Impaired Cognitive Inhibition in First Episode Major Depressive Disorder

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Abstract

The aim of this study was to investigate cognitive inhibition in first episode major depressive disorder (MDD) at a one-year follow-up. Forty-three participants were assessed on the Behavior Rating Inventory of Executive Function—Adult Version (BRIEF-A) and the Color Word Interference Test (CWIT) one year after first being diagnosed with MDD. Previous studies of the same sample have shown that the patient group, especially those who relapsed, had impaired inhibition performance as measured by the CWIT, even after remission. This study investigated self-reported inhibition, as well as general executive functioning, measured by BRIEF-A. The patient group (n=26) reported more problems with inhibition and general executive functioning than controls (n=27), despite remission from other depressive symptoms. CWIT inhibition scores did not correlate with BRIEF inhibition, but did correlate with the BRIEF Global Executive Composite. There was no significant difference between the BRIEF-A scores of those who had relapsed and those who had not. These results support previous findings of persistent deficits after remission, but it is possible that the two measures should be treated as complimentary, not interchangeable, measures of inhibition in MDD. Inhibition has promise in the study of MDD, but needs to be carefully defined.
Sammendrag

Major depressive disorder (MDD) is a debilitating illness with an estimated lifetime prevalence of 11.1%-14.6% globally (Bromet et al., 2011). It is associated with significant negative consequences in a number of areas, including occupational performance (Baune et al., 2010; Preiss et al., 2009), social functioning (Segrin, 2000) and quality of life (Gostautas, Pranckeviciene, & Matoniene, 2006), as well as being linked to morbidity and mortality for a number of diseases (Lépine & Briley, 2011). MDD also constitutes a large economic burden to society in the form of direct and indirect healthcare costs (Sobocki, Jönsson, Angst, & Rehnberg, 2006), lower work productivity even after remission (Adler et al., 2006), and a reduced workforce due to disablement (Ferrari et al., 2013). Depression is considered the second largest cause of disability worldwide (Ferrari et al., 2013). The burden on the individual and on society is compounded by the high rate of relapse and chronicity of depression; more than 75% will have more than one episode (Boland & Keller, 2009), with about 50% relapsing within the first two years of an initial episode (Mueller et al., 1999). Naturally, mechanisms involved in the development of depression, in causing related disability and increasing risk of relapse are of great interest. Cognitive impairment has been associated with these different aspects of depression, and may possibly serve as a link between them.

There is broad consensus that depression involves cognitive impairment in important domains such as memory, attention, psychomotor- and processing-speed, and problem solving (Austin, Mitchell, & Goodwin, 2001; Baune, Fuhr, Air, & Hering, 2014; Hammar & Årdal, 2009; Lee, Hermens, Porter, & Redoblado-Hodge, 2012; Papakostas, 2014). Previous studies have indicated that deficits in executive function are present in the first episode of depression, specifically impairments on the measures
of inhibition and semantic fluency (Schmid & Hammar, 2013a). These executive function deficits were found to persist despite symptom recovery after one year (Schmid & Hammar, 2013b). Impaired performance during the acute phase on an inhibition task with additional demands on cognitive flexibility was found to differentiate between depressed participants who relapsed within a year and those who did not (Schmid & Hammar, 2013b). Thus, poor performance on this inhibition measure may reflect a cognitive impairment linked to the risk of relapse in MDD.

The current study further examined the role of inhibition in depression. The measure of inhibition in the study that found persistent impairment (Schmid & Hammar, 2013b) was the Color Word Interference Test (CWIT) from the Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001). The present study looked at the self-reported inhibition deficits for this same sample on the Behavior Rating Inventory of Executive Function–Adult Version (BRIEF-A; Roth, Isquith, & Gioia, 2005). It aimed to investigate whether impairments detectable in controlled testing environments are associated with comparable problems in daily life, addressing the unity of the inhibition construct as well as its utility in the study of depression vulnerability, disability and relapse. There are a number of conceptual and methodological obstacles to defining and investigating the presence, type and impact of cognitive impairment, but increasing evidence indicates that it may be a central part of understanding the depression cycle (Austin et al., 2001; Baune et al., 2014; Hammar & Årdal, 2009; Jaeger, Berns, Uzelac, & Davis-Conway, 2006).

**Cognitive Impairment in Depression**

The subjective experience of depression frequently involves reduced mental acuity, articulated in the Diagnostic and Statistical Manual of Mental Disorders-IV
(DSM-IV; American Psychiatric Association, 2000) as “diminished ability to think or concentrate, or more indecisiveness” (p.356). It is also generally accepted that cognitive content such as negative automatic thoughts and schemas play an important role in depression (Scher, Ingram, & Segal, 2005). Biased information processing in the domains of attention, appraisal and memory are well established as characteristics of the depressive cycle (Gotlib & Joormann, 2010; Mathews & MacLeod, 2005). Many studies have indicated that individuals with depression may be more vulnerable to these kinds of biased information processing, particularly when experiencing stress or negative affect (Ingram, 2003; Scher et al., 2005). However, research has suggested that affective disorders are associated with impairments in a number of important cognitive domains that exceed what can be explained by the affective symptoms and stress-related biases in information processing (Preiss et al., 2009; Thompson et al., 2005).

Suboptimal functioning of central cognitive processes may constitute an important source of vulnerability in depression.

There are a number of reasons why cognitive impairment in depression is of interest. The proposed roles for cognitive impairment include (a) a link with psychosocial disability; (b) a link with illness characteristics and outcomes; and (c) a vulnerability to depressive episodes. Relevant in all these areas is the relative stability and independence of cognitive impairment from other symptoms. These elements will be discussed in turn before exploring whether executive functions, and more specifically inhibition, is a good way to conceptualize this deficit.

**Psychosocial disability.** It has been shown in a variety of psychiatric conditions, including depression, that neuropsychological deficits impede life functioning in many areas, including, work, education, family relationships and other social relations
(Hammar & Årdal, 2009; Jaeger et al., 2006). Some studies have indicated that cognitive impairment may be the largest source of psychosocial disability for people with MDD by impairing occupational performance (McIntyre et al., 2013; Preiss et al., 2009). Both self-reported and objectively measured cognitive impairments have been found to be unique and strong predictors of disability in MDD, particularly in the area of physical disability, which involves impediments to physical activity, daily routines, and hobbies (Naismith, Longley, Scott, & Hickie, 2007).

It is not just during acute depressive episodes that MDD has been associated with reduced psychosocial functioning. Judd, Paulus, Wells, and Rapaport (1996) found that the disability for subsyndromal depression was equal to that of clinical depression, implying a role for a more persistent disadvantage. Impaired cognitive functioning may be persistent, occur independently of affective symptoms, and contribute to psychosocial disability beyond the effects of other symptoms. There are indications that cognitive symptoms do not always subside at the same rate as other symptoms, and might persist even after remission from MDD (Austin et al., 2001; Baune et al., 2010; Bhardwaj, Wilkinson, Srivastava, & Sharma, 2010; Bortolato, Carvalho, & McIntyre, 2014; Hammar & Årdal, 2009; Neu et al., 2005; Reischies & Neu, 2000), which could mean that the detrimental consequences for daily social, occupational and academic activities continue even after the other symptoms are reduced. Sarapas, Shankman, Harrow, & Faull (2013) have reported that cognitive performance could predict social functioning for people with depression after 18 years, even when controlling for baseline social functioning and symptoms of depression. Jaeger et al. (2006) found that neuropsychological impairments during hospitalization for MDD predicted disability in life functioning 6 months later. Not all studies have found the persistence of cognitive
symptoms after remission (Biringer et al., 2005; Neu et al., 2005), so causes of these discrepancies warrant further research.

It is possible that these psychosocial consequences in themselves contribute to increased stress and persistent illness, for example through less social support and thus reliance on less effective coping strategies (Jaeger et al., 2006). Reduced cognitive function may, however, also impair the ability to self-regulate, cope and problem solve more directly (Gotlib & Joormann, 2010). More knowledge on the topic could help determine whether interventions aimed at remediating or living with reduced cognitive function are useful ways to reduce disability in MDD.

**Outcomes and illness characteristics.** There are indications that, in addition to disability, cognitive impairment is correlated with other important MDD illness characteristics, such as chronicity, the number of relapses and long-term average severity (Bhardwaj et al., 2010; Buist-Bouwman et al., 2008; Sarapas et al., 2013; Fennig & Mottes, 2002). In a recent review of the literature, Papakostas (2014) found multiple and consistent indications that cognitive deficits were associated with earlier onset, longer duration and impediment to treatment and functional recovery. Schmid and Hammar (2013b) found that in first episode depression, cognitive functioning was related to the risk of relapse within a year. Some exceptions do exist; Reischies and Neu (2000) found no such correlation between cognitive functioning and duration of disease or number of episodes, despite persistence of impairment after remission and a relatively high median age of the participants ($Md = 53.7$ years). Determining when and how cognitive functioning is impaired in depression, and how it relates to illness characteristics, may offer valuable insights into the heterogeneity of MDD outcomes.
Vulnerability to depressive episodes. The association between poorer cognitive functioning and depression has been demonstrated repeatedly, but the exact nature of this relationship is complex. Three commonly cited hypotheses will be discussed briefly, as any possible causality is central to understanding the role of cognitive impairment in depression. The relationship between cognitive dysfunction and depression has been variously posited as a mood-dependent effect, a detrimental consequence of depression, or a more stable trait that constitutes a predisposition to depression.

Cognitive impairment as mood-dependent effect. It has been suggested that mild residual affective symptoms could be a factor contributing to apparent cognitive deficits in remission for bipolar patients (Clark, Iversen, & Goodwin, 2002). Scheurich et al. (2008) argued for a motivational origin of cognitive impairment in major depression, related to mood, task specific self-efficacy, and interest. However, motivation and effort have not always been found to co-vary with the symptoms (Jaeger et al., 2006). Furthermore, after controlling for mood effects and for hypercorticoalaemia, the impairments on a number of cognitive tests were still significant for MDD and bipolar disorder (Gorwood, Corruble, Falissard, & Goodwin, 2008; Jaeger et al., 2006; Thompson et al., 2005). For remitted patients, it has been reported that the concurrent levels of depressive symptoms were unrelated to cognitive impairment (Preiss et al., 2009; Schmid & Hammar, 2013b). Thus residual mood-dependent effects do not appear to explain all cognitive difficulties.

Cognitive impairment as a consequence of detrimental effects on the brain caused by depression, possibly resulting from neurotoxic effects. There is some research suggesting that depression has a detrimental effect on cognitive function,
specifically the fact that impairments have been found to correlate with the number of previous episodes (Kessing, 1998), get worse with each episode (Baune et al., 2010) and to correlate with the duration of the illness (Elgamal, Denburg, Marriott, & MacQueen, 2010; Gorwood et al., 2008; Sweeney, Kmiec, & Kupfer, 2000; Thompson et al., 2005). In a longitudinal study, past but not current symptom severity correlated with cognitive deficits, and cognitive impairment did not fluctuate with the other symptoms (Sarapas et al., 2013).

The detrimental effects of depression might involve endocrine and autonomic changes. One common finding in depression is hypersecretion of corticotropin-releasing factor (CRF) and consequently hyperactivity of the hypothalamic-pituitary-adrenocortical (HPA) axis and sympathetic activation (Arborelius, Owens, Plotsky, & Nemeroff, 1999; Gillespie & Nemeroff, 2005; Nemeroff, 1988). A review by Schlosser, Wolf and Wingenfeld (2011) found moderate support for the hypothesis that HPA-related changes are linked to cognitive performance, as 14 out of 20 studies reported significant correlations between these. Central to the dysregulation of the HPA is an excess of corticosteroids, which have the potential to cause neural damage and to negatively impact cognitive and affective function (Herbert et al., 2006). This, however, does not mean that depressive episodes themselves cause this damage. HPA-function, commonly linked to stress, may be influenced by genetic factors as well as life events (Herbert et al., 2006). In addition, cognitive impairment has been reported as early as the first episode in MDD (Hammar & Årdal, 2009; Lee et al., 2012), indicating that not all cognitive impairment can be explained simply by the detrimental effects of long term illness.
Cognitive impairment as the reflection of a trait or other vulnerability to depression. In addition to being reported in first episode MDD (Hammar & Årdal, 2009; Lee et al., 2012), some aspects of cognitive dysfunction might even predate the first episode, and thus serve as a predictive marker of depression. Compromised performance on cognitive tests has been demonstrated three years before depression develops (Airaksinen, Wahlin, Forsell, & Larsson, 2007) and with participants who are not yet depressed but genetically at risk for depression (Mannie, Barnes, Bristow, Harmer, & Cowen, 2009). Neuroanatomical abnormalities associated with recurrent depression have been found in first episode MDD (Lee et al., 2012), further supporting the hypothesis that some dysfunction may predate any potential long-term effects of depression. Performance in a number of cognitive domains has proven stable over six years for depressed patients (Sarapas, Shankman, Harrow, & Goldberg, 2012). It has been suggested that cognitive impairment in affective disorders are the result of a trait-level dysfunction in neural circuitry (Smith, Muir, & Blackwood, 2006; Thompson et al., 2005). This could represent a stable and possibly heritable (Smith et al., 2006) vulnerability to depression.

A combination. The nature of the relationship between cognitive impairment and depression could depend on several variables. One of these may be way in which cognitive dysfunction is measured and defined. There are some indications that some cognitive processes are only temporarily compromised in acute episodes, whereas others might represent longer-lasting traits (Douglas & Porter, 2009; Huang, 2009; Lee et al., 2012). In a meta-analysis of cognitive function in first episode MDD by Lee et al. (2012), psychomotor speed and memory were described as state-dependent, whereas attention and executive function were more persistent. This was also reported by
Douglas and Porter (2009). Huang (2009) has found poor executive and motor functioning to persist after remission, but not memory and attention.

The causality could also be reciprocal and complex. Longitudinal research reveals this complexity better than cross-sectional studies. For instance; cognitive measures at baseline predictive of psychosocial functioning 18 years later beyond the effect of illness severity (Sarapas et al., 2013), but cognitive functioning at 26 years was also strongly associated with average illness severity retrospectively (Sarapas et al., 2012). It thus seems that cognitive functioning at baseline might at least be predictive of a more debilitating form of depression. Regardless of the causal relationships, assessment of cognitive function may therefore be useful for early screening (Jaeger et al., 2006; Sarapas et al., 2012). It is possible that an initial cognitive vulnerability may be exacerbated by repeated depressive episodes. Understanding the causal relationship might also require taking into account shared influences, such as HPA-dysregulation (Arborelius et al., 1999; Schlosser et al., 2011).

**Divergence due to Heterogeneity**

One reason for the disagreements over the role of cognitive deficits in depression might be variations in methodology and the populations studied. Treating all MDD patients as one group has led to contradictory findings (Hammar & Årdal, 2009; McClintock, Husain, Greer, & Cullum, 2010). In order to further assess the independent impact of cognitive impairment, its effects needs to be differentiated from correlated characteristics of the illness, such as duration and number of episodes, depression subtype and other factors that have an impact on functioning (McIntyre et al., 2013). Numerous other variables have also been found to predict the degree to which cognitive impairment is associated with depression, such as comorbidity, age at onset, premorbid
intelligence and education level, current age, gender and profession (Elgamal et al., 2010; Gorwood et al., 2008; Grant, Thase, & Sweeney, 2001; Kessler, 2003; Levin, Heller, Mohanty, Herrington, & Miller, 2007; McIntyre et al., 2013). These variables have additionally been reported to interact; as with for instance cognitive flexibility, which correlated with more severe illness for unipolar but not bipolar depression (Sarapas et al., 2012). The present study used a well-defined population of outpatients experiencing first episode MDD with little or no co-morbidity. Even in well-defined populations, however, studying different parts of cognitive functioning, or using different tasks, might still lead to divergent conclusions on the role of cognitive impairment in depression (Jaeger et al., 2006). Identifying what tasks are sensitive to dysfunction in MDD is therefore essential.

**Identifying which Specific Deficits are Associated with Depression**

Attempting to describe the cognitive impairment more precisely has yielded widely varying results. Some of the domains that have been frequently linked to depression are: memory, problem solving, psychomotor speed and executive functions (Austin et al., 2001; Baune et al., 2014; Hammar & Årdal, 2009; Lee et al., 2012). However, in all these areas, studies are also reported which have found no impairment, and dividing each domain further into components such as working memory versus episodic memory, or cued versus free recall yields further discrepant findings (Austin et al., 2001; Baune et al., 2014; Hammar & Årdal, 2009; Lee et al., 2012). It might therefore be that crucial variables still remain unaccounted for in much of the previous literature that confound the question of which specific cognitive deficits are associated with depression. Characteristics of the study participants such as comorbidity, depression subtype, age, education, presence of psychosis, symptom severity and
medication have all been reported to influence what type of cognitive impairment that is found (Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, & Lönnqvist, 2008; Hammar & Årdal, 2009; Jaeger et al., 2006; Kizilbash, Vanderploeg, & Curtiss, 2002; Lee et al., 2012; Levin et al., 2007), suggesting that various cognitive processes might be differentially associated with depression for dissimilar groups. Using small and differing test batteries may also make comparison between studies problematic, potentially leading to divergent conclusions (Jaeger et al., 2006).

Executive Functions

Despite these differences, there have also been frequent overlaps between findings. Executive functioning is one of the areas that have been often researched in relation to MDD, and results on a wide range of executive tasks have been associated with depression (Baune et al., 2010; Hammar & Årdal, 2009; Ottowitz, Dougherty, & Savage, 2002; Snyder, 2013; Stordal et al., 2004). The term executive function is used to describe higher-order cognitive control-mechanisms involved in the regulation of thought, emotion and behavior (Garavan, Ross, Murphy, Roche, & Stein, 2002; Miyake et al., 2000; Roth et al., 2005). Executive functions coordinate, monitor and modulate cognitive subprocesses to enable a flexible approach to varying demands and conditions (Miyake et al., 2000).

Examples of proposed executive function tasks include the ability to select and redirect attentional focus; make, select and switch strategies; monitor performance; utilize feedback; deploy cognitive resources efficiently; and inhibit inappropriate responses (P. Anderson, 2002; Baune et al., 2010; Ridderinkhof et al., 2004). Basic cognitive skills such as attention, memory, language and perception are utilized in order to produce complex, abstract and creative thought and behavior (Alvarez & Emory,
2006; Swanson, 2005), making executive functions essential parts of a wide range of activities, including problem solving; decision making; goal-directed thought and behavior; planning; and self-regulation (Miyake et al., 2000; Rose, Feldman, & Jankowski, 2011; van der Plas, Crone, van den Wildenberg, Tranel, & Bechara, 2009). When executive functions are operating successfully; thoughts, emotions and behaviors are internally generated to a larger extent, and less stimulus-driven and automatic (Miyake et al., 2000), allowing for our actions to be consistent with internal goals in a complex and changing environment (Waskom et al., 2014).

In general, executive functions are thought to be specifically relevant in effortful tasks that require novel or non-routine processing (Shallice, 1988), and when there's a conflict between competing responses (Posner & DiGirolamo, 1998). It has therefore been posited that they are especially important in less defined or constrained tasks, where the participant needs to direct attention to relevant material and inhibit other elements (Lezak, 1982).

**Executive Functions and Research on Cognitive Impairment**

Executive functions might be of particular interest when studying cognitive impairment in MDD for a number of reasons. One reason is that executive functions are likely to be important to a large number of tasks in everyday life as well as in controlled settings (P. Anderson, 2002; Baune et al., 2010; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004; Waskom, Kumaran, Gordon, Rissman, & Wagner, 2014). A deficit in this area is therefore plausible as an explanation for varied and numerous problems. Hertel (1997) has suggested that the memory problems experienced in depression are caused by poor cognitive control. Gotlib & Joormann (2010) ruled out memory problems in one group of depressed individuals by structuring the task in a way
that reduced the opportunity for rumination; a process linked to executive control according to the authors. The fact that task characteristics, such as structure, have this impact on executive function performance could explain some of the variability previously discussed in cognitive function-results. It may be interesting to review the task structure in studies where results have contradicted many previous findings. For instance, in the study by Reischies and Neu (2000), in which illness duration or number of episodes did not correlate with cognitive dysfunction, the authors made a point of using short tasks.

Some cognitive function tasks might be placing greater demands on higher-level processes in charge of organizing and planning, in other words executive functions. In a review article, Castaneda et al. (2008) reported that in relation to depression, evidence of deficient executive functioning is more consistently found than problems with memory and learning. Comparisons of multiple tests in comprehensive test batteries have shown indications of executive dysfunction even when performance was unimpeded in a range of other domains (Hammar & Årdal, 2009; Porter, Gallagher, Thompson, & Young, 2003).

**Executive Functions and Depression**

Executive functioning has been liked to affective regulation and behavioral control (Gotlib & Joormann, 2010), and might consequently be a good candidate for exploring the relationship between cognition and affective disorders. The inability to disengage attention from negative material has been seen in depression (Beevers & Carver, 2003; Compton, Heller, Banich, Palmieri, & Miller, 2000), as have problems manipulating and discarding negative material from working memory (Gotlib &
Joormann, 2010). In people prone to rumination, impairments have been shown on tasks measuring executive function (Joormann & Gotlib, 2010).

Poor executive function might also be a good candidate for exploring disability in depression. Performance on tests of executive function has directly predicted daily functioning for a range of populations, including those with dysexecutive syndrome (Burgess, Alderman, Evans, Emslie, & Wilson, 1998), non-demented elderly people (Kiosses & Alexopoulos, 2005) and schizophrenia patients (Weinberger & Gallhofer, 1997). Executive functioning has also been found to predict important variables such as academic ability (Blair & Razza, 2007; Toll, Van der Ven, Kroesbergen, & Van Luit, 2011) and social skills (Hensler et al., 2014; Schonfeld, Paley, Frankel, & O’Connor, 2006).

Executive functions may prospectively influence affective adjustment; Ghassabian et al., (2014) found that executive functions mediated the link between low positive emotionality at 3 years of age, and withdrawn temperament at 6 years. Impaired executive functioning has additionally been evidenced in remission from affective symptoms, and is therefore a potential source of trait-like vulnerability or lasting disability (Bhardwaj et al., 2010; Douglas & Porter, 2009; Huang, 2009; Lee et al., 2012). It may be that not all patients with MDD suffer from executive dysfunction to an equal extent. According to a review by McIntyre et al. (2013), pronounced deficits in this area were present in about 20–30% of those with MDD.

Different Models of Executive Functions

The question of how cognitive functions are coordinated and selected has been approached from a variety of angles. Many models of various aspects of cognitive functioning have involved some form of control mechanism, such as Baddeley's central
executive for working memory (Baddeley, 1996); Posner and Petersen's top down control of attention (Posner & Petersen, 1990); and Shallice and Norman’s supervisory attentional system (Shallice & Norman, 1986) necessary for controlled, complex and novel information processing. Even in more general conceptualizations, terminology used as well as the importance awarded different control functions varies. Some have argued for the primacy of goal-activation and maintenance (Duncan, Emslie, Williams, Johnson, & Freer, 1996; Nieuwenhuis, Broerse, Nielen, & de Jong, 2004), the ability to maintain an active representation of context information in working memory (Braver & Cohen, 2001; Waskom et al., 2014) or navigating goals through a generalized cognitive search process (Hills, Todd, & Goldstone, 2010). Regardless of what is considered the main mechanism of executive control, it is clear that most researchers consider complex cognitive operations to be dependent on some form of regulation and selection, whether this is an emergent feature of the systems themselves or a separate process. The exact operationalization of the term, including the number, nature and organization of these executive functions, as well as the best way to assess them, varies somewhat in the literature (Alvarez & Emory, 2006; Hammar & Årdal, 2009; Miyake et al., 2000).

**One Executive Function**

Some have argued for a unitary view of executive function, seeing it as a single construct. Della Sala, Gray, Spinnler, and Trivelli (1998) asserted that the most common neuropsychological tests all draw on a common resource, and that there is no support for fractionation into subcategories of executive functions. De Frias, Dixon, and Strauss (2006) found, using confirmatory factor analysis, that a single-factor structure of executive functions provided the best fit. It has also been suggested that the proposed
functions of executive processes are better viewed as the outcome of other processes, such as working memory functioning (Kimberg, D’Esposito, & Farah, 1997).

**Multiple Executive Functions**

Most models, however, have posited a fragmentary view, consisting of distinct though often correlated mechanisms (P. Anderson, 2002; Lehto, Juujarvi, Kooistra, & Pulkkinen, 2003; Miyake et al., 2000; Stuss & Alexander, 2000). Although some executive functions have been found to depend on a general intelligence factor (Obonsawin et al., 2002), research has found that at least some functions (e.g. shifting and inhibition; Miyake et al., 2000) are independent of IQ. Ardila, Pineda, & Rosselli (2000) reject the correlation with traditional IQ measures altogether. Further support for the fractionation of executive functions comes from the fact that various pathologies have been associated with distinct executive functions (Ozonoff & Jensen, 1999; Pennington & Ozonoff, 1996) as well as variable developmental trajectories (P. Anderson, 2002). Behavioral symptoms of poor executive regulation have also been reported to load on multiple distinct factors (Burgess et al., 1998). Many of the tasks used to measure executive functions have been found to correlate with each other (Miyake et al., 2000; Della Sala et al., 1998), but these correlations have usually been low ($r = .40$ or less; Jurado & Rosselli, 2007).

Delis, Jacobson, Bondi, Hamilton, & Salmon (2003) have pointed out that in non-clinical populations, there tends to be a high degree of shared variance between tests of different cognitive functions, whereas the fragmentation becomes more apparent when comparing homogenous groups with focal neurological impairment to normative samples (Delis et al., 2003). There is still no consensus about the structure of executive
functions. Even if there are a number of subprocesses, these could still reflect different functions of one unitary system (Baddeley, 1996).

**Measuring Executive Functions**

Features of executive function make it a difficult domain to study. The extent to which a task requires deliberate and superordinate processes may vary between individuals and situations; even within a single test session performance on a given task can become increasingly automatized, and therefore less dependent on controlled processes (Hughes & Graham, 2002). Slight variations in task characteristics can shift the processing from automatic to controlled (Hughes & Graham, 2002). This can also potentially make repeated measures designs tricky, as a task is only new once (Hughes & Graham, 2002). Another potential obstacle to repeated testing, and consequently to assessing the reliability of executive function task measures, is that complex tasks involving a number of processes are likely to result in more sources of variation, and therefore a higher chance of measurement error (Delis, Kramer, Kaplan & Holdnack, 2004). Establishing measures that are both complex and novel can thus come into conflict with the need for tests that are replicable and standardized. By definition, executive functions also integrate and depend on the intact operation of other cognitive domains, making it challenging to develop tasks with good discriminant validity, and to precisely describe the mechanisms of the executive functions (Alvarez & Emory, 2006; Jurado & Rosselli, 2007; Nieuwenhuis et al., 2004; Stuss & Alexander, 2000).

**Real Life Implications of Executive Function Measurements**

As discussed, assessment of executive functions involves measuring processes that are relevant to performance and self-regulation in a complex environment. This implies another inherent problem with measuring executive functions, as some
problems experienced in daily life may not be evident on structured laboratory tasks. Simple, short tasks with explicit requirements ordered one at a time and initiated by the experimenter do not require the same type of planning and organization of multiple tasks over time that is needed in real life (Shallice & Burgess, 1991). The need for testing procedures that replicate this element has been widely recognized (Goel, Grafman, Tajik, Gana, & Danto, 1997; Levine, Dawson, Boutet, Schwartz, & Stuss, 2000; Shallice & Burgess, 1991; Wilson, 1993). Goel et al., (1997) found that patients with frontal lobe damage performed adequately on specific, local tasks but were impaired when the task required more global, higher-level structuring of the problem, such as planning, shifting, allocating effort and generating feedback.

There have been numerous observations of inconsistencies between scores on neuropsychological measures and difficulties in daily functioning (P. Anderson, 2002; Levine et al., 2000; Toplak, West, & Stanovich, 2013). Naismith et al. (2007) found that self-report and neuropsychological measures were concordant only for memory performance, but not for speed, learning or executive functions. Daily functioning could be influenced by compensatory strategies that are not available in the test situation, and performance may depend on specific environmental demands (Chaytor & Schmitter-Edgecombe, 2003). Chaytor, Schmitter-Edgecombe and Burr (2006) found that task measures only predicted 18-20% of the variance in everyday life functioning for a group of outpatients with various neurological conditions. This percentage was 51% when the non-executive components environmental cognitive demand and compensatory strategy were added.

Other variables may also differentially influence performance in a testing situation and the daily environment. For instance, tests done in ideal environments may
be less sensitive to effects of fatigue, distractions, emotions, and motivation; and high premorbid functioning and high IQ could mean that a large drop in daily functioning is experienced even with relatively small effects on specific tasks (Chaytor & Schmitter-Edgecombe, 2003).

The Components of Executive Function

Regardless of these difficulties with measurement, there is mounting evidence that a number of proposed subcategories of executive functions are useful to our understanding of cognitive mechanisms, behavior and performance. The most commonly cited executive functions are, according to Miyake et al. (2000): “Shifting between tasks or mental sets”, “updating and monitoring of working memory representations” and, “inhibition of dominant or prepotent responses” (p.54).

Using confirmatory factor analysis they found the three factors; Shifting, Updating and Inhibition, to be moderately correlated but also distinguishable in tasks aimed at testing complex problem solving (Miyake et al., 2000). Thus, they concluded that although they may to a certain degree tap common resources or constitute aspects of a common construct, there is value in studying these elements separately when seeking to describe the mechanisms of executive function (Miyake et al., 2000). In a meta-analysis, Alvarez and Emory (2006) summarized previous findings and identified the factors constituting executive function as: “inhibition and switching”, “working memory” and “sustained and selective attention” (p.17). As this article examines the role of inhibition, it is relevant to note that this construct is common to various models, and well established as an independent subprocess of executive functions.
Inhibition

Inhibition as an executive function refers to the capacity to overcome automatic, prepotent, routine, or dominant responses (Miyake et al., 2000; Shallice & Burgess, 1993; Shuster & Toplak, 2009). There are indications that inhibition could be valuable to understanding the relationship between affective symptoms, information processing and neurological impairments. In a recent review of the ways in which poor cognitive control causes a vulnerability to low affect (Joormann & Gotlib, 2010), inhibition had a central role. The authors suggested that the inability to disengage from, or inhibit, negative stimuli could be detrimental to the regulation of affect in multiple ways, leading to negatively biased appraisals and increased exposure to stressful stimuli (Gotlib & Joormann, 2010). Joormann and Gotlib (2010) reported that depression was associated with reduced inhibition of negative material. Furthermore, they found a correlation between emotion regulation strategies and inhibition (Joormann & Gotlib, 2010). Specifically, reduced inhibition was linked to more rumination, more expressive suppression and less reappraisal. Others have also linked impaired inhibition of negatively valenced material to depression (Goeleven, De Raedt, Baert, & Koster, 2006; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005). Impairment of inhibitory functions could therefore plausibly contribute to depression vulnerability.

It has further been suggested that inhibition has a central role in executive functioning. Inhibition has been found to correlate even with cognitive tests that were not viewed as executive function tasks, prompting Burgess et al. (1998) to suggest that this factor might be viewed as a «primary form of executive function» (p.555) that underlies problems on many other tests of cognitive functioning, and issues concerning social behavior, as well as being linked to the concept of general intelligence. A factor-
solution, highlighting the primacy of inhibition, was found when measuring every-day dysexecutive symptoms in a non-clinical population (Chan, 2001). Others have also suggested that the different executive functions require intact inhibition to operate optimally (Barkley, 1997; Bull & Scerif, 2001; Diamond, Carlson, & Beck, 2005). This could potentially explain the correlations between different executive functions (Bull & Scerif, 2001). As previously mentioned, executive functions become especially relevant in novel situations and in cognitive conflicts (Goel et al., 1997; Levine et al., 2000; Lezak, 1982; Shallice & Burgess, 1991). Inhibition of the habitual, competing response is therefore conceptually plausible as an essential part of cognitive control. Inhibition has been shown to be one of the earliest executive functions to develop in children (Jurado & Rosselli, 2007), so other capacities could theoretically build on the ability to inhibit. The primary role of inhibition is not undisputed; it has for instance been suggested that the purported effects of inhibition are better accounted for by differences in processing speed or working memory (Conway & Engle, 1994; Salthouse & Meinz, 1995).

**Inhibition in Daily Life**

The previously discussed methodological issues concerning executive functions in general are also significant obstacles to describing and operationalizing inhibition, and subsequently determining what role it has in depression. As cognitive inhibition cannot be observed directly, the presence and mechanisms of inhibition must be inferred from behavior (Klein & Taylor, 1994). The types of tasks or behaviors used to define the construct of inhibition have varied substantially, and there have been doubts about the coherence of the inhibition construct across these different tasks and behaviors (Klein & Taylor, 1994; Rabbit, 1997). MacLeod (2007) has suggested that a common
source of confusion in inhibition research is that “in cognition, inhibition is sometimes a measurable phenomenon, sometimes a theory about the cause of that phenomenon, and often both” (p.5). As discussed, much of the research into executive functions has used performance-based measures, and the degree of overlap with behavior in daily life is not well established (MacLeod, 2007). Experienced problems with inhibitory control and impulsivity have been associated with deficits on many typical neurological tasks (Chan, 2001; Enticott, Ogloff, & Bradshaw, 2006; Fino et al., 2014; Posner et al., 2002), but there have also been a lot of contradictory findings (Mcauley, Chen, Goos, Schachar, & Crosbie, 2010; Samyn, Roeyers, Bijttebier, Rosseel, & Wiersema, 2015; Toplak et al., 2013).

Burgess et al. (1998) identified inhibition as a factor that accounted for symptoms of disinhibition on a behavioral level, as well as on a range of executive function tests. Zhou, Chen and Main (2012) have pointed out that the concept of inhibition in daily life has a lot in common with the temperamental dimension effortful control, which is often assessed using the same types of tasks (e.g. Stroop and Go/No-Go; Zhou et al., 2012). Effortful control involves self-regulation of emotion, thought and actions (Rothbart, Sheese, & Posner, 2007) and has been found to moderate the effects of negative or positive affect, rumination and depressive symptoms (Verstraeten, Vasey, Raes, & Bijttebier, 2009). Low effortful control has been reported to constitute a vulnerability to depression (Verstraeten et al., 2009). Zhou et al. (2012) identified inhibition, in addition to attentional control, as a central common characteristic that unites the terms executive functions and effortful control from cognitive neuroscience and personality/temperament perspectives respectively. However, they also point out the important distinction between them; where inhibition in the framework of effortful
control typically centers on emotional or motivational responses, the executive control paradigm typically assesses the inhibition of “cold” cognitive responses (Zhou et al., 2012). Blair and Razza (2007) have observed that while executive functions are usually thought of as volitional control mechanisms, effortful control often involves automatic or non-conscious regulation. They found that these two concepts were moderately correlated and accounted for distinct variance in children's academic ability (Blair & Razza, 2007). It is a possibility that this reflects a difference between the kinds of regulatory mechanisms that are relevant in daily life, and those that are needed for specific, limited tasks.

**Multiple Inhibiting Processes**

Nigg (2000) outlined eight different inhibitory functions grouped into *executive*, *motivational* and *automatic* processes. In his taxonomy, the Stroop task, similar to the CWIT in the present study (Delis, 2001), was classified as a measure of interference control (Nigg, 2000). Other common performance measures of inhibition, such as the Stop task and the Go/No-Go were regarded measures of *behavioral inhibition*, whereas suppressing irrelevant ideas and intrusive thoughts were considered examples of *cognitive inhibition* (Nigg, 2000). There were all under the umbrella of executive inhibition, while motivational inhibition effects denoted responses to punishment and novelty (Nigg, 2000). This is noteworthy as everyday life involves demands on inhibition in a variety of ways. Friedman and Miyake (2004), using structural equation modeling, found that they could separate two kinds of cognitive inhibition. One factor involved inhibiting prepotent responses as well as resisting distraction interference (Friedman & Miyake, 2004). This was related to problems such as everyday cognitive failures and task switching (Friedman & Miyake, 2004). The other factor consisted of
the ability to resist proactive interference and related to, amongst other things, resisting unwanted intrusive thoughts (Friedman & Miyake, 2004). Thus, the type inhibition task may affect the results in studies of inhibition, and tasks might need to be carefully selected based on the goals of the study. It remains to be established whether certain types of inhibition are more relevant to depression, and whether different forms of inhibition are differentially related to various aspects of depression such as regulation of affect, and disability.

Even on tasks apparently measuring very similar inhibition capacities, discrepancies in performance have been found. Khng and Lee (2014) compared the performance of adolescent pupils on two inhibition tasks that are both supposed to require inhibition of an inappropriate prepotent response, the Stroop task and the Stop-Signal task, and found no correlation. They also reported that they were associated with different neural substrates and developmental trajectories, affected clinical subgroups differently and loaded on different factors (Khng & Lee, 2014). Attempting to explain this discrepancy, they distinguished between inhibiting intrusion by recently learned responses, and intrusion by habitual responses, the former being related to general intelligence (Khng & Lee, 2009). In their account, the Stop-Signal task is among the tests that measure intrusion of recently learned material, whereas Stroop measures intrusion from more entrenched material (Khng & Lee, 2009), a distinction supported by other researchers as well (Bull & Scerif, 2001). The CWIT, at least the pure inhibition condition, is therefore according to this account presumably an example of inhibition of well-learned responses as it is similar to Stroop (Delis et al., 2001). If the different types of inhibition are fragmented in this way, the utility of any one type of measure in predicting functioning in another setting becomes questionable. It is possible
that these various forms of inhibition are manifestations of one core cognitive process, but this form of hierarchy has not been adequately described (MacLeod, 2007), and thus inferences about what tests better capture this hypothetical core process cannot be made.

**Comparing Measures of Inhibition**

Direct comparisons between self-reported inhibition on BRIEF-A and performance on the CWIT for depressed adults were not found, but limited research was available for other populations, and for the relationship between task and scale measures of executive functions more generally. Chaytor & Schmitter-Edgecombe (2003) reviewed six studies on the ecological validity of various executive functioning tests, and concluded that over all, they did not significantly correlate with self-report measures. Samyn et al. (2015) have suggested that the two types of measurement reflect different underlying constructs, as revealed by a latent-variable analysis of multiple task and scale measures of executive function, including inhibition. For an adult sample with mild to moderate traumatic brain injury, self-reported cognitive problems predicted some objective performance in attention/processing speed, but depressive symptomatology was a much better predictor of objective executive functioning than self-reports (Schiehser et al., 2011).

There are some studies that have compared the CWIT, or Stroop (similar to the CWIT; Delis et al., 2001) with scale rating measures other than BRIEF. For example Heflin et al. (2011), who found no correlation between Stroop results and a behavioral rating of disinhibition for patients with mild cognitive impairment or dementia. Chan (2001) found a barely significant \( p = .05 \) and weak \( r = .18 \) correlation between Stroop results and the factor Inhibition, created from responses to the Dysexecutive Questionnaire, amongst adults from the general population.
There have also been studies of the relationship of BRIEF-A and a variety of task measures. Toplak et al. (2013) reviewed thirteen different studies of patients with mild cognitive impairment or dementia that had compared BRIEF-A scores with various objective measures of executive functions. They found a median correlation of \( r = .15 \) for the various possible scale correlations, described by the authors as “extremely weak” (p.136). This was also the case for other rating scales; the Dysexecutive Questionnaire and scale measures of impulsivity, indicating that the problem here was not just with BRIEF (Toplak et al., 2013). Toplak et al. (2013) explained these findings by proposing that the measures pertain to different levels of cognitive control. According to the authors, everyday functioning, rated on scales such as BRIEF-A, is a matter of the reflective mind (Toplak et al., 2013). At the reflective level, thinking style, goals, beliefs and decisions play a large part (Stanovich, 2011; Toplak et al., 2013). Objective measures on the other hand, supposedly examine the relatively narrow functions at the level of the algorithmic mind (Toplak et al., 2013), which reflects the functional efficiency of the cognitive mechanisms (Stanovich, 2011). Stanovich (2011) has argued that constrained executive function tasks cannot assess strategic control processes, which occur at the level of the reflective mind. Toplak et al. (2013) also noted that a common methodological problem in studies comparing measures of executive function, including inhibition, is the use of multiple measures yielding a large number of correlations, of which only some are reported.

**Relationship Between the CWIT and BRIEF-A**

Of the studies reviewed by Toplak et al. (2013), only a few looked at both BRIEF and the CWIT. Parrish et al. (2007) reported correlations between parent-reported BRIEF scores and the CWIT, but in a sample of children with epilepsy. They
found that while both could distinguish the epilepsy from the non-epilepsy groups, the only correlation with CWIT was the Metacognition Index from BRIEF (Parrish et al., 2007). V. A. Anderson, P. Anderson, Northam, Jacobs and Mikiewicz (2002) found moderate positive correlations (from $r = .27$ to $r = .48$) between self-corrections on the Contingency Naming Task, a test similar to Stroop (Riddle & Suhr, 2012), and scores on BRIEF, particularly on scales belonging to the Metacognition Index. The population in this study was children with brain disease (V. A. Anderson et al., 2002). Shuster and Toplak (2009) reported a negative correlation ($r = -.32, p > .01$) between the Inhibit scale on BRIEF self-report and Stroop interference in a non-clinical population. This implies that more interference on Stroop was actually related to fewer self-reported difficulties on BRIEF (Shuster & Toplak, 2009). Post-hoc analyses revealed that the number of self-corrections positively correlated with Stroop interference score, but negatively with BRIEF scores (Shuster & Toplak, 2009). The authors suggest that self-corrections may be an example of something that contributes to good inhibition scores on self-reports but poor performance scores (Shuster & Toplak, 2009). They also found that performance measures of other types of inhibition significantly predicted responses on BRIEF independently of Stroop, suggesting the different measures might be assessing independent aspects of performance (Shuster & Toplak, 2009). Hummer et al. (2011) also reported a negative correlation between Stroop Interference and the Inhibit scale from BRIEF ($pr(35) = -0.37, p = .02$), when controlling for gender. Their sample consisted of adolescents that had attention deficit hyperactivity disorder (ADHD) and/or disruptive behaviour disorders and healthy controls (Hummer et al., 2011).

In a study not included in the review by Toplak et al. (2013), Lalonde, Henry, Drouin-Germain, Nolin, and Beauchamp (2013) reported that amongst typically
developing adolescents, the CWIT did not predict results on parent-reported BRIEF. Interestingly, their virtual-reality version of the Stroop test did, however, leading the authors to suggest that it is a matter of ecological validity (Lalonde et al., 2013).

In summary, the reported correlations between BRIEF and the CWIT (or similar tasks) have been at best partial and moderate, and the direction of the relationship is inconsistent. Findings regarding the relationships between specific subscales on the two tests have also been inconsistent. The two kinds of test may be sensitive to different pathologies, as the discussed research has used widely varying populations. In the study by Samyn et al. (2015) only the objective measure was able to identify children with ADHD, or autism spectrum disorder. V. A. Anderson et al. (2002) found that the BRIEF-profile was distinct for children with focal frontal lesions when compared to more diffuse brain damage, whereas the objective measures could not. The correlation between the two types of measures might be determined in part by the clinical picture of the participants. It has been suggested that the correlation between task measures and daily problems may be higher for clinical populations, for whom the symptoms are more obvious, than for the general population (Chan, 2001). In line with this, Heinonen et al. (2013) found that BRIEF correlated with task measures of executive functions, including Stroop, for term-born control participants but not for very low birthweight adults. For the latter group, only parent ratings correlated with objective measures (Heinonen et al., 2013). With so many different populations and measures used, it's therefore not possible to draw any conclusions about adults with MDD. The fact that there have been reports of poor correspondence between tests, and differing underlying factor solutions, might indicate a need for a great degree of specificity when comparing findings.
The present study did focus on a specific group, MDD patients at one year after their first depressive episode. Previous studies of this sample by Schmid and Hammar (2013a, 2013b) have indicated that a deficit in cognitive inhibition as measured by the CWIT is present during first episode MDD and persists one year after initial diagnosis, despite symptom reduction (Schmid & Hammar, 2013b). The patients were particularly impaired on the inhibition condition with an added demand on cognitive flexibility (Inhibition/Switching; Schmid & Hammar, 2013b). Performance on the Inhibition/Switching condition was also worse at both testing times for the group that relapsed compared to those who did not (Schmid & Hammar, 2013b). There was no relationship between cognitive flexibility measured by other tasks and relapse or persistent impairment, suggesting inhibition is of primary interest (Schmid & Hammar, 2013b).

Groups that relapsed or experienced little or no change in their symptoms had a higher rating for illness severity at both testing times, but the effects of illness severity were controlled for when the relationship between cognitive function and relapse was assessed (Schmid & Hammar, 2013b). The correlation between illness severity and inhibition impairment was not significant, indicating that the impact of inhibition was not a result of illness severity (Schmid & Hammar, 2013b). Schmid & Hammar (2013b) also found that the slower reaction times on the inhibition task were not simply the result of lower processing speed.

The Current Study

The present study examined the relationship between self-reported and objective measures of inhibition at a one-year follow up of participants after they had presented with their first episode of depression. The goal was to investigate whether BRIEF-A
measures corroborated the results obtained using the CWIT which have indicated a lasting executive function deficit for previously depressed patients, especially in the area of inhibition (Schmid & Hammar, 2013b). Another objective was to see how the task measure of inhibition correlated with self reported general executive functioning and inhibition. I also wanted to know if the relationship between self-reported inhibition deficits and relapse was similar to that of task measured inhibition and relapse at follow up. Three questions were addressed:

1. Do participants diagnosed with MDD have a higher score on BRIEF-A than controls?
2. Do self-reported problems with general executive functioning, and cognitive inhibition specifically (BRIEF-A), correspond to the objective measure of cognitive inhibition (D-KEFS) for all groups?
3. Do those participants who experienced relapse within a year have a higher self-reported (BRIEF-A) deficit in inhibition than those who did not?

As previous analysis of the same participants using objective test measures have shown a persistent deficit in inhibition for the patient group (Schmid & Hammar, 2013b), it was expected that this would also be evident on self-report measures of the same cognitive construct. It was therefore hypothesized that the scores on BRIEF-A would be higher for depressed participants (Hypothesis 1) and that the two measures would be positively correlated (Hypothesis 2). Furthermore, a larger deficit in inhibition was detected amongst patients who experienced a relapse, both at initial testing and at a one-year follow-up. It was therefore hypothesized that patients who experienced a relapse would also have a higher score on self-reported inhibition problems as measured by BRIEF-A at one year follow up compared to those who did not relapse (Hypothesis 3).
Method

The current study is an extension of a larger longitudinal study of cognitive functioning in depression (Schmid and Hammar 2013a, 2013b). It is a crossectional analysis of data collected at a one-year follow-up. The participants have therefore been tested at two times, but the present analyses focused on the results from the second testing, a year after participants presented with their first depressive episode. Results will be discussed in the context of previous findings using this sample.

Participants

Fifty-three participants (27 male, 26 female) between the ages of 20 and 42 ($M = 27.1, SD = 5.3$) were included in the analyses. They consisted of 26 patients who had met the criteria for MDD one year previously and 27 control subjects. See Table 1 for descriptive data. The participants included in this study were tested at two times; first at inclusion (T1), then one year later (T2). The present analysis concerns the results from T2, and used only those participants for whom the relevant data was available. This means that there were slightly fewer cases included in this analysis ($N = 53$) than at T1 ($N = 60$), due to dropout and lacking data.

Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient group ($n = 26$)</th>
<th>Control group ($n = 27$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>Age</td>
<td>26.12</td>
<td>5.55</td>
</tr>
<tr>
<td>Education (years)</td>
<td>13.92</td>
<td>1.71</td>
</tr>
<tr>
<td>Males/females</td>
<td>13/13</td>
<td>-</td>
</tr>
<tr>
<td>MADRS score</td>
<td>9.72</td>
<td>5.54</td>
</tr>
</tbody>
</table>

Note. MADRS = Montgomery Åsberg Depression Rating Scale (Montgomery & Åsberg, 1979). MADRS scores not reported for healthy controls without symptoms of depression.
Inclusion. Inclusion criteria for the patient group at T1 was meeting the DSM-IV (2000) criteria for a unipolar first-episode MDD diagnosis, as assessed using the MINI-International Psychiatric Structural Interview (Leiknes, Leganger, Malt, & Malt, 1999). In addition, a minimum score of 20 on the Montgomery Åsberg Depression Rating Scale (MADRS), indicating a moderate to severe depression (Montgomery & Åsberg, 1979), was required. Patients who reported having previously experienced serious symptoms of depression, or who had been diagnosed with or treated for depression were excluded. As were patients with known brain damage, severe somatic disorders, alcohol and/or substance abuse, those who had been treated with electroconvulsive therapy, were psychotic or had experienced psychosis earlier in life.

Participants were recruited through primary health care. Doctors and psychologists informed patients about the study and determined who qualified for inclusion based on the criteria. The study coordinator then contacted those who were considered appropriate for inclusion and who consented to participate. At inclusion in the study, the MINI (Leiknes et al., 1999) was administered by a trained clinical psychologist in order to get a broad evaluation of psychiatric illnesses. Two patients reported symptoms of panic disorder, and met the criteria of a co-morbid diagnosis of panic disorder with agoraphobia.

All patients were outpatients. Some received either medical treatment (13.3%), psychological treatment (30%) or both (33.3%), and some (23.3%) received no treatment. Fourteen patients were prescribed antidepressant medication. Twelve patients were prescribed selective serotonin reuptake inhibitors (SSRIs; Cipralex, Citalopram); one used a serotonin noradrenaline reuptake inhibitor (SNRI; Effexor), and one used a tetracyclic antidepressant (TeCA; Remeron/Mirtazapine).
A control group \((n=30)\) was matched to the patients at T1, based on gender, age and years of education (within a ±2 year limit). IQ scores were similar for the patient group \((M = 118.53, SD = 8.12)\) and the control group \((M = 120.97, SD = 8.23)\). Control participants were 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 controls were interviewed to rule out those who reported a history of mental disorder, brain damage, alcohol abuse or substance abuse.

**Follow-up.** At T2, one year later, the mean MADRS score for patient group was 9.72, indicating minimal symptoms of depression, and no need of treatment (Hawley et al., 2002). Of the 26 patients included in the present analysis, nine participants were using medication at T2, of which eight used SSRIs (Cipralex, Citalopram) and one used an SNRI (Venlafaxine).

At T2, the patients were grouped based on whether or not they had experienced a relapse since T1. The sample was therefore divided into a *relapse group* \((n = 10)\), a *no relapse group* \((n = 7)\) and a *no change group* \((n = 2)\). The relapse and no relapse groups had a mean score on MADRS below 10, indicating that at the time of testing, depression severity for these groups was low and treatment not required (Hawley et al., 2002). The no change group reported experiencing their depression as more chronic, with short periods (lasting days or weeks) of minor symptoms. At follow-up, they had a mean MADRS score of 18, indicating a mild to moderate depression requiring treatment (Hawley et al., 2002). There were no major differences between the relapse group and the no relapse group regarding treatment variables across T1 and T2.
Measures

The Delis-Kaplan Executive Function System. The participants were assessed using the The Delis-Kaplan Executive Function System (D-KEFS; Delis et al., 2001) at T1 and T2. The D-KEFS is a neuropsychological battery made up of nine tests that can be used on their own or together with other parts of the set. It contains measures of verbal and non-verbal executive function, and assesses the function of more basic cognitive processes as well as executive functions, in order to distinguish the performance of each (Delis et al., 2001). A large number of studies with varied clinical populations have established that the D-KEFS tests are sensitive to impairments in executive functions and have adequate reliability compared to other executive measures (Delis et al., 2004).

The Color Word Interference Test. The test from the D-KEFS that was used for analysis in in the present study was the Color Word Interference Test (CWIT). It is based on the classic Stroop test developed to study verbal interference effects (Delis et al., 2001). There have been numerous investigations of the reliability and validity of Stroop, showing good reliability and sensitivity to executive deficits, even though some practice effects have been reported (Homack & Riccio, 2004). The CWIT is intended to measure the ability to inhibit dominant, perseverative and impulsive verbal responses as well as requiring cognitive flexibility. It is made up of four conditions. In the Color Naming condition (C), the subject is presented with color patches and asked to name the color as fast as they can. In Word Reading (W), they are asked to read a word written in black, as fast as possible. The third condition, Color Word (CW), a measure of inhibition, is the classic Stroop condition, where participants are presented with color names written with different color fonts and asked to name the ink color. This requires
inhibition of word reading. In the fourth condition, Inhibition/Switching (IS), the subject has to switch between saying the color and reading the word depending on whether the word is in a frame, requiring set shifting as well as inhibition (Delis et al., 2001; Lippa & Davis, 2010; Swanson, 2005). The IS condition is assumed to be more difficult, although Lippa & Davis (2010) have shown that this is not always the case.

Faster reaction times on the CW and IS conditions are thought to reflect a better ability to inhibit interference and switch between mental sets (Delis et al., 2001).

**The Behavior Rating Inventory of Executive Function—Adult Version.**

Participants were also given a Norwegian translation of the Behavior Rating Inventory of Executive Function—Adult Version (BRIEF-A; Nicholas & Solbakk, 2006). The adult version is based on the original BRIEF (Gioia, Isquith, Guy & Kenworthy, 2000), created to measure childrens’ executive functioning as rated by their parents and teachers. The BRIEF-A contains a self-report form as well as an informant-report (Roth et al., 2005), but only the self-report was used in the current study to assess self-reported executive functioning in daily life.

The BRIEF-A self-report contains 75 items that assess the respondent on nine clinical scales and three validity scales. For each of the 75 items, the respondent marks whether they experience the statement as true frequently, sometimes or never. The nine clinical scales reflect distinct and empirically validated constructs that capture different facets of executive functioning. They are: Inhibit, Shift and Emotional Control, which grouped together are referred to as the Behavioral Regulation Index; and Self Monitor Initiate, Working Memory, Plan/Organize, Task Monitor and Organization of Materials, which make up the Metacognition Index. The overall summary score is the Global Executive Composite (GEC). BRIEF-A has been used with a wide range of disorders to
assess executive functioning in adults between 18 and 90, and its reliability, validity and utility have been established empirically (Roth et al., 2005).

**Inhibition.** The Inhibit scale in BRIEF (Roth et al., 2005) consists of eight questions meant to capture «the ability to inhibit, resist or not act on impulse» (p.20). It can manifest as inappropriate, unsafe or disruptive behavior, a tendency to interrupt others and problems on delayed response tasks (Roth et al., 2005). Poor behavioral inhibition is often observed in ADHD and after traumatic brain injury (Roth et al., 2005). Some example questions from this scale are: item 16, «I have trouble sitting still»; item 43, «I make decisions that get me into trouble (legally, financially, socially)»; item 55, «People say that I am easily distracted» and item 73, «I am impulsive» (Roth et al., 2005).

The participants were also evaluated on a comprehensive test battery that included measures of IQ and other standardized experimental tests. They were not analyzed in the present study, but have been discussed previously (Schmid & Hammar, 2013a, 2013b).

**Testing Procedure**

Testing took place at the Institute of Biological and Medical Psychology, University of Bergen, Norway, and was administered by a trained senior test technician. The test technician was not blinded to which participants belonged to control group and which were patients, because of the nature of the recruitment procedure. Testing occurred in the same sequence for all participants, took about four hours to complete and was done during regular work-hours.
Ethical Considerations

The Regional Committee for Medical Research Ethics and The Norwegian Data Protection Authority has approved the study. The study was performed in accordance with the Helsinki Declaration of the World Medical Association Assembly. All participants gave their informed consent to participate at inclusion. The participants diagnosed with MDD were offered the normal treatment independently of the study.

Scoring and Data Analyses

For all analyses, raw scores were used. For both conditions on the CWIT, this consisted of the time taken to complete the tasks in seconds. For BRIEF, each item was converted to a score, with 1 corresponding to Never, 2 corresponding to Sometimes and 3 corresponding to Often. These were then summarized to produce the raw score. The statistical data analysis was done in the Statistical Package for the Social Sciences (SPSS) version 22. Effect sizes were calculated using the formula $r = z / \sqrt{N}$.

Results

The data violated assumptions of normality. Kolmogorov-Smirnov and Levene's test were significant at the .01 level for the GEC and Inhibit scales of BRIEF-A. CWIT Condition 4 had a very high level of kurtosis (6.81) and high positive skewness (1.75). Non-parametric tests were used. The results for the three hypotheses are discussed in turn.

Hypothesis 1

The first hypothesis was that previously depressed participants would have a higher score on the Global Executive Composite (GEC) and Inhibit scales on BRIEF-A than controls. See Table 2 for an overview of the raw scores and T-scores.
A Mann-Whitney U test revealed significant differences between the patient group ($Md = 116$, $n = 26$) and the control group ($Md = 79$, $n = 27$) on the GEC, $U = 39$, $z = -5.55$, $p = .00$, $r = - .76$. There was also a significant difference between the patient group ($Md = 11$, $n = 26$) and the control group ($Md = 9$, $n = 27$) on the Inhibit subscale, $U = 176$, $z = -3.164$, $p = .00$, $r = - .43$.

Table 2

<table>
<thead>
<tr>
<th>Score</th>
<th>Patient group ($n = 26$)</th>
<th>Control group ($n = 27$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>BRIEF-A GEC (raw)</td>
<td>119.19</td>
<td>23.58</td>
</tr>
<tr>
<td>BRIEF-A Inhibit (raw)</td>
<td>11.73</td>
<td>2.96</td>
</tr>
<tr>
<td>BRIEF-A GEC T-score</td>
<td>57.27</td>
<td>11.26</td>
</tr>
<tr>
<td>BRIEF-A Inhibit T-score</td>
<td>49.27</td>
<td>9.88</td>
</tr>
<tr>
<td>CWIT (CW)</td>
<td>47.62</td>
<td>1.33</td>
</tr>
<tr>
<td>CWIT (IS)</td>
<td>56.73</td>
<td>2.33</td>
</tr>
</tbody>
</table>

*Note.* GEC = Global Executive Composite. CWIT = Color Word Interference Test. CW = Color Word. IS = Inhibition/Switching. BRIEF-A T-scores $\geq 65$ considered clinically significant. For T-scores, $M = 50$, $SD = 10$ (Roth et al., 2005).

A look at the T-scores of the two groups revealed that nine cases (34.6%) in the patient group, and none of the controls, had a GEC score of 65 or, the cutoff score for clinical significance (Roth et al., 2005).

Additional analyses of the change in BRIEF-A raw scores from T1 and T2 were done to assess the stability of these reported problems with inhibition and executive functioning across one year. T1 BRIEF-A data was only available for 31 cases, of which 18 were patients and 13 controls. A Wilcoxon Signed Rank Test revealed a statistically significant reduction in the GEC scores from T1 ($Md = 94.00$) to T2 ($Md = 90$), $z = -2.11$, $p = .04$, with a small to medium effect size ($r = .27$).
An examination of the subscales using the Wilcoxon Signed Rank Test revealed that the only significant change was on the scales that make up the Metacognition Index, $z = 3.18, p = .001$, which decreased from T1 ($Md = 53$) to T2 ($Md = 50$), with a large effect size ($r = .57$). Splitting the group into control and patients revealed that the change on the GEC measured by the Wilcoxon Signed Rank Test was significant for the patient group ($n = 13$), $z = -2.48, p = .013$ but not for the control group ($n = 18$), $z = -1.92, p = .05$. It is possible that splitting the groups this way resulted in insufficient power, as the total number of cases was only 31. These analyses of change scores constitute a tentative indication that scores on BRIEF-A were not completely stable after one year. It was only the scales subsumed by the Metacognition Index that changed significantly. It appears that the scores changed more for the patient group, but this is speculative, as there are probably too few data points to examine the statistical significance of this difference in change scores reliably.

The BRIEF-A Inhibit and GEC scores were compared with the depression symptoms, as measured on MADRS. Spearman's Rank Order Correlation revealed a significant correlation between the GEC and MADRS scores, $r = .63, n = 25, p = .001$. There was no significant correlation between MADRS scores and the BRIEF-A Inhibtion scale, $r = .312, n = 25, p = .13$

**Hypothesis 2**

The second hypothesis was that self-reported problems with cognitive inhibition (BRIEF-A) would correlate with the objective measure of cognitive inhibition (CWIT) for all groups.

The relationships between the Inhibit scale on BRIEF-A and reaction time on Condition 3 (Inhibit) and Condition 4 (Inhibit/switch) of the CWIT were investigated
using Spearman's Rank Order Correlation. See Table 3 for correlations.

There was no significant correlation between the Inhibit scale and reaction time on Condition 3, \( r = .05, n = 53, p = .75 \). There was also no correlation between the Inhibit scale and Condition 4, \( r = .13, n = 53, p = .35 \). There was a significant positive correlation of medium effect size between the GEC on BRIEF-A and condition 3, \( r = .30, n = 53, p = .03 \). There was also a significant positive correlation between the GEC and condition 4, \( r = .37, n = 53, p = .006 \). Adjusting for multiple correlations using a Bonferroni correction, only the correlation between Condition 4 and the GEC were significant at the required .01 level, indicating a positive correlation between the total number of self-reported problems with executive functioning and longer reaction times on the Inhibition/Switching condition of the CWIT.

Table 3

*Correlations (Spearmans rho) between raw scores on the BRIEF-A and the CWIT for the whole sample (N = 53).*

<table>
<thead>
<tr>
<th>BRIEF-A Scale</th>
<th>Condition 3 (CW)</th>
<th>Condition 4 (IS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GEC</td>
<td>.297*</td>
<td>.370**</td>
</tr>
<tr>
<td>Inhibit</td>
<td>.045</td>
<td>.132</td>
</tr>
</tbody>
</table>

*Note. GEC = Global Executive Composite; CW = Color Word; IS = Inhibition Switching.*

*\( *p < 0.05 \) (2-tailed). **\( *p < 0.01 \)(2-tailed).*

**Hypothesis 3**

The third hypothesis was that those participants who had relapsed within a year would have a significantly higher self-reported (BRIEF-A) deficit in inhibition at T2 than those who didn't.

A Mann-Whitney U test revealed no significant differences between the relapse group (\( Md = 133.5, n = 10 \)) and the no relapse group (\( Md = 105, n = 7 \)) on the GEC
scores, $U = 26, z = -0.878, p = .42$. There was also no significant difference between the 
relapse group ($Md = 10.5, n = 10$) and the no relapse group ($Md = 12, n = 7$) on the 
Inhibit subscale, $U = 30, z = -0.45, p = .67$. The low number of participants means there 
is a greater chance of type II error for this test.

In the relapse group, 50% had a GEC T-score above 65, in the no relapse group 
28.6% had a GEC T-score of more than 65.

An exploration of the relationship between BRIEF-A scores at T1 and relapse 
was severely limited by the sample size, as BRIEF-A data was only available for five 
cases in the relapse group and three cases in the no relapse group at T1. For these, the 
difference between the relapse group ($Md = 115, n = 5$) and the no relapse group ($Md = 
131, n = 3$) in T1 GEC scores was non-significant, $U = 5.5, z = -0.60, p = .57$.

**Discussion**

The aim of this article was to examine persistent impairment of executive 
function, particularly inhibition, in first episode depression. Scores from the Behavior 
Rating Inventory of Executive Function—Adult Version (BRIEF-A) and the Color 
Word Interference Test (CWIT) from a one-year follow up were analyzed. The results 
supported a hypothesis of significantly elevated BRIEF-A scores for the patient group 
despite symptom remission, indicating persistent self-reported problems with inhibition 
and general executive functioning. This was consistent with findings of persistently 
impaired performance on the CWIT of inhibition in the same sample (Schmid & 
Hammar, 2013a). There were, however, significant differences between the two 
measures. There was some support for the hypothesis of a correlation between BRIEF-A 
and CWIT measures of executive function, but this was only the case for the BRIEF-A 
General Executive Composite (GEC) scale and the Inhibition/Switching (IS)
condition of the CWIT. There was no significant correlation of between the Inhibit scale on BRIEF-A and inhibition as measured by CWIT. There was no support for the hypothesis of a significant difference between the BRIEF-A scores of the relapse group and the no relapse groups, even though CWIT measured inhibition was persistently worse for the relapse group (Schmid & Hammar, 2013b). These findings will be discussed in turn.

Scores on BRIEF-A

There was support for the first hypothesis; the patient group reported significant problems with executive function, including inhibition, despite symptom remission. The effect size was large for the GEC and moderate for the Inhibit scale.

Some additional analyses were done to address the stability of the self-reported deficit, and the independence from affective symptoms. It was apparent that the scores decreased somewhat from T1 to T2, although the effect was small. It was also mainly the items subsumed by the Metacognition Index (MI) that improved. The MI score reflects the ability plan, organize and approach problems systematically and consists of five clinical scales that do not include Inhibit (Roth et al., 2005). The scores appeared to have improved more for the patient group, several of whom have gone from being in a depressive episode to no longer experiencing a depressive episode, although a larger data set is necessary to adequately investigate whether this group difference in change scores is significant. Some improvement has also been reported for CWIT scores (Schmid & Hammar, 2013b), with performance on the Color Word condition improving significantly for the no relapse group and Inhibition/Switching scores improving significantly for the control group.
There was a significant correlation between the patients' MADRS scores and GEC scores, but not the Inhibit scale. Despite the mean score for MADRS being at a level interpreted as remission (Hawley, Gale, & Sivakumaran, 2002), 10 out of 26 cases still had a score of 11 or above, indicating mild depression, and one case had a score of 22, just above the cutoff for moderate depression. This indicates that some influence of low-level residual symptoms on the GEC cannot be ruled out. It is also possible that persistent problems with executive functioning could lead to low-level symptoms associated with depression.

It was noted that nine of the patients, but none of the controls, had a BRIEF-A T-score of 65 or more, indicating clinically significant elevation (Roth et al., 2000). This allows for the possibility that a subset of MDD patients experience more significant problems with executive functions. McIntyre et al. (2013) suggested executive deficits were present in about 20-30% of depressed individuals. In this study, about 34.6% have clinically significant scores on BRIEF-A.

In summary, some of the self-reported problems with executive functioning in daily life appear to improve somewhat after symptom remission, but BRIEF-A scores for the patient group nonetheless remain significantly higher than for controls. Scores on the Inhibit scales are also significantly different for the patient and control groups, and appear to be more stable across one year. There is a significant correlation between the GEC scale and MADRS scores, but not the Inhibit scale and MADRS scores.

**The Relationship between the BRIEF-A and the CWIT**

There was partial support for the second hypothesis. There was no correlation between self-reported inhibition measured by BRIEF-A and inhibition as measured by the CWIT. There was, however, a significant correlation between the
Inhibition/Switching condition in the CWIT and the GEC in BRIEF-A. Before a Bonferroni correction, the Inhibition condition was also significantly correlated with the GEC. This suggests that self-reported executive functioning in general was correlated with inhibition performance, but self-reported inhibition was not.

It is possible that with a larger sample, the Inhibit scale would also have reached significance. The fact that the GEC, but not Inhibit, was correlated with MADRS score could mean that the relationship between GEC score and CWIT performance was somehow related to low level affective symptoms, even if there was no direct correlation between CWIT and MADRS. It could also be that inaccurate self-perception affects scores on the self-report measure. Alternatively, the results may reflect some real difference between the underlying constructs being measured. If the results are replicated in other samples, they may reflect a notable difference between the CWIT and the BRIEF-A.

It could be argued that even the specific scales on BRIEF-A assess functioning more broadly than the CWIT task. A number of variables, such as motivation, goals, and situational variables may contribute to performance in a real world environment, as pointed out by for example Chaytor et al., (2006). Items on the BRIEF-A Inhibit scale refer to phenomena such as decisions, distractions and impulsivity, which all involve interactions with the environment. Certain issues, such as “... trouble sitting still” (Roth et al., 2005) are mostly noticeable in environments that demand specific behaviors. Even if two individuals share a common cognitive deficit, this might therefore present itself differently depending on their environment, resources and personality characteristics. Different individuals may also utilize different compensatory mechanisms (Chaytor et al., 2006). Some of these, such as self-correction (Shuster &
Toplak, 2009), may lead to slower reaction times on the CWIT but less apparent inhibition problems in daily life. Thus it is possible that the CWIT and BRIEF-A are sensitive to different manifestations of an inhibition deficit.

It might also be that the two measures do not measure the same underlying cognitive mechanism. The validity of the inhibition construct is weakened if the operationalization if the term depends on tests that do not correlate with each other. The convergent validity of inhibition as measured by the different measures is debatable. The discriminant validity of inhibition is called into question by the fact that the CWIT measure of inhibition correlates more with general executive functions (GEC) than with the Inhibit scale. Rabbitt (2004) has asserted that much of the divergence in the executive function literature can be attributed to the fact that the study of executive functions try to describe these functions at more than one level at once. He further argues that the proposed components of executive functioning, including inhibition, are actually just “descriptions of task demands” (p.1), and that performance on various purported executive tasks may be excessively task-specific (Rabbitt, 2004).

The fact that it was only the combined Inhibition/Switching (IS) condition that correlated with the GEC on BRIEF-A at a .01 level suggests that this condition might be more strongly related to the experience of executive function in daily life. The unique thing about this condition was the added demand of switching, or cognitive flexibility. As other measures of cognitive flexibility were not correlated significantly with depression in this sample (Schmid & Hammar, 2013a), it allows for the possibility that the higher difficulty or complexity of the task is the factor that makes it more sensitive to inhibition deficits than the pure inhibition condition (CW). As previously discussed, task characteristics such as complexity and novelty may be crucial when assessing
executive functions (Goel et al., 1997; Levine et al., 2000; Shallice & Burgess, 1991). A problem with this important aspect of the concept of executive functions is that novelty and complexity are task characteristics that would probably also be more sensitive to global reductions in processing efficiency (Rabbit, 2004). Based on the fact that performance on other tasks requiring effortful processing are intact, Hammar et al. have concluded that the observed impairments probably are not the result of higher demands. Schmid & Hammar (2013b) also ruled out processing speed as an explanation of the slower reaction times by subtracting the reaction times of the simple naming conditions from the inhibition scores (calculating the contrast score). There may still be other variables that affect the performance on this measure preferentially, such as the ability to learn from the preceding CW condition (Lippa & Davis, 2010).

It might also be the case that the mean performance on certain tests is not the most informative metric. Nieuwenhuis et al. (2004), have suggested that variability in performance between trials and between different tasks are more informative about executive dysfunction than the group means for each trial or any specific task. They have proposed that variability in performance can be elicited by variations in the task characteristics; specifically whether features of the task encourage strategic attention to demanding task characteristics and therefore encourage goal activation (Nieuwenhuis et al. 2004). In other words, they propose a more general process rather than the operation of specific executive domains to explain variability in results (Nieuwenhuis et al. 2004).

Another possibility is that instead of a different or more general mechanism, more specificity is needed. These findings are consistent with propositions that there are separate components of inhibition, allowing for the possibility that these two measures tap into different components of the same umbrella term as suggested by a number of
previously discussed studies (Bull & Scerif, 2001; Friedman & Miyake, 2004; Khng & Lee, 2009; Nigg, 2000).

**Relapse and No Relapse Groups**

No difference was found between those who had relapsed in the last year and those who hadn't, however it is important to note that the number of participants for which this data was presently available, was very low. With only seventeen cases, the analysis might lack the statistical power to justify rejecting the hypothesis that those who relapsed have more significant problems with self-reported inhibition. The median score of the relapse group was slightly (though not significantly) higher than the no relapse group, but the scores on inhibition were actually slightly (though not significantly) lower for the relapse group. A larger sample is needed to examine whether this reflects a real trend, or simply random variation as suggested by the present analysis. If it really is the case that CWIT is correlated with the risk of relapse, but not BRIEF-A, this is further reason to suggest there is some difference in the sensitivity of the measures or in the underlying process that is assessed. Knowing which measures of inhibition deficits are associated with risk of relapse could be valuable to developing screening tools aimed at identifying at-risk groups.

**Summary and Conclusions**

These results added to previous findings of persistent executive function impairments in first episode MDD by analyzing a self-report measure. Inhibition was of particular interest, as this was the executive deficit most strongly related to depression in previous studies (Schmid & Hammar, 2013a, 2013b). The relationship between the inhibition scores on CWIT and BRIEF-A is ambiguous.
There was some congruence; both the CWIT and BRIEF-A inhibition scores seem to reveal relatively persistent and stable inhibition deficits, despite remission from MDD. Even though further analyses indicated a small significant decrease in the GEC score, especially for the patient group, this was apparently not the case for the Inhibit scale, suggesting its relative stability. The Inhibit scale was also not significantly correlated with concurrent depressive symptoms, and neither were the CWIT scores at T2 (Schmid & Hammar, 2013b)

Despite this, the direct correlation between the two measures of inhibition was not significant. CWIT was furthermore related to the risk of relapse, whereas there was no indication that BRIEF-A was. The large variations in previous research, as well as the low correlation between BRIEF-A and CWIT in the current study raises questions about the construct validity of inhibition as defined by these two measures.

Determining the cause of the discrepancy might provide valuable insights about cognitive functioning in depression. Information about the patterns of impairment could perhaps be instrumental to expanding models of depression, or to identifying subgroups of MDD patients.

**Limitations**

A few methodological considerations impact the generalizability of these results. The inclusion criteria for participation were highly specific, with a relatively low age, no previous history of depression, outpatient status and little or no co-morbidity. While this is also one of the strengths of the study, the results may not extend to other groups. Some of the participants were receiving antidepressant medication and/or psychological treatment. Assessing the impact of these on BRIEF-A was beyond the scope of the present study, but CWIT scores did not appear to be affected by medication at follow-up
(Schmid & Hammar, 2013b). The average IQs of the patient \((M = 118.53)\) and control group \((M = 120.97)\) as measured at T1 were quite high (Schmid & Hammar, 2013a).

Low sample sizes may have contributed to falsely accepting null-hypotheses, particularly when comparing relapse groups. Low statistical power due to few participants may also be one of the reasons why only one correlation between BRIEF-A and CWIT reached significance.

Random sampling was not possible for this study. The control group was matched on important characteristics, but it cannot be completely ruled out that there are confounding variables not accounted for. Testing was not blinded.

It is not possible to draw conclusions about causality as it is a crosssectional study. The results do suggest that the cognitive impairment is not the result of long-term depressive illness. In lieu of information about pre-morbid performance, however, it is not possible to establish whether poor executive functioning, including inhibition, contributed to the onset of depression, results from the first episode or is caused by other variables. The positive correlation of MADRS scores with the BRIEF-A, but not with CWIT, suggests that the former may be more closely linked to residual depressive symptoms. The causal direction of this relationship is not possible to determine based on the current study.

It cannot yet be determined with certainty that the measured impairments on either measure really do reflect an underlying executive function or inhibition deficit in the way it has been described in common models. The fact that only two different tests of inhibition were analyzed limits any inferences about the underlying cognitive constructs. Although there seems to be a variable that is affecting task performance on the CWIT and executive function in daily life, it is still a matter of debate whether this
best explained by general or specific processes. The inherent problems with measuring executive functions (e.g. Alvarez & Emory, 2006; Hughes & Graham, 2002; Jurado & Rosselli, 2007; Nieuwenhuis et al., 2004; Stuss & Alexander, 2000) are relevant to the current study. The participants had been tested on the same task measures twice, although with one year in between sessions.

**Implications and Future Research**

The present findings corroborate previous results (Schmid & Hammar, 2013b) suggesting that the inhibition problems in first episode MDD persist despite remission from affective symptoms. This indicates the possibility of a trait-like vulnerability. Follow-up studies after more than one year could answer whether cognitive function is simply slower to normalize than affective symptoms, or if this impairment constitutes a more permanent trait. Prospective studies with participants that have not previously been depressed are needed in order to answer whether the deficit is present before the first episode, occurs simultaneously with, or is a consequence of this first episode.

More research is needed to determine that the difference between control and patient groups on BRIEF-A scores after remission are, like the CWIT scores (Schmid & Hammar, 2013b), independent of the concurrent low-level depressive symptoms, as the current analysis suggests a link between the BRIEF-A and MADRS scores. Similarly, the work of ruling out other cognitive, affective and demographic third-variables requires replication studies with various samples.

Correlations between the scale items and task performance were inconsistent, in line with some previous findings (Mcauley et al., 2010; Samyn et al., 2015; & Toplak et al., 2013), suggesting caution is needed when generalizing from performance on specific task measures to daily life, and when comparing studies that have utilized
different forms of measurement. It could be that the two approaches are not interchangeable, but complimentary in capturing a broader picture of inhibition difficulties in MDD. More than one type of assessment may necessary to assess cognitive functions in depression, as suggested by Burgess (1998). Research with precisely defined populations and multiple types of measures could establish what impact specific deficits have on more general problem solving and daily situations.

Conceptual and methodological issues relating to executive function measurement in general, such as poorly defined and divergent descriptions of the various processes, and variable approaches to measurement (Rabbitt, 2004), need to be resolved before any conclusions can be drawn about the underlying constructs measured by the CWIT and BRIEF-A in MDD. The relationship between BRIEF-A and informant-reports on BRIEF-A, as well as other scale measures and objective measures of inhibition performance could provide valuable information.

It remains to be established whether various measures of inhibition are equally predictive of relapse. CWIT performance can be linked to the rate of relapse beyond what is explained by symptom severity (Schmid & Hammar, 2013b), but the present analyses could not determine reliably whether this was the case for BRIEF-A. Highly specific populations and thorough assessment of possible confounding variables are probably necessary in order to establish specific relationships between cognitive function measures and clinical features such as the risk of relapse.

Future studies may also reveal whether the pattern of impairment is specific to MDD. Multiple studies using various precisely defined populations could establish the specificity of this impairment. Research with other populations could furthermore decide if inhibition deficits are present for most of those who become depressed; or only
for some, as suggested by McIntyre et al. (2013). It could be that this impairment is associated with more severe subtypes of depression, or that it only constitutes a predisposition to depression in combination with other variables.

Determining whether there is a link between cognitive inhibition and vulnerability to depression is only the first step to developing interventions. If future research shows further support for a statistically and clinically significant effect of inhibition in predicting depressive episodes, the reasons for this need to be explored. Cognitive inhibition may impact mood and or general functioning directly (Gotlib & Joormann, 2010), but it could also be symptomatic of more severe depression subtypes, or of more important cognitive, clinical or environmental characteristics. If inhibition does have a causal role, the mechanisms through which this occurs, whether by affecting regulation of emotions, thoughts or behavior; or the ability to perform daily tasks, need to be described. Research on specific interventions is needed to test whether the impairment, or its effects on disability and the chance of experiencing depressive episodes, can be ameliorated. In addition to extending theories of cognitive impairment and developing targeted treatments, describing the patterns of impairment also has a more immediate application; if there are more chronic subtypes of depression associated with cognitive impairment, or certain groups that are at risk due to reduced cognitive performance, measures of cognitive functions could constitute a helpful tool in identifying these. Early interventions might then be developed with the aim of improving long-term outcomes and psychosocial functioning.
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