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26th European Congress of Psychiatry

Plenary Lecture

Plenary Lecture: Schizophrenia is a Myth with a Strong Genetic Component

PL0001

Schizophrenia is a myth with a strong genetic component

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Traditional psychiatric textbooks describe schizophrenia as a disease of unknown aetiology. However, this is wrong on two counts. First we now realise that schizophrenia itself is not a disease but rather a clinical syndrome, and one with very fuzzy boundaries. It is simply the name that we give to the severe manifestation of psychosis, and we now know there exists a continuum which stretches into other so-called psychotic disorders, and indeed into the general population. Thus, psychosis is distributed though the population like hypertension, and schizophrenia is the equivalent of severe hypertension.

A recent study encompassing 16 sites across 5 European countries shows that the incidence varied widely, with the figures for South London and Amsterdam being more than 5 times higher than in some Southern European sites. Curiously, psychosis is more common in those living in large cities than in rural areas in Northern but not Southern Europe. These differences presumably reflect differences in exposure to risk factors.

We now know a great deal about the risk factors, or contributory causes, of psychosis. These turn out to be largely the same as for severe psychosis, i.e. schizophrenia. These can be roughly divided into two main types; those which result in (a) aberrant neurodevelopment and (b) those which cause dopamine dysregulation; both characteristic abnormalities found in schizophrenia.

Genetic factors are, of course, pre-eminent. In 2014 a landmark GWAS study of 37,000 people with schizophrenia and 113,000 healthy controls identified 108 loci significantly associated with schizophrenia. Each of these polygenes has only a very small effect but cumulatively they account for about 30% of the variance in occurrence of schizophrenia. Some such as neurexin or TCF4 subtly impair neurodevelopment while others such as DRD2 or AKT3 impact on dopamine signalling. A small proportion of schizophrenia (perhaps 3%) results from copy number variants (CNVs) impacting on neurodevelopmental genes; these CNVs can have a much bigger effect size, increasing risk 3- to 20-fold.

Various environmental factors have been consistently associated with schizophrenia. Some such as adverse obstetric events (e.g. prenatal infection, perinatal hypoxia) impair neurodevelopment. Others such as abuse of drugs such as amphetamines, cocaine and cannabis which increase striatal dopamine, also increase risk. In recent years it has become clear that heavy use of high potency cannabis is responsible for a significant proportion of psychosis (>20% in South London). Synthetic cannabinoids are an increasing cause of acute psychosis sometimes termed "spiceophrenia". A range of social adversities such as child abuse, adverse life events, migration/minority ethnicity appear also to facilitate dopamine dysregulation and consequent psychosis.

Disclosure of interest.– The authors have not supplied their declaration of competing interest.

PL0002

High risk and resilience studies: Lessons for prevention, care and research

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Background.– Severe mental illnesses like schizophrenia and bipolar disorder are known to be diseases that to some extent, but not entirely can be understood genetically. For schizophrenia the dominating hypothesis is that it is a neurodevelopmental disorder, and that genes, environment as well as gene-environment-interactions contribute to the risk of developing the disease. Children born to parents with severe mental illness are at a higher risk of growing up under unstable life conditions and more frequently show early signs of vulnerability for mental illness themselves.

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Aim.– We aim to analyse the influences of genetic risk and environmental factors in a population of 7-year-old children with either 0, 1 or 2 parents diagnosed with schizophrenia spectrum psychosis or bipolar disorder. Main outcomes are psychopathology, neurocognition and development.

Methods/design.– We have established a cohort of 522 7 year old children and their parents for a comprehensive investigation of the children's neurocognitive, social, behavioural and neuromotor function and psychopathology. The test battery included a large neurocognitive battery, home visits evaluating the degree of stimulation and support in the home, information from teachers, and measures of stress, and attachment. The parents take part in an anamnestic interview about the child. The participants were recruited via Danish registers to ensure representativity. Data from registers concerning social status, birth complications, somatic illnesses and hospitalization are included.

Results.– Generally the children with parents with schizophrenia had lower neurocognitive function, more psychiatric symptoms and poorer motor function. Children with parents with bipolar disorder were not different from control children.

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