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Testing a hypothesis arising from the epidemiology of schizophrenia in New Zealand

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A 2011 publication in *World Psychiatry* thoughtfully, but somewhat pessimistically, addressed the question of how schizophrenia might be prevented.[1] A review of causal evidence[2] a decade later offers little further hope. We hypothesise that an emergent increase in syndromal schizophrenia in New Zealand, particularly in Maori (descendants of the indigenous Polynesian population), may present opportunities for prevention.

The concept and epidemiology of schizophrenia have generally recognised it as a syndrome, rather than a disease or specific illness. Many theories of aetiology or pathogenesis have been enthusiastically propounded, often to fall by the wayside as unconfirmed or as only minor contributors to pathogenesis. Variable genetic predisposition with multiple possible environmental triggers has been the main fallback position.[3]

In recent years two groups of researchers have attempted to quantify the relative epidemiology of schizophrenia in New Zealand Maori (15% of the population) and non-Maori.[4 5] Using quite different research designs, the researchers both demonstrate significantly increased prevalence and incidence of schizophrenia in Maori compared with the remainder of the New Zealand population. The reported two to threefold increased incidence far exceeds a plausible contribution of misdiagnosis arising from cultural factors. Moreover, there is no evidence that this ethnic imbalance in incidence and prevalence existed prior to the turn of the century. Notably, the increases are at double the rate in Maori compared with non-Maori (71% vs 35%) in the context of observed population-corrected increases in mental health service use in the first 16 years of this century (data provided by the New Zealand Ministry of Health). When analysis is limited to those discharged from inpatient services with a diagnosis of schizophrenia (F20 in the International Classification of Diseases 10th Revision), the increases are 85% and 54%, respectively. Thus, in the absence of significant changes in mental health service availability, there is good evidence of increased mental health service delivery to people diagnosed with schizophrenia, particularly in the Maori population.

A prospective Swedish study[6] published in *The Lancet* in 1987, and more recently replicated by others,[7 8] identified the ability of some illicit drugs, particularly amphetamines and potent cannabinoids, to produce a disorder mimicking schizophrenia in course and chronicity. Accordingly, a proportion of patients with syndromal schizophrenia experience the disorder as a consequence of drug use. With both cannabinoids and methamphetamine, the increased risk of schizophreniform psychosis is dose-related,[9] and thus depends on both frequency and quantity of use.

It is now clear that the availability and use of methamphetamine in particular has risen markedly in New Zealand over the last 10 years. A New Zealand Police Insight Report of April 2018 notes that the numbers arrested and detained while under the influence of methamphetamine increased approximately tenfold between 2010 and 2017 (data provided by the New Zealand Health Quality & Safety Commission). Also of note is the observation that the tetrahydrocannabinol content, and thus psychoactive potency, of New Zealand cannabis has increased markedly since 1996.[10] A major study of the community prevalence of mental disorders in New Zealand found substance abuse significantly over-represented in Maori.[11] Dharmawardene and Menkes[12] also found cannabis use/abuse twice as common in a Maori compared with non-Maori clinical population.

The correlational evidence strongly suggests that significant increases in the availability

of potent cannabinoids and methamphetamine may be related to the apparent surge in the rate of schizophrenia in the general population of New Zealand and particularly to the now apparent greater rate in those of Maori ethnicity. These associations are consistent with those suggested previously regarding clinical populations in Japan, China, various European countries, and Brazil.[13–16] While correlation does not establish causality, the apparent dose–response relationship[9] supports causal inference as does the fact that the findings are consistent across changes in time, place and culture.

Although adequately powered, prospective, longitudinal studies will be required to establish causality, we propose that the available correlational evidence is sufficiently strong to encourage the planning and commitment of resources to relevant preventive measures.

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