BJPsych The British Journal of Psychiatry (2023)
223, A37. doi: 10.1192/bjp.2023.110

Highlights of this issue

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Can't make bricks without clay

When I was growing up (well, when I was completing my PhD), I wanted to be an epidemiology detective.[1] I imagined myself using clues hidden in numbers to deduce the culprit in clinical problems while wearing a silly-looking hat. In this issue of the *BJPsych*, several population studies reveal many hidden and unexpected clues for clinical practice.

First, Tiihonen and his team have done it once again. I first fell in love with his work when I read the FIN11 study as a first-year trainee.[2] The latest Finnish national cohort study examines over 60 000 patients with bipolar disorder to investigate the real-world effectiveness of various medications (pp. XX–XX). Two main take-home messages for me were: (a) many long-acting injectable antipsychotics are effective at reducing psychiatric hospital admission (olanzapine, haloperidol and zuclopenthixol, but not paliperidone, aripiprazole or risperidone); and (b) despite increasing clinical hesitancy to use it because of perceived intolerability, lithium is associated with a significantly reduced risk of somatic hospital admission. Further, the study found that although only two oral antipsychotic medications (clozapine and levomepromazine) were effective at reducing the risk of psychiatric hospital admission, most oral mood stabilisers (lithium, carbamazepine, lamotrigine, sodium valproate and even pregabalin) were effective in the real world. Given its combination of effectiveness and tolerability, it appears that lithium remains *numero uno* for bipolar disorder more than 70 years after John Cade's discovery.

Moving from Tiihonen to Taiwan, Tsai et al (pp. 465–470) linked the Taiwan National Insurance Research Database (consisting of healthcare data for more than 99.7% of the entire Taiwanese population) and the Database of All-Cause Mortality to investigate the familial coaggregation of deaths by suicide with major psychiatric disorders. Using case–control matching, they found that first-degree relatives of individuals who died by suicide were at increased risk of death by suicide and accident, as well as being at higher risk of being diagnosed with various psychiatric disorders including schizophrenia, bipolar disorder, major depressive disorder and attention-deficit hyperactivity disorder (ADHD), compared with those without a first-degree relative who died by suicide. Of note, the sons of mothers who died by suicide and the mothers of daughters who died by suicide were at the highest risk of suicide themselves. These findings suggest that family history of suicide is a significant risk factor for many psychiatric outcomes.

Thapar et al (pp. 472–477) used longitudinal data from the National Child Development Study in the UK to examine whether childhood ADHD-like problems at age 7 were associated with cardiovascular risk factors at age 44 or 45. Although the study found statistically significant increases in body mass index (28.3 kg/m^2 *v.* 27.3 kg/m^2), systolic blood pressure (129.9 mmHg *v.* 126.4 mmHg) and diastolic blood pressure (80.9 mmHg *v.* 78.7 mmHg) among those with childhood ADHD-like problems, the clinical significance of these differences appears doubtful. Nevertheless, given that most participants in the cohort (who were born in 1958) were unlikely to have received ADHD medications, these findings reinforce the importance of monitoring cardiovascular risk factors for those with ADHD, especially with escalating awareness of and treatment for ADHD today.

In another study from the UK in this issue, Hotham et al (pp. 478–484) examined the cross-sectional associations of cannabis, cannabidiol and synthetic cannabinoid use with depression, anxiety or conduct-disorder-like symptoms, as well as auditory hallucinations, among 13- to 14-year-olds. The study found that young people who used cannabis, cannabidiol or synthetic cannabinoids were more likely to report mental health symptoms than those who did not. They were, however, also more likely to live with parents who were unemployed or had low income, and more likely to drink alcohol and smoke cigarettes. Correlation does not equal causation, but there is enough in the data for us to be concerned about the increasing access to cannabis, cannabidiol and synthetic cannabinoid in many countries worldwide.

Finally, Bonivento et al (pp. 485–492) compared the neurocognitive profiles of individuals with recent-onset psychosis, recent-onset depression or clinical high risk for psychosis with those of healthy controls. Those with recent-onset depression did not show any significant neurocognitive deficit compared with healthy controls, but significant deficits were present among those with recent-onset psychosis and, to a lesser degree, those at clinical high risk for psychosis. The authors argue that the findings may point to a specificity of neurocognitive deficits as markers of vulnerability to psychosis. To me, however, this is a clue that has yet to reveal a clear culprit.

'Data! Data! Data!' Sherlock Holmes cried impatiently. 'I can't make bricks without clay.' Like Holmes, we psychiatrists can't make solid clinical decisions without reliable research evidence. Thankfully, this issue of the *BJPsych* contains tons of data to help us solve clinical mysteries.

References

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2 Tiihonen J, Lönnqvist J, Wahlbeck K, Klaukka T, Niskanen L, Tanskanen A, et al. 11-year follow-up of mortality in patients with schizophrenia: a population-based cohort study (FIN11 study). *Lancet* 2009; **374**(9690): 620–7.