

COMMENTARY

What neuroscience has already done for us

Commentary on... Why hasn't neuroscience delivered for psychiatry?[†]

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Summary Each of the components of the biopsychosocial model of mental illness is important for understanding mental illness. Biological and genetic abnormalities have been demonstrated in major mental illnesses. These are leading to changes in our understanding of these conditions, as well as our understanding of the link between life events and mental illness.

Declaration of interest None.

Keywords Schizophrenia; depressive disorders; neuropathology.

As readers will be well aware, the biopsychosocial model has underpinned psychiatry for several decades.¹ Each component of this model is important for our understanding of mental illness. Professor Kingdon is therefore correct to say in his interesting editorial that neuroscience is unlikely to hold all of the answers to why people develop mental disorders and when they occur in their lifetime.² I challenge, however, his assertion that 'biological changes have yet to be shown to be relevant to the major mental disorders'.

Brain imaging and schizophrenia

Taking schizophrenia as an example, there are clearly demonstrable differences in the brains of individuals with schizophrenia compared with those of controls. It was first shown in the 1970s that people with schizophrenia had enlarged cerebral ventricles.³ Since then abnormalities in both grey and white matter have been convincingly demonstrated in the disorder.⁴ More sophisticated brain-imaging techniques have allowed the discovery in recent years that differences in brain volume are present even in medication-naïve individuals with first-episode psychosis.⁵ It seems increasingly likely that

there are differences in volume even before the at-risk mental state, although this remains difficult to prove definitively.^{6–8}

Recently developed scientific techniques such as the use of induced pluripotent stem cells to create a 'cortex in a dish' (*aka* brain organoids) have allowed tantalising insights into why these imaging abnormalities may emerge. Using cells from individuals with schizophrenia (some with a range of predisposing genetic abnormalities) multiple studies have shown abnormalities such as impaired cellular differentiation and synapse formation.^{9–11}

These studies add to the emerging hypothesis that the brain of someone at risk of schizophrenia differs from controls at an early stage and that these differences increase as psychosis emerges. These neuroscientific findings are a good fit with the long-standing findings from more psychosocially focused research that differences can be seen in childhood behaviour in those who later develop schizophrenia.^{12–15} They also suggest that, to develop better treatments for schizophrenia, we need to look beyond compounds targeting dopamine receptors.

Genetics in intellectual disability and depression

I would also challenge Professor Kingdon's assertion that no genetic findings of use to the practising psychiatrist have

[†] See this issue.

been found for the major mental illnesses. Genetic testing for copy number variants is starting to form part of practice in intellectual disability services.^{16,17} Using his example of depression he is correct to say that the much vaunted candidate genes studied in the 1990s and 2000s have not been replicated in later, large studies.¹⁸ However, more recent, vastly better powered studies have produced findings of greater potential use. The most recent genome-wide association study on depression found 87 independent loci that were associated with depression, with a startling lack of genes involved in the 5-HT system.¹⁹ This may suggest that, although drugs acting on the 5-HT system are effective in treating depression for many people, disturbances in 5-HT are not the cause of depression. Findings such as these are likely to be of great benefit in developing new treatments.

Epigenetics and treatment targeting

Neuroscience can also help us to explain the link between life events, which are frequently assessed in psychosocial research, and mental health outcomes. For example, epigenetic studies have shown that maternal behaviour influences the expression of genes, including those involved in the glucocorticoid stress response.^{20,21} Because this work was done in rats it was possible to demonstrate that this effect was not genetic as it was abolished by cross-fostering with more affectionate mothers.²¹ Childhood maltreatment such as physical abuse has long been recognised as a risk factor for mental illness. Recent genetic and epigenetic studies are helping us to understand why some people are more resilient to the effects of this abuse than others.^{22,23} It has been suggested by some authors that this information could be used to better target childhood interventions, such as providing more intensive interventions to those likely to be least resilient to the effects of childhood maltreatment.

Conclusions

Our understanding of mental health problems has started to change radically in the past few decades. It is only 50 years since it was widely believed that parents could be responsible for their offspring developing schizophrenia.²⁴ Within the past 50 years patients with intractable epilepsy were cared for in psychiatric hospitals, something that would now be unthinkable, and the parent-child relationship was seriously considered as a cause of epilepsy.^{25,26}

This improvement in understanding has the potential to reduce stigma, to ultimately lead to new treatments and to provide patients with a better understanding of what is happening to them and why. It is critical that mental health researchers work together, rather than in methods-based silos, to further improve our understanding of why and how patients develop mental health problems.

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SPECIAL ARTICLE

The emotional and mental health needs of young carers: what psychiatry can do

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Aims and method To review the literature on the emotional and mental health needs of young carers of parents with mental illness and the extent to which such needs are recognised and supported by professionals. Three databases were systematically searched from 2008 to 2018, and five studies met the inclusion criteria.

Results The key findings were that young caregivers had a significantly higher dose-response mortality risk than their peers; were at increased risk of mental health difficulties, especially where the ill family member was a parent and had mental illness or misused substances; were overlooked by professionals owing to a lack of awareness; but could derive benefits from their caring role when appropriately supported.

Clinical implications Young carers are at increased risk regarding emotional and mental health needs; this risk could be mitigated by professionals recognising the young carer's role and including them in their parent's treatment plan.

Declaration of interest None.

Keywords Young carers; mental health; psychiatry; systematic review.

Background

Societal awareness of young carers and the potential effects of caring on their health and development has increased in the past 20 years.¹ A 'young carer' is 'a child or young person under 18 who provides regular or ongoing care and emotional support to a family member who is physically or mentally ill, disabled or misuses substances'.^{2,3} The 2011 Census in England and Wales showed that 166 363 children in England

cared for their parents, siblings or family members, an increase of 20% on the number recorded in the 2001 Census.^{4,5} However, this was thought to be an underestimate.² The prevalence of informal caring in the underage population was estimated as a minimum of 2–4% in Western countries.⁶

Dearden and Becker reported that most young carers cared for parents, particularly mothers, although some provided support for grandparents, siblings or other relatives.