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Vulnerability indicators in bipolar disorder

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Background

Trait cognitive dysfunction in Bipolar Disorder (BD) may be an expression of genetic vulnerability. The aim of this project was to delineate core cognitive deficits in unaffected BD siblings and offspring.

Materials and methods

We recruited 75 unaffected relatives and 71 controls. 33 had lifetime (23 offspring and 10 siblings) diagnoses of major depressive disorder (n=21), anxiety disorders (n=4), substance abuse (n=11) and eating disorder (n=1). All participants underwent assessment of their general intellectual ability, memory, working memory, response inhibition and emotional learning (EL). Level of symptomatology was assessed using Hamilton Depressive Rating Scale (HDRS), Young Mania Rating Scale (YMRS) and Brief Psychiatric Rating Scale (BPRS). We conducted two analyses; one with whole sample siblings and offspring and another including asymptomatic ones defined as scoring 24 on the BPRS.

Results

Whole sample: Siblings analysis: Compared to controls (a) Healthy siblings showed deficits in inhibition and EL (b) Siblings with a lifetime diagnosis were additionally impaired in auditory delayed memory. Healthy siblings over performed both lifetime diagnosis siblings and controls in the working memory task Offspring analysis: (a) Compared to controls both healthy and lifetime diagnosis offspring were impaired in response inhibition and EL. Healthy offspring performed similar to controls in the working memory task. Asymptomatic: Siblings and offspring showed inhibition deficits but were not impaired in visual immediate memory and EL. The pattern of performance in working memory was similar to the whole sample.

Conclusions

Response inhibition may reflect genetic predisposition to BD, irrespective of phenotype while abnormalities in delayed auditory memory may relate to disease expression, irrespective of specific diagnosis. Enhanced performance in working memory may protect against disease expression.

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