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Review article

Neuropsychological changes in melancholic and atypical depression: A systematic review

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ABSTRACT

There is not a consensus as to whether neuropsychological profiling can distinguish depressive subtypes. We aimed to systematically review and critically analyse the literature on cognitive function in patients with melancholic and atypical depression. We searched in databases PubMed, SCOPUS, Web of Knowledge and PsycInfo for papers comparing the neuropsychological performance of melancholic patients (MEL) to non-melancholic depressive patients (NMEL), including atypical depressives, and healthy controls (HC). All studies were scrutinised to determine the main methodological characteristics and particularly possible sources of bias influencing the results reported, using the STROBE statement checklist. We also provide effect size of the results reported for contrasts between MEL; patients and NMEL patients. Seventeen studies were included; most of them demonstrated higher neuropsychological impairments of MEL patients compared to both NMEL patients and HC on tasks requiring memory, executive function, attention and reaction time. Detailed analysis of the methodologies used in the studies revealed significant variability especially regarding the participants' sociodemographic characteristics, clinical characteristics of patients and differences in neuropsychological assessment. These findings suggest that MEL may have a distinct and impaired cognitive performance compared to NMEL depressive patients on tasks involving verbal and visual memory, executive function, sustained attention and span, as well as psychomotor speed, this last especially when cognitive load is increased. Additional studies with adequate control of potentially confounding variables will help to clarify further differences in the neuropsychological functioning of depressive subtypes.

Contents

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1. Introduction

Depression is a common disease, which is frequently highly recurrent (Kandel et al., 2000; Sayer, 2001). In addition to causing great suffering, depression also generates numerous financial and personal losses, and it is high in the world ranking of disabling diseases (Bromet et al., 2011; Vos et al., 2012). Nevertheless, currently, available treatments have limited effectiveness. One estimate is that 30–60% of patients do not respond adequately to treatment (Nierenberg et al., 2007; Souery et al., 2007). Potentially an explanation for this is the existence of different clinical conditions within the depression spectrum (Juruena and Cleare, 2007). Important clinical differences among patients may contribute to the nature of depression as a heterogeneous syndrome with distinct subtypes which respond differentially to the treatments (Harald and Gordon, 2012; Henkel et al., 2006). Although the diagnostic manuals adopt a concept of depression as a single entity (American Psychiatric Association – APA, 2013), some subtypes of depression are described within this by modifier terms leaving open the discussion about the current status of depressive subtypes. Within this broad heterogeneity, two subtypes of depression can be particularly discriminated: melancholic depression and atypical depression (APA, 2013).

Melancholic (MEL) depression is characterised as a severe depressive syndrome with a putatively distinguishable biological aetiology and represents between 25 and 30% of cases of depression (Gold and Chrousos, 2002). Melancholic depressive patients present with symptoms such as psychomotor disturbances (Parker, 2007), loss of appetite and sleep; additionally, they tend to feel worse in the morning compared to the rest of the day (Juruena et al., 2011; Parker et al., 1994; Parker and Hadzi-Pavlovic, 1996). Moreover, patients with the melancholic depressive subtype are commonly anxious and exhibit low responsiveness to environment stimulation (Gold and Chrousos, 2002). Thus, psychotherapeutic treatments are often found to be less effective than pharmacotherapy (Brown, 2007).

In contrast, atypical depression (NMEL) differs from endogenous or MEL depression as it is characterised by reactive mood and vegetative symptoms contrasting those found in MEL depression (American Psychiatric Association (APA), 2013; Stewart et al., 2007). Moreover, it is often associated with HPA axis down-regulation, inflammatory and metabolic dysregulation (Lamers et al., 2013). Frequently, atypical depression begins early in life and affects women in a higher proportion than men (Angst et al., 2002). There is a significant genetic factor, and it is common for patients with atypical depression to show comorbidity with dysthymia, substance abuse, antisocial personality disorder, panic disorder and social phobia (Juruena and Cleare, 2007). Furthermore, patients with atypical depression usually have higher rates of a history of neglect and abuse during the childhood and familial alcohol and drug disorders (Matza et al., 2003; Sullivan et al., 1998). Moreover, a relationship exists between atypical depression and lifetime trauma that may be more complicated than the etiologic pathways outlined in prior research and trauma and atypical depression may be interrelated throughout life (Withers et al., 2013).

In addition to differences in pathophysiological mechanisms, the range of subtypes included in MDD may also differ in cognitive functioning. Although studies indicate that depression negatively impacts on cognitive function, the nature and severity of these impairments need to be better understood (Beblo et al., 2011). In a meta-analysis, Zakzanis et al. (1999) demonstrated that the cognitive domains that tended to be more impaired in depressive patients were the declarative or episodic memory and attention. However, numerous studies have shown that the most common cognitive deficits in depression include problems related to executive function combined with decreased performance in tasks involving changing the focus of attention and impaired memory (Elliott et al., 1996; Purcell et al., 1997; Tavares et al., 2003). Thus, the results described in the literature are not completely in agreement with each other. While some studies have shown, for instance, attentional changes in depressed patients (Kaymak et al., 2010; Reppermund et al., 2007). Other studies found no significant differences when comparing patients to healthy controls (Castaneda et al., 2008; Fossati et al., 2004; Westheide et al., 2007). Similarly, divergences have been found among the results reported on the performance of patients on neuropsychological tests of memory and executive functions. Authors such as Kaymak et al. (2010) and Neu et al. (2005) showed that depressive patients had deficits in executive functions, in contrast with other studies that found no differences when patients were compared to healthy controls (Castaneda et al., 2008; Fossati et al., 2004; Grant et al., 2011; Reppermund et al., 2007; van Wingen et al., 2010). In the memory domain, studies have shown evidence of poor performance of depressed patients compared to controls on mnemonic tasks (Kaymak et al., 2010; Reppermund et al., 2007). Meanwhile, other studies found no differences between those groups (Castaneda et al., 2008; Fossati et al., 2004; Ilonen et al., 2000).

As a possible explanation for the variability of results on the neuropsychological performance of depressed patients, McClintock et al. (2010) argue that depressive subtype is an important factor to be considered. Taking depression as a single entity, studies assessing cognitive function in depression may include different subtypes of the disease, which could explain some of the discrepant findings. Several studies have been conducted seeking to identify neuropsychological deficits in depression with the particular focus on factors that could influence cognitive performance (Beblo et al., 2011; Day et al., 2015; Lee et al., 2012).

Overall, the neuropsychological impairments in depressed patients have been associated with a decrease in psychosocial and occupational functioning, which remains independent of other depressive symptoms or clinical remission (Baune et al., 2010) and increased risk of suicide (Westheide et al., 2008). Considering bipolar disorder, patients in the depressive phase with untreated neuropsychological deficits tend to show a poorer adherence to antidepressant treatment (Martinez-Aran et al., 2009) Therefore, many authors agree that the identification of neuropsychological markers can be useful for the development of new therapeutic