Brain

Towards a Natural History of Schizophrenia

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All scientific knowledge relies on observation, but with complex phenomena there is often an awful lot to observe. Schizophrenia is a neuropsychiatric disorder characterized by a wide range of symptoms such as delusions, hallucinations, lack of motivation, cognitive difficulties, impaired speech and aberrant motor functioning. Beyond the behavioural level of symptoms, however, additional hallmarks of the disorder have been discovered across every level of neuroscientific investigation. Epidemiological studies have shown, for example, strong heritability, late-adolescent onset, and higher incidence in males than females. Neuroimaging studies have found characteristic alterations in both structural and functional brain connectivity. Pharmacological studies have uncovered disruptions in dopamine function. Histological studies have implicated parvalbumin-containing GABAergic neurons. Transcriptomic studies of post-mortem brain tissue have highlighted hundreds of differentially expressed genes, while genome-wide association studies have identified scores of risk loci in the genome. However, this rich, multi-scale description of 'what' the disorder is makes it ever more challenging to advance mechanistic hypotheses for 'how' these complex phenotypes come about. In this issue of *Brain*, van den Heuvel and co-workers cannily propose that we may get closer to understanding 'how' schizophrenia emerges by focusing first on the apparently harder question of 'why' it exists at all (van den Heuvel *et al.*, 2019).

Schizophrenia has long been understood as a heritable—and therefore genetic—disorder. As such, its relatively high population-wide prevalence (~1%) presents an evolutionary puzzle

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