Psychomotor disturbances in bipolar disorder - investigations using structural and functional neuroimaging

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Abstract

Psychomotor disturbances in bipolar disorder are an expression of alterations in volition, cognition, emotion and motor control. While psychomotor disturbances remain a classic hallmark of severe mood disorders, there is limited knowledge about its mediators and neural correlates.

The general aim of this thesis was to obtain a better understanding of psychomotor disturbances in bipolar disorder. Our specific objectives were to investigate whether symptoms of psychomotor disturbances in bipolar depression relate to neural activation in frontal-striatal networks that mediate motor function, whether there is a differential activation within these frontal-striatal motor networks between depressed patients with bipolar disorder and healthy controls, and whether morphometric changes in the basal ganglia and thalamus are associated with clinical variables in euthymic bipolar disorder that may predict impairments in psychomotor function.

In paper I, we validated a self rating scale with respect to established observer rating scales. In a post-hoc analysis we found a psychomotor factor that we investigated further with functional magnetic resonance imaging (fMRI) in paper II and III.

In paper II, we investigated motor execution in patients with bipolar depression. We used task based fMRI to investigate whether self paced finger tapping would reveal any differences in neural activation between groups, and whether neural activation at different levels in the frontal-striatal motor loop is predicted by the functional deafferentiation theory - framework used to explain slowed movement in Parkinson’s disease. We could not confirm our hypotheses. In paper III, we investigated different parts of the production of voluntary movement using fMRI and a motor imagery task. We found significant between-group differences in medial parieto-occipital regions during motor imagery and all other tasks, and in cortical motor areas during motor execution. We also found decreased activations in motor regions when there was an increase in psychomotor disturbances.

In paper IV, we investigated whether tests of psychomotor function were associated with morphometric change in the basal ganglia or thalamus. We could not confirm our hypothesis. However, we found significant between-group differences in the shape of the right putamen in the absence of impaired psychomotor function. Shape differences were located in regions connected to frontal executive regions and motor areas. In paper V, we investigated morphometric differences in a subgroup of bipolar disorder characterized by greater impairment of psychomotor function in their euthymic phase. We also investigated clinical variables associated with disease expressions, and the effect of antipsychotic treatment, on morphometric change. We found that antipsychotic medication, the number of manic episodes and duration of illness were associated with local shape changes in the basal ganglia.

In summary, we found that psychomotor disturbances may be considered both a symptom and a sign, and that the neural signature of these appear to involve both structural and functional alterations in brain regions of frontal-striatal networks.