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A prospective randomized controlled training study investigating the relationship between cognitive control and rumination using ERPs in healthy subjects

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Abstract

Rumination is an established risk factor for the emergence of various psychological diseases. Evidence from behavioral and neuroimaging studies has linked rumination to deficits in cognitive control (CC). In contrast, the relationship between rumination and event-related potentials (ERPs) indexing CC is less understood, while the causal relation between CC and rumination in healthy individuals is also not sufficiently described. In the current study CC-related ERPs of an n-back task were linked to state and trait rumination as well as state rumination changes in response to induced negative mood in a total sample of 33 healthy participants. A subgroup (n=14) conducted a CC training to enhance CC and was compared to a control group (n=16). Causal influences of cognitive control on rumination were investigated using this randomized controlled prospective study design. Results indicated that training reduced the susceptibility to ruminate. ERP amplitudes which are linked to inhibition and conflict monitoring (N2 and N4) were larger in individuals with a high susceptibility to ruminate. Results also showed a tendency towards a correlation between current state of rumination and an ERP component, which is linked to working-memory updating (P3). Possible diverging relationships between CC and rumination are discussed.

1. Introduction

As human beings, we tend to manage our emotions in different ways. Let's say we experienced a stressful event, like getting in trouble by blowing an important test, getting upbraided by our supervisor or having a fight with our partner. All these events could make us feel bad at least for a while and force us to deal with upcoming unpleasant emotions. We can visualize the memory of what happened again and again or we may search for reasons, why these events occur, either in us or externally. We can try to extract the positive sides and reappraise the whole situation, look for help by others or simply refuse to deal with it and try to forget all about it. Those different ways to regulate our emotions have become subject of psychological research (Gross, 2015). Different emotion regulation strategies have been linked to heath, wellbeing and psychological diseases. In this context, one of the most important emotion regulation strategies is rumination and its categorization, operationalization, causes and consequences have been subject of a large body of research.

1.1. Theoretical Background

The psychological concept of rumination has largely been influenced by the work of Susan Nolen-Hoeksema, who established the Response Styles Theory (RST; Nolen-Hoeksema, 1991). The RST describes rumination as a mode of responding to negative affect (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), with

> behaviors and thoughts that focus one's attention on one's depressive symptoms and on the implications of these symptoms (….). Ruminative responses may include isolating oneself to think about one's symptoms (…), possible causes of the depression (…), and the possible consequences of depression (…). (Nolen-Hoeksema, 1991, p. 596)

A ruminative response style is a trait like pattern of ruminative thinking in response to negative emotions (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008). The ruminative response style has so far mostly been measured with the Response Styles Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991), which distinguishes two oppositional styles of responses to negative emotion, i.e. rumination and distraction. Consecutive research differed between two subscales

of the ruminative response style, i.e. reflective pondering and brooding, which are problem focused and emotion focused rumination styles, respectively (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Other concepts of rumination define ruminative processes more as a reaction to an event and not as an individual thinking style in reaction to negative mood per se. Thus, the respective inventories focus on rumination in reaction to specific events, rather than on general reaction styles (Smith & Alloy, 2009). Nolen-Hoeksema's concept of rumination is different to the precedent approaches of cognitive theorists (Beck, 1967; Nolen-Hoeksema et al., 2008) who had rather focused on the specific contents of thoughts than the process of repetitive thinking and it's perseverative character. Attempts to integrate Beck's Theory of depression (Beck, 1967) with the RST revealed, that the development of depressive cognitive schemas, in adolescents and adults, is moderated by the ruminative response style brooding. Nevertheless, integrating these models does not enhance the prediction of depressive symptoms compared to Beck's theory alone (Black & Pössel, 2014; Pössel, 2011).

Rumination is related to the concept of worrying, but is also clearly differentiated thereof. While worrying describes anxious thoughts that are focused on future events, involve more cognitive effort and have problem solving characteristics, rumination is directed to the past and describes more passive and less confident thinking style (Nolen-Hoeksema et al., 2008; Papageorgiou & Wells, 2003; Wimmer, 2010). Ruminative thoughts are focused on reduced self-worth and seek to solve problems by understanding the meaning of past events, while unconsciously avoiding the initiation of problem solving actions (Nolen-Hoeksema et al., 2008). Rumination is also distinguishable from other self-referential thoughts, like negative automatic thoughts, private self-focus, self-focused attention and repetitive thoughts, since rumination describes a more specific style of thinking about problems and the self. Reviewing rumination literature, Smith and Alloy (2009) discriminated rumination from these fundamental concepts as follows: negative automatic thoughts differ in length and repetitiveness, so automatic thoughts may be part of rumination, but have a more transitory nature. Private self-focus is a general processing style, which does not necessarily depend on mood state and rumination could hence be subsumed under private self-focus as a

higher order category. Self-focused attention, which is closely related to private self-focus, could also be considered as a higher order category, which then contains reflection as well as rumination. Repetitive thoughts can generally be distinguished from rumination regarding valence, context, and construal level (Watkins, 2008). When directly compared to each other, rumination, as a specific mode of repetitive thinking, has negative valence, refers to a negative context and has an abstract level of construct (Smith & Alloy, 2009).

1.1.1. Rumination and psychopathology

In the early development of the RST rumination has primarily been linked to depression, negative affect and anxiety (Mor & Winquist, 2002; Nolen-Hoeksema et al., 2008). It has been shown, that induced rumination, in contrast to distraction, increases depressed mood in subjects diagnosed with a major depressive disorder (MDD; Nolen-Hoeksema & Morrow, 1993). Moberly and Watkins (2008) found a longitudinal relationship between trait rumination and increased reactivity to negative affect. They further describe how a ruminative self-focus mediates the influence of stressful events on negative affect. Rumination has also been found to increase the probability of the onset of depressive episodes and diagnosis of depression (Kuehner & Weber, 1999; Mor & Winquist, 2002; Nolen-Hoeksema et al., 2008). Longer durations of depressive episodes have been linked to higher levels of trait rumination (Nolen-Hoeksema et al., 2008). Exploring known gender differences regarding higher rumination tendencies and higher prevalence of depression in women (Abate, 2013; Copeland et al., 1999; Nolen-Hoeksema & Jackson, 2001), it has been demonstrated that gender differences in depression are moderated by ruminative response styles (Nolen-Hoeksema, 1987; Nolen-Hoeksema, Larson, & Grayson, 1999).

Rumination has been linked to various other psychopathologies, such as anxiety, binge drinking, binge eating, self-harm and substance abuse (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Nolen-Hoeksema et al., 2008): a meta-analysis by Aldao, Nolen-Hoeksema and Schweizer (2010) found rumination to be the emotion regulation strategy, with the strongest relation to psychopathologies in general compared to any other emotion regulation strategy.

In the view of the recent discussion on future developments in the field of psychiatric diagnostics, accompanied by the publication of the DSM-5 (American Psychiatric Association, 2013), rumination became an illustrative example of a transdiagnostic approach to psychiatric diseases (Nolen-Hoeksema & Watkins, 2011; Seligman, 2014). Following new ideas of future diagnostics, rumination may become a phenomenon which will not further be viewed as a risk factor or cause of specific psychiatric disorders but as a diagnostic entity, that itself may be sufficiently worth treatment (Seligman, 2014).

1.1.2. Rumination and cognitive control

As previously discussed, rumination plays an important role in the emergence and appearance of affective disorders, thus the majority of literature on rumination originates from clinical studies investigating the development and symptoms of depression.

Rumination has been associated with a large scale of cognitive control (CC) deficits, most of which are associated with inhibitory functions. Several reviews have examined the correlates of CC deficits and rumination, stating that rumination is associated with deviations in information processing, particularly in depressed individuals, but also in healthy subjects (Joormann, Yoon, & Zetsche, 2007; Koster, De Lissnyder, Derakshan, & De Raedt, 2011; Whitmer & Gotlib, 2013). Rumination related concepts like mind wandering have also been related to reduced performance in attentional tasks (Randall, Oswald, & Beier, 2014). On the other hand, emotion regulation strategies, which are more adaptive than rumination, have been linked to increased CC. Cohen, Daches, Mor and Henik (2014) reviewed and compared findings about inhibition of negative information in association with rumination and reappraisal. The authors suggest that inhibition is a key mechanism, which is oppositely related to rumination and reappraisal. According to Cohen et al. decreased inhibition relates to a tendency to ruminate in response to negative events, while an intact ability to inhibit negative content supports reappraisal.

A large body of research including multiple domains of CC and working memory, as well as various forms of stimulus material has been reviewed by Whitmer and

Gotlib (2013). While systematically taking account of the sample and objectives used in these studies, following conclusions were drawn from the available literature:

- Trait rumination is associated with difficulties to forget or to disengage from no longer relevant information in depressed and dysphoric individuals and healthy controls (HC).
- During performance of tasks which demand ignoring of external distracters, trait rumination leads to better selective attention in healthy subjects, but not in depressed individuals. Dysphoric subjects have difficulties ignoring distracting information when they are in a state of rumination, which is not observed in HC.
- The ability to inhibit prepotent responses is negatively related to state rumination in MDD but not in healthy subjects. Trait-rumination findings are mixed.
- The ability to switch attention between different objects or tasks is negatively correlated with state rumination and trait rumination.
- Trait rumination is related to greater costs of working memory (WM) updating, in depressed and non-depressed individuals.

Several attempts have been made to elucidate the causal relationship between CC deficits and rumination. Although several reviews propose causal frameworks explaining the relationship, no consensus has yet been found.

A common explanation for CC deficits in rumination is the resource depletion account. The resource depletion account assumes that rumination influences CC in a negative way. As state rumination involves cognitive processes which demand executive resources, it reduces resources which are available for task related processes. Thereby depletion of resources leads to deficits in task-relevant processing (Whitmer & Gotlib, 2013). The influence of state rumination on CC has sufficiently been demonstrated in studies, inducing ruminative state in experimental settings (Hertel, 1998; Lo, Lau, Cheung, & Allen, 2012; Philippot & Brutoux, 2008; Whitmer & Gotlib, 2012).

Other approaches suggest that decreased CC domains increase habitual tendencies to ruminate. Some reviews concentrate on valence specific deficits in attentional control, due to mood congruent processing in depressive ruminators (Joormann & Gotlib, 2008; Joormann et al., 2007; Koster et al., 2011). Koster, de Lissnyder, Derakshan and de Raedt (2011) developed a model describing the interaction between rumination and CC domains, which promotes depressive episodes. Koster et al. argue that external or internal stressors, which conflict with personal goals, cue rumination. Subsequently two important cognitive mechanisms play a crucial role for the tendency to ruminate; conflict signaling and attentional control. In individuals with little tendency to ruminate, intact conflict signaling indicates, that the currently processed negative or self-referential information is in conflict with self-schemas and thoughts about the self or current goals. Attentional control is the mechanism, which usually turns the attention on goal congruent information, therefore reallocates attentional resources for reappraisal, distraction and mood repair. Impairment of one of the two domains might lead to prolonged rumination as follows: Insufficient conflict signaling makes rumination more probable, because the reallocation of recources is not initiated properly. Reduced attentional control makes it more difficult to stop rumination, as attention cannot sufficiently be withdrawn from negative self referential thoughts. The integration of the resource depletion account into Koster et al.‘s model emphasizes the role of reduced CC as follows: Impaired attentional control is additionally reduced by rumination (Philippot & Brutoux, 2008; Whitmer & Gotlib, 2012), such that reduced CC and increased rumination intensify each other reciprocally.

Koster et al. (2011) assume that CC is not generally impaired in depression, but moreover for inhibition of negative valenced information, as several reviews suggest (Gotlib & Joormann, 2010; Joormann, 2010; Joormann et al., 2007). Joormann, Yoon and Zetsche (2007) already stated that CC deficits in MDD are not caused by a general lack of executive resources. They further argue that deficits occur only in tasks, which give the opportunity to ruminate during task performance. According to Joormann et al. deficits in CC demanding tasks might occur only, when rumination or task unrelated thoughts interfere by depletion of

cognitive resources. Joormann (2010) argues, that depression specific difficulties in the inhibition of negative information leads to increased processing of negative material and decreased expel of negative information from WM. This in turn worsens mood-repair and exacerbates rumination, which increases negative mood and aggravates the negative attentional bias.

Contrary to the suggestion that executive functions are not generally impaired in MDD, a meta-analysis of executive functions in MDD by Snyder (2013) supports the assumption, that depression in fact is associated with deficits in a wide range of executive functions. Snyder found that those deficits are: Firstly, not present only in current depressive episodes, but also in remitted depressive participants, secondly, these impairments are not caused only by lack of vigilance an psycho-motor speed reduction, as other authors suggested (Egeland et al., 2003), but performance differences exceed a simple psychomotor speed reduction. Although a psychomotor speed reduction is present in major depression, it does not explain executive function differences to control groups (Egeland et al., 2003; Snyder, 2013).

Whitmer and Gotlib (2013) found no evidence for the hypothesis that inhibitory deficits are exclusively valence specific. Particularly in HC, CC deficits in rumination seem to be restricted to inhibition of previous relevant task sets and irrelevant information in WM as well as activation of novel information. Healthy ruminators show increased inhibition of distracting information. Whitmer and Gotlib propose that a narrow attentional scope, seen as a trait, moderates the correlation between CC and rumination, i.e. ruminators may tend to process information more selectively and longer. They hence would better be able to ignore distracting information, but have problems attending to new information and to disengage current processes. As noted by Koster et al. (2011), an attentional scope is narrowed by negative mood, thus rumination about negative self-referential information might additionally restrict the attention to negative content. Thus a resource depletion account would still explain CC restriction after induced rumination but problems inhibiting previous relevant information as well as switching and updating difficulties would indicate a susceptibility to ruminate and not be a consequence of rumination (Whitmer & Gotlib, 2013).

Different approaches have been used to prove causal influences of CC on rumination. Connolly et al. (2014) conducted longitudinal study in a sample of 200 adolescent (age 12-13) participants. Results indicated, that reduced executive functions did not predict enhanced rumination in later stages of development. Moreover, higher rumination scores predicted reduced executive function measures, thus the resource depletion hypothesis was supported. Siegle, Ghinassi and Thase (2007) used a cognitive control training (CCT) to enhance CC in a clinically depressed sample. Rumination trait measures were reduced by the training. A part of the CCT was also used by Brunoni et al. (2014) in combination with transcranial direct current stimuliation (tDCS) to enhance CC. The intervention successfully decreased depressive symptoms. In the same sample a reduction of brooding between pre- and posttraining sessions was found. Additionally a correlation between training-performance improvement and brooding reduction was observed (Vanderhasselt et al., 2015).

In summary, the different research approaches have shown that CC and rumination are clearly related in depressive individuals. For healthy samples the interrelation between rumination and CC is less clear, Whitmer and Gotlib (2013) have shown that trait rumination is associated with CC domains which demand flexible operations with information in WM, i.e. disengaging from information or withdraw attention from objects. Contrarily healthy ruminators perform better on tasks demanding selective attention such as ignoring external distracters. Even less is known about the causal relationship between rumination and CC: Training studies have supported the assumption, that increasing CC reduces rumination in depressive subjects. These results do not account for healthy subjects, because in contrast to healthy subjects, depressive individuals are known to show deficits in executive functioning. Therefore further research is necessary to illuminate the causal relationship between rumination and those CC domains which have been shown to be correlated with trait rumination in healthy subjects.

1.1.3. Neuronal correlates of rumination

Disner, Beevers, Haigh and Beck (2011) presented a neuronal model, linking rumination tendencies to depression. According to Disner et al. ventrolateral prefrontal cortex (vlPFC) and dorsolateral prefrontal cortex (dlPFC) activity is

impaired in this setting. Reduced activity may result in decreased CC over limbic structures, such as the hippocampus and the amygdala, which are linked to memory processing and emotion. Several findings regarding rumination and prefrontal cortex (PFC) activity support this model (Cooney, Joormann, Eugène, Dennis, & Gotlib, 2010; Kühn, Vanderhasselt, De Raedt, & Gallinat, 2014; Mandell, Siegle, Shutt, Feldmiller, & Thase, 2014). Other prefrontal structures, that showed altered activity in rumination are linked to inhibition, like the inferior frontal gyrus (IFG; Kühn et al., 2013, 2012, 2014) and self-referential processing, as the medial prefrontal cortex (mPFC; Cooney et al., 2010). According to Disner et al. activation in the mPFC and the amygdala stimulate each other reciprocally, which leads to prolonged self-referential processing in rumination. Increased activity in the amygdala and hippocampus during rumination has been associated to increased activity in the subgenual anterior cingulate cortex (sgACC), which integrates signals from limbic structures to prefrontal areas (Disner et al., 2011). Increased activity in the sgACC is correlated with rumination (Berman et al., 2011, 2014; Cooney et al., 2010), negative bias in MDD (Eugène, Joormann, Cooney, Atlas, & Gotlib, 2010) and deficits in mood regulation (Kohn et al., 2014).

Another important structure, involved in rumination, is the dorsal anterior cingulate cortex (ACC), which according to Disner et al. (2011) is associated with increased emotional processing in depression. Correlations between ACC activity and rumination have been found in different ways: Altered functioning of ACC has been linked to trait rumination (Kühn et al., 2012; Vanderhasselt et al., 2013), state rumination (Piguet et al., 2014) and intrusive thoughts (Kühn et al., 2013). Reduced gray matter in the ACC has been linked to depression (Kühn et al., 2012) and treatment resistance in depression (Machino et al., 2014).

Another approach to neuronal correlates of rumination in depression comes from default mode network (DMN) literature (Marchetti, Koster, Sonuga-Barke, & de Raedt, 2012). Activation in a task negative network, which is activated during resting state (RS), i.e. when no task is performed (Raichle & Snyder, 2007), is negatively correlated to a task positive network, which is deactivated during rest and activated during task involvement. Marchetti, Koster, Sonuga-Barke and de Raedt (2012) link depression, and also rumination to increased activity of the task

negative over the task positive DMN. Cerebral regions of the task negative DMN involve mPFC, rostral ACC (rACC), precuneus, posterior cingulate cortex (PCC), parts of the lateral parietal cortex and mediolateral temporal cortex as well as hippocampus. Associated cognitive processes during activation are mind wandering, self-related processing, autobiographical memory and theory of mind. Activation in these parts impairs successful goal directed, attention demanding processes, provided by activation in the task positive network, consisting of dlPFC, inferior parietal cortex, supplementary motor areas, frontal eye fields and extrastriate cortex. The DMN hypothesis of rumination is supported by studies, linking DMN activity to state (Andersen, Moore, Venables, & Corr, 2009; Berman et al., 2011; Cooney et al., 2010) and trait (Freton et al., 2014) rumination. Switches between activation in task positive and negative DMN are associated with activity in the right frontal insula (Marchetti et al., 2012). Activity in the frontal insula was negatively correlated to rumination in cognitive and emotional tasks (Mandell et al., 2014; Piguet et al., 2014). Insufficient dlPFC function and increased mPFC, amygdala and hippocampal activity are common assumptions of Disner et al. (2011) and the DMN model (Marchetti et al., 2012).

Compared to neuronal imaging studies, so far only little EEG research has been conducted to investigate neuronal correlates of rumination. Lo, Lau, Cheung and Allen (2012) found that neuronal resource allocation is altered by rumination: in a switching task cognitive resource allocation of switching costs was indicated by a late positive potential (LPP). Ruminators displayed stronger expression of the LPP in an emotional task version and after a negative mood induction. Krompinger and Simons (2011) conducted an ERP study, linking N450 amplitudes to the RSQ rumination score. The N450 has been linked to recruitment of attentional control for the inhibition of conflicting information (Larson, Clayson, & Clawson, 2014). Comparing participants with high and low scores of depressive symptoms, they found a correlation between trait rumination and N450 amplitude differences between incongruent and congruent trials. This indicated that depressive trait ruminators needed to allocate more neuronal resources to perform the cognitive tasks sufficiently. In the same study, no correlation was found between trait rumination and the P300 amplitude, which indexes context updating of WM

(Polich, 2007). Aldermann et al. (2014) investigated the N2 amplitude of a flanker task in individuals with MDD. The N2 is also linked to conflict monitoring and inhibition (Larson et al., 2014; Pires, Leitão, Guerrini, & Simões, 2014). They found that the N2 amplitude was smaller for individuals with high rumination scores. In the same study, the P300 was not linked to rumination.

1.1.4. Measuring CC

The *n*-back task is a commonly used task for the measurement of CC (Chen, Mitra, & Schlaghecken, 2008; Owen, McMillan, Laird, & Bullmore, 2005; Snyder, 2013). Commonly the *n*-back task consists of a stream of sequentially presented stimuli, each stimulus representing one trial. The task requires the subject to compare the stimulus of each trial to the stimulus which was presented *n* trials before. Therefore the term *n*-back task subsumes similar tasks with different WM load, which is indicated by the corresponding *n*. *N*-back tasks of different WM-load have been linked to different subprocesses of WM, i.e. matching, replacing and switching (Chen et al., 2008; Watter, Geffen, & Geffen, 2001). Kearney-Ramos et al. (2014) compared different neuropsychological measures to performance in a letter *n*-back task. Performance in the *n*-back task converged with other WM tasks of other sensory modalities, as well as timing and accuracy in visual attention-switching tasks. Analysis revealed an important discrimination from vigilance, psychomotor speed, general language processing, selective attention and sustained attention under distracting circumstances. Thus the letter *n*-back task seems to be more sensitive to WM control and attention switching ability than selective attention, which is, as described above, enhanced in healthy individuals with ruminative response styles.

In the current study, event related potentials (ERP) will be used to measure neuronal correlates of cognitive control via EEG. In accordance to previous studies investigating neuronal effects of cognitive training (Millner, Jaroszewski, Chamarthi, & Pizzagalli, 2012; Oelhafen et al., 2013), typical correlates of CC mechanisms, especially the N2 and P3 and N4 components, will be acquired for neuronal analysis.

N2: The N2 is a fronto-central negative potential peaking between 200 and 350 ms after stimulus onset (Pires et al., 2014). The N2 component has largely been associated with inhibitive behavioral functions, regardless of the used paradigm (Pires et al., 2014), and conflict monitoring (Larson et al., 2014). The majority of findings emerged from studies using the go/nogo task (Bekker, Kenemans, & Verbaten, 2005; Falkenstein, Hoormann, & Hohnsbein, 1999; Lamm et al., 2014; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Rietdijk, Franken, & Thurik, 2014), the Stroop Task (Holmes & Pizzagalli, 2008), or the Flanker Task (Millner et al., 2012; van Veen & Carter, 2002). The N2 amplitude was positively correlated to working memory load in a working memory tasks (Pinal, Zurrón, & Díaz, 2014). The N2 has further been found to be reduced with growing probability of the eliciting stimulus (Bekker et al., 2005) and is enhanced after self-referential stimuli (Knyazev, 2013). A study by Aldermann et al. (2014) depicted also a negative correlation between N2 amplitude size and trait rumination measures. Dipoles of the N2 have been primarily found in the ACC (Bekker et al., 2005; Nieuwenhuis et al., 2003; van Veen & Carter, 2002).

P300: The P300 is another prominent ERP component, with a positive potential peaking between 250 and 500 ms after stimulus onset (Pires et al., 2014). Its distribution can be separated in an early fronto-central P3a and a later parietal P3b. In the current study we will refer only to the P3b. The P300 has been used for neuronal measures of CC (Polich, 2007), inhibitive functions of CC (Pires et al., 2014), as well as WM (Pinal et al., 2014) and context updating (Polich, 2007; Schmitt, Ferdinand, & Kray, 2014). The P300 has also been found to be sensitive to training of CC (Oelhafen et al., 2013; Rietdijk et al., 2014). Oelhafen et al. (2013) used a dual *n*-back task in a training of cognitive control and found transference effects on the P300 of an attention network test. Dipoles of the P300 have been found in the ACC, middle and inferior frontal gyrus and parietal lobule (Li, Wang, & Hu, 2009). Amplitude variations in response to negative valence of stimuli are linked to neuronal sources in the hippocampus (Jiajin, Hong, Antao, & Yuejia, 2007). Stimulation of the left dlPFC increases amplitudes of P300 and also working memory performance (Keeser et al., 2011).

N450: The N450 is a negative frontocentral potential, peaking around 400 ms after stimulus onset (Pires et al., 2014). The N400 has originally been used for neural measures of semantic processing of sentence endings. Recent research enquired the association of the N400 to facilitated processing in WM tasks (Mangina & Sokolov, 2006), as well as to recognition (Kutas & Federmeier, 2011) or to controlled inhibitive processes (Pires et al., 2014) and to conflict monitoring (Krompinger & Simons, 2011; Larson et al., 2014). In the context of conflict detection, the component is named N450, because peak amplitudes show a maximum at 450 ms after stimulus onset. Regarding trait rumination, the N450 has already found to be decreased in high ruminators in a sample with depressive symptomatology (Krompinger & Simons, 2011). Dipoles of the N450 have been found in the ACC (West, Bowry, & McConville, 2004). Involvement of other prefrontal structures seems to variate N450 amplitudes, as observed in a study by Vanderhasselt et al. (2013). Here the peak amplitude of a N450 when inhibiting prepotent reaction to happy, compared to sad faces, was enhanced by tDCS stimulation of the left dlPFC.

1.2. Present Study

Though high effort to find links between CC and rumination has been spent, most conclusions are limited to correlational relationships. Experimentally enhancing CC by training in an experimental group, this prospective study will enlarge correlational statements by adding a causal component. Links between training induced changes in rumination and neuronal as well as behavioral CC changes will be analyzed.

Little research has yet been conducted, linking ERPs to state and trait rumination. In the current study neuronal correlates of CC will be measured via EEG records during the *n*-back task. Inhibition related ERPs will be analyzed to illuminate the interrelations and support behavioral findings.

In the present study, three measures of rumination will be recorded in a nondepressed sample: Firstly, trait rumination measures. Secondly, state rumination without induced rumination, to account for naturally occurring state of rumination during task performance. Thirdly, susceptibility to ruminate

operationalized by changes in state rumination in reaction to a negative mood and rumination induction.

In order to explore the relation between rumination measures and CC, representing a common task for executive functions, a letter *n*-back task will be conducted.

1.2.1. Hypotheses

The present study investigates rumination in several forms: State rumination and trait rumination, as well as neural correlates of CC functions; i.e. conflict monitoring and inhibition related ERPs. Thereby the following hypotheses will be tested:

1. Trait rumination is correlated to inhibition related ERPs.
2. State rumination is correlated to inhibition related ERPs.
3. CC Training changes behavioral measures of CC.
4. CC Training changes inhibition related ERPs.
5. CC Training changes trait rumination.
6. CC Training changes susceptibility to increase state rumination in response to a combined negative mood and rumination induction.
7. Changes in inhibition related ERPs moderate training effects on rumination.

2. Methods

2.1. Research Design

The current study includes correlational and causal analyses of the relationship between CC and rumination. Therefore two experimental sessions were conducted, which consisted of CC measures, EEG recordings, state rumination measures and a combined mood and rumination induction. Between the two experimental sessions, a CCT was implemented to achieve a manipulation of CC. Participants were assigned randomly to either a control group or an experimental group, which conducted the CCT. The time course of the study is illustrated in Figure 1.

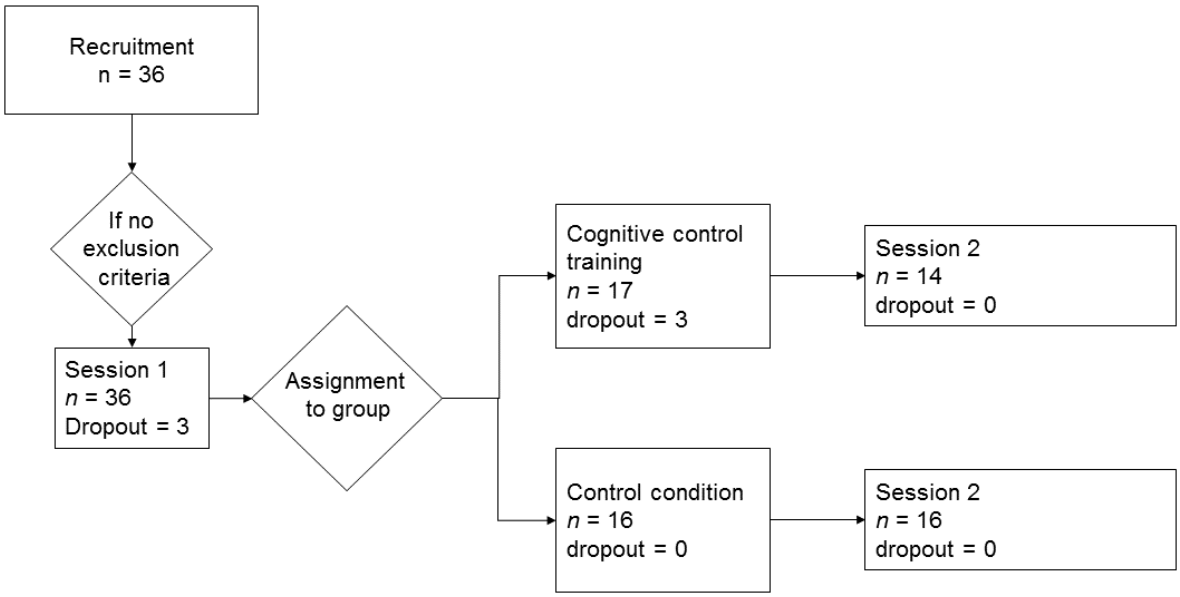


Figure 1. Flowchart illustrating the time course of the present study.

2.2. Participants

A total number of 36 healthy subjects participated in the study. Participants were recruited via advertisements on an internet job platform, flyers and placards inside buildings of the University of Vienna and the Medical University of Vienna. All participants with acute or former psychiatric diseases, medical or psychotherapeutic treatment and any other consummation of psychotropic substances were excluded from the study. To ensure feasibility and reasonability of EEG-recordings, additional exclusion criteria in the selection of participants such as blood, infectious, skin or neurological diseases, any number of cutaneous nevi located on the scalp, skull fractures, hyper-sensitivity of the scalp, claustrophobia and fear of needles applied.

All participants were assigned either to the control or the experimental group, matched by age and gender. The participants were regarded as matching, when they had the same sex and belonged both to the same age group. The age groups were separated by decades as follows: participants aged less than 20 years, aged between 20 and 30 years and or age of more than 30 years. If no complete match was available in the sample, participants were randomly assigned to either the control or the experimental group. If a matching participant was already included in

the sample, the participant was assigned to the opposite group of the respective match.

2.3. Questionnaires

2.3.1. RSQ German version.

Trait rumination was measured using the German version of the RSQ (RSQ-D; Kühner, Huffziger, & Nolen-Hoeksema, 2007), consisting of 32 items, corresponding to 2 subscales, rumination (21 items) and distraction (11 items). Subjects rated their habitual reactions to negative mood on a scale, reaching from 1 (*almost never*) to 4 (*almost always*). The RSQ-D has been validated in a depressive and a control group and internal consistency is given for both subscales, in both samples (rumination subscale: Cronbach's $\alpha \geq .89$; distraction: Cronbach's $\alpha \geq .79$). In the present study only the rumination subscale was used for further analysis, as distraction seems to have unclear divergent relationships to rumination and depression (Nolen-Hoeksema et al., 2008).

Items of the RSQ-D were administered to the participants in an online version using the software Soscipanel (Leiner, 2014). Subjects completed the questionnaire once between the first experimental session and the first CCT session, and once between the last training session and the second experimental session.

2.3.2. Beck's Depression Inventory.

To account for depressive symptoms, the Beck's Depression Inventory II (BDI-II; Beck, Steer, & Brown, 2006) was conducted in both experimental sessions in paper-pencil form.

2.3.3. Positive and Negative Affect Schedule.

In order to observe changes of affect before and after manipulation of mood, the Positive and Negative Affect Schedule (PANAS; Krohne, Egloff, Kohlmann, & Tausch, 1996; Watson, Clark, & Tellegen, 1988) was conducted in paper-pencil form twice in each experimental session, once before and once after the mood and rumination induction.

2.3.4. Visual analog scale mood.

Additionally, three questions concerning mood were administered via a visual analogue scale (VAS). These questions were adopted from Moberly and Watkins (2008). Participants rated their current sadness, anxiety and irritation on a VAS reaching from 1 (*not at all*) to 7 (*very much*). Presentation to the subjects was conducted using the software E-Prime 2.0 (Psychology Software Tools, Inc., Sharpsburg, PA).

2.3.5. State-rumination questionnaire.

State rumination was measured using a state-rumination questionnaire (SRQ), developed by de Jong and Parthe (2009). It consisted of 8 questions which correspond to 2 different subscales, i.e. state rumination and cognitive flexibility, each containing each 4 items. The questionnaire has been validated via factor analysis. Internal consistency was acceptable for the cognitive flexibility scale (SRQ flexibility; Cronbach's $\alpha = .79$) and good for the situational rumination scale (SRQ rumination; Cronbach's $\alpha = .87$). The questions were presented in computer format, using the software E-Prime 2.0 (Psychology Software Tools, Inc., Sharpsburg, PA).

2.3.6. Visual analog scale rumination.

Additionally, a state-rumination measure was adopted from Moberly and Watkins (2008). Participants rated their current focus on feelings and their problems using a VAS, which reached from 1 (*not at all*) to 7 (*very much*). Presentation was conducted using the software E-Prime 2.0 (Psychology Software Tools, Inc., Sharpsburg, PA).

2.4. Combined Mood and Rumination Induction

In order to test the individual susceptibility to ruminate, participants subwent a combined mood and rumination induction (cMRI). Several authors have claimed that healthy subjects do not easily tend to ruminate in an experimental situation, if a rumination induction or mood induction is given alone (de Jong-Meyer et al., 2009; Nolen-Hoeksema & Morrow, 1993; Nolen-Hoeksema et al., 2008). Therefore, a combined approach of rumination, autobiographic and musical mood

induction was chosen, which was adopted from Rood, Roelofs, Bögels and Arntz (2012). The mood induction began with a written and spoken prompt to remember a stressful event, to visualize the event like a movie before the inner eye and to bring up all corresponding feelings and thoughts that were attached to it. The instruction was given by a male voice and combined with a musical mood induction; Prokofiev's "Russia under the Mongolian Yoke" was played at half speed, which has been shown to have a negative effect on mood (Västfjäll, 2002; Westermann, Spies, Stahl, & Hesse, 1996). After 6 minutes, participants were asked to type in a few words describing the situation, which were recorded to check the participants' compliance. Subsequently, the participants were prompted to think again about the event, but this time in a special way, i.e. about the causes, consequences and personal implications. The cMRI lasted in sum about 8 min. The full instruction can be found in Rood et al. (2012). Presentation to the subjects was conducted using the software E-Prime 2.0 (Psychology Software Tools, Inc., Sharpsburg, PA).

2.5. *N*-Back Task

Participants were engaged in a letter variant of the *n*-back task, similar to versions in previous studies examining WM functions in depression (Harvey et al., 2004), their age related variations (Saliasi, Geerligs, Lorist, & Maurits, 2014) and their neural correlates (Braver et al., 1997; Enge, Fleischhauer, Lesch, Reif, & Strobel, 2011; Gevins, Smith, McEvoy, & Yu, 1997). In general the *n*-back task consisted of 4 different subtasks with different *n*-back load (*n*), the 0-back, 1-back, 2-back and 3-back task. Subjects responded to letters, successively presented on the screen, each letter representing one trial. In the 1-, 2- and 3-back task, participants had to decide whether the actual letter was the same letter as *n* trials before (match) or not (mismatch), according to *n* of the current task. In case of mismatch, subjects were asked to respond by pressing the number key 2 on the keyboard, while pressing number key 1 in case of a match. Responses should be made with the right index finger. In the 0-back task, participants decided wither the letter presented was *X* (match) or not *X* (mismatch).

Letter stimuli comprised 20 capital consonants of the German alphabet, that is: *B*, *C*, *D*, *F*, *G*, *H*, *J*, *K*, *L*, *M*, *N* ,*P*, *Q*, *R*, *S*, *T*, *V*, *W*, *Y* and *Z*, as well as *X* in the 0-

back task. The letters appeared for 1 s in the middle of the screen, followed by an inter-stimulus interval (ISI) showing a fixation cross for 1.5 seconds. For each *n*-back task, 2 successive blocks were conducted separated by a pause of 1 min. Each block consisted of 20 match and 40 mismatch trials. The match and mismatch letters as well as the position of matching trials in the line of presentation were selected randomly before each block. The *n*-back subtasks were presented in order of increasing *n*, starting with a 0-back task. The first block of each *n*-back task was preceded by a training task, consisting of 12 stimuli (4 targets), and followed by a summarizing feedback. If the performance indicated that the task had not been fully understood, the option was given to repeat the training. Each training and test block was preceded by 3 additional mismatch trials, which were not included in the analysis. Stimuli were presented using the software E-Prime 2.0 (Psychology Software Tools, Inc., Sharpsburg, PA). The whole n-back task lasted in sum about 30 min.

2.6. Cognitive Control Training

In the present study, the aim to manipulate CC was approached by a cognitive training. The training was adopted from a study by Siegle et al. (2007), who effectively changed patterns of CC in a clinically depressed sample. The training consisted of two primary tasks: the adaptive Paced Auditory Serial Addition Task (PASAT; Gronwall, 1977; Siegle et al., 2007) and Wells Attention Training (WAT; Siegle et al., 2007; Wells, 2000). The training was conducted by each participant privately at home on their personal computers. Before each training session, participants rated their current affect on three VASs, which were the same as in the VAS mood in the experimental sessions described above (Moberly & Watkins, 2008). Additionally, they completed three items regarding state of rumination in the last 24 hours, which were adopted from Takano and Tanno (2011). Participants rated (a) how much they had been focused on their feelings and problems, (b) the unpleasantness of their thoughts and (c) how controllable their thoughts had been, on a seven-point scale, reaching from *not at all* (1) to *very much* (7). In the experimental group, these questions were followed by one of the two training tasks, PASAT or WAT. The order of tasks was different from session to session, but balanced so that three sessions began with the PASAT and three began with

WAT. The control group did not conduct the PASAT and WAT, but completed the same questions during each of the six sessions. They used the same software but completed only two five-minute blocks of the PASAT, one in the first and one in the last training session. This procedure was chosen to create the impression of a cognitive training in the control group, thereby increasing the blindness of participants in the control group.

The training was programmed using the Java-based open-source programming framework Tatool (von Bastian, Locher, & Ruflin, 2012). Sessions were self-administered by the participant at home using the open source software Tatool (von Bastian et al., 2012). In the experimental group six sessions were conducted, each session lasted about 35 min. In the control group participants also conducted 6 session, the first and last session lasted about 10 minutes, the other 4 sessions lasted about 5 minutes. After completion of a training session, results of the questionnaires and PASAT responses were uploaded to a web server located at the University of Vienna running Tatool online (von Bastian et al., 2012). This enabled the researcher to observe the progression of each participant's training sessions.

2.6.1. PASAT.

The PASAT consisted of a continuous stream of acoustically presented numbers. The participant's task was to calculate the sum of the ongoing number and the last number presented. Thereby each presentation of a number represented one trial. A circle of number buttons was presented visually on the screen, allowing the person to click on the number according to the result of the last addition. The speed by which the task was conducted varied with performance of the participant; the task became faster, when a performance was good and slowed down when the performance became worse.

The first session of the training started with an instruction and a prompt to use earphones for the training. The subject was told that the task is difficult and that in case of a disruption of attention, the person should reallocate attention back on the task. From the third session on, the instruction was reduced to an illustration of the basic rules. In the first training session the ISI lasted 3 seconds. Each time a

participant had four consecutive trials correct, the ISI became 100 ms shorter and when four consecutive trials were incorrect, the ISI grew by 100 ms. In the following training sessions the task started with the median ISI of all trials in all sessions previously conducted by the participant. Each training session included 3 blocks of PASAT, with an equal duration of 5 minutes each, separated by a pause of one minute.

2.6.2. Wells Attention Training.

WAT consisted of several attention tasks, which required the participant to concentrate on different sounds in an environment of simultaneously presented noises, in this case bird voices. Before the task began, participants were asked to use earphones, keep their eyes open and to listen carefully to the given instructions. Then a screen appeared, presenting a colored dot on white background. The task instruction was given acoustically by a male human voice at the same time as the bird voices were presented. The instructor's voice directed the participants attention to different sounds: firstly, varying sounds had to be focused for a certain time while the other sounds had to be ignored; secondly, the attention was directed to switch quickly from one sound to another; and thirdly, the participant was asked to widen the focus of attention and to perceive all sounds simultaneously at once. During the WAT no behavioral responses were collected.

The stimuli used in this experiment were the same as those used in the original study by Siegle et al. (2007), involving 13 different bird sounds, of which four had to be focused plus one tapping sound. The sounds were presented on different channels of the sound output system, so they appeared either from the right or from the left earphone. The distribution of target sounds to the earphones varied from session to session and was balanced regarding the sound direction, i.e. the same number of sounds was presented on each earphone and sounds occurred from different directions in each session.

2.7. Procedure

Participants were invited for experimental EEG sessions at the EEG laboratory of the biological department of the psychological faculty of the University of Vienna.

In the beginning of the first experimental session, exclusion criteria were assessed and subjects gave informed consent, if none present. Subsequently, participants filled in the Edinburgh Handedness Inventory (Oldfield, 1971) and for reasons of data-privacy protection created their own personal code which was used for further data naming. Subsequently, the EEG-electrodes were attached to the participants scalp using an equidistant cap (model M10, EASYCAP GmbH, Herrsching, Germany) and participants were placed in a separate room, surrounded by a faraday cage, with soundproof walls and dimmed light. The test person was seated 70 cm in front of a 21” CRT monitor (Sony GDM-F520), with a refreshing rate of 75 Hz. Immediately before the computer experiment started, subjects filled out the BDI-II (Beck et al., 2006) in paper-pencil form.

The following computer test consisted of two cognitive tasks and mood and state rumination measures. The first part of the computer session started with the previously described *n*-back task. The *n*-back task was followed by a short Navon Letter Task (Braboszcz et al., 2013; Navon, 1977). The second part of the session began with a baseline evaluation of affect and state rumination. This included PANAS in paper-pencil form, and VAS mood, VAS rumination and the SRQ in computer-administered format. Subjects also performed an internal counting task, interrupted by short questions about distracting thoughts and mind-wandering. Subsequently the cMRI was conducted, followed again by the state rumination and mood measures. Thus, the whole protocol, i.e. questionnaires and counting task were performed once before and once after cMRI. At the end of the experimental session, subjects underwent a positive mood induction, starting with a prompt to think about a positive event in the past. This induction was supported by a piece of music favored and brought to the session by the respective subject.

After the first experimental session, participants received the training software and were advised to install the training on their own computer at home. Participants agreed to conduct the training a total number of six times within two weeks and not more than once a day and three times a week. They were also provided with an internet address linking to an individual training calendar. It was updated by the researcher each time a training session was completed and showed the optimal date for the succeeding training session. After completion of the fourth training

session, the participants were invited to make an appointment for the second experimental session.

The second experimental session after training was conducted exactly as the first session.

2.8. EEG Data Acquisition

During the two experimental sessions, electrophysiological data were acquired using a set of 61 Ag/AgCl electrodes mounted on an equidistant cap (model M10, EASYCAP, GmbH, Herrsching, Germany). The Cz was fixed at the center of the scalp, which was defined as half of the distance between nasion and inion, as well as half of the distance between both mastoid bones. The ground electrode was placed on the nasion. For purposes of eye movement control, two additional electrodes were placed below and above the left eye. The electrophysiological signal was amplified by a DC amplifier set-up (NeuroPrax, neuroConn GmbH, Ilmenau, Germany) and digitalized online at a sample rate of 500 Hz.

2.9. EEG Data Preprocessing

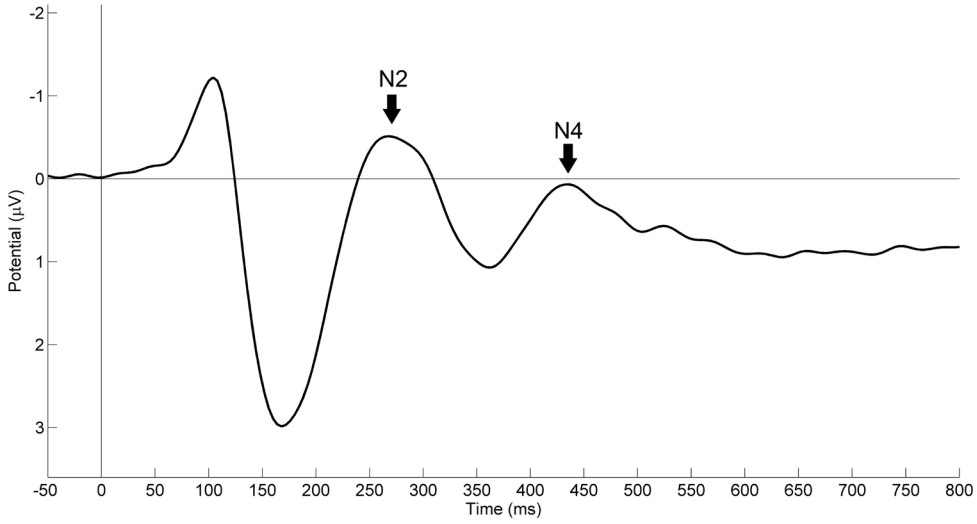
Offline data processing was conducted using EEGLAB 12.0.2.1b (Delorme & Makeig, 2004), on Matlab R2012b (The MathWorks, Inc., Natick, MA, USA). Data were sampled down to 250 Hz and filtered by a 30 Hz lowpass filter and a 0.1 Hz highpass filter. Trials of the *n*-back task were extracted in segments; each limited by the time interval of -1 s before and 2 s after stimulus presentation.

For purposes of artifact rejection, independent component analysis (ICA; Bell & Sejnowski, 1995) was conducted. Resulting components representing ocular movements and blinks were visually identified and rejected. For further analysis, epochs were shortened to the time limits 200 ms before and 1000 ms after stimulus onset. For each trial a baseline correction was conducted: the mean amplitude of the time window of 200 ms before stimulus onset was calculated and subtracted from every data point of the corresponding epoch. For additional artifact rejection an algorithm was applied to the data removing all epochs with a data range exceeding +/- 70 µV. Trials without artifacts were averaged per group and *n*-back condition.

2.10. ERP Identification

To identify time windows for peak determination, grand averages including each group and condition were inspected at Fz and Pz (Figure 2). The *n*-back task revealed a N2, P3 and N4 component. Time windows of maximum peak values were identified at following times after stimulus onset: 180 ms to 380 ms for N2, 280 ms to 430 ms for P3 and 380 to 600 ms for N4.

Fz



Pz

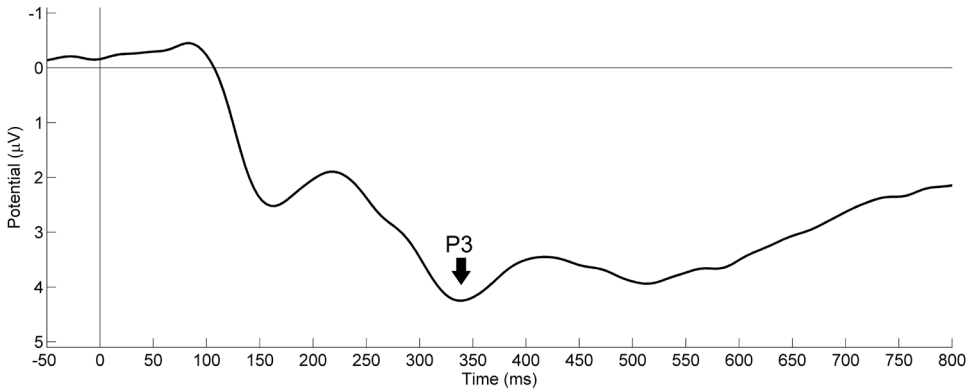


Figure 2. Grand averages of the *n*-back task across all subjects, times and conditions at Fz and Pz.

According to the grand average maximum amplitudes, spatial signal distribution was inspected at times 280 ms for N2, 380 ms for P3 and 440 ms for N4. ERP maps revealed a frontocentral minimum for the N2 amplitudes, a parietal maximum for P3 components and frontal minimum for N4. Therefore midline electrode Fz was selected to measure N2 and N4 and Pz for P3 amplitudes (Appendix).

Consequently the N2 was defined as the minimal peak amplitude in the time window 180 ms to 380 ms after stimulus onset. According to the sequence of the components, the P3 was defined as the maximal peak between latency of N2 and 440 ms after stimulus onset and N4 amplitude between P3 latency and 600ms after stimulus onset, for each participant, time and condition separately.

2.11. Data Analysis

Behavioral measures were recorded in term of accuracy and retention time (RT) for each *n*-back condition. Accuracy scores were calculated as the total percentage of correct answered trials separately for each *n*-back condition. For calculation of RTs, only correct answered trials were included with RTs larger than 200 ms and shorter than 1500 ms post stimulus presentation. Scores correspond to the mean RT of the selected trials.

Effects of the mood and rumination induction were validated with *t*-tests for dependent measures. Considering accumulation of type 1 error, *p*-values were corrected using the Bonferroni method. In order to evaluate susceptibility to ruminate, changes induced by the cMRI were further evaluated using difference scores. Differences between post cMRI and pre cMRI measures were calculated for the SRQ rumination subscale (dSRQ rumination), SRQ flexibility subscale (dSRQ flexibility) and for the VAS rumination (dVAS rumination). Positive values indicate increases after cMRI. Correlations of state rumination were calculated for scores before cMRI, indexing state of rumination during the *n*-back task.

2.11.1. Correlations between CC and rumination measures

The relationship between rumination scores and ERPs or behavioral measures related to CC was investigated using the Pearson correlation coefficient, if data

was normally distributed. Otherwise, Spearman correlations are reported. Correlations of posttraining measures were conducted with partial correlations, including group as a control variable to account for training-related bias. Correlations with ERPs were conducted two tailed, as no converging findings about relationship with ERPs exist. Correlations with behavioral measures were conducted one-sided, expecting longer RTs and lower accuracy for higher rumination scores.

Therefore, each combination of rumination and ERP component comparison included 8 correlations, i.e. 2 (time) × 4 (*n*-back load). Corresponding *p*-values are reported as significant, if they passed the Holm-Bonferroni correction (Holm, 1979) at $\alpha = .05$. The statistical software R was used for alpha-correction (R Development Core Team, 2008). Measures of CC included RTs and *n*-back accuracy on the behavioral level and N2 amplitude, P3 amplitude and N4 amplitude on the neuronal level. Measure of trait rumination was the RSQ-D rumination subscale. Measures for naturally occurring state of rumination were SRQ rumination pre cMRI, SRQ flexibility pre cMRI and VAS rumination pre cMRI. For susceptibility to ruminate, dSRQ rumination, dSRQ flexibility and dVAS rumination scores were included.

2.11.2. Training effects

In order to shed light on the question, if the CCT had an effect on rumination and CC, changes after training in behavioral measures, ERP amplitudes as well as rumination trait, state and susceptibility measures were analyzed using mixed-design analysis of variance (ANOVA). For CC measures, within-subject factors were time (Session 1 and Session 2) and condition (0-, 1-, 2-, and 3-back). Rumination analysis included only time as within-subject factor. Between-subject factor was group (control vs experimental). Training effects are represented by Group × Time interactions. If assumption of homogeneity of variance was violated, dependent *t*-tests were applied to data separately. When time effects or interactions with factor time were found, dependent *t*-tests were calculated to highlight significant changes in the different groups. To account for accumulation of Type 1 error in the post hoc dependent *t*-tests, significance was reported with a

corrected threshold of $p < .00625$, holding an overall $\alpha = .05$ for eight comparisons of Group (2) × Condition (4).

2.11.3. Common changes of CC and rumination by training

Intending to find out if changes in CC moderate training induced changes in rumination measures, hierarchical multiple regression models were calculated. A reference model of training effects on rumination measures was compared to enlarged models, including training effects and CC changes of neuronal and behavioral measures. Regression models were analyzed only for rumination measures, which showed significant changes after training in the mixed-design ANOVA or dependent *t*-test.

The rumination differences between sessions were entered as dependent variables. The reference model included only group as independent variable, representing training effects. In order to check if training effects were due to changes in neuronal and behavioral data, for every variable the reference model was enlarged separately by adding data of all *n*-back conditions at once to the model. For this purpose, changes in each *n*-back measure were calculated, subtracting Session 1 values from Session 2 values. Conclusively the corresponding models included five predictors, with group and four variables presenting behavioral or neuronal changes in all four *n*-back tasks. Improvement of the reference model by the added data is reported for every measure. If the reference model was improved significantly ($p < .05$) or with a trend towards significance ($p < .10$) by the added variables, all coefficients will be reported too. It is important to note, that because of high correlations between *n*-back measure changes across session (Appendix), the coefficients of the performance and neuronal data are not interpretable. Nevertheless, the influence of the parameters of factor group, which indexes training effects on rumination, can be evaluated after controlling for changes in the other CC related predictor variables. Additionally correlations between training related changes in ERPs as well as behavioral performance in the *n*-back task and changes in rumination measures in the experimental group were investigated.

3. Results

3.1. Descriptives

From the total sample of 36 participants, three subjects with BDI-II scores higher than 20 at the beginning of the study were excluded from analysis. The final sample consisted of 15 male and 18 female participants, aged between 19 and 35 years (M = 25.36, *SD* = 4.22). All subjects were right handed, according to the Edinburgh Inventory (Oldfield, 1971). For analysis of the second experimental session, three male participants could not be included, because they withdrew consent for participation after the first session. Therefore, Session 2 measures could be analyzed in a total sample of only 30 participants (18 female).

In the final analysis set, the experimental group included 14 (nine female) and the control group 16 (nine female) participants. The two groups did not differ significantly in distribution of gender, $\chi^2(1) = 0.20$, $p = .72$. Training duration, age and BDI-II scores did not differ in the two groups. Of note, significant higher baseline RSQ-D measures were observed in the experimental group (Table 1). In the experimental group, all 14 participants completed the training six times and the training duration varied between 9 and 23 days. Two participants in the control group completed only five sessions and in this group duration between the first and last training session varied between 6 and 27 days.

Table 1:
Mean Sample Characteristics of Different Groups

	Control (*n* = 16)	Experimental (*n* = 14)
	M (*SD*)	*M* (*SD*)
Age	24.94 (4.40)	23.07 (3.97)
BDI-II	5.50 (5.37)	7.14 (4.69)
RSQ-D*	44.63 (8.33)	53.29 (10.00)
Training duration (days)	13.31 (5.97)	14.79 (4.59)

Note. Participants with BDI-II score >20 and dropouts between pre- and post-training sessions are excluded. BDI-II = Beck's Depression Questionnaire. RSQ-D = Response Styles Questionnaire German version rumination subscale. *M* = mean. *SD* = standard deviation.
*group means differ significantly ($p < 0.05$).

3.2. Mood Induction

Each mood and rumination measure showed significant differences before and after the cMRI except for measures in the SRQ flexibility subscale in the second session, $t(29) = 2.53$, $p = .21$. The induction decreased the positive affect scale of PANAS and increased the negative affect scale of PANAS as well as negative affect scores measured by the VAS. SRQ rumination measures were increased by cMRI. The cognitive flexibility subscale was decreased by the cMRI (Table 2).

Table 2
Mood and State Rumination Measures Before and After cMRI

	Pre cMRI		Post cMRI	
	M	(*SD*)	*M*	(*SD*)
Session 1				
SRQ rumination**	97.57	(61.98)	167.47	(97.11)
SRQ flexibility**	293.53	(69.55)	242.20	(77.85)
VAS mood**	37.4	(54.83)	109.67	(69.60)
VAS rumination**	53.9	(42.90)	117.83	(52.66)
PANAS negative**	12.73	(3.35)	16.13	(6.19)
PANAS positive**	28.83	(7.21)	23.3	(8.07)
Session 2				
SRQ rumination*	92.13	(80.63)	134.93	(82.92)
SRQ flexibility	283.1	(71.16)	254.40	(73.93)
VAS mood**	33.40	(39.47)	93.83	(62.30)
VAS rumination**	43.97	(44.94)	111.47	(35.04)
PANAS negative**	11.83	(2.93)	14.03	(3.89)
PANAS positive**	29.79	(7.31)	23.9	(7.39)

Note. Comparisons correspond to *t* tests. All *p* values are corrected using Bonferroni method, i.e. were multiplicated by factor 12, corresponding to the number of comparisons. For Session 2, PANAS measures were conducted with $n = 29$, because of one uncompleted questionnaire. For all other comparisons $n = 30$. Asterisks indicate significant mean differences. SRQ = state-rumination questionnaire. VAS = visual analog scale. PANAS = Positive And Negative Affect Schedule. cMRI = combined mood and rumination induction.
$^{+}p < 0.10$. $^{*}p < 0.05$. $^{**}p < 0.01$.

3.3. Correlations

3.3.1. Behavioral.

No significant correlations between *n*-back accuracy and any rumination trait, state or change scores were found. Nevertheless, a moderate positive correlation between state flexibility and accuracy scores in the 3-back task of the first session was found, with a trend towards significance, $r = .40$, $p < .09$. Another trend

towards significance was observed regarding a moderate negative correlation between 1-back accuracy and RSQ-D scores in the first session (Table 3). No other trends were observed and correlations had different directions across *n*-back conditions.

The RTs of the 0-back task correlated significantly with RSQ-D scores in the pre training session (Figure 3). This correlation was positive and had moderate size (Table 3). All other correlations in this session were positive and revealed small effect sizes. In the second session, correlations ranged from small to moderate and had varying directions. No other rumination scores, neither trait nor state nor changes by cMRI revealed significant correlations with RT in the *n*-back task. Coefficients were heterogeneous across *n*-back conditions and revealed small effect sizes.

Table 3
Correlations of Behavioral N*-back Measures With RSQ-D Rumination Subscale*

		ACC	RT
		r	*r*
Session 1			
	0-Back	-.03	.46*
	1-Back	-.40^{+}	.31
	2-Back	.19	.07
	3-Back	.23	.05
Session 2			
	0-Back	-.08	.38
	1-Back	-.08	.32
	2-Back	-.07	-.02
	3-Back	.25	-.08

Note. Session 1: $n = 32$. Session 2: $n = 30$. Correlations of Session 2 are partial correlations, controlling for group. *r* = Pearson's correlation coefficient. For each behavioral measure, *p*-values are corrected using Bonferroni-Holm method. ACC = accuracy. RT = reaction time. *r* = Pearson's correlation coefficient. RSQ-D = Response Styles Questionnaire German version rumination subscale.
$^{+}p < 0.10$. $^{*}p < 0.05$

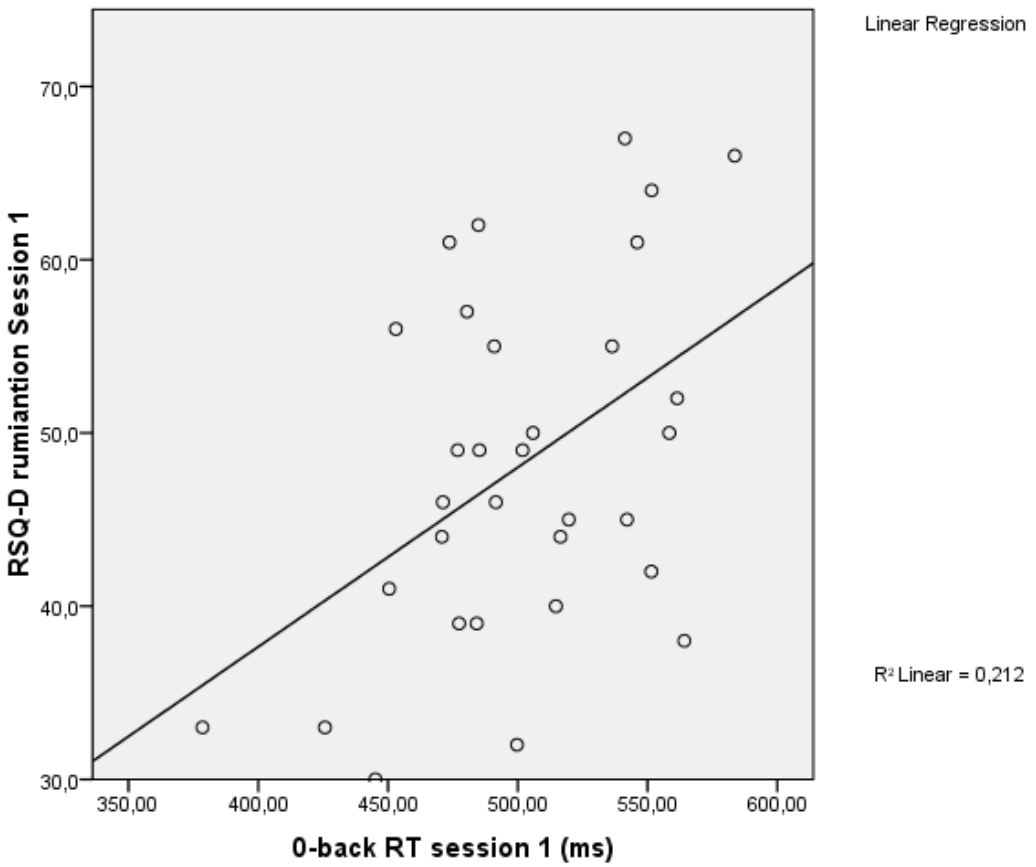


Figure 3. Relationship between RT in 0-back task and trait rumination in Session 1. RSQ-D = Response Styles Questionnaire German version. RT = reaction time.

3.3.2. N2.

3.3.2.1 Trait rumination.

The N2 amplitudes did not show significant correlations with RSQ-D measures (Table 4). Directions of correlation coefficients were negative, except for one small positive correlation in the 1-back task of the first session. Sizes varied between small and moderate across *n*-back conditions.

3.3.2.2 State rumination.

The N2 amplitudes did not show significant correlations with state rumination. Directions of correlation coefficients with SRQ rumination were negative and small. Sizes of correlations with other measures varied between small and medium and showed no overlapping directions.

Table 4
Correlations of ERP Amplitudes With RSQ-D Rumination Subscale

	N2 *r*	P3 *r*	N4 *r*
Session 1			
0-Back	-.13	.09	.20
1-Back	.09	.18	.11
2-Back	-.07	.18	-.09
3-Back	-.21	.21	-.06
Session 2			
0-Back	-.37	-.16	-.27
1-Back	-.23	-.11	-.09
2-Back	-.34	-.25	-.01
3-Back	-.28	-.20	.10

Note. In Session 1 $n = 32$. In Session 2 $n = 30$. Correlations in Session 2 are partial correlations, controlling for group. For all rumination measures, *p*-values are corrected using Bonferroni-Holm method. *r* = Pearson's correlation coefficient. RSQ-D = Response Styles Questionnaire German version rumination subscale
$^{+}p < 0.10$. $^{*}p < 0.05$

3.3.2.3 Susceptibility to ruminate.

Correlations between mood induced changes in state rumination and the N2 amplitudes are displayed in Table 5. Mood induced changes of SRQ rumination correlated significantly and strong with the N2 amplitude in the 2-back task of the second experimental session (Figure 4). Nonsignificant trends occurred for moderate negative correlations in the 2-back task of the first session and in the 0-back task of the second experimental session. All correlations were negative by direction, thus indicating that higher (more negative) N2 amplitudes correlate with increased dSRQ rumination measures.

Mood induced changes of the VAS rumination did not correlate significantly with the N2 amplitude. Trends towards significance could be observed for negative moderate correlations in the 0-back and 2-back condition of the first experimental session. Direction of correlation coefficient differed in both sessions; the first session revealed negative, the second session positive correlations.

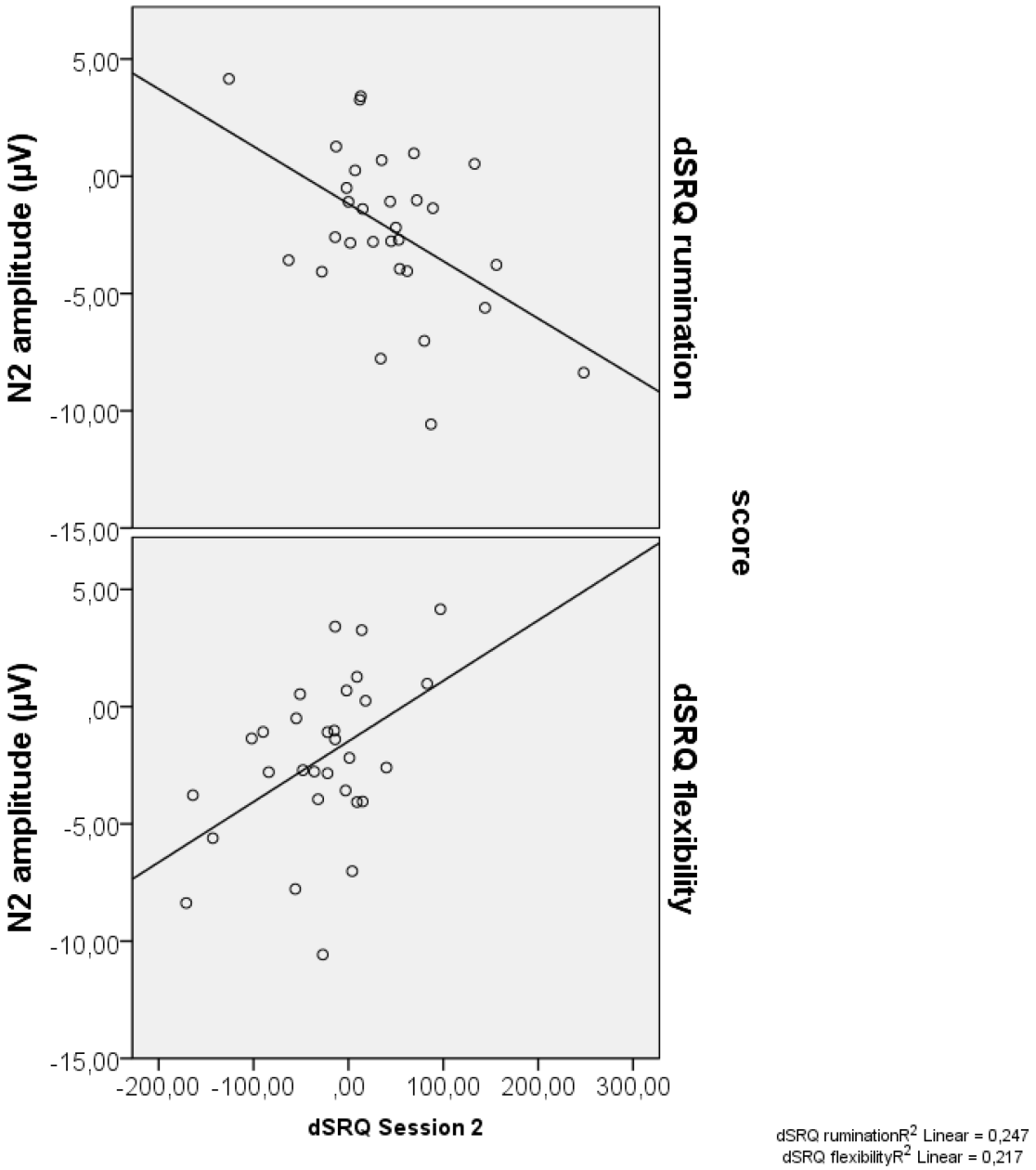


Figure 4. Relationship between mood induced changes in SRQ scores and the N2 amplitudes of the 2-back task in Session 2. SRQ = state rumination questionnaire. dSRQ = difference in SRQ score between post- and pre-cMRI. cMRI = combined mood and rumination induction.

In the second experimental session, dSRQ flexibility showed a strong significant positive correlation with the N2 amplitudes in the 2-back task (Table 5) and a positive trend could also be observed in the 0-back task. All correlation coefficients

were positive, indicating that a greater N2 amplitude is related to greater decreased of situational flexibility measure after cMRI (Figure 4).

Table 5
Correlations of the N2 Amplitudes With State Rumination Changes After cMRI

	dSRQ rumination	dSRQ flexibility	dVAS rumination
	r	*r*	*r*
Session 1			
0-Back	-.29	.25	-.45^{+}
1-Back	-.18	.14	-.30
2-Back	-.44^{+}	.22	-.43^{+}
3-Back	-.34	.29	-.22
Session 2			
0-Back	-.45^{+}	.47^{+}	.05
1-Back	-.25	.28	.13
2-Back	-.50*	.51*	.05
3-Back	-.41	.36	.06

Note. Session 1: *n* = 33. Session 2: *n* = 30. Correlations of Session 2 are partial correlations, controlling for group. For all rumination measures, *p*-values are corrected using Bonferroni-Holm method.
SRQ = state rumination questionnaire. dSRQ = difference between post and pre-cMRI SRQ scores. cMRI = combined mood and rumination induction. VAS = visual analog scale. dVAS = difference between post- and pre-cMRI VAS scores.
$^{+}p < 0.10$. $^{*}p < 0.05$

3.3.3. P3.

3.3.3.1 Trait rumination.

RSQ-D measures did not correlate significantly with the P3 amplitude and correlations yielded only weak or small coefficients with different signs (Table 4).

3.3.3.2 State rumination.

A nonsignificant positive trend between VAS rumination measure and P3 amplitude was found in the 3-back condition of the posttraining session. Across session and conditions, all correlations had a positive sign and strengths ranged from weak to moderate (Table 6).

Correlations between the SRQ rumination subscale and the P3 amplitude were homogenously positive, except for a negative correlation in the 0-back task of the second session. Strengths ranged from weak to moderate, but no significance or trend could be observed (Table 6).

Correlations between the SRQ flexibility subscale and the P3 amplitude were homogenously negative, except for one positive correlation in the 0-back task of the second session. Strengths of relationships were weak or small and no significance or trend could be observed (Table 6).

Table 6
Correlations of P3 Amplitudes With Rumination State Measures.

	SRQ rumination	SRQ flexibility	VAS rumination
	r	r	r
Session 1			
0-Back[a]	.29	-.27	.01
1-Back	.42	-.27	.04
2-Back	.38	-.20	.17
3-Back	.41	-.26	.17
Session 2			
0-Back	-.05	.16	.19
1-Back	.10	-.02	.35
2-Back	.14	-.11	.30
3-Back	.20	-.09	.47^{+}

Note. Session 1: n = 33. Session 2: n = 30. Correlations of Session 2 are partial correlations, controlling for group. For all rumination measures, p-values are corrected using Bonferroni-Holm method. SRQ = state rumination questionnaire. VAS = visual analog scale. r = Pearson's correlation coefficient.
[a]Spearman's correlation coefficient
$^{+}p < 0.10$. $^{*}p < 0.05$

3.3.3.3 Susceptibility to ruminate.

Changes of VAS rumination measures did not correlate significantly with P3 amplitudes and no trends were observed. The correlation coefficients were homogenously negative by direction across conditions and sessions. Strengths reached from weak to moderate.

Changes of SRQ rumination subscale did not yield significant correlations or trends. However, directions of correlation were unitarily negative, with strengths ranging from small to moderate.

Mood-induced changes of SRQ flexibility were not significantly correlated with P3 amplitude, nor were any trends observed. Signs of coefficients were heterogeneous and strength varied between weak and small.

3.3.4. N4.

3.3.4.1 Trait rumination.

The N4 amplitude did not correlate significantly with the RSQ-D rumination subscale (Table 4). No significant correlations or trends were found, the direction of relationship was diverse across sessions and conditions, the strength of correlation varied between weak and small.

3.3.4.2 State rumination.

Scores of the VAS rumination did not show any significant correlations or trends with the N4 amplitude. Directions of the correlation coefficients were heterogeneous across conditions and sessions. The strength of relationship varied between weak and small.

The SRQ rumination subscale did not show any significant correlation with the N4 amplitude, nor were any trends observed. In all sessions and conditions the direction of the relationship was positive, indicating larger rumination scores for participants with smaller N4 amplitudes in the present sample. The correlation coefficients yielded weak up to small sizes.

The SRQ flexibility measures were not significantly correlated to N4 amplitude in any *n*-back condition. No trends were found either and all correlation coefficients had a different sign. Sizes of effect varied between weak and small.

3.3.4.3 Susceptibility to ruminate.

Regarding changes in the VAS rumination measure after mood induction, no significant correlation was found and no trend or homogeneous directions across conditions and sessions could be observed. Sizes of the correlation coefficients ranged from weak to moderate.

Changes found in the SRQ rumination subscale were not significantly correlated to the N2 amplitude. Nevertheless, trends towards significance were observed in the 0-, 2- and 3-back task of the second session. All correlations were negative and had medium size. Across conditions and sessions all correlations were negative, indicating that increased N4 amplitudes (more negative) correlate with increases

of SRQ rumination scores after cMRI. The observed correlations were small or medium (Table 7).

Table 7
Correlations of N4 Amplitudes With State Rumination Changes After cMRI

	dSRQ rumination	dSRQ flexibility	dVAS rumination
	r	*r*	*r*
Session 1			
0-Back	-.18	-.02	-.34
1-Back	-.15	.08	-.20
2-Back	-.34	.15	-.29
3-Back	-.16	.20	-.13
Session 2			
0-Back	-.45^{+}	.50*	.02
1-Back	-.15	.22	.04
2-Back	-.48^{+}	.60**	-.02
3-Back	-.45^{+}	.52*	-.03

Note. Session 1: n = 33. Session 2: n = 30. Correlations of Session 2 are partial correlations, controlling for group. For all rumination measures, *p* values are corrected using Bonferroni-Holm method.
SRQ = state rumination questionnaire. dSRQ = difference between post and pre-cMRI SRQ scores. cMRI = combined mood and rumination induction. VAS = visual analog scale. dVAS = difference between post- and pre-cMRI VAS scores. *r* = Pearson's correlation coefficient.
$^{+}p < .10$. *$p < .05$. **p < .01.

Changes in SRQ flexibility subscale after cMRI correlated significantly with N4 amplitudes in the 0-, 2- and 3-back task of the second session. Correlations were positive and had strong sizes. All correlations were positive across sessions and conditions, except for a small negative correlation in the 0-back task of the first session. Positive correlations indicate smaller (i.e. more positive) amplitudes for individuals who report less decrease in SRQ flexibility scores after mood induction (Figure 5).

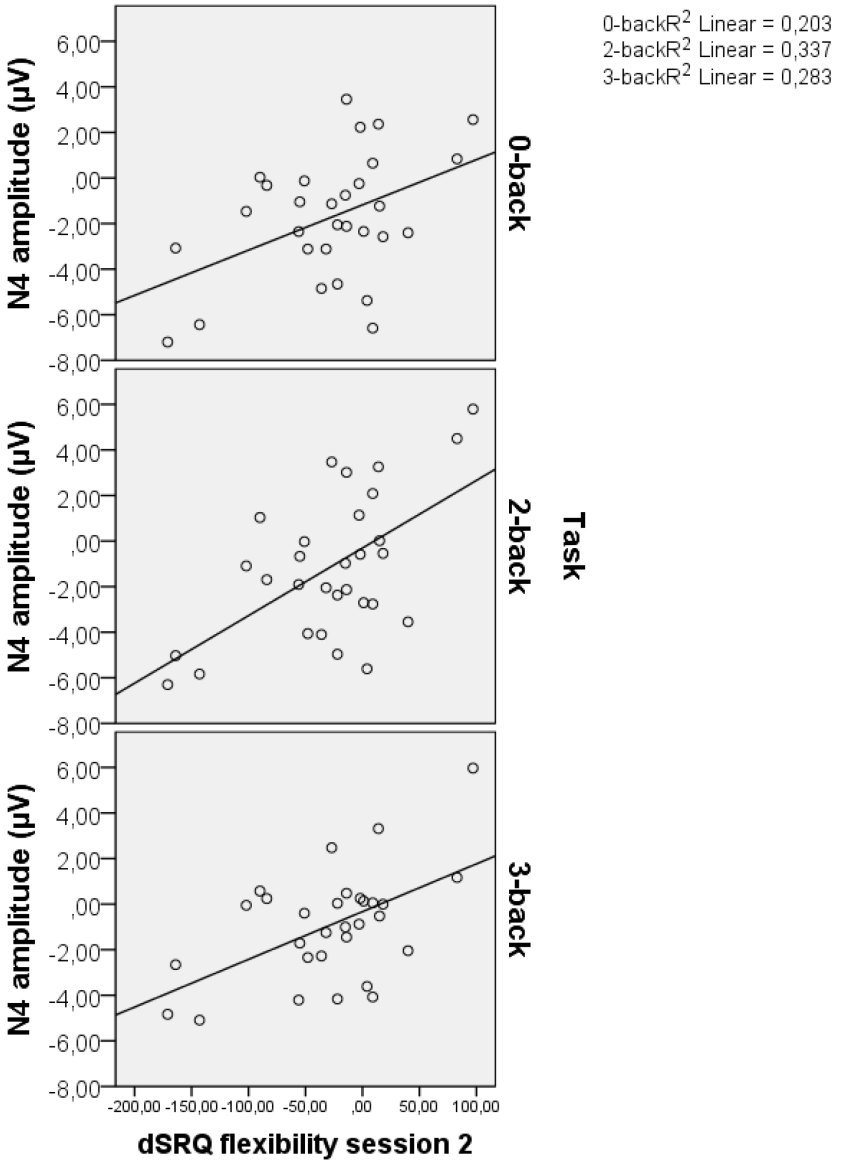


Figure 5. Relationship of dSRQ flexibility scores and N4 amplitudes in 0-, 2-, and 3- back task. SRQ = state rumination questionnaire. dSRQ = difference between SRQ measures post- and pre-cMRI. cMRI = combined mood and rumination induction.

3.4. Training Effects

3.4.1. Behavioral.

Regarding the comparison of behavioral performance measures in the *n*-back task, ANOVA of accuracy scores showed a significant main effect of time, $F(1,28) = 4.34$, $p < .05$, *n*-back load, $F(2.05,57.48) = 130.11$, $p < .01$ and a non-significant trend towards an main effect of group, $F(1,28) = 3.98$, $p < .06$. These findings

indicate that accuracy increased over time and that the control group performed slightly better. A mixed effect of Condition × Group was found, $F(2.05,57.48) = 4.59$, $p < .02$ and a trend towards a three way interaction of Condition × Time × Group was observed, $F(2.34,65.56) = 2.66$, $p < .07$. Post hoc *t*-tests analyzing session differences in each group and condition revealed that significant increases of accuracy could be observed only in the control group during the 3-back condition, $t(15) = -3.35$, $p < .04$, $r = .65$, but not in the experimental group.

Analysis of RT revealed main effects for time, $F(1,28) = 58.42$, $p < .01$ and *n*-back load, $F(1.73,48.54) = 78.53$, $p < .01$. RT decreased over the two sessions, and increased with an accelerating *n*-back load. A significant interaction effect between Condition × Time was found, $F(2.24, 62.76) = 5$, $p < .01$. Training effects were revealed in a trend towards significance regarding interaction between Time × Group, $p < .09$, $F(1,28) = 3.26$. Training effects in the *n*-back task are illustrated in Figure 6.

Post hoc *t*-tests revealed that significant changes were observed in the experimental group across all *n*-back conditions (Table 8). In contrast, the control group revealed only one significant effect which was measured in the 2-back condition. All effects caused a reduction of RT over sessions.

Table 8

Post hoc Dependent T-*Tests of Changes in RT Between Sessions*

	Control Group		Experimental Group	
RT	*t*(15)	*r*	*t*(13)	*r*
0-back	3.07+	.62	5.42***	.83
1-back	3.04+	.62	5.57***	.84
2-back	3.5*	.67	6.29***	.87
3-back	2.37	.52	4.51**	.78

Note. All *p* values are Bonferroni corrected. RT = reaction time. *r* = Pearson's correlation coefficient. $^{+}p < .10$. *p < .05. **p < .01. ***p < .001.

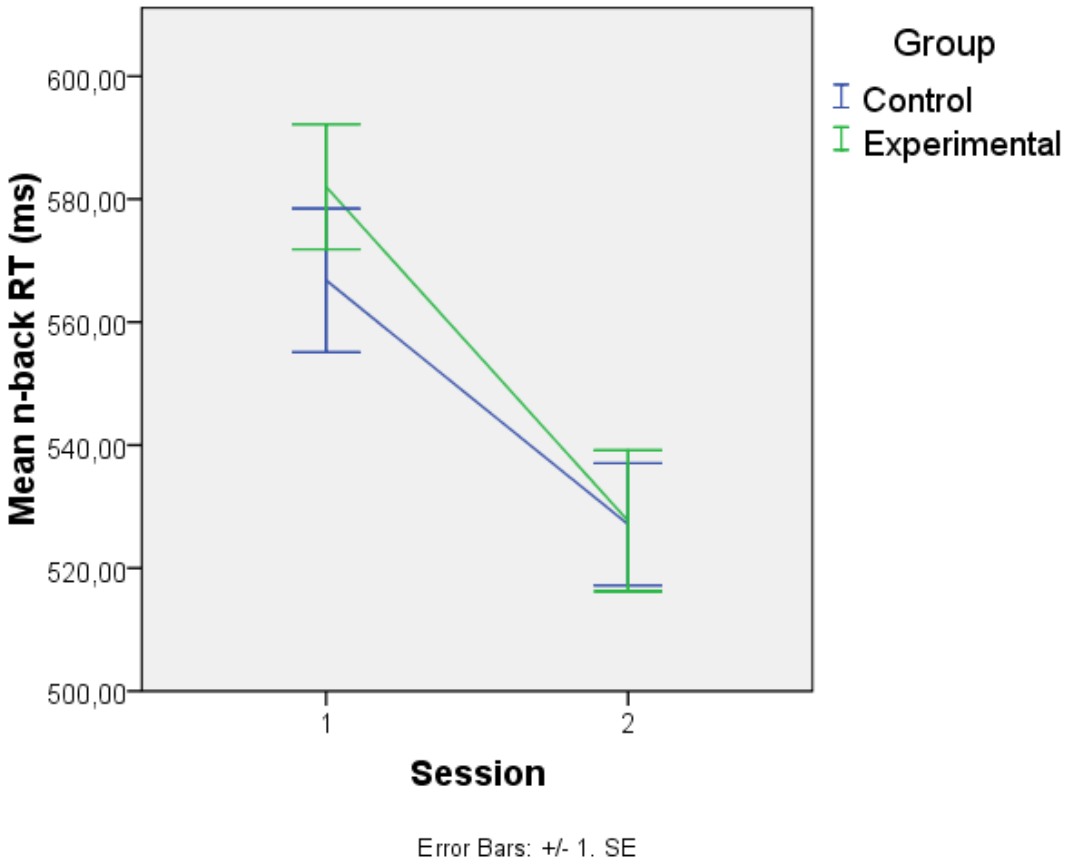


Figure 6. Mean RT across all *n*-back tasks. Error bars indicate standard errors. RT = reaction times. SE = standard error.

3.4.2. Neuronal.

Mixed measures ANOVA of the N2 amplitude revealed a main effect of time, $F(1.87,52.27) = 13.63$, $p < .01$, indicating an overall increase of N2 negativity over the two sessions. Post hoc analyses revealed no significant changes in any group or condition after Bonferroni correction, though effect sizes reached large effects in the 2-back and 3-back task of the control group with $t(15) = 2.26$, $p < .40$, $r = .50$ and $t(15) = 2.24$, $p < .41$, $r = .50$ respectively (Figure 7). Another main effect regarding *n*-back load was found, $p < .02$, $F(1,28) = 6.17$, indicating a higher N2 negativity in *n*-back conditions with higher n-back load.

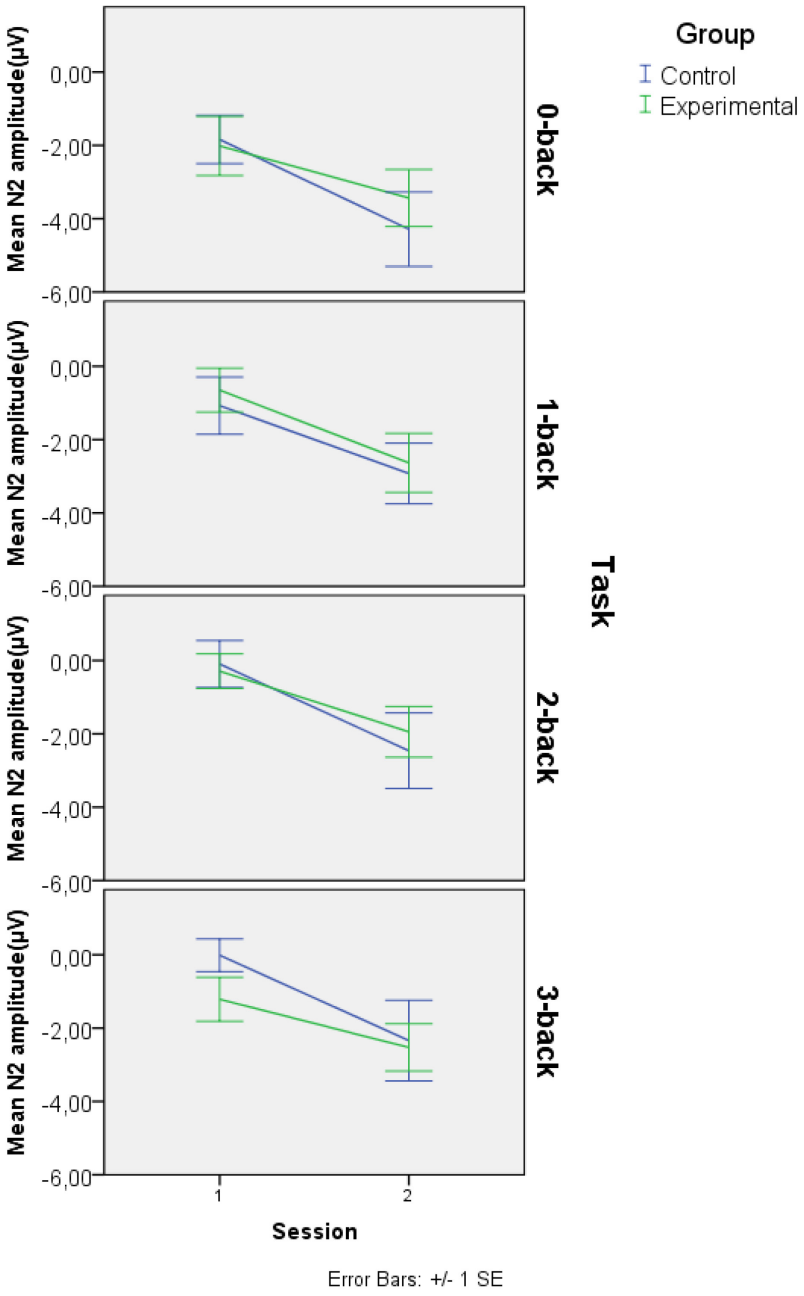


Figure 7. Mean amplitudes in all *n*-back tasks, sessions and groups. Error bars indicate standard errors. SE = standard error.

Analysis of the P3 amplitude revealed a main effect of *n*-back load, $F(2.42,67.75) = 10.64$, $p < .01$. Results indicate that amplitude was lower in the 2-back and 3-back load condition than in the 0- and 1-back condition. Amplitudes in the 3-back task were smaller than amplitudes in the 2-back task. No interaction effects were found.

Analysis of N4 amplitudes revealed a non-significant trend towards a main effect for *n*-back load, $F(3,84) = 2.66$, $p < .06$. No other effects were found.

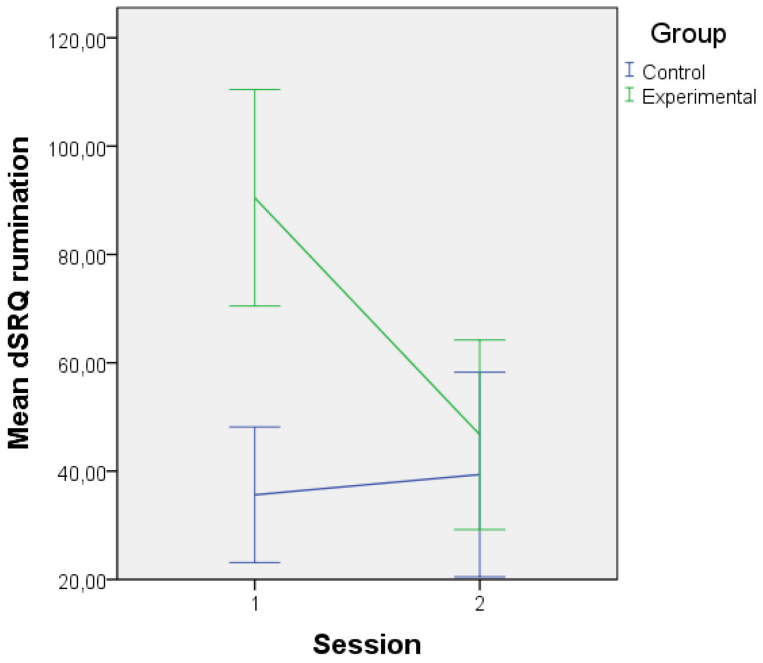
3.4.3. Rumination.

ANOVAs of trait scores in the RSQ-D revealed neither a main effect of time nor any interaction effects. Nevertheless, a significant main effect of group was found, $F(1,28) = 4.99$, $p < .04$, indicating overall higher trait rumination scores in the experimental group. In contrast, analysis of state rumination scores revealed no significant effects.

Regarding dSRQ rumination measures, a significant main effect of time was found, $F(1,28) = 5.28$, $p < .03$, indicating a decrease in dSRQ scores over time, therefore the training reduced the influence of the cMRI on state rumination. Another trend towards a main effect of group was found, $F(1,28) = 3.57$, $p < .07$, pointing at overall higher state rumination changes in the experimental group. Training effects were displayed in a significant interaction of Time × Group, $F(1,28) = 6.71$, $p < .02$. Post hoc *t*-test revealed stronger reductions of SRQ rumination changes by cMRI in the experimental group, compared to the control group (Figure 8), represented by a large effect in the training group, $t(13) = 3.01$, $p = .01$, $r = .65$, compared to no effect in the control group, $t(15) = -.23$, $p = .82$, $r = .06$.

Analysis of changes in the dVAS rumination measure revealed a non-significant trend towards an interaction of Time × Group, $F(1,28) = 3.69$, $p < .07$. Post hoc *t*-tests revealed that this trend was not reproduced by significant changes in any of the two groups separately. Nevertheless, two non-significant opposite effects in both groups, i.e. a small effect of reduction of dVAS rumination in the experimental group, $t(13) = 1.02$, $p < .33$, $r = .27$, and a medium effect of increase of dVAS rumination in the control group, $t(15) = -1.83$, $p < .09$, $r = .43$, were observed (Figure 8).

A



B

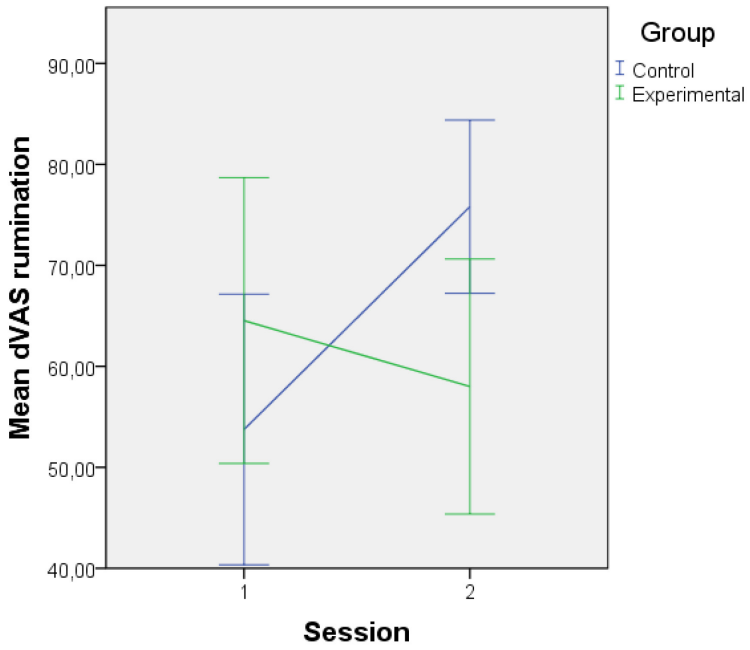


Figure 8. Mean in different groups and sessions of mood induced changes in SRQ rumination subscale (A) and VAS rumination (B). Error bars indicate standard errors. VAS = visual analog scale. dVAS = difference of scores in the visual analog scale post- and pre-cMRI. cMRI = combined mood and rumination induction. SRQ = state rumination questionnaire. dSRQ = difference in SRQ score post- and pre-cMRI.

No significant main effects were found for the dSRQ flexibility measure. Group differences revealed a non-significant trend, $F(1,28) = 3.98$, $p < .06$ towards overall elevated scores in the experimental group. A non-significant main effect of time pointed at a decrease in dSRQ flexibility over time, $F(1,28) = 3.62$, $p < .07$.

3.5. Common Changes of Cognitive Control and Rumination by Training

3.5.1. Correlations of changes in rumination and *n*-back measures.

In the experimental group, changes over sessions in dVAS rumination correlated negatively with changes in accuracy in the 0-back task, $r = -.70$, $p < .03$. This strong effect indicated that increasing performance in the 0-back task was accompanied by decreasing susceptibility to ruminate. A similar nonsignificant trend was found for a strong negative correlation between dSRQ rumination changes over sessions and accuracy in the 0-back task, $r = -.68$, $p < .09$. No other correlation yielded significance after Bonferroni-Holm correction. Nevertheless, notable strong and medium, positive correlations were found between changes in N4 amplitudes over sessions and changes in susceptibility to ruminate (Table 9). The effects indicated that participants who revealed increased N4 amplitudes after training were less prone to induced rumination after training than before.

Table 9
Correlations of N4 Amplitude Changes Over Sessions and Susceptibility to Ruminate Over Sessions

	dSRQ rumination difference	dSQR flexibility difference	dVAS rumination difference
	r	*r*	*r*
N4			
0-back	.44	-.14	.53
1-back	.42	-.03	.51
2-back	.18	.25	.47
3-back	.57	-.10	.47

Note. For all correlations: $n = 30$. *N*-back measures are differences between Session 2 and Session 1. For all rumination measures, *p* values are corrected using Bonferroni-Holm method. SRQ = state rumination questionnaire. dSRQ = difference between post and pre-cMRI SRQ scores. cMRI = combined mood and rumination induction. VAS = visual analog scale. dVAS = difference between post- and pre-cMRI VAS scores. *r* = Pearson's correlation coefficient.

3.5.2. Regression models.

As explicated above, significant effects of training on rumination could be reproduced only in dSRQ rumination values when compared with the control group. Additionally, a trend towards significant training effects was found in mood induced state rumination changes measured by the dVAS rumination scale. Consequently, these two variables were further examined regarding common changes with neuronal and behavioral measures via multiple regression analysis as described above.

3.5.2.1 State questionnaire rumination.

The reference model yielded significance, as presented in Table 10. Group had a negative influence on changes over time in dSRQ rumination, indicating that differences in the experimental group were larger than in the control group, which has already been shown by effects in the mixed design ANOVA, reported above.

Table 10
Multiple Regression Models of Differences in dSRQ Rumination Between Sessions

	Reference model		N2 model		Accuracy model	
	β	*t*	β	*t*	β	*t*
Constant		0.22		-0.36		-0.66
Group[a]	-0.40	-2.59*	-0.30	-1.75^{+}	-0.20	-1.44
0-back[b]			-0.80	-2.19*	-0.60	-3.99**
1-back[b]			1.08	3.08**	0.07	0.43
2-back[b]			-0.30	-0.75	-0.30	-1.90^{+}
3-back[b]			0.00	-0.02	0.21	1.26
R^2	0.19		0.44		0.56	
F	6.72*		3.79*		6.21**	
ΔR			0.25		0.36	
ΔF	0.36**		2.66^{+}		4.91**	

Note. For all models: *n* = 30. Dependent variable is the difference between dSRQ measures between Session 2 and Session 1. *N*-back measures are differences between Session 2 and Session 1. SRQ = state rumination questionnaire. dSRQ = difference between post and pre-cMRI SRQ scores. cMRI = combined mood and rumination induction.
[a]Group is coded 1 for the experimental and 0 for the control group.
[b]Coefficients of changes in *n*-back measures cannot be interpreted, because of high multicollinearity between *n*-back measures.
$^{+}$p < .10. *p < .05. **p < .01.

The explanatory power of the reference model did not accelerate significantly, by adding changes in RT, N4 amplitudes or P3 amplitudes to the predictors.

By adding *n*-back accuracy measures to the independent variables, the quality of the model was enhanced significantly (Table 10). This indicates that changes in accuracy measures over sessions predict changes dSRQ over sessions. The coefficient of factor group was smaller and did not contribute significantly to the model, after controlling for accuracy changes over time.

When the N2 amplitude changes were added to the predictors, the quality of the model was enhanced, yielding a non-significant trend, as presented in Table 10. The coefficient of group was smaller in this model and had no significant contribution to the model, though a trend towards significance was observed.

3.5.2.2 VAS rumination.

The reference model of dVAS rumination changes over sessions revealed a trend towards significance. The factor group contributed to the model with a trend towards significance. Converging with the results from the mixed design ANOVA reported above, a decrease in dVAS rumination measures over sessions was observed for the experimental group.

The explanatory power did not accelerate significantly, when adding session differences in the N2 or N4 amplitude or RT to the model for dVAS rumination change.

By adding accuracy measures to the model, the quality of the model was enhanced significantly (Table 11). The coefficient of group did not reach significance anymore, when controlling for accuracy changes over time.

When adding changes in P3 amplitudes to the predictors, the quality was enhanced significantly (Table 11). In this model the coefficient of factor group contributed significantly to the model.

Table 11
Multiple Regression Models of Changes in dVAS Rumination Between Sessions

	Reference model		P3 model		Accuracy model	
	β	*t*	β	*t*	β	*t*
Constant		1.56		1.84^{+}		0.47
Group[a]	-0.3	-1.92^{+}	-0.4	-2.34*	-0.20	-1.06
0-back[b]			0.65	1.59	-0.50	-3.00*
1-back[b]			0.36	0.57	-0.10	-0.86
2-back[b]			-0.20	-0.42	0.24	1.41
3-back[b]			-0.80	-1.80^{+}	0.04	0.22
R^2	0.12		0.38		0.48	
F	3.69^{+}		2.89*		4.43**	
ΔR			0.26		0.36	
ΔF			2.49^{+}		4.20*	

Note. For all models: *n* = 30. Dependent variable is the difference between dVAS Rumination measures between Session 2 and Session 1. *N*-back measures are differences between Session 2 and Session 1. VAS = visual analogue scale. cMRI = combined mood and rumination induction. dVAS = difference between post and pre-cMRI VAS rumination scores.
[a]Group is coded 1 for the experimental and 0 for the control group.
[b]Coefficients of changes in P3 and accuracy measures cannot be interpreted, because of high multicollinearity between *n*-back measures.
$^{+}$p < .10. *p < .05. **p < .01

4. Discussion

The aim of the current study was to investigate the relationship between neural correlates of inhibition related CC domains and various aspects of rumination, i.e. trait rumination, naturally occurring state of rumination and the susceptibility to ruminate in response to negative mood. For this purpose, 33 participants participated on a prospective controlled single blind study. A manipulation of CC was put into effect by implementation of a CCT in the experimental group while the control group was not trained in CC. Before and after training the participants conducted a letter *n*-back task and completed measures of trait rumination, and state rumination in an experimental setting. State rumination was measured using questionnaires once before and once after a combined induction of rumination and negative mood. Naturally occurring state of rumination in a neutral and unconstrained situation was objectified by the state rumination measures before

the induction, ruminative susceptibility in response to sad mood was measured by changes in state rumination induced by the mood and rumination induction. EEG recordings were conducted during the *n*-back task and inhibition related ERPs were calculated. Correlations between CC and rumination measures were calculated, additionally analysis of causal influences of CC on these rumination domains were examined by analyzing effects of CC changes.

We hypothesized, that behavioral performance and inhibition related ERPs, i.e. N2, P3 and N4 components of a letter *n*-back task, correlate with different rumination measures. We further hypothesized, that CCT would change CC as well as rumination. We finally hypothesized that changes in rumination measures after training would no longer persist, when controlling for changes in CC measures.

A medium positive correlation between accuracy and state of cognitive flexibility indicates that participants who ruminate less during a neutral unconstrained situation have higher accuracy in the *n*-back task. Another medium sized effect indicates that trait ruminators perform the *n*-back task with less accuracy. An effect of medium size indicated additionally that trait ruminators have longer RT in the *n*-back task. Strong effects were observed for a positive relationship between susceptibility to ruminate and both N2 and N4 amplitude sizes. Larger amplitudes thus index a susceptibility to ruminate in response to negative mood. A moderate sized effect between P3 amplitudes and state of rumination indicates that participants, who reported higher degrees of state rumination during the *n*-back task revealed larger P3 amplitudes.

The efficacy of the CCT was validated by stronger reductions in RT in the experimental group in contrast to the control group. A causal relationship between CC and rumination could be shown by moderate effects of the training on ruminative susceptibility. In contrast, for trait rumination measures no effects of the CCT could be observed. Strong sized correlations in the experimental group between 0-back changes and dVAS rumination changes as well as 1-back accuracy changes and dVAS rumination changes over sessions indicated that increasing accuracy was accompanied by decreasing ruminative susceptibility.

Multiple regression analyses of training effects on susceptibility for rumination revealed that training effects were not persistent, when models were adjusted for changes in the N2 amplitude and *n*-back accuracy. This indicates group differences in changes of ruminative susceptibility were no longer present in both groups, when controlling for changes in CC measures, i.e. accuracy and the N2 amplitude. This further implicates that these training related changes were moderated by changes in CC, which validates the effects of the CCT and supports the hypothesis that rumination is causally influenced by CC.

In contrast, a model adjusted for changes in P3 amplitudes showed significant training effects. This indicates that P3 amplitudes represent a cognitive correlate of ruminative susceptibility, which was not altered by implementation of CCT.

4.1. Behavioral

According to our hypotheses, we found results indicating that RTs of the 0-back task correlate with trait rumination measures. A moderate positive relationship between situational flexibility and accuracy in the 3-back task indirectly points at a similar trend for state rumination, because state rumination is negatively associated with situational flexibility. However, no relationship between behavioral performance in the 1-back task and susceptibility to ruminate in reaction to negative mood could be observed.

Present findings in behavioral measures of the *n*-back task support Whitmer and Gotlib's (2013) suggestion, that deficits in inhibition of previous relevant information, as well as in switching and updating can be found in ruminators without history of MDD. Switching and updating both are components of the 1-, 2- and 3-back task (Chen et al., 2008; Watter et al., 2001). Conclusively the correlation of accuracy in the 1-back task and changes in cognitive flexibility are in line with the assumption that trait ruminators reveal poor performance in tasks involving inhibition and updating of WM content. The 0-back task does only involve matching of the current stimulus with a target letter, thus demands little WM load (Chen et al., 2008). The correlation of 0-back RTs and RSQ-D thus promotes the suggestion that trait rumination decreases performance of WM tasks with relatively low cognitive load.

Performance of the *n*-back task increases activity in multiple brain regions which in contrast have been found to show less activation in rumination. Involvement of the ACC during the *n*-back task has been shown in studies using different neuropsychological measures. Gevins, Smith, McEvoy and Yu (1997) compared frequency bands during performance of an *n*-back task with different difficulties. They observed increases in frontal theta power with augmenting *n*-back load and during practice. Source localization of frontal theta displayed a dipole in the ACC. Performance loss by increased difficulty in the *n*-back task has been associated with a right lateralized frontoparietal network (FPN), including the ACC (Saliasi et al., 2014). The dlPFC has been found to show greater activity with augmenting *n*-back load (Kearney-Ramos et al., 2014; Owen et al., 2005). Another important region which is disabled in depressive rumination is the ventrolateral prefrontal cortex (vlPFC; Disner, Beevers, Haigh, & Beck, 2011), which shows increased activity with augmenting *n*-back load (Kearney-Ramos et al., 2014; Owen et al., 2005; Saliasi et al., 2014). Activity in the vlPFC is generally linked to conscious intention about retaining information in WM and related attentional control processes (Dove, Rowe, Brett, & Owen, 2001). On the other hand, DMN activity, which is related to rumination, shows the opposite pattern: The PCC, which is part of the DMN, is higher activated in tasks with low *n*-back load in contrast to high *n*-back load. A similar pattern also appeared for prefrontal midline structures, which are associated with self-referential processing (Kearney-Ramos et al., 2014). The behavioral correlations of trait rumination with 0-back task performance in the current study might therefore be due to hyperactivity in task negative and hypoactivity in task positive DMN structures (Marchetti, Koster, Sonuga-Barke, & de Raedt, 2012).

As previously discussed, effects of the CCT on *n*-back performance could be shown. These effects are represented by reduced RT in the experimental group, when compared to the control group. Contrary to our hypothesis, accuracy scores of the 3-back task increased over the two experimental sessions in the control group compared to the experimental group. Therefore training effects were present in RT measures, while accuracy scores did not show consistent effects across conditions. Even though no effects of CCT on trait rumination could be

found, CCT reduced the susceptibility to ruminate in reaction to negative mood in an experimental setting. The CCT implemented in the present study was adopted from Siegle et al. (2007), who also compared a training group to a control group. In Siegle et al.'s study, each group additionally received psychotherapy and the training effects on CC were assessed by physiological and behavioral measures. Additionally, a small subset (N = 6) of the training group was assessed regarding training effects on involvement of dlPFC in a digit sorting task using fMRI. Results indicated increased dlPFC activity during difficult tasks and decreased activity during easier tasks after training. Most important, depressive symptoms as well as rumination trait measures were reduced by the intervention, when compared to the control group. A recent study by Brunoni et al. (2014), implemented the same training in combination with transcranial direct-current stimulation (tDCS) over the left dlPFC to a clinical depressed sample. Improvement of CC was associated with reduction of depressive symptoms. A consecutive analysis of trait rumination measures in the same study population (Vanderhasselt et al., 2015) revealed a reduction of brooding from pre- to posttraining sessions as well as positive correlations between PASAT improvement and brooding reduction. Therefore the results in the present study are in line with previous findings on effects of a CCT. They furthermore legitimize the suggestion that dlPFC functioning was enhanced by the training in the current experimental group.

Although no relevant correlations of accuracy with measures of susceptibility to rumination were found, multiple regression analysis indicates, that training effects on ruminative susceptibility no longer persist when controlling for changes in accuracy measures in the *n*-back task. This indicates that training effects, which target ruminative susceptibility are moderated by changes in *n*-back accuracy. Though training effects for RT were larger and existed in every *n*-back subtask, RTs did not show a relation to training effects on rumination.

Moreover, our findings of decreases of susceptibility in rumination indicate that this rumination measure is sensitive to CC changes. Correspondingly, changes in state rumination post- vs. pre-cMRI might be a more sensitive measure for ruminative tendencies in healthy subjects than the RSQ-D. The RSQ-D includes items that involve complex and active behaviors associated with trait rumination, e.g.

isolation from social activities or listening to mood-congruent music (Kühner et al., 2007). Furthermore it depends on self-judgements about personal habitual thinking tendencies. In contrast, the experimental measure of susceptibility to rumination specifically objectifies current changes in state rumination in reaction to negative mood. This rumination measure may be more sensitive to CC changes and therefore reveal correlations with CC measures in healthy samples. Ruminative susceptibility could function as a precondition for the development of a habitual ruminative thinking style. On the other hand, it could possibly be the factor of trait rumination, which has been altered by CCT training in the other studies described above (Brunoni et al., 2014; Siegle et al., 2007; Vanderhasselt et al., 2015).

4.2. N2 and N4 Amplitudes

In the present study, large correlations of the N2 amplitudes with susceptibility to ruminate were found. Negativity of the N2 amplitudes in the 2-back task was larger when subject reported either increases in state rumination or decreases in situational cognitive flexibility after mood and rumination induction. Correlations between the N4 amplitude and ruminative susceptibility revealed a similar pattern. Strong correlations between amplitude size and loss in cognitive flexibility after cMRI could be observed in three subtasks of the *n*-back task. Additionally, nonsignificant trends towards medium sized correlations between increases in state rumination after cMRI and N4 amplitude size were found in the same subtasks of the *n*-back task. Trait rumination or naturally occurring state of rumination did not correlate with N2 or N4 amplitudes.

N2 and N4 amplitudes have previously been linked to similar cognitive functions, such as conflict monitoring (Larson et al., 2014) and behavioral inhibition (Pires et al., 2014), therefore similar findings for both components are not surprising. The relation of rumination to N2 and N4 amplitudes has so far been heterogeneous. In a study by Krompinger and Simons (2011) conflict related increases of N450 amplitudes of a Stroop Task indicated higher scores of trait rumination in a student sample with enhanced grades of depressive symptoms. In contrast, Alderman et al. (2014) found decreased N200 amplitudes in a flanker task performed by subjects with diagnoses of MDD. While Krompinger and Simons interpreted their

findings as increased allocation of neuronal resources to perform the task sufficiently, Alderman et al. concluded decreased allocation of resources and diminished CC in ruminators, though in their study no relationship to behavioral measures was found.

A common approach to interpret increased neuronal activity as a compensatory phenomenon argues as follows: In the group with increased activity, additional resource allocation is needed to achieve the same task performance (Krompinger & Simons, 2011; Saliasi et al., 2014; G. Wagner et al., 2006). Therefore never depressed trait-ruminators would need more resources than low trait-ruminators to overcome CC deficits for equivalent behavioral performance. This explanation does not yield for the present study, since decreased RTs were linked to trait rumination. A similar trend was also found for accuracy, indicating that task performance differs between high and low ruminators.

An alternative explanation for enlarged amplitudes during the *n*-back task could be a negative attentional bias. Depression, but also rumination is linked to a negative attentional bias. A negative attentional bias causes preferred perception of negative information (Gotlib & Joormann, 2010) and interpretation of neutral information in a specific negative, rumination related manner (Mor, Hertel, Ngo, Shachar, & Redak, 2014). In healthy and depressed individuals N4 amplitudes are increased during the perception of negative vs. neutral or positive information, and this effect is stronger in MDD (Dai & Feng, 2011). If this effect is as well present for rumination in healthy samples, the increased N4 amplitudes could be due to negative attentional bias. This interpretation still remains speculative, as no attentional bias was measured in the present experiment.

4.2.1. The conflict monitoring hypothesis and rumination

In other studies investigating N200 (Alderman et al., 2014) or N450 (Krompinger & Simons, 2011) amplitudes in rumination, EEG recordings were conducted during the performance of Flanker or Stroop Tasks. Stroop and Flanker Tasks both demand inhibition of conflicting and irrelevant information. Especially this abilities have not been found to be decreased, but even increased in healthy ruminators (Whitmer & Gotlib, 2013). Alderman et al. (2014) as well as Krompinger and

Simons (2011) did not depict correlations between rumination and ERPs in healthy controls with low depressive symptomatology.

Some researchers doubt the direct association of N2 amplitude with inhibition and instead assign the frontal N2 exclusively to monitoring functions. According to this assumption, N2 amplitudes index the need for inhibition (Pires et al., 2014), but do not represent inhibitive activity. Although conflict monitoring is not unitarily necessary for performance of the *n*-back task, it is part of it. Successful task performance mainly involves switching and updating of WM (Chen et al., 2008; Harvey et al., 2004; Watter et al., 2001) and is measured in RT and accuracy scores. Even if conflict monitoring is not a characteristic element of the *n*-back task, it is needed for successful task performance. Each *n*-back subtask involves the decision between two competing response representations, which is quite similar to demands of a go/nogo task, with the difference that the nogo condition is replaced by a second go-condition. As the N2 amplitudes of go and nogo trials represent conflict monitoring processes (Nieuwenhuis et al., 2003), they might as well represent the same mechanisms in the *n*-back task. Conclusively N2 and N4 amplitudes could index the same functions Go/Nogo N2, Flanker N2 and Stroop N450. Conflict monitoring demands might also be increased by augmenting task demands, as more information has to be processed for adequate conflict resolution.

The current sample and Krompinger and Simons's (2011) sample had in common, that subjects with history of MDD were excluded and hence samples differed only in depressive symptoms. Krompinger and Simons suggested that their sample of high functioning depressed participants belonged to a population with hypersensitivity to conflict and that trait rumination involves the expectation of such conflict, leading to inefficient ACC engagement. Under the assumption that N2 and N4 amplitudes of the *n*-back task represent conflict monitoring, results of the current study may correspond to Krompinger and Simons's findings. Increased amplitudes in this context indicate enhanced resource allocation for conflict monitoring and findings for increased susceptibility to ruminate might therefore be due to increased sensibility to conflict. Increased conflict signaling in healthy individuals might lead to increased perception of conflicts in ambiguous or neutral

situations and therefore promote ruminative states, which could be a reason for trait-ruminators to dwell deeper and prolonged about personal conflicts (Krompinger & Simons, 2011).

Hypersensitivity to conflict constitutes an alternative explanation for better behavioral performance of trait ruminators in the Stroop and Flanker task in healthy samples (Whitmer & Gotlib, 2013). If inhibitory mechanisms are not impaired, increased conflict signaling should lead to more effective inhibition of distracting information.

The quantification of susceptibility to ruminate by the induction of state of rumination could be a more sensitive measure for ruminative tendencies in heathy samples than the RSQ. This assumption might explain, why the current study reveals correlations of ERPs with susceptibility to ruminate, in contrast other studies, which failed to link RSQ to ERPs in healthy samples (Alderman et al., 2014; Krompinger & Simons, 2011).

A general impairment of executive functions in MDD is widely observed (Snyder, 2013) and divergent findings of ERPs in rumination could therefore be due to different sample characteristics. Several findings and their interpretation support this suggestion: Alderman et al.'s (2014) findings indicate that MDD patients show smaller N2 and N450 amplitudes than healthy subjects. N2 and N450 amplitudes are less increased by conflict in subjects with history of MDD than HC (Holmes & Pizzagalli, 2008). This effect is enhanced, the more depressive episodes in the patients history of MDD occurred (Vanderhasselt & De Raedt, 2009). A diverging relationship between CC and rumination has also been found in a developmental study with a large sample of healthy (N = 486) adolescents (age 12-13). The findings indicate an alternating direction of relationship between sustained attention and rumination depending on depressive symptoms (C. A. Wagner, Alloy, & Abramson, 2014). While low depressive symptoms indicated a positive correlation, high depressive symptoms indicated a negative relationship between sustained attention and trait rumination. These findings support the assumption that neuronal resource allocation is impaired in MDD, which leads to higher trait rumination. The disengagement hypothesis by Koster et al. (2011), which

associates decreased conflict signaling to increased rumination, might therefore yield only for individuals with manifest depression.

Dipoles of the N2 and N450 have previously been found primarily in the ACC (Bekker et al., 2005; Nieuwenhuis et al., 2003; van Veen & Carter, 2002; West et al., 2004). According to the conflict monitoring theory (Botvinick, Braver, Barch, Carter, & Cohen, 2001), this cerebral structure signals to CC related structures. Particularly the vlPFC and dlPFC reduce conflict activation by biasing posterior regions and implementing attentional focus (Larson et al., 2014). With augmenting number of performed trials, conflict signals should therefore be reduced. Consequently current findings of increased conflict signals in rumination could be caused by reduced conflict adaptation due to insufficient dlPFC function in subjects with a high susceptibility to ruminate. This hypothesis corresponds in turn to the neuropsychological model of rumination by Disner et al. (2011), who link rumination to decreased dlPFC functioning.

No significant training effects could be observed for N2 or N4 amplitudes. Nevertheless, the N2 amplitudes generally increased after training, but less in the experimental group than in the control group.

According to the conflict monitoring theory discussed above, dlPFC training could lead to more efficient conflict reduction, represented by lower N2 amplitudes over training sessions (Clayson & Larson, 2011; Larson et al., 2014). Millner, Jaroszewski, Chamarthi and Pizzagalli (2012), measured neuronal changes after a CCT and found a decrease in the N2 amplitude in ERPs of a flanker task after training. Since their study did not involve a control group, training effects could not exclusively be attributed to the training.

The changes of the N2 amplitudes in the control, but also in the experimental group of the present study could also be due to practice related effects, as the N2 amplitude increases with practice (Schapkin, Falkenstein, Marks, & Griefahn, 2007). If CCT generally decreases N2 by increasing dlPFC functioning, these two competing modulations of N2, i.e. training and practice, may explain the unexpected findings in the current sample as follows: Practice effects of the *n*-back task led to larger N2 amplitudes in both groups equally, but nonsignificantly

stronger in the control group as the effect is slightly counteracted by the influence of the training in the experimental group.

4.3. P3 Amplitudes

In contrast to our findings on N2 and N4, analysis of the P3 amplitudes showed no relationship to susceptibility to ruminate or trait rumination. This result converges with findings in other studies, which were also unable to find correlations between trait rumination and P3 amplitudes. A nonsignificant trend towards a positive correlation of medium strength between state rumination and P3 amplitude in the 3-back task might indicate that higher degrees of naturally occurring ruminative state occur with larger P3 amplitudes.

The P3 in the current study corresponds to the parietal P3b component of the P300 complex, as described by Polich (2007). The P3b represents inhibition of ongoing activity in parietal and temporal structures in favor of integration of task related processes. As state rumination represents an ongoing, task-unrelated cognitive process, findings of larger P3 amplitudes could indicate successful inhibition of rumination related processes. Larger P3 amplitudes should therefore be correlated with successful WM performance in the *n*-back task too, as described by Saliasi, Geerligs, Lorist and Maurits (2014). This assumption supports the resource depletion account for rumination, since resources, which are demanded by state rumination, are needed for successful task performance. The role of the resource depletion account in the relationship of CC deficits in state rumination has previously been emphasized by Whitmer and Gotlib (2013). The negative influence of state rumination on CC has mainly been observed in studies including CC measures after a rumination induction. In contrast, in the current study CC was measured in an emotionally neutral setting, such that *n*-back performance might be unaffected or only slightly effected by state rumination. Of note, the current sample included only nondepressed subjects, who may additionally be able to successfully inhibit ongoing activity in favor of task performance. Subjects, who ruminated more in a neutral situation, indicated by the state rumination measure, are probably also prone to ruminate during the *n*-back task, but they might at the same time be able to interrupt ruminative tendencies and switch their attention back to the task when a stimulus appears. The positive

correlation between P3 and state rumination might therefore indicate intact inhibition of ruminative state during task performance. In this scenario the P3 does not indicate a general ability to inhibit memory processes, which is generally not impaired in healthy subjects, but an actual inhibition of ruminative processes, which is stronger present in individuals with high state rumination scores. This assumption may additionally be supported by findings of neuronal correlates of the *n*-back task: A recent study by Luu et al. (2014) investigated neural correlates of the *n*-back task via source localization. A component was found in the P3 time window between 280 ms and 430 ms after stimulus onset. Dipoles of the component were found in several DMN structures: the middle superior temporal gyri, inferior parietal lobe, temporal poles, mPFC, bilateral inferior temporal lobe and precuneus. As activation of DMN structures has been linked to state rumination, inhibition of these structures and reallocation of attentional resources for task related demands might be represented by enhanced P3 amplitudes. In contrast, decreased ability to reallocate neuronal resources for task performance should lead to decreased amplitudes and increased susceptibility to rumination or trait rumination. In the present study, no such correlation yielded significance, but overlapping negative signs of correlations between P3 and measures for susceptibility to ruminate are in line with this assumption (Appendix).

The current finding might also offer novel explanations for general P3 characteristics. Increased P3 amplitudes have constantly been observed in infrequent trails, mainly measured in go/nogo tasks (Polich, 2007). If frequent tasks become more automatized during task performance, less CC and therefore less neuronal resources may be needed for task execution. This could lead to increased task-unrelated processes during task performance. Infrequent trails, which are less automatized, might cue attention reallocation for task performance and lead to inhibition of this task unrelated activity, thereby leading to enhanced P3 amplitudes.

When the observed marginal training effects on susceptibility to rumination were controlled for changes in P3 amplitudes, these training effects persisted and had significant contributions to the model. This indicates that P3 amplitudes index a factor of ruminative susceptibility, which influences susceptibility to ruminate, but is

not effected by CCT. This finding is in line with the nonexistence of training effects on P3 amplitudes. On the other hand, this result supports the association of P3 amplitudes with state rumination during task performance as follows: If P3 amplitudes represent a state characteristic of CC, which is linked to a state of rumination, controlling for P3 changes might increase sensitivity for longterm effects of CCT on trait like patterns of rumination, represented by susceptibility to ruminate.

Increased P3 amplitudes during naturally occurring state of rumination could alternatively be observed due to increased self-referential processing. State rumination is a type of self-focused attention and P3 amplitudes have been found to be increased in reaction to self-relevant information (Knyazev, 2013). If self-focused attention or state rumination leads to self-referential processing of external stimuli, *n*-back letters might be perceived as self-relevant during the task. The neutral letter stimuli have no self-referential character per se, but self-reference could emerge during the performance situation. Self-referential processing bias has previously also been linked to MDD and trait rumination (Beckwé, Deroost, Koster, de Lissnyder, & de Raedt, 2014; Gotlib & Joormann, 2010). Another cognitive bias linked to rumination and depression is a negative interpretation bias (Mor et al., 2014). In a study by Schick, Wessa, Vollmayr, Kuehner and Kanske (2013) a late positive potential (LPP) was defined in the time window from 300 to 500 ms, which corresponds to the time window of P300. In this study a negative cognitive bias interpreting ambiguous stimuli correlated with reflective pondering, an adaptive form of rumination. Responses indicating negative interpretation of ambiguous stimuli yielded larger LPP amplitudes. Conclusively a negative interpretation bias in high ruminating individuals might lead to enlarged P3 amplitudes. These hypotheses still remain speculative, as no measures of cognitive bias were included in the current study.

4.4. Limitations

Some methodological limitations of the current study should be taken into account when drawing conclusions from the results reported.

The *n*-back task performed in this study consisted of four subtasks, varying in WM load. ERPs have been correlated for each subtask separately. Though task demands differed between subtasks, high inter task correlations pointed at similar processes represented by ERPs (Appendix). Therefore results were interpreted cumulatively as CC measures. To account for accumulation of Type I error, Bonferroni-Holm correction was conducted, leading to increased Type II error. Therefore results of the current study might be reported conservatively. The use of a wide range of *n*-back load might be advised only in less complex within-subject designs.

N2 and P3 reliability has been reported with Cronbach's $\alpha > .54$ (Kinoshita, Inoue, Maeda, Nakamura, & Morita, 1996) for both components. In this study, correlations between sessions did not exceed $r = .38$ (Appendix) and measures were sometimes negatively correlated. This fact questions trait characteristics of these measures and could also explain the nonexistence of correlations with trait rumination measures and missing significance in training effects. It might generally question the comparison between trait measures and ERPs, as internal consistency might not sufficiently be given.

Trait measures of rumination were completed at home, in order to control for state rumination influences of the experimental situation. This fact might increase the probability for nonfindings regarding correlations with trait rumination in healthy samples, but on the other hand increase the ecological validity of data. Studies measuring trait rumination and CC in the same experimental setting might possibly be affected by state rumination as a confounding variable, which affects CC and answer behavior in the trait rumination questionnaire, thus increase the correlation between CC and trait rumination. In this regard, experimental measures of ruminative susceptibility might offer a more valid approach.

In addition, also the training sessions were completed at home. Although the progression of performance was observed by the researcher, no standardization of the setting during a training session was given. This could decrease the training effects, and increase vulnerability for confounding variables.

4.5. Conclusion and Future Directions

Trait rumination in healthy individuals is linked to decreased measures of cognitive control in the *n*-back task. Increases of CC lead to decreases of ruminative susceptibility in healthy samples. Ruminative susceptibility is linked to increased ERP signals which are known to index conflict monitoring. Hypersensitivity to conflict might be a factor contributing to rumination in healthy samples exclusively, while conflict signaling might be reduced in depressive samples (Alderman et al., 2014; Koster et al., 2011). Hypersensitivity to conflict might contribute to increased Stroop and Flanker Task performance observed in high ruminators within nondepressed samples (Whitmer & Gotlib, 2013). A marginal effect of increased P3 amplitudes during naturally occurring state of rumination encourages further investigations on the influence of different states of rumination on P3 amplitudes.

5. References

Abate, K. H. (2013). Gender disparity in prevalence of depression among patient population: A systematic review. *Ethiopian Journal of Health Sciences*, *23*(3), 283–288.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, *30*(2), 217–237.

Alderman, B. L., Olson, R. L., Selby, E. A., Bates, M. E., Buckman, J. F., & Shors, T. J. (2014). Rumination in major depressive disorder is associated with impairments in ERP correlates of inhibitory cognitive control. *Psychophysiology*, *51*, S77–S77.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Andersen, S. B., Moore, R. A., Venables, L., & Corr, P. J. (2009). Electrophysiological correlates of anxious rumination. *International Journal of Psychophysiology*, *71*(2), 156–169.

Beck, A. T. (1967). *Depression: Clinical, experimental and theoretical aspects*. New York: Harper & Row.

Beck, A. T., Steer, R. A., & Brown, G. A. (2006). *Beck-Depressions-Inventar* (2nd edition). Frankfurt/Main: Harcourt.

Beckwé, M., Deroost, N., Koster, E. H. W., de Lissnyder, E., & de Raedt, R. (2014). Worrying and rumination are both associated with reduced cognitive control. *Psychological Research*, *78*(5), 651–660.

Bekker, E. M., Kenemans, J. L., & Verbaten, M. N. (2005). Source analysis of the N2 in a cued Go/NoGo task. *Cognitive Brain Research*, *22*(2), 221–231.

Bell, A., & Sejnowski, T. (1995). An information maximization approach to blind separation and blind deconvolution. *Neural Computation*, *7*(6), 1129–1159.

Berman, M. G., Misic, B., Buschkuehl, M., Kross, E., Deldin, P. J., Peltier, S., … Jonides, J. (2014). Does resting-state connectivity reflect depressive rumination? A tale of two analyses. *NeuroImage*, *103*, 267–279.

Berman, M. G., Peltier, S., Nee, D. E., Kross, E., Deldin, P. J., & Jonides, J. (2011). Depression, rumination and the default network. *Social Cognitive and Affective Neuroscience*, *6*(5), 548–555.

Black, S. W., & Pössel, P. (2014). Integrating Beck's cognitive model and the response style theory in an adolescent sample. *Journal of Youth and Adolescence*, *44*(1), 195–210.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*(3), 624–652.

Braboszcz, C., Cahn, B. R., Balakrishnan, B., Maturi, R. K., Grandchamp, R., & Delorme, A. (2013). Plasticity of visual attention in Isha yoga meditation practitioners before and after a 3-month retreat. *Consciousness Research*, *4*, 1–9.

Braver, T. S., Cohen, J. D., Nystrom, L. E., Jonides, J., Smith, E. E., & Noll, D. C. (1997). A parametric study of prefrontal cortex involvement in human working memory. *NeuroImage*, *5*(1), 49–62.

Brunoni, A. R., Boggio, P. S., De Raedt, R., Benseñor, I. M., Lotufo, P. A., Namur, V., … Vanderhasselt, M.-A. (2014). Cognitive control therapy and transcranial direct current stimulation for depression: A randomized, double-blinded, controlled trial. *Journal of Affective Disorders*, *162*, 43–49.

Chen, Y.-N., Mitra, S., & Schlaghecken, F. (2008). Sub-processes of working memory in the N-back task: An investigation using ERPs. *Clinical Neurophysiology*, *119*(7), 1546–1559.

Clayson, P. E., & Larson, M. J. (2011). Conflict adaptation and sequential trial effects: Support for the conflict monitoring theory. *Neuropsychologia*, *49*(7), 1953–1961.

Cohen, N., Daches, S., Mor, N., & Henik, A. (2014). Inhibition of negative content—a shared process in rumination and reappraisal. *Emotion Science*, *5*, 1–4.

Connolly, S. L., Wagner, C. A., Shapero, B. G., Pendergast, L. L., Abramson, L. Y., & Alloy, L. B. (2014). Rumination prospectively predicts executive

functioning impairments in adolescents. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*(1), 46–56.

Cooney, R. E., Joormann, J., Eugène, F., Dennis, E. L., & Gotlib, I. H. (2010). Neural correlates of rumination in depression. *Cognitive, Affective, & Behavioral Neuroscience*, *10*(4), 470–478.

Copeland, J. R. M., Beekman, A. T. F., Dewey, M. E., Hooijer, C., Jordan, A., Lawlor, B. A., … Wilson, K. C. M. (1999). Depression in Europe - Geographical distribution among older people. *British Journal of Psychiatry*, *174*, 312–321.

Dai, Q., & Feng, Z. (2011). Deficient interference inhibition for negative stimuli in depression: An event-related potential study. *Clinical Neurophysiology*, *122*(1), 52–61.

De Jong-Meyer, R., Parthe, T., & Projektgruppe. (2009). Einfluss von Achtsamkeitsübung und Dezentrierung auf Rumination und Spezifität autobiographischer Erinnerungen. *Zeitschrift Für Klinische Psychologie Und Psychotherapie*, *38*(4), 240–249.

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*(1), 9–21.

Disner, S. G., Beevers, C. G., Haigh, E. A. P., & Beck, A. T. (2011). Neural mechanisms of the cognitive model of depression. *Nature Reviews Neuroscience*, *12*(8), 467–477.

Dove, A., Rowe, J. B., Brett, M., & Owen, A. M. (2001). Neural correlates of passive and active encoding and retrieval: A 3T fMRI study. *Neuroimage*, *13*(6), S660–S660.

Egeland, J., Rund, B. R., Sundet, K., Landrø, N. I., Asbjørnsen, A., Lund, A., … Hugdahl, K. (2003). Attention profile in schizophrenia compared with depression: Differential effects of processing speed, selective attention and vigilance. *Acta Psychiatrica Scandinavica*, *108*(4), 276–284.

Enge, S., Fleischhauer, M., Lesch, K.-P., Reif, A., & Strobel, A. (2011). Serotonergic modulation in executive functioning: Linking genetic variations to working memory performance. *Neuropsychologia*, *49*(13), 3776–3785.

Eugène, F., Joormann, J., Cooney, R. E., Atlas, L. Y., & Gotlib, I. H. (2010). Neural correlates of inhibitory deficits in depression. *Psychiatry Research: Neuroimaging*, *181*(1), 30–35.

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychologica*, *101*(2–3), 267–291.

Freton, M., Lemogne, C., Delaveau, P., Guionnet, S., Wright, E., Wiernik, E., … Fossati, P. (2014). The dark side of self-focus: brain activity during self-focus in low and high brooders. *Social Cognitive and Affective Neuroscience*, *9*(11), 1808–1813.

Gevins, A., Smith, M. E., McEvoy, L., & Yu, D. (1997). High-resolution EEG mapping of cortical activation related to working memory: Effects of task difficulty, type of processing, and practice. *Cerebral Cortex*, *7*(4), 374–385.

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology*, *6*, 285–312.

Gronwall, D. M. (1977). Paced auditory serial-addition task: A measure of recovery from concussion, *44*(2), 367–373.

Gross, J. J. (2015). Emotion regulation: Current status and future prospects. *Psychological Inquiry*, *26*(1), 1–26.

Harvey, P. O., Le Bastard, G., Pochon, J. B., Levy, R., Allilaire, J. F., Dubois, B., & Fossati, P. (2004). Executive functions and updating of the contents of working memory in unipolar depression. *Journal of Psychiatric Research*, *38*(6), 567–576.

Hertel, P. T. (1998). Relation between rumination and impaired memory in dysphoric moods. *Journal of Abnormal Psychology*, *107*(1), 166–172.

Holmes, A. J., & Pizzagalli, D. A. (2008). Response conflict and frontocingulate dysfunction in unmedicated participants with major depression. *Neuropsychologia*, *46*(12), 2904–2913.

Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics*, *6*(2), 65–70.

Jiajin, Y., Hong, L., Antao, C., & Yuejia, L. (2007). Neural correlates underlying humans' differential sensitivity to emotionally negative stimuli of varying valences: An ERP study. *Progress in Natural Science*, *17*, 115–121.

Joormann, J. (2010). Cognitive inhibition and emotion regulation in depression. *Current Directions in Psychological Science*, *19*(3), 161–166.

Joormann, J., & Gotlib, I. H. (2008). Updating the contents of working memory in depression: Interference from irrelevant negative material. *Journal of Abnormal Psychology*, *117*(1), 182–192.

Joormann, J., Yoon, K. L., & Zetsche, U. (2007). Cognitive inhibition in depression. *Applied & Preventive Psychology*, *12*(3), 128–139.

Kearney-Ramos, T. E., Fausett, J. S., Gess, J. L., Reno, A., Peraza, J., Kilts, C. D., & James, G. A. (2014). Merging clinical neuropsychology and functional

neuroimaging to evaluate the construct validity and neural network engagement of the n-back task. *Journal of the International Neuropsychological Society*, *20*(07), 736–750.

Keeser, D., Padberg, F., Reisinger, E., Pogarell, O., Kirsch, V., Palm, U., … Mulert, C. (2011). Prefrontal direct current stimulation modulates resting EEG and event-related potentials in healthy subjects: A standardized low resolution tomography (sLORETA) study. *NeuroImage*, *55*(2), 644–657.

Kinoshita, S., Inoue, M., Maeda, H., Nakamura, J., & Morita, K. (1996). Long-term patterns of change in ERPs across repeated measurements. *Physiology & Behavior*, *60*(4), 1087–1092.

Knyazev, G. G. (2013). EEG correlates of self-referential processing. *Frontiers in Human Neuroscience*, *7*, 1–14.

Kohn, N., Falkenberg, I., Kellermann, T., Eickhoff, S. B., Gur, R. C., & Habel, U. (2014). Neural correlates of effective and ineffective mood induction. *Social Cognitive and Affective Neuroscience*, *9*(6), 864–872.

Koster, E. H. W., De Lissnyder, E., Derakshan, N., & De Raedt, R. (2011). Understanding depressive rumination from a cognitive science perspective: The impaired disengagement hypothesis. *Clinical Psychology Review*, *31*(1), 138–145.

Krohne, H. W., Egloff, B., Kohlmann, C.-W., & Tausch, A. (1996). Untersuchungen mit einer deutschen Version der “Positive and Negative Affect Schedule” (PANAS). *Diagnostica*, *42*(2), 139–156.

Krompinger, J. W., & Simons, R. F. (2011). Cognitive inefficiency in depressive undergraduates: Stroop processing and ERPs. *Biological Psychology*, *86*(3), 239–246.

Kuehner, C., & Weber, I. (1999). Responses to depression in unipolar depressed patients: An investigation of Nolen-Hoeksema's response styles theory. *Psychological Medicine*, *29*(06), 1323–1333.

Kühner, C., Huffziger, S., & Nolen-Hoeksema, S. (2007). *RSQ-D Response Styles Questionnaire - deutsche Version□; Manual*. Göttingen: Hogrefe.

Kühn, S., Schmiedek, F., Brose, A., Schott, B. H., Lindenberger, U., & Lövden, M. (2013). The neural representation of intrusive thoughts. *Social Cognitive and Affective Neuroscience*, *8*(6), 688–693.

Kühn, S., Vanderhasselt, M.-A., De Raedt, R., & Gallinat, J. (2012). Why ruminators won't stop: The structural and resting state correlates of rumination and its relation to depression. *Journal of Affective Disorders*, *141*(2–3), 352–360.

Kühn, S., Vanderhasselt, M.-A., De Raedt, R., & Gallinat, J. (2014). The neural basis of unwanted thoughts during resting state. *Social Cognitive and Affective Neuroscience*, *9*(9), 1320–1324.

Kutas, M., & Federmeier, K. D. (2011). Thirty years and counting: Finding meaning in the N400 component of the event-related brain potential (ERP). *Annual Review of Psychology*, *62*(1), 621–647.

Lamm, C., Walker, O. L., Degnan, K. A., Henderson, H. A., Pine, D. S., McDermott, J. M., & Fox, N. A. (2014). Cognitive control moderates early childhood temperament in predicting social behavior in 7-year-old children: An ERP study. *Developmental Science*, *17*(5), 667–681.

Larson, M. J., Clayson, P. E., & Clawson, A. (2014). Making sense of all the conflict: A theoretical review and critique of conflict-related ERPs. *International Journal of Psychophysiology*, *93*(3), 283–297.

Leiner, D. J. (2014). SoSci Survey (Version 2.4.00-i). Retrieved from https://www.soscisurvey.de

Li, Y., Wang, L.-Q., & Hu, Y. (2009). Localizing P300 generators in high-density event- related potential with fMRI. *Medical Science Monitor: International Medical Journal of Experimental and Clinical Research*, *15*(3), MT47–53.

Lo, B. C. Y., Lau, S., Cheung, S., & Allen, N. B. (2012). The impact of rumination on internal attention switching. *Cognition & Emotion*, *26*(2), 209–223.

Luu, P., Caggiano, D. M., Geyer, A., Lewis, J., Cohn, J., & Tucker, D. M. (2014). Time-course of cortical networks involved in working memory. *Frontiers in Human Neuroscience*, *8*, 1–15.

Machino, A., Kunisato, Y., Matsumoto, T., Yoshimura, S., Ueda, K., Yamawaki, Y., … Yamawaki, S. (2014). Possible involvement of rumination in gray matter abnormalities in persistent symptoms of major depression: An exploratory magnetic resonance imaging voxel-based morphometry study. *Journal of Affective Disorders*, *168*, 229–235.

Mandell, D., Siegle, G. J., Shutt, L., Feldmiller, J., & Thase, M. E. (2014). Neural substrates of trait ruminations in depression. *Journal of Abnormal Psychology*, *123*(1), 35–48.

Mangina, C. A., & Sokolov, E. N. (2006). Neuronal plasticity in memory and learning abilities: Theoretical position and selective review. *International Journal of Psychophysiology*, *60*(3), 203–214.

Marchetti, I., Koster, E. H. W., Sonuga-Barke, E. J., & de Raedt, R. (2012). The default mode network and recurrent depression: A neurobiological model of cognitive risk factors. *Neuropsychology Review*, *22*(3), 229–251.

Millner, A. J., Jaroszewski, A. C., Chamarthi, H., & Pizzagalli, D. A. (2012). Behavioral and electrophysiological correlates of training-induced cognitive control improvements. *NeuroImage*, *63*(2), 742–753.

Moberly, N. J., & Watkins, E. R. (2008). Ruminative self-focus, negative life events, and negative affect. *Behaviour Research and Therapy*, *46*(9), 1034–1039.

Mor, N., Hertel, P., Ngo, T. A., Shachar, T., & Redak, S. (2014). Interpretation bias characterizes trait rumination. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*(1), 67–73.

Mor, N., & Winquist, J. (2002). Self-focused attention and negative affect: A meta-analysis. *Psychological Bulletin*, *128*(4), 638–662.

Navon, D. (1977). Forest before trees: The precedence of global features in visual perception. *Cognitive Psychology*, *9*(3), 353–383.

Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience*, *3*(1), 17–26.

Nolen-Hoeksema, S. (1987). Sex differences in unipolar depression: Evidence and theory. *Psychological Bulletin*, *101*(2), 259–282.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, *100*(4), 569–582.

Nolen-Hoeksema, S., & Jackson, B. (2001). Mediators of the gender difference in rumination. *Psychology of Women Quarterly*, *25*(1), 37–47.

Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality*, *77*(5), 1061–1072.

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta earthquake. *Journal of Personality*, *61*(1), 115–121.

Nolen-Hoeksema, S., & Morrow, J. (1993). Effects of rumination and distraction on naturally occurring depressed mood. *Cognition & Emotion*, *7*(6), 561–570.

Nolen-Hoeksema, S., & Watkins, E. R. (2011). A heuristic for developing transdiagnostic models of psychopathology: Explaining multifinality and divergent trajectories. *Perspectives on Psychological Science*, *6*(6), 589–609.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, *3*(5), 400–424.

Oelhafen, S., Nikolaidis, A., Padovani, T., Blaser, D., Koenig, T., & Perrig, W. J. (2013). Increased parietal activity after training of interference control. *Neuropsychologia*, *51*(13), 2781–2790.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, *9*(1), 97–113.

Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping*, *25*(1), 46–59.

Papageorgiou, C., & Wells, A. (Eds.). (2003). *Depressive rumination: Nature, theory and treatment* (1st edition). Chichester, England; Hoboken, NJ, USA: Wiley.

Philippot, P., & Brutoux, F. (2008). Induced rumination dampens executive processes in dysphoric young adults. *Journal of Behavior Therapy and Experimental Psychiatry*, *39*(3), 219–227.

Piguet, C., Desseilles, M., Sterpenich, V., Cojan, Y., Bertschy, G., & Vuilleumier, P. (2014). Neural substrates of rumination tendency in non-depressed individuals. *Biological Psychology*, *103*, 195–202.

Pinal, D., Zurrón, M., & Díaz, F. (2014). Effects of load and maintenance duration on the time course of information encoding and retrieval in working memory: From perceptual analysis to post-categorization processes. *Frontiers in Human Neuroscience*, *8*, 165.

Pires, L., Leitão, J., Guerrini, C., & Simões, M. R. (2014). Event-related brain potentials in the study of inhibition: Cognitive control, source localization and age-related modulations. *Neuropsychology Review*, *24*(4), 461–490.

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, *118*(10), 2128–2148.

Pössel, P. (2011). Can Beck's theory of depression and the response style theory be integrated? *Journal of Counseling Psychology*, *58*(4), 618–629.

Raichle, M. E., & Snyder, A. Z. (2007). A default mode of brain function: A brief history of an evolving idea. *NeuroImage*, *37*(4), 1083–1090.

Randall, J. G., Oswald, F. L., & Beier, M. E. (2014). Mind-wandering, cognition, and performance: A theory-driven meta-analysis of attention regulation. *Psychological Bulletin*, *140*(6), 1411–1431.

R Development Core Team. (2008). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://www.R-project.org

Rietdijk, W. J. R., Franken, I. H. A., & Thurik, A. R. (2014). Internal consistency of event-related potentials associated with cognitive control: N2/P3 and ERN/Pe. *PLoS ONE*, *9*(7), 1–7.

Rood, L., Roelofs, J., Bögels, S. M., & Arntz, A. (2012). The effects of experimentally induced rumination, positive reappraisal, acceptance, and distancing when thinking about a stressful event on affect states in adolescents. *Journal of Abnormal Child Psychology*, *40*(1), 73–84.

Saliasi, E., Geerligs, L., Lorist, M. M., & Maurits, N. M. (2014). Neural correlates associated with successful working memory performance in older adults as revealed by spatial ICA. *PLoS ONE*, *9*(6), 1–13.

Schapkin, S. A., Falkenstein, M., Marks, A., & Griefahn, B. (2007). Practice-related effects in a go-nogo task. *Perceptual and Motor Skills*, *105*(3f), 1275–1288.

Schick, A., Wessa, M., Vollmayr, B., Kuehner, C., & Kanske, P. (2013). Indirect assessment of an interpretation bias in humans: Neurophysiological and behavioral correlates. *Frontiers in Human Neuroscience*, *7*, 1–11.

Schmitt, H., Ferdinand, N. K., & Kray, J. (2014). Age-differential effects on updating cue information: Evidence from event-related potentials. *Cognitive, Affective, & Behavioral Neuroscience*, *14*(3), 1115–1131.

Seligman, M. E. P. (2014). The real mental illnesses: Susan Nolen-Hoeksema (1959–2013) in memoriam. *Journal of Abnormal Psychology*, *123*(1), 1–2.

Siegle, G. J., Ghinassi, F., & Thase, M. E. (2007). Neurobehavioral therapies in the 21st century: Summary of an emerging field and an extended example of cognitive control training for depression. *Cognitive Therapy and Research*, *31*(2), 235–262.

Smith, J. M., & Alloy, L. B. (2009). A roadmap to rumination: A review of the definition, assessment, and conceptualization of this multifaceted construct. *Clinical Psychology Review*, *29*(2), 116–128.

Snyder, H. R. (2013). Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: A meta-analysis and review. *Psychological Bulletin*, *139*(1), 81–132.

Takano, K., & Tanno, Y. (2011). Diurnal variation in rumination. *Emotion*, *11*(5), 1046 – 1058.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*, *27*(3), 247–259.

Vanderhasselt, M.-A., Baeken, C., Van Schuerbeek, P., Luypaert, R., De Mey, J., & De Raedt, R. (2013). How brooding minds inhibit negative material: An event-related fMRI study. *Brain and Cognition*, *81*(3), 352–359.

Vanderhasselt, M.-A., & De Raedt, R. (2009). Impairments in cognitive control persist during remission from depression and are related to the number of past episodes: An event related potentials study. *Biological Psychology*, *81*(3), 169–176.

Vanderhasselt, M.-A., De Raedt, R., Namur, V., Lotufo, P. A., Bensenor, I. M., Boggio, P. S., & Brunoni, A. R. (2015). Transcranial electric stimulation and neurocognitive training in clinically depressed patients: A pilot study of the effects on rumination. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *57*, 93–99.

Van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience*, *14*(4), 593–602.

Västfjäll, D. (2002). Emotion induction through music: A review of the musical mood induction procedure. *Musicae Scientiae*, *5*(1 suppl), 173–211.

Von Bastian, C. C., Locher, A., & Ruflin, M. (2012). Tatool: A Java-based open-source programming framework for psychological studies. *Behavior Research Methods*, *45*(1), 108–115.

Wagner, C. A., Alloy, L. B., & Abramson, L. Y. (2014). Trait rumination, depression, and executive functions in early adolescence. *Journal of Youth and Adolescence*, *44*(1), 18–36.

Wagner, G., Sinsel, E., Sobanski, T., Köhler, S., Marinou, V., Mentzel, H.-J., … Schlösser, R. G. M. (2006). Cortical inefficiency in patients with unipolar depression: An event-related fMRI study with the Stroop Task. *Biological Psychiatry*, *59*(10), 958–965.

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, *134*(2), 163–206.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality*, *54*(6), 1063–1070.

Watter, S., Geffen, G. M., & Geffen, L. B. (2001). The n-back as a dual-task: P300 morphology under divided attention. *Psychophysiology*, *38*(6), 998–1003.

Wells, A. (2000). *Emotional disorders and metacognition innovative Cognitive Therapy*. New York: Wiley.

Westermann, R., Spies, K., Stahl, G., & Hesse, F. W. (1996). Relative effectiveness and validity of mood induction procedures: A meta-analysis. *European Journal of Social Psychology*, *26*(4), 557–580.

West, R., Bowry, R., & McConville, C. (2004). Sensitivity of medial frontal cortex to response and nonresponse conflict. *Psychophysiology*, *41*(5), 739–748.

Whitmer, A. J., & Gotlib, I. H. (2012). Switching and backward inhibition in major depressive disorder: The role of rumination. *Journal of Abnormal Psychology*, *121*(3), 570–578.

Whitmer, A. J., & Gotlib, I. H. (2013). An attentional scope model of rumination. *Psychological Bulletin*, *139*(5), 1036–1061.

Wimmer, T. (2010). *Positive und negative Metakognitionen über die Rumination und ihre differentiellen Effekte auf die kognitive Flexibilität bei dysphorisch/depressiven Frauen* (Doctoral dissertation, Westfälische Wilhelms-Universität Münster, Münster, Deutschland). Retrieved from http://miami.uni-muenster.de/Record/2ff91faf-5c87-47c5-9dc2-aaac555a0962

6. Appendices

6.1. Deutsche Zusammenfassung

Rumination ist ein etablierter Risikofaktor für die Entstehung von verschiedenen psychischen Erkrankungen. Erkenntnisse aus behavioralen und bildgebenden Studien haben Rumination mit Defiziten in kognitiver Kontrolle (KK) in Verbindung gesetzt. Dagegen ist die Beziehung zwischen Rumination und ereigniskorrelierten Potenzialen (EKP), die KK indizieren, weniger bekannt, während auch die kausale Beziehung zwischen KK und Rumination bei gesunden Individuen nicht ausreichend beschrieben ist. In der vorliegenden Studie wurden in einer Stichprobe von 33 gesunden Probanden EKPs eines *n*-back Tasks, die mit KK in Verbindung stehen, mit aktueller und habitueller Rumination, sowie mit Veränderungen in aktueller Rumination in Reaktion auf induzierte negative Stimmung in Zusammenhang gebracht. Eine Teilstichprobe (n=14) führte ein KK Training aus, um KK zu erhöhen und wurde mit einer Kontrollgruppe verglichen (n=16). Kausale Einflüsse von KK auf Rumination wurden durch dieses randomisierte prospektive kontrollierte Studiendesign untersucht. Die Ergebnisse zeigten, dass das Training die Anfälligkeit für Rumination reduzierte. EKP Amplituden, die mit Inhibition und Konfliktüberwachung (N2 und N4) verbunden sind, waren bei Personen mit hoher Anfälligkeit für Rumination größer. Die Ergebnisse zeigten auch eine Tendenz hinsichtlich einer Korrelation zwischen aktueller Rumination und einer EKP-Komponente, die mit Arbeitsgedächtnis-Aktualisierung verknüpft ist (P3). Mögliche divergierende Beziehungen zwischen KK und Rumination werden diskutiert.

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1. Descriptive Statistics

1.1. Descriptive Statistics of Trait Measures in Session 1

		Session 1					Session 2				
Group		*N*	*M*	*SD*	Min	Max	*N*	*M*	*SD*	Min	Max
Control											
	BDI-II	16	5.5	5.37	0	17	16	5.19	6	0	22
	RSQ-D rumination	16	44.63	8.33	33	64	16	44.88	10.66	31	70
Experimental											
	BDI-II	17	7.24	4.29	2	16	14	6.21	5.65	1	17
	RSQ-D rumination	16	51.63	10.98	30	67	14	50.86	8.69	29	62

1.2. Descriptive Statistics of State Measures

	Pre cMRI					Post cMRI				
Measure	*N*	*M*	*SD*	Minimum	Maximum	*N*	*M*	*SD*	Minimum	Maximum
Session 1										
PANAS NA	33	12.91	3.26	10	23	33	15.97	5.98	10	33
PANAS PA	33	28.88	7.13	12	43	33	23.88	8.23	12	42
VAS affect	33	39.24	55.76	0	257	33	107.67	71.46	1	248
VAS rumination	33	55.27	45.19	0	150	33	114.58	54.58	10	200

	Pre cMRI					Post cMRI				
Measure	*N*	*M*	*SD*	Minimum	Maximum	*N*	*M*	*SD*	Minimum	Maximum
SRQ rumination	33	98.85	72.21	0	303	33	162.73	100.18	0	315
SRQ flexibility	33	285.09	73.98	117	392	33	241.73	78.30	106	400
Session 2										
PANAS NA	29	11.83	2.93	10	24	30	14.43	4.40	10	26
PANAS PA	29	29.79	7.31	19	46	30	24.00	7.28	12	44
VAS affect	30	33.40	39.47	0	153	30	93.83	62.30	6	213
VAS rumination	30	43.97	44.94	0	165	30	111.47	35.04	21	180
SRQ rumination	30	92.13	80.63	3	264	30	134.93	82.92	11	297
SRQ flexibility	30	283.10	71.16	159	380	30	254.40	73.93	140	383

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1.3. Descripitve Statistics of N-Back Measures Session 1

		Control Group					Experimental Group				
Measure		N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
accuracy											
	0-Back	16	0.99	0.01	0.97	1.00	17	0.99	0.01	0.97	1.00
	1-Back	16	0.97	0.02	0.92	0.99	17	0.96	0.04	0.83	1.00
	2-Back	16	0.94	0.04	0.84	0.99	17	0.92	0.05	0.84	1.00
	3-Back	16	0.87	0.05	0.79	0.96	17	0.83	0.08	0.66	0.98
reaction time											
	0-Back	16	494.33	49.22	378.51	564.18	17	506.07	41.11	445.10	583.58
	1-Back	16	527.13	49.63	419.14	591.90	17	537.97	30.56	489.73	588.61
	2-Back	16	618.21	71.09	466.70	740.18	17	646.99	65.99	545.67	779.05
	3-Back	16	639.42	78.76	525.70	737.53	17	649.80	105.18	444.87	852.09
N2 amplitude in µV											
	0-Back	16	-1.84	2.63	-7.63	4.30	17	-2.02	3.32	-10.41	4.52
	1-Back	16	-1.07	3.12	-7.42	5.38	17	-0.65	2.47	-4.07	4.94
	2-Back	16	-0.10	2.56	-5.22	6.22	17	-0.29	1.94	-3.04	3.34
	3-Back	16	-0.01	1.80	-3.04	3.15	17	-1.21	2.47	-7.71	1.71
P3 amplitude in µV											
	0-Back	16	4.28	4.30	-0.02	13.83	17	6.95	5.67	-1.05	17.80
	1-Back	16	4.88	6.34	-0.58	23.90	17	7.22	6.24	-1.23	21.20
	2-Back	16	4.20	5.84	-0.48	23.02	17	6.18	5.70	-1.24	17.64
	3-Back	16	2.84	4.12	-1.29	15.67	17	5.18	3.81	-1.08	13.51

		Control Group					Experimental Group			
Measure		N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
N4 amplitude in µV											
	0-Back	16	-0.89	2.33	-5.01	4.22	17	-0.47	1.87	-2.50	3.39
	1-Back	16	-0.06	2.97	-4.23	6.22	17	-0.43	2.23	-4.19	5.57
	2-Back	16	0.26	2.02	-2.41	3.75	17	-0.47	2.03	-4.97	3.57
	3-Back	16	0.03	2.02	-4.01	4.23	17	-0.63	1.79	-5.79	2.26

1.4. Descriptive Statistics of N-Back Measures Session 2

		Control Group					Experimental Group			
Measure		N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
accuracy											
	0-Back	16	0.98	0.01	0.96	1.00	14	0.99	0.01	0.98	1.00
	1-Back	16	0.97	0.03	0.90	1.00	14	0.97	0.03	0.89	1.00
	2-Back	16	0.96	0.03	0.89	0.99	14	0.94	0.04	0.86	0.98
	3-Back	16	0.90	0.04	0.84	0.99	14	0.84	0.05	0.77	0.94
reaction time											
	0-Back	16	-4.29	4.07	-13.24	0.52	14	-3.44	2.90	-7.00	2.86
	1-Back	16	-2.92	3.32	-10.12	2.12	14	-2.63	3.02	-8.11	2.30
	2-Back	16	-2.46	4.12	-10.58	4.16	14	-1.94	2.59	-5.61	3.41
	3-Back	16	-2.34	4.40	-12.14	4.04	14	-2.53	2.42	-6.05	0.94
N2 amplitude in µV											
	0-Back	16	-1.97	2.58	-7.20	2.56	14	-1.49	2.98	-6.59	3.46
	1-Back	16	-1.60	3.45	-6.81	6.93	14	-1.49	3.34	-8.33	4.46
	2-Back	16	-1.12	3.62	-6.30	5.79	14	-1.20	2.72	-5.84	3.02
	3-Back	16	-0.65	2.86	-4.83	5.97	14	-1.25	1.93	-5.09	0.58

		Control Group					Experimental Group				
Measure		N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
P3 amplitude in µV											
	0-Back	16	3.42	4.84	-2.47	12.82	14	6.22	4.79	-1.94	13.54
	1-Back	16	4.36	5.64	-2.98	14.50	14	5.58	4.08	-2.58	11.86
	2-Back	16	3.46	4.53	-3.48	11.31	14	5.12	4.14	-2.44	9.42
	3-Back	16	2.51	3.21	-2.02	7.23	14	4.09	4.18	-2.45	11.57
N4 amplitude in µV											
	0-Back	16	460.02	40.87	373.77	534.67	14	461.43	37.27	404.98	539.13
	1-Back	16	501.74	50.01	414.58	608.94	14	486.67	37.19	429.77	541.93
	2-Back	16	553.92	54.48	471.91	656.23	14	561.23	69.75	449.47	689.85
	3-Back	16	592.80	65.36	493.86	710.43	14	601.46	82.18	447.76	751.86

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1.5. Comparison of Sample Characteristics in Groups

1.5.1. Normality distribution of sample characteristics

Group		Age in years		BDI-II		RSQ-D rum.		training duration in days	
	df	*D*	*p*	*D*	*p*	*D*	*p*	*D*	*p*
control	16	0.17	.68	0.18	.62	0.18	.58	0.21	.40
experimental	14	0.22	.44	0.24	.34	0.14	.92	0.23	.40

1.5.2. T-tests of sample-characteristics differences between groups

	equality of variances		independent *t*-test		
Measure	*F*	*p*	*t*(28)	*p*	*r*
Age in years	0.64	.43	1.21	.24	.22
BDI-II	0.24	.63	-0.89	.38	.17
RSQ-D rumiantion	0.87	.36	-2.59	.02	.44
training duration in days	0.72	.40	-0.75	.46	.14

2. Validation of Combined Mood and Rumination Induction

2.1. Test of Normality for Difference Scores of State Measures Pre- and Postinduction

Measure		df	*D*	*p*
Session 1				
	PANAS NA	29	0.22	.11
	PANAS PA	29	0.14	.53
	VAS rumination	30	0.10	.88
	SRQ rumination	30	0.12	.73
	SRQ flexibility	30	0.13	.64
	VAS affekt	30	0.15	.45
Session 2				
	PANAS NA	33	0.11	.76
	PANAS PA	33	0.11	.80
	VAS rumination	33	0.12	.69
	SRQ rumination	33	0.12	.71
	SRQ flexibility	33	0.13	.58
	VAS affekt	33	0.09	.95

2.2. Dependent *T*-Tests for Changes in State Measures Across Mood and Rumination Induction

	Session 1				Session 2			
Measure	*t*	df	*p*	*r*	*t*	df	*p*	*r*
PANAS NA	-4.12	32	.000	.59	-4.00	28	.000	.60
PANAS PA	4.37	32	.000	.61	6.29	28	.000	.77
VAS Rumination	-6.14	32	.000	.74	-8.99	29	.000	.86
SRQ rumination	-5.02	32	.000	.66	-3.35	29	.002	.53
SRQ flexibility	3.82	32	.001	.56	2.53	29	.017	.43
VAS affect	-6.70	32	.000	.76	-6.94	29	.000	.79

3. Correlations

3.1. Test of Normality Distribution in Rumination and *N*-Back Measures

		Session 1			Session 2		
		df	*D*	*p*	df	*D*	*p*
Accuracy							
	0-back	33	0.23	.06	30	0.21	.13
	1-back	33	0.22	.07	30	0.21	.11
	2-back	33	0.15	.38	30	0.19	.22
	3-back	33	0.14	.51	30	0.10	.89
N2							
	0-back	33	0.16	.33	30	0.15	.47
	1-back	33	0.18	.22	30	0.10	.88
	2-back	33	0.16	.32	30	0.13	.65
	3-back	33	0.11	.74	30	0.16	.38
N4							
	0-back	33	0.18	.19	30	0.11	.83
	1-back	33	0.19	.15	30	0.16	.37
	2-back	33	0.13	.63	30	0.09	.95
	3-back	33	0.11	.75	30	0.14	.59
P3							
	0-back	33	0.25	.03	30	0.18	.25
	1-back	33	0.17	.27	30	0.10	.92
	2-back	33	0.21	.10	30	0.19	.21
	3-back	33	0.14	.47	30	0.16	.36
RT							
	0-back	32	0.08	.97	30	0.10	.89
	1-back	33	0.10	.84	30	0.08	.98
	2-back	33	0.09	.92	30	0.10	.91
	3-back	33	0.08	.96	30	0.09	.93
VAS rumination		33	0.13	.63	30	0.11	.84
SRQ rumination		33	0.14	.47	30	0.20	.15
SRQ flexibility		33	0.12	.66	30	0.15	.42
dVAS rumination		33	0.09	.92	30	0.13	.61
dSRQ rumination		33	0.12	.69	30	0.10	.88
dSRQ flexibility		33	0.12	.71	30	0.12	.73
RSQ-D rumination		33	0.13	.58	30	0.13	.64

3.2. Pearson's Correlation Between *N*-Back Measures and Rumination Measures in Session 1

	SRQ rumination			SRQ flexibility			VAS rumination			dSRQ rumination			dSRQ flexibility			dVAS rumination			RSQ-D rumination		
	r	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*
Accuracy																					
0-back	-.17	.346	33	.13	.461	33	-.20	.267	33	.05	.761	33	.02	.896	33	.04	.845	33	-.03	.873	32
1-back	-.20	.270	33	-.11	.535	33	-.31	.076	33	-.02	.911	33	.13	.478	33	-.05	.802	33	-.40	.025	32
2-back	-.09	.607	33	.18	.326	33	-.32	.068	33	.00	.994	33	-.11	.553	33	.25	.159	33	.19	.294	32
3-back	.03	.851	33	.40	.020	33	-.24	.172	33	.01	.949	33	-.23	.202	33	.25	.168	33	.23	.208	32
RT																					
0-back	.29	.103	33	-.04	.843	33	.11	.560	33	-.20	.268	33	.15	.401	33	-.28	.113	33	.46	.008	32
1-back	.04	.813	33	-.04	.817	33	.08	.671	33	-.03	.884	33	.17	.343	33	-.27	.131	33	.31	.083	32
2-back	.01	.952	33	-.13	.458	33	-.04	.832	33	.12	.495	33	-.01	.952	33	-.16	.370	33	.07	.698	32
3-back	-.19	.290	33	.25	.161	33	-.10	.584	33	.31	.082	33	-.23	.199	33	.07	.681	33	.05	.793	32
N2																					
0-back	-.03	.859	33	-.21	.232	33	.05	.767	33	-.29	.102	33	.25	.165	33	-.45	.008	33	-.13	.467	32
1-back	-.04	.824	33	-.27	.127	33	-.03	.864	33	-.18	.312	33	.14	.450	33	-.30	.095	33	.09	.625	32
2-back	-.01	.948	33	-.14	.433	33	.10	.579	33	-.44	.010	33	.22	.222	33	-.43	.012	33	-.07	.703	32
3-back	-.08	.662	33	-.25	.160	33	.02	.902	33	-.34	.054	33	.29	.101	33	-.22	.218	33	-.21	.250	32
P3																					
0-back	.42	.015	33	-.21	.241	33	.05	.798	33	-.10	.572	33	-.07	.702	33	-.08	.667	33	.26	.147	32
1-back	.42	.016	33	-.27	.134	33	.04	.831	33	-.17	.334	33	-.04	.814	33	-.15	.404	33	.18	.313	32
2-back	.38	.029	33	-.20	.261	33	.17	.359	33	-.36	.038	33	.07	.719	33	-.38	.029	33	.18	.326	32
3-back	.41	.018	33	-.26	.142	33	.17	.355	33	-.26	.144	33	.00	.999	33	-.29	.103	33	.21	.260	32

	SRQ rumination			SRQ flexibility			VAS rumination			dSRQ rumination			dSRQ flexibility			dVAS rumination			RSQ-D rumination		
	r	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*
N4																					
0-back	.08	.676	33	-.09	.632	33	.07	.697	33	-.18	.316	33	-.02	.912	33	-.34	.050	33	.20	.268	32
1-back	.06	.735	33	-.24	.185	33	-.07	.703	33	-.15	.393	33	.08	.645	33	-.20	.264	33	.11	.539	32
2-back	.20	.265	33	-.10	.596	33	.14	.432	33	-.34	.050	33	.15	.415	33	-.29	.101	33	-.09	.628	32
3-back	.02	.899	33	-.30	.087	33	.07	.692	33	-.16	.379	33	.20	.268	33	-.13	.473	33	-.06	.758	32

Note: All *p*-values are reported two-tailed and uncorrected for familywise error rate. *r* = Pearson's correlation coefficient.

3.3. Pearson's Correlations Between *N*-Back Measures and Rumination Measures in Session 2

	SRQ rumination			SRQ flexibility			VAS rumination			dSRQ rumination			dSRQ flexibility			dVAS rumination			RSQ-D rumination		
	r	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*
Accuracy																					
0-back	-.13	.516	27	.11	.582	27	.03	.863	27	-.03	.857	27	.07	.703	27	-.01	.940	27	-.08	.682	27
1-back	.13	.501	27	-.24	.200	27	.03	.897	27	.16	.420	27	-.05	.811	27	.20	.307	27	-.08	.695	27
2-back	-.21	.276	27	-.01	.954	27	-.39	.038	27	.02	.918	27	-.02	.915	27	.41	.027	27	-.07	.709	27
3-back	-.20	.288	27	.02	.905	27	-.02	.904	27	-.03	.896	27	-.06	.758	27	.07	.726	27	.25	.195	27
RT																					
0-back	.00	.999	27	.10	.595	27	-.01	.941	27	.11	.555	27	-.07	.711	27	.01	.973	27	.38	.041	27
1-back	-.13	.512	27	.07	.700	27	-.02	.931	27	.17	.387	27	-.02	.925	27	-.07	.708	27	.32	.087	27
2-back	.10	.597	27	-.04	.824	27	-.04	.839	27	-.13	.507	27	.14	.468	27	-.08	.688	27	-.02	.927	27
3-back	.03	.886	27	.01	.945	27	-.12	.521	27	-.12	.542	27	.29	.130	27	-.10	.619	27	-.08	.689	27

	SRQ rumination			SRQ flexibility			VAS rumination			dSRQ rumination			dSRQ flexibility			dVAS rumination			RSQ-D rumination		
	r	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*
N2																					
0-back	-.16	.400	27	.12	.530	27	-.42	.023	27	-.45	.014	27	.47	.009	27	.05	.777	27	-.37	.050	27
1-back	-.30	.117	27	.20	.289	27	-.33	.079	27	-.25	.199	27	.28	.145	27	.13	.507	27	-.23	.227	27
2-back	-.11	.570	27	.06	.765	27	-.35	.063	27	-.50	.005	27	.51	.004	27	.05	.786	27	-.34	.067	27
3-back	-.13	.489	27	.16	.404	27	-.36	.054	27	-.41	.026	27	.36	.057	27	.06	.745	27	-.28	.138	27
P3																					
0-back	-.05	.812	27	.16	.398	27	.19	.324	27	-.21	.270	27	.21	.284	27	-.03	.896	27	-.16	.402	27
1-back	.10	.602	27	-.02	.912	27	.35	.064	27	-.14	.481	27	.20	.287	27	-.05	.783	27	-.09	.626	27
2-back	.14	.483	27	-.11	.569	27	.30	.111	27	-.24	.206	27	.31	.104	27	-.09	.654	27	-.01	.960	27
3-back	.20	.288	27	-.09	.656	27	.47	.011	27	-.25	.187	27	.24	.205	27	-.21	.282	27	.10	.590	27
N4																					
0-back	.05	.779	27	.01	.974	27	-.08	.694	27	-.45	.014	27	.50	.006	27	.02	.915	27	-.27	.150	27
1-back	.04	.846	27	.03	.875	27	.10	.603	27	-.15	.426	27	.22	.262	27	.04	.829	27	-.11	.557	27
2-back	.22	.245	27	-.23	.238	27	.03	.886	27	-.48	.008	27	.60	.001	27	-.02	.904	27	-.25	.186	27
3-back	.22	.260	27	-.11	.558	27	.05	.781	27	-.45	.015	27	.52	.004	27	-.03	.863	27	-.20	.310	27

Note: All *p*-values are reported two-tailed and uncorrected for familywise error rate. Correlation coefficients refer to partial correlations, adjusting for factor group. *r* = Pearson's correlation coefficient.

3.4. Correlations of Changes Across Sessions in *N*-Back Measures and Rumination Measures in the Experimental Group

		RSQ-D difference		SRQ rumination difference		SRQ flexibility difference		VAS rumination difference		dSRQ rumination difference		dSQR flexibility difference		dVAS rumination difference	
		r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
ACC															
	0-back	.17	.57	-.26	.38	.35	.22	.41	.15	-.61	.02	.27	.36	-.70	.01
	1-back	-.23	.44	-.09	.76	.37	.19	.25	.38	-.29	.31	-.05	.86	-.38	.18
	2-back	.16	.58	.27	.35	.00	.99	-.39	.16	-.38	.18	.30	.30	.15	.61
	3-back	.28	.34	-.22	.45	.29	.32	-.07	.82	-.26	.38	.15	.60	-.31	.28
RT															
	0-back	.13	.66	.18	.53	-.01	.98	-.15	.61	-.07	.81	.06	.84	.05	.87
	1-back	.21	.47	-.42	.13	.05	.88	.01	.98	.09	.77	.06	.85	-.15	.60
	2-back	.16	.59	-.28	.34	.21	.46	.05	.86	-.02	.94	.14	.63	-.19	.52
	3-back	-.02	.95	.11	.72	.14	.63	.07	.82	-.06	.84	.03	.92	-.12	.69
N2															
	0-back	-.23	.44	-.31	.28	-.10	.74	.06	.85	.04	.89	.08	.78	-.07	.81
	1-back	-.10	.73	-.52	.06	.08	.79	-.04	.90	.20	.49	-.15	.62	-.02	.94
	2-back	.12	.67	-.28	.33	-.14	.63	.19	.50	.00	1.00	.03	.92	-.21	.48
	3-back	.20	.49	-.14	.62	-.22	.46	.39	.17	.06	.84	.03	.92	-.27	.35
P3															
	0-back	.14	.64	-.21	.47	.21	.47	-.51	.07	-.13	.66	-.05	.87	.16	.58
	1-back	.14	.63	-.23	.43	.20	.50	-.54	.05	-.04	.88	-.08	.79	.21	.46
	2-back	.34	.24	-.30	.29	.09	.75	-.39	.17	-.11	.70	-.01	.98	.03	.93

	RSQ-D difference		SRQ rumination difference		SRQ flexibility difference		VAS rumination difference		dSRQ rumination difference		dSQR flexibility difference		dVAS rumination difference	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
3-back	.23	.42	-.26	.38	.21	.47	-.20	.49	-.09	.76	-.15	.60	-.05	.88
N4														
0-back	-.17	.56	-.23	.42	-.16	.58	-.50	.07	.44	.12	-.14	.63	.53	.05
1-back	-.15	.61	-.33	.25	-.06	.85	-.49	.07	.42	.13	-.03	.91	.51	.06
2-back	-.18	.54	-.11	.70	-.41	.14	-.42	.14	.18	.54	.25	.40	.47	.09
3-back	-.08	.79	-.18	.54	-.35	.22	-.21	.47	.57	.03	-.10	.73	.47	.09

Note: All *p*-values are reported two sided and uncorrected for familywise error rate

3.5. Correlation of N2 Amplitudes Within and Between Sessions

		N2											
		0-back			1-back			2-back			3-back		
		r	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*
N2	0-back	-.16	.41	30	.88	.00	30	.88	.00	30	.79	.00	30
	1-back	.77	.00	33	.04	.82	30	.90	.00	30	.85	.00	30
	2-back	.59	.00	33	.82	.00	33	.19	.31	30	.93	.00	30
	3-back	.42	.02	33	.55	.00	33	.79	.00	33	.09	.65	30

Note: All *p*-values are calculated two sided and uncorrected for familywise error rate Correlations beyond the diagonal indicate correlations in Session 1, correlations above the diagonal indicate correlations in Session 2. Correlations on the diagonal indicate correlations between Sessions.

3.6. Correlations of P3 Amplitudes Within and Between Sessions

		0-back			1-back			2-back			3-back		
P3	0-back	.26	.16	30	.88	.00	30	.88	.00	30	.90	.00	30
	1-back	.93	.00	33	.10	.59	30	.91	.00	30	.85	.00	30
	2-back	.81	.00	33	.91	.00	33	.27	.14	30	.93	.00	30
	3-back	.84	.00	33	.91	.00	33	.93	.00	33	.33	.07	30

Note: All *p*-values are calculated two sided and uncorrected for familywise error rate Correlations beyond the diagonal indicate correlations in Session 1, correlations above the diagonal indicate correlations in Session 2. Correlations on the diagonal indicate correlations between Sessions.

3.7. Correlations of N4 Amplitudes Within and Between Sessions

		0-back			1-back			2-back			3-back		
		r	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*	*r*	*p*	*n*
N4	0-back	.15	.44	30	.81	.00	30	.85	.00	30	.82	.00	30
	1-back	.80	.00	33	.09	.64	30	.81	.00	30	.74	.00	30
	2-back	.50	.00	33	.70	.00	33	.22	.24	30	.86	.00	30
	3-back	.55	.00	33	.75	.00	33	.61	.00	33	.18	.35	30

Note: All *p*-values are calculated two sided and uncorrected for familywise error rate. Correlations beyond the diagonal indicate correlations in Session 1, correlations above the diagonal indicate correlations in Session 2. Correlations on the diagonal indicate correlations between Sessions.

4. Training Effects on *N*-Back Measures

4.1. Test for Normality Distribution Within Groups

	Session 1				Session 2			
	Control Group		Experimental Group		Control Group		Experimental Group	
	D(16)	*p*	*D*(14)	*p*	*D*(16)	*p*	*D*(14)	*p*
Accuracy								
0-back	0.21	.42	0.25	.30	0.19	.56	0.21	.48
1-back	0.23	.33	0.27	.20	0.19	.55	0.28	.18
2-back	0.19	.52	0.11	.99	0.21	.43	0.18	.66
3-back	0.13	.91	0.15	.84	0.16	.72	0.11	.99
RT (ms)								
0-back	0.13	.91	0.17	.74	0.11	.97	0.11	.99
1-back	0.15	.82	0.10	1.00	0.16	.75	0.10	.99
2-back	0.11	.99	0.18	.72	0.19	.55	0.16	.79
3-back	0.16	.75	0.14	.90	0.17	.72	0.22	.45
N2 (µV)								
0-back	0.11	.98	0.23	.40	0.26	.20	0.18	.70
1-back	0.15	.82	0.22	.46	0.13	.91	0.12	.97
2-back	0.24	.26	0.23	.37	0.22	.37	0.20	.56
3-back	0.13	.91	0.15	.86	0.29	.11	0.16	.83
N4 (µV)								
0-back	0.19	.56	0.21	.51	0.16	.77	0.15	.85
1-back	0.21	.44	0.21	.50	0.21	.41	0.16	.83
2-back	0.15	.80	0.19	.65	0.19	.57	0.13	.94
3-back	0.13	.92	0.23	.41	0.14	.86	0.22	.45
P3 (µV)								
0-back	0.30	.08	0.21	.50	0.20	.46	0.16	.82
1-back	0.30	.09	0.10	1.00	0.17	.71	0.13	.95
2-back	0.32	.06	0.22	.47	0.20	.49	0.21	.48
3-back	0.19	.57	0.15	.87	0.19	.58	0.15	.88
VAS rumination	0.19	.57	0.20	.56	0.20	.49	0.20	.59
SRQ rumination	0.15	.80	0.18	.70	0.24	.29	0.13	.94
SRQ flexibility	0.15	.80	0.17	.77	0.20	.49	0.11	.99
dVAS rumination	0.13	.93	0.13	.94	0.09	1.00	0.15	.84
dSRQ rumination	0.16	.75	0.17	.72	0.20	.47	0.12	.97
dSRQ flexibility	0.14	.86	0.14	.92	0.17	.66	0.18	.69
RSQ-D rumination	0.18	.58	0.14	.92	0.13	.93	0.15	.89

4.2. Test of Assumption of Sphericity

Within Subjects Effect		Mauchly's W	χ(5)2	*p*
N-back load				
	N2	.37	26.80	.00
	P3	.59	14.17	.01
	N4	.75	7.53	.18
	RT	.30	32.43	.00
	ACC	.45	21.47	.00
Group×Time				
	N2	.57	15.03	.01
	P3	.60	13.58	.02
	N4	.64	11.79	.04
	RT	.44	21.70	.00
	ACC	.60	13.73	.02

4.3. Test of Homogeneity of Variances

	Session 1		Session 2	
	F(1,28)	*p*	*F*(1,28)	*p*
N2				
0-back	2.18	.15	0.57	.45
1-back	0.05	.83	0.11	.74
2-back	1.40	.25	0.03	.86
3-back	2.87	.10	1.09	.31
P3				
0-back	0.00	.96	3.34	.08
1-back	3.37	.08	0.28	.60
2-back	0.46	.50	0.56	.46
3-back	0.79	.38	0.01	.92
N4				
0-back	0.64	.43	0.03	.86
1-back	0.01	.94	0.44	.51
2-back	0.95	.34	0.61	.44
3-back	1.43	.24	1.48	.23
RT				
0-back	0.31	.58	0.05	.83
1-back	2.49	.13	0.24	.63
2-back	0.00	.97	0.44	.51
3-back	0.17	.68	0.14	.71
ACC				
0-back	0.04	.84	0.09	.77
1-back	1.49	.23	0.23	.64
2-back	0.71	.41	1.76	.20
3-back	0.02	.89	0.63	.43

4.4. ANOVA of ERP Measures

Source		df	*F*	*p*	η^2_p
Within Subject Effects					
NBACK					
	N2 GG	1.87, 52.27	13.63	.00	.33
	P3 GG	2.42, 67.75	10.64	.00	.28
	N4	3, 84	1.80	.15	.06
NBACK*Group					
	N2 GG	1.87, 52.27	1.80	.15	.06
	P3 GG	2.42, 67.75	1.98	.15	.07
	N4	3, 84	1.80	.15	.06
Time					
	N2	1, 28	6.17	.02	.18
	P3	1, 28	0.62	.44	.02
	N4	1, 28	2.76	.11	.09
Time * Group					
	N2 GG	1, 28	0.32	.58	.01
	P3 GG	1, 28	0.05	.82	.00
	N4 GG	1, 28	0.16	.69	.01
NBack * Time					
	N2 GG	2.22, 62.15	0.13	.90	.00
	P3 GG	2.40, 67.08	0.06	.96	.00
	N4 GG	2.49, 69.70	0.87	.46	.03
NBack * Time * Group					
	N2 GG	2.22, 62.15	0.95	.40	.03
	P3 GG	2.40, 67.08	0.26	.81	.01
	N4 GG	2.49, 69.70	0.48	.66	.02
Between Subject Effects					
Group					
	N2	1, 28	.00	.95	.00
	P3	1, 28	2.405	.13	.08
	N4	1, 28	.139	.71	.00

4.5. Contrasts of Factor *N*-Back Load for Neuronal Measures

	N2			P3			N4		
Comparison	*F*	*p*	*r*	*F*	p	r	*F*	*p*	*r*
n-back load									
0-back vs. 1-back	18.06	.00	.63	0.45	.51	.13	1.62	.21	.23
1-back vs. 2-back	7.14	.01	.45	5.42	.03	.40	0.64	.43	.15
2-back vs. 3-back	4.74	.04	.38	12.30	.00	.55	0.05	.83	.04
N-back load × Group									
0-back vs. 1-back	0.04	.85	.04	2.98	.10	.31	3.06	.09	.31
1-back vs. 2-back	0.45	.51	.13	0.00	.98	.01	0.60	.44	.14
2-back vs. 3-back	7.82	.01	.47	0.18	.67	.08	0.15	.70	.07
N-back × Time									
0-back vs. 1-back	0.02	.88	.03	0.05	.82	.04	0.07	.80	.05
1-back vs. 2-back	0.00	.99	.00	0.11	.74	.06	0.51	.48	.13
2-back vs. 3-back	0.49	.49	.13	0.03	.87	.03	0.44	.51	.12
N-back load × Group × Time									
0-back vs. 1-back	2.19	.15	.27	0.66	.42	.15	0.41	.53	.12
1-back vs. 2-back	1.71	.20	.24	0.50	.48	.13	0.16	.69	.08
2-back vs. 3-back	0.36	.56	.11	0.39	.54	.12	0.56	.46	.14

Note: For *F* statisitics: df = 1,28

4.6. ANOVA of Behavioral Measures

Source		df	*F*	*p*	η^2_p
Within Subject Effects					
NBACK					
RT	GG	1.73, 48.54	78.53	.00	.74
ACC	GG	2.05, 57.48	130.11	.00	.82
NBack * Group					
RT	GG	1.73, 48.54	0.58	.54	.02
ACC	GG	2.05, 57.48	4.59	.01	.14
Time					
RT		1, 28	58.42	.00	.68
ACC		1, 28	4.34	.05	.13
Time * Group					
RT		1, 28	3.26	.08	.10
ACC		1, 28	0.29	.60	.01
NBack * Time					
RT	GG	2.24, 62.76	5.73	.00	.17
ACC	GG	2.34, 65.56	1.76	.18	.06
NBack * Time * Group					
RT	GG	2.24, 62.76	0.34	.74	.01
ACC	GG	2.34, 65.56	2.66	.07	.09

Source	df	*F*	*p*	η^2_p
Between Subject Effects				
Group				
RT	1, 28	1.04	.32	.04
ACC	1, 28	3.98	.06	.12

4.7. Contrasts of Factor *N*-Back Load for Behavioral Measures

		RT			ACC		
Effect	Comparison	*F*(1,28)	*p*	*r*	*F*(1,28)	*p*	*r*
n-back load							
	0-back vs. 1-back	34.15	.00	.74	33.68	.00	.74
	1-back vs. 2-back	81.90	.00	.86	15.32	.00	.59
	2-back vs. 3-back	8.86	.01	.49	132.83	.00	.91
N-back load × Group							
	0-back vs. 1-back	0.46	.50	.13	0.41	.53	.12
	1-back vs. 2-back	1.77	.19	.24	1.49	.23	.22
	2-back vs. 3-back	0.02	.90	.02	3.30	.08	.32
N-back load × Time							
	0-back vs. 1-back	0.00	.97	.01	0.47	.50	.13
	1-back vs. 2-back	12.50	.00	.56	1.72	.20	.24
	2-back vs. 3-back	3.06	.09	.31	0.10	.76	.06
N-back load × Time × Group							
	0-back vs. 1-back	1.80	.19	.25	0.27	.61	.10
	1-back vs. 2-back	0.04	.84	.04	0.34	.56	.11
	2-back vs. 3-back	0.10	.76	.06	3.84	.06	.35

4.8. Follow-up *T*-Tests of Factor Time for *N*-Back Measures

	Control Goup			Experimental Group		
Measure	*t*(15)	*p*	*r*	*t*(13)	*p*	*r*
Accurracy						
0-back	1.31	.21	.32	-0.72	.49	.20
1-back	0.28	.79	.07	-0.84	.41	.23
2-back	-1.59	.13	.38	-1.33	.21	.35
3-back	-3.35	.00	.65	0.19	.85	.05
RT						
0-back	3.07	.01	.62	5.42	.00	.83
1-back	3.04	.01	.62	5.57	.00	.84
2-back	3.50	.00	.67	6.29	.00	.87
3-back	2.37	.03	.52	4.51	.00	.78

Measure	Control Goup			Experimental Group		
	t(15)	*p*	*r*	*t*(13)	*p*	*r*
N2						
0-back	1.87	.08	.43	0.95	.36	.25
1-back	1.71	.11	.40	1.78	.10	.44
2-back	2.26	.04	.50	1.64	.12	.41
3-back	2.24	.04	.50	0.92	.38	.25
P3						
0-back	0.55	.59	.14	0.53	.60	.15
1-back	0.24	.81	.06	0.85	.41	.23
2-back	0.42	.68	.11	0.62	.54	.17
3-back	0.28	.79	.07	0.98	.34	.26
N4						
0-back	1.16	.26	.29	1.56	.14	.40
1-back	1.27	.22	.31	1.09	.30	.29
2-back	1.44	.17	.35	0.49	.64	.13
3-back	0.89	.39	.22	0.56	.58	.15

Note: All *p*-values are given uncorrected for accumultation of Type 1 error.

5. Training Effects on Rumination Measures

5.1. Test for Normality Distribution Within Groups

	Session 1				Session 2			
	Control		Experimental		Control		Experimental	
	D(16)	*p*	*D*(14)	*p*	*D*(16)	*p*	*D*(14)	*p*
SRQ rumination	0.15	.20	0.18	.20	0.24	.02	0.13	.20
SRQ flexibility	0.15	.20	0.17	.20	0.20	.09	0.11	.20
VAS rumination	0.19	.14	0.20	.13	0.20	.09	0.20	.15
dSRQ rumination	0.16	.20	0.17	.20	0.20	.08	0.12	.20
dSRQ flexibility	0.14	.20	0.14	.20	0.17	.20	0.18	.20
dVAS rumination	0.13	.20	0.13	.20	0.09	.20	0.15	.20
RSQ-D rumiantion	0.18	.15	0.14	.20	0.13	.20	0.15	.20

5.2. Test for Homogeneity of Variances

Measure	Control Group		Experimental Group	
	F(1,28)	*p*	*F*(1,28)	*p*
SRQ rumination	0.21	.65	1.11	.30
SRQ flexibility	0.66	.42	0.83	.37
VAS rumination	0.08	.78	1.42	.24
dSRQ rumination	2.54	.12	0.02	.89
dSRQ flexibility	0.87	.36	0.26	.62
dVAS rumination	0.19	.67	3.41	.08
RSQ-D rumiantion	0.87	.36	0.49	.49

5.3. Mixed-Measures Design ANOVA of Rumination Measures

	$F(1,28)$	p	η^2_p
Wthin-subject effects			
Time			
SRQ rumination	0.15	.697	.005
SRQ flexibility	0.66	.422	.023
VAS rumination	1.18	.286	.041
dSRQ rumination	5.28	.029	.159
dSRQ flexibility	3.62	.067	.115
dVAS rumination	0.05	.829	.002
RSQ rumination	0.92	.345	.032
Time × Group			
SRQ rumination	0.00	.970	.000
SRQ flexibility	1.10	.302	.038
VAS rumination	0.51	.482	.018
dSRQ rumination	6.72	.015	.194
dSRQ flexibility	0.00	.952	.000
dVAS rumination	3.69	.065	.116
RSQ rumination	1.39	.248	.047
Between-subject effects			
Group			
SRQ rumination	0.66	.42	.02
SRQ flexibility	0.56	.46	.02
VAS rumination	1.43	.24	.05
dSRQ rumination	3.57	.07	.11
dSRQ flexibility	3.98	.06	.12
dVAS rumination	0.02	.89	.00
RSQ rumination	4.99	.03	.15

5.4. Follow up *T*-Tests of Factor Time

	Control Group			Experimental Group		
Measure	*t*(15)	*p*	*r*	*t*(15)	*p*	*r*
RSQ-D	-.168	.87	.04	1.405	.18	.36
SRQ rumination	.273	.79	.07	.281	.78	.08
SRQ flexibility	1.125	.28	.28	-.240	.81	.07
VAS rumination	1.323	.21	.32	.256	.80	.07
dSRQ rumination	-.233	.82	.06	3.099	.01	.65
dSRQ flexibility	-1.483	.16	.36	-1.219	.24	.32
dVAS rumination	-1.828	.09	.43	1.019	.33	.27

Note: All *p*-values are given uncorrected for accumultation of Type 1 error.

6. Regression Models of Training Effects

6.1. ANOVA of Rregression Models

	dSRQ rumination			dVAS rumination		
Model	df	*F*	*p*	df	*F*	*p*
Reference	1,28	6.72	.02	1,28	3.69	.07
N2	5,24	3.79	.01	5,24	0.84	.53
P3	5,24	1.46	.24	5,24	2.89	.04
N4	5,24	1.91	.13	5,24	0.88	.51
RT	5,24	1.84	.14	5,24	1.00	.44
ACC	5,24	6.02	.00	5,24	4.43	.01

6.2. Model Quality Changes to Reference Model

				Change Statistics			
Dependent Variable	Model	*R*	R^2	Δ*R*	Δ*F*	df	*p*
dSRQ rumination							
	Reference	0.44	0.19				
	N2	0.66	0.44	0.25	2.66	4,28	.06
	P3	0.48	0.23	0.04	0.31	4,24	.87
	N4	0.53	0.28	0.09	0.76	4,25	.56
	RT	0.53	0.28	0.08	0.69	4,24	.60
	ACC	0.75	0.56	0.36	4.91	4,24	.00
dVAS rumination							
	Reference	0.34	0.12				
	N2	0.39	0.15	0.03	0.23	4,28	.92
	P3	0.61	0.38	0.26	2.49	4,24	.07
	N4	0.39	0.15	0.04	0.27	4,25	.89
	RT	0.42	0.17	0.06	0.41	4,24	.80
	ACC	0.69	0.48	0.36	4.20	4,24	.01

6.3. Normal Distribution of Model Residuals

	Dep.variable changes dSRQ rum.		Dep. variable changes dVAS rum.	
Model	*D*(30)	*p*	*D*(30)	*p*
Referenc e	0.15	.49	0.10	.88
N2	0.10	.87	0.15	.44
P3	0.18	.27	0.11	.80
N4	0.12	.73	0.09	.94
RT	0.13	.68	0.13	.65
ACC	0.10	.92	0.13	.67

6.4. Coefficients of Regression Models With DSRQ as Dependent Variable

Model		B	SE	β	*t*(24)	*p*
Reference						
	(Constant)	3.75	17.42		0.22	.831
	Group	-66.11	25.50	-0.44	-2.59	.015
N2						
	(Constant)	-6.28	17.47		-0.36	.722
	Group	-42.58	24.27	-0.28	-1.75	.092
	N2 diff.					
	0-back	-12.31	5.63	-0.82	-2.19	.039
	1-back	19.76	6.41	1.08	3.08	.005
	2-back	-6.88	9.20	-0.34	-0.75	.462
	3-back	-0.11	5.93	-0.01	-0.02	.985
P3						
	(Constant)	1.07	18.73		0.06	.955
	Group	-61.96	27.81	-0.41	-2.23	.036
	P3 diff.					
	0-back	-3.56	5.61	-0.29	-0.63	.532
	1-back	7.63	7.00	0.76	1.09	.286
	2-back	-3.36	6.67	-0.28	-0.50	.619
	3-back	-3.31	8.24	-0.20	-0.40	.692
N4						
	(Constant)	5.78	18.67		0.31	.759
	Group	-73.09	26.91	-0.49	-2.72	.012
	N4 diff.					
	0-back	-12.18	9.56	-0.52	-1.27	.215
	1-back	6.85	5.63	0.37	1.22	.235
	2-back	0.80	6.68	0.04	0.12	.906
	3-back	5.25	9.92	0.20	0.53	.602
RT						
	(Constant)	-20.37	24.61		-0.83	.416
	Group	-73.16	28.98	-0.49	-2.52	.019
	RT diff.					
	0-back	-0.55	0.42	-0.29	-1.33	.198
	1-back	0.19	0.55	0.09	0.34	.735
	2-back	-0.08	0.24	-0.07	-0.34	.739
	3-back	-0.10	0.26	-0.09	-0.39	.698

Model		B	SE	β	*t*(24)	*p*
ACC						
	(Constant)	-10.73	16.20		-0.66	.514
	Group	-33.40	23.27	-0.22	-1.44	.164
	ACC diff.					
	0-back	-4295.77	1075.58	-0.62	-3.99	.001
	1-back	134.40	309.44	0.07	0.43	.668
	2-back	-488.83	257.19	-0.30	-1.90	.069
	3-back	314.65	250.22	0.21	1.26	.221

6.5. Coefficients of Regression Models With DVAS as Dependent Variable

Model		B	SE	β	*t*(24)	*p*
Reference						
	Constant	22.06	14.10		1.56	.13
	Group	-39.63	20.64	-0.34	-1.92	.07
N2						
	Constant	18.80	16.67		1.13	.27
	Group	-32.43	23.15	-0.28	-1.40	.17
	N2 diff.					
	0-back	-2.60	5.37	-0.22	-0.48	.63
	1-back	5.58	6.11	0.40	0.91	.37
	2-back	-1.70	8.78	-0.11	-0.19	.85
	3-back	-1.39	5.66	-0.10	-0.25	.81
P3						
	Constant	23.98	13.07		1.84	.08
	Group	-45.34	19.40	-0.39	-2.34	.03
	P3 diff.					
	0-back	6.22	3.91	0.65	1.59	.13
	1-back	2.76	4.88	0.36	0.57	.58
	2-back	-1.95	4.66	-0.21	-0.42	.68
	3-back	-10.34	5.75	-0.79	-1.80	.08
N4						
	Constant	24.57	15.69		1.57	.13
	Group	-40.54	22.62	-0.35	-1.79	.09
	N4 diff.					
	0-back	3.43	8.03	0.19	0.43	.67
	1-back	-1.43	4.73	-0.10	-0.30	.76
	2-back	3.38	5.62	0.19	0.60	.55
	3-back	-5.38	8.34	-0.26	-0.65	.52

Model		B	SE	β	*t*(24)	*p*
RT						
	Constant	8.44	20.35		0.41	.68
	Group	-47.86	23.96	-0.41	-2.00	.06
	RT diff.					
	0-back	-0.25	0.35	-0.17	-0.72	.48
	1-back	-0.16	0.45	-0.10	-0.34	.73
	2-back	-0.14	0.20	-0.16	-0.70	.49
	3-back	0.17	0.21	0.20	0.78	.44
ACC						
	Constant	6.35	13.56		0.47	.64
	Group	-20.57	19.47	-0.18	-1.06	.30
	ACC diff.					
	0-back	-2702.47	900.29	-0.50	-3.00	.01
	1-back	-221.72	259.01	-0.14	-0.86	.40
	2-back	303.03	215.28	0.24	1.41	.17
	3-back	46.26	209.44	0.04	0.22	.83

6.3. Figures

Content

1. ERP Maps I

1.1. N2-ERP MAP at FZ, 280 ms Post Stimulus I

1.2. P3-ERP MAP at PZ, 380 ms Post Stimulus I

1.3. N4-ERP MAP at FZ, 440 ms Post Stimulus II

2. Grand Averages II

2.1. Grand Averages of the Control Group at FZ II

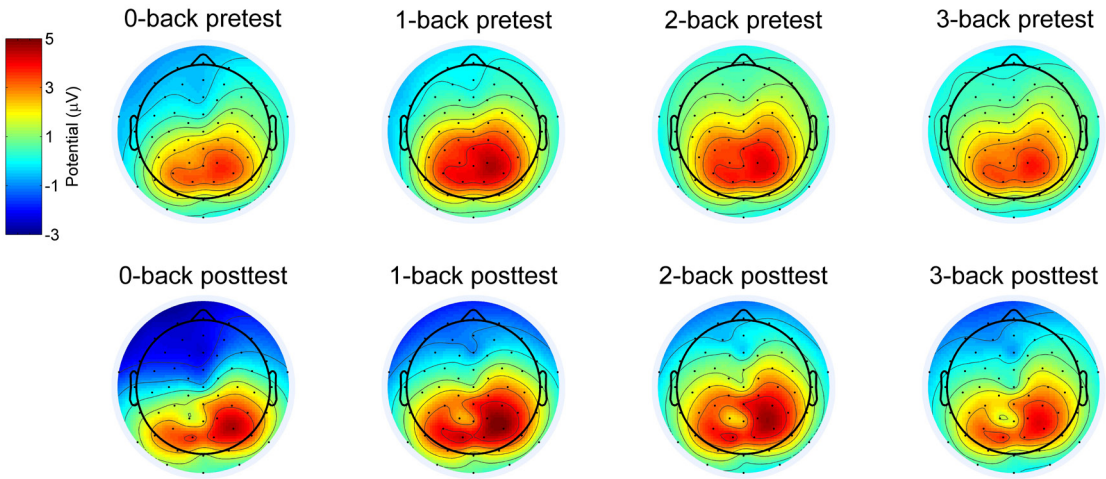
2.2. Grand Averages of the Experimental Group FZ III

2.3. Grand Averages of the Control Group at PZ III

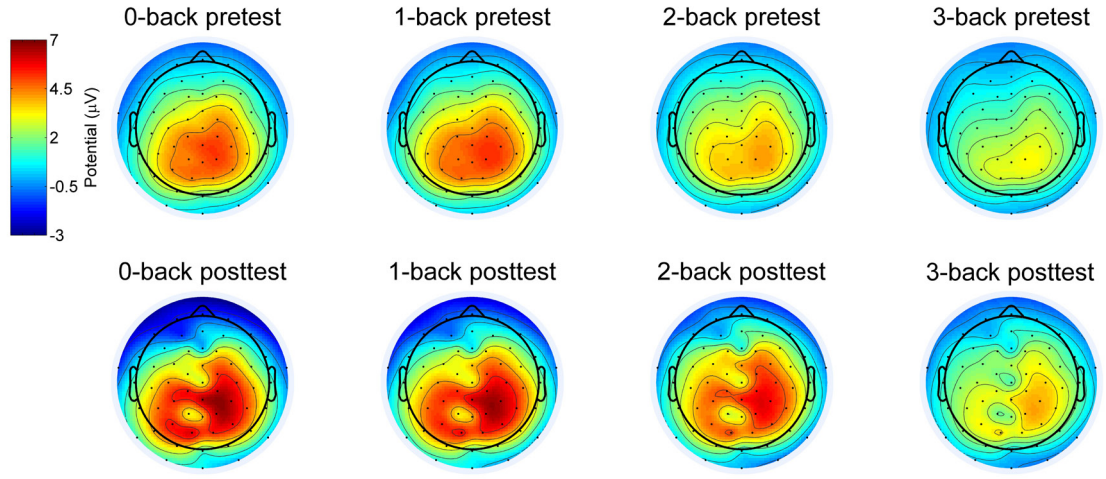
2.4. Grand Averages of the Experimental Group PZ IV

1. ERP Maps

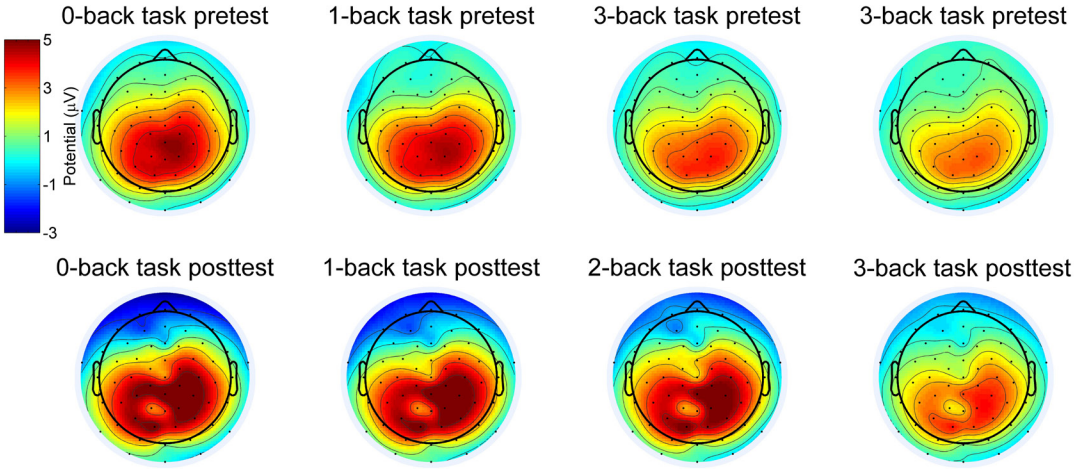
1.1. N2-ERP MAP at FZ, 280 ms Post Stimulus



1.2. P3-ERP MAP at PZ, 380 ms Post Stimulus

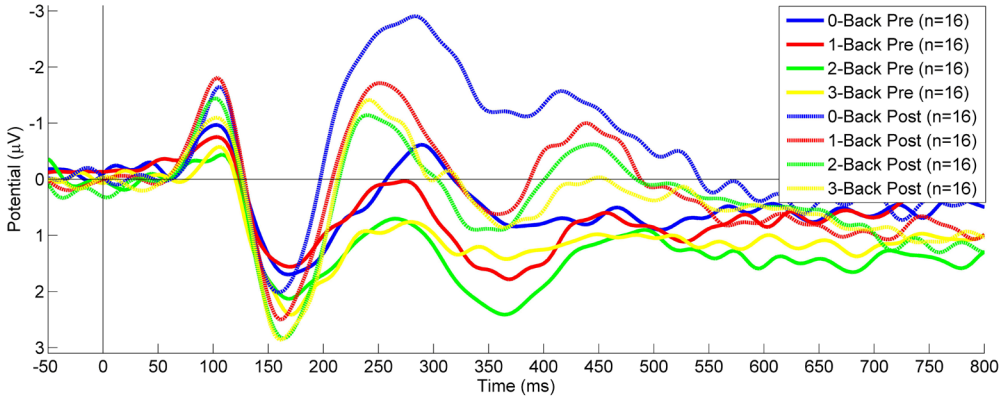


1.3. N4-ERP MAP at FZ, 440 ms Post Stimulus

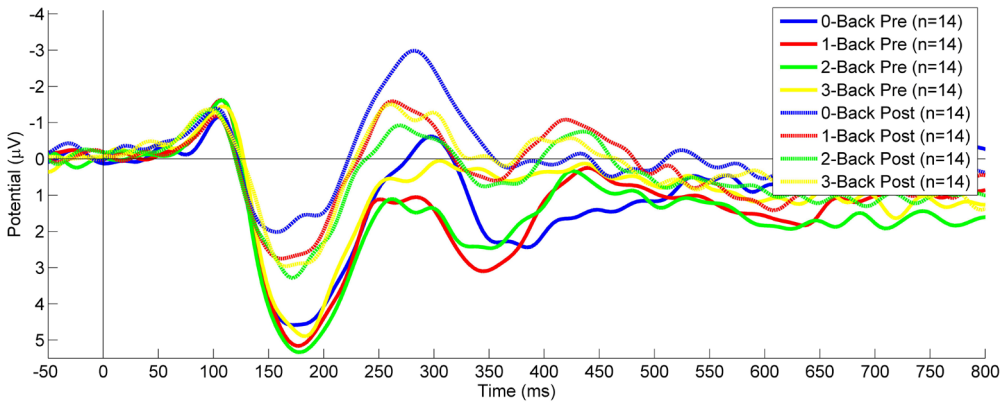


2. Grand Averages

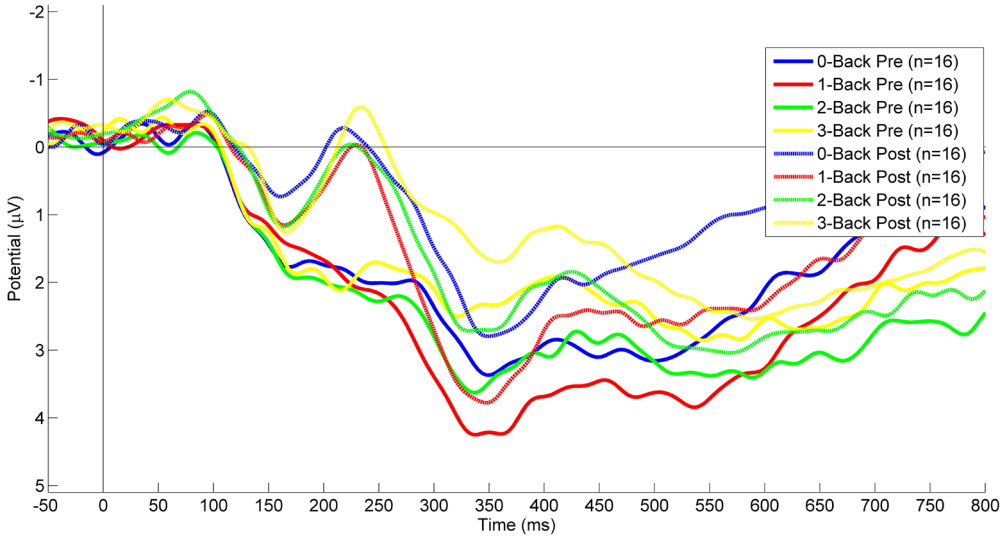
2.1. Grand Averages of the Control Group at FZ



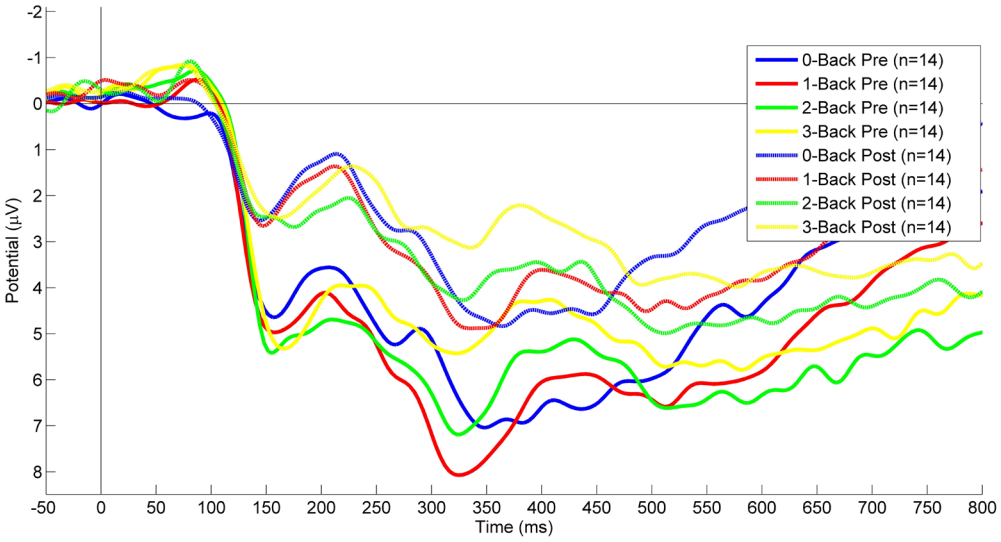
2.2. Grand Averages of the Experimental Group FZ



2.3. Grand Averages of the Control Group at PZ



2.4. Grand Averages of the Experimental Group PZ



6.4. Eidestattliche Erklärung

Ich versichere, dass ich die Diplomarbeit ohne fremde Hilfe und ohne Benutzung anderer als der angegebenen Quellen angefertigt habe, und dass die Arbeit in gleicher oder ähnlicher Form noch keiner anderen Prüfungsbehörde vorgelegen hat. Alle Ausführungen der Arbeit, die wörtlich oder sinngemäß übernommen wurden, sind als solche gekennzeichnet.

Wien, am _____

Unterschrift _____

6.5. Curriculum Vitae

Akademischer Werdegang

06/2013	Förderstipendium der Universität Wien zur Durchführung einer Diplomarbeit
08/2012 – 02/2013	Auslandssemester, Université catholique de Lyon
02/2010	Erste Diplomprüfung
03/2008 – dato	Psychologiestudium Universität Wien
10/2007 – 02/2008	Mathematikstudium, Universität Bonn
06/2006	Gymnasium Köln Nippes, Abschluss Abitur (Note 2.2)

Berufserfahrung

05/2015 – dato	Honorartätigkeit, Dipl. Psych. Scheele-Wirtz, Assistenz bei Therapieanträgen
05/2015 – dato	Geringfügige Beschäftigung, Alexianer Köln GmbH, Sitzwache und Einzelbetreuung, geschützte gerontopsychiatrische Station
07/2014 – 10/2014	Praktikum Charité Berlin, Klinik für Psychiatrie und Psychotherapie, Forschungsgruppe Mind and Brain
04/2012 – 07/2012	Praktikum, Universität Wien, Lehr- und Forschungspraxis der psychologischen Fakultät
10/2006 – 07/2007	Zivildienst, St. Marien Hospital Köln, Pflegehelfer, Ausbildung zum Pflegediensthelfer durch Malteser Hilfsdienst Aachen e.V.

Sonstige Fähigkeiten

Fremdsprachenkenntnisse	Deutsch, Englisch, Französisch
EDV Kenntnisse	Microsoft Office, LATEX, HTML
Programmierkenntnisse	Matlab, Java, PHP, SPSS, E-Prime
Musikinstrumente	Piano, Accordeon, Gitarre