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Review

Cognition-related brain networks underpin the symptoms of unipolar depression: Evidence from a systematic review

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ABSTRACT

This systematic review sources the latest neuroimaging evidence for the role of cognition-related brain networks in depression, and relates their abnormal functioning to symptoms of the disorder. Using theoretically informed and rigorous inclusion criteria, we integrate findings from 59 functional neuroimaging studies of adults with unipolar depression using a narrative approach. Results demonstrate that two distinct neurocognitive networks, the autobiographic memory network (AMN) and the cognitive control network (CCN), are central to the symptomatology of depression. Specifically, hyperactivity of the introspective AMN is linked to pathological brooding, self-blame, rumination. Anticorrelated under-engagement of the CCN is associated with indecisiveness, negative automatic thoughts, poor concentration, distorted cognitive processing. Downstream effects of this imbalance include reduced regulation of networks linked to the vegetative and affective symptoms of depression. The configurations of these networks can change between individuals and over time, plausibly accounting for both the variable presentation of depressive disorders and their fluctuating course. Framing depression as a disorder of neurocognitive networks directly links neurobiology to psychiatric practice, aiding researchers and clinicians alike.

Contents

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1. Introduction

Sufferers of unipolar depression each present with a unique constellation of cognitive, affective, and somatic symptoms. These features were first described in ancient texts, and remain relevant today: disturbed mood, self-loathing, difficulty concentrating, a wish to die, bodily complaints, indecision, and delusions of guilt (Davison, 2006). In line with current concepts of brain function, depressive symptoms are thought to arise from the failed regulation of large-scale anatomical and functional brain networks (Insel et al., 2010; Mayberg, 2003; Menon, 2011; Palmer et al., 2015; Sporns, 2011), with different depressive symptoms or symptom clusters reflecting different neurobiological substrates (Aktas et al., 2010). Research has often focused on mapping the networks that give rise to affective symptoms (Goulden et al., 2012; Hamilton et al., 2012; Heller et al., 2009; Le Doux, 2000; Mayberg et al., 1999; Phillips et al., 2003), given that the "cardinal" features of the disorder are low mood (dysphoria) and the inability to experience pleasure (anhedonia). Growing evidence, however, points to the fundamental role that abnormal interactions between cognition-related networks may play in the expression of many of the diverse features of depression, including somatic and affective symptoms as well as cognitive phenomenology (Davidson et al., 2002; Ochsner and Gross, 2005; Phan et al., 2005; Phillips et al., 2003).

Cognitive disturbance is recognised as an "accompanying" feature of unipolar depression in current diagnostic criteria (American Psychiatric Association, 2013; World Health Organization, 2008). Common cognitive manifestations include maladaptive and distorted styles of thinking about the self and the world (Beck and Alford, 2009), as well as subjective and objective impairments in cognitive control, memory, processing negative information, and other cognitive domains (Antikainen et al., 2001; Clark et al., 2009; Gotlib and Joormann, 2010; Rock et al., 2013). Studies using structural and functional magnetic resonance imaging (MRI) have identified a midline web of prefrontal-limbic regions thought to underpin deficits in cognition that relate to a range of negative affective experiences (Bremner et al., 2004; Cocchi et al., 2014; Levin et al., 2007; McDermott and Ebmeier, 2009). Moreover, altered functioning of cognitive brain networks has been hypothesised to impair the downregulation of cortico-subcortical mood networks, potentially accounting for some of the somatic features and phenotypes of unipolar depression (Ochsner and Gross, 2005; Wilson, 2011).

The aim of this review is to comprehensively characterise the role of cognitive networks in depressive symptomatology. To achieve this, we (i) investigated whether cognition-related brain networks show altered functioning in adults with unipolar depression; (ii) assessed research detailing whether the functional relationships between various cognitive networks are abnormal in unipolar depression; (iii) specifically reviewed how abnormal interrelationships between cognitive networks might impact on other affect-regulating brain networks; and (iv) examined how these altered dynamics relate to the symptoms of unipolar depression. It builds on previous (neuro)cognitive models of depression (Disner et al., 2011; Gotlib and Joormann, 2010; Marchetti et al., 2012; Northoff et al., 2011; Whitfield-Gabrieli and Ford, 2012) that use self-selected or purely behavioural data by (i) doing a *systematic* review of the functional imaging literature (ii) to explain the cognitive, affective, and somatic symptoms of depression in terms of neurocognitive network dysfunction.

The functional neuroimaging research reviewed here links the symptoms of depression to two abnormal cognition-related brain networks. The Autobiographic memory network (AMN) is commonly known in resting-state form as the "default mode network". It focuses on internal mental states but in depression is overactive, leading to pathological introspection and symptoms such as rumination and distorted information processing. In contrast, the goal-directed cognitive control network (CCN) is underengaged in people with depression, leading to characteristic difficulties in efficiently attending and responding to environmental demands. The anatomical and functional configurations of these two networks can change between individuals and over time, plausibly accounting for both the idiosyncratic symptom presentation of depressive disorders and their often-fluctuating course.

This model of depression has the advantage of being able to map the abnormal function of complex brain systems to the clinical reality: a patient consumed by a maladaptive internal monologue, too lethargic from poor sleep and nutrition and too self-focused to efficiently marshal cognitive resources to appropriately engage in the world around them. Better understanding the underlying neurobiology of depressive symptoms has the potential to improve the precision of psychiatric medicine. Conceivable advances to stem from future neurocognitive studies include: (i) narrowing the search for in vivo biomarkers of depression that are evident on non-invasive investigations such as neuroimaging, (ii) broadening research parameters to include patients with a depressive biomarker but subclinical depressive symptom, and (iii) linking a patient's unique clinical presentation to proximal brain networks in order to define individually tailored anatomical, cognitive, or neurochemical targets for treatment.

2. Method

In May 2015, we conducted literature searches on EMBASE and MEDLINE using the Topic-Add MeSH or search terms (1) "all fields = cognition" AND "all fields =network" AND "keyword = depression" and (2) "all fields = symptom" AND all fields = ("neuro*" and "network") AND "keyword = depression", searching the years 1980–2015 for peer-reviewed articles in English with prospective or retrospective data. The abstracts of retrieved articles were examined by G.R. and included if they met all of the following stepwise criteria: (i) described original research or meta-analyses, and (ii) reported functional neuroimaging (e.g., positron emission tomography (PET), fMRI, single-photon emission computed tomography (SPECT)) of neurocognitive networks OR neuroimaging relevant for understanding the neurobiological basis of depressive symptoms, and (iii) compared adult patients with unipolar depression to healthy adult controls. Studies of late-onset (>50 years) depression were excluded, as current evidence suggests that unique pathophysiologies underpin early onset versus late-onset unipolar depression (Naismith et al., 2012). For similar reasons, studies of context-dependent unipolar depression such as seasonal affective disorder and postnatal depression were also excluded. Finally, since the existential symptoms of depression