



Short communication

Cognition in depression: Can we THINK-it better?



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ABSTRACT

Background: Cognitive compromise is a common experience for patients with depression and other mood disorders. Depressed patients sustain deficits in working memory and attentional distortions in emotional processing and negative attention biases, which may contribute to maintaining their depressive state.

Methods: The Mood Assessment and Classification (MAC) Committee comprised academic psychiatrists with clinical expertise in the management of mood disorders. The independently convened committee met to discuss contentious aspects of mood disorders diagnosis and assessment with the express aim of informing clinical practice and future research.

Results: The Committee specifically identified cognition as an important aspect for clinicians to consider in the context of depression and mood disorders. This article highlights some of the barriers to assessment and proposes tools that have the potential to be implemented in clinical practice.

Limitations: The conclusions drawn within this article are based on expert opinion. We have noted the limitations of the literature that informs this opinion.

Conclusions: As cognitive ability has been closely linked to patients' ability to achieve functional recovery, it is imperative that clinicians are able to identify patients with cognitive deficits and are equipped with tools to conduct effective cognitive assessments. Examining cognitive factors may generate a deeper understanding of the pathogenesis of depression and mood disorders which can ultimately be used to inform treatment.

1. Introduction

In addition to mood symptoms, it is increasingly recognized that major depressive disorder (MDD) is characterised by emotional, psychological, behavioural, and physical symptoms. However, in recent years, cognitive symptoms such as neuropsychological dysfunction have attracted greater interest (Baune et al., 2009; Beblo et al., 2011) because cognition has been found to be fundamental to patient

functioning and resolution of cognitive dysfunction is necessary to achieve meaningful long-term outcomes. Clinically this is borne out by the fact that deficits of cognition impact psychosocial and workplace engagement and contribute significantly to the health economic burden accrued by MDD (Krol et al., 2011; Olesen et al., 2012; Thomas and Morris, 2003).

Empirically it has been found that cognitive symptoms in depression last longer than the acute episodes of depression (Conradi et al., 2011) –

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further supporting the contention that conceptualising MDD around mood as its principal symptom is limiting and likely flawed. Specifically, the cognitive symptoms of depression feature prominently from the acute state through to remission (Conradi et al., 2011) and this is likely to be more evident in more severe forms of depression as well as in melancholic depression – although some degree of cognitive impairment is likely to be a core feature of all ‘kinds’ and all ‘grades’ of depression (Beblo et al., 2011b). It should be noted, that the debate on which domains of cognition are affected in depression is still ongoing (Malhi et al., 2015). Although a meta-analysis revealed significant cognitive deficits in the domains of executive function, memory and attention in patients with depression when compared to controls (Rock et al., 2013), other research concludes that a broad range of cognitive domains are affected in depression without a clear pattern of specific deficits (Beblo et al., 2011). Significant correlations have also been shown in a meta-analysis between depression severity and cognitive performance in the domains of episodic memory, executive function, and processing speed, but not for semantic memory or visuo-spatial memory (McDermott and Ebmeier, 2009). The lack of effect for visuo-spatial and semantic memory may be due to the small sample size of patients in these particular cognitive domains (both $N < 50$), while the other domains had data from 126 to 1150 patients. Particularly interesting, and worthy of note, is the suggestion that some of these deficits may remain after remission of MDD (Baune et al., 2010; Hasselbalch et al., 2011; Jaeger et al., 2006; Rock et al., 2013).

In practice, detailed in-depth objective assessment of cognition is rarely carried out as it can be difficult and time consuming. It also should be accompanied by careful evaluation of subjective reports. While it is important to bear in mind that patients tend to over-report cognitive symptoms when explicitly asked (Iverson and Lam, 2013), at the same time, patients seldom report cognitive problems to health care professionals spontaneously because of embarrassment and/or fear of being diagnosed with incipient dementia. It is also common for patients to not regard their cognitive symptoms as part of their depressive syndrome or assume that their cognitive complaints will automatically resolve as the depressive illness improves.

Cognitive dysfunction in depression can be meaningfully examined by refining the concept into “cold” and “hot” cognition as proposed by Sahakian and colleagues (Roiser and Sahakian, 2013). This separates the different components of cognition in relation to emotion, such that “cold” (emotion-independent) cognition refers to functions such as executive function, attention, memory and cognitive speed that are *not* modified by mood states, whereas ‘hot’ (emotion-laden) cognitions describe processes that are susceptible to modulation by emotions. The latter, emotionally coloured cognitive responses, can be assessed by tests of perception, attention and working memory. Repeatedly, results of such experimental studies in depression indicate a negative emotional bias, which may also affect reward and punishment processing (Porter et al., 2015; Roiser and Sahakian, 2013). Emotional processing distortions in MDD, namely negative attention bias to sad facial expressions (Weightman et al., 2014) and oversensitivity to negative feedback (Elliott et al., 1997), are perhaps key clinical features of depression that may also be present in remission. By the same token, social cognition (e.g. theory of mind), that is the ability to identify, perceive, and interpret socially relevant information in relation to oneself and to others, has a significant role in successful interpersonal and day-to-day functioning. Difficulties with social interaction as observed in MDD, may, in part, be due to an altered ability to correctly interpret (and make sense of) emotional stimuli and the mental states of others (Weightman et al., 2014). These too seem to persist in remission especially if MDD does not respond to treatment (Aker et al., 2016) and can result in functional impairment. Although emotion processing and social cognition have been considered to be at the core of depression, their functional importance has been largely underappreciated. Given that several aspects of cognition (‘cold’, ‘hot’ and social cognitive processing abilities) seem to be intimately related to psychosocial function,

it is likely that the broad cognitive dimension of depression extends beyond the phenomenological level and may represent a crucial pathogenetic element that contributes to the development and long-term course of depression. However, there are other important factors that contribute to cognitive dysfunction in depression, such as illness-related socio-economic stressors, medical (e.g. diabetes, coronary heart disease) and psychiatric (e.g. anxiety, substance abuse) comorbidities, being overweight and taking prescribed medication along with physical and emotional abuse. Because these factors may negatively affect cognitive dysfunction as well as other symptoms in depression, these additional burdens may require specific clinical assessment and treatment, which in turn may assist with improving cognitive dysfunction and depression overall.

Along the lines of subgrouping patients with depression according to a development of long-term functional impairment (Conradi et al., 2011), cognitive symptoms and possibly neuroprogression may be important contributors to functional decline. While evidence on the direct relationship between cognitive dysfunction and general functioning is emerging, some evidence suggests that persistent cognitive deficits in depression may play an important role in patients’ inability to fully recover functionally over time (in addition to other critical factors such as confidence, personality, comorbidity, subthreshold symptoms, social supports influencing functional recovery; Baune et al., 2013; Jaeger et al., 2006). For example, poor cognitive function may be partly responsible for patients’ failure to return to work, as a study revealed that only cognitively recovered patients were able to successfully recommence their employment (Baune et al., 2010), indicating that cognitive function is indeed an important factor that contributes to the burden of disease of depression. Furthermore, a preliminary study suggests that deficits in executive functioning exert a mediating effect on the relationship between depression and impaired activities of daily living (Kiosses and Alexopoulos, 2005). A systematic review confirms the relevance of cognitive function in day-to-day functional activities in MDD (Evans et al., 2014). For clinical practice it is important to note that MDD patients with neuropsychological deficits tend to be less adherent with antidepressant treatment (Martinez-Aran et al., 2009) and exhibit an increased risk for suicide (Westheide et al., 2008), with both findings highlighting the clinical importance of examining for neuropsychological deficits in the clinical management of patients with depression.

2. Assessment

The assessment of cognition in clinical practice comes with numerous practical challenges. One major challenge is that cognitive symptoms are conceptualised as a transient disturbance that will resolve automatically with the resolution of mood and other symptoms of depression leading to non-assessment in many patients with actual acute cognitive deficits. Such a distorted understanding of the nature of cognitive problems in depression may fail to prompt proper clinical and/or formal assessment in the first place. Relatively simple questions about day-to-day function can reveal important clues as to the cognitive problems the patient experiences; however, these questions should not be the sole source of assessment as they may correlate poorly with objective measures. As previously mentioned, another significant challenge is related to an assumption that cognitive problems only occur in the elderly with depression and, if they occur, then only as an early symptom of dementia. The notion that the identification of cognitive dysfunction may indicate neurodegeneration may present as a concern for patients and families when testing cognition because they are worried that they are being tested for dementia. However, it needs to be better appreciated and properly communicated that cognition is an important function in itself and is a mediator of other real-life outcomes rather than being solely a precursor to a potential dementing process in the elderly. Therefore, in practice, it is advisable that all concerned (general practitioners, psychiatrists and other health care

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