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Review

Cognitive control interventions for depression: A systematic review of findings from training studies

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HIGHLIGHTS

- Depression is associated with cognitive control impairments.
- Cognitive control training can elucidate the causal status of this risk factor.
- Cognitive control training may have therapeutic benefits.
- We critically review the extant literature.
- Cognitive control training shows promise in depression but more research is needed.

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ABSTRACT

There is a strong interest in cognitive control training as a new intervention for depression. Given the recent promising meta-analytical findings regarding the effects of cognitive training on cognitive functioning and depressive symptomatology, the current review provides an in-depth discussion of the role of cognitive control in depression. We consider the state-of-the-art research on how manipulation of cognitive control may influence cognitive and depression-related outcomes. Evidence for the effectiveness of cognitive control training procedures are discussed in relation to three stages of depression (at-risk, clinically depressed, remission) as well as the training approach that was deployed, after which the putative theoretical mechanisms are discussed. Finally, we provide ways in which cognitive control training can be utilized in future research.

Contents

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1. Introduction

Depression is the leading cause of disability worldwide, and is a major contributor to the global burden of disease (World Health Organization, 2012). Moreover, depression is one of the most common and debilitating psychiatric disorders with an estimated 8 to 20% of the population experiencing at least one depressive episode during their lifetime. Despite the availability of well-established psychological and pharmacological treatment options for depression, that have acceptable short-term effectiveness, various challenges in the treatment of depression remain. Major challenges are that relapse or recurrence rates after remission or recovery remain very high and tend to increase (up to 80%) with the number of episodes (Beshai, Dobson, Bockting, & Quigley, 2011). Moreover, there is a substantial proportion of patients who fail to respond to treatment (Thomas et al., 2013). Treatment-resistant and recurrent depressive episodes are strongly associated with poor psychosocial outcomes due to increasing social problems (e.g., elevated divorce rates) and financial problems (e.g., multiple sick leaves, unemployment).

A crucial idea is that current treatments insufficiently target key underlying vulnerability factors of depression, causing depression to remit insufficiently or, when remitted, to still act as a risk factor for new depressive episodes. Although cognitive impairments in concentration, memory, and attention were initially considered side effects of the affective problems, recent neurobiological as well as cognitive research indicates that diminished cognitive control over information in working memory may be a key psychological vulnerability factor (Joormann, Yoon, & Zetsche, 2007; Millan et al., 2012; Siegle, Ghinassi, & Thase, 2007). Information processing factors are thought to have proximal links with rumination, a key maladaptive emotion regulation strategy, that can in turn influence depressive symptoms (Joormann & D'Avanzato, 2010; Joormann & Vanderlind, 2014). Importantly, recent findings suggest that existing antidepressant treatments do not impact cognitive impairments in depression (Shilyansky et al., 2016).

Cognitive control involves executive processes that allow information processing and behavior to vary adaptively over time depending on current goals, rather than remain rigid and inflexible. These cognitive control processes include a broad class of mental operations including goal or context representation and maintenance, and strategic processes such as attention allocation and stimulus-response mapping. Miyake et al. (2000) have suggested that executive functions mapping cognitive control can be operationalized into three major, interrelated yet separable functions: mental set shifting (shifting), information updating and monitoring of working memory representations (updating), and inhibition of prepotent responses (inhibition). Joormann et al. (2007) have argued, based on the work of Hasher and Zacks (1979), that cognitive control processes play a crucial role in determining the content of working memory, conceptualized as a limited-capacity system for the temporary storage of information (Baddeley & Hitch, 1974; Jonides et al., 2008). Difficulties in exerting cognitive control over negative information operations could explain the proliferation of negative information in working memory (Joormann et al., 2007), directly linking cognitive control impairments to perseverative negative thinking (depressive rumination), a well-supported vulnerability factor for depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008).

There is converging evidence from psychopathology and neurobiological research to indicate that depression is associated with broad impairments on cognitive control tasks (for a recent meta-analysis, see Snyder, 2013). Moreover, across a variety of different tasks individuals at-risk for depression have also been found to display reduced cognitive control. For instance, cognitive control deficits have been observed in participants showing heightened trait rumination (e.g., Beckwé, Deroost, Koster, De Lissnyder, & De Raedt, 2014) and subclinical levels of depressive symptomatology (dysphorics; e.g., Derakshan, Salt, & Koster, 2009; Joormann, 2004; Owens, Koster, & Derakshan, 2012). Similarly, cognitive control impairments have been observed in a vast amount of studies exploring cognitive functioning in depressive patients (e.g., Deveney & Deldin, 2006; Goeleven, De Raedt, Baert, & Koster, 2006; Harvey et al., 2004; Levens & Gotlib, 2010; Merriam, Thase, Haas, Keshavan, & Sweeney, 1999; Murphy et al., 1999), and remain evident following remission from depression (e.g., Demeyer, De Lissnyder, Koster, & De Raedt, 2012; Levens & Gotlib, 2015; Paelecke-Habermann, Pohl, & Leplow, 2005; Vanderhasselt & De Raedt, 2009). Importantly, impaired cognitive control is mainly observed in at-risk samples when individuals are processing emotionally negative information (e.g., angry faces or negative self-referring words), whereas the impairments appear to be more broadly present in individuals that meet clinical levels of depression (Snyder, 2013). Furthermore, several studies suggest that cognitive control deficits are most apparent when engaging in rumination (e.g., Philippot & Brutoux, 2008; Whitmer & Gotlib, 2012). Research indicates that these impairments are not merely correlates of depression, but predict future rumination and the development of new depressive symptoms in prospective studies in healthy (e.g., Pe, Brose, Gotlib, & Kuppens, 2016; Zetsche & Joormann, 2011) and at-risk samples (e.g., Demeyer et al., 2012).

At the neuropsychological level, fronto-limbic disruptions are thought to play a crucial role in cognitive impairments involved in emotion regulation (for reviews, see Pizzagalli, 2011; Roiser, Elliott, & Sahakian, 2012). Key findings from neuroimaging studies have shown that depression is associated with disrupted brain activity in the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC) (Davidson, Pizzagalli, Nitschke, & Putman, 2002; Etkin, Gyurak, & O'Hara, 2013; Pizzagalli, 2011), with decreased activation in these