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Titel der Arbeit

## Effects of learned helplessness on error processing investigated with event-related potentials

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Für Maximilian und Roman

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# Theoretical Part

# 1. Introduction

In order to adopt an effective behavior, humans must be capable of monitoring their actions and therefore successfully detect errors of any kind. In the last decades, neuroscientific findings not only shed light on the neural correlates of action-monitoring but also revealed a specific error-related neural reaction. This specific error-related neural response seems to be influenced by a series of factors, such as emotional aspects or psychopathological variables.

The goal of the present thesis is to examine the interaction between error processing and specific emotional states, by focusing on the impact of induced helplessness on the neural correlate of error monitoring, the so-called error-related negativity (Falkenstein, Hohnsbein, Hoormann & Blanke, 1991; Gehring, Goss, Coles, Meyer & Donchin, 1993).

Helplessness was determined to be of importance in the etiological chain of depression but also within the context of academic achievement (Abramson, Metalsky & Alloy, 1989; Mikulincer, 1994). Helplessness is usually characterized by emotional, motivational and cognitive deficits. More specifically, feelings of anxiety, passivity and difficulty to learn that events can be influenced by one's own behavior can be observed (Seligman, 1975).

In experimental settings, helplessness was first generated in dogs and other animals (Mikulincer, 1994). In human experiments, helplessness was mostly induced by withdrawing the control of the participants over different tasks which require the selection, organization and implementation of responses to solve a problem (Mikulincer, 1994). In our study, participants were exposed to a helplessness inducing treatment consisting of a series of numbers followed by a flanker task. During the flanker task, we recorded the EEG of the subjects in order to analyze the error-related neural correlate and compared the subjects who became helpless vs. those who did not.

# 2. Theoretical background of learned helplessness

## *2.1 The concept of learned helplessness – a chronological overview*

The origins of experimentally induced helplessness can be found in the work of Solomon, a theoretician in the field of the psychology of learning. In one of his experiments with dogs, he observed that some of his test animals showed evidence of helpless and passive behavior, an aspect that occurred rather accidentally and hindered his empirical study (cf. Meyer, 2000). Such animals became lethargic and did not try to escape electrical shocks even when they were given the possibility to do so.

These entirely unforeseen results became the subject of further investigations of several researchers such as Overmier and Seligman (1967), or Seligman and Maier (1967).

As the appearance of passive and helpless behavior in dogs could have had more than one explanation, e.g. the habituation of the dogs to electrical shocks, Overmier and Seligman (1967) proposed a triadic design and examined the behavior of three groups of dogs. The control group received no training at all, one of the two treatment groups was exposed to uncontrollable electrical shocks and the other to controllable electrical shocks. The results clearly showed that only the dogs exposed to the uncontrollable electrical shocks were the ones that developed passive behavior. Seligman (1972) referred to that kind of behavior as learned helplessness. The same behavior was also shown in several animals e.g. rats, cats and fish (cf. Maier & Seligman, 1976). Hence, a large series of experiments on humans followed, all aiming to investigate human helplessness.

One of the pioneers of helplessness studies in humans was Hiroto (1974), who exposed his subjects in a triadic design to loud and aversive noise. Comparable to the animal experiments, his results showed that the subjects who belonged to the treatment group whose training-phase was made up of the uncontrollable situation were less likely to exercise control over the aversive noise. The majority of them were not able to learn how to escape the noise when they were given the possibility to do so.
Hiroto subsequently dedicated one of his next studies to the issue of generalization of human helpless behavior (Hiroto & Seligman, 1975) and observed that uncontrollability induced via a noise escape task decreased posttest performance also in anagrams or concept formation tasks.

## *2.2 Theory of learned helplessness – the first version*

Seligmans' first version of the theory of human learned helplessness (LH, 1975) was partially determined by the results of the just mentioned experiments.

Within this theory there is an important term that has to be explained: the uncontrollability of an event E.

Seligman (1975) postulates an event as uncontrollable when the relation between this certain event and the reaction of a person is non-contingent. In concrete terms, if the occurrence probability of an event E remains the same as well after somebody is executing a behavior R as in the absence of that certain behavior R [p (E/R) = p (E/non - R)], the concerned event is uncontrollable through that behavior R.

The time course of LH can be summarized in the following way:

The relation between an event and the reaction to that event is being cognitively represented as non-contingent if subjects either figure out or only wrongly believe to receive the same amount of reinforcement, whether or not they exercise a certain behavior. The cognitive representation of non-contingency leads to expectations of future uncontrollability of the certain event and generalizes on other future events. The final outcome of that process is characterized by motivational, cognitive and emotional deficits (i.e. clinical symptoms).

Uncontrollable situations generate motivational deficits such as a cessation in attempts to escape the aversive stimulus (Seligman, 1975) because the incentive to initiate voluntary responses is undermined by the expectations of uncontrollability. The cognitive disturbances refer to difficulties to learn that controllable events can be influenced by one´s own behavior and the emotional disturbances comprise feelings of anxiety and depression.

Figure 1 schematically illustrates the time course of LH.

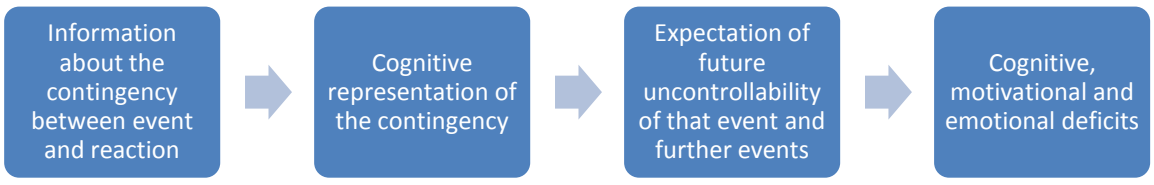


**Fig.1.** First version of the theory of LH modified after Seligman (1975, p.47) and Maier and Seligman (1976) as cited in Meyer ( 2000, p.39).

This briefly described theory became an intensively disputed research topic. The following subchapters will give a short overview on the subsequent milestones of this field of research.

## *2.3 An upgrading perspective of human learned helplessness: reactance, perceived task value and further points of critique*

Further studies on human LH revealed several inconsistencies. Thornton and Jacobs (1972) for example, showed that subjects increased their performance in the test-phase after running through a training-phase designed to generate uncontrollability while the performances of the subjects who experienced a controllable training-phase remained constant.

Wortman and Brehm (1975) explained these and other comparable results (Roth & Bootzin, 1974, as cited in Meyer, 2000, p. 54) by proposing the integration of reactance theory into the theoretical framework of LH. They considered the length of the training-phase designed to generate uncontrollability as playing a major role for the outcome. If the training is not too long, so that the subject still expects to be able to gain control over the event, the result of such training would be the generation of reactance. If the training is long enough, then the subjects realize they are not able to control the event and therefore become helpless.

Furthermore, Wortman and Brehm (1975) proposed task value as a moderator variable in the process of coping with uncontrollable situations. When the task is not important enough for a subject, then neither reactance nor the typical helplessness deficits occur. The assignment of importance to a task leads to reactance and - provided that the situation still remains uncontrollable - to particularly pronounced LH deficits. In other words, the higher the task value, the stronger the learned helplessness deficits will be.

Kuhl (1984) suggested the „action versus state orientation“as further moderators of the LH effect. Action orientation describes the aptitude of a person to regulate his/her emotions, thoughts or behaviors in order to be able to fulfill his/her intention. State orientation refers to the opposite, hence the incapacity to modulate these emotions, thoughts or behaviors and as a consequence the incapacity to modify emerging anxiety, confusion, or uncertainty (Kuhl, 1981). Kuhl (1984) hypothesized that the state orientation of a person is responsible for the performance losses after a helplessness-training. More precisely, he assumed the thoughts and mechanisms that are characteristically for the state orientation to interfere and cause a decrease of performance.

## *2.4 The attributional theory of learned helplessness*

Abramson, Seligman and Teasdale (1978) assigned a crucial role to attributional styles in explaining the developmental process of the LH deficits and closed thereby two gaps of Seligman’s (1975) first version of the theory.

The first shortcoming which Abramson et al. (1978) emphasized in this context was the fact that the original version did not distinguish between universal and personal helplessness. This differentiation is important because the experience of universal helplessness does not cause any self-esteem deterioration, while the personal helplessness causes a self-esteem reduction. Universal helplessness occurs if a person expects the outcome to be not contingent on any response in his/her repertoire and also not contingent on responses in the repertoire of any relevant other (Abramson et al., 1978). On the other hand, personal helplessness occurs if a person expects the outcome to be not contingent on any response in his/her repertoire but contingent on a response in the repertoire of a relevant other (Abramson et al., 1978).

The second point of critique was the generalization of LH deficits. The implication of the original theory (Seligman, 1975) that helplessness deficits following uncontrollability are highly general has been refuted by a series of studies with inconclusive results (see Mikulincer, 1994, for an overview).

Abramson et al. (1978) specified that the solution for resolving these problems would be the integration of the concept of causal attributions into the model of LH. They considered the dimensions internal vs. external, stable vs. unstable and global vs. specific to play a major role in the genesis of LH as follows:

I. Internal attributions[1] lead to personal helplessness while external explanations[2] are associated with universal helplessness. Therefore, the internal attributional styles presumably cause deterioration of self-esteem whereas external attributional styles seem not to damage the self-esteem.
II. An explanatory style based upon stable causes[3] determines persistent helplessness while an attributional style based upon unstable[4] causes seems to favor the appearance of transient helplessness deficits.
III. Global attributions[5] seem to open the gates for the generalization of the helplessness-deficits to other situations whereas specific[6] ones affect similar outcomes or situations.

Finally, as far as the nature or characteristics of helplessness deficits are concerned, the authors agree on the symptoms hypothesized in Seligman's (1975) original version of the theory.

[1] Explanations of events based on internal causes (e.g. intelligence, personality)
[2] Explanations of events based on external causes (e.g. the behavior of other persons)
[3] e.g. lack of ability.
[4] e.g. lack of motivation
[5] If a person believes that a certain outcome affects all aspects of life
[6] If a person believes that a certain outcome does not affect all aspects of life

## *2.5 Helplessness and Psychopathology*

According to the existing literature, there are indubitable ties between learned helplessness and psychopathology.

As one of the very first, Seligman (1975) showed similarities between both cognitive and behavioral symptoms of LH and depression and concluded that the exposure to uncontrollable situations can be understood as an antecedent of the reactive[7] depression. Within this context, he drew parallels between LH and depression by emphasizing the reduced motivation for deliberate reaction, the negative thought patterns, the time course, the reduced aggressiveness as well as the loss of appetite and interest for sexual and social activities that are characteristic for LH and depression.

Seligman (1975) also referred to LH and depression as having common aspects in their etiology since reactive depression as well as LH are typically preceded by critical life events (e.g. death of a loved person, illness, financial difficulties) which are uncontrollable.

However, also Mikulincer's (1994) perspective of the above mentioned connection added valuable insights. He offered an integrative solution and took into account several findings mentioned in subchapter 2.3. In his work, he described an interesting defragmentation of the time course of LH which concomitantly can be of interest also for translational research such as academical achievement issues (see Figure 2).

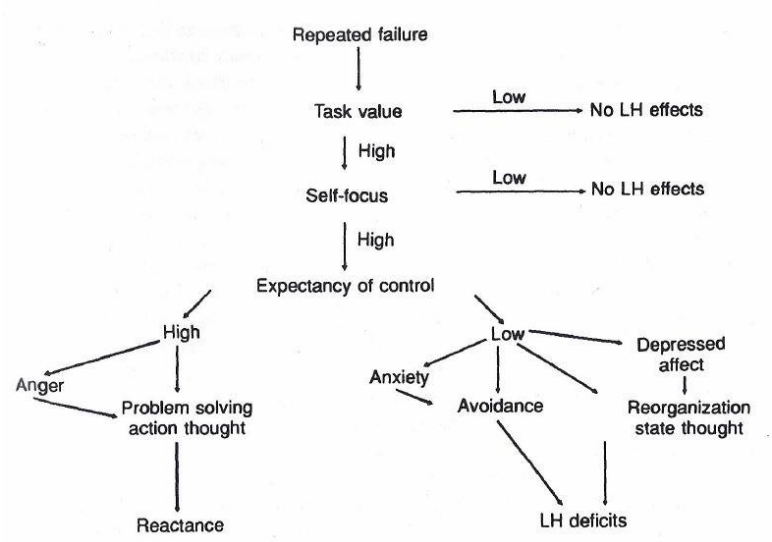


**Fig. 2**. Coping flowchart of helplessness training (Mikulincer, 1994, p. 243).

[7] Term is no longer used in classification systems as for instance ICD-10 (International Classification of Diseases, World Health Organization). Reactive depression was supposed to be triggered by external events (e.g. loss of a loved one)

Regarding psychopathology, Mikulincer (1994) hypothesized the decisive point in uncontrollable situations to be the adoption of an avoidance coping style. He suggested that intensive and prolonged avoidance coping leads to a "structural fragmentation of thought and disorganization of action" (Mikulincer, 1994, p. 269) and causes dysfunctional LH deficits. These deficits anyhow, can indicate also other psychopathological reactions, such as paranoid or panic reactions as well as obsessive-compulsive and posttraumatic stress disorders.

Finally, to better describe the connection between helplessness and psychopathology, it can be stated that two revisions of the initial theory of LH were particularly striking: the attributional reformulation of the learned helplessness theory (Abramson et al., 1978) – as described in the previous subchapter 2.4. – and the hopelessness theory of depression proposed by Abramson, Metalsky and Alloy (1989), which will be presented in the following subchapter.

## *2.6 The Hopelessness Theory of Depression*

Abramson, Metalsky and Alloy (1989) focused on a more clinical research line and postulated the existence of a specific subtype of depression, the so called hopelessness depression.

The hopelessness theory of depression is a diathesis-stress[8] approach which describes the causal chain of hopelessness depression. Even though Abramson et al. (1989) posit that LH is playing a role in the genesis of the above mentioned subtype of depression, the core point of their concept is no longer the LH but the hopelessness state. In concrete terms, they specify that LH arises in consequence of non-contingency between an outcome and one's own behavior while the hopelessness state involves also negative expectations about the occurrence of highly valued outcomes in addition to helplessness expectancies. The model itself can be described in the following manner:

### *2.6.1 Basic assumptions*

Hopelessness is the only sufficient cause of the symptoms of hopelessness depression. All other causes only increase the occurrence-likelihood of hopelessness depression symptoms but are neither necessary nor sufficient for their occurrence (Abramson et al., 1989). Furthermore, all causes differ in operating either at the beginning or towards the end of the etiological chain of hopelessness depression and are therefore called distal respectively proximal causes. Figure 3 depicts the model, as postulated by Abramson et al. (1989).

[8] Diathesis-stress models explain a disease based on the interaction between a (mainly biological and genetic) vulnerability component and a stress component which arises from external events.

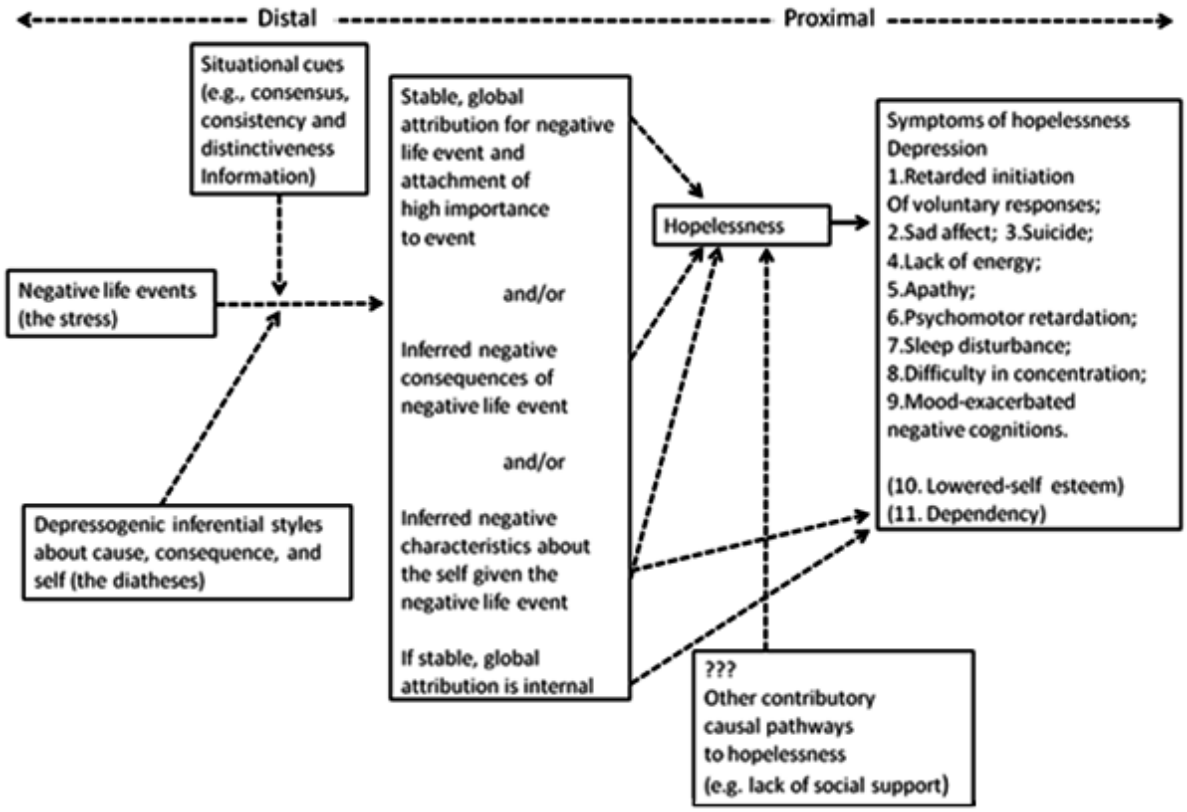


**Fig.3.** Causal chain of hopelessness depression (Abramson et al.,1989, p. 360). The solid lines indicate sufficient causes, the broken lines contributory causes.

### *2.6.2 Diathesis-stress components of the model*

The negative life events are regarded to be the stress-component in the model. The diathesis-component of the model refers to the depressogenic attributional styles of a person. To Abramson et al. (1989), depressogenic attributional styles are the cognitive styles of people who tend to attribute negative events to stable and global factors respectively to view these events as very important. Both, the diathesis component and the stress component should be understood as continuous. That is because some people may exhibit more negative styles than others and some events may generate hopelessness in most people (e.g. being a prisoner in a concentration camp) whereas among cognitively vulnerable people, also minor events, chronic stressors or daily hassles may act as a stressor-component. Furthermore, the authors also assume that “the less negative a person’s cognitive style, the more negative an event needs to be in order to interact with that style and contribute to formation of [depressive] symptoms” (Abramson et al., 1989, p. 362).

### *2.6.3 Time course*

The causal chain of hopelessness depression begins with the occurrence of a negative life event. The interaction between negative life events and depressogenic attributional styles may lead to specific inferences, which in turn modulate whether somebody becomes hopeless. Those specific inferences are (see also Figure 3):

I. Inferred stable and global causes of a negative life event and a high degree of importance attached to the event and/or
II. inferred negative consequences resulting from the occurrence of the negative event and/or
III. inferred negative characteristics about the self given that the event occurred

Due to the nature of the negative life events, those inference-types are not equally important in contributing to whether or not a person becomes hopeless. To exemplify this issue, the authors describe how a young girl may become hopeless after her mother's death rather because of the negative consequences than because of the death's cause or the immediate implications for the view of herself. In other words, the concerned child might become hopeless because the suffered loss of a high valued person is irreversible rather than due to the death's cause or the immediate implications for the view of the self. At the same time, other negative life events do not necessarily imply the same hierarchy of inferences.

Additionally to the cognitive factors enumerated above, also personal differences regarding the experienced social support can play a role in modulating one person's pathway to hopelessness.

Finally, the hopelessness culminates in symptoms such as retarded initiation of voluntary responses, sad affect, suicidal ideation, lack of energy, apathy, psychomotor retardation, sleep disturbance, concentration problems and negative cognitions determined by the negative mood or low self-esteem and dependency on persons from the living environment. Those symptoms are characteristic for hopelessness depression.

## *2.7 Sex and Helplessness*

The question regarding the differences between helpless men and women has been analyzed with respect to different settings: the academical achievement as well as the clinical setting.

Referring to the academical achievement issues, some contradictory results have been reported. Erkut (1983) as well as Stipeck (1984, as cited in Mikulincer, 1994, p.133), conclude in a developmental study that girls are more likely to make stable attributions for failure and to expect lesser control for a new task after a helplessness training. The findings of Parsons, Meece, Adler and Kaczala (1982) however, suggest no differences as they did not detect any evidence to support the hypothesis that girls are generally more helpless in mathematics than are boys.

Results from clinical studies clearly show sex differences in depressive symptoms, which appear in early adolescence and then remain throughout the adult life span (Nolen-

Hoeksema, Larson & Grayson, 1999). More precisely, it seems that beginning with the age of 15, girls and women are twice as likely as boys and men to experience depression (Nolen-Hoeksema & Girgus, 1994, p. 424). Recent studies also indicate that females are more vulnerable to depression than males (Stone, Gibb & Coles, 2010).

At the same time, women tend to focus more strongly on themselves and their mood when they are in a depressed mood, which, in turn, leads them to experience longer periods of depressed mood (Butler & Nolen-Hoeksema, 1994, p. 331). Furthermore, also other studies indicate different reactions to depressed mood in males and females. Thus, males are supposed to rather distract themselves than to ruminate as a consequence of depressed mood (Nolen-Hoeksema, 2002, as cited in Addis, 2008, p. 156).

Against this backdrop and also considering the role LH is playing in the causal chain of hopelessness depression, it seems conceivable to assume that sex differences in experiencing LH might occur. We assume to find such differences on cortical level, related to a specific neural reaction to errors that will be described in the following chapter of the thesis.

# 3. Error Processing

It is inevitable for humans to attain a certain level of psychological and physiological well-being without being able to adapt themselves to the complexity of their living environment. Adaptational actions and processes in turn, require the allocation of numerous resources on the part of an organism and implicate, among other skills, the ability to detect and correct one´s own errors.

The following section of the thesis will expand on the topic of error processing and describe the ERP components associated with error processing, the hypothesized neural substrate of error processing as well as behavioral findings related to error processing.

## *3.1 Anterior Cingulate Cortex*

The cingulate cortex can be regarded as *"*a neural interface between emotion, sensation, and action*"* (Hayden & Platt, 2009, p. 887). It comprises the cingulate gyrus, which is practically encircling the corpus callosum, *"*as well as the cortex lining the superior and inferior banks of the cingulate sulcus" (Hayden & Platt, 2009, p. 887).

The anterior cingulate cortex (ACC) is the more rostral region of the cingulate cortex consisting of Brodmann`s areas[9] (BA) 24, 25, 32, 33, while the posterior cingulate cortex (PCC) is a region which lies more caudal and consists of BA 29, 30, 23, and 31 (Hayden & Platt, 2009). These two divisions of the cingulate cortex can be differentiated from one

[9] Brodmann's areas were originally defined by Korbinian Brodmann, based on the cytoarchitectural organizations of the neurons

another based on their “cytoarchitecture and patterns of projections, as well as function” (Bush, Luu & Posner, 2000, p. 215). For the ACC, Bush et al. (2000) proposed a functional segregation model, in a rostro-ventral affective division (ACC; originally termed ventral ACC) and a dorsal cognitive division (midcingulate cortex - MCC; originally termed dorsal ACC).

However, with regard to the entire cingulate cortex, more recent cytoarchitectural studies rather favor a four-region model (Vogt, 2005) as follows (see also Figure 4):

**ACC** [with its subgenual (s) and pregenual (p) subdivision], **MCC** [divided in an anterior (a) and a posterior (p) subdivision], **PCC** [divided in a dorsal (d) and a ventral (v) part] and **retrosplenial cortex** (RSC) situated “on the ventral bank of the posterior cingulate gyrus that is not exposed on the gyral surface” (Vogt, 2005, p. 535). More importantly, all those regions and subregions reflect a circuitry and functional organization and not only locations in a three-dimensional coordinate system.

Given our research interest and the above-mentioned circuitry and functional organization of the cingulate cortex proposed by Vogt (2005), it appears appropriate to put the spotlight on the MCC and on its function of response selection.

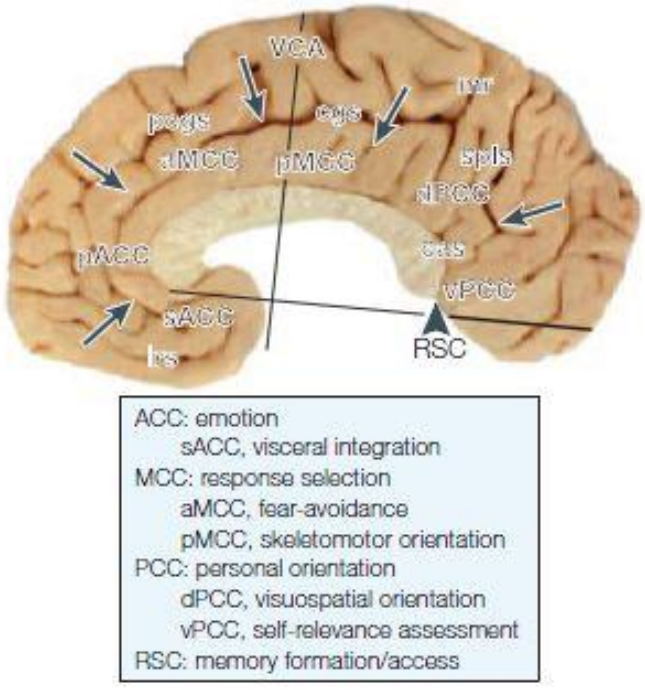


**Fig. 4.** Distribution of the four cingulate regions and subregions (Vogt, 2005, p.535): aMCC - anterior midcingulate cortex; cas - callosal sulcus; cgs - cingulate sulcus; dPCC - dorsal posterior cingulate cortex; irs – inferior rostral sulcus; mr – marginal ramus of cgs; pACC – pregenual anterior cingulate cortex; pcgs – paracingulate sulcus; pMCC – posterior midcingulate cortex; RSC – retrosplenial cortex; sACC – subgenual anterior cingulate cortex; spls – splenial sulci; vPCC – ventral posterior cingulate cortex

In their recent work, Shackman, Salomons, Slagter, Fox, Winter and Davidson (2011) give an overview of various results and suggest that “negative affect, pain and cognitive control are anatomically and functionally integrated at the subdivision level in aMCC, likely within RCZ, the premotor area harboured in the dorsal portion of aMCC” (Shackman et al., p. 164). Thus, they conclude that the aforementioned functional segregation model of Bush and his colleagues (2000) is no longer tenable and propose “the adaptive control hypothesis, which suggests that aMCC uses information about punishment to bias responding when the most adaptive course of action is uncertain” (Shackman et al., p. 164).

## *3.2 Error Related Negativity (ERN)*

Two independently working research groups provided insight into the neuronal processes of action monitoring and error detection of humans by identifying a negative going waveform that could be seen in the averaged EEG and which was time-locked with incorrect responses (Falkenstein, Hohnsbein, Hoormann & Blanke, 1991; Gehring, Goss, Coles, Meyer & Donchin, 1993). Falkenstein and his colleagues (1991) termed this ERP component as error negativity (Ne) while the team of scientists led by Gehring named it error related negativity (ERN). The present thesis will refer to this component as ERN. The ERN (Figure 5) peaks approximately 50-100 ms post response, reaches its maximum at fronto-central recording sites (Falkenstein et al., 1991; Gehring et al., 1993) and is a component which can be observed in connection with a motor response following both visual and acoustic cues (Yeung, Botvinick & Cohen, 2004; Falkenstein, Hoormann, Christ & Hohnsbein, 2000). Moreover, it can be observed also in connection with various response modalities (Holroyd, Dien & Coles, 1998) and is assumed to be generated in the aMCC (Debener, Ullsperger, Siegel, Fiehler, von Cramon & Engel, 2005). The hypothesis concerning the neural source of ERN is sustained by several studies conducted using different methods such as source localization (Dehaene, Posner & Tucker, 1994; Holroyd et al., 1998), magnetoencephalography (MEG), an approach that combines both electric and magnetic data (Miltner, Lemke, Weiss, Holroyd, Scheffers & Coles, 2003), functional magnetic resonance imaging (fMRI, e.g. Carter, Braver, Barch, Botvinick, Noll & Cohen, 1998; Kiehl, Liddle, & Hopfinger, 2000; Menon, Adleman, White, Glover, & Reiss, 2001) and intracerebral recordings (Brazdil, Roman, Daniel, & Rektor, 2005). Additionally, the implication of the aMCC as the neural substrate of error processing has been substantiated also by findings which show diminished ERN in patients with damage to the medial prefrontal cortex, including the anterior cingulate region (Stemmer, Segalowitz, Witzke & Schonle, 2004).

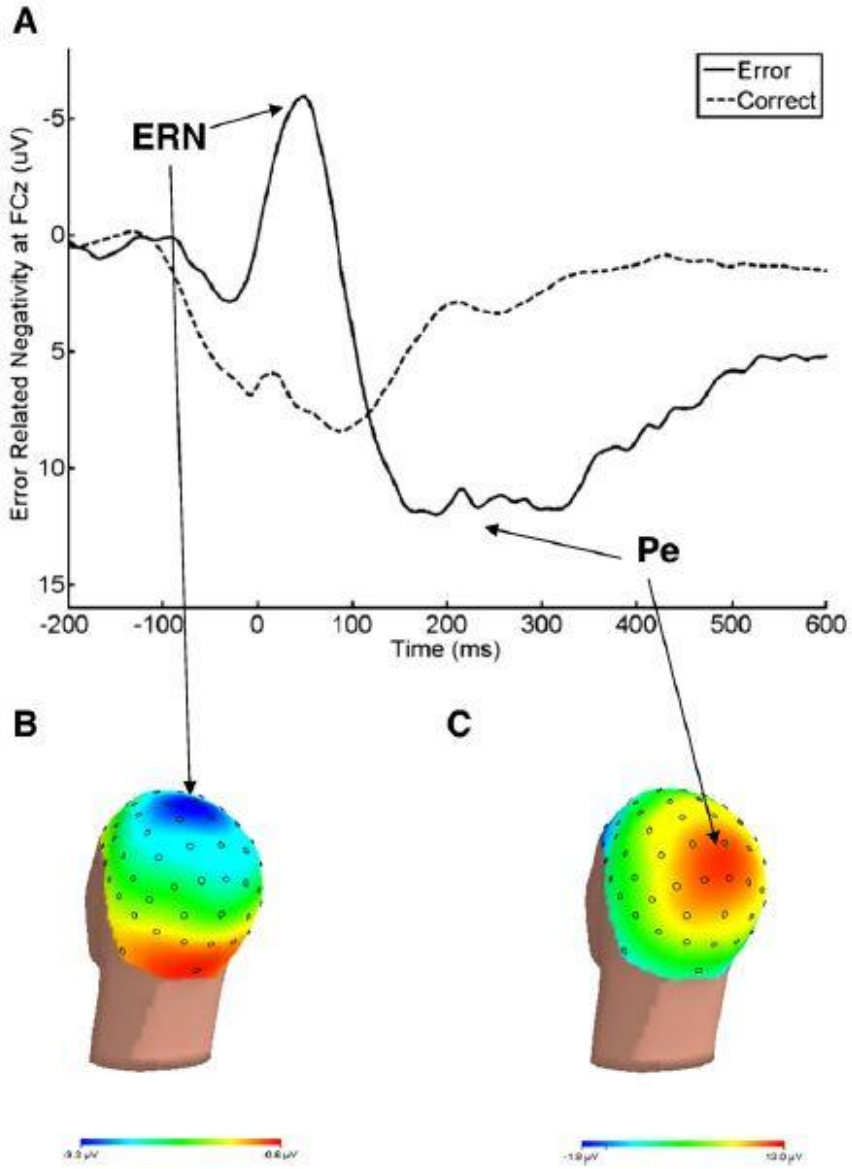


**Fig. 5. A -** Response locked ERPs for error and correct trials at location FCz ; **B** - Typical scalp distribution of ERN; **C** - Typical scalp distribution of Pe (Olvet & Hajcak, 2008, p. 1345).

As to the functional aspects of the ERN, several approaches try to offer an explanation but, so far, there is no scientific agreement on a unifying theory. In general, two main categories of approaches can be distinguished: evaluative and response-selection approaches.

Two prominent evaluative approaches, for instance, differ from one another in terms of how the function of the ERN is understood: error-detection or conflict-detection.

The error-detection approach assumes the ERN to signal errors after the detection of a mismatch between the produced response and the intended, correct response (Coles, Scheffers & Holroyd, 2001; Falkenstein et al., 1990, 2000; Gehring et al., 1993; Scheffers & Coles, 2000). Figure 6 schematically illustrates the theory of the ERN related to error-detection. Coles and his colleagues (2001) describe the above mentioned mismatch theory

as follows: The error processing system is composed of a monitoring system which detects errors and a remedial action system, which initiates remedial actions, like, for example, the slowing of responses. The core of the monitoring system is a comparator, which compares the representation of the produced response with the representation of the correct response.

The dynamics involving this system run along these lines:
People mostly act before they have entirely processed the stimulus and thus commit errors due to impulsive responding. However, as the processing of the stimulus continues after the error, people extract further information necessary to generate a correct response. Hence, a representation of the correct response is derived from it. Evidence for the implicit subsequent processing of a stimulus following an error can be found in Rabbitt, Cumming and Vyas (1978, as cited in Yeung, Botvinick & Cohen, 2004, p. 932) who showed that error correcting responses can be observed within 20 ms of the original incorrect response. Moreover, a motor efference copy of the actual response is sent to the monitoring system. The arrival of the efference copy to the monitoring system triggers its comparison with the representation of the correct response. If a mismatch is detected, an error signal sent to the second error-processing component, the remedial system, follows. The ERN is generated by the arrival of the error-signal at the remedial action system.

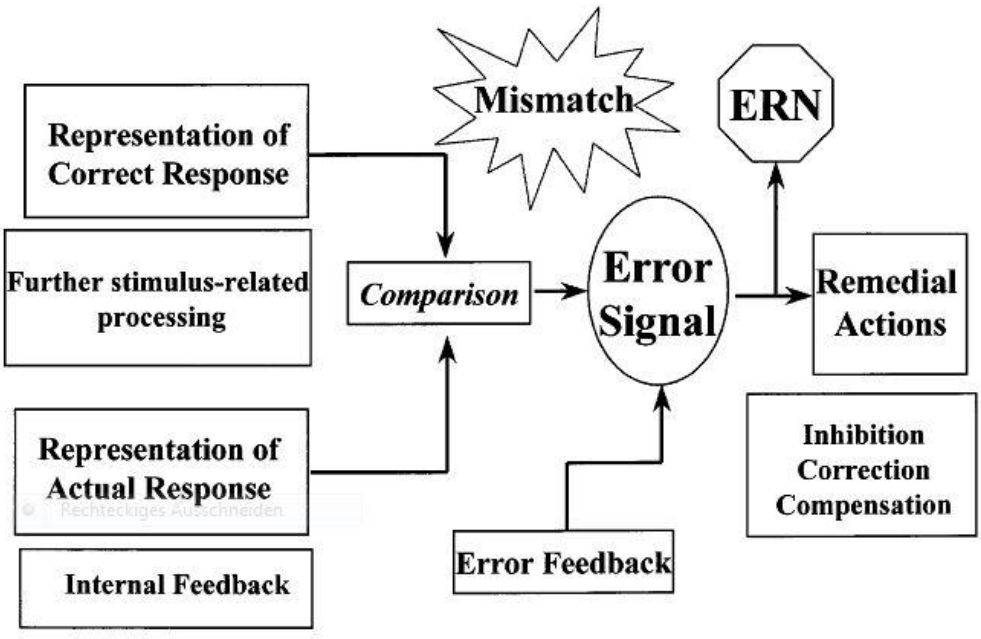


**Fig. 6.** Mismatch theory of ERN/Ne (Coles et al., 2001, p. 174). The ERN/Ne reflects neuronal activity in the ventral bank of the anterior cingulate sulcus.

Though agreement prevails within several research groups on the aspect of the theory assuming a comparison between the representation of the actual response with the representation of the correct response, there is an ongoing debate about what exactly the ERN reflects, as some scientists also propose the ERN to represent the error-detection

process itself (Falkenstein et al., 2000; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001) or an emotional response to the error (Bush et al., 2000; Gehring & Willoughby, 2002; Pailing, Segalowitz, Dywan, & Davies, 2002). However, the latter conception offers the possibility to interpret the ERN not only as a cognitive correlate of error processing but also as an affective correlate of error processing. Data supporting such assumptions will be presented in the last part of this subchapter.

The conflict monitoring theory of the ERN (Botvinick, Braver, Barch, Carter & Cohen, 2001; Yeung, Botvinick & Cohen, 2004) suggests the ERN to reflect processes of conflict-monitoring. In fact, the authors are using computational simulations of connectionist models of response conflict to show that error detection respectively the occurrence of the ERN do not imply an explicit comparator mechanism and a direct evaluation of response accuracy.
Nevertheless, a complete exposition of all the simulations and explanations in the work of Botvinick et al. (2001) and Yeung et al. (2004) would go beyond the scope of the present thesis. To gain insight into the rationale of the authors, the section below will briefly describe one of these simulations. Basically, the assumptions are grounded on models composed of "identical processing units, each carrying a real valued activity level, which excite or inhibit one another through weighted connections" (Botvinick et al., 2001, p. 630) of already established paradigms, such as for instance the flanker task (Eriksen & Eriksen, 1974).
A schematic illustration of the connectionist view of information processing on the model of a flanker task is depicted in Figure 7. This model allows the simulation of the performance in a task requiring a key-press reaction that indicates if one of the letters H or S appears in the center of a three letter array, with the flanking letters being the same as the letter in the middle (congruent condition) or differing from the target letter (incongruent condition).
The input layer consists of 6 position-specific letter units (e.g. Hl meaning H left, Hc meaning H center and Hr meaning H right) and bidirectional excitatory weights connect the different layers, whereas the units within each layer are connected by inhibitory links to each other.
Conflict is operationalized through the energy of the response layer (Hopfield, 1982, as cited in Yeung et al., 2004, p.935), meaning that conflict can be calculated with the help of the equation(1):

$$\text{Conflict} = -\sum_{i=1}^{N}\sum_{j=1}^{N} a_i a_j \, w_{ij} \qquad (1)$$

Here, *a* denotes the activity of the unit, *w* denotes the weight of the connection between a pair of units and *i* respectively *j* index the units of interest.
Furthermore, on the basis of equation (1), it is obvious that when one response unit is active and the other inhibited, conflict is low or zero. If there are two or more units active, the degree of conflict is high.

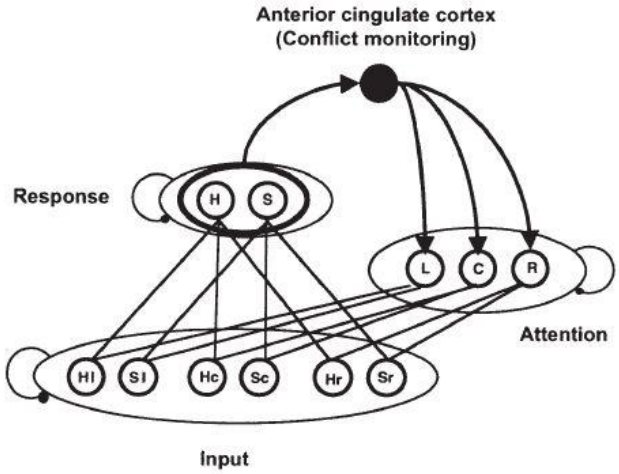


**Fig. 7.** Model of a flanker task with feed-back loop simulating the role of ACC in performance monitoring, Botvinick et al. (2001) as cited in Yeung et al. (2004, p. 935)

The results of the simulations after 10 runs of 1000 trials each, with randomized order of the stimuli, showed an increased conflict mirrored by higher energy levels of the response layer respectively an energy peak following error trials. These results are considered to simulate the occurrence of the ERN component.

To summarize, the theory hypothesizes the ERN to be generated due to the occurrence of conflict after committing errors. Conflict is defined as the simultaneous activation of mutually inhibiting units and it is exactly what happens after an error as the continued processing of stimuli is leading to post-error activation of the correct response in parallel to the activated incorrect response. So, conflict monitoring is hypothesized to be an inherent property of the system, manifesting through the occurrence of the ERN.

The assumptions of the conflict monitoring theory of ERN are supported through findings of several neuroimaging studies, indicating the ACC to be active not only following errors, but also on incongruent correct trials, which also elicit a certain level of response conflict (e.g. Botvinick, Nystrom, Fissell, Carter & Cohen, 1999; Carter, Braver, Barch, Botvinick, Noll & Cohen, 1998; Pardo, Pardo, Janer & Raichle,1990).

In the response selection approach (Holroyd & Coles, 2002), the mesencephalic dopamine (DA) system for reinforcement learning is assumed to generate the ERN. This theory can be summarized in the following manner:

The human nervous system is constituted (among other components) of multiple motor controllers (see Figure 8). The model conceptualizes the ACC to act as a motor control filter, which allows one of those motor controllers to acquire command of the motor system. In this model, the mesencephalic DA system is training the ACC to recognize the best suited motor controller for the given situation through the modulation of the firing rate of its DA neurons.

After an error has occurred, a phasic decrease in the mesencephalic dopaminergic activity follows as the ongoing events are worse than expected. Due to the reduction of the mesencephalic dopaminergic activity, the neurons of the motor area in the ACC disinhibit and generate, in this way, the ERN. Conversely, the consequence of correct responses is a phasic increase in the mesencephalic dopaminergic activity. Moreover, the authors hypothesize the basal ganglia to compute the value and the change in value of the ongoing events, thus assigning it to play the role of an adaptive critic. Findings supporting the reinforcement learning theory of the ERN stem from De Bruijn, Hulstijn, Verkes, Ruigt and Sabbe (2004) who observed increased ERN in healthy subjects after administration of the DA agonist D-amphetamine, which increases DA availability.

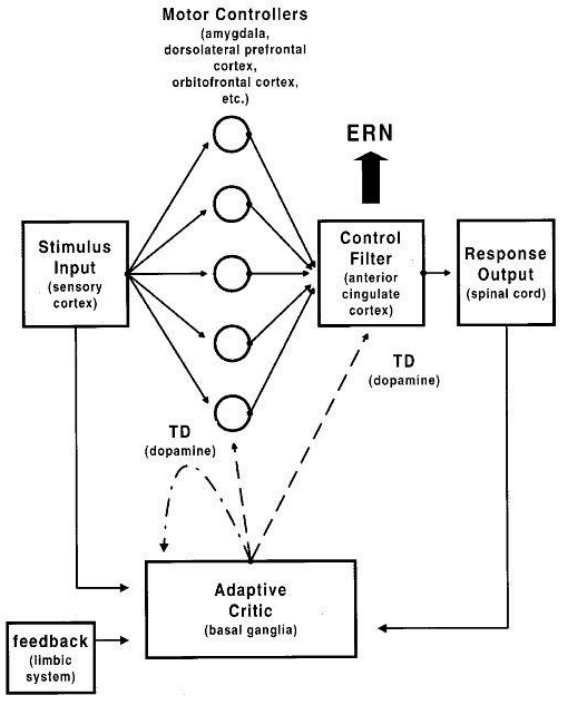


**Fig. 8.** Schematic illustration of the reinforcement learning theory of the ERN (Holroyd & Coles, 2002, p.685) . TD stands for temporal difference error, symbolizing an algorithm often used in computational models to simulate reinforcement learning.

After the previous confrontation with the logic of three ERN-theories, it becomes obvious that these approaches commonly agree on only a few features of the ERN. Still, its occurrence has always been described following an error. On this account, the results of some studies (e.g. Falkenstein, Hoormann, Christ & Hohnsbein, 2000; Scheffers & Coles, 2000; Vidal, Hasbroucq, Grapperon & Bonnet, 2000) which revealed a small ERN-like component after correct trials seem, at first glance, counterintuitive. Nevertheless, several explanations have been proposed to interpret the aforementioned component (also termed as correct response negativity - CRN). Coles et al. (2001) as well as Scheffers and Coles (2000) suggest two conceivable explanations: either measurement artifacts reflecting residual stimulus-related

activity that was not removed in the averaging process or the subject's uncertainty of a correct response.

Last but not least, the possibility of the ERN to reflect alternatively motivational or emotional aspects of error processing should not be left unmentioned.
Vidal et al. (2000) suggested the ERN to represent one of two presumptions: a response evaluation process only secondarily leading to error detection or an emotional process. Gehring et al. (1993) measured enhanced ERN amplitudes for the subjects instructed to react accurate compared to the participants instructed to react fast. Hence, they suggested that "the process manifested by the ERN is influenced by the importance of errors to the subject: The more costly the error, the greater the likelihood or strength of the activity manifested by the ERN" (Gehring et al., p.389). Referring to the above mentioned study, Hajcak, Moser, Yeung and Simons (2005) assumed Gehring and his colleagues(1993) might have made the errors more salient and therefore more negatively valent by emphasizing the accuracy in the instructions. Consequently, Hajcak et al. (2005) investigated the influence of error value on the ERN and found more valuable errors to be associated with an enhanced ERN. This pattern of results has been assessed in absence of behavioral differences, which means that the accuracy of the participants on valuable and less valuable trials did not differ. Therefore, they concluded that their results are consistent with the view that the ERN reflects the motivational significance of errors.

### *3.2.1 ERN and Psychopathology*

To obtain a complete picture of the up to date research on the phenomenon ERN, there is undoubtedly a considerable amount of findings left to be referred to. However, it is beyond the capacity of this thesis to review all the research work related to the ERN. Consequently, while the previous subchapter only mentioned some basic characteristics of the ERN, the present subchapter will focus on findings concerning internalizing disorders (Figure 9), as they are more related to the research question addressed in this study (see Olvet & Hajcak, 2008, for a more detailed review).

Olvet and Hajcak (2008) stated in their review that exacerbated error-sensitivity is one characteristic of internalizing disorders. In this respect, some empirical evidence corroborating their statement refers to data about the ERN in depressed people and in pathologically anxious people. Here are some results supporting the interconnection between error processing and internalizing disorders:
Chiu and Deldin (2007) showed that depressed patients displayed an enhanced ERN compared to non-psychiatric controls, probably reflecting a heightened sensitivity to discrepancies between affective goal states and actual states. In fact, also patients suffering from obsessive compulsive disorder (OCD), a disorder which seems connected with

abnormal action monitoring (Olvet & Hajcak, 2008), are shown to display an increased ERN compared to healthy subjects (Gehring, Himle & Nisenson, 2000). Moreover, children suffering from both OCD and generalized anxiety disorder were also found to generate an increased ERN (Ladouceur, Dahl, Birmaher, Axelson & Ryan 2006; Hajcak, Franklin, Foa & Simons, 2008).

*Excursus*

The (IE) internalizing-externalizing model (Krueger, Chentsova-Dutton, Markon, Goldberg & Ormel, 2003) of mental disorders assumes to explain the correlation-pattern among common psychopathological-syndromes by using two broad dimensions, the internalizing dimension and the externalizing behavior dimension (see Figure 9).

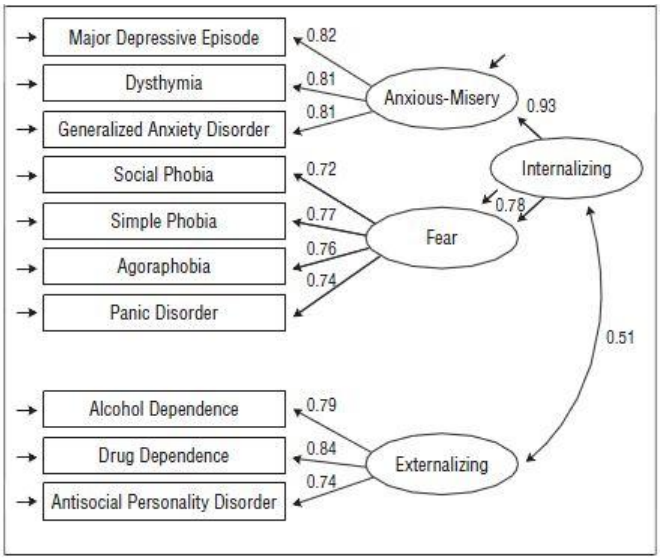


**Fig. 9.** Best-fitting model for US National Comorbidity Survey as described by Krueger (1999, p. 924) based on the results of confirmatory factor analyses of patterns of comorbidity among 10 common mental disorders.

However, the ERN is also reported to be increased in healthy subjects scoring high on an anxiety scale (Hajcak, McDonald & Simons, 2003) as well as on negative affect scales (Hajcak, Mc.Donald & Simons, 2004), thus suggesting personality traits related to pathological anxiety to be connected with enhanced ERNs.

Considering these and a whole series of other similar results, Olvet and Hajcak (2008) argue that the ERN might reflect an underlying condition characteristic to the mentioned disorders, namely negative affect. In the same vein as Hajcak et al. (2008), they even conclude that “the ERN might be a useful endophenotype for internalizing disorders” (Olvet & Hajcak, 2008, p. 1350).

Finally, to provide a more comprehensive idea about the current state of knowledge, it is also necessary to point out that especially the relation between depression and the ERN seems to

be less straightforward than the previously mentioned findings lead to believe, since recent results indicate also a decreased ΔERN[10] as being related to the severity of the symptoms in MDD[11] (Olvet, Klein & Hajcak, 2010). In responding to these various issues, Olvet et al. (2010) proposed a unifying approach and hypothesized a non-linear relation between the ERN and depressive disorder: while mild and moderate depression are related to numerically larger ERN, severe depression is associated with reduced ERN-amplitudes, as the "debilitating apathy and anhedonia found in more severe depression diminish the abnormally large ERN that might typically characterize individuals with depression " (Olvet et al., 2010, p. 33).

### *3.2.2 Mood induction and the ERN*

The effect of short lasting affective modulations on the ERN has been analyzed to a lesser extend in the existing work about the ERN.

Larson, Perlstein, Stigge-Kaufman, Kelly and Dotson (2006) found their subjects in the context of presenting them target stimuli superimposed on a background of positive, negative, or neutral pictures, to display the largest ERN after the trials including pleasant pictures. Larson et al. (2006) interpreted their findings in terms of mismatch occurring when an error is committed in a positive emotional context.

Compton, Carp, Chaddock, Fineman, Quandt and Ratliff (2007) analyzed data indicating a similar pattern. Their participants had to make judgments about the gender of faces varying in emotional expression, which were presented for only 14 ms. They observed that participants high in state anxiety displayed smaller ERNs for angry-face blocks and larger ERNs for happy-face blocks, compared to less anxious participants. To account for these results, Compton et al. (2007) hypothesized anxious participants to have altered expectations for success and failure in the context of happy and angry facial cues and therefore to display greater ERN amplitudes when expectations are violated.

Yet, in the light of apparently opposing results, addressing the issue of mood induction and the ERN does not seem to be that simple.

Wiswede, Münte, Goschke and Rüsseler (2009a) used a flanker-task to evoke an ERN and presented, prior to each flanker stimulus, either a neutral, a pleasant or an unpleasant picture from the International Affective Picture System (IAPS, Lang, Bradley & Cuthbert, 1999). As a consequence, they report that especially during the first part of their experimental manipulation, the presentation of unpleasant IAPS pictures led to increased ERN amplitude compared to the conditions preceded by the presentation of pleasant or neutral IAPS pictures. Thus, they suggest the following possibility to explain the pattern of their data: affect-related changes in dopamine levels together with the noradrenergic system -

[10] The amplitude of erroneous trials minus the amplitude of correct trials
[11] Major Depressive Disorder

presumably mediating through arousal - modulate the ERN, causing enhanced ERN after unpleasant stimuli.

Wiswede, Münte and Rüsseler (2009b) also investigated the ERN after derogative and encouraging feedback related to individual performance levels in a flanker task. The feedback was based on the individual reaction times and was provided every 30 trials during the flanker task. They found higher ERN amplitude after derogatory feedback and interpreted this modulation as a consequence of negative affect.

In a more recent study van Wouwe, Band and Ridderinkhof (2011) reported their subjects to display a reduced ERN after watching a positive film clip and then accomplishing a continuous performance task, compared to the participants in the neutral film-clip condition. The authors understand these results as a consequence of an increase of dopaminergic activity in the striatum following the induced positive affect, which limits the phasic reduction of DA resulting from errors. This, in turn, is leading to a less pronounced dopaminergic error signal to the MFC resulting in a smaller ERN.

According to a literature search, the last up-to-date reported steps in the incremental process of investigating the effect of different induced affective states on the ERN are the findings of Olvet and Hajcak (2011). The authors induced sad, neutral and positive mood using a film clip and a song before asking their participants to complete a flanker-task. Even though the mood induction appeared successful, no ERN differences between the negative, neutral and positive group could be assessed. However, changes in sadness from pre- to post-mood induction were related to increased error-related brain activity. This effect appeared to be larger among individuals scoring higher on neuroticism. Olvet and Hajcak (2011) interpreted this pattern of results by suggesting that neuroticism moderates the relationship between the ERN and changes in sad mood.

Given the findings summarized above, the need of further investigation becomes evident. Therefore, one of the main goals of this thesis is to contribute to the research of the dynamics between mood induction and the ERN by analyzing the effect of induced helplessness on the ERN.

## *3.3 Error Positivity (Pe)*

The positive deflection following the ERN (see Figure 5) has been identified to reach its maximum mostly within the 200ms to 400ms time window and to have a more posterior midline scalp distribution than the ERN (Falkenstein, Hoormann, Hohnsbein & Kleinsorge, 2003; Nieuwenhuis, Ridderinkhof, Blom, Band & Kok 2001; Overbeek, Nieuwenhuis & Ridderinkhof, 2005). The just mentioned phenomenon is regarded as a further error-processing related component and is known under the term “error positivity” (Pe). Unfortunately, the Pe has not been investigated with the same intensity like the ERN has been, and “available data do not allow strong inferences to be drawn about the functional

significance of the Pe" (Overbeek et al., 2005, p. 327). There are, though, some proposals about the functionality of the Pe. Falkenstein et al. (2000) for instance, assume the Pe to reflect additional processing after errors - apart from error detection or response checking - which could represent conscious error recognition, adjustment of response strategies, or the subjective emotional assessment of errors. However, Falkenstein, Willemssen, Hohnsbein and Hielscher (2005) emphasize that all these three possible explanations for the occurrence of the Pe are based on a common precondition, namely the conscious error-recognition. Therefore, they explain the Pe as being "the reflection of the conscious recognition of an error or of further cognitive or affective-motivational processing after having recognized a full error" (Falkenstein et al., p. 305).

Overbeek et al. (2005) conducted a literature review surveying 32 published studies on the topic of the Pe. Here are some statements of their work:

The Pe seems to be very dissimilar to the ERN. Whereas all drugs directly or indirectly affecting the dopaminergic activity in the brain seem to impinge on the ERN, the Pe component is not affected through the administration of such substances. Therefore, they presume that "the dopamine-mediated performance-processes subserved by the MFC [Medial frontal cortex] " (Overbeek et al., 2005, p.321) do not underlie the Pe.

Moreover, young adults display a larger ERN than young children do but the Pe seems to be similar across these two groups. When older adults are compared to younger ones, a reduction of amplitude can be noted as well for the ERN as for the Pe with the older participants displaying reduced amplitudes.

Furthermore, while the patients suffering from OCD have larger ERN compared to healthy controls, no differences with respect to the Pe could be found in this context. Interestingly, effects of personality traits on the ERN and the Pe have been documented (Hajcak et al., 2004) as affecting the two components in opposite directions: while the ERN was higher in subjects scoring high on a negative-affect scale compared to those scoring low on the negative-affect scale, the Pe was smaller in those scoring high on negative- affect compared to those scoring low on negative- affect.

It is an established fact that the Pe is present solely on erroneous trials on which the subjects are aware of the committed errors (Nieuwenhuis et al., 2001). Moreover, Overbeek et al. (2005) summarize that the Pe-amplitude seems to covary not only with the degree of awareness of the error but also with the salience of the error-inducing stimulus.

Finally, as far as the functional significance of the Pe is concerned, Overbeek et al. (2005) propose another hypothesis and assume the Pe component to possibly be a P3b (Rösler, 1983, as cited in Overbeek et al., 2005, p. 326) associated with the motivational significance of the error.

## *3.4 Behavioral correlates of error monitoring*

Two almost self-explanatory behavioral correlates of action-monitoring processes are the reaction times and the accuracy rates. The accuracy rates measure the quality of a completed performance. As far as the reaction times are concerned, there are three types that can be distinguished: the simple reaction times, the recognition reaction times and the choice reaction times. The simple reaction times measure the reaction speed when the appearance of a certain acoustic, visual or audio stimulus must be signaled, the recognition reaction times measure the reaction speed when there are stimuli that should be responded to whereas other stimuli should not be responded to, and choice reaction times refer to the speed of a reaction, which must correspond to a certain stimulus like, for instance, pressing a key (Luce, 1986).

Since the research on error monitoring processes requires tasks which elicit errors, experiments measuring choice reaction times are often the method of choice. There are several established speeded response tasks which can be used for this, such as for instance the arrow version of the flanker-task (Eriksen & Eriksen, 1974). This task consists of five arrowheads being displayed either with all arrowheads pointing in the same direction – congruent condition - (e.g. >>>>>) or with the middle arrow pointing in the opposite direction of its flaking arrowheads – incongruent condition - (e.g. >><>>). Thereby, the participants are instructed to respond to the direction of the middle arrowhead by pressing a corresponding key.

The choice reaction times (CRT) in the flanker paradigm are always found to be increased for incongruent conditions compared to those for congruent trials. Regarding this flanker interference, Sanders and Lamers (2002) found evidence sustaining the view that it might occur because the parallel processing of the target and the incongruent flankers leads to competition among conflicting response tendencies.

Rabbit (1981, as cited in Hajcak; McDonald & Simons, 2003b) reported longer choice reaction-times for correct trials following erroneous trials compared to those following correct responses. This so-called post-error slowing (PES) is supposed to happen due to compensatory actions taken in order to increase the likelihood of correct responses after committed errors and could therefore be interpreted as a behavioral measure of response monitoring (Hajcak et al., 2003b). However, the use of post-error slowing measurements as a marker of cognitive control should be carefully deployed (Notebaert, Houtman, Van Opstal, Gevers, Fias & Verguts, 2009). According to Notebaert and colleagues (2009), the post-error slowing seems to be caused at least partly by the relative infrequency of errors which causes attentional capture. They refer to this hypothesis as the orienting account and support it through findings showing that “post-error slowing was observed when errors were infrequent,

but when errors were frequent, slowing was observed after correct trials" (Notebaert et al., 2009, p. 278).

Against this backdrop, it remains unclear whether the PES can be disentangled of attentional-capture components. Based on previous findings on error-processing (e.g. Olvet and Hajcak, in press; Wiswede et al., 2009a; Wiswede et al., 2009b), we assume the PES to be a behavioral measure of response monitoring.

## *3.5 Sex and error processing*

There is not much research focusing on sex differences in error processing as reflected by the ERN and Pe. Up to now, only one study (Larson, South & Clayson, 2011) investigated these differences in adults. The authors reported increased ERN and Pe amplitude for their male participants compared to their female participants. Thus, they concluded that the variable sex must be also taken into consideration when it comes to researching the emotional and cognitive correlates of performance monitoring.

On the behavioral level, choice reaction times differences (CRT) were also observed, with the female participants achieving longer CRT than males (Larson et al., 2011). Such differences in reaction times were documented also by Der and Deary (2006) who analyzed the data of 7130 participants and detected significant sex differences of simple reaction times and CRT, with females being slower than males.

In the context of sex differences and the quantification of ERP's however, it should be borne in mind that also anatomical aspects such as individual variations in the conductive properties of the volume between the cortex and the scalp surface could influence the outcome of the EEG recordings (Hagemann, Hewig, Walter & Naumann, 2008). More specifically, the greater the electrical resistance of the volume, the more reduced are the surface EEG amplitudes, where the resistance is a function of the conductivity and the thickness of the layers [e.g. cerebrospinal fluid (CSF), skull, scalp]. (Nunez & Shrinivasan, 2006, as cited in Hagemann et al., 2006). Hence, also the following sex related differences in the scalp anatomy must be taken into account:

> The skull of an adult female is, as a rule, lighter and smaller, and its cranial capacity about 10 per cent. less, than that of the male. Its walls are thinner and its muscular ridges less strongly marked; the glabella, superciliary arches, and mastoid processes are less prominent, and the corresponding air sinuses are small or rudimentary. The upper margin of the orbit is sharp, the forehead vertical, the frontal and parietal eminences prominent, and the vault somewhat flattened." (Gray's Anatomy, no date, "The Interior of the Skull" para. 20).

Against this background, even though the overall cranial vault thickness of males is not significantly greater in comparison with females (Lynnerup, Astrup & Sejrsen, 2005; Hatipoglu, Ozcan, Hatipoglu & Yuksel, 2008), it is conceivable that some of the above mentioned sexual dimorphism[12] features of the skull still influence the EEG measurements on the scalp. This might be also the reason why, women generally display larger ERP amplitudes than men (Guillem, Mendrek, Lavoie, Pampoulova & Stip, 2009). However, such results are inconsistent, most likely depending on the site where the ERP's were measured (Guillem et al., 2009). To summarize, when looking for sex differences on ERP level, there are also anatomical and not only functional sources of variation that might act as confounds and have to be taken into consideration.

## *3.6 Research interest of the present thesis*

To sum up, the helplessness state is associated with cognitive, emotional and motivational deficits (Seligman, 1975).

Furthermore, the concept of helplessness is supposed to play an important role in the etiology of the hopelessness depression (Metalsky & Alloy, 1989) and the existing literature documents enhanced ERN in depressed patients (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008).

On the other hand, investigations on the influence of induced, short time emotions on the ERN revealed evidence about larger ERN-amplitudes being displayed in connection with negative feedback about one´s own performance. This modulation has been explained in terms of negative affect (Wiswede et al., 2009b).

Moreover, sex differences in the reaction to depressed mood have been determined (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema, 2002, as cited in Addis, 2008). Likewise, also increased ERN amplitudes have been observed in males compared to females (Larson et al., 2001) but sexual dimorphism aspects should be also considered in this context.

In the light of the above reported findings, the current work will focus on the effects of induced helplessness on the ERN and Pe, already established neural correlates of error processing. Thereby, also the issue of possible sex differences will be addressed.

### *3.6.1 Hypotheses EEG Data*

I. Subjects who become helpless display an increased ERN compared to those subjects who become not helpless (Wiswede et al., 2009a; Wiswede et al., 2009b)

[12] Sexual dimorphism is the difference in morphology between male and female members of the same species (Wikipedia, 2011)

II. Helpless subjects do not display any differences in the Pe time window compared to not helpless subjects (e.g. Falkenstein et al., 2005)

III. There are significant differences of ERN-amplitudes between male and female helpless subjects, with females displaying significantly higher amplitudes compared to males (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema, 2002, as cited in Addis, 2008; Wiswede et al., 2009a; Wiswede et al., 2009b)

### *3.6.2 Hypotheses Behavioral Data*

I. The reaction times and the flanker task error rates are higher for helpless subjects compared to not helpless subjects (Seligman, 1975)

II. The CRT are higher for females compared to males (Der & Deary, 2006; Larson et al., 2011)

III. The flanker task error rates are higher for helpless females compared to helpless males (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema, 2002, as cited in Addis, 2008; Seligman, 1975)

IV. The CRT are higher for incongruent stimuli compared to those for congruent stimuli (e.g. Sanders & Lamers, 2002)

V. A post-error slowing effect can be replicated (e.g. Hajcak et al., 2003b)

# Empirical Part

# 4. Method and Materials

## *4.1 Subjects*

Initially, the sample consisted of 50 healthy persons, 25 females and 25 males. The participants were recruited using an advertisement published in the web portal www.unijobs.at and posters placed in different places in the university building. All persons were paid 20 Euro for their participation.

The subjects were right-handed, had normal or corrected-to-normal vision, did not take any antipsychotic drugs and had no psychiatric-history. In order to ensure the mental health of the subjects, the German version of the screening tool “Structured Clinical Interview questionnaire” (SCID-I, Wittchen, Wunderlich, Gruschwitz & Zaudig, 1996) was conducted prior to the beginning of the experiment. Handedness was assessed using the Edinburgh handedness Inventory (Oldfield, 1971). Moreover, all persons were informed about the procedure used for the EEG-recordings according to the regulations of the Brain Research Laboratory of the University of Vienna and their written informed consent was collected. The experiment was conducted in accordance to federal and local ethic standards.
Finally, either due to a high number of artifact contaminated erroneous trials (2 subjects) or simply due to a too small total amount of committed errors (7 subjects) respectively a too high amount of committed errors (4 subjects), data of only 37 subjects was considered for further analysis. Thus, the final sample consisted of 17 males and 20 females, aged between 19 – 34 years ($M$=25.27, $SD$=3.89). The male and female participants did not differ significantly in age.

## *4.2 Experimental Design*

Before the helplessness induction, the subjects completed the Positive and Negative Affect Schedule (PANAS, Watson, Clark & Tellegen, 1988). Then, a helplessness training was conducted by withdrawing the control of the subjects in a reasoning task. Between the helplessness induction and the following flanker task, the subjects again completed the PANAS (Watson et al., 1988) as well as a questionnaire tapping helplessness (Bauer, Pripfl, Lamm, Prainsack & Taylor, 2003). Furthermore, after the flanker task, the Attributionsstilfragebogen für Erwachsene (ASF-E, Poppe, Stiensmeier-Pelster & Pelster, 2005) tapping attributional style, as well as the German version of the Interpersonal Reactivity Index [Saarbrückener Persönlichkeitsfragebogen (SPF), Paulus, 2009] were completed. The ASF-E as well as the SPF questionnaires were not relevant for the present thesis.

## *4.3 Procedure and Task*

The subjects were seated about 60 – 70 centimeters in front of a 19'' CRT (Cathode ray tube) monitor. As the recordings took place in two different laboratories, the types of monitors used were Sony-Multiscan G520 and Sony GDM-F520. Due to technical problems, the participants had to use either a computer keyboard or a response box with five buttons (Psychology Software Tools, Inc., Sharpsburg, USA) in order to type in their responses. The stimuli were presented and synchronized with the EEG-data collection with the help of the software E-prime 2.0 (Psychology Software Tool, Inc., Sharpsburg, USA).

The induction of helplessness occurred in a preceding experiment by means of cognitive reasoning tasks or, more specifically, by the presentation of 60 series of numbers (Czapla, 2011). The stimuli in the helplessness induction procedure were grouped in 2 blocks, each block being made of 30 series of numbers. In the first block, 24 Items were solvable and 6 unsolvable whereas in the second block 6 stimuli were solvable and 24 unsolvable.

Before and after completing the reasoning task, the questionnaires mentioned in the previous sections were filled in. Then, the participants were given following verbal instructions:

*"You will now perform a reaction-speed task. Thereby, please try to move as little as possible and to blink as little as possible during the task. You will have 7 breaks whose length will depend on you and during which you can move your head, legs or hands if you need to.*

*The task consists of 5 arrowheads that are going to be displayed on the screen for a very short time. Please concentrate only at the arrowhead in the middle and press the corresponding key depending on whether it points to the left or to the right. Please try to be as quick as possible and to react accurately."*

Afterwards, an adapted, speeded arrowhead version of the Erikson Flanker Task (Eriksen & Eriksen, 1974) consisting of 20 training items was presented. Prior to the training session, all verbal instructions were also presented on the screen in written form. The written instructions indicated also which keys had to be pressed for the arrowheads pointing to the left or to the right. After completing the 20 training items, the subjects were informed - in written form on the monitor - that the training session was over and the real experiment consisting of 400 items is about to begin following button press.

Subsequently, an adapted, speeded arrowhead version of the Erikson Flanker Task (Eriksen & Eriksen, 1974) consisting of 400 Items grouped in 8 blocks was presented. The timeline of the Flanker task can be described in the following manner:

I. Fixation cross 0 - 1000ms
II. 1000 ms -1100 ms flankers without the target arrow in the middle
III. 1100 ms - 1130ms flankers full, with the target arrow in the middle
IV. 1130 ms - max. 2000 ms black screen during response registration
V. 550 ms black screen after the response

VI. Inter Stimulus Intervall (ISI) 450 ms - 640 ms, fixation cross

Figure 10 depicts the timeline of the trials. Furthermore, following combinations of arrowheads were possible:

| | | |
|---|---|---|
| a) | **<<<<<** | congruent stimulus |
| b) | **>>>>>** | congruent stimulus |
| c) | **<<><<** | incongruent stimulus |
| d) | **>><>>** | incongruent stimulus |

Altogether, the entire flanker task procedure (including the verbal instructions and the breaks) lasted approximately 30 minutes.

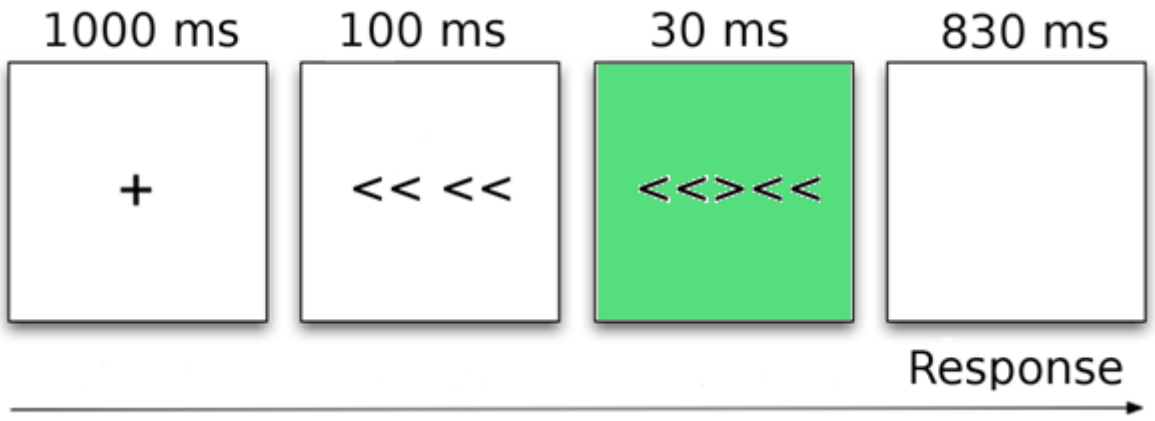


**Fig. 10** Timeline of a flanker trial. Hereby, only one of 4 possible stimuli is depicted in green color for illustrative purposes. The ISI varied between 450ms - 640ms.

## *4.4 EEG Recordings*

Scalp potentials were recorded from 61 Ag/AgCl electrodes embedded in an elastic cap (EASYCAP GmbH, Herrsching, Germany, model M10) and equidistantly spread over the entire scalp. Further electrodes were placed below and above the left eye and at the outer canthi of each eye in order to facilitate the recording of an electro-oculogram (EOG).

All EEG-channels were referenced to a non-encephalic sternovertebral reference (Stephenson & Gibbs, 1951) constituted of electrodes placed on the vertebra prominens and on the sternal end of the right clavicula. The ground electrode was fixed on the forehead.

The impedances of the electrodes were measured by a manual impedance meter and kept below 3kΩ by carefully scratching the skin of the participants with a single-use sterile needle

(Picton & Hillyard, 1972) and filling into each electrode degassed electrode-gel (Electrode-Cap International, Inc., Eaton/Ohio; USA).
The recorded signals were within a frequency range of 0.016 to 125 Hz and were sampled at 250 Hz for digital storage. Moreover, the EEG recordings took place using an EEG AC-amplifier with a time constant of 10 sec (Ing. Kurt Zickler GmbH, Pfaffstätten, Austria).

## *4.5 Questionnaires*

### *4.5.1 Positive and Negative Affect Schedule (PANAS, Watson, Clark & Tellegen, 1988)*

The PANAS questionnaire consists of two scales, the negative affect scale (NA) and the positive affect Scale (PA), with each scale containing 10 Items. Watson et al. (1988) defined these scales as follows:

> (…) High PA is a state of high energy, full concentration, and pleasurable engagement, whereas low PA is characterized by sadness and lethargy. In contrast, Negative Affect (NA) is a general dimension of subjective distress and unpleasurable engagement that subsumes a variety of aversive mood states, including anger, contempt, disgust, guilt, fear, and nervousness, with low NA being a state of calmness and serenity. (Watson et al., 1988, p. 1063)

In the present work, the German version of PANAS was used to assess the data of the participants. Thereby, subjects responded on a five-point scale with a range from 1 (not at all) to 5 (extremely). All Items refer to how a person completing the questionnaire has felt in the last minutes.

### *4.5.2 Helplessness assessing questionnaire (Bauer et al., 2003)*

This questionnaire assesses the feelings of individuals after being exposed to a helplessness training comprising a reasoning task made out of solvable and unsolvable series of numbers. It consists of 11 questions, which can be answered on a 5-point scale and a twelfth question with an open answering format. All questions deal with aspects such as experienced control during the helplessness training, the motivation to succeed, the feelings during the helplessness training such as depression, demotivation, passivity, despondency, depression or aggression and with the personal importance to succeed.

# 5. Data Analysis

## *5.1 Blink and EOG movement correction*

Prior to the EEG recordings for the Flanker task, EOG calibration trials were conducted, whereby each participant was asked to perform guided vertical and horizontal eye-movements.

Based on these calibration trials, individual weights were calculated subject- and channel-wise, representing the covariance between each EEG channel and the EOG divided by the variance within the EOG channels (Bauer & Lauber, 1979). Those weights were used to remove the eye movement artifacts, by successively subtracting the weighted horizontal and vertical EOG data from each EEG channel, trial by trial.

The eye blink artifacts were also taken into account as a template matching procedure was used to identify the time windows containing blinks. Then, the identified time windows were corrected using blink correction coefficients that have been calculated for each EEG channel with help of linear regression (c.f. Vitouch, Bauer, Gittler, Leodolter & Leodolter, 1997; Lamm, Fischmeister & Bauer, 2005, for detailed information).

## *5.2 EEG Data*

The data analysis was conducted off-line, using EEGLAB 6.03b (Delorme & Makeig, 2004), a software implemented in Matlab 7.5.0 (The MathWorks Inc., Natick, USA).

Thereby, the EEG-data was low-pass filtered with a cut-off frequency of 30 Hz (roll-off 6 dB per octave). Further aspects of the performed analysis were the exclusion of the two pre-auricular channels and the extraction of EEG epochs lasting 650 ms, with a baseline starting 100 ms before response onset. Then, as the subjects either responded correctly or pressed the wrong key, datasets for following conditions were merged by using the corresponding epochs:

(1) Congruent correct
(2) Congruent error
(3) Incongruent correct
(4) Incongruent error

For the detection and exclusion of artifactual trials, a semi-automatic artifact removal procedure was applied to the data. First, all trials with a voltage value exceeding the threshold of ± 75 µV and/or a linear voltage drift with a slope exceeding the threshold of 50 µV were marked with help of the artifact-rejection option in EEGLAB. After a visual inspection, the labeled trials were rejected, together with other trials that were contaminated by muscular or movement artifacts and were detected via detailed visual inspection of all trials. In the final sample, the data of the participants contained a minimum of 5 errors

(Hajcak & Simons, 2008; Amodio, Harmon-Jones; Devine, Curtin, Hartley & Covert, 2004) and a maximum of 33 errors (*M*=12.43; *SD*=7.87).

However, some of the participants did not commit any errors on congruent trials at all. Therefore, the datasets for congruent and incongruent errors respectively for congruent and incongruent correct trials had to be merged together. The following datasets resulted from the merging procedure for each subject:

(1) Erroneous trials
(2) Correct trials

Moreover, the artifact-corrected data were averaged for each of the above listed two conditions per participant and for all subjects per condition (grand averages). Thus, in order to enable visual inspection of the relevant data, the grand averages for the following conditions were calculated:

(1) helpless/male/error;
(2) helpless/female/ error;
(3) not helpless/female/error;
(4) not helpless/male/error;
(5) helpless/male/correct
(6) helpless/female/correct;
(7) not helpless/female/correct;
(8) not helpless/male/correct;
(9) helpless/error;
(10) helpless/correct;
(11) not helpless/error;
(12) not helpless/correct;

In order to assess the peak amplitude-values and the latencies of the peak amplitude-values for the ERN and the Pe, each averaged dataset per subject was visually inspected by using a peak-detection tool (BRL peak finder v.0.1b) implemented in EEGLAB. Both components were investigated at location Cz. While the ERN was quantified as the most negative peak occurring in the time window 10 ms to 120 ms post response, the Pe was quantified as the most positive data point in the window between 200 ms to 400 ms following response execution. The time windows for the ERN and Pe measurement were chosen after the visual inspection of all averages per subject.

For the correct trials averages of three participants, no peaks could be detected in the time window 10 ms to 120 ms post response. Therefore, the sampling points corresponding to the peak amplitude-values of their erroneous trials in the time window 10 ms to 120 ms post response were determined and subsequently applied also on their correct trials data. Thereby, the recorded amplitude values at exactly those sampling points were collected.

One additional aspect was the comparability of the erroneous trials data of helpless vs. not helpless participants. Therefore, we also separately z-transformed the data for males and for females, in order to eliminate sex dimorphism confounds and to be able to compare the measured values with respect to their relative position in the distribution.

## *5.3 Behavioral Data*

As far as the behavioral data are concerned, the performance of the participants has been assessed on the basis of error rates and choice reaction times (CRT). Thus global CRT, CRT for post-erroneous trials, CRT for post matched-correct trials, CRT for all correct and all erroneous trials as well as for the incongruent and congruent trials were obtained for further statistical analysis.

The post matched-correct trials were determined in order to examine the post-error slowing effect (PES) by using a similar procedure as in Wiswede et al. (2009) and Hajcak, McDonald and Simons (2003). The just mentioned procedure aims to exclude the possibility that the slowdown effect is determined by means of a simple regression toward the mean, as error trials are typically faster than correct ones. In view of these considerations, the slowdown effect was determined for post-error trials versus trials following a subset of correct trials which were matched to the error trials in terms of total number and reaction times. In this process, the fastest correct trials for each subject were chosen.

Furthermore, only responses given within 200 ms to 800 ms after the flanker stimulus onset were included in the subsequent data analysis (Wiswede et al., 2009).

## *5.4 The operationalization of the helplessness state*

For the operationalization of helplessness, the scores of only three questions of the helplessness questionnaire were used, as a principal component analysis of the own data and the data of other recent studies on helplessness (Deichstätter, in work) indicated three components of interest inquired by the following enumerated questions:

*" How did you feel after not being able to solve the presented Items, in the long term ?"*

***(1) "Did you feel demotivated?"***

***(2) "Did you feel passive?"***

***(3) "Did you become despondent?"***

The assignment to one of the three initially possible helplessness states was based on two cut-off points, with people reaching a total score of 6 to 9 points being termed as not helpless, participants attaining 10 to 11 points being termed as middle-helpless and subjects achieving a total score of 12 to 15 points being assigned to the helpless group.

At the beginning, this decision was based on general considerations regarding the quality assurance of the induced helplessness state. After performing the appropriate statistical analysis though, we found extreme group comparisons to be more suitable for assessing our research question. Our results indicated that not helpless and middle helpless participants reported significant differences in feelings of demotivation and despondency but that their ratings didn't reveal significant differences in the experienced passivity. Therefore, as the

passivity is a key aspect when dealing with helplessness (Seligman, 1975), we assumed the middle-helpless group to have experienced a kind of negatively valenced affect but no helplessness. Nevertheless, despite the fact that extreme group comparisons seemed more appropriate in context with the assessment of helplessness effects on error processing, we report also the results of the analyses including the three-leveled variable helplessness in the appendix.

# 6. Statistical analysis

## *6.1 Helplessness questionnaire*

The nonparametric Kolmogorov–Smirnov tests applied to the helplessness questionnaire data were significant, which is why we investigated the skew and kurtosis values of the relevant variables. Furthermore, as all the values fell within the range +1 to -1, we concluded that the data were reasonably close to normality in order to be analyzed using parametric tests. (George & Mallery, 2003, as cited in Meyers, Gamst & Guarino, 2006). Thus, we computed four two-way independent ANOVA's, all of them having the same between-subject factors *helplessness* (not helpless/helpless) and sex. The dependent variables were *Despondency-Scores*, *Passivity-Scores*, *Demotivation-Scores* and *Total Helplessness-Scores.* The assumption of homogeneity of variances was met in all cases.

Note that these ANOVA models were also computed with the three-leveled between-subject factor *helplessness*. However, the test-values of the latter ANOVA's are going to be reported only in the appendix in table-format.

### *6.1.1 PANAS questionnaire*

First, the normality of the data was confirmed by means of nonparametric Kolmogorov–Smirnov-tests. Then, a four-way mixed-design ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the within-subject factors *Positive Affect* (PA before/PA after) and *Negative Affect* (NA before/NA after) was conducted. For the NA after scores, the Levene tests indicated statistical significance. Therefore, the ANOVA was recomputed with log-transformed data. However, the findings of the ANOVA applied to the transformed data did not differ from the findings of the ANOVA conducted with the untransformed data on the interpretational level. This is why only the results of the latter one will be reported. Furthermore, the sphericity assumption was not an issue as our within-subjects factors were two-leveled.

The test-values of the ANOVA with the three-leveled between-subject factor *helplessness* are reported in the appendix. Finally, significance level of $\alpha = 0.05$ was used for all analyses.

## 6.2 Behavioral Data

### 6.2.1 Error rates

Due to the exclusion of the trials with CRT out of the range of 200 ms to 800 ms, the error rates of each participant were calculated relative to the own total amount of trials left after the filtering procedure, which differed from participant to participant. The acquired accuracy data consisted of global error rates per subject as well as the error rates of the subjects for congruent and incongruent stimuli. Three two-way independent ANOVA's were computed, all of them having the same between-subject factors *helplessness* (not helpless/helpless) and sex. The dependent variables were *global error rates*, *congruent error rates* respectively *incongruent error rates*. The normality of these data was confirmed by means of nonparametric Kolmogorov–Smirnov-tests.

As the assumption of homogeneity of variances was not met for the dependent variable *global error rates*, arcsine transformed error-rates, as suggested by Neter, Wasserman and Kutner (1985, as cited in Larson, Fair, Good & Baldwin, 2010,) were used to recompute the ANOVA affected by the above mentioned violation. However, since heterogeneity of variances was assessed even for the transformed data, the alpha level was adjusted to 0.01 in order to control for Type I errors (Tabachnick & Fidell, 2007). Otherwise, significance level of $\alpha = 0.05$ was used for all other analyses.

Furthermore, since the findings of the ANOVA applied to the transformed data did not differ from the findings of the ANOVA conducted with the untransformed data on the interpretational level, only the results of the latter one will be reported.

Also a paired t-test to explore differences of congruent error rates vs. incongruent error rates was performed.

Finally, all three ANOVA's described above were conducted also with the three-leveled between-subject factor *helplessness*. The test-values of these ANOVA's are going to be reported in the appendix in table-format.

### 6.2.2 Reaction times

For the global CRTs, the congruent trials, incongruent trials respectively also for the post-erroneous trials, post matched-correct trials, correct and erroneous trials, individual median-CRTs were calculated. The decision to use individual CRT-medians was taken following the example of Ratcliff (1993, as cited in Larson, Fair, Good & Baldwin, 2010, p. 418) who proposed the use of median reaction times (RT) in consideration of the positive skew frequently associated with RT data and to reduce the influence of outliers. The normality of the resulting data was confirmed by means of nonparametric Kolmogorov–Smirnov-tests. In order to assess the questions motivating this work, following statistical procedures have been conducted:

I. a three-way mixed-design ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the within-subject factors *congruency* (CRTs congruent trials/ CRTs incongruent trials)
II. a three-way mixed-design ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the within-subject factors *correctness* (CRTs correct trials/ CRTs erroneous trials)
III. a three-way mixed-design ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the within-subject factor *post-error slowing* (CRTs post-erroneous trials/CRTs post matched-correct trials)
IV. a two-way independent ANOVA using the dependent variable *global CRTs* and the independent variables *helplessness* (helpless/not helpless) and *sex*

For the global CRTs, the Levene tests indicated statistical significance even for the log-transformed data. Therefore, the alpha level was adjusted to 0.01 in order to control for Type I errors (Tabachnick & Fidell, 2007). Furthermore, since the findings of the ANOVA applied to the transformed global CRT did not differ from the findings of the ANOVA conducted on untransformed data on the interpretational level, only the results of the latter one will be reported.

Moreover, the Levene tests indicated again statistical significance as well for the raw as for the log-transformed data of the correct trials. As a consequence, the alpha level of the involved ANOVA was adjusted to 0.01 in order to control for Type I errors (Tabachnick & Fidell, 2007). Besides that, since the findings of the involved ANOVA (as mentioned in point II) conducted on log-transformed data did not differ from the findings of the ANOVA conducted on untransformed data on the interpretational level, only the results of the latter one will be reported.

As far as the CRTs of the congruent and incongruent trials are concerned, the applied log-transformation did not entirely stabilize the heterogeneity of the data. Additionally, the ANOVA findings of the log-transformed data differed from the ANOVA findings of the untransformed data also on the interpretational level. In addressing these issues, we focused on the ANOVA of the log transformed data - the values of which will be reported, together with the resulting back-transformed means and confidence intervals (CI) (Bland & Altman, 1996) - and we adjusted the alpha level to 0.01 in order to control for Type I errors (Tabachnick & Fidell, 2007).

Aside from that, the sphericity assumption was not an issue for any of the mixed-design ANOVAs since the whithin-subject factors were two-leveled.

For the remaining analysis as mentioned at point III, we used significance level of $\alpha = 0.05$.

All ANOVA's described above were conducted also with the three-leveled between-subject factor *helplessness*. The test-values of these ANOVA's are going to be reported in the appendix in table-format.

## *6.3 Correlation Analyses*

We computed correlations between questionnaires data, behavioral data and ERP data as follows:

I. Pearson Correlations between:
*PA after* and *Total Helplessness Score*; *PA after* and *ERN Amplitude; PA after* and *ΔERN; PA after* and *Pe Amplitude; PA after* and *Global CRT; PA After* and *PES CRT; PA after* and *Error rates; Global CRT* and *ERN Amplitude; Global CRT* and *ΔERN; Global CRT* and *Pe Amplitude; PES CRT* and *ERN Amplitude; PES CRT* and *ΔERN; PES CRT* and *Pe Amplitude; Error rates* and *ERN Amplitude; Error rates* and *ΔERN; Error rates* and *Pe Amplitude; Total Helplessness Score* and *ERN Amplitude*; Total Helplessness Score* and *ΔERN*; Total Helplessness Score* and *Pe Amplitude*, Total Helplessness Score* and *Global CRT*; Total Helplessness Score* and *PES CRT; Total Helplessness Score* and *Error rates*;*

II. Spearman Correlations (*NA after* data were not normally distributed) between:
*NA after* and *Total Helplessness Score*; *NA after* and *ERN Amplitude*; *NA after* and *Δ ERN*; *NA after* and *PE Amplitude*; *NA after* and *Global CRT*; *NA after* and *PES CRT*; *NA after* and *Error rates*;

The significance tests for the correlations of the variable-pairs marked with (*) were conducted one-tailed whereas the significance tests for the correlations of the unmarked variable-pairs were performed two-tailed. Furthermore, all correlations enumerated above were calculated also for the entire sample (N=37), including the middle helpless group. The latter are reported in the appendix in table-format.

## *6.4 ERP-Data*

With regard to the hypotheses of the present thesis, the measured parameters – peak amplitudes and latencies - were subjected to following statistical procedures:

### *6.4.1 Time window 10 ms to 120 ms post response*

I. a three-way mixed-design ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the within-subject factor *correctness* (amplitudes correct-trials/amplitudes erroneous trials). Therefore, also a priori planed linear contrasts were conducted. Hence, we compared the error trials amplitudes and the correct trials amplitudes of helpless vs. not helpless subjects respectively the

erroneous trials amplitudes and the correct trials amplitudes of males relative to females via t-tests. Both t-tests pairs were conducted using Bonferroni adjusted alpha levels of 0.025 (0.05/2) per test.

II. a two-way mixed-design ANOVA with separately z-transformed amplitudes for males and females having the within-subject factor *correctness* (z-transformed amplitudes correct trials/z-transformed amplitudes error trials) and the between-subject factor *helplessness* (helpless/not helpless). Therefore, also planed linear contrasts were conducted. Thus, we compared the z-transformed error-trials amplitudes of helpless vs. not helpless subjects via t-tests.

III. a two-way independent ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the dependent variable *latency* for erroneous trials

*6.4.2 Time window 200 ms to 400 ms post response*

I. a three-way mixed-design ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the within-subject factor *correctness* (Amplitudes correct-trials/ Amplitudes erroneous trials) for the time window 200 ms to 400 ms post response

II. a two-way independent ANOVA with the between-subject factors *helplessness* (helpless/not helpless) × *sex* and the dependent variable *latency* for erroneous trials in the time window 200 ms to 400 ms post response

As a part of the statistical investigation, the normality of the ERP-data was confirmed with the help of nonparametric Kolmogorov–Smirnov tests and the homogeneity of variances assumption was confirmed by using Levene-tests. With the exception of the Bonferroni adjustement mentioned in the subchapter 6.3.1 point I, significance level of $\alpha = 0.05$ was used for all analyses. Moreover, since our within-subject factors were only two-leveled, the assumption of sphericity was not an issue we had to deal with.

With the exception of point II in subchapter 6.4.1, all ANOVA's described above were conducted also with the three-leveled between-subject factor *helplessness*. The test-values of these ANOVA's are going to be reported in the appendix in table-format.

# 7. Results

## *7.1 Descriptive statistics of questionnaires-data*

In total, the sample considered for preliminary statistical analysis consisted of 37 persons, 17 males and 20 females. Of these participants, 10 became not helpless and 13 became helpless. The remaining 14 participants were classified as middle-helpless and excluded from further interpretational work. Therefore, the calculations including the entire sample data respectively the 3-leveled variable Helplessness are presented only in the Appendix, in table format.

Furthermore, 40 % of the not helpless and 70% of the helpless participants were females. A chi-square test indicated no significant differences in the distribution of helplessness depending on sex ($\chi^2$= 1.965, $p$=.161, $df$ =1).

## *7.2 Helplessness questionnaire*

All four ANOVAs revealed significant main effects of ***helplessness*** for the variables in question, *Despondency* $F(1,19) = 25.394$, $p<.001$, $\eta^2 = .572$ ; *Demotivation*, $F(1,19) = 19.636$, $p<.001$, $\eta^2 = .508$; *Passivity*, $F(1,19) = 14.881$, $p = .001$, $\eta^2 = .439$ and the *Total Helplessness-Score*, $F(1,19) = 100.374$, $p<.001$, $\eta^2 = .841$. These results attest significant differences in *Despondency-*, *Demotivation-*, *Passivity-* and *Total Helplessness-Scores* between helpless and not helpless participants. Furthermore, all above mentioned ANOVAs showed no significant main effect of ***sex*** for the variables in question, *Despondency*, $F(1,19) = 0.002$, $p = .969$*; Demotivation,* $F(1,19) = 0.785$, $p = .387$*; Passivity,* $F(1,19) = 0.019$, $p = .892$; and the *Total Helplessness-Score*, $F(1,19) = 0.217$, $p = .647$, indicating no significant differences between male and female subjects with respect to those reported *Despondency-*, *Demotivation-*, *Passivity-* and *Total Helplessness-Scores*. Finally, all interactions of the ANOVAs applied to the helplessness questionnaire data were not significant, with all p's>.635

## *7.3 PANAS Questionnaire*

The mixed-design ANOVA yielded a significant effect of ***PA*** [$F(1,19)=62.182$, $p<.001$, $\eta^2=.766$], denoting the fact that ***PA*** scores were lower after the helplessness induction. Otherwise, the results of all main effects were not significant [$F(1,19)=0.339$, $p=.567$, for ***NA***; $F(1,19)=0.495$, $p=.490$, for ***helplessness***; $F(1,19)=0.240$, $p=.630$ for ***sex;***]. Furthermore, also a significant interaction ***PA×NA*** has been identified [$F(1,19)=7.045$, $p=.016$, $\eta^2=.270$], which mirrors following mood dynamics in the experiment: whereas the ***NA*** scores of the participants increased after the mood induction procedure, the ***PA*** scores of the participants decreased after the helplessness inducing treatment (see also Figure 11).

The interaction ***helplessness* × *sex*** [$F(1,19)=3.519$, $p=.076$, $\eta^2=.156$] reached trend-level significance, indicating that not helpless females tended to achieve higher overall scores compared to not helpless males whereas helpless females tended to make lower overall ratings (***NA*** and ***PA***) relative to helpless men. For the remaining interactions all *p's* >.155.

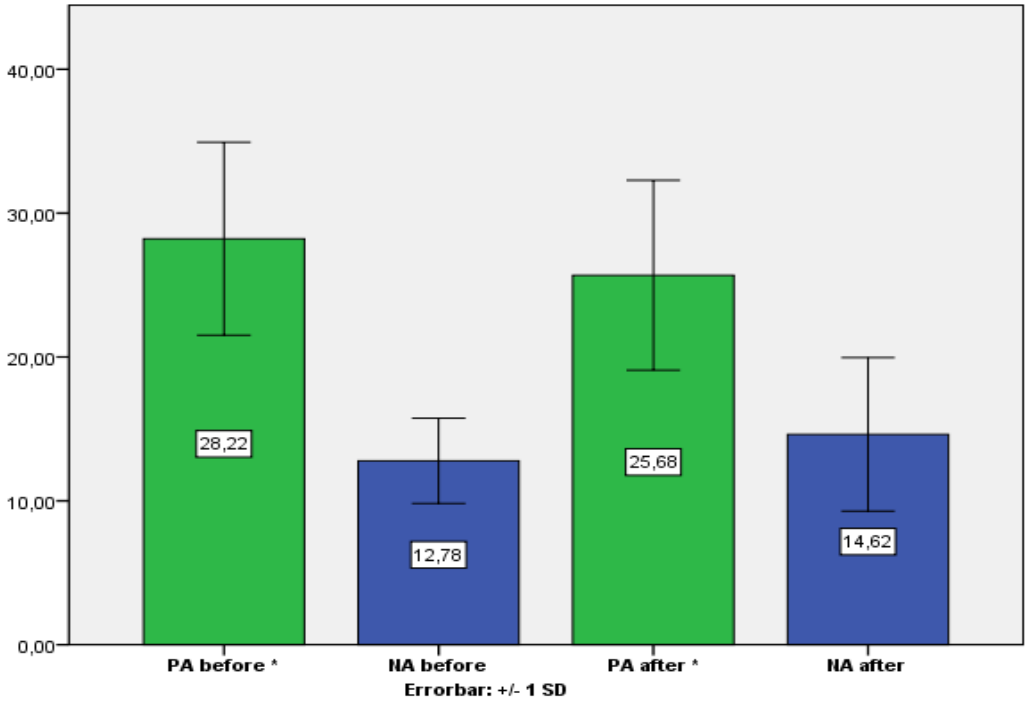


**Fig. 11** Mean scores of Positive and Negative Affect, measured before and after the helplessness treatment. The asterisks indicate significant results and the error bars represent +/- 1 SD.

## *7.4 Behavioral Data*

### *7.4.1 Error rates*

Analysis of global error rates showed neither a significant effect for ***helplessness*** [$F(1,19) = 0.674$, $p = .422$] nor for ***sex*** [$F(1,19) = 0.003$, $p = .955$]. Also no significant interaction ***helplessness* × *sex*** [$F(1,19) = 0.054$, $p = .819$] was observed. These results indicate that helplessness state and sex did not influence the quality of the global accuracy data.

Analysis of congruent error rates also did not reveal any significant results [***helplessness***, $F(1,19) = 0.185$, $p = .672$; ***sex***, $F(1,19) = 1.630$, $p = .217$; interaction ***helplessness* × *sex***, $F(1,19) = 0.147$, $p = .706$].

The ANOVA for incongruent error rates likewise yielded no significant results [***helplessness***, $F(1,19) = 0.185$, $p = .672$; ***sex***, $F(1,19) = 1.630$, $p = .217$; interaction ***helplessness* × *sex***, $F(1,19) = 0.147$, $p = .706$]. Therefore, it can be stated that neither the helplessness state nor

the sex of the participants seemed to influence the amount of committed incongruent or congruent errors. Table 1 reproduces all error rates data.

Finally, a paired t-test determined significant differences between error types, with the incongruent errors being committed significantly more often than congruent errors [$t(22)= -28.385$, $p<.001$].

| | **Helplessness** | | | | | |
|---|---|---|---|---|---|---|
| | non-helpless | | | helpless | | |
| | **Sex** | | **Total** | **Sex** | | **Total** |
| | male | female | | male | female | |
| **Global error rates *** | 0.04 (±0.02) | 0.05 (±0.02) | 0.05 (±0.02) | 0.06 (±0.03) | 0.06 (±0.04) | 0.06 (±0.03) |
| **Error rates incongruent trials **** | 0.96 (±0.06) | 0.93 (±0.05) | 0.95 (±0.05) | 0.99 (±0.02) | 0.93 (±0.11) | 0.95 (±0.09) |
| **Error rates congruent trials ***** | 0.04 (±0.06) | 0.07 (±0.05) | 0.05 (±0.05) | 0.01(±0.02) | 0.07 (±0.11) | 0.05 (±0.09) |

**Table 1.** Means of error rates (%) for congruent and incongruent trials as well as for the global error rates. The parenthesized values represent the SD of the data

***** *Note that the global error rates were calculated relative to the total number of trials per subject*

*** *** Note that the congruent and incongruent error rates were computed only relative to the total amount of erroneous trials per participant*

### *7.4.2 Reaction times*

Looking at the global reaction time performances, no significant effects emerged for ***helplessness*** [$F(1,19) = 0.13$, $p = .912$] or ***sex*** [$F(1,33) = 2.359$, $p = .141$] and no significant ***helplessness × sex*** interaction could be detected [$F(1,19) = 0.229$, $p = .637$].

For congruency, analysis of reaction times revealed a significant effect of ***congruency*** [$F(1,19) = 621.152$, $p < .001$, $\eta^2 = .970$], indicating that all subjects achieved significantly longer CRTs for incongruent trials compared to the CRTs of congruent trials. Moreover, no significant effect of ***helplessness*** [$F(1,19) = 0.013$, $p = .911$] or ***sex*** [$F(1,19) = 2.009$, $p = .173$] could be measured, meaning that neither distinct helplessness-states nor sex could cause significant differences in the reaction-time performance of the participants for congruent and incongruent trials. Finally, no significant interactions were observed [all p values above $p > .259$]. Table 2 reproduces the means and SD for these data.

Regarding correctness, a significant effect of ***correctness*** [$F(1,19) = 68.224$, $p<.001$, $\eta^2 = .782$] was identified, showing that the correct trials of the participants lasted significantly longer than their erroneous trials. No significant effects of ***helplessness*** [$F(1,19) = 0.011$, $p = .918$] or ***sex*** [$F(1,19) = 2.197$, $p = .155$] were ascertained, which reveals that all participants, regardless of their sex or helplessness state, needed more time to react correctly than to commit errors. None of the observed interactions reached statistical significance [all p values above $p > .527$].

| | Helpless | | | | Total |
|---|---|---|---|---|---|
| | Males | | Females | | |
| | Mean | [CI] | Mean | [CI] | Mean [CI] |
| **Congruent CRT** | 312.49 | [250.05; 390.52] | 335.86 | [296.21; 380.80] | 328.48 [299.19; 360.64] |
| **Incongruent CRT** | 401.97 | [352.94; 457.83] | 427.81 | [383.98; 476.70] | 419.72 [389.04; 452.77] |
| | **Not Helpless** | | | | |
| | Males | | Females | | |
| | Mean | [CI] | Mean | [CI] | |
| **Congruent CRT** | 309.63 | [272.70; 351.56] | 344.98 | [227.71; 522.69] | 323.30 [283.38; 368.89] |
| **Incongruent CRT** | 388.38 | [353.57; 426.58] | 448.67 | [310.34; 648.65] | 411.45 [365.11; 463.68] |

**Table 2.** Back-transformed means and confidence intervals [CI] of helpless respectively not helpless male and female participants for congruent and incongruent stimuli (Bland & Altman, 1996)

Further analysis revealed a significant **PES** effect [$F(1,19) = 21.734$, $p < .001$, $\eta^2$ .534], showing that trials subsequent to error trials go along with slower CRTs in comparison with CRTs of post matched-correct trials. However, there was no influence of ***helplessness*** [$F(1,19) = 0.132$, $p = .721$] but a trend for a ***sex*** *effect* [$F(1,19) = 4.087$, $p = .058$, $\eta^2$ =.177] was observed. The latter indicated differences between the overall CRT-performance of males and females, with females achieving slower CRTs relative to males. In addition to this, no significant interactions have been detected [all p values above $p > .111$]. Table 3 reports the PES- data.

| | **Helplessness** | | | | | |
|---|---|---|---|---|---|---|
| | not helpless | | | helpless | | |
| | **Sex** | | **Total** | **Sex** | | **Total** |
| | male | female | | male | female | |
| **CRT post error trials** | 361.7(±39.55) | 430.5(±94.55) | 389.2(±71.48) | 348.7(±53.47) | 403.0(±73.24) | 386.3(±70.51) |
| **CRT post matched corr. trials** | 332.5(±44.54) | 384.5(±93.31) | 353.3(±68.75) | 331.0(±54.92) | 385.2(±64.08) | 368.6(±64.59) |
| **Global CRT** | 351.8(±36.56) | 405.0(±93.68) | 373.1(±66.49) | 361.5(±45.46) | 389.4(±60.04) | 380.8(±55.67) |
| **CRT correct trials** | 358.8(±35.89) | 407.7(±93.25) | 376.0(±66.03) | 369.7(±41.1) | 395.1(±53.28) | 387.3(±49.64) |
| **CRT erroneous trials** | 262.6(±31.26) | 305.7(±71.40) | 279.9(±52.32) | 266.2(±32.22) | 311.6(±110.13) | 297.65(±93.92) |

**Table 3. C**RTs in ms. represent the means of individual median CRTs for post error trials and post matched correct trials as well as for the global performance of the participants and for their correct and erroneous trials. The parenthesized values represent the SD of the data.

## *7.5 Correlations*

We found 2 significant correlations and 3 correlations reaching trend level of significance as listed below:

I. ***Total Helplessness Score*** and ***ΔERN*** [$r(21) = -.362$, $p = .045$] were correlated, indicating that, in our sample, larger absolute values of the ΔERN go along with higher Total Helplessness Scores

II. ***PES CRT*** and ***ERN Amplitude*** [$r(21) = .498$, $p = .016$] were also significantly correlated, revealing that longer PES CRT were related with less pronounced ERNs respectively less negative ERN-amplitudes

III. The correlations between ***PES CRT*** and ***Pe Amplitude*** [$r(21) = -.369$, $p = .0.083$], ***Global CRT*** and ***ERN Amplitude*** [$r(21) = .383$, $p = .0.071$] and ***Global CRT*** and ***Pe Amplitude*** [$r(21) = -.398$, $p = .0.060$] reached trend-level of significance. The first analysis indicated that less pronounced Pe-amplitudes tended to be related to longer PES CRTs. The second correlation showed that shorter Global CRTs tended to be related to more negative ERN amplitudes. Finally, the third correlation revealed that more positive Pe-amplitudes tended to go along with shorter Global CRTs.

The results of all the other correlational analyses were not significant (all p`s>.100).

## *7.6 ERP Data*

### *7.6.1 Time window 10 ms to 120 ms post response*

As hypothesized, after a first visual inspection of the 10 ms to 120 ms post response time window, an ERN characteristic negative deflection was detected for erroneous trials compared to correct ones. Figure 12 displays the ERPs at channel location Cz. Furthermore, also a more pronounced ERN has been remarked in the helpless group compared to the not helpless subjects (see also Figure 13). However, the data reflecting these observations were ascertained by several analyses.

Applying the aforementioned ANOVA, no significant effect of ***helplessness*** [$F(1,19) = 1.677$, $p = .211$] but a significant ***sex*** effect emerged [$F(1,19) = 11.322$, $p = .003$, $\eta^2 = .373$], with male participants displaying generally higher voltages in connection with both erroneous and correct trials relative to female subjects. Additionally, a significant effect of ***correctness*** occurred [$F(1,19) = 62.150$, $p < .001$, $\eta^2 = .766$], indicating significant amplitude differences between erroneous and correct trials. Moreover, a significant ***helplessness × correctness*** [$F(1,19) = 8.045$, $p = .011$, $\eta^2 = .297$] as well as a trend for a ***correctness × sex*** interaction [$F(1,19) = 4.041$, $p = .059$, $\eta^2 = .175$] occurred. All other interactions were not significant [all p values above p >. 283]. Table 4 depicts the relevant data for this analysis.

Post-hoc contrasts revealed no significant result, neither for the erroneous trials [*t(21)* = -1.187, *p* = .248] nor for the correct trials amplitudes [*t(21)* = 1.254, *p* = .223] of helpless vs. not helpless subjects, denoting that the ***helplessness*** × ***correctness*** interaction indicates significantly higher Δ ERN for helpless compared to not helpless participants.

Moreover, further follow up contrasts showed males participants to display a significantly enhanced ERN compared to their female counterparts [*t(21)* = 2.42, *p* =.024, $\eta^2$ =.218]. Due to the applied Bonferroni correction though, the correct trials voltages of males relative to females were enhanced only at trend level [*t*(21) = 2.396, *p* =.025].

| | **Helplessness** | | | | | |
|---|---|---|---|---|---|---|
| | non-helpless | | | helpless | | |
| | **Sex** | | **Total** | **Sex** | | **Total** |
| | male | female | | male | female | |
| **Amplitude correct trials** | -9.6(±2.94) | -2.9(±5.41) | -6.9(±5.11) | -5.6(±2.39) | -3.9(±5.07) | -4.4(±4.39) |
| **Amplitude erroneous trials** | -20(±5.72) | -7.6(±2.46) | -15.1(±7.85) | -24.8(±5.32) | -16.9(±9.40) | -19.3(±8.97) |
| **Peak latency** | 64.7(±26.34) | 61.0(±36.86) | 63.2(±29.02) | 38.0(±16.49) | 32.0(±15.62) | 33.8(±15.46) |

**Table 4**. Mean amplitudes in µV for erroneous and correct trials and latencies in ms for error-trials in the time-window 10 ms -120 ms

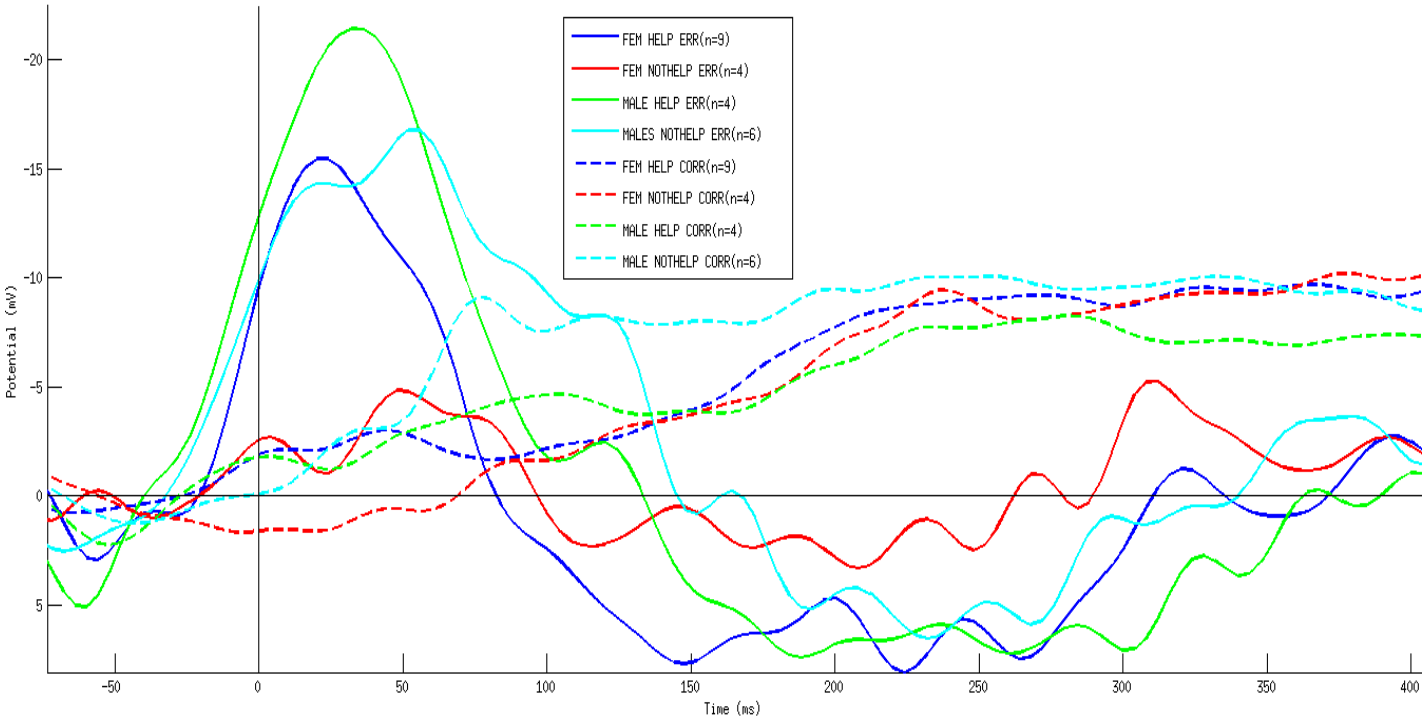


**Fig. 12** Grand means of all conditions at location Cz

Additionally, in order to be able to analyze the effect of the helplessness induction procedure without sex confounds, the data were separately z-transformed for males and for females. Subsequently, a mixed-design ANOVA having the within-subject factor ***correctness*** (z-transformed amplitude erroneous trials/z-transformed amplitude correct trials) and between-subject factor ***helplessness*** (not helpless/helpless) was conducted. The main factors ***correctness*** [$F(1,21) = 0.119$, $p = .734$] and ***helplessness*** [$F(1,21) = 0.498$, $p = .488$] were not significant. However, the analysis yielded a significant interaction of ***correctness*** × ***helplessness*** [$F(1,21) = 6.967$, $p = .015$, $\eta^2 = 0.249$]. Post-hoc contrasts indicated the z-transformed erroneous trials amplitudes to be enhanced for helpless participants compared to not helpless ones [$t(21) = -2.269$, $p = .033$, $\eta^2 = .196$].

As far as the peak latencies in the time window 10 ms to 120 ms post response are concerned, a significant main effect of ***helplessness*** was detected [$F(1,19) = 7.340$, $p = .014$, $\eta^2 = 0.279$] indicating helpless subjects to reach the peak-amplitudes of their ERN significantly earlier than the not helpless participants. All other results of the above mentioned procedure were not significant [***sex****: $F(1,19) = 0.221$, $p = .643$; interaction* ***sex*** × ***helplessness****: $F(1,19) = 0.013$, $p = .911$, $\eta^2 = 0.001$].

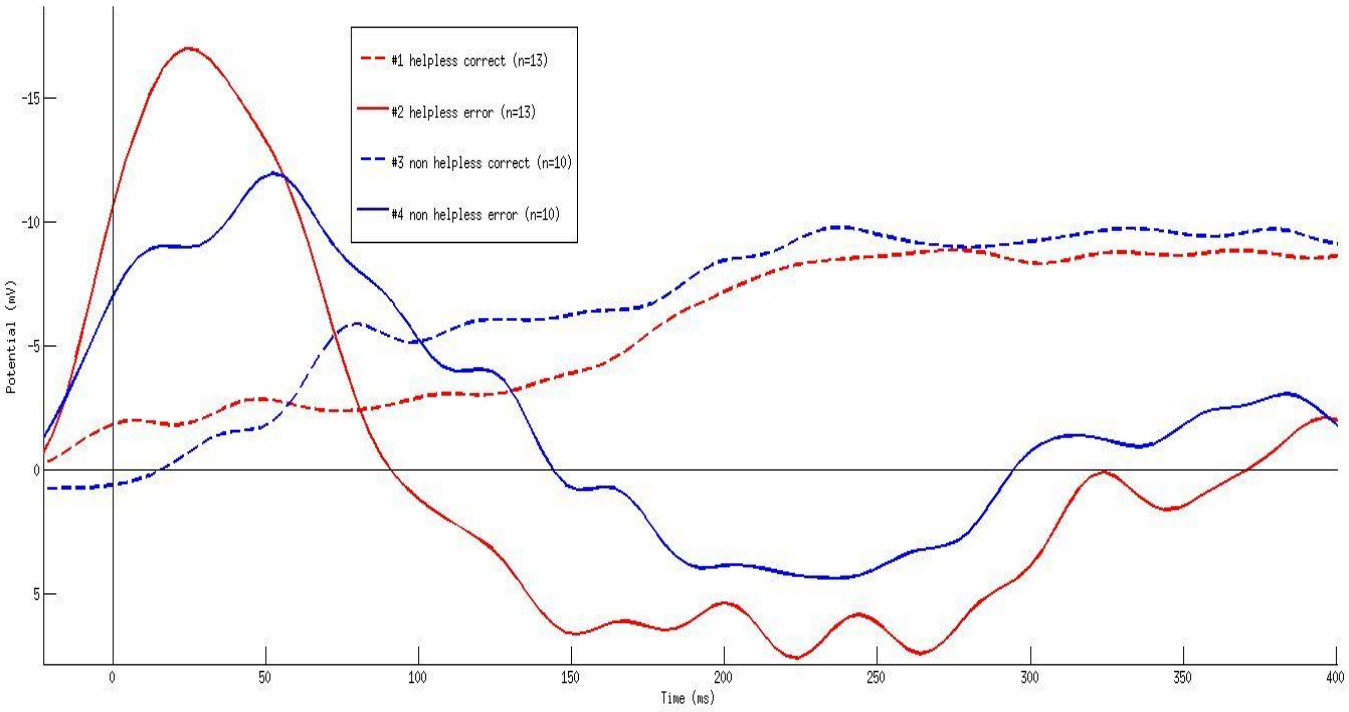


**Fig. 13** Grand means of helpless vs. not helpless subjects at location Cz.

*7.6.2 Time window 200 ms to 400 ms post response*

The visual inspection in the time window 200 ms to 400 ms yielded a potential significant amplitude difference between erroneous and correct responses (see Figure 12). Results of the ANOVA analyzing the amplitude data quantifying the visible Pe-characteristic positive deflection showed a significant effect of ***correctness*** [$F(1,19) = 44.242$, $p < .001$, $\eta^2 = 0.70$], indicating that the voltages of the error condition were significantly more positive than voltages in the correct condition. Furthermore, no significant effects of ***helplessness*** [$F(1,19) = 0.387$, $p = .541$] or ***sex*** [$F(1,19) = 0.022$, $p = .883$] emerged. Besides, no interactions reached statistical significance [all p values above p > .429]. For the data inspection concerning the above mentioned procedure, please consult Table 6 and Figure 12 and 13.

With regard to the peak latencies of the Pe-component in the time window 200 ms to 400 ms, again no significant main effects of ***helplessness*** [$F(1,19) = 0.330$, $p = .573$] or ***sex*** [$F(1,19) = 0.074$, $p = .789$] was revealed and no significant ***helplessness × sex*** interaction [$F(1,19) = 0.544$, $p = .470$] occurred. Table 5 shows the means of these data.

| | **Helplessness** | | | | | |
|---|---|---|---|---|---|---|
| | non-helpless | | | helpless | | |
| | **Sex** | | **Total** | **Sex** | | **Total** |
| | male | female | | male | female | |
| **Amplitude correct trials** | -8.3(±4.49) | -6.8(±7.05) | -7.7(±5.33) | -6.4(±4.18) | -7.5(±7.61) | -7.2(±6.58) |
| **Amplitude erroneous trials** | 10.0(±8.40) | 6.3(±6.01) | 8.5(±7.41) | 9.9(±4.70) | 11.6(±12.44) | 11.1(±10.46) |
| **Peak latency** | 242.7(±23.14) | 266(±80.83) | 252(±51.19) | 273.0(±63.86) | 262.2(±47.08) | 265.6(±50.24) |

**Table 5**. Mean amplitudes in µV for erroneous and correct trials and latencies in ms for error- trials in the time-window 200 ms - 400 ms.

# 8. Discussion

The aim of the current study was to investigate whether experimentally induced helplessness affects error-monitoring processes reflected by the two psychophysiological indices, the ERN and Pe. Moreover, the impact of sex as a moderator variable on the relationship between helplessness and the ERN respectively Pe was investigated.

According to the existing literature (Wiswede et al., 2009a, b), induced negative affect is thought to determine enhanced ERN amplitudes in healthy participants. Therefore, we hypothesized that subjects who would become helpless after being exposed to helplessness training, would display an enhanced ERN compared to the not helpless subjects. Furthermore, as the Pe is supposed to be related to the conscious recognition of an error (e.g. Falkenstein et al., 2000), we expected to measure no significant differences in Pe-amplitude when comparing not helpless and helpless participants. Similarly to previous

studies, we measured significantly higher ΔERN[13] in helpless participants compared to the not helpless participants while the Pe data revealed no significant results. Besides that, after eliminating sex confounds through the separate z-transformation of data sets for males and females, we even assessed significant differences in ERN-amplitudes between helpless and not helpless subjects.

Regarding sex, we expected female participants to experience a more pronounced helplessness-state mirrored in significantly enhanced ERNs compared to helpless males according to results observed in clinical samples (Chiu & Deldin, 2007, Holmes & Pizzagalli, 2008; Nolen-Hoeksema, 1987; Nolen-Hoeksema, 2002 as cited in Stone, Gibb & Coles, 2010). However, there were no significant ERN differences between helpless males and helpless females.

On the behavioral level, our findings did not verify most of our hypotheses. Although we were able to asses significantly longer CRT (choice reaction times) for incongruent stimuli compared to congruent ones and replicated a post-error slowing effect (PES-effect) with our data, no other hypotheses were confirmed. We did not asses any error rates differences or CRT differences between helpless and not helpless subjects and, no sex differences in error rates or CRTs were observed.

## *8.1 Questionnaire Data*

The learned helplessness questionnaire-data revealed, with only one exception, significant differences in reported feelings of passivity, demotivation and despondency between the initially as not helpless, middle helpless and helpless designated participants. While these significant differences supported our 3-leveled operationalization of learned helplessness, the above mentioned exception documented the fact that middle helpless participants achieved their total helplessness scores rather due to high ratings in demotivation and despondency but not due to experienced passivity. Hence, against the theoretical background indicating passivity to be a core symptom of learned helplessness (e.g. Seligman, 1975) the terminology of middle helpless was no longer tenable. We therefore concluded that our helplessness induction procedure failed to induce helplessness in our initially as middle helpless termed group. Instead, we assumed it just induced negatively-valenced feelings and focused on the two-group solution during statistical analyses respectively excluded the not eligible data from further interpretational work.

Additionally to the learned helplessness questionnaire, we also used the PANAS to assess the affect dynamics of our participants. In this context, helpless subjects reported significantly lower PA scores after the mood induction procedure. If we consider the description of high PA scores as mirroring the extent to which a person feels enthusiastic, active and alert by

[13] amplitude errors minus amplitude correct

Watson et al. (1988), this finding confirms our presumption about enhanced levels of passivity and demotivation in the helpless participants due to the helplessness training. Moreover, despite the non-significant alteration of NA-scores after the helplessness induction, we drew the conclusion that the procedure successfully induced negatively valenced affect in our helpless subjects. According to Watson et al. (1988), the aversive mood states captured by the NA scale rather imply a state of unpleasurable engagement. Unpleasurable engagement however, could be understood, in terms of the circumplex model of affect[14] (Russel, 1980) as being connected with high arousal or high activation. This, in turn, is not in conformity with the reported passivity state of the helpless subjects and could be the reason why our participants did not report significant NA changes after the helplessness induction. Furthermore, we believe that despondency is a negative affect state characterized by low arousal levels and might therefore be better suited to assess helplessness states, as described by the existing literature.

To summarize, we assume the successful induction of helplessness in the participants assigned to the helpless group, even though the reported heightening of negative affect, as measured by the NA scale of PANAS, was not statistically significant. As our data indicate high levels of despondency in the participants who became helpless, we suggest they just experienced low arousal negatively valenced affect, which is more characteristic to the helplessness state.

## *8.2 Behavioral data*

Due to our research question, we were interested in whether or not sex respectively induced helplessness would influence behavioral correlates of error monitoring. We hypothesized the helpless subjects to have prolonged CRT and higher error rates than the not helpless participants and the male participants to achieve faster CRT compared to females. Additionally, we assumed helpless women to produce higher error rates compared to helpless male participants. Surprisingly, none of these hypotheses could be confirmed. However, we were able to detect a sex influence on the PES-effect at trend level.

### *8.2.1 Coice Reaction Times*

In this context, the most intriguing finding was the fact that, even if we observed the pattern of the typical sex differences in CRT, the results were statistically not significant. Considering the fact that “the largest and most consistent difference across the range of ages is the increased CRT of women” (Der & Deary, 2006, p.64), we propose that this attenuation of sex differences might be due to the helplessness training preceding the flanker task. There is

[14] The circumplex model of affect proposes affective states to arise from two neuropsychological systems, one related to valence and the other to arousal

little known about the exact reason of such gender-related differences in CRT. Adam, Paas, Buekers, Wuyts, Spijkers and Wallmeyer (1999) for example, suggest that these reaction times differences may reflect differences in processing strategy.

However, in order to be able to make further statements in this direction, a direct comparison of the CRT performance of female and male participants without helplessness induction would be necessary. In this way, we would be able to find out whether a lowering of performance in males or an increase of performance in females occurred and to better understand those hypothesized mechanisms of action of our helplessness training. Unfortunately, up to now, no such data were available.

Furthermore, even though our subjects experienced a certain kind of helplessness induction, this might not have been strong enough to elicit behavioral effects apparent in CRT differences. Previous work on reaction times (e.g. Welford, 1980; Broadbent, 1971) indicates that arousal influences the reaction time performance in humans. The fastest reaction times go along with intermediate arousal levels while too low or too high arousal levels slow the reaction time. This is why we presume that especially the arousal lowering achieved through our helplessness training was not strong enough to cause the expected significant slowdown of the CRT performance in our helpless participants.

### *8.2.2 Error rates*

Regarding attentional focus, Gable and Harmon-Jones (2010) used a global-local letter task (Navon, 1977) to measure attentional breadth and demonstrated that low-motivation negative affect caused attentional broadening. For our paradigm, it was conceivable that attentional broadening might lead to higher error rates due to an increased distractibility of flankers. Therefore, we assume that the helplessness treatment was too short to induce sufficient demotivation and cause an attention broadening shift. However, for the verification of the latter possibility, the inclusion of a control group in our experimental design would have been something to strive for.

Moreover, we proposed that helpless female participants, due to a more pronounced focus on themselves in depressed moods (Butler & Nolen-Hoeksema, 1994), might commit more errors than their male counterparts. Our results did not confirm our expectations. At the same time, a chi-square test detected no sex related distributional differences in our helpless and not helpless samples. Hence, we assume that the female participants as a whole were not more vulnerable to our mood manipulation than their male counterparts or, at least, not enough to cause the behavioral differences we expected.

### *8.2.3 Post-error slowing and congruency effect*

The PES effect (e.g. Hajcak et al., 2003b) refers to the deceleration of the CRT after the commission of an error and the congruency effect (e.g. Sanders & Lamers, 2002) describes the fact that CRTs for incongruent stimuli are always increased compared to CRTs of congruent stimuli. The acquired behavioral data enabled the confirmation of the hypotheses regarding those effects and we successfully replicated them.

Our approach to the investigation of the PES was two-fold. Primarily, we wanted to ensure the measurement of a genuine PES. Given the fact that CRT of error trials are typically faster than those of correct trials, it was necessary to exclude the possibility of a regression toward the mean effect. Therefore, we used a similar procedure as in Wiswede et al. (2009) and Hajcak, McDonald and Simons (2003b) and examined the CRT of post-error trials versus trials following a subset of correct trials which were matched to the error trials in terms of total number and reaction times. Then, we went ahead in a more explorative manner. We wanted to figure out if any of our experimental conditions would have an impact on the PES-phenomenon. We found it to be independent of helplessness state but assessed a sex effect that reached trend level of significance. However, the latter rather reveals longer overall CRTs for females compared to males than an enhanced PES in females. Likewise, the just mentioned sex effect was not identified in the global CRT-analysis, which is more representative due to the much more numerous trials involved in it.

Furthermore, we assessed a congruency effect but were not able to measure any influence of sex or helplessness state within this context. Thus, it seems that all our subjects experienced a comparably strong flanker-interference when reacting to incongruent stimuli.

## *8.3 EEG Data*

### *8.3.1 ERN*

Due to partly opposing findings, the current status of knowledge on the topic regarding the influence of short lasting negative affect on error monitoring processes as reflected by the ERN seems rather poor. More specifically, whereas some authors did not ascertain any significant modulations of ERN-amplitude after experimentally induced negative affect (Olvet & Hajcak, 2011), other research-groups found significant alterations of ERN-amplitudes which obviously originated from induced negative affect (Wiswede et al., 2009a; Wiswede et al., 2009b). In our study we observed that experimentally induced helplessness modulates the amplitude of the ERN and, that higher Total Helplessness Scores are related to larger absolute values of the ΔERN. Thus, our results are strongly supported by those reported by Wiswede and colleagues (2009a, b). We furthermore believe that Olvet and Hajcak's (2011) null findings might be explained based on efficacy matters of their mood induction procedure (MIP).

Whilst Wiswede et al. (2009a) used a flanker-task to evoke an ERN and presented, prior to each flanker stimulus, either a neutral, a pleasant or an unpleasant picture from the IAPS (Lang et al., 1999), Wiswede et al. (2009b) induced negative affect by providing derogative performance related feedback during the flanker task after blocks of every 30 trials. That is, in both experiments, the above mentioned authors measured the ERN only very shortly after the mood induction and observed ERN modulations.

In contrast, Olvet and Hajcak (2011) induced sad, neutral and positive mood using a film clip which lasted 2-5 minutes and a song played in the background throughout the entire experiment. More precisely, after the film clip, they first asked their participants to rate their mood on the valence scale of the Self-Assessment Manikin (Lang, 1980) and to complete a flanker task training block consisting of 30 trials. The measurement of the ERN occurred only afterwards, during a flanker task which lasted approximately 10 minutes. Hence, it is conceivable that the mood induction paradigm applied by Olvet and Hajcak (2011) generated mood states which were not stable enough to last in the same intensity throughout the training block and the following flanker task.

Consequently, as the time span between the end of the mood induction procedure (MIP) and the end of the flanker task was comparably long in our experiment, it seems appropriate to broach the issue of why we believe that our MIP might have been more effective than the MIP used by Olvet and Hajcak (2011). Apart from the fact that our helplessness training lasted much longer than Olvet and Hajcak's (2011) film clip, we confronted our subjects with misinforming instructions about the alleged simplicity of the tasks. In reality, 50% of the reasoning tasks were unsolvable. Under those circumstances, it might be that the subjects made unpleasant competence inferences about the self. This, in turn, might have led to a negative mood which was more salient and therefore more stable since the subjects, as it were, put the blame on themselves for not being able to solve the series of numbers. In comparison, it is likely that viewing a sad film-clip and listening to sad music did not evoke such self-referential thoughts. That is, by giving rise to self-esteem related thoughts, our MIP might have ensured enough stability for the induced affect.

The ERN modulation we report about was first observed as Δ ERN which was shown to be significantly higher for helpless participants compared to not helpless participants.

Furthermore, as we investigated a sex-mixed sample due to our research interest, we z-transformed our data in order to avoid sex related confounds when comparing the ERN amplitudes between different helplessness groups. After controlling for sex related confounds by separately z-transforming the amplitudes for males and females, we were able to detect also significantly enhanced ERN-amplitudes for helpless subjects compared to not helpless ones.

In view of the observed ERN modulation, we assume that “to the extent to which the ERN amplitude can be taken as an index of the activity level of the action monitoring system” (Wiswede et al., 2009a, p. 88), our results suggest that the brain’s action monitoring system is more active when action is taking place on the background of negative affect.

The effect of negative affect on the ERN amplitude can be explained based on the reinforcement learning theory of ERN (Holroyd & Coles, 2002) and on the conclusions of Ashby, Isen and Turken (1999) respectively Ashby and Casale (2003). Taken together, the previously mentioned literature suggests that “the influence of affect on cognition might be moderated by changes in dopamine release from the mesencephalic dopamine projections to (among others) the ACC and the Nucleus accumbens*”* (Wiswede et al., 2009a, p.89).

Additionally, Wiswede et al. (2009a) hypothesized that the ERN enhancement could be mediated by higher arousal levels via the noradrenergic system. Our data do not support this assumption, since we induced low arousal negative affect.

#### 8.3.1.1 ERN and Sex differences

First of all, it has to be noted that sex confounds may arise due to anatomical variation sources of the electrical potentials recorded on the scalp (Nunez & Shrinivasan, 2006). We observed that our male participants generally displayed enhanced ERPs relative to their female counterparts. This result is inconsistent with exactly the opposite pattern reported by Guillem et al. (2009). Nevertheless, the same authors (Guillem et al., 2009) mentioned that females do not generally display larger ERPs at all scalp sites. Consequently, our results still can be explained as being related to sexual dimorphism in the human scalp.

Besides that, our male subjects displayed also an enhanced ERN when compared to their female counterparts which is in line with the results reported by Larson et al. (2001) who also detected enhanced ERN in males compared to females.

Furthermore, since we did not asses a significant sex × correctness × helplessness interaction, the hypothesized significant differences of ERN amplitude between helpless males and females were not confirmed.

Against this backdrop, we cannot entirely rule out the possibility that the enhanced ERN of our male participants might be originating only through the influence of the MIP on the generally enhanced ERPs of the male sample. Therefore, we also cannot tell if our results are mirroring the male characteristic to generally display enhanced ERN amplitudes compared to females, as hypothesized by Larson et al. (2001).

Consequently, if we cannot draw any ultimate conclusion about the origins of the enhanced ERN in our entire male sample, we also cannot definitely tell how the lack of significant amplitude differences between helpless males and females might be interpreted. In effect, that means we can neither understand why the activity level of the action monitoring system was comparably high in our helpless female and male participants nor are we able to fully

confirm the findings of Larson et al. (2001) who found enhanced ERN for males relative to females.

To sum it up, due to the lack of conclusive results, we cannot make any clear statement about whether or how sex is moderating the interconnection of helplessness and ERN.

#### 8.3.1.2 ERN and Depression

Helplessness is supposed to play a role in the etiology of hopelessness depression (Abramson et al., 1989). On the other hand, depressed subjects were found to display an increased ERN compared to healthy controls. (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008). Given this background, we observed that experimentally induced helplessness modulated the ERN in the same direction as depression does. It is therefore conceivable that immediately after the MIP our helpless subjects experienced a comparable emotional state as the depressed people do during a mild episode of depression. Since “the ERN might be a useful endophenotype for internalizing disorders” (Olvet & Hajcak, 2008, p. 1350), it is imaginable that persons, who become helpless after an experience such as our helplessness training, might be more predisposed to depression.

However, future studies might address this issue by applying an inverse approach and investigating how (recovered) individuals with a psychiatric history react to such a helplessness training compared to individuals with no psychiatric history.

### *8.3.2 Pe*

We found no influence of helplessness status or sex on the amplitudes of the Pe component. Our null finding is in line with the results of Wiswede and his co-workers (2009b) and favors the assumptions that the Pe might be related to the conscious recognition of an error and that negative affect induction does not seem to increase sensitivity for conscious error recognition.

## *8.4 Correlations*

We assessed 2 significant correlations and 3 correlations reaching trend level of significance. Whereas the strong relationship between larger absolute values of ΔERN and higher Total Helplessness Scores supports our hypothesis respectively the effect of our experimental manipulation on the ERN, the other correlations between behavioral and ERP data are difficult to be interpreted.

For instance, the fact that longer PES CRTs were significantly related with less pronounced ERNs is conflicting with the findings of Debener et al. (2005) who showed that higher single-trial ERN amplitudes were associated with longer CRTs on trials after errors. Thus, it appears that the above mentioned relationship between longer PES CRTs and less negative ERN-amplitudes may have come into being due to the PES matching procedure and the

sexual dimorphism in our sample. At the same time we also found that larger Pe-amplitudes were associated with less post-error slowing which is not in accordance with former results (Hajcak et al., 2003b; Nieuwenhuis et al.,2001). Additionally, the relation between shorter Global CRTs and more negative ERN-amplitudes respectively between more positive Pe-amplitudes and shorter Global CRTs might be determined by sexual dimorphism aspects rather than by our experimental manipulation.

To sum it up, either due to the method we used to asses PES or due to sex dimorphism related differences between male and female ERPs, the results of the correlations between behavioral and EEG measures of error monitoring may be distorted.

### *8.5 Limitations*

Overall, considering our behavioral findings, we want to stress that the lack of significant results on behavioral level might be partly determined by our experimental design. The fact that all our participants went through the same mood induction procedure before completing the flanker task might be the reason, why some differences on behavioral level were too small to reach the threshold of statistical significance. To the extent to which it can be implied that behavioral measures of error monitoring are not that sensitive as EEG-measures of error monitoring processes, we assume this is also the reason why the behavioral and the EEG results point in different directions. Furthermore, it is conceivable that such inconveniences could have been avoided when using a control group or, if the not-helpless participants would have been asked to complete the flanker task to a different, later time point.

Last but not least, it should be also pointed out that additional data of a female and a male control group might have contributed to clarifying the circumstances that have arisen within the context of ERN and the hypothesized moderator variable sex.

## 9. Conclusion

We were able to show that induced short time negative affect has an influence on the ERN amplitude. Thus, it can be stated that we provided a further piece of evidence about the interactive connection between affect and performance monitoring processes on a short time scale. Due to the role helplessness can play in the etiology of depression, further research seems mandatory to investigate whether participants who report high helplessness levels after a helplessness training might be more susceptible to depression when compared to persons who do not become helpless in such a setting.

Moreover, since the question regarding the mediating role of sex remained unanswered, future research should attempt to clarify all conceivable interactions between sex, ERN and helplessness.

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# 11. Appendix

*11.1 Helplessness questionnaire with three-leveled between-subject factor helplessness (not helpless/middle helpless/helpless)*

| **Dependent variable Despondency** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 12.109 | **<.001***[15] | .439 |
| **sex** | 1/31 | 0.094 | .761 | .003 |
| **sex×helplessness** | 2/31 | 0.222 | .802 | .014 |
| **Dependent variable Passivity** | | | | |
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 7.759 | **.002*** | .334 |
| **sex** | 1/31 | 0.075 | .786 | .002 |
| **sex×helplessness** | 2/31 | 0.134 | .875 | .009 |
| **Dependent variable Demotivation** | | | | |
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 11.420 | **<.001*** | .424 |
| **sex** | 1/31 | 1.034 | .317 | .032 |
| **sex×helplessness** | 2/31 | 0.126 | .882 | .008 |
| **Dependent variable Total Helplessness Score** | | | | |
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 71.826 | **<.001*** | .823 |
| **sex** | 1/31 | 0.613 | .440 | .019 |
| **sex×helplessness** | 2/31 | 0.158 | .854 | .010 |

[15] * denote significant results

*11.2 PANAS questionnaire with three-leveled between-subject factor helplessness (not helpless/middle helpless/helpless)*

| **Mixed-design ANOVA within-subject factors PA and NA** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **PA** | 1/31 | 128.289 | **<.001*** | .805 |
| **PA×NA** | 1/31 | 12.149 | **.001*** | .289 |
| **NA** | 1/31 | 0.494 | .487 | .016 |
| **Helplessness** | 2/31 | .253 | .778 | .016 |
| **Sex** | 1/31 | 1.633 | **.**221 | .050 |
| **All other interactions with *p*-values above *p* = .126** | | | | |

*11.3 Error rates ANOVAs with three-leveled between-subject factor helplessness (not helpless/middle helpless/helpless)*

| **Dependent variable Global error rates** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.249 | .781 | .016 |
| **sex** | 1/31 | 0.024 | .879 | .001 |
| **sex×helplessness** | 2/31 | 0.028 | .973 | .002 |
| **Dependent variable incongruent error rates** | | | | |
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.205 | .816 | .013 |
| **sex** | 1/31 | 4.769 | **.037*** | .133 |
| **sex×helplessness** | 2/31 | 0.213 | .809 | .014 |
| **Dependent variable congruent error rates** | | | | |
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.205 | .816 | .013 |
| **sex** | 1/31 | 4.769 | **.037*** | .133 |
| **sex×helplessness** | 2/31 | 0.213 | .809 | .014 |

*11.4 Choice reaction times ANOVAs with three-leveled between-subject factor helplessness (not helpless/middle helpless/helpless)*

| **Mixed-design ANOVA within-subject factor correctness** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.213 | .809 | .014 |
| **sex** | 1/31 | 2.484 | .125 | .074 |
| **correctness** | 1/31 | 177.404 | **<.001*** | .851 |
| **All interactions with *p*-values above *p* = .597** | | | | |

| **Mixed-design ANOVA within-subject factor congruency** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.072 | .930 | .005 |
| **sex** | 1/31 | 2.782 | .105 | .082 |
| **congruency** | 1/31 | 1394.064 | **<.001*** | .978 |
| **congruency×sex×helplessness** | 2/3 | 3.374 | **.047*** | .179 |
| **congruency×sex** | 1/31 | 2.989 | .094 | .088 |
| **All remaining interactions with p-values above *p* = .596** | | | | |

| **Mixed-design ANOVA within-subject factor PES** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.079 | .924 | .005 |
| **sex** | 1/31 | 4.519 | **.042*** | .127 |
| **PES** | 1/31 | 25.006 | **<.001*** | .446 |
| **All interactions with p-values above *p* = .336** | | | | |

| **Dependent variable global CRT** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.148 | .871 | .129 |
| **sex** | 2.092/31 | 4.474 | .163 | .681 |
| **sex×helplessness** | 2/31 | 0.510 | .605 | .032 |

*11.5 Correlations for the entire sample (including middle helpless subjects)*

| Variables | *p*-value | Pearson's correlation coefficient (r) |
|---|---|---|
| **PA after** *and* **Total Helplessness Score** | **.096****[16] | -.278 |
| **PA after** *and* **ERN Amplitude** | **.064**** | .307 |
| **PA after** *and* **ΔERN** | .102 | .273 |
| **PA after** *and* **Pe Amplitude** | .509 | .112 |
| **PA after** *and* **CRT post-error (PES)** | .869 | .028 |
| **PA after** *and* **Global CRT** | .621 | -.084 |
| **PA after** *and* **Error rates** | .699 | -.066 |
| **CRT post-error (PES)** *and* **ERN Amplitude** | **.027*** | .364 |
| **CRT post-error (PES)** *and* **ΔERN** | .252 | .193 |
| **CRT post-error (PES)** *and* **Pe Amplitude** | .355 | -.156 |
| **Global CRT** *and* **ERN Amplitude** | .106 | .270 |
| **Global CRT** *and* **ΔERN** | .690 | ..068 |
| **Global CRT** *and* **Pe Amplitude** | .352 | -.157 |
| **Error rates** *and* **ERN Amplitude** | .505 | .113 |
| **Error rates** *and* **ΔERN** | .348 | .159 |
| **Error rates** *and* **Pe Amplitude** | .412 | -.139 |
| **Total Helplessness Score** *and* **ERN Amplitude** | .228 | -.164 |
| **Total Helplessness Score** *and* **ΔERN** | **.045*** | -.362 |
| **Total Helplessness Score** *and* **Pe Amplitude** | .438 | .034 |
| **Total Helplessness Score** *and* **Error rates** | .253 | .113 |
| **Total Helplessness Score** *and* **CRT post-error (PES)** | .450 | .128 |
| **Total Helplessness Score** *and* **Global CRT** | .130 | .190 |

[16] ** Trend level

| Variables | *p*-value | Spearman's ρ (rho) |
|---|---|---|
| **NA after** *and* **Total Helplessness Score** | .116 | .263 |
| **NA after** *and* **ERN Amplitude** | .624 | .083 |
| **NA after** *and* **ΔERN** | .556 | .100 |
| **NA after** *and* **Pe Amplitude** | .120 | .260 |
| **NA after** *and* **CRT post-error (PES)** | .728 | .059 |
| **NA after** *and* **Global CRT** | .360 | .155 |
| **NA after** *and* **Error rates** | .797 | -.044 |

*11.6 ERN ANOVAs with three-leveled between-subject factor helplessness (not helpless/middle helpless/helpless)*

| **Mixed-design ANOVA within-subject factor correctness** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 1.433 | .245 | .085 |
| **sex** | 1/31 | 6.322 | **.017*** | .169 |
| **sex×helplessness** | 2/31 | 6.147 | **.006*** | .28 |
| **correctness** | 1/31 | 93.854 | **<.001*** | .752 |
| **correctness×helplessness** | 2/31 | 3.509 | **.042*** | .185 |
| **correctness×sex** | 1/31 | 1.253 | .272 | .039 |
| **corectness×helplessness×sex** | 2/31 | 1.765 | .188 | .102 |

| **Dependent variable latency time window 10 ms-120 ms post response** | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 3.504 | **.042*** | .184 |
| **sex** | 1/31 | 0.790 | .381 | .025 |
| **sex×helplessness** | 2/31 | 0.110 | .896 | .007 |

*11.7 Pe ANOVAs with three-leveled between-subject factor helplessness (not helpless/middle helpless/helpless)*

| Mixed-design ANOVA within-subject factor correctness | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.604 | .553 | .037 |
| **sex** | 1/31 | 0.451 | .507 | .014 |
| **sex×helplessness** | 2/31 | 0.307 | .738 | .019 |
| **correctness** | 1/31 | 95.409 | **<.001*** | .755 |
| **correctness×helplessness** | 2/31 | 0.108 | .898 | .007 |
| **correctness×sex** | 1/31 | 0.226 | .638 | .007 |
| **corectness×helplessness×sex** | 2/31 | 0.459 | .636 | .029 |

| Dependent variable latency time window 200 ms-400 ms post response | | | | |
|---|---|---|---|---|
| **Factors** | **df** | ***F*-value** | ***p*-value** | $\eta^2$ |
| **helplessness** | 2/31 | 0.281 | .757 | .018 |
| **sex** | 1/31 | 0.673 | .418 | .021 |
| **sex×helplessness** | 2/31 | 0.541 | .588 | .034 |

## *11.8 Zusammenfassung (Abstract Deutsch)*

Das Hauptziel dieser Untersuchung war, die Zusammenhänge zwischen Hilflosigkeit und neuronalen Korrelaten der Fehlerverarbeitung (ERN und Pe) zu erfassen. 25 Männer und 25 Frauen nahmen an einem Hilflosigkeitstraining teil, basierend auf 30 lösbaren und 30 unlösbaren Zahlenreihen. Anschließend baten wir unsere Probanden einen Hilflosigkeitsfragebogen (Bauer et al., 2003) auszufüllen. Anhand ihres Ratings bei dem Fragebogen teilten wir sie einer unserer 3 Hilflosigkeitsgruppen zu. Es erfolgte auch ein modifizierter Flanker Task (Eriksen & Eriksen, 1974), im Zuge dessen wir die ERN und Pe der Teilnehmer mittels EEG aufnahmen. Wir verglichen die ERN und Pe von 23 Probanden, die aufgrund ihrer Ratings im Hilflosigkeitsfragebogen der Extremgruppen hilflos und nicht hilflos angehörten. Wir fanden signifikant höhere ΔERN für hilflose verglichen mit nicht hilflosen Probanden. Nachdem wir durch eine z-Transformation auch die Wirkung der konfundierenden Variable Geschlecht ausgeschlossen hatten, konnten wir auch signifikant höhere ERN Amplituden für Hilflose feststellen verglichen mit den Nicht-Hilflosen. Hinsichtlich der Pe-Komponente konnten keine Unterschiede zwischen Hilflosen und Nicht-Hilflosen festgestellt werden. Diese Ergebnisse bestätigen unsere Hypothesen und wir interpretieren die ERN Unterschiede zwischen hilflosen und nicht hilflosen Probanden basierend auf der Wirkung des induzierten negativen Affekts.

Zusätzlich konnten wir keine signifikanten Amplituden-Unterschiede hinsichtlich der ERN hilfloser Männer und Frauen feststellen. Die angenommene Moderator-Rolle des Geschlechts in diesem Zusammenhang konnte jedoch weder bestätigt noch abgewiesen werden.

## *11.9 Abstract in English*

This diploma thesis aimed to investigate whether induced short time negative affect in the form of helplessness would modulate the amplitudes of the ERN and the Pe event-related potentials. Both components are established neural correlates of error processing. Thus, we exposed 25 female and 25 male participants to a helplessness training comprising 30 solvable and 30 unsolvable cognitive tasks (series of numbers). Subsequently, participants had to complete a modified version of a flanker task (Eriksen & Eriksen, 1974) while their EEG-signals were recorded. We compared ERN and Pe amplitudes of helpless (13 subjects, 9 females) vs. not helpless (10 subjects, 4 females) participants and found a significantly higher ΔERN for helpless subjects compared to their not helpless counterparts. The helplessness status of the participants was assessed using a helplessness questionnaire (Bauer et al., 2003).

After separate z-transformation of the ERN amplitudes for males and females which was conducted in order to eliminate sex confounds caused by sexual dimorphism, we also found significantly larger ERN amplitudes for helpless vs. not helpless subjects. We explain the induced modulations of the ERN in terms of negative affect.

Additionally, we did not asses any differences regarding the Pe of helpless vs. not helpless subjects and no ERN related differences between helpless males and females were found. However, the hypothesis about the moderator role of the variable sex was neither confirmed nor rejected and future studies addressing this issue should consider an experimental design including a female and a male control group.

Finally, it should also be mentioned that further research seems mandatory to investigate whether participants who report high helplessness levels after a helplessness training might be more susceptible to depression when compared to persons who do not become helpless in such a setting.

## *11.10 List of figures*

## Curriculum vitae

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**Vorname:** Diana Roxana
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| | |
|---|---|
| **1988 - 1990** | Bundesrealgymnasium für Mathematik und Physik in Bukarest |
| **1990 - 1991** | AHS Kenyongasse in Wien |
| **1991 - 1993** | AHS Kandlgasse in Wien |
| **Juni 1993** | Matura mit Schwerpunkt Mathematik |
| **Oktober 2004** | Studium der Psychologie an der Universität Wien |
| **Ende März 2012** | Voraussichtlicher Abschluss des Psychologiestudiums |