voet (foot)
zomer (summer)	zweer (sore)	wolk (cloud)

Procedure

Go/noGo task and evaluative categorization task

Participants performed a standard speeded Go/noGo task (Vocat, Pourtois, & Vuilleumier, 2008) interleaved with a visual word categorization task (see Figure 1). Actions performed during the speeded Go/noGo task actually served as primes whereas words were used as targets in analogy with a conventional prime-target sequence during evaluative priming. Each trial started with a fixation cross that lasted for 500 ms. Afterwards, a black arrow, either oriented up or down, was presented at the position previously occupied by the fixation cross. After a variable interval ranging from 1000 ms to 2000 ms, the black arrow became either green or turquoise while its orientation could either remain identical or shift in the opposite direction compared to the initial black arrow. When the black arrow turned green and the orientation remained unchanged, participants were instructed to press a pre-defined button of the response box as fast as possible with the index finger of their left (non-dominant) hand (Go trials). However, participants had to withhold responding when either the arrow became green but changed orientation, or when the arrow became turquoise and kept its initial orientation, enabling two noGo trial types. Instructions emphasized both speed and accuracy, such that not only accuracy, but also the perceived speed was later evaluated as being either correct or incorrect. For each trial, speed was evaluated using an individually calibrated RT limit computed during a training block that preceded each session of two test blocks. This limit was thus calculated and updated three times in total (before Blocks 1 and 2 – Session 1, before Blocks 3 and 4 – Session 2, and before Blocks 5 and 6 – Session 3). This procedure allowed us to deal with unspecific learning effects over time and maintain a high number of response errors throughout the experimental session. For the first session, the upper limit was set to 70% of the mean RT from the first training block. For the two subsequent sessions, this upper limit was updated and set to 80% of the mean RT during the respective training block. Hence, this procedure required participants to respond at least 30% faster (first session) or 20% faster (second and third sessions) on Go trials

than their average speed during the respective training block. This procedure ensured a sufficient number of response errors on noGo trials and allowed us to distinguish between Fast Hits (i.e. correct responses on Go trials that were made faster than the individually-titrated RT limit) and Slow Hits (i.e. correct responses on Go trials that were made slower than the RT limit). Errors were formally defined as overt responses on noGo trials (i.e. False Alarms - FAs), while correct inhibitions corresponded to correctly withheld responses on the same noGo trials.

Three hundred milliseconds after an action was executed, a target word was presented. For correct inhibitions, the target word was presented 1800 ms after the presentation of the colored arrow. Participants were instructed to categorize the valence of the target word (positive or negative) as fast and as accurately as possible by pressing one of two predefined keys of the response box using their dominant hand. Hence, the evaluative word categorization task was executed with a different effector than the Go/noGo task. The target word remained on the screen until the participant responded or 3000 ms elapsed. In order to balance the presentation of positive vs. negative words following Fast Hits, Slow Hits, Correct Inhibitions, and FAs, the target word that was presented following an action was selected randomly on each trial. After the word categorization, participants received feedback informing them about their accuracy for the two consecutive tasks. The feedback for the Go/noGo task indicated whether the performed action was correct (and fast enough), incorrect or too slow, while the feedback for the word categorization could be either correct or incorrect. Both feedback signals remained on the screen for 2000 ms.

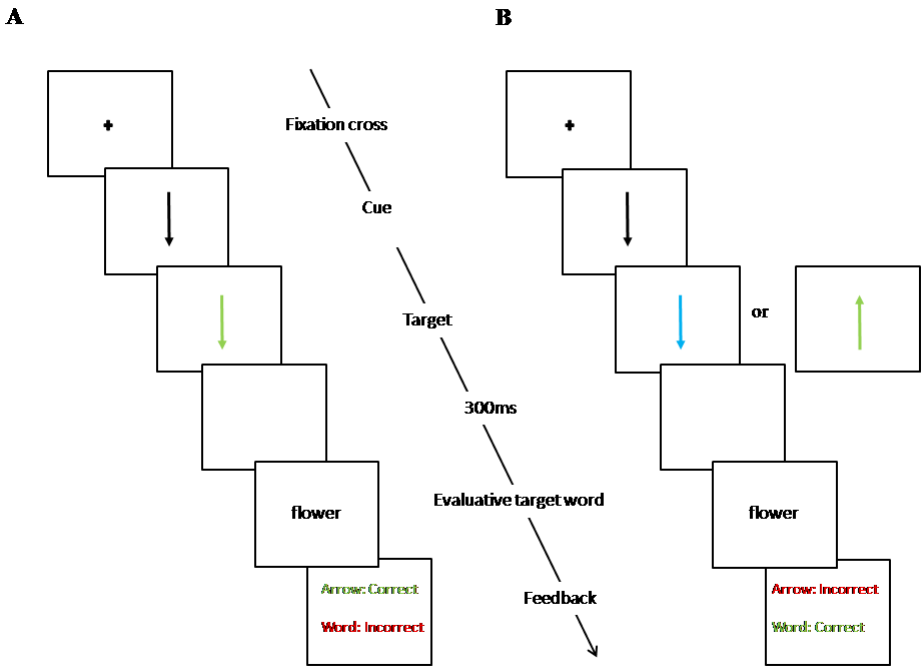

Figure 1. Stimuli and task. (A) Go trial, (B) NoGo trial.

After a practice phase including 24 trials, the experiment was divided into 3 sessions, each starting with a training block (containing 28 trials: 20 Go and 8 noGo trials), followed by two test blocks (each containing 72 trials: 48 Go and 24 noGo trials). Note that participants were unaware that training blocks were actually used as calibration blocks to compute the RT limit used during the two following test blocks. Trial presentation was randomized within blocks. Between blocks, a small break (no longer than 5 min) was introduced. The whole experiment included 540 trials and lasted on average 50 min. Stimulus presentation and response recording were controlled using E-prime software (V2.0., http://www.pstnet.com/products/e-prime/).

Word repetition detection task (localizer)

In this task (always following the Go/noGo plus evaluative categorization tasks), participants had to press a predefined button on the response box when they detected a word that was identical to the previous one (i.e. one-back task). Hence, we used a low-load memory task requiring a shallow lexical and semantic processing of the words, while task demands were balanced across the three emotion word conditions. Every word (N=30 per emotion category) was

presented once in random order for 550 ms and immediately followed by a blank screen (1000 ms). In total, 6 words (2 words of each emotion category) out of 90 were repeated and had to be overtly detected. The appearances of these 6 immediate repetitions in the word list were alternated across participants.

Analyses of behavioral data

Go/noGo task

Accuracy and RTs were analyzed separately using repeated measures analyses of variance (ANOVAs) with the type of action (FA, Fast Hit or Slow Hit) as within-subject factor.

Evaluative categorization task

Accuracy and RTs (for correct responses) were analyzed using ANOVAs as a function of (i) the valence of the target word (either positive or negative) and (ii) the type of action (FA, Fast Hit or Slow Hit) preceding word presentation. We did not include in these analyses trials corresponding to correct inhibitions because no overt action was performed in this condition. Separate statistical analyses performed on these trials showed that the evaluative categorization was not significantly influenced by these correct inhibitions. Following a correct inhibition, the speed to categorize negative words was similar to that used to categorize positive words ($t < 1$).

Word repetition detection task (localizer)

Accuracy was analyzed using repeated measures ANOVA with the type of emotion word (negative, neutral, positive) as within-subject factor.

EEG acquisition and pre-processing

Go/noGo task

Continuous EEG was acquired at 512 Hz using a 128-channel (pin-type) Biosemi Active Two system (http://www.biosemi.com) referenced to the CMS-DRL ground. ERPs of interest were computed offline following a standard sequence of data transformations (Picton et al., 2000): (1) -500/+1000 ms segmentation around the motor response, (2) pre-response interval baseline

correction (from -500 ms to 0 ms), (3) vertical ocular correction for blinks (Gratton, Coles, & Donchin, 1983) using the difference amplitude of two electrodes attached above and below the left eye, (4) artifact rejection [*M* = -72/+72, *SEM* = 2.0 amplitude scale (μV) across participants], (5) averaging of trials for each of the two main conditions separately (FA vs. Fast Hit), and (6) 30 Hz low pass digital filtering of the individual average data. We primarily contrasted FAs to Fast Hits but not Slow Hits, because these latter trials were more frequent than FAs, whereas Fast Hits were almost as frequent as FAs. Moreover, unlike FAs or Fast Hits, Slow Hits did not lead to any significant evaluative priming effect (see behavioral results here below).

Evaluative categorization task

The sequence of data transformations was similar to the one used for the Go/noGo task with the notable exception that the baseline correction was not performed using the entire pre-stimulus interval (500 ms preceding word onset), but using the -50/+50 ms around word stimulus onset in order to downplay as far as possible lingering effects of the preceding response-related ERPs (e.g. ERN/Ne and Pe components following error commission) on the current visual ERPs elicited by the emotion word, which always followed the action with a constant interval of 300 ms. Four different ERP averages were computed for each participant: negative words following FAs; positive words following FAs; negative words following Fast Hits; positive words following Fast Hits.

Word repetition task (localizer)

The sequence of data transformations was similar to the one used for the Go/noGo task and three individual ERP averages corresponding to the three main emotion word conditions were eventually computed. The deviant immediate repetitions of words (n=6) requiring overt detection were not included in these averages.

ERP data analyses

Go/noGo task

We primarily focused on two well-documented error-related ERP components following incorrect response onset (Falkenstein et al., 2000), on the ERN/Ne, with a maximum negative amplitude over fronto-central electrodes along the midline (electrode FCz) early on following motor execution (~0 - 100 ms post-response onset), immediately followed by the Pe component (~150 - 300 ms post-response onset), with a maximum positive amplitude over more posterior and central electrode locations along the midline (electrode Cz). For each ERP component and each condition separately (FA vs. Fast Hit), we calculated the area under the curve, during the 0-60 ms interval post-response onset at electrode FCz for the ERN/Ne amplitude, and during the 170-210 ms interval post-response onset at electrode Cz for the Pe component. The selection of these two specific scalp locations (and time windows) was based on the topographic properties of the present dataset, as well as based on converging results obtained in previous ERP studies using the same task (Aarts & Pourtois, 2010).

Statistical analyses were performed on the mean amplitude of each area using a paired t-test (FA vs. Fast Hit). We also performed brain-behavior correlation analyses using the amplitude of the ERN/Ne (or CRN in the case of Fast Hits) and RTs for the evaluative categorization task. We sought to assess whether the error-related brain reactions occurring during the Go/noGo task might predict the size of the RT facilitation for the immediate orthogonal emotion word categorization task. More specifically, we assessed whether the ERN/Ne-CRN amplitude difference (reflecting accuracy sensitivity roughly) might be related to the RT facilitation for congruent trials (FA-negative word and Fast Hit-positive word) compared to incongruent trials (FA-positive word and Fast Hit-negative word). We therefore computed an evaluative priming effect by subtracting congruent trials from incongruent trials and evaluated, using a Pearson coefficient correlation, whether this priming effect might be related to amplitude changes occurring at the level of the ERN/Ne-CRN component. We

also assessed whether the evaluative priming effect may be predicted by amplitude changes occurring at the level of the Pe component and accordingly we computed a similar amplitude difference between FAs and Fast Hits for this later deflection.

Word repetition task (localizer)

A major problem arose for the visual ERPs recorded during the main evaluative categorization task because the baseline of these ERPs was strongly contaminated by the residual activity from the preceding action. To indirectly overcome this problem and to be able to explore the electrophysiological time-course of emotion word processing depending on the accuracy of the preceding action (FA vs. Fast Hit) despite an obvious distortion of the ERP signal/morphology, we used an additional word repetition task as an independent localizer. This localizer was primarily used to establish time intervals during which a reliable ERP difference could be detected between emotional and neutral words, with a proper pre-stimulus baseline. To formally isolate these emotion-sensitive time periods, we submitted the ERP data of the localizer to a standard topographical mapping analysis. The rationale and basic principles of this analysis have been extensively described elsewhere (Michel, Seeck, & Landis, 1999; Murray, Brunet, & Michel, 2008; Pourtois, Delplanque, Michel, & Vuilleumier, 2008). The topographical analysis was run on the ERP data from stimulus onset until 500 ms after emotion word stimulus onset (i.e. 256 consecutive time frames at 512 Hz sampling rate), using a standard clustering (or spatio-temporal segmentation) method (K-means; Pascual-Marqui et al., 1995). Following standard practice, the dominant scalp topographies (identified in the group-averaged data) that were found to discriminate between neutral and emotional words (with a focus on the EPN and LPP components) were then fitted to the ERPs of each individual subject using spatial fitting procedures to quantitatively determine their representation across subjects and conditions. For each time interval (either EPN or LPP), the resulting Global Explained Variance (GEV) values were finally entered in a repeated-measure ANOVA with two within-subject factors, emotion (neutral, negative and positive) and map

configuration (i.e. the electric field distributions previously identified by the spatial cluster analysis). These analyses were carried out using CARTOOL software (Version 3.34; developed by D. Brunet, Functional Brain Mapping Laboratory, Geneva, Switzerland).

Evaluative categorization task

The previous analysis enabled us to identify two non-overlapping time intervals (corresponding to the EPN and LPP) during which the processing of emotional (either positive or negative) words differed from neutral words. These specific time intervals were then used during the main evaluative categorization task to assess whether the accuracy of the preceding action influenced emotion word processing or not. In a first step, we ran paired t-tests (negative vs. positive words; alpha level set to .01) for all 128 electrodes concurrently, separately for FAs and Fast Hits, on the amplitude of the ERP signal during these two specific emotion sensitive time intervals (EPN and LPP). Given the obvious distortion of the ERP signal induced by the preceding action, we had to perform this first analysis comparing positive to negative words separately for FAs and Fast Hits. This first-pass statistical analysis allowed us to reveal clusters of electrodes where a reliable difference occurred between the processing of negative vs. positive words, separately for FAs and Fast Hits. In a second step, we verified, using repeated measures ANOVAs whether the amplitude of the ERP signal at these pre-selected clusters and during these two specific time-intervals was reliably influenced by the type of action (FA vs. Fast hit) as well as the valence of the word (negative vs. positive).

RESULTS

Behavioral results

Outliers

Trials with RTs shorter than 150 ms (FAs: *M* = 3.02, *SEM* = .87; Fast Hits: *M* = 4.47, *SEM* = 1.76) or longer than 500 ms (FAs: *M* = .90, *SEM* = .39; Slow Hits: *M* = 3.73, *SEM* = 1.23) during the Go/noGo task were discarded, as were trials of the

evaluative categorization task for which the RT exceeded 2.5 *SD* above or below the mean RT computed per condition (Negative: *M* = 2.91, *SEM* = .22; Positive: *M* = 3.10, *SEM* = .28).

Go/noGo task

The number of actions (FA, Fast or Slow Hit) differed significantly, $F(2, 28) = 44.22$, $p < .001$. Participants made as many FAs as Fast Hits, $t(14) = -1.55$, $p > .10$. Moreover, participants made significantly less Fast Hits compared to Slow Hits, $t(14) = -5.58$, $p < .001$, as well as less FAs compared to Slow Hits, $t(14) = -11.06$, $p < .001$. Action types also reliably differed regarding speed, $F(2, 28) = 138.97$, $p < .001$. RTs for Slow Hits were longer than RTs for Fast Hits, $t(14) = -15.02$, $p < .001$, while RTs for FAs were shorter than RTs for Slow Hits, $t(14) = -17.30$, $p < .001$, but longer than RTs for Fast Hits, $t(14) = 3.32$, $p < .01$ (see Table 2). These results were compatible with previous findings obtained with the same Go/noGo task (Aarts & Pourtois, 2010, 2012).

Table 2.

Mean number of actions and RT latencies (ms) during the Go/noGo task, separately for each condition.

		Number		Speed (ms)	
		M	*SEM*	*M*	*SEM*
FAs	M	60.47	5.73	222.40	2.66
Fast Hits	M	78.87	10.49	204.93	5.76
Slow Hits	M	198.20	11.02	276.60	3.80

Evaluative Categorization Task

Speed

The ANOVA performed on the mean RTs for correct responses revealed a significant interaction effect between action type and word type, $F(1, 28) = 13.60$, $p < .001$. This interaction resulted from faster evaluative categorizations when the valence of the word was congruent with the putative affective value of the action. More specifically, RTs for negative words following FAs were shorter compared to RTs for positive words following FAs, $t(14) = -3.28$, $p < .01$, while

symmetrically, participants tended to categorize positive words faster compared to negative words when they followed Fast Hits, $t(14) = 6.30$, $p < .05$. Following Slow Hits, no significant RT difference emerged between negative and positive words, $t < 1$. The main effect of word type was not significant, $F(1, 28) = 2.02$, $p > .10$. By contrast, the ANOVA revealed a significant main effect of action type, $F(2, 28) = 26.80$, $p < .001$, reflecting overall longer RTs for words following FAs compared to words following either Fast Hits, $F(1, 14) = 32.47$, $p < .001$, or Slow Hits, $F(1, 14) = 31.29$, $p < .001$, an effect in line with a systematic post-error slowing (Danielmeier & Ullsperger, 2011; Rabbitt, 1966) (see Figure 2A).

Accuracy

The ANOVA performed on accuracy data (i.e. % correct responses) revealed a significant interaction effect between action type (FA, Fast Hit, Slow Hit) and word type (Negative Word, Positive Word), $F(1, 28) = 14.39$, $p < .001$. This interaction indicated that participants were less accurate to categorize words as positive following FAs, compared to negative words following FAs, $t(14) = 4.32$, $p < .001$. Accuracy was similar for categorizing positive vs. negative words following either Fast, $t < 1$, or Slow Hits, $t < 1$. Furthermore, the main effect of action type approached significance, $F(2, 28) = 4.17$, $p < .05$, indicating higher accuracy following Fast Hits compared to FAs, $F(1, 28) = 13.41$, $p < .01$. Finally, the main effect of word type was also significant, $F(1, 28) = 6.11$, $p < .05$ (see Figure 2B).

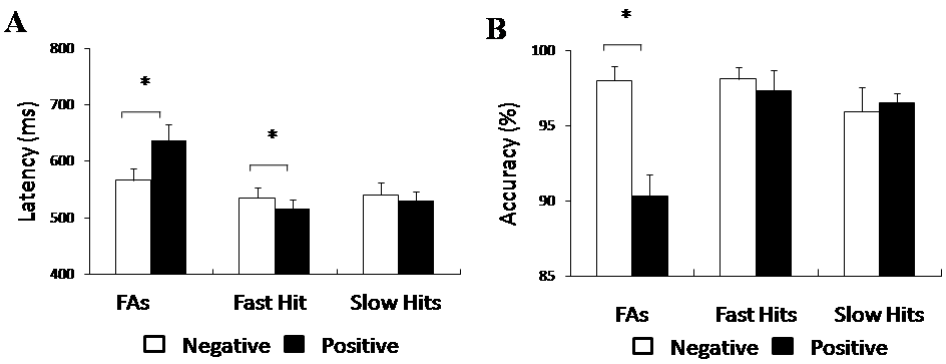

Figure 2. (A) Mean RTs (+ 1 *SEM* for bars) for correct evaluative categorizations as a function of prime type (FA, Fast Hit, or Slow Hit) and word type (Negative or Positive Words). (B) Mean accuracy in percentages (+ 1 *SEM* for bars) for correct evaluative categorizations as a function of prime type (FA, Fast or Slow Hit) and word type (Negative or Positive Words).

ERP results

Go/noGo task

When participants committed FAs, there was a clear sharp negative deflection that peaked roughly ~30 ms post-response onset, with a maximum amplitude at fronto-central electrodes along the midline, including FCz. These electrophysiological properties are consistent with the ERN/Ne. Consistent with previous ERP studies (Falkenstein et al., 1991; Gehring et al., 1993), the amplitude of the ERN/Ne was reliably larger for FAs (i.e. response errors), relative to Fast Hits (i.e. correct responses) where a smaller negative component (CRN) was also visible, $t(14) = -4.0$, $p < .001$ (see Figure 3A).

This early negative component was immediately followed by a large positive potential, with maximum amplitude over more posterior scalp positions, including Cz. This error-related positive component was strongly attenuated for Fast Hits, $t(14) = 5.06$, $p < .001$ (see Figure 3A). These electrophysiological properties were compatible with the generation of a genuine error-related Pe component (Falkenstein et al., 2000; Ridderinkhof, Ramautar, & Wijnen, 2009.

Remarkably, we found that the evaluative priming effect [defined as the RT difference between incongruent (FA-positive word and Fast Hit-negative word) and congruent trials (FA-negative word and Fast Hit-positive word)] was actually related to the difference between the ERN/Ne and CRN component, $r = -.50$, $p =$

.05 (see Figure 3B). This result was important, because it suggests that the more the early fronto-central negative deflection following response onset differentiated between incorrect and correct actions, the larger the evaluative priming effect (i.e. RT facilitation for categorizing the valence of a word that was presumably compatible with the inferred value of the preceding action). These results confirmed that this early action-monitoring component is not only responsible for coding the accuracy of the action (correct vs. incorrect), but also probably its concurrent emotional or motivational significance (good for correct actions vs. bad for response errors). Moreover, despite the modest sample size (n=15), we found an almost significant correlation between the size of the ERN/Ne and the RT difference between negative and positive words following errors, r = -.49, p = .07 (see Figure 3C). This correlation showed that participants with a larger ERN/Ne component had subsequently a larger RT facilitation for categorizing negative relative to positive words. Symmetrically, we also observed a trend for an association between the CRN generated for Fast Hits and the subsequent RT facilitation to categorize positive relative to negative words following these correct actions, r = .39, p = .15 (see Figure 3D). This suggested that smaller CRN amplitudes were related to larger RT facilitations for positive compared to negative words. We did not find a similar correlation between the Pe component and the general evaluative affective priming effect, r = -.31, p > .10.

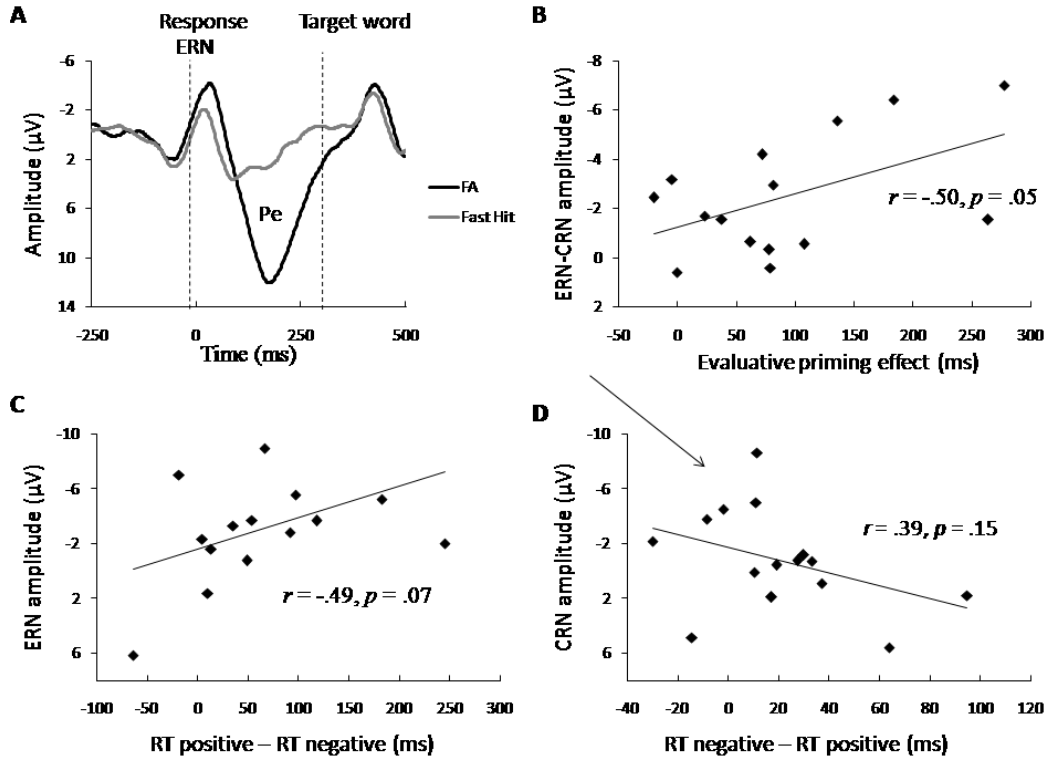

Figure 3. ERP results during the speeded Go/noGo task. (A) Grand average ERP waveforms (electrode FCz) for FAs and Fast Hits. FAs elicited an early negative component (ERN/Ne), followed by a large positive deflection (Pe), whereas Fast Hits elicited only a smaller early negative activity (CRN). (B) A significant positive correlation was found between the evaluative priming effect and the absolute amplitude difference between the ERN/Ne and CRN component. (C) A negative correlation was found between the ERN/Ne amplitude and the absolute RT difference between positive and negative words. (D) By contrast, a positive correlation was found between the CRN amplitude and the absolute RT difference between negative and positive words.

Word repetition detection task (localizer)

Using the topographical analysis, we found that the ERP signal was reliably influenced by the emotional content (positive or negative, relative to neutral) of the word during two non-overlapping time intervals. This analysis accounted for 90% of the variance. The first interval was 184-203 ms post-word onset, and the second spanned from 326 until 393 ms post-word onset. These latencies were compatible with an EPN and LPP effect, respectively (see Figure 4AB). Consistent with a sensitivity of these two ERP components to the emotional or arousal value conveyed by the words, statistical analyses performed on the GEV values extracted for these two topographical components confirmed that the EPN

topography explained more variance for emotional compared to neutral words [negative: $t(14) = 2.38$, $p < .05$; positive: $t(14) = -1.82$, $p = .09$] (see Figure 4C), and the LPP topography alike [negative: $t(14) = 2.75$, $p < .01$; positive: $t(14) = -2.83$, $p < .05$] (see Figure 4D).

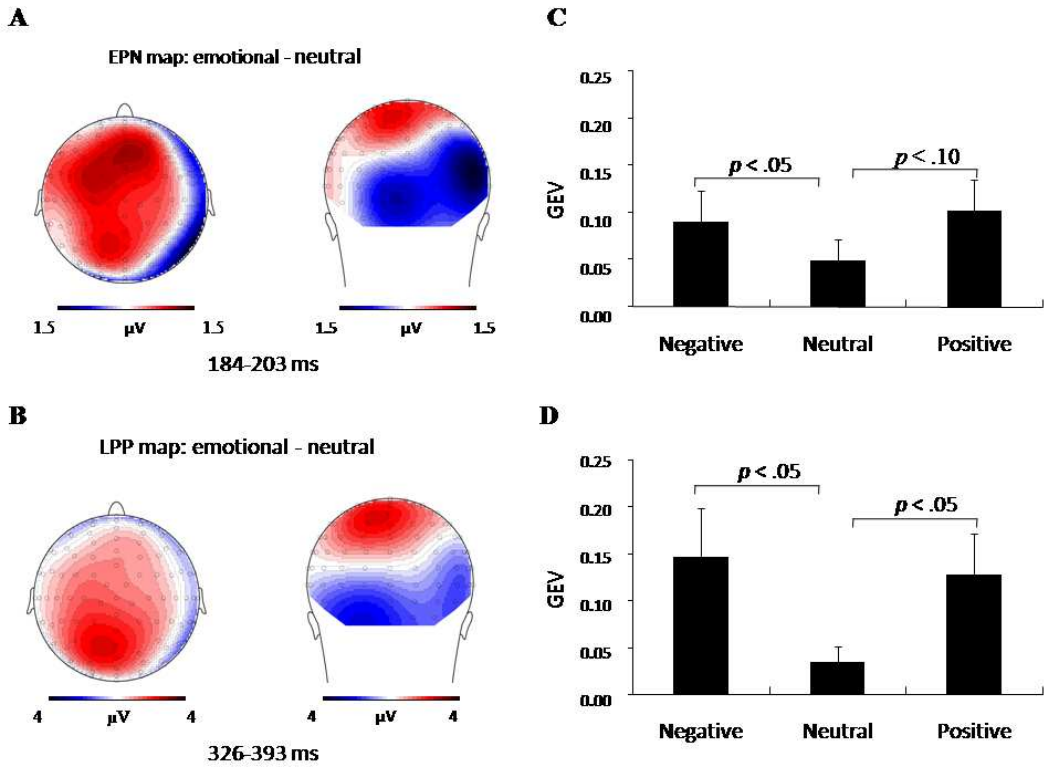

Figure 4. ERP results obtained for the localizer experiment. (A) The voltage map (horizontal and back views) of the EPN (184-210 ms post-word onset) for emotional vs. neutral words was characterized by a negative activity mainly at right occipital electrodes. (B) The voltage map (horizontal and back views) of the LPP (326-393 ms post-word onset) for emotional vs. neutral words showed a broad positive activity over centro-parietal electrode positions. (C) The EPN topographical component explained more variance for emotional compared to neutral words (see results section for exact numerical values). (D) Likewise, the LPP topographical component explained more variance for emotional compared to neutral words (see results section for exact numerical values).

Evaluative categorization task

Self-generated actions had a deleterious effect on the morphology and expression of visual ERPs generated in response to the visual words during the main evaluative categorization task. Action type (either FAs or Fast Hits) had a major influence on the expression of these ERPs time-locked to the onset of the word (see Figure 5AB). This substantial distortion of the ERP signal and the

presence of large response-locked ERP components during the pre-stimulus onset baseline (ERN/Ne and Pe components for errors, see also Fig. 3A) led us to establish the presence of reliable EPN and LPP effects during emotion word processing using an independent localizer (see Figure 4), where no self-generated actions were interleaved and could alter visual ERPs to word onset, as in the main experiment (see methods).

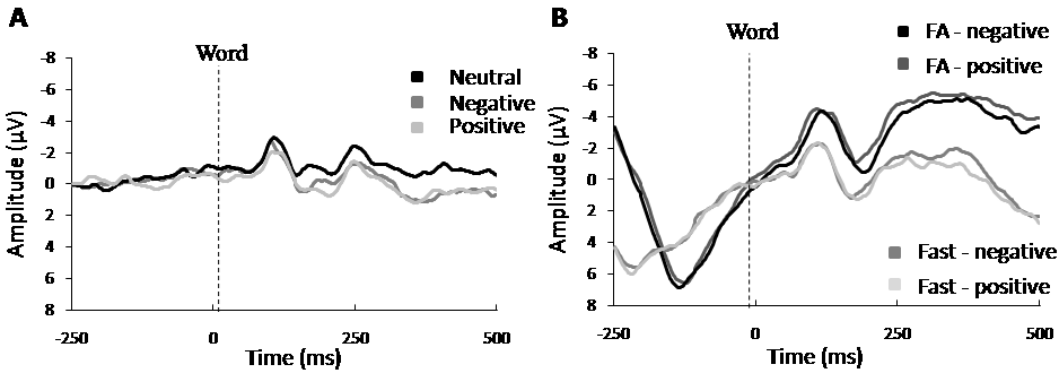

Figure 5. Grand average ERP waveforms (electrode Cz) time-locked to the onset of the word (A) during the localizer (neutral, positive and negative) and (B) during the main evaluative categorization task (positive and negative words either following FAs or Fast Hits).

A first statistical analysis based on running t-tests (see methods) showed that following errors (but not correct responses), a significant difference occurred between positive and negative words during the time-interval corresponding to the EPN at right occipital (B11, B14, B15) and left frontal electrodes (D2, D12, D19, D28 and D25). At these electrodes, the amplitude of the ERP signal was reliably larger for incongruent (positive words) compared to congruent (negative words) trials. By contrast, following correct actions (Fast Hits but not FAs), a reliable difference emerged between positive and negative words mainly during the time interval corresponding to the LPP component, mainly at right frontal electrodes (C1-C7 and C23), as well as at some additional scalp positions (A21, A22, A23, A30, B13, D7, D22, D23 and D24). At these electrode locations, the LPP signal was larger for incongruent (negative words) compared to congruent (positive words) trials alike.

In a second step, we ran repeated measures ANOVAs on the mean amplitude of the ERP signal extracted during these two non-overlapping time

intervals and verified whether the processing of positive vs. negative words was reliably influenced earlier (i.e. during the EPN interval) following errors (FAs) compared to correct responses/Fast Hits, that primarily influenced the ERP signal during the interval, likely corresponding to the LPP component. The ANOVA performed on the ERP signal during the EPN interval with the within-subject factors electrodes (n = 12: B5-B16 corresponding to the right occipital cortex), action type (FA vs. Fast Hit) and word valence (negative vs. positive) revealed a significant interaction between action type and word valence, $F(1, 154) = 5.22$, $p < .05$ and showed a larger ERP (EPN) signal for incongruent compared to congruent trials. Moreover, this congruency effect was driven by smaller amplitudes for positive words following a FA compared to negative words, $F(1, 154) = 9.37$, $p < .01$, while no such differential effect was observed following Fast Hits, $F < 1$ (see Figure 6AB).

Interestingly, for the LPP component, we found a mirror-symmetric result. The repeated measures ANOVA run on the mean amplitude of the LPP signal with the within subjects factors electrodes (n = 7; C1-C7 corresponding to right frontal cortex), action type (FA vs. Fast Hit) and word valence (negative vs. positive) revealed a significant interaction between action type and word valence, $F(1, 84) = 6.34$, $p < .05$. This significant interaction translated generally larger LPP amplitudes for incongruent compared to congruent trials, $F(1, 84) = 6.34$, $p < .05$ but in contrast to the earlier evaluative priming effect found at the level of the EPN, this later LPP effect was driven by a significant differentiation between positive and negative words following fast hits, $F(1, 84) = 27.78$, $p < .001$, but not following FAs, $F < 1$ (see figure 6CD).

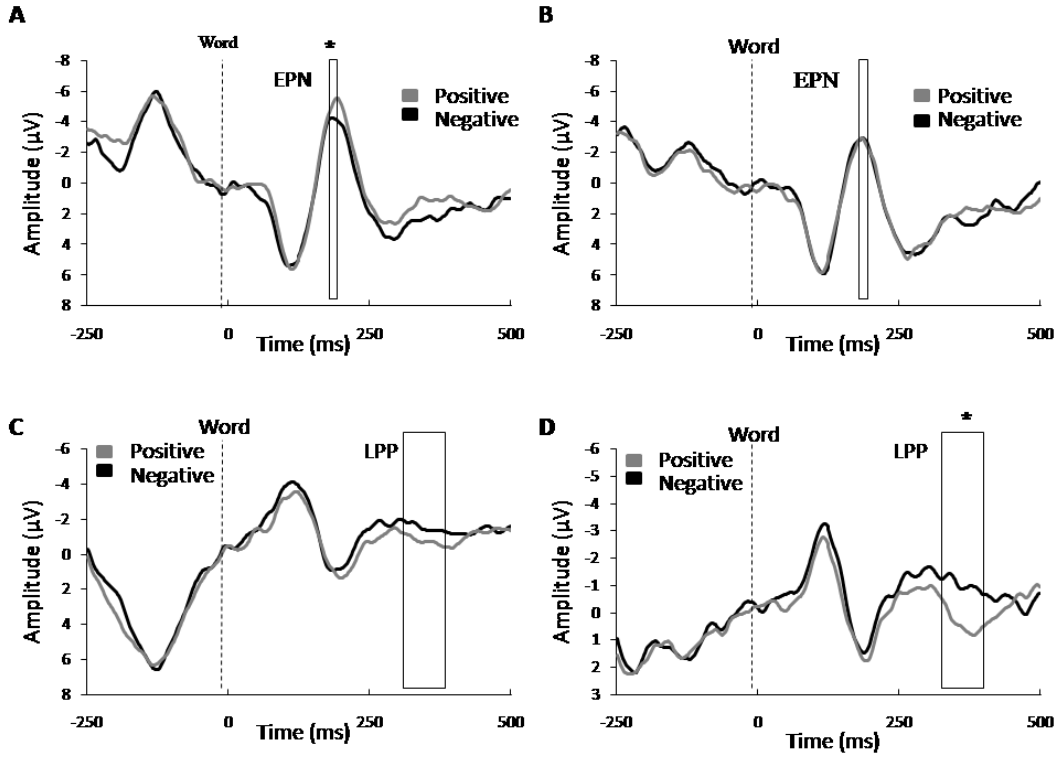

Figure 6. Main ERP results during the evaluative categorization task. (A) Grand average ERP waveforms (for a representative right occipital electrode - B6), separately for positive and negative words following FAs. The amplitude of the EPN was larger for positive compared to negative words. (B) No similar differential effect was seen (same electrode B6) for positive vs. negative words following Fast Hits. (C) Grand average ERP waveforms (for a representative right fronto-central electrode - C2), separately for positive and negative words following FAs. No LPP difference was seen between these two conditions. (D) By contrast, the amplitude of the LPP (same electrode C2) was enhanced for negative compared to positive words following Fast Hits. Asterisks indicate p <. 05.

DISCUSSION

The goal of the present ERP study was twofold. (i) To establish whether the magnitude of the ERN/Ne (error) – CRN (correct response) component generated "automatically" early on following action execution might actually be related to how quick participants could later discriminate positive vs. negative visual words, in line with an action-word evaluative priming effect (see Aarts et al., in revision). (ii) Furthermore, we aimed at clarifying the actual electrophysiological manifestations of this action/prime – word/target evaluative priming effect, by focusing on visual ERPs generated in response to these emotional words (and

more specifically the emotion-sensitive EPN and LPP ERP components). Our results show that the evaluative priming effect was related to the ERN/Ne-CRN difference. Because the ERN/Ne-CRN difference provides a reliable estimate of how well or strong participants differentiated "online" incorrect from correct actions during the Go/noGo task early on following response onset based on internal motor representations, this significant correlation with the evaluative priming effect suggests that these former monitoring brain processes are somehow related to the automatic processing of the affective or motivational value of self-generated actions. Given the compelling early electrophysiological time-course of these monitoring processes (ERN/Ne and CRN components), these "emotional" effects are likely to be automatic or preconscious (Nieuwenhuis et al., 2001). To the best of our knowledge, these ERP results provide the first direct electrophysiological evidence for the involvement of the ERN/Ne-CRN component in the processing of the affective values of self-generated actions.

Moreover, our new ERP results allowed us to clarify what are the actual electrophysiological correlates of this evaluative priming effect, here triggered by the rapid affective evaluation of self-generated actions. More specifically, we found that during an early time interval following emotional word onset (180-200 ms post-stimulus onset, corresponding to the early phase of the EPN component, as established using an independent "localizer" experiment), a significant ERP difference arose between positive vs. negative words following FAs over right occipital electrodes, indicated by a larger ERP signal for incongruent, compared to congruent action-word pairs in terms of "shared" affective content (positive words following FAs vs. negative words following FAs, respectively). During this early EPN time period, no differential valence effect between positive and negative words was found following Fast Hits. By contrast, such a valence-related ERP difference was well found for these correct responses during a later and non-overlapping time period, likely corresponding to the LPP component (320-390 ms post-stimulus onset). During this second time period, incongruent action-word pairs (negative words following Fast Hits) elicited a

larger ERP signal than congruent pairs (positive words following Fast Hits), following Fast Hits only, but not FAs. These ERP results suggest therefore that incongruent action-word pairs may be associated with an enhanced emotional or arousal reaction during the sensory processing of the emotional words, this effect being earlier for emotional words following FAs than Fast Hits.

The ERN/Ne-CRN is linked to the online automatic processing of the affective value of self-generated actions

Whereas previous studies already showed that unwanted response errors unlocked psycho physiological emotional reactions consistent with the detection and processing of aversive events (Hajcak & Foti, 2008), as well as differential brain responses in the amygdala (Pourtois et al., 2010), the evidence linking response errors to negative affect (or conversely correct responses to positive emotions) was primarily correlational in nature in these earlier studies. Moreover, the accumulating neurophysiological evidence linking enhanced ERN/Ne-CRN amplitudes to internalized psychopathological traits or characteristics, including anxiety and depression (Olvet & Hajcak, 2008; Vaidyanathan, Nelson, & Patrick, 2012), does not enable to draw strong conclusions regarding an altered emotional tagging of response errors in these anxious or depressed participants. In all these ERP studies, no significant change in behavior or emotional reactions following errors was seen or reported between high vs. low anxious, or between depressed vs. non-depressed patients. Accordingly, our new behavioral and ERP results are important because they show for the first time that self-generated actions performed during a standard Go/noGo task are rapidly appraised along a genuine affective dimension (FAs were evaluated as more negative compared to Fast Hits while conversely Fast Hits were automatically "tagged" as more positive than FAs; see behavioral results). This evaluative priming effect was related to inter-individual variations at the level of the magnitude of the response-locked ERN/Ne and CRN component, unambiguously linking this early action-monitoring ERP component to the automatic affective marking of actions (Aarts & Pourtois, 2010; Hajcak & Foti, 2008; Luu et al., 2000), presumably operating via specific meta cognitive

control systems working on the byproduct of an internal representation of motor actions, given the extremely rapid time-course and unfolding of these ERN/Ne-CRN brain effects in ACC (Fernandez-Duque et al., 2000; Winkielman et al., 2003). Our novel results show that across participants, the ones who showed a large difference between the ERN/Ne (errors) and CRN (correct responses) had a larger RT facilitation for processing the valence of the subsequent emotional words when it was actually "shared" with that of the actions (i.e. congruent vs. incongruent action-word pairs), compared to participants showing a more modest ERN/Ne-CRN differentiation.

Our new results may thus help interpret indirectly the functional meaning of these abnormal and enhanced ERN/Ne-CRN components typically seen in either high anxious or clinically depressed participants. These overactive early action-monitoring effects could reflect a selective impairment in extracting "online" the normal emotional/affective value of self-generated actions in these participants, a somewhat deleterious generic action-monitoring deficit that could potentially cause (i) a reduced affective priming effect at the behavioral level (see Aarts et al., in revision), as well as (ii) specific problems in integrating online the affective value of self-generated actions with the valence of external evaluative feedback stimuli shown after these specific actions (Aarts & Pourtois, 2012). Future studies are needed to establish what factors may cause the onset and maintenance of these early overactive action-monitoring effects at the level of the ERN/Ne and CRN, because they seem to underlie selective problems related to the rapid/automatic decoding of the emotional values of self-generated actions. Dysfunctional primary reinforcement learning signals generated in the basal ganglia and swiftly guiding action-monitoring processes at the cortical level within the dACC could potentially account for these early abnormal affective reactions following action execution (Cavanagh, Figueroa, Cohen, & Frank, 2011; Frank et al., 2005; Holroyd & Coles, 2002).

Interestingly, our correlation analysis also showed that when using the mean ERN/Ne amplitude alone (instead of the ERN/Ne-CRN amplitude difference), inter-individual changes in the size of this error-related component

alone were almost significantly linked (despite a modest sample size, n=15) to the subsequent RT facilitation for negative compared to positive words following the onset of these adverse events ($r > .35$, $p = .07$). This trend suggests that the larger the ERN/Ne amplitude, the quicker negative words were discriminated from positive words, a finding in line with earlier psycho physiological results showing an enhanced startle responses following errors compared to correct responses during a flanker task (Hajcak & Foti, 2008). In this earlier study alike, inter-individual variations at the level of this automatic defensive response (Shi & Davis, 2001) were actually predicted by the magnitude of the ERN/Ne component.

Action valence influences early stages of emotional word processing

A second major finding of our ERP study concerns the actual electrophysiological time-course and manifestations of this action-word affective priming effect. Early on following word onset (180-200 ms post-stimulus onset; EPN effect), we found that positive words led to a larger EPN ERP signal than negative words, following FAs. No such modulation was seen after Fast Hits. Later on, 320-390 ms post-word onset (LPP effect) negative words led to a larger LPP ERP signal than positive words, following Fast Hits, but not following FAs. Importantly, these two specific time intervals (EPN and LPP) were identified and based on the results obtained from an independent localizer experiment that allowed to reveal these two time periods of interest during which the visual processing of emotional words (either positive or negative) reliably differed from neutral words in the same participants. The first effect likely corresponded to an EPN effect, whereas the later effect to a LPP effect. Our new ERP results therefore suggest that FAs led to an earlier influence during the sensory processing of the emotional value of the words, than did Fast Hits.

Since the occipital EPN component has mainly been related to a motivated attentional capture effect depending on arousal and possibly depending on direct feedback effects from deeper limbic structures (Sabatinelli, Bradley, Fitzsimmons, & Lang, 2005; Sabatinelli, Flaisch, Bradley, Fitzsimmons, & Lang, 2004); a larger EPN is typically found for more arousing compared to less

arousing pictures or words (Herbert et al., 2008; Kissler et al., 2007; Kissler et al., 2009; Schacht & Sommer, 2009a, 2009b). Accordingly, the results of the present study suggest that an incongruency between the valence of the word and the accuracy of the action (i.e. FA-positive word) led to an enhanced arousal reaction 180-200 ms post-word onset, relative to congruent FA-negative words pairs. Response errors are usually deviant events that "automatically" call for a change in the behavior and are accompanied by defensive emotional (Hajcak & Foti, 2008) or attentional orienting reactions (Notebaert et al., 2009), such that their potential influence on the subsequent emotion word processing could take place earlier than the concurrent and symmetrical priming effect triggered by Fast Hits/correct responses. Because Fast Hits and FAs had similar frequencies of occurrence during the Go/noGo task, this differential priming effect during emotional word processing between these two action types cannot easily be related to uncontrolled endogenous attentional factors. At any rate, this EPN effect manifested itself as an augmented arousal for positive words following FAs, compared to negative words following similar FAs. Hence, the present behavioral evaluative priming effect found at the behavioral level could actually result not so much from a sensory facilitation for negative emotional words that necessarily shared the same intrinsic valence than the preceding self-generated actions (i.e. response errors; see Grill-Spector, Henson, & Martin, 2006), but instead, from an interference effect created by the perceived mismatch for the association of positive words with earlier response errors.

On the other hand, the LPP component has generally been associated with top-down fronto-parietal (endogenous) attention selection mechanisms (H. T. Schupp et al., 2000) and was usually larger for high compared to low arousing stimuli alike (Olofsson, Nordin, Sequeira, & Polich, 2008; Schupp, Flaisch, Stockburger, & Junghöfer, 2006). Accordingly, the presentation of negative (compared to positive) words following Fast Hits might unlock an enhanced endogenous orienting reaction, given the perceived affective incongruence between the preceding action, and the current emotional valence of the (negative) word. Unlike the early (perhaps automatic) EPN effect found following

errors during emotional word processing, this later LPP effect following Fast Hits could likely translate an attention-dependent change in the perceived emotional arousal of the words. As a result, a differential processing of negative compared to positive words would occur during a later time interval following Fast Hits (LPP effect), than FAs (EPN effect).

In sum, our results show that self-generated actions (performed during a standard speeded Go/noGo task with simple, non-emotional visual symbolic cues) are automatically appraised along an affective dimension, such that unwanted response errors (FAs) facilitate the processing of negative words shown after these specific actions, whereas conversely, correct responses (Fast Hits) lead to RT facilitation for positive words. This behavioral effect unambiguously indicated that response errors are implicitly and automatically perceived as more negative events compared to correct responses. Our new ERP findings show that the earliest action-monitoring brain effect (ERN/Ne-CRN component generated for the responses performed during the Go/noGo task) predicted the subsequent RT facilitation during emotion word processing, suggesting that this former ERP deflection is linked to the motivational significance of self-generated actions. Finally, we also found that whereas response errors automatically influenced the early sensory processing of the subsequent emotional words (EPN effect), correct responses influenced the processing of these emotional words alike, but during a later and non-overlapping time interval (LPP effect).

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CHAPTER 7: GENERAL DISCUSSION

Whereas response errors have for a long time been considered in psychology primarily as interfering events reflecting attentional lapses or stochastic breakdowns in cognitive control (to be eventually removed from subsequent data analyses), the last two decades have witnessed a tremendous paradigm shift whereby the processing of response errors (and more generally performance monitoring) has now become a major and central theme in cognitive and affective neuroscience (O(Connell et al., 2007; Padilla, Wood, Hale, & Knight, 2006). Response errors are associated with specific reinforcement learning signals in the human brain (Frank, Woroch, & Curran, 2005; Holroyd & Coles, 2002), and they usually set free a cascade of affective and/or attention orienting effects (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Hajcak, McDonald, & Simons, 2003b; Notebaert et al., 2009). Response errors also often have a negative connotation for participants who inadvertently commit them, and accordingly they are seen as aversive events, even though this aspect has actually received much less attention in the literature. However, it seems to be especially the case in high anxious, dysphoric or depressed individuals who usually show a hypersensitivity (and hyperactivity in specific brain regions, including the dACC) towards these adverse events. The main goal of my doctoral research was to better characterize these links between error-monitoring brain functions and negative affect, using both standard behavioral measures, as well as concurrent scalp ERP measurements informing about the brain dynamic underlying this process.

In this dissertation, I used specific task settings enabling to unlock a large number of unwanted response errors in well-controlled laboratory conditions, within a short period of time, and in every participant, without inducing excessive frustration. These errors correspond to false alarms in the present case, or failures to inhibit a prepotent response tendency (Miyake et al., 2000). I primarily focused on well-defined electrophysiological markers of error detection

and performance monitoring, namely the ERN/Ne, CRN, Pe and FRN ERP components. Previous ERP studies have already shown that individuals with high negative affect and internalizing disorders are characterized by an overactive error-monitoring brain system, in particular when focusing on the ERN/Ne component (Olvet & Hajcak, 2008; Vaidyanathan, Nelson, & Patrick, 2012). However, what this early overactive error monitoring in negative affect exactly reflects, remains largely underspecified. Does it correspond to a more global executive control deficit, or rather, a differential emotional reaction when facing these specific negative events (Carter et al., 1998; Luu, Collins, & Tucker, 2000; Vidal, Hasbrouckq, Grapperon, & Bonnet, 2000)? These questions form the core of the experiment work reported in this doctoral dissertation.

To address these questions, I first investigated error-monitoring brain functions in (sub clinical) trait anxious participants (Chapter 2) or clinically depressed (major depression disorder – MDD) patients (Chapter 3), using standard ERP peak analyses, but also complementing topographic and source localization methods. Comparing the results of these two studies (Chapters 2 and 3) enabled me to demonstrate that sub clinical trait anxiety and MDD have actually dissociable effects on early error-monitoring brain processes. The results of these two studies (Chapters 2 and 3) show that error-monitoring deficits in MDD are not corresponding to a mere amplification (or reduction) of the neural alterations seen in high anxious individuals; they seem to be qualitatively different. Second, I compared effects of sub clinical trait anxiety on internal (Chapter 2) vs. external action-monitoring brain processes (Chapter 4). Although these two systems may share a common or generic fronto-striatal neural network involved in reinforcement learning (Frank et al., 2005; Holroyd & Coles, 2002; Ullsperger & von Cramon, 2006), the question arose whether detrimental effects of trait anxiety on early error monitoring based on internal motor representations (Chapter 2) were also visible when performance had primarily to be monitored based on external evaluative feedback (Chapter 4). The ERP results of the study carried out in Chapter 4 confirmed that effects of trait anxiety on action monitoring are not restricted to the swift monitoring of internal cues, but

also present during the processing of external evaluative feedback. Accordingly, ERP studies carried out in Chapters 2 to 4 informed about the electrophysiological time-course (and probable loci in the human brain) during which either (sub clinical) trait anxiety or depression influenced error-detection or performance-monitoring brain processes. However, these ERP studies alone did not immediately highlight what may eventually be changed in these anxious participants or depressed individuals during early error detection or performance monitoring, given that these electrophysiological effects were evidenced without obvious changes in the behavior (i.e. accuracy or speed with the speeded Go/noGo task) as a function of negative affect. To further address this question, I also devised a new experimental method (based on the priming phenomenon) suited to infer indirectly the actual affective value of self-generated actions, including response errors (Chapter 5). Results of this study confirmed the prediction that response errors were automatically marked as negative events (and this effect was blunted in high anxious participants, see Chapter 5). I therefore ran a last ERP study (Chapter 6) to gain insight into the electrophysiological markers of this automatic affective tagging of self-generated actions. Results of this ERP study showed that (i) the ERN/Ne-CRN component likely encodes not only the perceived accuracy but also concurrently the affective or motivational value of self-generated actions; and (ii) this affective marking of self-generated actions is different (at the ERP level) for response errors vs. correct responses. I now provide an outline of the main new experimental findings presented in each chapter, before discussing more thoroughly each of the three main research questions addressed in this dissertation.

In Chapter 2, I tested the prediction that early detection brain mechanisms (as reflected by the ERN/Ne component) are not simply overactive in high anxious individuals, as previously put forward in the literature (Olvet & Hajcak, 2008; Vaidyanathan et al., 2012), they are also undergoing qualitative and traceable changes compared to low anxious participants. Low vs. high anxious individuals performed a speeded Go/noGo task while high-density (128 channels) EEG was recorded concurrently. Trait anxiety did not influence the error making

behavior (i.e. the number and speed of unwanted false alarms made on noGo trials). However, the earliest electrophysiological marker of error detection (i.e. ERN/Ne) was not only larger in high compared to low anxious individuals, it was also associated with different and non-overlapping rostral/ventral ACC effects in high anxious individuals. By contrast, the subsequent Pe component was not influenced by levels of trait anxiety. These results therefore suggest an early differential emotional or motivational processing of response errors in anxiety, or at least the recruitment of non-overlapping ACC regions during early error detection.

Because anxiety and depression are overlapping and usually considered as comorbid negative affect disorders (Clark & Watson, 1991; Mineka, Watson, & Clark, 1998; Watson et al., 1995), I used a very similar method and task in **Chapter 3**, but explored early error-monitoring brain functions in clinically depressed patients, as opposed to sub clinical anxious individuals in Chapter 2. The same speeded Go/noGo task was administered to a sample of depressed patients vs. healthy controls, while EEG was recorded concurrently. Like it was already found for anxiety (Chapter 2), depression did not change the expression of the error making behavior in this task; it mainly slowed down RTs, regardless of the experimental condition. However, ERP results showed that during early error processing, the ERN/Ne amplitude was not numerically augmented in these depressed patients, although additional topographical and source localization analyses showed that they recruited additional dorsolateral prefrontal areas (besides the classical medial frontal and dorsal ACC regions) during this early time interval following error commission, compared to the controls. This result suggests the engagement of auxiliary cognitive control processes in depression early on following error detection, probably related to interfering ruminative thinking processes in these patients. More importantly, I found that the subsequent error-related Pe component was significantly smaller in depressed individuals, and this effect was mainly explained by an abnormal recruitment of ventral cingulate areas. Interestingly, this later electrophysiological effect during error monitoring was strongly related to the specific trait-related rumination

characteristic of these patients, suggesting that probably rumination, more than depression per se, may actually influence this specific stage of (conscious) error detection. Altogether, the new ERP results reported in Chapters 2 and 3 point to dissociable effects of anxiety vs. depression on the early detection and monitoring of response errors.

In **Chapter 4**, I evaluated whether trait anxiety could influence "external" performance-monitoring brain processes equally well as "internal" error-monitoring processes (see Chapter 2), in keeping with the assumption of a shared neural system in these two cases (Holroyd & Coles, 2002). Therefore, a modified version of the speeded Go/noGo task was used, in which salient feedback on task (speed) performance was presented after each and every action, and low vs. high trait anxious individuals were recruited in this ERP experiment. In contrast to what was found in Chapter 2 (internal monitoring processes), no clear effect of anxiety was seen at the level of the ERN/Ne-CRN or Pe component in this experiment (Chapter 4), but the FRN effect time-locked to the onset of the evaluative feedback (i.e. externally-oriented performance monitoring) was reliably reduced in high compared to low anxious participants, suggesting that trait anxiety may actually produce generic action-monitoring impairments, not restricted to the processing of internal (motor) representations (as reflected by the ERN/Ne component, see Chapter 2). Results reported in Chapter 4 confirmed that high anxious individuals experience difficulties to evaluate whether their actions are correct (fast) or not (slow), when uncertainty is high, and external evaluative feedback stimuli need therefore to be processed rapidly in order to eventually assist or guide performance monitoring.

In **Chapter 5**, I devised a new behavioral experiment based on a standard evaluative priming technique suited to decipher the putative affective value of self-generated actions, including response errors. The basic principle of the experiments reported in Chapter 5 was to verify whether response errors were "automatically" marked as more negative events, compared to correct responses. In this study (Chapter 5), every self-generated action made during the Go/noGo task was, after a specific delay (i.e. 300 ms in Experiments 1 and 2; 600

ms in Experiment 3; 1000 ms in Experiment 4) followed by the presentation of a visual evaluative word that had to be quickly categorized as either positive or negative (hence I used a dual task setting). In accordance with a classical evaluative priming effect (Fazio, Sanbonmatsu, Powell, & Kardes, 1986; Hermans, De Houwer, & Eelen, 2001), I observed that the putative valence of the self-generated actions reliably influenced the processing of the subsequent emotional words: Negative words were categorized faster following response errors than correct responses, with a symmetrical effect for positive words. This priming effect was dependent on the length of the SOA: only at short (300 or 600 ms), but not at long SOAs (1000 ms) did the self-generated actions prime the processing of the subsequent evaluative words, in accordance with the hallmark of an evaluative priming effect. These results confirm that response errors are "automatically" marked as more negative events compared to correct responses, probably via dedicated meta-cognitive control systems (Fernandez-Duque, Baird, & Posner, 2000; Winkielman, Schwarz, Fazendeiro, & Reber, 2003). Moreover and importantly, I found that this action-word priming effect was smaller in high compared to low anxious individuals, suggesting that trait anxiety may influence the early automatic decoding of the emotional or motivational value of self-generated actions, including response errors (see also results of Chapter 2).

In **Chapter 6**, I explored, using EEG methods, the actual electrophysiological correlates of the action-word evaluative priming effect found in the previous chapter. First, I found that across participants, the magnitude of the evaluative priming effect (i.e. RTs for incongruent action-word pairs vs. congruent pairs) could partly be predicted by the ERN/Ne-CRN amplitude difference time-locked to the onset of the action, confirming that this early action-monitoring ERP component is somehow involved in the affective processing of self-generated actions. Moreover, I found that response errors actually influenced early perceptual stages of emotion word processing, namely at the level of the early posterior negativity (EPN) ERP component (Herbert, Junghöfer, & Kissler, 2008; Schupp, Flaisch, Stockburger, & Junghöfer, 2006), during this action-word evaluative priming effect, while correct responses mainly

influenced a later stage of emotional word processing, namely at the level of the late positive potential (LPP) ERP component (Hajcak, MacNamara, & Olvet, 2010; Olofsson, Nordin, Sequeira, & Polich, 2008; Schupp et al., 2000). These ERP results provide thus important information regarding the electrophysiological time-course of this action-word evaluative priming effect. They show a temporal precedence of response errors compared to correct responses during this priming effect, which may tentatively be explained by the enhanced motivational or affective value of these (deviant) aversive events.

I now turn to the discussion of the three main research questions addressed in this dissertation and by doing so, I provide an integration of the results obtained in the five different chapters into a broader theoretical context. First, the dissociable effects of trait anxiety vs. depression on the ERN/Ne vs. Pe ERP component are discussed. Second, the differential effects of trait anxiety on "internal" vs. "external" action-monitoring processes are carefully reviewed. Finally, I discuss more thoroughly the notion of an automatic affective tagging of self-generated actions, and to which extent this process may be impaired selectively in anxiety or depression, and backed up by amplitude changes at the level of the ERN/Ne-CRN ERP component. The discussion ends with possible implications and relevance of the present ERP and behavioral results for the classification of internalizing disorders in psychopathology (with a focus on anxiety and depression), followed by the presentation of two possible future research perspectives.

ERN/NE-ANXIETY VS. PE-DEPRESSION?

The results of the studies reported in Chapters 2 and 3 show that neither sub clinical trait anxiety (Chapter 2) nor depression (Chapter 3) simply disrupts error detection or adaptation effects at the behavioral level, at least with the specific Go/noGo task used in these studies and which promotes a fast and impulsive response mode. The number of response errors was similar between high vs. low anxious individuals (Chapter 2) or between healthy controls vs. clinically depressed patients (Chapter 3). RTs of high anxious individuals were as

fast as controls while depressed patients were overall slower compared to healthy controls, but importantly, this effect was general and not condition specific. These observations were important because (1) they confirm that negative affect (either trait anxiety or depression) does not simply alter executive functions or performance monitoring in general when investigated using a simple speeded Go/noGo task (Eysenck, Derakshan, Santos, & Calvo, 2007), and moreover (2) they allow to exclude the possibility that unbalanced behavioral performance between groups could account for the observed ERP differences during early error-monitoring processes. Because the signal to noise ratio of error-related ERP components, including the ERN/Ne and Pe, strongly depends on the actual number of trials included in the averages (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Olvet & Hajcak, 2009b), the reported ERP differences between high anxious or depressed vs. healthy controls could not be ascribed to asymmetries in the resulting error-related ERP signal across groups.

Interestingly, a direct comparison of the ERP results obtained in Chapters 2 and 3 points to notable dissociable effects of trait anxiety vs. depression on the error-monitoring brain machinery. Whereas trait anxiety mainly influenced the earliest stage of error monitoring (i.e. ERN/Ne-CRN component) but left unchanged the subsequent Pe component (Chapter 2), I found a symmetric outcome for depression (Chapter 3). In the former case, a larger ERN/Ne (relative to the CRN) was found in high anxious participants, and this early effect was associated with the activation of rostral ACC regions, while in the latter case a blunted Pe component was found, with a corresponding decrease of ventral ACC activations. These results therefore confirm that trait anxiety and depression have different (remote) effects on early error-monitoring brain processes. These findings also lend support to the notion that these two early error-related ERP components (ERN/Ne vs. Pe) likely reflect different stages of processing during early error monitoring (Nieuwenhuis, Ridderinkhof, Blow, Band, & Kok, 2001; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; Ridderinkhof, Ramautar, & Wijnen, 2009). Previous ERP studies already showed a functional dissociation between the ERN/Ne and the Pe component, either based on specific

experimental manipulations (e.g. error awareness; Dhar, Wiersema, & Pourtois, 2011; Endrass, Franke, & Kathmann, 2005; Klein et al., 2007; Nieuwenhuis et al., 2001; O'Connell et al., 2007), or based on pharmacological manipulations (e.g. dopaminergic drugs; De Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004). The results of these earlier studies were compatible with the assumption that the ERN/Ne reflects a rapid, perhaps automatic (in the sense of unconscious, see Moors & De Houwer, 2006) stage of error detection, which is dependent upon fronto-striatal dopaminergic brain systems (Chase, Swainson, Durham, & Benham, 2011; Frank et al., 2005; Holroyd & Coles, 2002). By contrast, the subsequent error-related Pe component could translate a more elaborate process during error detection, maybe related to the accumulation of evidence about error commission (Steinhauser & Yeung, 2010), or error awareness (Dhar et al., 2011; Endrass et al., 2005; Nieuwenhuis et al., 2001; O'Connell et al., 2007){ #45; #511; #266; #450}, or the (conscious) detection of a salient event (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). These latter processes would be less dependent upon direct dopaminergic inputs, although few ERP studies have actually explored possible links between this specific neurotransmitter (or other ones, including serotonin or maybe norepinephrine; see Nieuwenhuis, Aston-Jones, & Cohen, 2005) and amplitude changes at the level of the Pe component. The results reported in Chapters 2 and 3 show that different forms of negative affect (trait anxiety vs. depression) may have different influences on these early error-monitoring brain processes. Previous ERP studies already reported dissociations between the ERN/Ne and Pe component in patients with obsessive compulsive disorders (Ruchsow et al., 2005), in schizophrenic patients with and without psychosis (Bates, Liddle, Kiehl, & Ngan, 2004), or in individuals with high vs. low negative affect (Hajcak, McDonald, & Simons, 2004).

An increased ERN/Ne accompanied by the recruitment of rostral ACC regions in high anxious individuals (Chapter 2) was interpreted as reflecting either a higher motivational significance of response errors in these participants (Hajcak & Foti, 2008; Hajcak et al., 2003b; Luu et al., 2000) or alternatively, a

change in effort or attentional control (Eysenck et al., 2007). In this latter framework, anxious individuals are characterized by excessive worrisome thoughts concerning negative events that might happen in the future, and this narrowing down of the action-thought repertoires has a deleterious effect on executive functions and performance monitoring. However, the attentional control theory posits that high anxious individuals somehow compensate for these problems by increasing efforts or the amount of resources, which leads to a drop in efficiency when task demands or complexity increases. Although still speculative at this stage, I interpreted an increased ERN/Ne component in high anxious, associated with rostral ACC activations (Chapter 2), along these lines. This specific interpretation (i.e. the overactive ERN/Ne in high trait anxious individuals resulting from a rostral ACC activation would somehow translate a compensatory mechanism in these individuals) is also indirectly supported by earlier findings in the literature linking effort to an increased ERN/Ne (Luu et al., 2000; Tops, Boksem, Wester, Lorist, & Meijman, 2006), as well as to stronger ACC activity (Paus, Koski, Caramanos, & Westbury, 1998). However, because I did not manipulate or measure directly "efforts" (or efficiency, for example by comparing the processing of response errors made during a simple vs. dual-task setting) in Chapter 2, additional studies are needed to corroborate the assumption that an increased ERN/Ne in trait anxiety could reflect a change in "efforts" and/or efficiency. Using the same framework, one could thus conclude that the absence of an increased ERN/Ne in depressed individuals (Chapter 3) indicate that they are probably not using (online) a similar compensatory mechanism to deal with their response errors, early on following their onset. In contrast, because of their distinctive ruminative thinking style, the Pe component is substantially reduced in depressed patients. Because the Pe component likely reflects other processes involved in error-awareness per se (Nieuwenhuis et al., 2001), or attention orienting towards motivationally significant events (Davies, Segalowitz, Dywan, & Pailing, 2001; Ridderinkhof et al., 2009), or the accumulation of evidence for error commission (Steinhauser & Yeung, 2010), it appears that the omnipresent ruminative thoughts in depressed

patients may consume (attentional) resources away from the efficient (conscious) monitoring and registration of response errors.

In sum, anxiety-related alterations at the level of the ERN/Ne might be accounted for by changes in attention control mechanisms, whereas depression-related alterations at the level of the Pe would result from the intrusion of rumination. It is interesting to note that in the former case, the enhanced ERN/Ne could very well arise due to a proactive compensatory mechanism used by high anxious individuals to deal with these somehow adverse or unexpected events challenging self-efficacy, while in the latter case the ruminative thinking style of depressed patients somehow block or interfere with reactive monitoring processes during an efficient and rapid (conscious) registration of response errors (Pe effect). I come back to these considerations and conjectures in the section 5.1 below.

ANXIETY-RELATED CHANGES OF INTERNAL (ERN/NE) VS. EXTERNAL (FRN) DRIVEN PERFORMANCE MONITORING

Whereas in Chapter 2, I found a clear modulation of the ERN/Ne-CRN component as a function of trait anxiety, this effect was no longer seen in Chapter 4, where the same Go/noGo task was used however, but salient performance feedback, based on static emotional faces, was now presented to low. vs. high anxious participants. This apparent discrepancy might also be due to the fact that the average level of trait anxiety was actually lower in the high anxious group selected in Chapter 4 (STAI-T: *M* = 45; *SEM* = 1), compared to the high anxious group included in Chapter 2 (STAI-T: *M* = 52; *SEM* = 1). Given that previous ERP studies reported larger ERN/Ne amplitudes with higher levels of trait anxiety (Hajcak, McDonald, & Simons, 2003a; Olvet & Hajcak, 2008; Vaidyanathan et al., 2012; Weinberg, Olvet, & Hajcak, 2010), this factor might have played a significant role. However, we note that it is also extremely difficult to balance properly trait anxiety levels of samples of student participants across different studies. Alternatively, the use of salient (emotional facial) feedback after each and every action made during the Go/noGo task (Chapter 4) may

modulate the link between the ERN/Ne-CRN component and trait anxiety, as demonstrated earlier (Olvet & Hajcak, 2009a). Interestingly, when action monitoring is substantially helped out or assisted by the presentation of external evaluative feedback (informing about the accuracy or speed of self-generated actions), the overactive ERN/Ne component in trait anxiety is usually no longer visible (Olvet & Hajcak, 2009), suggesting that effects of trait anxiety on performance monitoring are not rigid, but they are instead context dependent. Accordingly, in (experimental) situations where evaluative feedback is salient and informative regarding task performance, participants might swiftly shift their action-monitoring processes from the use of internal (motor) to external (visual) cues. As a result, the overactive ERN/Ne in trait anxiety would somehow be corrected, and become comparable to the ERN/Ne of low anxious individuals.

More generally, these observations and the new ERP results reported in Chapters 2 and 4 somehow challenge the notion that the ERN/Ne provides a reliable endophenotype of internalizing disorders, including anxiety (Olvet & Hajcak, 2008). In this model, amplitude variations at the level of the ERN/Ne-CRN component are seen as a latent variable between a specific genetic makeup or biological predisposition, and a clear cut symptomatology (or phenotype) characterized by worry and distress (Gottesman & Gould, 2003). The results showing that low and high anxious participants show comparable ERN/Ne and CRN components during early error/action monitoring when external evaluative feedback was presented concurrently (Chapter 4) are not consistent with this general framework. Yet, they also show that the processing of this external evaluative feedback on task performance was altered in high, compared to low anxious individuals (Chapter 4). Whereas both groups showed a comparable early emotional effect at the level of the occipito-temporal N170 ERP component during feedback processing (this early face-specific component being larger for neutral faces indicating negative compared to positive feedback, see Bentin, Allison, Puce, Perez, & McCarthy, 1996; Vuilleumier & Pourtois, 2007), anxiety influenced selectively the subsequent medial frontal FRN component, which is involved in performance-monitoring processes (Hajcak, Moser, Holroyd, &

Simons, 2006; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Miltner, Braun, & Coles, 1997) . High anxious participants did not exhibit a normal FRN amplitude variation compared to low anxious individuals depending on the valence of the feedback. These results suggest that high anxious participants were still able to decipher properly the "extrinsic" emotional value of the feedback (N170 effect), but they could not relate it to the putative affective value of their action (FRN effect), even though it was made several hundred milliseconds before feedback delivery.

This selective FRN impairment in trait anxiety (Chapter 4) during evaluative feedback processing is noteworthy, given the tight overlap between (dopaminergic-dependent) fronto-striatal brain systems underlying the generation of the ERN/Ne and FRN components (Frank et al., 2005; Holroyd & Coles, 2002). These two ERP components are assumed to reflect similar or generic monitoring processes, either based on internal (motor) cues (ERN/Ne) or external (visual) cues (FRN). Therefore, the novel ERP results reported in Chapters 2 and 4 provide evidence for this specific interpretation. Note however that in one case, an overactive ERP component (ERN/Ne, Chapter 2) is observed during action monitoring based on internal cues, whereas in the other case, a blunted ERP activity is reported when the monitoring is driven by external evaluative cues (FRN, Chapter 4). However, in both cases, one may assume a common or generic action-monitoring deficit in high anxious participants (Holroyd & Coles, 2002). A blunted FRN component in high anxious participants could be interpreted as reflecting a breakdown in the swift integration process linking the valence of the evaluative feedback to the affective (or prediction error) value of the self-generated action, prior to feedback delivery (Chapter 4). This assumption was indirectly verified using a complementary correlation analysis across participants between amplitude variations at the level of the FRN component and a trait-like measure of attribution style and/or locus of control (Rotter, 1966). Results showed that participants who had an internal (as opposed to external) locus of control (i.e. meaning that they usually believe that changes in the environment are causally related or explained by their behavior or actions)

had a larger FRN component differentiating between positive and negative feedback (Chapter 4). However, this significant correlation was only found for low, but not high anxious participants, suggesting indirectly that external performance monitoring is disrupted in these individuals because they are no longer able to relate the content or value of their self-generated actions to external performance feedback shown after them and normally readily exploited by participants to gauge online the outcome of their behavior (FRN effect). Hence, trait anxiety appears to exert pervasive effects on action-monitoring brain systems. Although the actual expression of these anxiety-related impairments could vary depending on specific contextual or situational factors (see Chapters 2 and 4), the basic assumption is that they likely result from a generic deficit in generative brain mechanisms underlying performance-monitoring abilities, and likely encompassing (dopaminergic-dependent) fronto-striatal loops in the human brain (Frank et al., 2005; Holroyd & Coles, 2002), although the link between these specific brain systems and these error-related ERP components recorded at the scalp level remains by definition indirect.

SELF-GENERATED ACTIONS "AUTOMATICALLY" ACQUIRE AN AFFECTIVE COLOR

As outlined here above and in the introduction of this dissertation, error detection, and performance monitoring more generally, cannot be assimilated to a dry cognitive process encapsulated in the dACC (see also Figure 3 in the introduction). This process is rapidly and dynamically modulated or guided by phasic changes in levels of midbrain dopamine in order to eventually facilitate learning, as stated by the dominant reinforcement learning theory (Frank et al., 2005; Holroyd & Coles, 2002). Moreover, we already know that the processing of these motivational events (response errors or negative feedback) is also related to specific defensive emotional reactions (Hajcak & Foti, 2008), and differential amygdala effects (Polli et al., 2009; Pourtois et al., 2010). Finally, we have already reviewed abundant neurophysiological evidence linking overactive early error-related components with negative affect (Hajcak et al., 2003a, 2004; Vaidyanathan et al., 2012). The ERP results reported in Chapters 2-4 are broadly

consistent with this framework. Altogether, the present ERP results and the existing literature suggest that response errors are not only quickly evaluated as incorrect actions by dedicated brain systems, but they are also usually appraised in parallel as goal obstructive events, and in turn they necessarily bear a negative valence. However, all the results and findings reviewed so far remain somehow correlational in nature. Accordingly, in the last part of my doctoral dissertation (Chapters 5 and 6), I addressed this specific question and worked on a new method suited to infer indirectly the actual affective values of self-generated actions, including response errors. Such a "methodological" and theoretical development appears necessary to more directly relate and understand changes in early error-monitoring brain functions seen in negative affect (see Chapters 2 to 4), with possible alterations in this fundamental appraisal process.

Results obtained in Chapter 5 provide direct evidence for the rapid and automatic evaluation of self-generated actions. Errors are more negative than correct responses (i.e. fast hits during the Go/noGo task), while conversely, correct responses were treated as more positive than response errors (i.e. false alarms during the same Go/noGo task). This was the case when the interval (SOA) between the action (prime) and the word (target) was set to 300 or 600 ms, but not 1000 ms, suggesting an automatic tagging of these self-generated actions, likely via dedicated meta-cognitive control systems (Fernandez-Duque et al., 2000; Winkielman et al., 2003). How exactly does this affective marking of actions operate, and which brain regions or neural networks are involved, remain important questions for future research (see also Chapter 6). Moreover and importantly, this action-word priming effect was weaker in participants scoring high on a standard trait-related anxiety questionnaire. Remarkably, I also found in Chapter 6 that this effect was related to the size of the ERN/Ne-CRN component. Accordingly, the observation of an altered ERN/Ne (or FRN) component during performance monitoring in negative affect (see Chapters 2 to 4) could actually be explained by selective problems in ascribing a given affective value (either positive or negative) to self-generated actions (either correct or not). Therefore, I surmise that performance-monitoring deficits typically

observed in participants scoring high on negative affect scales could very well result from a specific impairment in deciphering online the actual affective value of their actions (positive vs. negative; good or bad; see Cacioppo & Gardner, 1999), more than a problem in decoding rapidly the perceived accuracy of these actions (correct vs. incorrect). This framework appears relevant and valid at first sight to account for a wide range of behavioral or ERP effects seen in high anxious individuals during error detection and/or action monitoring. However, future studies are needed to gain insight into the genesis and maintenance of this peculiar performance-monitoring deficit in high trait anxious individuals (or depressed patients).

Results of Chapter 6 shed light on the electrophysiological time-course of this action-word evaluative priming effect. This effect was found to influence earlier stages of processing after word presentation for response errors, compared to correct responses (Chapter 6). The EPN component (Herbert et al., 2008; Schupp et al., 2006) was larger for positive words than negative words following response errors. Symmetrically, the LPP component (Schupp et al., 2000) was larger for negative words than positive words following correct responses. This differential effect of action valence (or action type) on the electrophysiological time course of emotional word processing might be explained by the enhanced salience or behavioral relevance of response errors compared to correct responses (Pourtois et al., 2010; Sander, Grafman, & Zalla, 2003). Accordingly, response errors influenced early perceptual stages during emotion word processing (EPN effect), while correct responses did so too, but during a later time interval when other processes likely related to attention control or motivated attention mechanisms likely came into play (LPP effect). At any rate, these preliminary ERP results (Chapter 6) confirm the differential processing of response errors compared to correct responses, including regarding the putative affective coloring of these two different action types.

POSSIBLE CLINICAL RELEVANCE OF THE PRESENT FINDINGS

To better understand action monitoring in relation to negative affect from an affective neuroscience perspective appears valuable from a clinical point of view, in particular when considering the numerous attempts made in the literature to classify or organize mental disorders using specific taxonomies, while taking into account not only the phenotype, but also the underlying neurobiological or neurophysiological markers best characterizing specific mental disorders (Vaidyanathan et al., 2012). Classically, mental disorders are identified in psychopathology or psychiatry based on self-report measures and/or reliable and observable changes in the behavior, broadly defined (e.g. Diagnostic and Statistical Manual of Mental Disorders – DSM; American Psychiatric Association - DSM-IV, 2000). However, recently, the National Institute of Mental Health launched the Research Domain Criteria (RDoC) initiative, with the aim to characterize psychiatric disorders in terms of their neurobiological underpinnings (Insel & Cuthbert, 2009) by using and crossing experimental and neuroimaging methods. A similar attempt is provided by the "converging biomarker" approach, which is not focusing on specific neural circuits (and potential alterations), but on specific neurophysiological indicators, including specific ERP components, in order to clarify possible sources of heterogeneity or inconsistency within and across specific disorders or interrelated sets of disorders (Gilmore, Malone, & Iacono, 2010; Nelson, Patrick, & Bernat, 2011). With respect to anxiety and depression, this approach allows for example to identify and validate shared factors among these two disorders (e.g. negative affect), as well as isolate possible separate characteristics (e.g. arousal in anxiety vs. low positive affect in depression) as defined by Clark and Watson (1991), and eventually relate these common or distinct factors to specific neurobiological markers. Applied to our new neurophysiological findings, we would suggest that a large ERN/Ne (see Chapter 2) or a small FRN component (see Chapter 4) could provide reliable biomarkers of trait anxiety, while a blunted Pe component (see Chapter 3) would possibly characterize depression. We believe that such a modern approach, capitalizing on the cross-fertilization of

psychiatry/experimental psychopathology and brain-imaging/neurophysiology (e.g. ERP components), might provide a valuable alternative or tool from a clinical point of view, compared to other attempts currently put forward in the field, including the hypothesis stating that the ERN/Ne component alone provides a reliable endophenotype for internalizing disorders (Olvet & Hajcak, 2008).

As a caveat, we note however that these specific "neurobiological" markers (i.e. a large ERN/Ne and/or a smaller FRN component in high trait anxious individuals vs. a blunted Pe component in depressed patients) can probably not easily be generalized to other anxiety disorders without special attention (and careful validation work), given that although obsessive-compulsive disorder (OCD), post-traumatic stress disorder (PTSD), generalized anxiety disorder (GAD) or specific phobia (SP) may share some common basic features and alterations of specific brain regions, current neurobiological models of these disorders largely emphasize the lack of overlap in the underlying neural networks across them (Etkin & Wager, 2007; Shin & Liberzon, 2010). A potential way to address this problem would be to better constrain the neurobiological data and models available in the affective neuroscience literature with information gathered in neurophysiology (e.g. ERP) about the commonalities vs. differences in performance-monitoring abilities/impairments across these different negative affect disorders. Previous error-monitoring ERP studies already showed that an increased ERN/Ne component might actually be particularly related to the worry or apprehension component of anxiety, such as observed in OCD (Endrass et al., 2010) or GAD (Weinberg et al., 2010), but much less to the concurrent arousal component, such as in specific phobia (Moser, Hajcak, & Simons, 2005). Crossing results from neurophysiology, imaging and psychiatry might help delineate the boundaries between these non-overlapping negative affect disorders, and eventually guide their diagnosis and treatment in clinical practice.

FUTURE DIRECTIONS

ERN/Ne vs. Pe: Proactive vs. reactive control?

According to recent models of cognitive control (Braver, 2012), this fundamental ability is not a unitary construct, but it consists of proactive and reactive control mechanisms. In this view, proactive control is seen as a form of "early selection" through which a sustained and anticipatory maintenance of goal-relevant information allows for optimal cognitive performance. This function is related to dopaminergic inputs. By contrast, reactive control acts as a "late correction" mechanism that is mainly activated when needed, such as after the detection of an interfering event (e.g. response error or negative feedback). Unlike proactive control, reactive control does not depend on dopaminergic inputs. This general framework appears valuable to interpret some of our novel ERP findings, as well as generate specific predictions for future experiments or research.

I mainly interpreted the altered ERN/Ne component in anxious individuals (Chapter 2) as reflecting possibly a compensatory "attention control" mechanism (Eysenck et al., 2007), namely an increase in the amount of efforts or resources spent in the task in order to prevent in the future the re-occurrence of these negative events associated with worrisome thoughts, and eventually maintain a high accuracy. As such, this mechanism resembles a proactive control mechanism, which would therefore be exacerbated in high anxious individuals. Moreover, because the ERN/Ne (and FRN) component has previously been linked to specific dopaminergic-related reinforcement learning changes in the human brain (Frank et al., 2005; Holroyd & Coles, 2002), this early ERP component likely qualifies as a good candidate or ERP marker of proactive control (see Braver, 2012).

By contrast, the blunted Pe component seen in depressed patients (Chapter 3) during early error monitoring and likely indexing a decreased (conscious) action-monitoring control because of excessive interfering ruminative thoughts, could indirectly be linked to an impaired reactive control

mechanism during error monitoring in these patients. In one case (anxiety), the disorder would result from an overactive proactive control mechanism (ERN/Ne component) without changes at the level of the reactive control systems (Pe component). By contrast, in the other case (depression), a deleterious reactive control mechanism would explain the observed error monitoring deficits. Therefore, we propose that the dissociable effects of anxiety vs. depression on early error-related ERP components (ERN/Ne vs. Pe component) might actually arise thanks to selective alterations in proactive vs. reactive cognitive control mechanisms.

To put to the test this assumption, future studies could for example manipulate the valence or nature of the feedback on task performance during the same speeded Go/noGo task (see also Chapter 4), given that penalty incentives have been suggested to produce a shift towards reactive control, while the encounter of reward incentives produced a shift towards proactive control (Braver, Paxton, Locke, & Barch, 2009; Locke & Braver, 2008). Accordingly, it might be interesting to explore in future ERP studies changes in early error-related brain activities (ERN/Ne and Pe component) when correct responses are rewarded, or alternatively response errors are punished, producing an increase in proactive or reactive cognitive control, respectively. According to this dual mechanisms framework (Braver, 2012), the first manipulation should normally lead to a larger ERN/Ne component in high anxious individuals (Chapter 2), whereas the latter condition might actually restore a normal Pe component in depressed patients (Chapter 3).

The affective value of self-generated actions and the locus of control

A specific feature of high anxious individuals is that they usually show an increased tendency to attribute the causes of their actions to external factors or forces (i.e. external locus of control; Archer, 1979). These “externalizing” individuals are thus prone to believe that the outcome of their action is contingent upon events happening outside their personal control or volition. By contrast, “internalizing” participants usually believe that the outcome of their action is causally related to their behavior or “intrinsic” forces (Zimbardo, 1985).

The perceived link between self-generated actions and their outcome seems thus to be weaker in distressed individuals, and therefore it might result in a decreased capacity to tag or read out, using dedicated internal monitoring systems, specific affective or motivational values associated with these actions. The novel ERP results (Chapter 4) and behavioral findings (Chapter 5) reported in this doctoral dissertation provide indirect evidence for this account. However, additional studies are needed to verify whether the differential attribution style, or "locus of control" per se, in high anxious individuals can account for the observed changes at the electrophysiological level (e.g. ERN/Ne and FRN components; see Chapters 2 and 4), as well as the observed reduction of the action-word affective priming effect (Chapter 5) during error detection or performance monitoring.

This could be achieved for example by directly manipulating the perceived locus of control or overall perceived controllability or competence in low vs. high anxious individuals, and assess how these manipulations could eventually influence early error-monitoring brain functions (ERN/Ne and Pe effects; see also Inzlicht & Al-Khindi, in press), as well as feedback processing (FRN effect). In this context, the manipulation in well-controlled laboratory conditions of the content and/or veracity of the feedback given on task performance may provide an interesting avenue for future research (see also Chapter 4). A decreased controllability and a shift towards external evaluative cues may be obtained when feedback given on task performance become somehow unpredictable (see also Chapter 4). Such an effect should normally yield a shift from internal (motor-related) to external (visually-driven) action-monitoring processes, with traceable effects at the ERN/Ne and FRN levels (Eppinger, Kray, Mock, & Mecklinger, 2008; Frank et al., 2005). An opposite or symmetric manipulation could eventually be tested, whereby the perceived controllability and the use of internal monitoring cues could artificially be reinforced throughout the experimental session, for example by omitting now and then to present feedback on task performance. It would be particularly interesting to explore if and how trait anxiety or depression may reliably influence the unfolding of these action-monitoring processes.

NEDERLANDSTALIGE SAMENVATTING

INLEIDING

Een accurate en snelle detectie van fouten is noodzakelijk om ons gedrag aan te passen (Holroyd& Coles, 2002). Onderzoek heeft echter aangetoond dat fouten of incorrecte acties niet enkel dienen als motivationele signalen die het leren bevorderen, maar verschillende onderzoekers hebben ook aangetoond dat de detectie van een fout gepaard gaat met emotionele veranderingen zoals defensieve reacties, die bijvoorbeeld tot uiting komen in een sterkere "startle response" (Hajcak & Foti, 2008; Pourtois et al., 2010), en een verhoogde waakzaamheid, orienting of aandacht (Notebaert et al., 2009; Ridderinkhof, Ramautar, & Wijnen, 2009). Deze bevindingen worden ook ondersteund door studies in de affectieve neurowetenschappen die laten zien dat mensen die gevoelig zijn voor negatief affect, zoals angstige of depressieve mensen, over een overactief of hypersensitief foutenmonitoring systeem in de hersenen beschikken (Olvet & Hajcak, 2008; Vaidyanathan, Nelson, & Patrick, 2012). Wat dit hyperactief systeem in deze groepen met negatief affect betekent, is echter niet helemaal duidelijk. Heeft dit te maken met problemen in de executieve controle, of duidt dit op een verschillende motivationele/affectieve reactie t.o.v. fouten?

Fouten worden al zeer vroeg (in de orde van milliseconden) in de hersenen gedetecteerd m.b.v. de excellente temporeel sensitieve techniek "het elektro-encefalogram" (EEG). Wanneer het EEG wordt geregistreerd tijdens het maken van een fout en vervolgens gemiddeld worden over het totale aantal fouten dat gemaakt wordt tijdens een taak, ontstaat er in het EEG een 'event-related potential' die de cognitieve informatieverwerking tijdens een fout reflecteert. Onderzoekers hebben in deze foutengerelateerde potentiaal een zeer vroege, binnen de 100 ms, en scherpe negativiteit geobserveerd die geregistreerd wordt op frontocentrale elektroden (Fcz) en gegenereerd wordt door het dorsale

gedeelte van de anterieure cingulate cortex (ACC), en hebben dit de “error-related negativity” of fouten gerelateerde negativiteit (ERN/Ne) genoemd (Falkenstein, Hoormann, Christ, &Hohnsbein, 2000; Frank, Woroch, &Curran, 2005; Holroyd & Coles, 2002). Deze component wordt geacht de vroege, generieke en automatische reactie op fouten te weerspiegelen en wordt meer specifiek verondersteld de mismatch weer te geven tussen een gewenste of intentionele actie en de eigenlijke actie (Holroyd & Coles, 2002). Een gelijkaardige maar kleinere negativiteit wordt soms ook geobserveerd na correcte acties en wordt de correct gerelateerde negativiteit (CRN) genoemd. Sommige onderzoekers veronderstellen dat de ERN/Ne en de CRN dezelfde processen reflecteren (Falkenstein et al., 2000; Roger, Bénar, Vidal, Hasbroucq, & Burle, 2010; Vidal, Burle, Bonnet, Grapperon, &Hasbroucq, 2003). Deze ERN/Ne component wordt gevolgd door een grote positieve component, de “error positivity” (Pe), die het maximaal is 150-300 ms na het maken van een fout en wordt gegenereerd door meer rostrale ACC gebieden alsook door de posterieure cingulate cortex en de insula (Dhar, Wiersema, &Pourtois, 2011; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein et al., 2000; Herrmann, Rommler, Ehlis, Heidrich, & Fallgatter, 2004; Nieuwenhuis, Ridderinkhof, Blow, Band, & Kok, 2001; O'Connell et al., 2007; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). In tegenstelling tot de functionele significantie van de ERN/Ne, wordt verondersteld dat de Pe meer gecontroleerde of bewuste foutenverwerkingsprocessen weerspiegelt. De ERN/Ne, CRN en Pe zijn dus ERP componenten die gerelateerd zijn aan de interne detectie van fouten. Een component die echter gerelateerd is aan de externe detectie van fouten is de “feedback gerelateerde negativiteit” (FRN). Deze component is zeer gelijkaardig aan de ERN/Ne op elektrofysiologisch gebied en er wordt verondersteld dat deze component processen weerspiegelt die gelijkaardig zijn aan deze die gereflecteerd worden in de ERN/Ne component. De FRN is gewoonlijk groter voor negatieve vergeleken met positieve feedback en ook groter voor onverwachte vergeleken met verwachte feedback.

De foutengerelateerde processen in de hersenen blijken gevoelig te zijn voor veranderingen in negatief affect. Gedragsstudies hebben al aangetoond dat individuen die hoog scoren op negatief affect een verhoogde gevoeligheid vertonen voor fouten en negatieve feedback (Abela & D'Alessandro, 2002; Beats, Sahakian, & Levy, 1996; Conway, Howell, & Giannopoulos, 1991; Elliott, Sahakian, Herrod, Robbins, & Paykel, 1997; Elliott et al., 1996; Enns & Cox, 1999; Henriques & Leitenberg, 2002; Holmes & Pizzagalli, 2008; Pizzagalli et al., 2006; Shafran & Mansel, 2001). Dat negatief affect foutenverwerking beïnvloedt, werd ook bevestigd door ERP studies. In deze studies werd meer specifiek een verhoogde ERN/Ne en/of CRN amplitude geobserveerd (Olvet & Hajcak, 2008; Vaidyanathan et al., 2012) en sommige onderzoekers hebben daarom gesteld dat de ERN/Ne als een marker kan dienen voor de diagnose van stoornissen zoals angst en depressie, die beide gekarakteriseerd worden door een hoog niveau van negatief affect (Olvet&Hajcak, 2008). Angst wordt inderdaad systematisch gelinkt aan een vergrote ERN/Ne (Hajcak, McDonald, & Simons, 2003; Olvet & Hajcak, 2008; Simons, 2010; Vaidyanathan et al., 2012; Vocat, Pourtois, & Vuilleumier, 2008). Het plaatje voor depressie is echter minder duidelijk (Chiu & Deldin, 2007; Compton et al., 2008; Foti & Hajcak, 2009; Holmes & Pizzagalli, 2008; Pizzagalli, Peccoralo, Davidson, & Cohen, 2006; Santesso et al., 2008; Schrijvers et al., 2008; Schrijvers et al., 2009). Echter, wat deze modulaties door negatief affect in foutengerelateerde componenten zoals de ERN/Ne en CRN, alsook de Pe en FRN echter betekenen is niet geheel duidelijk. Is dit een gevolg van problemen in de executieve controle waarmee angstige en depressieve individuen gekenmerkt worden, of duidt dit op een deviante motivationele en/of emotionele reactie t.o.v. fouten in deze groepen vergeleken met gezonde controles?

Deze vraag heb ik getracht te beantwoorden in dit proefschrift door gebruik te maken van standaard experimentele methoden en EEG/ERP's. Het gebruik van deze methode en techniek maakte het mogelijk hersenprocessen gerelateerd aan de verwerking van fouten in hoog-angstige studenten en depressieve patiënten te onderzoeken en om de functionele betekenis van veranderingen in deze processen bloot te leggen. De experimentele taak die in al

de studies gebruikt werd in dit proefschrift, was de “Go/noGo” taak. Een belangrijk kenmerk van deze taak is dat de deelnemers zo snel mogelijk moeten reageren wanneer een bepaalde visuele stimulus, in dit geval een pijl (Go), op het scherm verschijnt, terwijl ze hun respons moeten onderdrukken wanneer er een andere specifieke stimulus (noGo) verschijnt. Fouten op deze taak worden gemaakt wanneer de deelnemer zijn/haar respons niet kan onderdrukken, dus als de inhibitie faalt (i.e. Vals Alarm) en de detectie van deze soort fout is gebaseerd op een interne voorstelling van de juiste actie. In deze taak was echter niet alleen de juistheid of accuraatheid, maar ook de snelheid van de actie belangrijk en daarom werd er ook feedback gegeven na elke actie. Zo werden deelnemers geïnformeerd of ze al dan niet juist en/of snel genoeg waren. In dit geval bevindt de voorstelling van de correcte actie zich dus buiten de persoon of in de omgeving. Zowel de “interne” verwerking van fouten (Hoofdstuk 2 en 3) als de “externe” (Hoofdstuk 3) werden onderzocht in dit proefschrift m.b.t. negatief affect (i.e. angst en depressie). Verder werd er ook een nieuw paradigma ontwikkeld om de affectieve waarde van acties af te leiden tijdens de Go/noGo taak (Hoofdstuk 5 en 6) om zo een beter inzicht te krijgen in de betekenis van veranderde foutengerelateerde hersenprocessen in angstige en depressieve mensen. Een gedetailleerd overzicht van deze bevindingen wordt hieronder gegeven.

OVERZICHT VAN DE BEVINDINGEN

In **Hoofdstuk 2** werd de predictie getest dat een grotere ERN/Ne component niet enkel een overactief of hypersensitief foutenmonitoring systeem in angstige mensen weerspiegelt, maar dat dit ook kan gepaard gaan met een afwijkende manier van fouten verwerken. Daarom hebben we in deze studie niet enkel naar kwantitatieve veranderingen in de ERN/Ne component (i.e. amplitude) gekeken maar ook naar kwalitatieve veranderingen (i.e. topografie en onderliggende hersenstructuren die de ERN/Ne component genereren). Laag- en hoog-angstige studenten voerden de Go/noGo taak uit terwijl hun EEG werd gemeten.De gedragsresultaten lieten zien dat zowel laag-

als hoog-angstige studenten evenveel fouten maakten en dat ze even snel reageerden. De ERN/Ne component bleek echter groter te zijn in hoog- vergeleken met laag-angstige studenten en meer ventrale ACC gebieden waren betrokken tijdens deze vroege detectie van fouten bij hoog-angstige studenten. De Pe component daarentegen was niet verschillend tussen de twee groepen. Deze resultaten suggereren dat angst de emotionele of motivationele verwerking van een fout beïnvloedt.

Omdat angst en depressie sterk overlappende en comorbide stoornissen zijn(Clark & Watson, 1991; Mineka, Watson, & Clark, 1998; Watson et al., 1995), werd een gelijkaardige methode gebruikt in **Hoofdstuk 3** om vroege foutendetectie processen in depressieve patiënten te onderzoeken. Dezelfde Go/noGo taak als in Hoofdstuk 2 werd gebruikt in dit experiment en het EEG werd opnieuw gemeten. Depressieve patiënten maakten evenveel fouten als gezonde controles en zoals verwacht waren depressieve mensen in het algemeen trager. Echter, in tegenstelling tot de ERN/Ne resultaten bij hoog-angstige studenten in de vorige studie, was de amplitude van de ERN/Ne niet verhoogd in depressieve patiënten.De bijkomende topografische en neurale bronlokalisatie analyses toonden echter wel aan dat er extra gebieden namelijk dorsolaterale prefrontale gebieden (naast de klassieke mediale frontale en dorsale ACC regio's) werden gerekruteerd tijdens de vroege verwerking van een fout bij depressieve patiënten. Deze activering zou gerelateerd kunnen zijn aan ruminatie tijdens de vroege detectie van een fout bij depressieve patiënten. De Pe daarentegen was kleiner bij depressieve individuen en ging gepaard met een sterkere rekrutering van ventrale cingulate gebieden. Dit laatste effect bleek gerelateerd te zijn aan het niveau van ruminatie. De studies die beschreven zijn in Hoofdstuk 2 en 3 laten dus een dissociatief effect van angst en depressie zien op de foutengerelateerde componenten, ERN/Ne en Pe.

In **Hoofdstuk 4** werd er vervolgens gekeken of trekangst ook, naast "interne" fouten detectie processen (zie Hoofdstuk 2), "externe" fouten detectie processen in de hersenen beïnvloedt, aangezien de ERN/Ne en de FRN verondersteld worden eenzelfde neurale systeem te delen (Holroyd & Coles,

2002). Daarvoor werd de Go/noGo taak aangepast en werd er meer bepaald salliante feedback aangeboden na elke actie terwijl EEG werd gemeten bij laag- en hoog-angstige studenten. In tegenstelling tot de resultaten die beschreven zijn in Hoofdstuk 2, werd in dit experiment geen effect van angst gevonden op de ERN/Ne of Pe amplitude. De FRN was echter wel gemoduleerd door angst. De FRN in hoog-angstige deelnemers bleek veel kleiner te zijn dan de FRN in laag-angstige deelnemers. Angst blijkt dus een effect te hebben op het monitoren van acties en meer specifiek op interne alsook op externe representaties van acties.

In **Hoofdstuk 5** stellen we een nieuw gedragsexperiment voor dat gebaseerd is op het evaluatieve priming paradigma en waarmee we gepoogd hebben om de affectieve waarde van zelf gegenereerde acties te meten. Hiermee wilden we meer specifiek te weten komen of de affectieve waarde van een actie automatische geactiveerd wordt en daarom werd in dit paradigma elke actie in de Go/noGo taak gevolgd (SOA = 300 in Experiment 1 en 2; SOA = 600 in Experiment 3 en SOA = 1000 ms in Experiment 4) door een positief of negatief woord dat zo snel mogelijk als positief of negatief moest worden geclassificeerd. Zoals voorspeld werd een evaluatief priming effect geobserveerd wanneer de SOA relatief kort was (i.e. SOA = 300 ms of 600 ms), maar niet wanneer deze langer was (i.e. 1000 ms). Negatieve woorden werden dus sneller gecategoriseerd na een fout dan na een correcte respons en positieve woorden sneller na een correcte respons. Als de valentie van het woord dus congruent was met de valentie van de actie, werd er sneller geantwoord. De affectieve waarde die gerelateerd is aan de juistheid van een actie blijkt dus snel en automatisch geëvalueerd te worden en fouten blijken als meer negatief te worden beoordeeld vergeleken met correcte reacties. Dit effect was echter kleiner wanneer het angstniveau van de deelnemer hoger was, wat zou kunnen wijzen op een verstoorde vroege automatische verwerking van de emotionele waarde van zelf gegenereerde acties.

In **Hoofdstuk 6** werden vervolgens de elektrofysiologische correlaten onderzocht van dit evaluatieve priming effect. De resultaten van deze studie toonden meer bepaald dat de grootte van het evaluatieve priming effect

gerelateerd was aan de sensitiviteit van de ERN/Ne/CRN aan de accuraatheid van acties; als het verschil tussen de ERN/Ne en de CRN groot was, werd er ook een groter verschil in de snelheid gevonden waarmee positieve en negatieve woorden werden gecategoriseerd. Dit suggereert dat de ERN/Ne/CRN component gerelateerd is aan de affectieve verwerking van acties. Verder vonden we ook dat fouten de vroege perceptuele componenten gerelateerd aan de emotionele verwerking van woorden beïnvloedde (i.e. "early posterior negativity" - EPN component; Herbert, Junghöfer, & Kissler, 2008; Schupp, Flaisch, Stockburger, &Junghöfer, 2006), terwijl correcte responsen latere processen beïnvloedde (i.e. "late positive potential" - LPP component; Hajcak, MacNamara, &Olvet, 2010; Olofsson, Nordin, Sequeira, &Polich, 2008; Schupp et al., 2000). Dit verschil in tijdsverloopgeeft aan dat fouten gerelateerd zijn aan een verhoogde motivatie of affect.

CONCLUSIE

Deze resultaten hebben belangrijke implicaties voor het verband tussen foutengerelateerde componenten zoals de ERN/Ne, Pe en FRN, en negatief affect. In wat volgt, zullen eerst de geobserveerde dissocieerbare effecten van angst en depressie op de ERN/Ne en Pe ERP componenten besproken worden. Vervolgens wordt het effect van angst op interne en externe foutenmonitoringsprocessen besproken (ERN/Ne vs. FRN). Tot slot wordt dieper ingegaan op het verband tussen affect en het monitoren van acties (correcte en incorrecte) in het algemeen en wordt er besproken of dit verband anders zou kunnen zijn in depressieve en angstige mensen.

ERN/Ne-angst vs. Pe-depressie?

In eerste instantie bestudeerden we het effect van twee stoornissen of trekken, die gerelateerd zijn aan negatief affect, op het verwerken van fouten. Olvet and Hajcak (2008) hebben gesteld dat negatief affect en internalizerende stoornissen zoals angst en depressie gepaard gaan met een grotere ERN/Ne. De resultaten die geobserveerd werden in de studies beschreven in Hoofdstuk 2 en 3, laten inderdaad zien dat negatief affect of internalizerende stoornissen

foutengerelateerde componenten beïnvloeden. Trekangst bleek echter de ERN/Ne te beïnvloeden: hoog angstige mensen hadden een grotere ERN/Ne en rekruteerden meer ventrale ACC gebieden. Depressie bleek echter meer de Pe te beïnvloeden: depressieve patiënten hadden een kleinere Pe en rekruteerden minder ventrale ACC gebieden. Deze resultaten bevestigen dus dat de ERN/Ne en Pe verschillende processen tijdens het monitoren van fouten omvatten (Nieuwenhuis et al., 2001; Overbeek et al., 2005; Ridderinkhof et al., 2009). Terwijl de ERN/Ne een snelle en automatische detectie van een fout weergeeft, vertaalt de Pe component zich meer in gecontroleerde of bewuste foutendetectie processen (Dhar et al., 2011; Endrass, Franke, & Kathmann, 2005; Nieuwenhuis et al., 2001; O'Connell et al., 2007) die de verdere accumulatie van evidentie omtrent het maken van een fout weergeeft (Steinhauser & Yeung, 2010).

Veranderingen in de ERN/Ne werden geïnterpreteerd in het kader van de "attentional control theory" (Eysenck, Derakshan, Santos, &Calvo, 2007), die stelt dat angstige mensen zich meer inspannen tijdens een cognitieve taak om zo te compenseren voor het feit dat ze zich zorgen maken over fouten die ze eventueel in de toekomst kunnen maken en om fouten in de toekomst te vermijden. Een verhoogde ERN/Ne zou dus wijzen op een grotere inspanning. Een verhoogde ERN/Ne werd niet gevonden in depressieve patiënten, wat zou kunnen wijzen op het feit dat deze patiënten geen compensatiegedrag vertonen om met hun intrusieve gedachten om te gaan. Daarentegen, depressieve mensen die geneigd zijn om te focussen op negatieve gebeurtenissen in het verleden, zouden meer focussen op fouten die ze "in het verleden" hebben gemaakt (i.e. rumineren) waardoor ze minder aandacht hebben voor de verdere "bewuste" verwerking van een fout, wat gereflecteerd zou worden in een kleinere Pe component.

Een verschillend effect van angst op interne en externe foutendetectie processen

In tweede instantie hebben we de vraag trachten te beantwoorden of angst, naast interne foutendetectie processen, ook externe foutendetectie

processen in de hersenen beïnvloedt. Hieruit bleek eerst en vooral dat, in een taak waarin duidelijke externe feedback wordt aangeboden, angst geen effect meer heeft op interne foutendetectie processen. Deze bevinding bevestigt dus niet de hypothese van Olvet en Hajcak (2008) die de ERN/Ne als een endophenotype van angst zien. Angst bleek echter wel een effect te hebben op externe foutendetectie: De FRN in hoog-angstige individuen discrimineerde minder tussen negatieve en positieve feedback, dan de FRN in laag-angstige individuen. Alhoewel deze observatie contrasteert met de interne foutendetectie (ERN/Ne) bevindingen in hoofdstuk 2 waar een grotere ERN/Ne was geobserveerd voor hoog- vergeleken met laag-angstige studenten, lijken deze FRN resultaten wel te bevestigen dat angst actie-monitoring processen (interne en externe) lijken te verstoren.

De kleinere FRN in hoog angstige individuen zou een gevolg kunnen zijn van een probleem in de monitoring van de actie die voorafging aan de feedback en zou meer specifiek de integratie tussen de waarde van deze actie en de valentie van de feedback beïnvloeden. Deze interpretatie werd indirect bevestigd door de correlatie tussen de FRN amplitude en de mate van het hebben van een interne locus of control (i.e. het niveau waarop mensen geloven dat veranderingen in de omgeving door hun acties worden veroorzaakt). Deze correlatie laat zien dat de FRN groter wordt naarmate mensen meer het gevoel hebben dat ze zelf (of hun acties) de controle hebben over of de oorzaak zijn van de veranderingen in hun omgeving. Echter, deze correlatie werd enkel in laag angstige individuen geobserveerd.

Zelf gegenereerde acties verwerven automatisch een affectieve waarde

Onderzoek heeft aangetoond dat fouten niet enkel het leren bevorderen (Frank et al., 2005; Holroyd& Coles, 2002), maar dat deze ook gepaard gaan met emotionele veranderingen. Zo werden er veranderingen in defensieve reacties (Hajcak & Foti, 2008) en in amygdala activiteit (Polli et al., 2009; Pourtois et al., 2010) geobserveerd na een fout. Deze eerdere bevindingen uit de literatuur werden verder indirect ondersteund door de effecten van negatief affect, op de

detectie van fouten, die geobserveerd werden in de studies beschreven in Hoofdstuk 2 t.e.m. 4.

De resultaten in de studies beschreven in Hoofdstuk 5 en 6 bevestigen verder het idee dat fouten gerelateerd zijn aan negatief affect. Door gebruik te maken van het evaluatieve priming paradigma observeerden we namelijk dat acties snel en automatisch geëvalueerd worden op een affectieve schaal (positief – negatief) en dat fouten vs. correcte acties vroege en late aandachtsprocessen tijdens emotionele woordverwerking op een verschillende manier beïnvloeden (fouten-EPN vs. correcte acties-LPP). Dit suggereert verder dat fouten vs. correcte acties anders verwerkt worden op affectief gebied.

Meer nog, het evaluatieve priming effect bleek kleiner te zijn naarmate het niveau van angst hoger werd en het bleek ook gerelateerd te zijn aan de mate waarin de ERN/Ne discrimineerde tussen correcte en incorrecte acties. Naarmate de ERN/Ne meer discrimineerde tussen correcte en incorrecte acties, was ook het evaluatieve priming effect groter. Deze observaties suggereren dat een afwijkende monitoring van fouten bij angstige of depressieve individuen zou kunnen te wijten zijn aan het feit dat deze individuen problemen vertonen in het automatisch koppelen van een affectieve waarde aan hun acties (positief vs. negatief; goed of slecht; zie Cacioppo & Gardner, 1999).

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