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# The influence of cognitive control training on stress reactivity and rumination in response to a lab stressor and naturalistic stress

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ABSTRACT

Cognitive control impairments have been identified as an underlying mechanism for rumination, a key predictor of depression. Literature suggests that cognitive control training (CCT) targeting working memory functioning can increase effectiveness of existing antidepressant treatments to reduce rumination. However, it remains unclear whether CCT can also be implemented as a preventive intervention for depression, increasing resilience. For this purpose, at-risk undergraduate students (high trait ruminators) were allocated to a CCT or active control condition, consisting of 10 online training sessions. Working memory functioning was assessed preceding and following the training and reactivity to a lab stressor was assessed directly following training. Finally, at four weeks follow-up, brooding – the maladaptive form of rumination – was re-assessed in response to a naturalistic stressor (examination period). Although we did not find direct transfer effects of CCT on working memory functioning, increase in working memory functioning following CCT was related to post-training brooding and resilience levels. Moreover, participants receiving CCT demonstrated lower stress reactivity in the lab and a decrease in brooding following a naturalistic stressor at follow-up, indicating temporal stability of our findings. These findings suggest that CCT can be considered a promising preventive intervention to reduce stress reactivity and rumination.



Depression is an important mental health problem (Kessler & Wang, 2009; WHO, 2012), associated with major individual suffering and high societal costs (IsHak et al., 2013; Luppa, Heinrich, Angermeyer, Konig, & Riedel-Heller, 2007). Current treatments of depression show rather limited success concerning effect size and long-term outcome (for a review, see Cuijpers, Andersson, Donker, & van Straten, 2011). This suggests that these interventions fail to influence key depressogenic mechanisms. Hence, identifying and changing such mechanisms is a major challenge for depression research.

Rumination – a maladaptive emotion regulation strategy that is characterized by the tendency to respond to a stressful event with repetitive, perseverative, and negative thinking – has been identified as an important risk factor for depression, influencing the course of a current episode as well as predicting future depressive episodes (Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Moreover, rumination shows relative stability when taking into account changes in depressive symptomatology (Nolen-Hoeksema et al., 2008). In particular brooding – the depressive subtype of rumination that is characterized by a passive style of moody pondering, self-blame and criticism (Treynor, Gonzalez, & Nolen-Hoeksema, 2003) – has been linked to negative information processing biases (Joormann, Dkane, & Gotlib, 2006) and future depressive symptomatology (Treynor et al., 2003). Furthermore, brooding has shown to moderate the relation between stress and depressive symptomatology (Cox, Funasaki, Smith, & Mezulis, 2012).

Accordingly, several researchers have argued that targeting rumination and stress reactivity in therapy could be an important strategy to prevent the (re-)occurrence of depressive episodes as well as enhance treatment (e.g., van Vugt, Hitchcock, Shahar, & Britton, 2012; Watkins et al., 2011). In the current study, we sought to examine whether training cognitive control, a key mechanism implied in rumination, can be beneficial to reduce stress reactivity, rumination, and brooding in particular in an at-risk sample characterized by high rumination scores. We start by

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considering the relationship between cognitive control, rumination, and depression.

Cross-sectional (Davis & Nolen-Hoeksema, 2000; Joormann, 2006; Joormann & Gotlib, 2010) as well as prospective studies (Connolly et al., 2014; Demeyer, De Lissnyder, Koster, & De Raedt, 2012; Zetsche & Joormann, 2011) have consistently linked rumination to impaired cognitive control (for a review, see Joormann & D'Avanzato, 2010).[1] Importantly, cognitive control impairments have been identified in at-risk (Owens, Koster, & Derakshan, 2012), currently depressed (De Lissnyder, Koster, Everaert, et al., 2012), and remitted depressed populations (Vanderhasselt & De Raedt, 2009), and predict higher levels of rumination and depressive symptoms in response to stress (De Lissnyder, Koster, Goubert, et al., 2012; Zetsche & Joormann, 2011). Moreover, it has been suggested that cognitive control impairments reflect increased biological vulnerability to depression (i.e., hypofrontality), which through rumination and its detrimental effects (e.g., sustained negative mood) is thought to further increase cognitive *and* biological vulnerability for recurrent depression (for a conceptual framework on the relation between cognitive control impairments and increasing biological and cognitive vulnerability in recurrent depression, see De Raedt & Koster, 2010).

To examine whether cognitive control plays a causal role in depression vulnerability, experimental designs manipulating cognitive control and examining subsequent effects on stress reactivity and rumination are of crucial importance. In recent years, important progress has been made in this area, using modified working memory training tasks such as the adaptive Paced Auditory Serial Addition Task (PASAT; e.g., Siegle, Ghinassi, & Thase, 2007) to train cognitive control. During the adaptive PASAT, participants are presented with a stream of auditory presented digits and are instructed to indicate the sum of the last two digits, which relies on continuously updating working memory. Depending on the accuracy of the responses, the inter stimulus interval (ISI) would decrease or increase, modifying task difficulty. Siegle et al. (2007) demonstrated the added value of combining cognitive control training (CCT) with treatment as usual (TAU), which led to a greater reduction in rumination and depressive symptomatology compared to a TAU control group. These findings have recently been replicated and extended, showing a reduced need for outpatient services one year following the combined intervention (Siegle et al., 2014). Importantly, Siegle et al. (2014) argue that changes in depressive symptomatology are secondary to changes in rumination.

These findings suggest a causal role of cognitive control in depressive rumination and demonstrate an added value of combining CCT with regular treatment (e.g., Siegle et al., 2014). However, until now it remains unclear whether working memory based CCT can also be implemented for *preventive purposes*. Rumination forms an important predictor for depression, and at-risk populations – characterized by heightened levels of rumination – might benefit from CCT given that cognitive control impairments predict higher levels of rumination in response to stress (De Lissnyder, Koster, Goubert, et al., 2012; Zetsche & Joormann, 2011). Moreover, rumination is known to mediate the relation between stressful events and depressive symptomatology (Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013). Training cognitive control thus holds the potential to improve emotion regulation in the wake of stress as increased cognitive control might reduce the extent to which subjects respond to a stressful event with rumination, increasing resilience to depression. This would fit the recent plea to invest in preventive programs and innovative treatment delivery methods to increase the quality of mental health care in order to reduce the burden of mental illness (Kazdin & Blase, 2011). Hence, a main goal of this study is to explore whether CCT can be used to increase stress resilience in an at-risk population.

Furthermore, there are still a number of remaining questions about clinically oriented CCT studies using the adaptive PASAT. First, in above mentioned studies (Siegle et al., 2007, 2014), CCT consisted of the adaptive PASAT *as well as* the Wells' attention training, during which participants are instructed to focus on auditory stimuli (Wells, 2000). Therefore, it was not clear to what extent observed improvements in cognitive control are due to the PASAT training. However, since performance on the PASAT has been related to DLPFC activity (Lazeron, Rombouts, de Sonneville, Barkhof, & Scheltens, 2003) and pilot work indicates that stimulating the left DLPFC can increase therapeutic effects of CCT (Segrave, Arnold, Hoy, & Fitzgerald, 2014; but see Brunoni et al., 2014), it is plausible that an important part of the therapeutic effects reported in previous CCT studies can be attributed to the adaptive PASAT component. Moreover, Brunoni et al. (2014) have recently provided evidence for the effectiveness of the adaptive PASAT in absence of the Wells' attention training in reducing depressive symptomatology in a clinical sample. Given these findings, we will only use the adaptive PASAT as CCT.

Second, while previous CCT studies have compared training effects with a passive control group, the lack of an active control group with regard to the computerized training does not allow to rule out placebo effects. Related to the latter point, Calkins, McMorran, Siegle, and Otto (2015) demonstrated the potential of the combined CCT in reducing depressive symptomatology compared to an adaptive version of the Peripheral Vision task. Other researchers have proposed to use the adaptive Visual Search task as an active control group in working memory training studies (Harrison et al., 2013; Redick et al., 2013). During this visual search training (VST), participants respond to the orientation of a target letter in the presence of distractors. Task difficulty is modified based on individual performance levels. The adaptive component allows researchers to control for effects of performing a computerized training (Shipstead, Redick, & Engle, 2012) without the task being related to working memory functioning (Kane, Poole, Tuholski, & Engle, 2006; Redick et al., 2013). Furthermore, in contrast to tasks used in previous CCT studies (Calkins, Deveney, Weitzman, Hearon, & Siegle, 2011; Calkins et al., 2015), the VST allows researchers to check whether training progress was made in both conditions leaving only the specific content (i.e., whether or not targeting working memory) as the experimental manipulation. Given that this approach allows a more clear interpretation of training effects (Harrison et al., 2013; Redick et al., 2013; Shipstead et al., 2012), we used the VST as an active control group.

## 1. Current study

Cognitive training focusing on remediating cognitive control impairments shows potential as an intervention for depression given that previous studies have demonstrated that impaired cognitive control increases the chance of deploying rumination in response to stressful events. This is important, as rumination – and more specifically, brooding – have shown to predict the occurrence of future depressive symptomatology. The current study examined whether working memory based CCT can heighten resilience to stress and reduce rumination in the wake of stress. Undergraduate students showing a tendency to ruminate were followed over time as they approached their examination period. Participants were randomly allocated to a CCT or VST condition, the latter being the

[1] 'Cognitive control' refers to the broad definition of the concept under which different executive functions are situated, including shifting, inhibition, and information updating in working memory (Collette et al., 2005; Miyake et al., 2000). Several researchers (Joormann & Quinn, 2014; Siegle et al., 2007) have argued that training cognitive control to improve working memory (CCT) could be of interest for treating the neurobiological and cognitive impairments underlying depression. Therefore, we focus on modified working memory training tasks.