Cortical Expression of Cognitive Control in the Bipolar Disorder Revealed by fMRI

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Introduction

The deficits in executive functioning, sustained attention, and memory in patients with BD have attracted several explanations. The residual deficits in executive functioning in euthymic BD patients merit particular attention, because they raise the possibility of primary cognitive changes that are at least independent of mood state. In general, this contributes to poor cognitive functioning, and any improvement would be of great clinical salience. Evidence to date suggests that the primary abnormality in BD is likely to be a dysfunctional prefrontal cortex. Prefrontal cortex serves several cognitive control functions. Comparisons among different cognitive tasks for the same BD group were barely conducted. Two cognitive control tasks, i.e. an n-back and a Go-NoGo task, were administered to a BD group and their control. Relationship of executive control and inhibition in BD can be examined and specified.

Method

Eight BD and eight control subjects were recruited. The BD patients were screened by the experienced psychiatrist. We used a 3-T MRI for fMRI experiment. Behavioral data were recorded at the same time. In the n-back task, 1- and 2-back conditions with different processing load were included. In the Go-NoGo task, the subjects responded to stimuli with higher proportion, and withheld the response for a target with lower proportion of its display. Functional imaging data were analyzed using SPM2.

Results and conclusion

Both tasks activated prefrontal cortex for cognitive control processing in the two groups. However, as compared to the control, BD patients showed activation weakness, especially in the prefrontal cortex. For the n-back results, BD caused decrease in responsiveness of the network for executive operation of working memory. In the contrast of 2- to 1-back condition, the BD group showed a pattern of right-lateralization while the pattern of the control group was different. These results seemed to suggest that BD results in inefficiency in resource recourse when the processing load of working memory increases.

In the Go-noGo task, while the BD subjects performed the Go response comparably to the control, activation in the prefrontal cortex for inhibition or monitoring processing disappeared, indicating a malfunction in the relevant neural networks. For the NoGo event, it is a transient effect of cognitive control to withhold or re-rout one's action plan. The results showed that the BD subjects lost activation in the ACC and bilateral DLPF, but not in the primary areas for motor execution. Implication seems to be that there is a difficulty to successfully recruit these cortical structures for cognitive control in the BD patients, even though the motor organ for action execution still works well.

Figure 1. Activation maps of the two subject groups for the two tasks