ABSTRACT

This study examined neurological and neurocognitive deficits as familial vulnerability factors to schizophrenia. Twenty-three Chinese schizophrenic patients, 21 of their non-psychotic siblings and 26 healthy volunteers, matched for age, sex and education, were assessed by using the Cambridge Neurological Inventory and a battery of neuropsychological tests including, Wisconsin Card Sorting Test (WCST), semantic verbal fluency, logical memory, digit span, information, comprehension and similarity. The patients fulfilled the DSMIV diagnostic criteria for schizophrenia. They were assessed when they were clinically stable upon discharge from hospital. The siblings and controls were screened for any psychiatric diagnosis by using the Structured Clinical Interview for DSMIIIR (non-patient version). The test performance of the patients was compared with that of their siblings and unrelated healthy controls. The results showed that siblings had significantly more global neurological signs, disinhibition signs and less word output in verbal fluency test as compared to controls. These abnormalities can be considered as inherited trait markers for schizophrenia and may point towards an underlying left fronto-temporal dysfunction. The other deficits including extra-pyramidal signs, perseverative errors in WCST and comprehension were found in patients but not their sibling counterparts. They are likely to be disease related state markers in view of their association with clinical symptoms particularly negative schizophrenic symptoms. Relationship between the residual symptoms after an acute psychotic episode and the magnitude of familial risk was examined. The result showed that more severe residual symptoms of probands at clinical remission could be predicted by their older age of onset and better verbal fluency performance in their non-psychotic siblings. This tentatively suggests that patients with milder genetic form of schizophrenic illness may have

more severe environmental contribution to cerebral insult according to the multifactorial/threshold model. The environmental cerebral insult may cause structural abnormalities leading to incomplete remission of clinical symptoms. The inherited trait deficits identified in this study can serve as potential candidate phenotypic markers for further molecular genetic research on schizophrenia.

Keywords: schizophrenia, neurological signs, neurocognitive dysfunction, inherited trait markers, discordant siblings

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