Cognitive Control in Depression: Toward Clinical Models Informed by Cognitive Neuroscience

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Abstract

Cognitive control dysfunctions are thought to contribute to the onset and maintenance of depression. However, the causes and nature of these dysfunctions remain unknown. Here, we critically review contemporary research on cognitive control in depression. We identify three main conceptual issues in this field: 1) uncritical use of the tripartite model; 2) reliance on descriptive explanations; and 3) lack of integration with emotional and motivational impairments. Recent advances in cognitive neuroscience offer possibilities to resolve these issues. We review this progress focusing on the ability to detect the need for control, the role of motivation, and the flexibility-stability balance. We propose that depression-related dysfunctions arise from issues in detecting when, how, and for how long to engage in goal-oriented processing. In conclusion, we argue that integrating advances in cognitive neuroscience into clinical research can help to move from a descriptive towards a more mechanistic understanding of cognitive dysfunctions in depression.

Keywords: depression; cognitive control; executive functions; motivation; anhedonia.

Introduction

Depression represents one of the leading causes of disability worldwide (Kessler & Bromet, 2013). This highly common disorder is linked to severe individual suffering and high societal costs (Kessler, 2012). Notably, the relapse and recurrence rates of depression remain high (Bockting, Hollon, Jarrett, Kuyken, & Dobson, 2015; Vittengl, Clark, Dunn, & Jarrett, 2007), indicating that improvement of existing treatments is urgently needed. To advance both theory and treatment of depression, it is crucial to uncover the mechanisms underlying hallmark features of depression. These mechanisms may include abnormalities in cognitive, emotional, and motivational processes which contribute to the onset and maintenance of depression (for recent reviews see: Admon & Pizzagalli, 2015; Crocker et al., 2013; Joormann & Vanderlind, 2014). One of the central cognitive abnormalities investigated in depression are cognitive control dysfunctions. These dysfunctions have become the focus of a rapidly increasing number of empirical and theoretical studies over more than a decade.

Cognitive control[1] refers to a set of mental processes that allow flexible adaptation of cognition and behavior in accordance with an individual's current goals (Braver, 2012; Friedman & Miyake, 2017; Shenhav, Botvinick, & Cohen, 2013). These processes are critical for goal-directed, non-automatic behavior and are found to be disturbed in a wide range of psychiatric disorders including depression (Millan et al., 2012; Snyder, Miyake, & Hankin, 2015). Three cognitive control processes that are most often investigated in relation to depression are: *inhibition* (overriding dominant or prepotent responses), *shifting* (switching between mental sets or tasks), and *updating* (adding or discarding of working memory contents) (Miyake et al., 2000; Miyake &

[1] Here we use the terms cognitive control, executive control, and executive functions interchangeably. We consider them to generally refer to the same set of processes. While the terms executive functions and executive control are mainly used in individual differences research, the term cognitive control is often used in cognitive neuroscience.

Friedman, 2012). Cognitive control dysfunctions are purported to represent a key vulnerability factor for depression (Joormann et al., 2007; Siegle et al., 2007). Dysfunctions in these processes have been observed in clinically depressed individuals (for a meta-analysis see Snyder, 2013), individuals with self-reported elevated levels of depressive symptoms (Derakshan, Salt, & Koster, 2009; Owens, Koster, & Derakshan, 2012), and patients with depression in remission (Demeyer, De Lissnyder, Koster, & De Raedt, 2012; Levens & Gotlib, 2015). These results from cross-sectional studies suggest that such impairments are not merely an epiphenomenon of a depressive mood or episode. Indeed, there is evidence that cognitive control may have a causal influence on depressive symptoms. Research indicates that improving cognitive control through training in depressed and at-risk populations helps to reduce depressive symptoms (Koster, Hoorelbeke, Onraedt, Owens, & Derakshan, 2017; Siegle et al., 2007). Also, longitudinal studies have provided initial evidence for the importance of cognitive control dysfunctions in predicting depressive symptoms (Demeyer et al., 2012; Pe, Brose, Gotlib, & Kuppens, 2015). Finally, cognitive control dysfunctions have also been linked to information-processing biases, increased emotional reactivity to stress, and difficulties to downregulate negative emotions (Joormann & Vanderlind, 2014; Koster et al., 2011; Siegle et al., 2007). Taken together, cognitive control represents an important construct in understanding vulnerability to depression.

Currently there is increasing recognition of the importance of cognitive control dysfunctions in depression, accompanied by a fast development of cognitive training procedures aimed at reducing depressive symptoms. We believe that now is the right time to take a step back and critically examine the current understanding of cognitive control in depression. Models and conceptualizations of cognitive control in depression have guided important discoveries of critical cognitive abnormalities. However, these models do not adequately address questions about causes and mechanisms of the depression-related cognitive control impairments. For example, it remains

unclear if depression is related to the general reduction of the ability to exert control, or it is more related to the problems in detecting when to engage in controlled processing and goal-oriented behavior, and how intensely to do so. Also, it is not clear if the cognitive control dysfunctions are general or specific to the processing of emotionally negative material. In short, the mechanisms behind these cognitive impairments remain unknown.

Recent advances in cognitive neuroscience have led to important improvements in understanding cognitive control mechanisms (for a broad overview see: Cohen, 2017). These advances offer a mechanistic view and plausible neurobiological substrates of cognitive control. The progress in research on depression-linked cognitive control dysfunctions notwithstanding, we advocate the view that future advances in clinical research should be informed by these novel developments in cognitive neuroscience. While current research and theorizing offer explanation at the descriptive level, there is a strong need for a mechanistic approach to dysfunctional cognitive control in depression. This will be a crucial next step in developing a more sophisticated understanding of cognitive control dysfunctions and may provide novel directions for treatment strategies. Hence, the aim of this paper is to bridge the current gap between clinical research and theories of cognitive control in depression and novel developments in cognitive neuroscience. In the following sections, we critically review the current state of theory and research on cognitive control in depression and provide an overview of recent developments in understanding cognitive control. By doing so, we aim to demonstrate how progress in cognitive neuroscience can be applied to research on depression to advance the understanding of cognitive control dysfunctions.

Cognitive Control and Depression

Theoretical Models of Cognitive Control in Depression

Contemporary cognitive models of depression (Disner et al., 2011) propose that genetic vulnerability in combination with adverse early experiences and stressful events (e.g., experiences

of loss) can result in depressogenic ways of processing emotional information. For instance, if someone has experienced low parental warmth in childhood, this person may develop beliefs that he/she is unlovable. Such core beliefs may determine how someone interacts with other people and stressful situations. This person may focus excessively on cues signaling that their current partner does not love them. This may result in difficulties to disengage from this type of information and regulating of the elicited thoughts and emotions. Indeed, research has documented specific emotional biases in cognitive processes of attention and memory in depressed and dysphoric individuals (for a review see: Everaert et al., 2012). Cognitive models have been put forward to explain such depressogenic information processing biases in terms of impaired disengagement of attention from negative stimuli (Koster et al., 2011) and cognitive control dysfunctions (Joormann, 2010; Joormann et al., 2007; Joormann & Vanderlind, 2014). These models are mainly descriptive and focused on detecting the processes that may be involved in depressogenic information processing.

The Impaired Cognitive Control account (Joormann, 2010; Joormann & Vanderlind, 2014; Joormann et al., 2007) is the most elaborate account linking cognitive control dysfunctions to depressive symptoms. Within this framework, cognitive control dysfunctions are defined as specific difficulties in controlling the contents of working memory (WM) (Joormann & Vanderlind, 2014). Following the tripartite model of cognitive control (Miyake & Friedman, 2012; Miyake et al., 2000), it is proposed that depression is related to decreased ability to limit the access of irrelevant negative information into working memory (*inhibition*) and a decreased ability to remove negative content that is no longer relevant from WM (*updating*). For example, negative cognitions about past failures that remain active in WM (e.g., "I failed my previous exam, why would I pass now") may interfere with current performance on a task (e.g., when one is preparing for the next exam). Reduced ability to perform inhibition and updating are theorized to further skew

information processing resulting in exaggerated processing of negative material and interrupting effective emotion regulation.

Several theoretical models have also elaborated on how cognitive control dysfunctions and depressogenic information processing biases contribute to the onset and maintenance of depressive symptoms. It is proposed that a decreased ability to exert cognitive control, particularly when processing negative information, underlies ineffective use of emotion regulation strategies to increase depressive symptoms (Joormann, 2010; Koster et al., 2011; Whitmer & Gotlib, 2013). Of particular importance in this context is the emotion regulation strategy called *rumination*, which refers to the tendency to engage in preservative negative thinking about the past and present and is closely linked to depressive symptoms (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). The indirect influence of cognitive control dysfunctions in processing negative information on depressive symptoms via changes in emotion regulation strategies has gained initial empirical support (Everaert et al., 2017; for a review see: Mor & Daches, 2015).

Theoretical models have also included neurobiological substrates of cognitive control dysfunctions in depression (Disner, Beevers, Haigh, & Beck, 2011). While cognitive control processes are supported by a wide range of interacting brain regions and circuitries, most research in the field of depression has been focused on two regions in particular. That is, depression has been linked to abnormal activation of the dorsolateral prefrontal cortex (dlPFC) and the anterior cingulate cortex (ACC). These two regions are strongly associated with cognitive control (Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Gotlib & Hamilton, 2008; Pizzagalli, 2011). Current theoretical models frequently relate diminished cognitive control in depression to hypoactivity of the dlPFC (but also ventral and medial PFC) and to hypoactivity of the ACC (Disner et al., 2011; Joormann et al., 2007). The main hypothesis is that depression-related hypoactivity in these areas is related to the inability to effectively regulate negative affect.

In summary, cognitive control dysfunctions are assigned a central role in depressogenic information processing and emotion regulation difficulties. These dysfunctions are currently understood as an impaired (i.e. attenuated) ability to engage cognitive control processes such as inhibition or WM updating. In the next section, we describe the empirical research that was guided by aforementioned theoretical models.

Research Findings on Cognitive Control in Depression

Behavioral studies

Depression-linked dysfunctions in cognitive control processes have been examined in the case of processing both neutral and emotional material. Standard cognitive control tasks present stimuli that are neutral in emotional tone. For example, the Stroop task presents a series of words (e.g. red, green, blue) printed in different colors (e.g. red, yellow, blue) and prompts participants to name the ink color of the printed word. This task requires participants to override an automatic response (i.e., processing the content of the word) and to execute a controlled behavior (i.e., naming the color of the printed word). Emotional variants of standard cognitive control tasks present emotionally salient stimuli. For example, in the emotional 2-back task, participants view streams of emotional faces and decide for each face if the current emotional expression is the same as the one presented two faces before. This task requires participants to update the WM contents continuously. The use of emotional stimuli allows for the investigation of valence-specific difficulties in WM operations. Depression research has been conducted using both single tasks which tap into a specific cognitive control component (e.g. using the Stroop task as a measure of inhibition) and multiple tasks tapping into the same component (i.e., the latent-variable approach; Miyake et al., 2000).

Meta-analytic work has provided robust evidence for dysfunctions in cognitive control *in response to neutral information*. A meta-analysis by Snyder (2013) integrated 113 studies

comparing the performance of participants with depression to healthy controls on a wide range of cognitive control tasks measuring different components such as inhibition, shifting, and updating. Snyder adopted the latent-variable approach by analyzing multiple tasks thought to measure the same cognitive control component. The results revealed depression-related impairments with medium effect size on *neutral* measures for several cognitive control processes, namely inhibition, shifting, updating, and others. Also, this study has provided evidence that the magnitude of the impairments can be related to depression symptom severity, with more severe impairments characteristic for severely depressed individuals. Another meta-analysis (Rock, Roiser, Riedel, & Blackwell, 2014) included 24 studies which used the same neuropsychological battery of tasks measuring cognitive control (i.e., the *Cambridge Neuropsychological Test Automated Battery*; Fray et al., 1996). This approach ensured the inter-study homogeneity of the tasks used to assess cognitive control. The study revealed medium effect size for the difference between depressed and non-depressed control groups on emotionally neutral measures of cognitive control. In sum, results from these meta-analyses provide robust evidence for depression-related dysfunctions in cognitive control on emotionally neutral tasks.

Research has also provided evidence for dysfunctional cognitive control *in response to emotional information* (for detailed reviews see: Gotlib & Joormann, 2010; Joormann & Vanderlind, 2014). In this research field, a variety of experimental tasks that presumably tap into different components of cognitive control have been used. Unfortunately, there are currently no meta-analyses to provide a systematic synthesis of previous work. This line of research has largely adopted the three-component model of cognitive control (Miyake & Friedman, 2012; Miyake et al., 2000). Several studies have investigated updating of working memory (WM) and found evidence of depression-related difficulties in manipulating material in WM, especially when processing negative material (Joormann et al., 2011). Depressed individuals are also slower to

discard sad faces and faster to discard happy faces from WM compared to healthy controls (Levens & Gotlib, 2010). With regard to the shifting function in depression, evidence for both general and emotion-specific dysfunctions has been found. While subclinical levels of depression may be characterized by emotion-specific dysfunctions in the form of shifting away from negative material, clinical depression levels may be characterized by dysfunctions in shifting between both negative and positive mental representations (De Lissnyder et al., 2012; Demeyer et al., 2012). Moreover, there is some evidence for depression-related deficits in inhibition of negative material. Using a negative affective priming task, Joorman and Gotlib have found that depressed individuals, compared to healthy controls, exhibit difficulties in inhibiting negative words (Joormann & Gotlib, 2010). In sum, studies in this field provide some evidence for depression-related dysfunctions in exerting cognitive control over emotional material.

Neuroimaging studies

Neuroimaging studies have started to investigate the neural substrates of cognitive control dysfunctions in depression. A recent review has pointed to depression-related decreases in activation of the dlPFC and dACC during both emotional and neutral tasks that demand increased cognitive control (Pizzagalli, 2011). This review also emphasized an important role of the hypoactivation and/or reduced deactivation of the rostral ACC – a region of the ACC related to evaluating emotional and motivational significance of events. For example, one study has shown that depressed patients have an increased activity of the dACC and parietal and bilateral insular cortices when removing positive compared to removing negative words from WM (Foland-Ross et al., 2013). Also, difficulties in shifting attention away from emotional stimuli in participants with mild to moderate depression levels has been related to weaker activation of the lateral PFC and parietal regions, regions associated with cognitive control and attentional processes (Beevers et al., 2010). A number of studies have focused on the processing of emotional material in depression,

and especially the processing of negative stimuli. Some of the meta-analyses in this domain have found evidence of abnormal activity in the dACC and decreased activity in the dlPFC, regions strongly associated with cognitive control (Hamilton et al., 2012), while others have failed to replicate these results (Müller et al., 2016; for a discussion see: Barch & Pagliaccio, 2017).

However, research on neural correlates of cognitive control dysfunctions in depression is in early stages. The number of studies in this domain is limited and sample sizes are often small. Also, the small number of studies and the heterogeneity of tasks used (e.g., emotional and neutral variants of various cognitive control tasks) limit the possibility of drawing strong conclusions about neural substrates of dysfunctional cognitive control in depression. Another important problem in this field is related to the issue of reverse inference – inferring the engagement of a psychological process from patterns of brain activity (Poldrack, 2006, 2011). For example, the observed difference between depressed and healthy individuals in the dlPFC activity when processing negative material cannot be interpreted in terms of the reduction in the ability of the depressed individuals to exert control over negative material. This type of reverse inference is commonly found in the interpretation of the neuroimaging results in depression. Finally, heterogeneity of depression leaves the possibility that different depression subtypes are related to different neurobiological changes, which could be one of the reasons for the lack of consistency of the neuroimaging studies (for a recent example of the work on neurophysiological subtypes of depression see: Drysdale et al., 2016).

State of the Art on Cognitive Control in Depression

Meta-analyses of behavioral studies reveal reliable depression-related dysfunctions in cognitive control processes when measured with neutral tasks. Neuroimaging studies have started to provide initial evidence for the neurobiological substrates of these dysfunctions, but this research is in early stages. A smaller number of studies offers initial evidence for emotion-specific

dysfunctions in different cognitive control components such as inhibition and WM updating. These findings provide support for theoretical models that ascribe an important role to reduced cognitive control in the onset and maintenance of depressive symptoms.

While the relationship between dysfunctional cognitive control and depressive symptoms is empirically supported, the causes and nature of these dysfunctions remain unclear. Questions about the relative importance of specific control dysfunctions in processing emotional material and the question of component-specific vs. general control dysfunctions remain unanswered. Crucially, current research is describing cognitive control dysfunctions, but not proposing mechanisms through which these dysfunctions originate and are maintained. We propose that current conceptualizations of cognitive control used in depression research contribute to the state of research in this field with lingering major research questions. In the next section, we outline some of the problems with views on cognitive control in this field and analyze how they are hampering further theoretical and empirical progress. We identify three main problems: (1) problems with the way in which the three-component model is used; (2) the problem of the depressed homunculus; and (3) lack of theoretical integration of cognitive control with emotional and motivational processes. We elaborate on each of these issues in the following sections.

Conceptual Problems with Cognitive Control in Depression Research

Problems with the Three-Component Model

The three-component model of cognitive control has been used to guide a large portion of empirical and theoretical work on depression, where most of the studies and frameworks have adopted the division of cognitive control into *inhibition*, *shifting*, and *updating* (Miyake et al., 2000). This three-component model of cognitive control is based on correlational and individual differences research and is primarily descriptive. The three components were extracted through confirmatory factor analysis of multiple (non-emotional) tasks known to engage cognitive control

processes and activate the PFC. The resulting components show both unity and diversity, which means that they represent correlated but separable facets of cognitive control. This approach to cognitive control has generated a wealth of research focusing on the relations between specific cognitive control components and other factors such as intelligence, genetic factors, and psychopathology (Braver, Cole, & Yarkoni, 2010; Diamond, 2012; Friedman & Miyake, 2017). In research on depression, studies on cognitive control have investigated depression-related deficits in one of the three components of cognitive control by selecting experimental tasks that are thought to primarily tap into a specific component. While this approach has led to some advances in charting cognitive control dysfunctions in depression, there are several issues with this approach that seem problematic from a methodological and conceptual point of view.

The task-impurity problem

One important methodological problem is related to specificity of the measurement of cognitive control processes. All tasks that have been used to assess cognitive control components involve multiple cognitive processes such that every task reflects an impure measure of the process (Miyake & Friedman, 2012). Apart from the processes targeted to address a particular research question, each cognitive control task also involves low-level visual processes (e.g., color processing in a Stroop task) and other non-targeted cognitive control processes (e.g., shifting between mental representations often involves updating the contents of WM). This problem is called the *task-impurity problem* (Miyake & Friedman, 2012). This issue is even more problematic in psychopathology research where complex tasks are often used to assess multiple cognitive control processes at once (for a detailed discussion see: Snyder et al., 2015). One of the solutions to address this problem is the use of the latent-variable approach. This approach involves measuring one component with multiple tasks tapping into the same process in order to extract a more pure measure of the cognitive control process involved in the tasks. This approach is rarely applied in

clinical research because it requires larger sample sizes and longer study protocols. Rather, most of the subclinical and clinical studies include only one task that is assumed to measure a single cognitive control component in relatively small samples. This imposes significant limitations on depression research. Specifically, the task-impurity problem and the lack of solutions to tackle this problem, challenge the claimed specificity of reported research findings in terms of cognitive control components. This hampers current understanding of the nature of cognitive control dysfunctions involved in depression.

Generalizability of the original component structure

Another problem related to the three-component structure of cognitive control is that this structure was extracted from data collected with tasks presenting emotionally neutral material. It remains unclear to what extent this structure can be replicated in the context of emotional stimulus materials. To date, research has yet to examine similarities and differences in the structure of cognitive control components in response to emotional vs. neutral material. Some indications of potential differences from the original component structure come from a recent study in which no correlations were found between inhibition, shifting, and updating when using emotional tasks (Everaert et al., 2017). In this respect, however, it is important to note that there is an increasing number of studies using control tasks with neutral material that did not replicate the initial three-component structure (Miyake & Friedman, 2012). As a result of these recent insights, changes in the initial conceptualization have recently been proposed (Friedman & Miyake, 2017; Miyake & Friedman, 2012).

The need for controlled processing

As we have stated earlier, the three-component model of cognitive control is primarily descriptive and reflects the factor structure of cognitive control components that provides the best fit with the dataset at hand. While this approach to cognitive control is very useful for investigating

possible dysfunctions in specific components, it is less useful when it comes to other questions relevant for depression research. A very important issue that has attracted limited attention in depression research concerns the question of when, with which intensity, and for how long individuals employ cognitive control. Cognitive control is regarded as a goal-directed and controlled process in opposition to more automatic and habitual processes (for a discussion of the concept of automaticity see: Moors & De Houwer, 2006). While the distinction between automatic and controlled processes has been implied as an important one in depression research (Beevers, 2005; Teachman et al., 2012), dysfunctional cognitive control has rarely been investigated from this perspective. Potential dysfunctions in switching from automatic to more controlled processing (i.e. engaging cognitive control), as well as in detecting the need for such a switch, are hard to address within the framework of the three-component model as it is currently used in depression research.

Interim summary

While the division of cognitive control into three components has led to some advances in clinical research, the use of the three-component model entails problems which cannot be resolved easily. This seems especially true for clinical research. The issues of measuring cognitive control components and the question of processing emotional material significantly contribute to the current state of depression research on cognitive control dysfunctions. Importantly, strictly adhering to the three-component model prevents depression research from posing questions about when and how individuals employ cognitive control.

The Depressed Homunculus

One of the long-standing issues of cognitive control research is the homunculus problem. The problem refers to the tendency of cognitive theories to attribute the ability of control over cognitive processes to a unitary controller – the homunculus (Verbruggen, McLaren, & Chambers,

2014). This problem has been long recognized and fractioning of the controller into more basic processes has been proposed as solution (Monsell & Driver, 2000). In this context, the division of control processes into inhibition, shifting, and updating is commonly understood as partitioning of the controller (A. D. Baddeley, 2012). Still, the homunculus is often merely replaced by multiple homunculi surviving in each of those processes. This problem can be tackled in several ways. In order to use cognitive control processes as explanatory concepts, further work is needed on understanding the simple sub-processes and their interactions which lead to what is termed *inhibition, shifting,* or *updating* (Verbruggen et al., 2014). Moreover, a complementary approach to tackling the homunculus problem comes from cognitive neuroscience. Models in this domain are trying to replace the homunculus by proposing explicit computational and neural mechanisms which underlie cognitive control processes (Hazy, Frank, & O'Reilly, 2006).

The homunculus problem is particularly visible in clinical psychology where there is a tendency to explain clinical symptoms by the malfunction of the homunculus. As we have previously stated, dysfunctional cognitive control is often used to explain a wide range of depressive symptoms as well as abnormalities in other processes such as emotion regulation. For example, the tendency to ruminate on negative aspects of an event can be explained by the inability to shift attention away from negative thoughts. Although this may seem like an explanation relying on a mechanism, it is just a re-description of the observed behavior in different terms, if no theoretical explanation is proposed for these inabilities (Verbruggen et al., 2014). In the case of cognitive control, there is a strong need for a more mechanistic explanation which could replace the one relying on the malfunction of the homunculus. This explanation should rely on a specific, mechanistic understanding of the dysfunctional cognitive control processes and well specified neural substrates of these processes.

Modern theories of depression that include cognitive control dysfunctions should aim to go beyond the explanations relying on general dysfunctions of one or multiple components of cognitive control. Instead, they should rely on mechanistic explanations and address the causes of the dysfunctions observed in cognitive control tasks. In this context, the questions of why and how individuals employ cognitive control are important because current explanations are largely homunculus-based. In this paper, we provide some of the building blocks for developing such a framework. We point to specific processes that are crucial for cognitive control and are likely altered in depression.

Cognitive Control is not an Isolated Mechanism: The Role of Motivation and Emotion

Models of cognitive control in depression rarely take into account crucial links between cognitive, emotional, and motivational processes. A growing number of researchers propose that emotion and motivation are crucial parts of cognitive control processes, and that the strong distinction between cognitive, emotional, and motivational processes is not theoretically and practically useful (Inzlicht, Bartholow, & Hirsh, 2015; Pessoa, 2008, 2009). Current views on cognitive control in depression research do not account for the interface between emotion, motivation, and cognitive control. Consequently, most research has focused on the influence of emotion on cognitive control in depression and conceptualized them as separate processes.

Relatedly, depression research has largely focused on cognitive processing of negative material, whereas the processing of motivationally salient material has been picked up only recently in this research domain. This is remarkable in light of the significant role assigned to motivational impairments (i.e., anhedonia) in depression (Pizzagalli, 2014; Treadway & Zald, 2013). Current theoretical models and empirical research have neglected the role of motivation to employ cognitive control processes in the context of depression. The link between motivational processes and cognitive control is crucial given the importance of control for goal-directed behavior. Indeed,

motivation has been shown to have strong effects on cognitive control and is of great importance in contemporary models (Botvinick & Braver, 2015). The lack of motivation to engage in controlled processing could play a pivotal role in cognitive control dysfunctions observed in depression, especially in individuals with anhedonic symptoms. However, the importance of motivational factors in this context is currently impossible to estimate given the lack of empirical studies in this domain. The potential role of motivation in cognitive control dysfunctions in depression is an important new avenue for both theoretical and empirical work. By taking into account the potentially different contributions of anhedonia and prolonged negative affect to dysfunctional cognitive control in depression this research field can start to investigate how different depression symptoms affect cognitive processes. This will be a crucial step towards taking into account the heterogeneity of depression which has been largely neglected in this field (Fried & Nesse, 2015).

In conclusion, an important challenge for future cognitive frameworks of depression is to integrate observed dysfunctions in emotional, motivational, and cognitive control processes rather than to investigate and conceptualize them as separable, but interacting processes. This will help to understand the complex nature of cognitive dysfunctions in depression, and relate them to emotional and motivational deficits – potentially revealing mechanistic relationships.

Summary

Our current knowledge about how cognitive control processes may be altered in depression is limited in several important ways. Some of the main problems in this domain are related to the conceptualization and measurement of cognitive control in depression research, reliance on descriptive and homunculus-based explanations, and the lack of integration between cognitive, motivational, and emotional processes. These problems are hindering progress in understanding the causes of dysfunctional cognitive control in depression. Below, we discuss how several

advances in contemporary models of cognitive control can provide novel insights and overcome some of the challenges we have described. We will focus on three big topics in cognitive control research, namely the problem of switching from automatic to more controlled processing, the role of motivation in cognitive control, and the flexibility of cognitive control.

Cognitive Control in Cognitive (Neuro)Science

When to Engage in Controlled Processing?

The distinction between automatic and controlled processing is one of the central topics of research in cognitive psychology (Posner & Snyder, 1975; Shiffrin & Schneider, 1977). While some situations demand control, in other situations, behavior can be automatic with no negative consequences. The ability to overcome habitual actions and engage in more strategic and goal-driven behavior is one of the hallmarks of human behavior. Here, a key question is how the need to switch from more automatic to more controlled processing and behavior is determined. In other words, how do individuals "know" when to engage in controlled processing or when to "turn on cognitive control"?

In this context, there is a prevailing notion that the cognitive system will stay in an automatic mode of processing until a need for cognitive control is detected via changes in current goals or via performance monitoring. Several theoretical models have adopted this approach and have posited the ACC as a key region responsible for detecting the need for control. Some of the proposals for the mechanism through which the need for control is detected include: the presence of response conflict (i.e., Conflict Monitoring Theory: Botvinick et al., 2001), commission of errors followed by omission of rewards (Holroyd & Coles, 2002), and the discrepancy between predicted and obtained outcomes (Alexander & Brown, 2011). The exact way in which the need for cognitive control is detected is still a subject of intense investigation (Brown, 2017; Holroyd & Yeung, 2012; Shenhav et al., 2013; Silvetti et al., 2014). Most theorists agree that the need for control is first

detected based on certain changes in the environment, which in turn signal the need to implement control. The role of detecting the need for cognitive control and the intensity of control is assigned to the ACC. The ACC transmits signals to other regions (such as the dlPFC) that in turn implement control.

Motivation and Cognitive Control

The importance of motivation in cognitive control processes is inherent in the definition of cognitive control as a set of processes that support goal-directed, non-automatic behavior. While it may seem that it would be most adaptive to be constantly engaged in controlled processing and behavior, new research and theoretical advances indicate that engaging in this type of processing carries an intrinsic cost which is named mental effort (Shenhav et al., 2017). It has been shown that engaging in tasks high in cognitive demand, such as cognitive control tasks, is inherently costly and that individuals tend to avoid it, even if this is tied to forgoing substantial rewards (Kool, McGuire, Rosen, & Botvinick, 2010; Westbrook, Kester, & Braver, 2013). In order to pursue a task that involves mental effort, individuals need to be sufficiently motivated. Indeed, a growing number of studies have demonstrated that in non-depressed individuals motivation can enhance a number of cognitive control processes such as response inhibition, task-switching, updating, etc. (Botvinick & Braver, 2015; Braver et al., 2014; Krebs & Woldorff, 2017). Research demonstrating motivational enhancements in cognitive control and costs of control (in terms of mental effort) has led to the development of novel theoretical approaches.

One of the novel theoretical developments which emphasizes the role of motivation in cognitive control comes from the authors of the three-component model (Miyake et al., 2000). Recently, the authors of this model have shifted their focus from diversity towards unity of cognitive control components. These authors have proposed the unity/diversity framework in which a common factor represents the unity of cognitive control processes, while updating-specific

and shifting-specific factors represent diversity (Friedman & Miyake, 2017; Miyake & Friedman, 2012). Importantly, the common factor shared by all control components is defined as the "ability to actively maintain task goals and goal-related information and use this information to effectively bias lower-level processing" (Miyake & Friedman, 2012, p. 11).

The open question in this field centers on the problem of how motivation (i.e., current goals) is translated to the allocation of cognitive control. One of the possible mechanisms that may determine when to allocate cognitive control (or effort) is the process reliant on cost-benefit decision-making including information on how much reward is expected from engaging control and how effortful this will be (for a review of these models see: Kool et al., 2017). One of the prominent theories taking this approach is the Expected Value of Control (EVC) theory (Shenhav et al., 2013). EVC theory proposes that a cost-benefit analysis underlies the decision about the amount, timing, and strength of control allocation. This cost-benefit decision-making process is based on three types of information: the expected payoff from the allocation of control, the amount of control needed, and the cost of the control in terms of cognitive effort. EVC theory proposes that the dorsal ACC integrates this information, calculates the expected value of control, and then signals this to regions which implement control such as the dlPFC.

The Balance Between Flexibility and Stability

The idea that information retained in WM can be used to control and guide subordinate cognitive processes and behavior is an enduring principle formulated in early models of WM such as the model of Baddeley and Hitch (1974). Flexible cognitive control poses conflicting functional demands on WM. On the one hand, it is important to maintain stable goal representations in order to guide attentional and decision making processes. In order to do this, the contents of WM have to be protected and any interfering representations must be inhibited. On the other hand, it has to be possible to flexibly update contents of WM in case of salient changes in the environment or

changes in one's goals. This poses conflicting demands on working memory and this problem has been termed "flexibility vs. stability paradox" (Bhandari, Badre, & Frank, 2017). While too much flexibility can promote distraction, too little flexibility can lead to rigidity. Hence, a balance between flexibility and stability is crucial for optimal allocation of cognitive control. Two important questions in this context are which mechanisms support flexibility and stability, and how individuals manage to obtain the optimal balance between these processes.

Cognitive neuroscience and computational models of working memory and cognitive control have proposed WM gating as the mechanism underlying flexibility and stability (Braver & Cohen, 1999; Chatham & Badre, 2015; Frank, Loughry, & O'Reilly, 2001; Hazy et al., 2006). If the gate is open WM is sensitive to external input, while when the gate is closed the existing representations are stably maintained. In this way, input gating serves as a selection mechanism that determines the time at which contents of WM can be updated. This gating mechanism is thought to rely on dopamine signaling within the PFC and the striatum (Braver & Cohen, 1999; Westbrook & Braver, 2016) and on fronto-striatal loops involving the PFC and basal ganglia (Frank et al., 2001; Hazy et al., 2006). More recently, a similar output gating mechanism has been proposed (Chatham & Badre, 2015; Chatham, Frank, & Badre, 2014). This mechanism determines which of the representations currently held in WM will control further processing (e.g. biasing attention towards goal-relevant stimuli).

While the described gating models provide an explicit mechanism that underlies the ability to flexibly update or stably maintain WM representations which underlie goal-directed behavior, an important open question is how the gating system learns when to be flexible or stable. The consensus in the field is that it is necessary to avoid potential homunculus-based explanation by relying on learning processes. One of the promising possibilities is that the system is trained by reward prediction errors, i.e. reinforcement learning (Bhandari et al., 2017). Another perspective

proposes that control can be grounded in associative learning and conceptualized as a process reliant on associative networks including perceptual, motor, and goal representations (Abrahamse, Braem, Notebaert, & Verguts, 2016; Verguts & Notebaert, 2009). These perspectives are not mutually exclusive and they offer a new way of circumventing the homunculus problem by linking cognitive control to learning processes while providing a neurobiologically plausible mechanism to support these proposals.

Summary

Cognitive neuroscience research has provided increasingly specific mechanistic views on cognitive control. The homunculus-based views are being replaced by fine-grained processes, such as detecting the need for controlled processing, motivation as an integral component of control, and learning-based flexibility of cognitive control. These novel advances are offering a detailed view of cognitive control as a high-order cognitive process emerging from the interactions between multiple processes with a strong neurobiological foundation. Although these models are still being developed, they provide a wide range of possibilities to solve some of the previously discussed issues in clinical research on cognitive control dysfunctions in depression. Current empirical work and theoretical models have begun to characterize cognitive control dysfunctions in depression and have provided ways in which these dysfunctions contribute to the onset and maintenance of symptoms via emotion regulation strategies. An important next step is to provide an explanation for the causes of these dysfunctions and to investigate their precise nature. In the next section, we discuss the ways in which the described developments in basic cognitive control research can be integrated into depression research and used to advance clinical science.

Updating Current Views on Cognitive Control in Depression

Engaging in Controlled Processing and Depression

The distinction between automatic and controlled processing has been addressed in depression research (Beevers, 2005; Teachman et al., 2012), but an integration with cognitive control processes is lacking. The question of the potential depression-related impairments in detecting when to switch from more automatic to more controlled processing modes (i.e., cognitive control processes) remains largely unaddressed. From a neurobiological point of view, there is evidence that depression is related to disrupted activity of the ACC, a region involved in detecting the need for cognitive control. For example, several studies have revealed abnormal error processing in depression leading to decreased cognitive control recruitment (Holmes & Pizzagalli, 2009; Holmes & Pizzagalli, 2007). This evidence of reduced ACC activation in depressed individuals coming from both neuroimaging and electrophysiological studies has inspired models which propose disrupted activity of the ACC as a possible biomarker for depression (Holroyd & Umemoto, 2016; Olvet & Hajcak, 2008; Pizzagalli, 2011).

Many of the cognitive models of depression include dysfunctional cognitive control, but do not specify the origin of these dysfunctions. The described models of cognitive control, which deal with the question of how switching between automatic and controlled processing occurs, offer a new avenue that could advance both cognitive research and theorizing in the domain of depression. Starting from the described models of cognitive control, it could be argued that failures to detect the need for increased control, which can be observed as disruptions in ACC activity, cause signals indicating the need to switch to more controlled behavior to be weaker. This is then observed as the decreased tendency of depressed individuals to engage cognitive control. This point of view offers a mechanism for observed cognitive control dysfunctions in depression and ties them closely to neurobiological mechanisms. Still, how to explain a disruption in the ability to detect the need for control? This is where some of the novel models that link cognitive control to motivation may play an important role.

Motivation and Cognitive Control in Depression

Sustained negative affect and anhedonia – the loss of interest or pleasure in previously pleasant activities, are regarded as cardinal symptoms of depression (Gotlib & Furman, 2015). While negative affect has received a lot of attention in cognitive models of depression, anhedonia has been somewhat disregarded. Recently, there is an upsurge of research interest in anhedonia in depression revealing impairments in reward processing and willingness to exert effort.

Research on anhedonia in depression has led to recent proposals to re-conceptualize anhedonia. While there is mixed evidence that depression is linked to reductions in consummatory value (i.e., loss of subjective pleasure coming from obtaining rewards), deficits in the ability to change behaviors in order to maximize reward attainment, in implicit reinforcement learning, and in reward-based decision making have been reliably demonstrated (Admon & Pizzagalli, 2015; Pizzagalli, 2014; Treadway & Zald, 2013). A recent meta-analysis has demonstrated that these depression-linked impairments in reinforcement learning are more related to reduced reward sensitivity then to the learning rates (Huys, Pizzagalli, Bogdan, & Dayan, 2013). Also, electrophysiological studies have demonstrated depression-related reduced responses to reward attainment (termed reward sensitivity), measured as reward prediction errors (Olvet & Hajcak, 2008) and reward positivity (Proudfit, 2015; Proudfit, Bress, Foti, Kujawa, & Klein, 2015). Another recent research line has demonstrated that depressed individuals are less willing to invest effort into obtaining rewards compared to controls (Treadway, Bossaller, Shelton, & Zald, 2012), and that their perceived level of invested effort differs from objective measures of actual effort invested (Cléry-Melin et al., 2011). These results suggest that depression is not necessarily related to decreased experience of subjective pleasure, but there is electrophysiological evidence for reduced sensitivity to obtaining rewards. Importantly, increased depression levels seem to be linked to a decreased willingness to modify behavior to obtain rewards, an impaired ability to learn from

obtaining rewards, and to a dissociation among experienced pleasure and willingness to invest effort into achieving pleasure.

Taking into account the importance of motivation and effort for cognitive control, the investigation of contributions of impairments in these processes to depression-linked dysfunctions in cognitive control seems crucial. Starting from the EVC theory of cognitive control and applying it to depression there are several important things to notice. The EVC theory proposes that decisions about timing and intensity of cognitive control are based on reward prospect related to engaging in controlled processing and expected amount of effort related to this. Both reward processing and effort expenditure are known to be changed in depression. It seems plausible to hypothesize that this could be one of the reasons underlying the depression-related cognitive control dysfunctions. In simplified terms, the idea could be that for depressed individuals the perceived gain from engaging in costly control processes seems small, while the effort seems too big, which could in turn lead to reduced exertion of control. This idea offers the possibility to reinterpret the neuroimaging results on the depression-related changes in the brain regions related to cognitive control. From the perspective of the EVC theory, changes in the perceived values of rewards and the willingness to exert effort would affect the cost-benefit calculation in the dACC which would decrease the intensity of control implemented by the dlPFC.

The contribution of anhedonic symptoms to dysfunctional cognitive control is a research field that – based on our analysis – could be highly informative. While there are theory-based indications that this link could be important, there is almost no empirical research on the topic. Future research should investigate the potential differences in the levels of cognitive control dysfunctions related to different symptoms of depression. The first step in this domain will be to compare cognitive control dysfunctions between individuals with elevated negative mood and individuals with increased anhedonic symptoms. This will be a crucial step towards uncovering the

potential heterogeneity within cognitive control dysfunctions in different subtypes of depression. Further on, research on the role of motivation in cognitive control dysfunctions should elucidate the potentially separable contributions of lack of motivation (e.g. "Is this reward worthy enough?") and the lack of willingness to exert effort (e.g. "How much effort am I willing to invest to attain this reward"). We are currently working on developing paradigms in which we can manipulate both reward prospect and the need to invest effort during a cognitive control task.

Flexibility and Stability in Depression

Psychological flexibility, largely reliant on flexible cognitive control, has been theorized as a fundamental aspect of mental health and may be disrupted in various forms of psychopathology (Kashdan & Rottenberg, 2010). There is evidence that depression is related to both problems in flexibly updating the working memory and in maintaining WM contents, which may guide further information processing. Most of the research and theoretical models in this field are directed towards finding precise impairments in these processes and how they are related to the valence of WM content. Yet, research evidence is still mixed. The cognitive neuroscience models we have described above allow to rephrase the initial question from searching for impairments towards examining *how* depressive symptoms are related to the balance between flexibility and stability. While there is no direct research on the topic yet, there is some relevant indirect evidence about the influence of mood on cognitive flexibility, showing that positive mood promotes flexibility at the cost of stability (Dreisbach, 2006; Dreisbach & Goschke, 2004). One of the possibilities is that prolonged negative mood could promote stable maintenance of WM representations at the cost of the ability to flexibly update content of WM with new information relevant for goal-directed behavior.

Another line of indirect evidence for a relation between flexibility-stability balance and depressive symptoms comes from neuroscience research on depression-related changes in the

activity of the striatum – a region central in the gating models of WM. As we have previously described, fronto-striatal loops are implicated in WM gating and neurobiological models of cognitive control. Importantly, dysfunctions of this circuitry are found in a wide range of psychiatric disorders (Gunaydin & Kreitzer, 2016). To date there is evidence that depression is associated with changes in activity of both the dorsal and the ventral striatum in response to pleasant and rewarding stimuli (Pizzagalli, 2014). Also, there is some evidence for depression-related changes in striatal-dACC connectivity in response to losses and rewards (Admon et al., 2015). However, it remains unclear if dysfunction of the striatal regions in depression can be observed only in the context of processing motivationally salient stimuli, or whether these dysfunctions are broader and can also be observed in neutral cognitive control tasks. An interesting avenue for further research would be to examine striatal activation in depression during neutral cognitive control tasks. The possibility of depression-related changes in the striatum – a region important for gating of information in or out of the WM – in neutral cognitive control tasks in depressed individuals would offer a plausible neurobiological mechanism that can account for some of the cognitive control dysfunctions in depression.

Finally, the theoretical accounts that relate cognitive control to learning mechanisms offer an opportunity to replace the depressed homunculus by well-defined basic mechanisms. For example, one of the proposals which we have described links reward prediction errors and reinforcement learning, known to be altered in depression (Gradin et al., 2011; Kumar et al., 2008; Pizzagalli, 2014), to adaptation of gating policies underlying cognitive control processes. Also, there is evidence for depression-related changes in the dopaminergic system which is crucial in gating models of cognitive control (Dunlop & Nemeroff, 2007; Nestler & Carlezon, 2006). Another proposal relies on associative learning and offers an interesting possibility to study potential

depression-related impairments in this type of learning and explore whether these are related to cognitive control dysfunctions (Abrahamse et al., 2016; Verguts & Notebaert, 2009).

Summary

The use of the three-component model has led to research on depression-related impairments in inhibition, shifting, and WM updating. While this approach has contributed to understanding of dysfunctional cognitive control, it has led to multiple problems as well. First of all, the important next step is to avoid homunculus-based explanations at the level of impaired cognitive control processes (e.g., depression-related impairments in updating). While these explanations are a good first step in guiding research, current progress in cognitive neuroscience provides useful avenues by which further research and theorizing can advance. By shifting the focus away from merely establishing specific dysfunctions, we can pose more specific questions such as: how and why do depressed individuals fail to detect the need to engage in controlled processing? Moreover, understanding cognitive control as a process reliant on reinforcement or associative learning will provide the field with the basic mechanisms through which the dysfunctions can occur. Importantly, by raising the questions of how "cognitive control is learned" depression research can investigate more direct links between stressful life events, which necessarily involve learning, with dysfunctional cognitive control. Finally, the important role of motivation and the willingness to exert effort in cognitive control processes has been largely overlooked in depression research. Novel models of cognitive control offer an exciting possibility of linking anhedonic symptoms with dysfunctional cognitive control in depression. Further integration of cognitive and neuroscience models of cognitive control into the clinical research on depression is a next step to take. Doing so carries the potential to elucidate causes of cognitive control dysfunctions and to enable the understanding of more fine-grained depression-related dysfunctions in cognitive processes.

Conclusions

Current cognitive models of depression consider cognitive control dysfunctions as an important risk factor for depression and an important hub in the depressogenic information processing. After more than a decade of establishing the presence and influence of dysfunctions in cognitive control, it is timely that research goes beyond this descriptive level. Current cognitive models of depression assume cognitive control dysfunctions, either general or more specific ones (e.g. inhibition of irrelevant negative material), without offering a cause and a mechanistic explanation for these dysfunctions. Moreover, most current cognitive models of depression assume that cognitive control is attenuated in depression, which is a very simplistic view of a complex set of dysfunctions. Starting from cognitive neuroscience models of cognitive control, we propose novel avenues for research and theorizing which offer solutions to current limitations of models of dysfunctional cognitive control. We offer a perspective in which deficits in cognitive control observed in depressed individuals do not stem from attenuated ability to exert cognitive control per se. Rather, we argue that dysfunctions arise due to the impaired ability to detect when, with which intensity, and for how long to engage in controlled processing. This process can rely on changes in the environment that signal the need for control (e.g. making a lot of errors) or a cost-benefit decision making process weighing between the prospect of reward related to switching to controlled processing mode and the costs of doing so. Alternatively, it can also rely on previous learning and knowing when to flexibly engage with the environment vs. when to stably maintain a goal. In this way, cognitive control dysfunctions can be viewed not as a structural problem arising from the malfunction of a particular brain region or a homunculus-based process, but rather as a systematic failure to effectively engage in controlled processing that supports goal-directed behavior.

This reconceptualization offers new perspectives on key depressive symptoms. For example, some of the maladaptive emotion regulation strategies can become an automatic mode of processing negative thoughts and emotions (e.g. rumination). In order to employ a more adaptive emotion regulation strategy, cognitive control might be needed to switch from ruminating and to start reappraising. If there is a failure to detect the need for a switch when ruminating (conflict detection), if there is a lack of motivation (anhedonia), and if switching is perceived as very effortful (effort expenditure problems), this can lead to a failure to employ cognitive control and switch to a different processing mode. In turn, this can then lead to further rumination. While this can be explained as a failure to switch from one task set to another (e.g. switching impairment), but we believe that a focus on potential causes of this process offers a much more in-depth perspective on depressed cognition. While these processes can be generally viewed as a failure to, for example, switch from one task set to another, we believe that the perspective which we provide here is much more specific.

From a clinical perspective, breaking down complex cognitive dysfunctions in depression into multiple smaller-scale problems offers new opportunities with regard to developing targeted treatments and cognitive control trainings. Importantly, cognitive control dysfunctions are also proposed to be a transdiagnostic mechanism as these dysfunctions are observed in a wide range of mental disorders (Goschke, 2014). Advances in the mechanistic understanding of cognitive control dysfunctions in psychopathology will lead to a better understanding of the relations between this and other transdiagnostic mechanisms which will be crucial in moving from the symptom-based towards the mechanism-based view of psychiatric disorders (Cuthbert & Insel, 2013; Insel et al., 2010). Finally, depression research offers an exciting field for testing cognitive neuroscience models of cognitive control in a more applied manner. In this way, the intersection between cognitive and clinical science offers productive avenues for advancing both fields.

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