THE RELATIONSHIP BETWEEN DEPRESSION AND COGNITIVE DEFICITS

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SUMMARY
In the last years cognitive impairment in depression has been widely reported. It is clear that cognitive symptoms persist after remission of psychopathological symptoms but little is known about the pathophysiological events linking depression and cognitive impairment. Novel biological, structural and functional neuroimaging techniques have allowed a better definition of this relation. Depression and cognitive dysfunction share a common neuropathological platform in cortical and sub-cortical brain areas implicated in emotional and cognitive processing which may be under the control of genetic and environmental factors.

Key words: depression - cognitive impairment - magnetic resonance imaging

INTRODUCTION
The public health implications of depression and cognitive impairment are enormous. The World Health Organization Global Burden of Disease Study ranked depression as the most burdensome disease in the world in terms of total disability-adjusted years among people in the middle years of life (Murray & Lopez 1996). Cognitive symptoms appear to represent one of the core features of depressive disorders with an impact on many functional outcomes (Atre-Vaidya et al. 1998, Martinez-Aran et al. 2004).

COGNITIVE DYSFUNCTION IN DEPRESSION
Several evidences have suggested that depression increases the risk of cognitive impairment and functional disability (Lebowitz et al. 1997, Charney et al. 2003). On the other hand, cognitive dysfunction during remission may also play a critical role in increasing the individual’s vulnerability for the first onset, maintenance and future recurrence of depressive episodes (Gotlib et al. 2010, Kessing et al. 2001). Cognitive symptoms, such as difficulty making decisions and poor concentration, are included in the DSM-IV diagnostic criteria for major depression. However, in the recent literature regarding depressive cognition we also find consistently implicated working memory, attention and executive dysfunction and processing speed (Doumas et al. 2012, Elderkin-Thomson et al. 2010, Rosenberg et al. 2010, Marazziti et al. 2010, Nakano et al. 2008, Weiland-Fiedler et al. 2004).

During the past years, there has been an increased interest in cognitive impairment in depression, as testified by numerous studies. Initially, cognitive impairment has been attributed to depressive symptoms and studies have involved patients during the acute phase of depression. However, in the last decade it has been widely reported that cognitive dysfunction remains unresolved even after remission of depressive symptoms (Reppermund et al. 2009, Smith et al. 2006, Biringer et al. 2005, Paelecke-Habermann et al. 2005, Weiland-Fiedler et al. 2004). Moreover, some authors have suggested that impairment of cognitive measures is not correlated to depression severity and psychiatric co-morbidity (Majer et al. 2004, Bearden et al. 2006, Wang et al. 2006, Reppermund et al. 2009, Castaneda et al. 2010). In addition, cognitive deficits have been reported in healthy first degree relatives of patients with unipolar depression (Christensen et al. 2006). Taken together these data provide evidences for a dissociation between cognitive function and psychopathological symptoms in depression.

Although it is clear that the presence of cognitive deficits in depression is independent of the clinical remission of psychopathological symptoms the reasons for poor cognitive performance in depression remains unclear and little is known about the pathophysiological events linking cognitive impairment and depression. In this article we tried to identify and briefly analyze some of these events with the contribution of data obtained with novel biological, structural and functional neuroimaging techniques.

STRUCTURAL NEUROIMAGING EVIDENCE
In vivo structural and functional imaging studies, as well as postmortem investigations suggest that frontal–striatal–thalamic and limbic–thalamic–frontal networks have an important role in the pathogenesis of depression by regulating mood, cognition and behaviour (Mayberg, 2003, Price & Drevets, 2010). White matter hyperintensities and abnormal gray matter in dorsolateral prefrontal cortex, cingulate cortex, orbito-frontal cortex and hippocampus are commonly reported in depression.
In our brief review we have tried to identify the main pathophysiological events relating depression and cognitive deficits. We suggest that cognitive impairment and depression are linked by structural and functional alterations in cortical and sub-cortical brain areas regulating processing of emotional and cognitive information. Genetic polymorphisms (BDNF, GSK3B, 5HTT-LPR) and negative life events have been correlated with emotional and cognitive control and some of the above mentioned brain alterations (Inkster et al. 2009, Gatt et al. 2009, Yang et al. 2010, Juhasz et al. 2011, Molendijk et al. 2012). However, little is known about gene-environment interaction and the complex functional architecture underlying the integration of depression and cognition. Future research in this field may add knowledge with potential clinical and therapeutic implications.

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