Migration and psychosis: our smoking lung?

To read the history of humankind is to read a history of migration. From the first human exoduses out of Africa, to Greek and Roman empires which sought territorial expansion, to the Ming dynasty's pioneering voyages of exploration, to the flight of ethnic, religious, political and sexual minorities escaping persecution from various authoritarian regimes or internal conflicts, to the economic migrants from continental Europe, Asia, the Middle East, and South and Central America who sought better lives for themselves and their families on new continents, migration is arguably the defining feature of a singular human experience that binds our past, present and future. The drivers and consequences of migration also leave indelible marks on the history of humankind. Perhaps in equal measure, they result in leaps forward for civilization – enriching cultural, social, genetic and economic diversity and human development – and pockmarks which serve to remind us of the seemingly ceaseless bounds of human savagery and brutality (see also Silove et al[1] in this issue of the journal).

To a psychiatric epidemiologist, migration is arguably associated with one of the defining public health inequalities of the last 100 years: that certain migrants, their children, and their children's children are as much as 10 times more likely to meet diagnostic criteria for psychotic disorder than the majority (usually white Caucasian) population in a given setting[2]. The exact magnitude of this risk varies, depending on the given migrant group and setting in which the study is conducted. In the UK, for example, psychosis risk ranges from slight increases (of 1.5 or less) for white migrants, to 2-4 times greater risk for people of Pakistani and Bangladeshi origin, and up to 10 times higher rates amongst black Caribbean and African groups[3]. Elsewhere, elevated risk also follows historical migration flows, such as amongst the Surinamese and Moroccan populations in the Netherlands[2], or East African migrants to Sweden[4]. Emerging research from countries which have experienced unprecedented contemporary immigration pressures[5] also shows that incidence rates are elevated amongst migrant groups.

It is only right that this epidemiological literature is subject to proper scrutiny to determine whether these patterns are causal. If they are not, then the alternatives are no less palatable: that other social or economic exposures are so entrenched within certain black and ethnic minority (BME) sections of society that they are powerful enough to increase the chance of experiencing a psychotic disorder by up to 1000%; or that the tools, practitioners and institutions tasked with making reliable and valid diagnostic assessments are so unfit for purpose, or so grossly inept at differentiating between normal cultural mores of behaviour and psychotic symptoms, that for every one migrant correctly diagnosed, a further nine may be misdiagnosed with psychotic disorder.

Scrutiny of the evidence in relation to misdiagnosis does not strongly support this as an explanation of higher rates. There may be poor inter-rater reliability between psychiatrists in agreeing on a specific psychotic diagnosis, but this does not appear to be racially biased[6]. Further, few modern epidemiological studies rely solely on clinician-rated diagnoses to measure outcomes, instead using carefully operationalized criteria to reach standardized diagnoses[3,7]. Finally, in the UK and elsewhere, the ethnic composition within clinical psychiatry is increasingly diverse, far from the monochromatic contrast that implicitly surrounds the misdiagnosis debate. In a recent study, for example, which also found elevated rates of psychotic disorder in BME groups in rural England[7], operationalized diagnoses were made by a panel of psychiatrists from over 13 different ethnic backgrounds.

Further new empirical data offer important directions. For example, raised rates do not seem to be entirely attributable to socioeconomic differences between BME groups and the majority population[8]. Other recent research, from Sweden, has demonstrated that refugee migrants are at considerably elevated risk of non-affective psychotic disorders compared with both the Swedish-born population and, importantly, other migrants from the same regions of origin[4]. The implication is that severe exposure to pre-migratory adversities, including war, famine and persecution, or the hazards involved in the transitory process of migration itself, may be aetiologically relevant to psychosis risk. Exposure to other severely traumatic migration-related experiences, such as witnessing genocide[8], also increases schizophrenia risk. Nonetheless, these data would not explain why elevated rates persist in successive generations following the index immigration event. Other factors must be relevant, possibly including experiences of racism and discrimination, although further research is needed on this issue.

We also require more integration of observational data with sociological, ethnographic, experimental psychology and neuroscience research to shed light on the possible pre-, peri- and post-migratory factors that increase psychosis risk amongst BME groups. A recent study from social neuroscience, for example, suggests that healthy volunteers from second generation migrant backgrounds exhibit elevated neural responses to stress following a sociocultural challenge[9]. If we can elucidate whether these putative stress pathways also contribute to the onset of psychosis – potentially encompassing complex interactions between genetic, biological and social factors – this will not only move us closer to understanding the excess risks among BME communities, but in society at large. Aside from psychosis (and, perhaps, post-traumatic stress disorder), there is less consistent evidence that migrants are at higher risk of other mental health conditions; this specificity would be one of several important criteria helping to establish causation.

Further studies are also required in settings where the increased psychosis risk amongst migrants is not observed, such as in people of Indian descent in the UK[3], Turkish descent in the Netherlands[2], or Hispanic origin in the US[10]. Canada is another putative counterfactual setting, given both its foundation on a

relatively recent migration history, and the effects on mental health of indigenous First Nations people in this context.

Studies in settings where white migrants form the minority group would also shed further light on the role of migration in psychosis risk. South Africa provides a possible example. Nonetheless, while white migrants in this context would be the minority in terms of population size, they also continue to hold a disproportionate balance of socioeconomic capital, which may negate any effect; in either case, the aetiological implications would be illuminating. For various reasons, and not without considerable challenges, Brazil, China, Japan and Zimbabwe present other settings for such counterfactual study.

Using data from the UK, we have previously estimated that, if we could identify the drivers of the elevated psychosis risk in BME groups, we could prevent up to 22% of new cases of first episode psychosis in the general population, and up to two thirds in BME groups specifically[11]. This major health inequality may be to psychiatry what nicotine exposure was to bronchogenic carcinomas over 65 years ago[12]: our smoking lung. The psychiatric research community has an unparalleled duty to advance our aetiological understanding on this issue in order to eradicate this gross social injustice.

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