Persistent early childhood developmental impairment may be associated with later schizophreniform disorder

Cannon M, Caspi A, Moffitt TE et al. Evidence for early-childhood, pan-developmental impairment specific to schizophreniform disorder. *Archives of General Psychiatry* 2002 May; 59: 449–56.

QUESTION: Can developmental impairments in childhood predict psychotic symptoms later in life?

Design

Population-based longitudinal cohort study.

Setting

Dunedin, New Zealand.

Participants

1037 children born in 1972–1973 enrolled in the Dunedin Multidisciplinary Health and Development Study. Full data were available for 96% (980) of participants alive at age 26.

Assessment of risk factors

Participants were assessed biannually for intelligence; emotional, behavioural and interpersonal problems, and motor and language development between 3–11 years using several different scales. At 11 years, psychotic symptoms were assessed.

Main outcome measures

At 26 years, DSM-IV diagnoses were made using the Diagnostic Interview Schedule. Those with

schizophreniform disorder (3.7%) were compared with healthy controls and people diagnosed with mania (2%) and non-psychotic anxiety or depressive disorders (29%).

Main results

Interpersonal difficulties and emotional problems during childhood were more likely among adults with general psychiatric disorders. Additional neuromotor, language and cognitive impairments were present only in children later diagnosed with schizophreniform disorder. Persistent developmental impairment also predicted self reported psychotic symptoms at 11 years, independent of socio-economic, obstetric and maternal factors.

Conclusions

Childhood developmental impairment may be associated with schizophreniform disorders in early adulthood.

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For correspondence:
T Moffitt, Social
Genetic and
Developmental
Psychiatry Research
Centre, Institute of
Psychiatry, London.
t.moffitt@iop.kcl.ac.uk

COMMENTARY

Many somatic diseases in adulthood may have their origin in childhood or even pregnancy. Mental disorders may also have childhood antecedents. Since the time of Kraepelin around the turn of the 19th century, subtle motor, emotional, cognitive and behavioural abnormalities have been observed in apparently healthy children who later develop schizophrenia.¹ This suggests that some aspects are established long before psychosis manifests. Past studies have found developmental delays at one point in time (for example, learning to stand, walk or speak; difficulties at school, or cognitive performance).² This study suggests early onset, pan-developmental and persistent impairments are specifically associated, not with DSM IV schizophrenia, but with the broader diagnostic group of schizophreniform psychoses.

It may be difficult to achieve statistical power in studies comparing uncommon risk factors (such as clear-cut childhood developmental impairments) and relatively rare outcomes (such as schizophrenia or schizophreniform psychoses). In this paper, repeated testing partially alleviated problems related to small sample size. The authors also attempted to overcome power problems by broadening diagnostic criteria from a narrow definition (DSM IV schizophrenia, n=10) to include all schizophreniform psychoses (n=36). Because diagnoses were pooled, the question of whether DSM IV schizophrenia differs from other psychoses remains unanswered.

It is difficult to estimate the risk of psychosis in healthy people or even those with non-psychotic psychiatric problems. Developmental delays are widespread and most are within the wide normal range. Most children with impairments will develop normally. It is difficult to separate those who will develop psychosis from the larger group with a more or less normal life span. Early recognition, intervention and prevention programmes based on developmental models are relatively recent. No single premorbid risk indicator has been identified that is specific to schizophrenia or that justifies active intervention before psychotic symptoms. No antecedent factor has been identified that is useful for prediction in the general population. The number needed to treat for any of the risk factors identified is high. Immediate prospects may be better in a clinical sample who may be experiencing the pre-psychotic phase – a sample which was not used in this article. Other advances come from selected 'ultra-high-risk' series suggesting strong genetic liability or clear prodromal symptoms.

Although there is little evidence for interventions among people at increased risk of psychosis, clinical experience justifies information, observation and psychosocial support for people at risk of any Axis I disorder. Major developmental impairments may be one important reason to lower the threshold of intervention, although this has not been empirically researched. The life course approach links neurobiology, epidemiology and clinical research into pathways of asymptomatic, subthreshold and threshold disease stages over the life span.³ To date, this has had more theoretical than practical relevance. Future predictive models may combine data from childhood and adolescent deficits, premorbid development and clinical symptomatology, genetic factors and structural brain and cognitive abnormalities.

Professor Matti Isohanni
Department of Psychiatry
University of Oulu, Finland

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