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Cognitive vulnerability indicators in bipolar disorder and schizophrenia

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Background

The aim of this project was to investigate the cognitive abnormalities in healthy individuals (No Axis I or II disorders) at high risk for bipolar disorder (BD) and schizophrenia (SZ).

Materials and methods

Participants were 17 BD and 15 SZ relatives (BD-R and SZ-R) respectively and 23 controls. All participants underwent assessment of Full Scale IQ (Wechsler Adult Intelligence Scale Revised, WAIS-R), verbal memory and learning (California Verbal Learning test, CVLT), working memory (N-back), inhibition (Hayling Sentence Completion Task, HSCT), verbal fluency (Controlled Oral Word Association, COWA), working memory and inhibition (alphabet and number words tasks). Lack of lifetime Axis I and II disorders was screened using Structured Clinical Interview for DSM-IV (SCID-I/II) and symptomatology was assessed with the Hamilton Depression Rating Scale (HDRS), Young Mania Rating Scale (YMRS) and Brief Psychiatric Rating Scale (BPRS).

Results

No difference was found in the WAIS-R. Loss of inhibition was found for both relatives' groups compared to controls (HSCT). BD-R and SZ-R underperformed compared to controls in short and long delay recall in the CVLT (CVLT) whereas SZ-R made additionally more intrusions and perseverations. SZ-R and BD-R produced fewer words compared to controls whereas the former group also made more errors (COWA). The SZ-R had fewer total correct responses overall and in the 3-back condition. In Alphabet and number words tasks the SZ-R failed to inhibit relatively fast erroneous responses, leading to an effect on error rates but not in reaction times. Effect of BPRS total score was found only for BD-R across all measures apart from the HSCT.

Conclusions

Genetic predisposition to SZ may be mediated by deficits in the Ventral Prefrontal Cortex (VPFC), Dorsal Prefrontal Cortex (DPFC) and temporal networks. In BD-R impairment was limited in the VPFC whereas the DPFC function was preserved. The two disorders share inhibition deficits associated with the VPFC.

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