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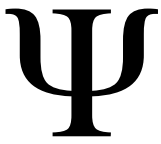

Inhibition and rumination in first-episode depressed individuals: A five-year follow-up study

HOVEDOPPGAVE

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Abstract

Det er økende enighet om at visse kognitive prosesser er kritiske i utvikling og opprettholdelse av Alvorlig Depressiv Lidelse (MDD). En ruminerende kognitiv stil har vært funnet å kunne predikere både alvorlighetsgrad og kronisitet i MDD og har blitt koblet til svakheter i eksekutive funksjoner som inhibisjon og mental fleksibilitet. I denne studien ble dette forholdet undersøkt nærmere gjennom en fem-årsoppfølging (T3) av førstegangsdeprimerte som tidligere hadde blitt testet ved første episode (T1) og ett år senere (T2). Det ble også undersøkt hvorvidt ruminering og/eller inhibisjon kunne predikere tilbakefall på T2 og T3. Tredve forsøkspersoner som møtte kriteriene for første episode av unipolar MDD og 30 kontrollpersoner ble innlemmet i studien på T1. Ruminering og inhibisjon ble målt ved hjelp av Ruminative Response Scale (RRS), Rumination Reflection Questionnaire (RRQ) og Color-Word Interference Test fra Delis-Kaplan Executive Function System. Resultatene viser en signifikant forskjell mellom pasient- og kontrollgruppe bade på ruminering og inhibisjon, som vedvarer over tid. Det ble funnet en sammenheng mellom inhibisjon og ruminering på tvers av gruppene. Resultatene viste imidlertid ingen sammenheng mellom disse faktorene og tilbakefallsrisiko. Det kan slås fast at svekket kognitiv inhibisjon og økt ruminering er karakteristiske trekk ved depresjon over tid.

Abstract

There is growing consensus that certain cognitive processes are critical in the development and maintenance of Major Depressive Disorder (MDD). A ruminative responsive style has been found to predict both severity and chronicity of MDD and has been linked to deficits in executive functions such as inhibition and mental flexibility. The present study investigated this relationship further in a five-year follow up (T3) of depressed individuals, who had previously been tested at first-episode MDD (T1) and at one-year follow up (T2). Whether rumination and/or inhibition could predict relapse at T2 and T3 was also studied. 30 subjects meeting the criteria for first-episode unipolar MDD and 30 controls were included in the study at T1. Rumination and inhibition was measured using the Ruminative Response Scale (RRS), Rumination-Reflection Questionnaire (RRQ) and the Color-Word Interference Test (CWIT) of the Delis-Kaplan Executive Function System. The results show a significant difference between patients and controls on both rumination and cognitive inhibition that persists over time. A correlation between cognitive inhibition and rumination was found across all subjects. However, the results did not show evicence of these factors being related to risk for relapse. Thus, impaired inhibition and increased rumination are long lasting characteristics in MDD.

Inhibiton and rumination in first-episode depressed individuals: A five-year follow-up study

Affecting 350 million people and being the leading cause of disability in the world, Major Depressive Disorder (MDD) is a serious threat to public health and wellbeing (World Health Organization, 2015). Depressive episodes affect behavior, cognition and emotion (Gotlib & Joorman, 2010), with symptoms such as difficulties concentrating, loss of interest and energy, and a pessimistic outlook on life (World Health Organization, 1992). Depressed individuals have more than twice the likelihood to commit suicide compared to the rest of the population (Haddad & Gunn, 2011). MDD also causes significant distress in the family and friends of the depressed individual, with the bond between romantic partners and parent and child being particularly affected (Haddad & Gunn, 2011). Findings from several studies suggest that the age of onset for depression is declining (Dalgård & Bøen, 2008), making identification and implementation of preventative measures all the more important for future generations.

In Norway, treatment of depression costs an estimated 1.5 billion kroner each year (Dalgård & Bøen, 2008). The lifetime prevalence of depression in Oslo has been found to be 17.8 percent, making it the most commonly occurring mental illness in Norway alongside alcohol dependency/abuse (Kringlen, Torgersen & Cramer, 2001). American epidemiological studies have found several factors that increase the likelihood of suffering from depression, including being female, having lower income, being unmarried and living in urban areas (Friedman & Anderson, 2009). MDD is frequently comorbid with other psychiatric illnesses as well as physical conditions. In a worldwide study on chronic illness and depression, Moussavi and colleagues (2007)

found that depression was much more prevalent in individuals with a chronic physical condition, such as diabetes. They also reported that people suffering from depression or depression in combination with a chronic illness had significantly poorer health compared to patients suffering only from physical conditions. People suffering from asthma, arthritis, diabetes, and cardiovascular disease have been found to be more likely to suffer from depression than healthy individuals (Chapman, Perry & Strine, 2005). Suffering from depression in addition to a chronic physical condition often has a negative impact on the treatment and course of the chronic disease, making screening for and treatment of depressive symptoms an important concern (Chapman et al., 2005). Kessler and associates (2007) found that 72 percent of people with MDD additionally met the criteria for one or more DSM-IV disorders. Anxiety disorders were the most frequently co-occurring with MDD, found in over 59 percent of cases, followed by impulse control disorders (30%) and substance abuse (24%) (Kessler et al., 2007).

A troubling aspect of depression, which contributes to the large personal and societal costs of the illness, is the high probability for reoccurrence. According to epidemiological data, more than 60 percent of people who have their first major depressive episode will experience another one, while a person with three previous episodes has a 90 percent chance of a new episode occurring (APA, 2000). Once a person has been in treatment for a depressive episode, they will in general spend 20 percent of their remaining life in a depressed state (Coyne, 2000). The likelihood of developing new depressive episodes appears to be greater in individuals who do not experience a complete recovery from depressive symptoms (Gopinath, Katon, Russo, & Ludman, 2007; Lin et al., 1998). Environmental stressors seem to play a bigger part as a precipitating factor in an individual's first and second depressive episode, while being of

less significance for the development of subsequent periods of depression (APA, 2000). The severity of the first depressive episode also predicts recurrence (APA, 2000). When studying previously depressed people perceived by their primary physician as being at risk for subsequent episodes, Gopinath and colleagues (2007) found low self-efficacy, negative perception of own health and low adherence to medication in the past month to be the strongest predictors of relapse. Maj, Veltro, Pirozzi, Lobrace and Magliano (1992) found that the strongest predictors for relapse in a five-year period were the number of previous episodes, underlying and chronic minor affective disorders, and family history of affective disorders respectively. They also found a pattern of increasing severity of subsequent episodes of depression (Maj et al., 1992). Patten (2013) points out that little research has been done on recurrence in depression, which makes evidence-based treatment difficult in long-running therapy.

Several researchers and theorists have tried to explain why people get depressed, and what makes some people more vulnerable to experience multiple depressive episodes over their lifespan. According to Beck's cognitive model of depression, our pre-existing schemas shape our view of the environment and direct our attention to stimuli that are congruent with these schemas (Teasdale, 1988). Thus, a depressed person tends to focus on negative aspects of the world because of the negative nature of her mental representations, which contain themes such as loss, hopelessness and despair (Gotlib & Joormann, 2010). These schemas will also be present when the person is not currently in an episode of depression, creating a persistent vulnerability for recurrence (Gotlib & Joormann, 2010). Within the framework of a cognitive model of depression, biased information processing and recall should also lead to difficulties in emotional regulation, as cognition would steer attention towards negative stimuli and memories.

Little evidence has been found in support of the hypothesis that depression affects all aspects of information processing, but there are strong indications that depressed individuals have deficits and biases in specific areas of cognition (Gotlib & Joormann, 2010). The research findings on the cognitive characteristics of depression will be presented in the following.

Neuropsychology, Cognition and Depression

Cognition is a broad term, capturing many different aspects of human functioning, and it is widely studied in relation to both normal functioning and mental disorders. It can be conceptualized as the internal processes that are involved when we make sense of the environment surrounding us and when deciding what action is appropriate in relation to the environment (Eysenck & Keane, 2015). Cognitive neuroscience is highly related to cognitive psychology, but adds to the study of human behavior by also studying the brain when people perform different cognitive tasks (Eysenck & Keane, 2015). There is general consensus that cognitive processes are closely linked to the development and effective treatment of mental disorders. Cognition and cognitive neuropsychology has been studied in relation to affective disorders, including depression, and in the following we will present some of the relevant research findings in this field of study.

Neuropsychological structures and depression.

A great amount of research has been conducted to explain the neurobiology of depression and other mood disorders. In 1937, Papez established the importance of the "system of emotion" in the brain, which includes major pathways in the limbic system including cingulate gyrus, hippocampus, hypothalamus and anterior thalamic nuclei. The research has been expanded to include other important areas, particularly the

prefrontal cortex (PFC), after the emergence of neuroimaging techniques (Beyer & Krishnan, 2002; Drevets, 2000; Videbech, 1997). These areas and their stability or malfunction is considered central to the pathophysiology of depression (Palazidou, 2012).

The PFC is the most widely studied brain structure in relation to depression, along with amygdala and hippocampus (Palazidou, 2012). Studies have shown a reduction in brain volume in depressed patients, with large reductions in areas of PFC, hippocampus, putamen and caudate (Beyer & Krishnan, 2002; Campbell, Marriott, Nahmias, & MacQueen, 2014; Hajek, Kozeny, Kopecek, Alda, & Hoschl, 2008; Sheline, Gado, & Kraemer, 2003; Videbech & Ravnkilde, 2004). Research also shows abnormalities in blood flow and glucose metabolism in several prefrontal cortical and limbic structures assumed to be involved in emotional processing (Drevets, 1998). This includes abnormal activation in the amygdala, which correlates with the severity of depression (Drevets, Bogers, & Raichle, 2002; MacDonald, Cohen, Stenger, & Carter, 2000). The PFC has rich connections to subcortical structures, like basal ganglia, but also sends and receives projections from nearly all the sensory and motoric cortical areas, which makes it well placed and connected in order to integrate higher-level representation from, and exert control over, different neurological systems in the brain (Gilbert & Burgess, 2008). The PFC seems to play an important role in the coordination of emotion and cognition by controlling limbic impulses and making emotional reactions appropriate to the situation (Ardila, 2008).

Hippocampus plays a central role in learning and memory (Squire & Knowlton, 2000), and a dysfunction in the hippocampus may be responsible for inappropriate context-dependent emotional responses (Fanselow, 2000). Hippocampus is closely

linked to the hypothalamus (Fanselow, 2000; Squire & Knowlton, 2000), and demonstrates a high capacity for neuroplasticity (Eriksson et al., 1998.). Memory impairment is seen in both first time depressed patients as well as patients with multiple episodes, but only in the latter group is the volume of hippocampus reduced (MacQueen et al., 2003; Videbach & Ravnkilde, 2004). It has been suggested that this structural abnormality may be a characteristic trait for recurrent depression (Frodl et al., 2004; Neumeister et al., 2005).

Based on the great amount of research done on this area, Palazidou (2012) advocates that depressive disorder has a multifactorial aetiopathogeneses, including a genetic diathesis, and with stress playing a major role. This includes abnormalities in pathophysiological mechanisms, such as reduced activity in the monoamine neurotransmission, a reduction in brain neurotrophins, and hyperactivity in the hypothalamus-pituitary-adrenal (HPA) axis. These are connected with functional and structural abnormalities, affecting the system balance. The hypothesis is that PFC, which becomes functionally and structurally impaired, is unable to regulate the overactivity within the cortical and limbic regions. This may result in the clinical manifestation of the depressive syndrome. Antidepressant drugs may reverse some of the structural changes in the hippocampus, and increase monoaminergic neurotransmission, and may have a beneficial modulatory effect on the disrupted neurobiological and neurostructural functions (Bunny & Davis, 1965, as cited in Hirschfeld, 2000; Schildkraut, 1965, as cited in Hirschfeld, 2000). Research on the neurobiology of mood disorders is important, as researchers seem to agree that the PFC is the neurobiological system that is closest related to executive functioning, though the

precise nature of this connection is still not completely understood (Hsu, Novick & Jaeggi, 2014).

In the present study, we investigate the role of rumination and inhibition in MDD, processes thought to be rooted in executive functioning. The above-presented evidence of unique variations in the brains of patients suffering from depression, can be seen in association with the cognitive impairments in MDD that will be explored in the following.

Cognitive functioning and depression.

Studies on how depressive symptoms affect people's performance on cognitive tasks, have found dysfunctions in attention focus, memory, and aspects of executive functioning. When investigating attention focus in depressed individuals, a review by Gotlib and Joorman (2010) found that when negative information is presented over an extended period of time, depressed people focus on it longer than healthy controls, and have problems disengaging from the information even though it is irrelevant to the task they are performing. In studies where people were asked to ignore information that was either positive or negative, the depressed participants were found to have difficulties not paying attention to negative information (Joormann, 2004). Joormann and Gotlib (2008) found that depressed individuals are less efficient in removing no-longer relevant negative information from working memory compared to people who have never been depressed. Gotlib and Joormann (2010) hypothesize that this extended attention focus on mood-congruent information can exacerbate depressed states and be an obstacle to recovery for people suffering from depression. In their meta-analysis of studies using the Cambridge Neuropsychological Test Automated Battery (CANTAB) on currently and previously depressed individuals, Rock, Poiser, Riedel and Blackwell (2014) found

moderate deficits on attention focus tasks in both depressed and remitted patients. It is worth to note that whereas many studies have demonstrated that people suffering from anxiety automatically focus on negative stimuli in the environment, no such assumptions can be made about depressed people based on the research available today (Gotlib & Joormann, 2010). Mathews and Macleod (2005) present several research findings that show no automatic attentional bias towards negative stimuli in depressed patients when the stimuli is presented too briefly to be processed consciously.

Memory is another part of cognition that has been demonstrated to be affected by a depressed state. Indeed, in their meta-analysis of mood-congruent memory, Matt, Vázquez and Campbell (1992) found that clinically depressed people are prone to remembering negative autobiographical information while non-depressed individuals have a bias towards remembering positive memories. Another characteristic found in memory functioning in depressed people, is over-general remembering. A substantial amount of research has found that when asked to recall specific events or memories, depressed people to a larger extent than healthy peers come up with generic information from long-term memory instead (Williams et al., 2007). This lack in specificity of recall is associated with poor problem-solving skills, and also with delayed recovery from depressive episodes (Williams et al., 2007). When induced to recall pleasant autobiographical information, non-depressed individuals have been found to recover from a sad mood (Joormann & Siemer, 2004). People who are dysphoric, however, do not experience this effect, a fact that suggests that people who are depressed are unable to use positive memories as a way of emotional regulation (Gotlib & Joormann, 2010). In the above-mentioned meta-study using CANTAB, currently depressed people had moderate impairments in memory tasks, while remitted individuals showed small

impairments (Rock et al., 2014).

Executive functioning can be conceptualized as a collective term for higher-level cognitive processes that facilitate new ways of behaving and help guide us in approaching new and unfamiliar situations (Gilbert & Burgess, 2008). These processes are separate, but closely related, and seen as necessary for the conduction of meaningful, goal-oriented behavior, and in planning the future or switching from one task or activity to another and when resisting temptations (Gilbert & Burgess, 2008; Wagner, Alloy & Abramson, 2015). It includes inhibition, mental flexibility, working memory, initiation of action, and the ability to filtrate interference and predict the consequences of our behavior among others (Ardila, 2008; Gilberg & Burgess, 2008). It has been suggested by many researchers that depression could be associated with a reduced capacity in executive processes, and the scientific evidence does support this idea to a certain extent (Gotlib & Joormann, 2010). Rock and colleagues (2014), found moderate deficits on executive functions in their meta-analysis on currently depressed patients, using CANTAB. This includes deficits on visual planning, reasoning and impulsivity (Stockings of Cambridge Test), working memory (Spatial Working Memory Test, Spatial Span Test) and cognitive flexibility (Intra-Extra Dimensional Set Shift). Moderate deficits in executive function were also found when analyzing studies on remitted patients (Rock et al, 2014). In the review by Gotlib and Joorman (2010), they found evidence for impaired inhibition for both emotionally neutral tasks, and emotionally laden tasks, in depressed individuals. Other reviews on the matter have added substantial support suggesting that depressed individuals show impairment in executive functions, especially inhibition and mental flexibility (Austin, Mitchell & Goodwin, 2001; Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, & Lönnqvist,

2008; Hammar & Årdal, 2009). This impairment in inhibition is seen in both first episode depressed indivduals (Schmid & Hammar, 2013b), as well as in longitudinal studies of recurrent depression (Schmid & Hammar, 2013a).

In summary, there are several biases in cognition and executive function that characterize depression. Once made aware of mood-congruent stimuli, depressed people tend to focus on this for an extensive amount of time and are also less able to stop thinking about this information when it is no longer relevant to the task they are performing. There is also evidence that people suffering from depression have difficulties recalling specific autobiographical memories and that they do not experience heightened mood when reminded of positive memories. When investigating executive functions, researchers have found impairments across several domains, demonstrating a profound impairment in abilities to inhibit both emotionally neutral, and emotionally laden information, as well as deficits in mental flexibility.

Rumination

The response styles theory (RST) presented by Susan Nolen-Hoeksema (1991) suggests that the way individuals respond to their depressed mood will affect the duration of this mood. A ruminative response style is conceptualized as a repeated focus on the depressed mood, the symptoms of depression and their meaning, as well as the cause and the consequences of the depression. People who are prone to this way of thinking, Nolen-Hoeksema (1991) suggests, will suffer from prolonged periods of depressed mood. An example of a ruminative response can for instance be to isolate oneself from one's surroundings in order to think about symptoms and the origin of these. An important feature of the RST is that it is the style of the negative cognition, and not the content, that is important (Nolen-Hoeksema, 1991). It is also important to

distinguish ruminative responses from problem solving. In active problem solving, the individual engages in different behaviors with the intention to solve a problem related to their depression. People who ruminate, however, do not take action, but spend their time only thinking about how they feel (Nolen-Hoeksema, 1991; Nolen-Hoeksema, Morrow & Fredrickson, 1993). It has been suggested that it is the focus on the individual's emotion and current state of feeling depressed that is most important, mainly due to the fact that most depressions are not related to specific events, but there are still symptoms present, which gives the individual something to ruminate about. Further, when depressed people focus on their emotional state they are focusing on a negative aspect, which again may cause their thinking to be affected by their mood (Nolen-Hoeksema, 1991). Studies have shown that people who score higher on measures of self-focus also tend to take less action, to ruminate more and to be sadder (Wood, Saltzberg, Neale, Stone & Rachmiel, 1990). Further, research has supported the hypothesis that response styles are consistent over time, as shown by Nolen-Hoeksema and colleagues (1993). More recent reviews have found that a ruminative response style appears to predict the onset of depression to a larger degree than the length of the depressive episode (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008). It has further been shown to maintain depressed mood (Donaldson & Lam, 2004), predict the severity of a depressive episode (Just & Alloy, 1997; Lam, Smith, Checkley, Rijsdijk & Sham, 2003) and the chronicity of a depressive disorder (Kuehner & Weber, 1999). Rumination has also been linked to low social support (Nolen-Hoeksema, 1991) and comorbid anxiety and depression (Nolen-Hoeksema, 2000). Finally, there is emerging evidence that rumination is related to an increased relapse risk in individuals who experience depressive mood (Huffziger,

Reinhard & Kuehner, 2009; Michalak, Hölz & Teismann, 2011; Nolen-Hoeksema, 2000).

The opposite of a ruminative response style is distraction, conceptualized as a meaningful attention switch from the depressive symptoms to a neutral or positive activity (Nolen-Hoeksema, 1991). An important feature of the RST is that whether people engage in either rumination or distraction as a response to depressed mood should strongly predict the duration of their negative mood, which according to the above-mentioned studies it appears to do.

Researchers often separate between trait rumination, a stable disposition to ruminate, and state rumination, which is the act of ruminating after a single stressful event (Just & Alloy, 1997; Key, Campbell, Bacon & Gerin, 2008). In studies, researchers often study trait or state rumination by either examining the cognitive correlates of scores on self-report measures of trait rumination (e.g. Altamirano, Miyake & Whitmer, 2010; Wagner et al., 2015; Whitmer and Banich, 2007; Whitmer and Gotlib 2013), or by investigating the effect of an experimentally induced state of rumination on executive function performance tasks (e.g., Phillipot & Brutoux, 2008; Watkins and Brown 2002; Whitmer & Gotlib, 2012). It seems that trait rumination is a quite stable tendency, and that there are individual differences in this trait, which are consistent over time and across levels of depressive symptoms and the content of ruminative thinking (Mandell, Siegle, Shutt, Feldmiller & Thase, 2014; Wagner et al., 2015).

Originally, rumination was seen as a unitary concept, but research has subsequently identified two different forms of rumination: brooding and reflection (Mor, Hertel, Ngo, Shachar & Redak, 2014). It is the brooding aspect, a passive, preservative, and judgmental focus, of rumination that is seen as maladaptive.

Reflection on the other hand is contemplative, intentional wondering about one's current state in order to solve a problem or a challenge (Mor et al., 2014), much like the problem solving mentioned previously. Many theorists have proposed that hopelessness regarding the future and negative evaluations of the self are core features of depressive disorders, and it seems that rumination also contributes to this, according to content analysis of rumination (Nolen-Hoeksema, 2000).

Rumination, Inhibition and Mental Flexibility

There is increasing evidence and growing consensus that there are cognitive processes that appear to be critical in the onset and maintenance of depressive disorders, such as the cognitive biases mentioned previously, as well as deficits in executive functioning (Joormann & Quinn, 2014). Much research has been conducted in order to understand the relationship between trait rumination and clinical depression. From the research mentioned above it seems that a ruminative response style affects the individual in many different ways, and a growing body of literature suggests that it also affects cognition and neuropsychological functioning. Specifically, the concept of executive functioning has been linked to rumination (Joormann & Quinn, 2014). There is increasing evidence that rumination is connected to deficits in executive functioning, specifically when processing emotionally laden information (Wagner et al., 2015).

In the relationship between deficits in executive functioning and a tendency to ruminate, it is particularly the concepts of inhibition and mental flexibility that have been studied. Inhibition can be defined as the ability to intentionally prevent or overrule the tendency to use dominant, automatic responses when its either not necessary or when they are no longer relevant (Gilbert & Burgess, 2008; Philippot & Brutoux, 2008). Mental flexibility can be defined as the ability to change between different stimulus-

response sets, or between tasks or operations (Gilbert & Burgess, 2008; Philippot & Brutoux, 2008). Switching is related to mental flexibility, conceptualized as the changing of the attention away from current representation and over to something new when the environmental conditions are changing (Whitmer & Gotlib, 2012). In the next section, we present studies that have researched the effect of trait and state rumination on performance on inhibition and mental flexibility tasks.

When examining state rumination and executive functioning, the subjects are usually presented with a manipulation that is meant to induce either rumination or distraction, before performing tasks involving inhibition or mental flexibility (see Morrow & Nolen-Hoeksema, 1990, for the most common rumination/distraction manipulation). A study by Whitmer and Gotlib (2012) predicted that induced rumination in depressed subjects would show more errors in inhibition and greater switching costs than non-depressed controls who were induced to ruminate and depressed individuals who were induced to distraction. Their results only partly confirmed their predictions, as they were not able to demonstrate that induced rumination impaired performance on inhibition tasks in depressed individuals. They did however demonstrate that induced rumination affected switching costs in the same group. These results suggest that deficits in task switching are dependent on a ruminative response style (Whitmer & Gotlib, 2012). Another study investigated whether it was only in the dysphoric individuals who were induced to ruminate one would see draining of executive resources (Philippot & Brutoux, 2008). In contrast to Whitmer and Gotlib's (2012) study, their results showed a relationship between deficits in inhibition and depressive rumination. The dysphoric subjects who were induced to ruminate made more errors, displaying a deficit in inhibition, and they also seemed to

be less flexible when they were induced to ruminate compared to non-dysphoric individuals (Philippot & Brutoux, 2008). They further suggest that flexibility might be fundamentally impaired in depressed individuals and that the induced rumination appears to exacerbate this tendency. This study replicates the finding of Watkins and Brown's study from 2002. They found that depressed individuals induced to ruminate made more errors on a random number-generation task meant to measure inhibition, compared to non-depressed subjects induced to rumination, and depressed and non-depressed people induced to distraction.

When examining trait rumination, the subjects are measured on one or several self-report questionnaires before performing the executive function tasks. The self-report questionnaires measure the individual level of current rumination, or a general tendency to ruminate. Trait rumination seems to be qualitatively distinct from rumination, and holds a particular pattern of association with performance on emotionally neutral cognitive tasks that also seem to affect the individual's ability to perform on inhibition and inhibition/switching tasks. Davis and Nolen-Hoeksema (2000) investigated whether trait ruminators would differ in their performance on the Wisconsin Card Sorting Test, a test thought to measure mental flexibility, from non-ruminators. Their results show that the trait ruminators made more errors of perseveration than the non-ruminators, adding to the evidence that rumination might be characterized by an inflexible cognitive style (Davis & Nolen-Hoeksema, 2000). Owens and Derakshan (2013) found that high ruminators performed poorer on a switching task that required them to switch between the former task and over to the new and relevant one. The high ruminators showed more interference from the currently irrelevant task and poorer filtering, which made them commit more errors compared to low ruminators

(Owens & Derakshan, 2013). Thus, their findings also add to research linking mental flexibility to impaired inhibition in rumination. They further argue that rumination in itself contributes to some of the cognitive deficits found in depression, and that impaired inhibition may cause a continuation of the ruminative pattern, even if the individual understands its maladaptive effect (Owen & Derakshan, 2013). Whitmer and Banich (2007) found that higher tendency to depressively ruminate is associated with a decreased ability to inhibit a previously relevant task set. In contrast, they found that when controlling for inhibition, set-switching cost was no longer predictive of ruminative tendencies, suggesting that executive dysfunction is more closely linked to difficulties in inhibition than to difficulties in switching task sets.

Zetche, D'Avanzato and Joormann (2012) found that rumination was related to having difficulties removing negative information that was no longer relevant from working memory. They conclude by suggesting that rumination might be driven by the inability to disengage from negative self-reflective thoughts and that high and low ruminators differ in the perseverance of rumination, rather than the initiation of these thoughts. They further suggest that it is not the immediate ruminating response that is maladaptive, but that it may become maladaptive if it persists over time and hinders the individual from engaging in activities that could help them recover (Zetche et al., 2012). Joormann (2006) also investigated whether differences in ruminative responses relate to deficits in inhibition of irrelevant emotional material in working memory. He found that those high in rumination also showed impaired inhibition in relation to emotionally laden information.

It has been proposed that a ruminative response to depression will increase the effect of negative expectation, and that its negative effect is exercised through making

negative cognition more available, specifically the cognitive biases that are related to depression (Mor et al., 2014; Nolen-Hoeksema, 1991). One of these biases is the interpretation bias, which is assumed to maintain negative emotional states and self-evaluations and also to enhance a negative memory bias (Mor et al., 2014). Mor and associates (2014) found that rumination was associated with an interpretation bias. They found that the subjects who had a tendency to ruminate interpreted ambiguous material as negative, and especially if this material was related to the content of their ruminative thoughts. Another study found associations between trait rumination and negative attentional bias in depressed individuals (Donaldson, Lam & Mathews, 2007). Rumination has also been related to an over-general style of retrieval of autobiographic memories (Park, Goodyer & Teasdale, 2004; Philippot & Brutoux, 2008). For instance, one study found that modification of the memory bias and training of dysphoric individuals in order to be more specific and less generalizing in their thinking style lead to a decrease in depressive symptoms (Watkins, Baeyens & Read, 2009).

In summary, trait rumination in depressive disorders appears to affect several aspects of executive functioning. While some studies demonstrated support for impaired mental flexibility (Davis & Nolen-Hoeksema, 2000; Owen & Derakshan, 2013; Whitmer & Gotlib, 2012), others find support for impaired inhibition (Watkins & Brown, 2002; Whitmer & Banich, 2007). Despite these disagreeing findings, the cumulative data on the matter suggests that both trait and state rumination impair performance on either inhibition or mental flexibility/switching tasks, or both, across clinical and random samples. Other areas of cognitive functioning, including interpretation, memory retrieval, and attention are also affected.

Background for the Current Study

As the results from the research presented above suggests, it is assumed that deficits in inhibition and mental flexibility may affect the ability to suppress and inhibit negative automatic thoughts and switch from ruminative patterns of thinking to new and more positive thoughts. This might provide the link between the findings that depressed individuals who display a ruminative response style have a greater risk of multiple depressive episodes and the findings that depression is characterized by cognitive deficits (Joormann & Quinn, 2014). This in the sense that difficulties inhibiting negative material that is no longer relevant may provide an explanation as to why depressed individuals respond to negative thoughts and moods with recurring and unintentional negative cognition. A lot of research has been conducted over the last few years in order to gain evidence for this possible link. However, as mentioned previously, there has been little research on recurrence in depression. Many people who experience a depressive episode once in their lifetime will also experience another episode later on. Given the results from research on rumination and deficits in inhibition and mental flexibility, it is possible that this will affect the recurrence of depressive episodes. However, there have been few studies investigating this relationship. We argue that it is important to study the link between performance on tasks on executive functioning and self-reported rumination in relation to recurrent depression. This will be of clinical relevance, as it will help guide which interventions to choose in treatment of recurrent depression, and possibly preventing future depressive episodes. Longitudinal studies further give a unique insight into the course of depression as an illness, giving information about relapse and recurrence and possible changes in cognitive functioning that cross-sectional studies cannot provide. As far as we are aware, there have never

previously been studies that investigate the relationship between cognitive functioning in first episode major depressive disorder and a five-year follow up on these patients in regard to cognitive functioning and rumination.

The aim of the present study is to investigate the relationship between inhibition and trait rumination in a five-year follow up of depressed individuals, who have previously been tested at first episode MDD and at one-year follow up.

We predict that the depressed individuals will perform poorer on measures of cognitive inhibition, and therefore still show prevailed cognitive inhibition at T3 (H1).

We further predict that the depressed individuals will report higher levels of rumination compared to the control subjects (H2).

We predict a correlation between cognitive inhibition and high self-reported rumination (H3).

Finally, we predict that cognitive inhibition will be related to a risk of relapse (H4a), that high self-reported rumination will be related to a risk of relapse (H4b) and that both these factors will be related to a risk of relapse (H4c).

Method

Clinical and Demographic Data

Patient group (PG). The subjects included in the study were tested at three points in time: in the acute phase of illness (T1), one year after inclusion (T2), and five years after inclusion (T3). At T1, 30 patients (16 males and 14 females) were included in the study, as they met the DSM-IV criteria for a unipolar first-episode MDD diagnoses, using the MINI - International Psychiatric Structural Interview (Lecrubier et al., 1997). The structural rating scale Montgomery Åsberg Depression Rating Scale

(MADRS) (Montgomery & Åsberg, 1979) was administered to measure depression severity at all test times.

The patients were recruited to the study through cooperation with primary physicians and psychologists, who in turn deemed their patient suitable, based on the inclusion and exclusion criteria. Inclusion criteria for the patient group were that the patient was diagnosed with first-episode MDD, with a minimum score of 20 on MADRS, indicating moderate to severe depression. Patients were excluded from the study if they reported having experienced former episodes of severe symptoms of depression, and if they had been diagnosed with depression and/or had received treatment for depression earlier in life. Patients with psychosis, known brain damage, severe somatic disorders, alcohol and/or substance abuse, and patients who had been treated with electroconvulsive therapy (ECT) were excluded from the study. The patients were outpatients receiving either medical treatment (13.3 %), psychological treatment (33 %) or both (33.3 %) for the first time, or no treatment at all (23.3 %).

Control group (CG). A control group (N = 30) was included at T1, with the subjects individually matched to the patient group on the basis of gender, age and years of education (within a ± 2 year limit). The CG was recruited from the University of Bergen and through acquaintances of employees of the Department of Biological and Medical Psychology of the University of Bergen. The prospective subjects of the control sample were interviewed to survey their history of mental or somatic disorder and were excluded if they reported a history of any mental disorder, brain damage and/or alcohol or substance abuse.

All subjects were asked to participate in follow-up assessments. At T2, data from two patients are missing due to dropout (N = 28). The mean score on MADRS

reported at T2 demonstrated that the patient group had minimal symptoms of depression. At T3, ten of the patients were unable to participate in the study (N = 23), and the ten individually matched control subjects were therefore not included. Mean score on MADRS was showing a normal to mild degree of symptoms of depression in the patient group.

Table 1

Descriptive data for the patient group and the control group at T1, T2 and T3.

	Patient Group		Control Group	
	M	*SD*	*M*	*SD*
T1				
n	30		30	
Age	26.20	5.94	26.17	5.68
Education	13.97	1.71	14.03	1.65
Males/females	16/14		16/14	
IQ**	118.53	8.12	120.97	8.23
MADRS score	24.77	3.77	*	*
T2				
n	28		28	
Age	26.93	5.33	26.79	5.26
Education	14.29	1.76	14.68	1.63
Males/females	14/14		15/13	
IQ**	118.43	8.40	121.58	8.18
MADRS score	9.96	6.01	*	*
T3				
n	23		22	
Age	30.35	5.74	30.00	5.98
Education	15.35	2.35	16.55	1.92
Males/females	11/12		10/12	
IQ**	119.05	8.45	119.58	8.48
MADRS score	8.87	8.13	*	*

* Control group, no history of illness.
** IQ measured at inclusion, T1.

Patient subgroups (PSG). At T2 the patient group was interview regarding the course of their symptoms, and further categorized into different subgroups based on this retrospective interview (Table 2). This categorization resulted in a relapse group (RG) (N = 11), a no-relapse group (NRG) (N = 5), and a third no-change group (NCG) (N = 5), that experienced little change in symptomology since inclusion. The NCG reported a mean MADRS score of 18, indicating mild to moderate depression requiring treatment. The RLG and NRG reported a MADRS mean of < 10, showing low depression severity.

At T3, a psychologist re-interviewed the PG. Thirteen subjects were categorized into the Relapse Group (N = 17) and five of the subjects were placed in the no-relapse group (N = 6). None of the subjects in the patient group reported no change since T2 (N = 0). MADRS score was reported to be < 10, showing a normal to moderate depression severity also at this point in time.

Relapse was defined as the subject returning to a fully symptomatic state of depression after a minimum three-week period with none or minimal levels of symptoms (Frank et al., 1991, as cited in Schmid & Hammar, 2013a). To fulfill the criteria of relapse, the subject had to report the relapse period as having lasted a minimum of two weeks. In the present study, difficulties performing at an optimal level in areas such as school, work or socials setting, was added to the definition of relapse.

Table 2

Descriptive data for the relapse group (RLG), the no-relapse group (NRG), the no change group (NCG) and the control group (CG) at T1, T2 and T3.

	RLG		NRG		NCG		CG	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
T1								
n	11		12		5		30	
Age	25.09	6.47	25.25	4.09	29.60	4.88	26.17	5.68
Education	14.27	1.62	14.25	1.96	13.00	1.41	14.03	1.65
Males/females	3/8		10/2		1/4		16/14	
IQ**	115.46	6.53	119.08	9.65	123.40	7.57	120.97	8.23
MADRS score	24.55	4.39	23.83	3.01	28.00	3.16	*	*
T2								
n	11		12		5		28	
Age	26.00	6.43	26.25	4.00	30.60	4.88	26.96	5.26
Education	14.64	1.69	14.42	1.93	13.20	1.30	14.68	1.63
Males/females	3/8		10/2		1/4		15/13	
IQ**	115.46	6.53	119.03	9.65	123.40	7.57	120.97	8.23
MADRS score	9.09	5.19	7.42	3.53	18	6.33	*	*
T3								
n	17		6		0		22	
Age	28.88	4.50	34.50	7.23	*	*	30.00	6.32
Education	15.06	2.41	16.17	2.14	*	*	16.55	1.92
Males/females	7/10		4/2		*	*	10/12	
IQ**	118.00	8.49	121.83	8.42	*	*	119.58	8.48
MADRS score	10.88	8.42	3.17	3.43	*	*	*	*
Number of relapse	2.27	3.25	.00	.00	*	*	*	*

* Control group, no history of illness
** IQ measured at inclusion, T1.

Procedure and Neuropsychological Assessment

The neuropsychological assessment was conducted at the Institute of Biological and Medical Psychology, University of Bergen, Norway. A trained senior test technician administered the testing. The test technician was not blinded to group membership for the patient and control subjects due to recruitment procedures. The neuropsychological tests were given to all patients in the same sequence. The tests were part of a larger test battery (the Delis-Kaplan Executive Function System), including IQ measurements (WASI) and other standardized and experimental tests. The procedure and tests used was the same at all test times.

D-KEFS Color-Word Interference Test (CWIT*).* The CWIT is a modified version of the Stroop procedure from 1935, and is meant to measure the subject's ability to inhibit an overlearned verbal response and comprises four conditions (Stroop, 1935). Condition 1: Color naming (C), condition 2: Word Reading (W), condition 3: Inhibition (CW) and condition 4: Inhibition/switching (IS). Conditions 1 and 2 are baseline conditions measuring key component skills of higher-level tasks, by naming color patches and reading color-words printed in black ink. Errors commited on one or both of these conditions could be related to fundamental skills, and/or perseverative tendencies. Errors in the latter occur when the subject is unable to inhibit the production of a previous response. Condition 3 is the traditional Stroop task, where the subjects must inhibit reading the words in order to name the dissonant ink color that the word is printed in. In condition 4 the subject is asked to switch back and forth between naming the dissonant ink colors and reading the words. This condition is meant to measure both inhibition and cognitive flexibility, as performance on this task requires adequate

naming speed, reading speed, verbal inhibition and cognitive flexibility. For further reference, Cognitive inhibition is often used as a collective term for the CWIT.

Rating Scales on Depression and Rumination

Montgomery Åsberg Depression Rating Scale (MADRS). The MADRS is a depression rating scale developed by Montgomery and Åsberg (1979). The MADRS is a rather short rating scale that is meant to be easily applied in clinical settings. All items included in the scale are related to core symptoms of depressive illness, and the scale is highly sensitive to change in severity of depressive symptoms. It consists of 10 items, where item 1 is based on the clinician's observation of the patient, whether he or she looks visibly sad or depressed. All items are rated on a scale from 0 (normal) to 6 (deep symptom severity), where 60 is the highest score possible to obtain.

Ruminative Response Scale (RRS). The RRS (Treynor, Gonzales & Nolen-Hoeksema, 2003) is the most commonly used measurement investigating rumination in depression. It is part of the Response Styles Questionnaire (RSQ), which is designed to screen for common responses to depressed mood. The RRS consists of 24 items, which upon analysis have been found to separate into two subsets of factors, namely brooding and reflection (Schoofs, Hermans & Raes, 2010). In the present study, the full-scale RRS has been used without separating between brooding and reflection. Each item is rated on a Likert scale from 1-4, where 1 is *almost never*, and 4 is *almost always*.

Rumination-Reflection Questionnaire (RRQ). The RRQ was developed by Trapnell and Campbell (1999) and is intended to separately measure anxious self-reflection and curious introspection. Trapnell and Campbell (1999) define reflection as a "self-attentiveness motivated by curiosity or epistemic interest in the self" (p. 297), while rumination is construed as "self-attentiveness motivated by perceived threats,

losses, or injustices to the self" (p. 297). Rumination and reflection appear to be essentially independent tendencies, as the correlation between these two factors was minimal (Trapnell & Campbell, 1999). The RRQ consists of 24 items, or statements, concerning rumination and reflection. The participants are asked to report how much they agree with each statement, using a scale from 1 to 5, where 1 represents *strongly disagree,* and 5 represents *strongly agree.* Items 1 to 12 are statements describing ruminative tendencies, while 13 to 24 hold reflective values. In the present study, only questions 1-12 have been included in the analysis as a specific measurement on rumination.

Descriptive data for the patient group and control group on the RRS and RRQ are presented in Table 3.

Table 3

Descriptive data for the patient group and control group on the RRS and RRQ.

	Patient Group		Control group	
	M	*SD*	*M*	*SD*
T1				
n RRS	14		0	
RRS total score	58.07	10.85	*	*
n RRQ	6		25	
RRQ-rumination score	46.00	5.62	33.72	6.55
T2				
n RRS	27		0	
RRS total score	45.15	11.95	*	*
n RRQ	25		28	
RRQ-rumination score	44.76	8.58	30.54	6.98
T3				
n RRS	23		22	
RRS total score	48.43	13.31	30.77	8.02
n RRQ	23		22	
RRQ-rumination score	48.43	12.57	32.41	9.50

* No subjects in the control group filled out the RRS at T1 and T2.

Informed consent was obtained from all participants at T1. The study was performed in accordance with the Helsinki Declaration of the World Medical Association Assembly. The Regional Committee for Medical Research Ethics and The Norwegian Data Protection Authority approved this study.

Results

Data Scoring and Analysis

Statistical analysis of the data was conducted using the Statistical Package for the Social Sciences (SPSS) version 23.0. An alpha level of $< .05$ was used for the statistical tests comparing the patient and control groups. To avoid making a type II error, we redid the analysis conducted on the PSG, using an alpha level of $< .10$. There

were no changes in the significant findings, thus all the following results are presented with an alpha level of < .05. The data was checked for outliers using boxplots. No outliers were removed however, as they were judged to be of clinical value. The data analyses were conducted in two main parts. First, preliminary analysis between the patient group and the control group was conducted to explore whether they matched on demographic variables and whether they differed on CWIT from T1 to T3. Secondly, the analysis concerning whether the control group and the patient group differed on performance on CWIT and responses on RRS and RRQ at T3 were conducted. Then, the analysis concerning the PSG were conducted, as well as the analysis concerning a possible relationship between rumination and performance on CWIT for the patient group, using the subgroups, and the control group. Finally, analysis of regression was performed to investigate the predictive value of the RRS/RRQ and CWIT.

Preliminary Analysis

Independent samples t-tests were computed to check whether the control group and the patient group matched on the demographic variables age, education and IQ at T1 and T3 separately. An independent sample t-test was also used to check whether the PSG differed in MADRS score at T1 and T3. Chi-square was used to check whether the control group and the patient group matched in gender. The results showed no significant differences on group means on the different variables, or in distribution of gender. One-way analysis of variance (ANOVA) was conducted at T1 and T3 to compare the relapse group, the no-relapse group, the no-change group and the control group on the mentioned demographic variables. No differences in means were found, except from a mean difference in MADRS score at T3 for the PSG, F (1,21) = 4.69, p = .04. Post-hoc comparisons, using Tukey HSD test, show a significant difference in

education between the RLG (M = 13.5, SD = 1.6) and the NRG (M = 15.5, SD = 1.2) at T1.

To investigate whether the control group and the patient group differed in performance on CWIT from T1 to T3, a mixed between-within repeated measures ANOVA was conducted. The basic design was Group (patient and control group) x Test occasion (T1 and T3) x Test condition (C, W, CW and IS). CWIT was measured by the number of seconds each subject required to complete the trial, and the data used was the raw scores. Assumptions of equality of variances were met, but Box's test of equality of covariance matrices was significant and one must therefore be careful when interpreting the results. The main effect of group was significant, F (1, 39) = 15.20, p = .000, partial eta squared = .280, suggesting there is a difference in performance between the patient and control group. The results are presented in Table 5.

To further investigate the difference between the control group and the patient group on the CWIT, a collective mean score for condition 1 and 2, and a collective mean score for condition 3 and 4 was computed. The mean score for condition 1 and 2 represents lower cognitive functioning, while the mean score for condition 3 and 4 represents higher cognitive functioning. Mean differences between the patient group and the control group on the Higher and Lower Cognition scores are shown in Figure 1. A mixed between-within repeated measures ANOVA was conducted using the two mean scores. The basic design was Group (patient and control group) x Test occasion (T1 and T3) x Test condition (Lower and Higher Cognition score). Assumptions of equality of variances were met, but Box's test of equality of covariance was significant. One must therefore be careful when interpreting the results. The main effect comparing the group difference was significant, F (1, 39) = 14.49, p = .000, partial eta squared =

.271, suggesting there is a difference in performance between the patient and control group. The results are presented in Table 5.

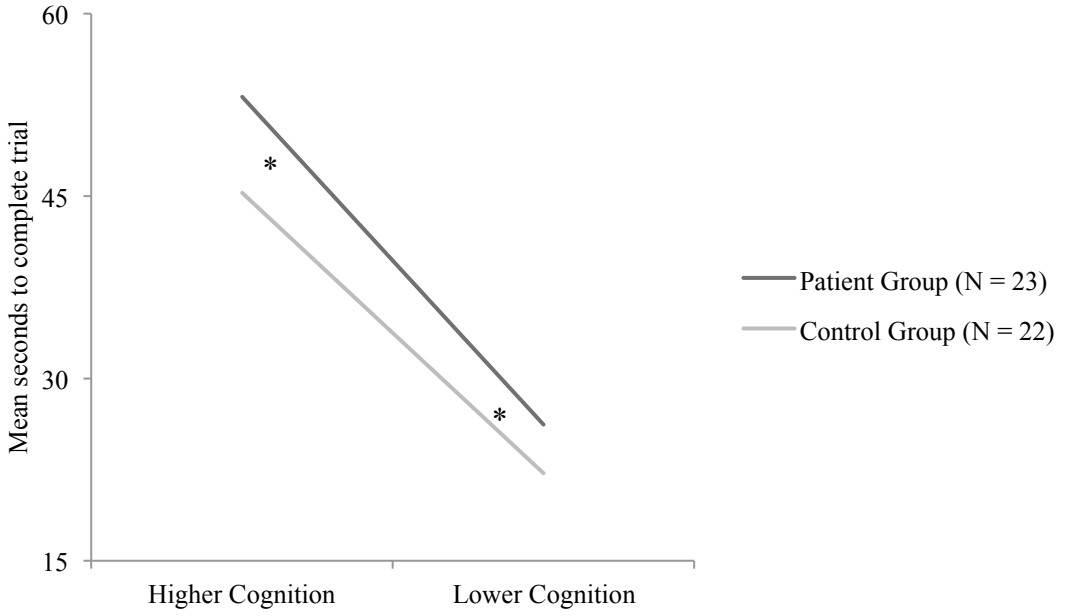

Figure 1
Mean scores and significant difference between patient group and control group on the Higher and Lower Cognition scores at T3.
* Significant on a < .05 level.

Table 5

Cognitive performance in the PG and CG across T1 and T3

		Main Effect		Interaction Effect			
		Condition	Time	Time x Group	Time x Condition	Group x Condition	Time x Condition x Group
CWIT	Wilks λ	0.038	0.675	0.962	0.792	0.885	0.943
	F (df)	309.60 (3,37)	18.81 (1,39)	1.54 (1,39)	3.24 (3,37)	1.60 (3,37)	0.75 (3,37)
	Eta sq.	0.962	0.325	0.038	0.208	0.115	0.057
	F-sig	*p* = .000*	*p* = .000*	*p* = .222	*p* = .033*	*p* = .205	*p* = .531
Lower/ Higher	Wilks λ	0.057	0.667	0.978	0.812	0.894	0.992
	F (df)	644.09 (1,39)	19.49 (1,39)	0.89 (1,39)	9.02 (1,39)	4.63 (1,39)	0.30 (1,39)
	Eta sq.	0.943	0.333	0.022	0.118	0.106	0.008
	F-sig	*p* = .000*	*p* = .000*	*p* = .351	*p* = .005*	*p* = .038*	*p* = .583

* Significant on a < .05 level.

Bivariate correlation was used to explore the relationship between depression severity, measured by MADRS, and the CW and IS raw scores, contrast scores and the Higher Cognition score, and between depression severity and rumination, measured by RRS sum score and RRQ rumination, on T3. Results are shown in Table 6.

Table 6

Correlation coefficients between depression severity, inhibition and rumination at T3

	Inhibition	Inhibition/ Switching	Higher Cognition	CW Contrast	IS Contrast	RRS	RRQ
MADRS	*r* = .122	*r* = .518*	*r* = .210	*r* = -.103	*r* = .395	*r* = .496*	*r* =.253
	n = 23	*n* = 23	*n* = 23	*n* = 23	*n* = 23	*n* = 23	*n* = 23
	p = .581	*p* = .011	*p* = .336	*p* = .639	*p* = .062	*p* = .016	*p* = .245

* Correlation is significant on a < .05 level (2-tailed)

Main Analysis

CWIT. A multivariate ANOVA was conducted in order to investigate the mean differences on the four conditions on CWIT between the control group and the patient group at T3. The independent variable was group, with two levels. The dependent variables were the four different conditions on CWIT. Box's test of equality of covariance matrices was significant, and one must therefore be careful when interpreting the results. The results show a significant difference between the two groups, F (4,40) = 5.44, p = .001, Wilk's Lambda = .648, partial eta squared = .352. The between-subjects effects results are shown in Table 7.

Table 7

Between-Subjects Effects - Patient Group and Control Group on CWIT

		Condition			
		Color Naming	Word Reading	Inhibition	Inhibition/ Switching
CWIT	*F* (df)	9.28 (1,43)	19.44 (1,43)	11.96 (1,43)	9.89 (1,43)
	Eta sq.	0.178	0.311	0.218	0.187
	F-sig.	$p = .004$*	$p = .000$*	$p = .001$*	$p = .003$*

* Significant on a < .05 level

A multivariate ANOVA was also computed to investigate the mean differences between the Higher and Lower Cognition scores between the patient group and the control group. The independent variable was group, with two levels, and the dependent variables were the two combination scores. Assumptions of equality of variance were met. The results show a significant difference between the groups, F (2,42) = 8.73, p = .001, Wilk's Lambda = .706, partial eta squared = .294. The between-subjects effects show a significant difference between the groups on the Higher Cognition variable, F (1,43) = 15.66, p = .000, partial eta squared = .267, and on the Lower Cognition variable, F (1,43) = 12.12, p = .001, partial eta squared = .220.

Rumination. A multivariate ANOVA was conducted to investigate the mean differences between the patient group and the control group on the two measures on rumination, RRS and RRQ, at T3. The independent variable was group, using two levels: patient group and control group. The dependent variables were RRS sum scores and RRQ rumination sum scores at T3. Assumptions of equality of variance were met. The results show a significant difference between the two groups, F (2,42) = 13.65, p = .000, Wilk's Lambda = .650, partial eta squared = .394. The between-subjects effects show a significant difference between the groups on RRS, F (1,43) = 26.03, p = .000, partial eta squared = .377, and between the groups on RRQ, F (1,43) = 9.29, p = .004,

partial eta squared = .178. When inspecting the mean scores for the PG and the CG for both RRS and RRQ, the patient group displays higher mean scores for both measures, although the difference is greater for RRQ.

Patient subgroups. A multivariate ANOVA was computed in order to investigate mean differences between the patient subgroups (RLG and NRG) and the control group on the four conditions on CWIT at T3. The independent variable was group, with three levels, and the dependent variables were the four conditions on CWIT. Assumptions of equality of variances were met. The results show a significant difference between the groups, F (8,78) = 2.63, p = .013, Wilk's Lambda = .620, partial eta squared = .212. The between-subjects effects results are shown in Table 8. Results from post-hoc comparisons, using the Tukey HSD test, are shown in Figure 2.

Table 8

Between-Subjects Effects - Relapse Group, No-relapse Group and Control Group on CWIT

		Condition			
		Color Naming	Word Reading	Inhibition	Inhibition/ Switching
CWIT	*F* (df)	4.57 (2,42)	9.51 (2,43)	5.97 (2,43)	5.20 (2,43)
	Eta sq.	0.179	0.312	0.221	0.199
	F-sig.	p = .016*	p = .000*	p = .005*	p = .01*

* Significant on a < .05 level

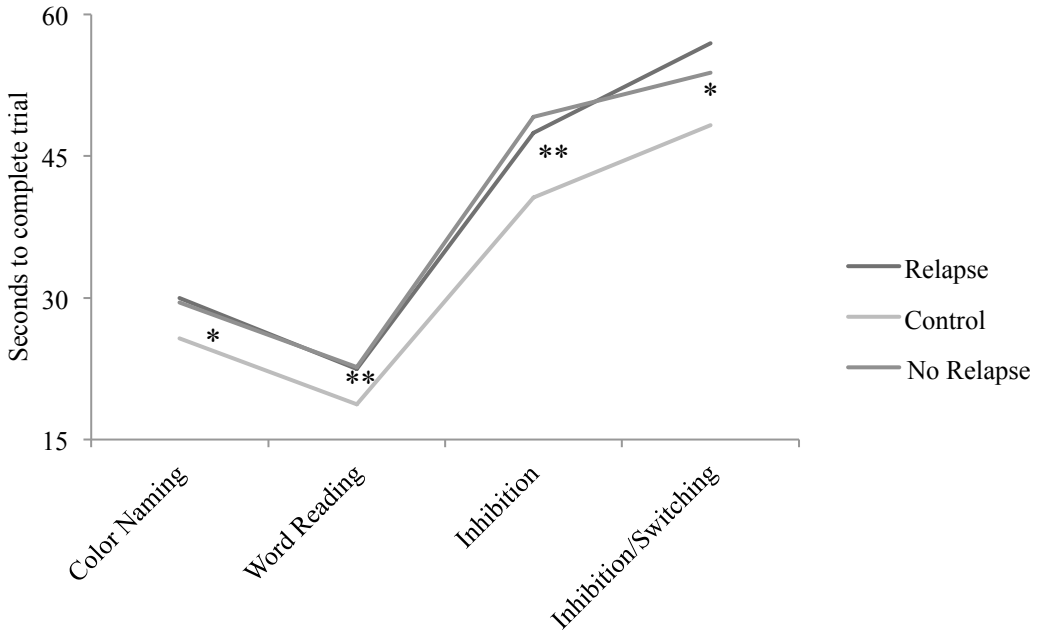

Figure 2
Mean Scores and Tukey HSD Significance Between RLG, NRG and CG on CWIT
* Significant difference between the RLG and the CG on a < .05 level
** Significant difference between the RLG and CG and between NRG and CG on a < .05 level

A multivariate ANOVA was also computed to investigate the mean group differences between the patient subgroups (RLG and NRG) and the control group on rumination, measured by sum scores on RRS and on RRQ rumination at T3. The independent variable was group, with three levels (RLG, NRG and CG), and the dependent variables were RSS sum score and RRQ rumination sum score. Assumptions of equality of variance were met. The results show a significant difference between the groups, F (4,82) = 8.39, p = .000, Wilk's Lambda = .291, partial eta squared = .291. The between-subjects effects show a significant difference between the three groups on RRS, F (2,42) = 18.78, p = .000, partial eta squared = .472, and on RRQ, F (2,42) = 8.97, p = .001, partial eta squared = .299. Results from post-hoc comparisons, using the Tukey HSD test, show a significant difference between the RLG (M = 52.1, SD = 10.9) and the NRG (M = 38, SD = 14.8) and between the RLG and the CG (M = 30.8, SD = 9.5) for RRS. For RRQ, post-hoc comparisons show a significant difference between

the RLG (*M* = 45.4, *SD* = 10) and the NRG (*M* = 32.7, *SD* = 15.3) and between the RLG and the CG (*M* = 32.4, *SD* = 8). Results are displayed in Figure 3.

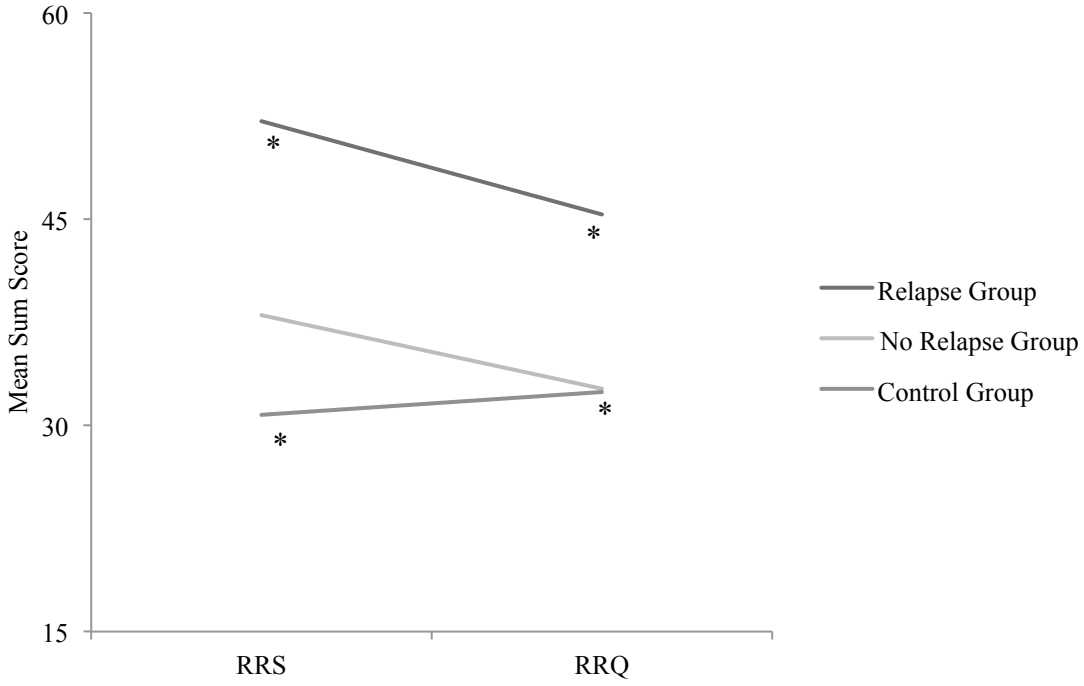

Figure 3
Mean Sum Scores and Tukey HSD Significance between the RLG, NRG and CG on RRS and RRQ
* Significant difference between RLG and NRG and between RLG and CG on < .05 level

A one-way between-groups ANOVA was computed to explore the mean group differences between the patient subgroups (RLG and NRG) and the control group on RRS at T3. Assumptions of equality of variances were met. The results show a statistical significance between the three groups, F (2,42) = 18.78, p = .000. The effect size was calculated using eta squared, and showed a large effect size, .472**.** Post-hoc comparisons using the Tukey HSD test show that the mean score for the RLG (*M* = 52.12, *SD* = 10.92) was significantly different from the NRG (*M* = 38, *SD* = 14.89), p = .023, and the CG (*M* = 30.77, *SD* = 9.50), p = .000. The NRG did not differ significantly from the CG.

A one-way between-groups ANOVA was also computed between the RLG, the NRG and the CG on RRQ at T3. Assumptions of equality of variances were met. The

results here show a statistical significant difference between the three groups, F (2,42) = 8.97, p = .001. Effect size was calculated using eta squared**,** .299**,** which is a large effect size, though smaller than what was found for RRS. Post-hoc comparisons using the Tukey HSD test show that the mean score for the RLG (M = 45.35, SD = 9.99) was significantly different from the NRG (M = 32.67, SD = 15.267) p = .026, and the CG (M =32.41, SD = 8.02), p = .001. The NRG did not significantly differ from the CG.

Correlation Analysis. Correlation analyses were performed using Pearson's R, in order to explore the relationship between the CW and IS conditions on CWIT and RRS sum scores on T3 and between the CW and IS conditions and RRQ rumination at T3. The analyses were conducted for all subjects, and for the control group and the patient group. Further, the analyses were also conducted for the patient subgroups (RLG and NRG). Correlation coefficients were further computed for the contrast scores and the Higher and Lower Cognition scores for all groups Results are shown in Tables 9 and 10.

Table 9

Correlation Coefficients (Pearson's R) between RRS, RRQ and CWIT at T3

		Inhibition	Inhibition/Switching	Lower Cognition	Higher Cognition	CW Interference Score	IS Interference Score	RRQ
All Subjects (n = 45)	RRS	.305*	.361*	.412**	.368*	.137	.242	.811**
PG (n = 23)	RRS	.124	.386	.376	.315	-.045	.280	.817**
CG (n = 22)	RRS	-.180	-.158	-.230	-.268	-.055	-.089	.668**
RLG (n = 17)	RRS	-.152	.202	.123	.086	-.237	.190	.654**
NRG (n = 6)	RRS	.868*	.571	.886*	.791	.784	.209	.943**

* Correlation is significant at a < .05 level (2-tailed)
** Correlation is significant at a < .01 level (2-tailed)

Table 10

Correlation Coefficients (Pearson's R) between RRQ and CWIT at T3

		Inhibition	Inhibition/Switching	Lower Cognition	Higher Cognition	CW Interference Score	IS Interference Score
All Subjects (n = 45)	RRQ	.293	.306*	.282	.302*	.205	.243
PG (n = 23)	RRQ	.293	.345	.235	.336	.251	.314
CG (n = 22)	RRQ	- .282	- .091	- .185	- .307	- .193	-.029
RLG (n = 17)	RRQ	.042	.063	- .218	.056	.151	.221
NRG (n = 6)	RRQ	.970**	.629	.922**	.879*	.938**	.266

* Correlation is significant at a < .05 level (2-tailed)
** Correlation is significant at a < .01 level (2-tailed)

Logistic Regression. A direct logistic regression was performed to explore the impact of poor performance on CWIT on the likelihood of experiencing relapse at T3 in the patient group. The full model containing the predictor (Higher Cognition at T2) was not significant $\chi^2_{(1, N=21)} = 0.95$, $p = .330$. This indicates that the model is not able to distinguish between those who relapsed at T3 and those who did not. The model as a whole explained between 4.4 % (Cox and Snell R Squared) and 6.3 % (Nagelkerke R Squared) of the variance and correctly classified 76.2 % of the cases. None of the variables contributed significantly to the model (Table 12)

Table 12

Logistic Regression Predicting Likelihood of Relapse at T3 from Higher Cognition

	B	S.E.	Wald	df	p	Odds Ratio	95.0 % C.I. for Odds Ratio	
							Lower	Upper
Higher Cognition	- .047	.050	0.903	1	.342	0.954	.866	1.051
Constant	3.368	1.622	1.622	1	.203	29.011		

A direct logistic regression was performed to explore the impact of a tendency to ruminate on the likelihood of experiencing relapse at T3 in the patient group. The full model containing the predictors (RRQ rumination sum score and RRS sum score at T2) was not significant $\chi^2_{(2, N=19)} = 1.86$, $p = .394$. This indicates that the model is not able to distinguish between those who relapsed at T3 and those who did not. The model as a whole explained between 9.3 % (Cox and Snell R Squared) and 13.1 % (Nagelkerke R Squared) of the variance and correctly classified 63.2 % of the cases. None of the variables contributed significantly to the model (Table 13).

Table 13

Logistic Regression Predicting Likelihood of Relapse at T3 from RRS and RRQ

	B	S.E.	Wald	df	p	Odds Ratio	95.0 % C.I. for Odds Ratio	
							Lower	Upper
RRS	.037	.060	.383	1	.536	1.038	.923	1.167
RRQ	.037	.074	.245	1	.620	1.037	.897	1.200
Constant	-2.487	2.610	.908	1	.341	0.083		

A direct logistic regression was performed to explore the impact of poor performance on CWIT and a tendency to ruminate on the likelihood of experiencing relapse at T3 in the patient group. The full model containing the predictors (Higher Cognition at T2, RRQ rumination sum score and RRS sum score at T2) was not significant $\chi^2_{(3, N=19)} = 3.91$, $p = .271$. This indicates that the model is not able to distinguish between those who relapsed at T3 and those who did not. The model as a whole explained between 18.6 % (Cox and Snell R Squared) and 26.1 % (Nagelkerke R Squared) of the variance and correctly classified 73.7 % of the cases. None of the variables contributed significantly to the model (Table 14).

Table 14

Logistic Regression Predicting Likelihood of Relapse at T3 from Higher Cognition, RRS and RRQ

	B	S.E.	Wald	df	p	Odds Ratio	95.0 % C.I. for Odds Ratio	
							Lower	Upper
RRS	.039	.068	.333	1	.564	1.040	.911	1.187
RRQ	.076	.087	.761	1	.383	1.079	.910	1.279
Higher Cognition	- .082	.060	1.890	1	.169	0.921	.819	1.036
Constant	.082	3.164	.001	1	.979	1.085		

Discussion

The results in the present study show that the hypothesis regarding poorer performance on measures of cognitive inhibition in the patient group, compared to the control group, was supported. The second hypothesis, regarding self-reported higher levels of rumination in the patient group compared to healthy controls was also supported. The results further show that the hypothesis regarding a positive correlation between cognitive inhibition and self-reported rumination was also supported. The hypotheses concerning the relationship between cognitive inhibition, rumination and a risk of relapse at T3 were, however, not supported. The results will be discussed in the following sections, along with discussions on methodological considerations and clinical implications.

Preliminary analyses show that the patient group and control group matched on demographic variables. The only exception was a difference in depression severity for the patient subgroups, but this was expected, as the no-relapse group has not experienced any depressive episodes since T2, in contrast to the relapse group.

Correlation analysis between depression severity and performance on Cognitive Inhibition and self-reported rumination show statistically significant positive correlations between MADRS and the condition four on CWIT, which might indicate that the performance on this task is confounded by the severity of the depressive symptoms in the patient group. There was further a statistically significant correlation between MADRS and RRS sum score. This correlation will be further discussed in the strengths and limitations section.

The preliminary analysis further shows that the patient group and control group differ significantly in performance on CWIT across time, which is in concurrence with

the findings from T1 and T2 (Schmid & Hammar, 2013b). When using the combination scores, we found the same results, suggesting that difference in performance is not due to poorer cognitive processing speed alone for the patient group. According to the D-KEFS examiners manual (Delis, Kaplan, & Kramer, 2001), one can separate between higher and lower cognitive functions on the CWIT, where the color naming and word reading conditions are considered to be lower mental functions that are automatic and do not require higher-level processes. The sum of the two first conditions of CWIT, the average of which comprise the Lower Cognition variable, has been conceptualized as a composite score of basic cognitive functioning (Lippa & Davis, 2010). On the other hand, conditions 3 and 4 are considered to be higher cognitive tasks, which rely on more sophisticated processes (Lippa & Davis, 2010). The patient group and the control group differed significantly on mean Higher Cognition combination scores and on mean Lower Cognition combination scores.

In order to test the first hypothesis, the difference between the patient group and the control group, as well as the patient subgroups, on the performance of the CWIT, and level of rumination at T3 alone was investigated. Based on former research, it was expected that the patient group would perform significantly poorer than the control group on the conditions 3 and 4 on the CWIT. It was further expected that the relapse group would have significantly longer response times than the no-relapse group. The results support the hypothesis and showed that the patient and control group differed significantly on the performance in all conditions. The results also demonstrated a significant difference between the groups on the Lower Cognition variable and the Higher Cognition variable. These findings are consistent with the findings of Schmid and Hammar (2013a, 2013b) from T1 and T2, which demonstrated that the patient

group performed significantly poorer on condition 4 when analyzing the raw scores on the CWIT.

When investigating the patient subgroups and control group on the four conditions of the CWIT, there was a significant difference between the groups. The post-hoc test demonstrated that the relapse group performed significantly poorer than the control group on all four conditions, while the no-relapse group performed significantly poorer than the control group on condition 2 and condition 4. There were no significant differences between the relapse group and the no-relapse group on any of the four CWIT conditions. Schmid and Hammar (2013b) reported a tendency for those patients who experienced relapse within the first year after initial episode to perform poorer on conditions 3 and 4 tested at T2, compared to the no-relapse group and the control group. It was not possible to fully replicate these findings at T3, as there was no significant difference between the relapse group and the no-relapse group. Research has demonstrated that both depressed and remitted patients struggle with moderate deficits on attention and executive functioning tasks (Rock et al., 2014), and since the relapse-group has experienced one or more depressive episode between T2 and T3, we expected the group to perform poorer than the no-relapse group.

In summary, when testing the first hypothesis, it was supported that the patient and control groups differ on measures of Cognitive Inhibition. This holds true both for the patients who had relapsed and for those who had experienced no new episodes of depression since T2. There was no difference in impairment of Cogntive Inhibition between the two patient subgroups.

In the second hypothesis, it was expected that the patient group would report higher levels of self-reported rumination than the control group. In relation to this, it

was further expected that the patients who experienced relapse since T2 would ruminate more than the patients who did not. The results demonstrated a significant difference between the patient group and the control group on scores on the RRS and the RRQ at T3 respectively. This indicates that the patient group reported significantly higher levels of rumination than the control group, on both questionnaires. This was expected, as trait rumination seems to be a quite stable tendency that is consistent over time and across levels of depressive symptoms (Nolen-Hoeksema & Davis, 1999; Nolen-Hoeksema et al., 2008). The patients who experienced relapse since T2 scored significantly higher than the no-relapse group and the control group on both RRS and RRQ. This is consistent with previous findings indicating that a ruminative response style has been a predictor of relapse risk in individuals who experience depressive mood (Huffziger et al., 2009; Michalak et al., 2011; Nolen-Hoeksema, 2000). These findings will be discussed later in the paper. There was no significant difference between the no-relapse group and the control group in rumination means scores. The no-relapse group reported a slightly higher mean score than the control group on the RRS, but not on RRQ rumination. One would expect that the no-relapse group would show higher levels of rumination than the control group, but this is not the case in the present study. Generally in our findings, the RRS has been more effective in reporting differences between groups, and this might explain the slight difference in mean score. RRQ is a more concentrated rumination questionnaire than RRS, and when investigating RRQ there was no difference between the control group and the no-relapse group. This will be further discussed later on.

Thus, the findings when testing the second hypothesis indicate that there is a difference between healthy controls and previously depressed individuals in their

tendency to ruminate. In addition, it seems that the patients who have relapsed since T2 ruminate more than those who have remained symptom-free, a finding that is in keeping with other research on the subject.

Correlation analysis was performed in order to test the third hypothesis. When looking at the correlations between RRS/RRQ and CWIT, results showed the strongest relationships when the analyses for all the subjects were performed. Across the patient and control groups, there was a significant correlation between RRS and CWIT conditions 3 and 4, and the Higher and Lower Cognition variables respectively. RRQ was significantly correlated with CWIT condition 4 and Higher Cognition. This is in accordance with the hypothesis. It was not part of the hypotheses that lower cognitive functions would correlate with rumination, and it is therefore interesting that this was the case with the RRS. It is worth to note, however, that the Lower Cognition variable was not significantly correlated with the RRQ. The correlations with CWIT condition 3 and 4 and Higher Cognition were expected, as these conditions require more effort and cognitive control, which is hypothesized to be related to rumination. That a higher score on the rumination measures is associated with a slower response on the more demanding Cognitive Inhibition tasks, indicates that people who tend to ruminate have impaired cognitive inhibition, which is in keeping with the hypothesis.

When separating between the patient and control groups, the strength of the above-mentioned correlations diminished and did not reach statistical significance. Further, looking at the relapse group and no-relapse group separately, there were no significant correlations between RRS/RRQ and inhibition in the relapse group. In the no-relapse group, however, both RRS and RRQ were significantly correlated with the Lower Cognition variable and condition 3 of CWIT. Thus, there is a stronger

relationship between Cognitive Inhibition and rumination for the previously depressed who have not relapsed, than for those who have had subsequent episodes since T2. It was not predicted that there would be a relationship between basic cognition and rumination, as was found in the no-relapse group. It was expected that the association between inhibition and rumination would be just as strong or stronger in the relapse group as in the no-relapse group, but the results did not support this.

Results showed a significant association between the RRS and RRQ-rumination scales across all subjects and subgroups in the study. Hence, the two questionnaires evoke a similar pattern of responses, which will be discussed later.

In summary, the measures of rumination used in this study have displayed a significant correlation with Cognitive Inhibition as measured by CWIT for all subjects included in the study. There was not found any significant correlations between Cognitive Inhibition and measures of rumination in previously depressed patients who have relapsed since their first depressive episode. On the measures used, there is no difference between the patient and control groups in the strength of the relationship between inhibition and rumination.

As mentioned earlier, there was no support for the hypotheses that poor performance on CWIT and high self-reported rumination would be able to predict who would experience relapse between T2 and T3. The model was able to correctly classify 73.7 percent of the cases, even though none of the variables of interest contributed in a statistically significant way. The predictive models for both RRS/RRQ and Higher Cognition separately were also not significant. Methodological consideration will be discussed in further detail later on, but it is important to note that the low *n* in the no-relapse group might affect the results here. To the extent of our knowledge, this is the

first longitudinal study investigating the relationship between impaired cognitive inhibition, rumination and a risk of relapse in clinically depressed patients. There is also a possibility that different results might be seen at the ten-year follow-up, but this is difficult to say at this point. At T2, logistic regression analysis did show that poor performance on condition 3 and 4 on CWIT, using contrast scores, was related to a risk of relapse (Schmid & Hammar, 2013a), and that it was condition 4 that contributed significantly to the model. It therefore seems that this predictive value is no longer relevant at the five-year follow-up, as we were not able to obtain similar results. However, it is important to note that rumination was not included in the analysis at T2, which might affect the results, even though none of the variables contributed significantly. It was the RRS and RRQ scores that had the highest Odd's Ratio (1.040 and 1.079), which might further indicate that the Higher Cognition factor (OR 0.921) no longer is as related to risk of relapse as it was at T2.

Previous longitudinal studies investigating the cognitive impairments in former depressed individuals have found differing results. There seems to be a general consensus that patients in the acute phase of a depressive disorder show cognitive impairments in different domains, like memory, executive functioning and attention (Hammar & Årdal, 2009). Douglas and Porter (2009) concluded in their review that executive functioning remains impaired over time. It is worth to note that the studies used in this review focused on relatively short periods of time, with most of them being ≤ 6 months. A meta-analysis and systematic review by Rock and colleagues (2014) showed that patients continue to show impaired cognitive abilities, specifically in the domain of executive functioning, when in a state of remission. However, this study was not conducted in order to investigate differences over time, but to investigate the

difference between patients who are currently depressed and patients in a state of remission (Rock et al., 2014). A two-year follow-up study by Biringer and associates (2005) found that patients who had recovered completely from a depressive episode also showed a recovery on executive functions, compared to healthy controls, however the results showed prevailed impairment in measures of cognitive inhibition. A study by Hammar and Årdal (2012) found no continuous impairment in effortful information processing at a ten-year follow-up of depressed patients. Another study by Årdal and Hammar (2011) found that at the ten-year follow-up the depressed patients show impairments in inhibition that was associated with the impairments shown at inclusion. Previous depressive episodes was not an exclusion criterion in this study, and they did not include rumination as a factor of interest (Årdal & Hammar, 2011). These results are of relevance to the current study, as it shows that impairments in inhibition are persistent over the course of ten years. It appears that it is difficult to conclude whether or not people suffering from recurrent depression will continue to show impairments in executive functioning over time (Hammar & Årdal, 2009). Reviews show that patients appear to continue to show impaired executive functioning (Douglas & Porter, 2009; Rock et. al, 2014), but few longitudinal studies that span across several years have been conducted (Hammar & Årdal, 2009).

As mentioned previously, studies have linked rumination to both an increase in number and severity in depressive symptoms over time (Nolen-Hoeksema, 2000), and there is emerging evidence that rumination is related to a risk of relapse. Nolen-Hoeksema (2000) found in her study that ruminative responses was correlated with depressive symptoms one year later, as well as with a depression diagnosis, though the effect on depression diagnosis was not significant when controlling for baseline

depression severity. A study by Michalak and colleagues (2011) showed that after going through mindfulness-based cognitive therapy, the subjects self-reported rumination predicted relapse, even when controlling for level of depressive symptoms and previous number of episodes. A study examining relapse risk factors in patients with comorbid avoidant personality disorder and MDD found that rumination was associated with personality pathology and increased risk of relapse in this population (van Rijsbergen, Kok, Elgersma, Hollon & Bockting, 2015). Huffziger and associates (2009) found in their longitudinal study of formerly depressed patients that ruminative symptom-focus predicted depressive symptoms over a period of three and a half years. Longitudinal studies on adolescents have found that high rumination can predict higher levels of depressive symptoms, increase in depressive symptoms and future symptoms (Calvete, Orue & Hankin, 2015; Wilkinson, Croudace & Goodyer, 2013). As mentioned previously, there are no studies that have investigated whether impairment in executive functioning and high self-reported rumination is related to an increased risk of relapse. Several studies, including those mentioned above, have investigated these variables, but not together. Even though our model was not able to predict a risk of relapse at T3, based on data from T2, RRS sum score and RRQ rumination were the variables with the highest odd's ratio in our model, and it appears that these are of more relevance when it comes to risk of relapse five years after the first depressive episode.

Strengths and Limitations

It has previously been mentioned that there is a lack of longitudinal research on depression in general, which makes this study, with its ten-year perspective, an important addition to the current literature. Another strength of this study is that the patient group consists of subjects who at T1 were going through their first episode of

depression. This provides a unique opportunity to study how initial impairments in inhibition and tendencies to ruminate develop in the years following onset.

This study also uses a patient group as opposed to inducing sad mood in healthy individuals, which has been the case in several other investigations of rumination. It is our opinion that using depressed individuals when studying rumination and inhibition increases the clinical relevance of our findings compared to studies using *e.g.* college students. In addition, this study did not induce rumination, but rather measured trait rumination through questionnaires. We believe that this is a more relevant procedure when looking at change in ruminative tendencies over time.

There are a few noteworthy limitations to the present study. The one that is perhaps the most pressing is the relatively small sample size. At the five-year follow-up, the patient group was reduced to 23 individuals, the no-relapse group consisting of merely six subjects. This reduces the power of the statistical analyses performed and therefore increases the risk of making a type II error when interpreting the results. As mentioned earlier, attempts were made to compensate for this by setting a more lenient alpha-level for the analyses with the patient subgroups. This adjustment did not have any effect on the findings. The low number of subjects still makes it important to use caution when interpreting the results.

Another aspect that is important to note is that the full-scale RRS was used in all analyses in this study. As previously stated, Schoofs and associates (2010), Treynor, and colleagues (2003) and several other researchers have found evidence that there are two dimensions in the RRS, and that the "brooding" dimension, compared to the "reflection" subcomponent, is most strongly associated with depressive symptoms. Treynor and associates (2003) recommend that researchers using the current version of

RRS separate their results into the brooding and reflection sub-components, but also note that this could lead to difficulties in analysis due to the relatively few items on each dimension. The results show that the full-scale RRS was strongly correlated with the RRQ-rumination measure, and we thus concluded that it was justified to continue analyses without dividing the scale into brooding and reflection.

Demographically, the participants in this study differ from the average population on intelligence level, the average IQ of both patient group and control group being ≈119 at T3. Hence, it is possible that the findings are not applicable to a population of average or below-average intelligence. The subjects are also relatively young, which might mean that the results are not generalizable to an older population. Haddad and Gunn (2011) state that the age of onset for depression ranges between 19 and 44 years, with an average age of onset of 32 years. The average age of the participants in this study (≈26 in both groups at T1) is well within the above-mentioned range, but is somewhat lower than the average age of onset.

Clinical Implications

The findings in the present study indicate that the two measures on rumination used in this study, RRS and RRQ-rumination, are strongly correlated. It therefore appears that these questionnaires measure the same tendencies. The strong correlation between RRS and RRQ indicates that both these questionnaires can be used as assessment tools for clinicians when planning effective treatment programs for depressive disorders.

Further, the difference found between the groups on rumination and cognitive inhibition, as well as the correlation between rumination and cognitive inhibition, indicates that those who tend to ruminate also show impaired cognitive inhibition. Since

these findings replicate those from T1 and T2, it supports the hypothesis mentioned in the literature that rumination is indeed a trait-like construct. It is important for therapists to treat rumination as a stable trait, and if a patient shows high rumination in initial depressive episodes this is likely to persist despite symptom reduction.

Results showed significant correlations between cognitive inhibition and rumination for all subjects. Since this relationship was found for all subjects, it indicates that this pattern is not only valid for depressed patients, but also for healthy controls. This is in accordance with the previously mentioned findings that rumination is a trait-like construct that is also present in healthy controls, though not to the same extent as in depressed individuals. Assessing rumination might be important in low-threshold intervention programs and in prevention of depression in at-risk populations. The healthy controls showing the pattern of high rumination and poorer cognitive inhibition might be at risk for developing a depressive episode, for example after experiencing negative events or a life crisis.

Conclusions and Closing Remarks

In this study we posited hypotheses regarding the effect of rumination and cognitive inhibition on first episode depressed patients and healthy controls, and the relationship between these variables on the risk of experiencing recurrent depressive episodes. In conclusion, the results show that depressed individuals perform poorer on measures of cognitive inhibition than the control group, as well as reporting higher levels of self-reported trait rumination. There was found a correlation between cognitive inhibition and self-reported a rumination, when investigating all subjects, where a higher score on the rumination measures is associated with a slower response on the more demanding cognitive inhibition tasks. The hypothesis regarding that poor

performance on CWIT and high self-reported rumination would be able to predict relapse in the patient group, was not supported. The results have not been able to demonstrate significant differences between the relapse groups and the no-relapse group, which might be a result of low power in the sample size.

This study strengthens previous findings on the topic of rumination, which across different studies have found impairment in inhibition and/or mental flexibility in high ruminators, both in a clinical and random sample. We argue that our study is an important contribution to this research, as it is the first to investigate this relationship over a longer time-span, by testing a clinical sample five years after initial depressive episode.

Based on the results, some lines for further research are suggested. For future studies, we recommend conducting more longitudinal studies on a clinical sample of first-episode depressed individuals, given that such a high percentage experience relapse after their first depressive episode. This would make future research more generalizable in the depressive population, and increase its relevance for clinical real-life situations. In the present study, there was a high dropout rate from T1 to T3, affecting the power in the study. One could avoid this by using a larger sample size if possible, considering that dropouts are to be expected over such a long period of time. As previously discussed, non-clinical subjects with higher levels of rumination and slower response time on cognitive inhibition tasks may contribute to the correlation between cognitive inhibition and self-reported inhibition. This relationship can be interesting for future research investigating vulnerability for depression.

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