CHAPTER 9

The epidemiological burden of major psychiatric disorders

Jennifer Dykxhoorn and James Kirkbride

Key definitions

Incidence: The number (frequency, rate, or proportion) of new health-related events in a defined population within a specified period of time [1].

Prevalence: The total number of individuals with a health-related event at a particular time/period in a defined population [1].

  Point prevalence: The proportion of individuals with the condition at a specified point in time.

  Period prevalence: The proportion of people with the condition at any point during a specified time period (e.g. annual **prevalence** or lifetime **prevalence**).

Measures of effect: Measures of effect summarize the strength of the relationship between an exposure and outcome, showing the amount of change a particular exposure has in the frequency or risk of the outcome [2, 3]. Measures of effect are used to compare the frequency of an outcome between two groups in relative or absolute terms [2]. Common relative effect measures include odds ratios and relative risks. Absolute effect measures include risk differences or 'number needed to treat.'

Measures of potential impact: Measures of potential impact are measures that estimate ~~the~~ how much of the risk of a disease can be attributed to an exposure and, further, what removing the exposure would have on the exposed group or the population. Measures of potential impact are necessary to translate epidemiological evidence into policy-relevant information [4]. Measures of impact assume a causal relationship between the exposure and the outcome [4]. Impact measures include attributable fraction among the exposed and attributable fraction for the population.

Attributable fraction (attributable proportion): The proportion of cases that can be attributed to a particular exposure. It is the proportion by which the risk would be reduced if the exposure would be eliminated. It can be estimated for exposed individuals (attributable fraction among the exposed) or for the whole population (attributable fraction for the population) [1].

  Attributable fraction among the exposed: The proportion by which the ~~incidence~~ of the outcome among the exposed would be reduced if the exposure were eliminated [1].

Attributable fraction for the population ('population attributable risk' or PAR): The proportion by which the ~~incidence~~ of the outcome in the entire population would be reduced if the exposure were eliminated [1].

The burden of major psychiatric disorders

One striking feature of psychiatric disorders is that they are found in every human population worldwide. These disorders, which include depression and anxiety, schizophrenia, bipolar disorder, and autism spectrum disorders, contribute substantially to global burden of disease estimates. For example, in 2015, the aforementioned disorders were ranked in the top 21 of all disorders contributing to years lived with disability, with depression, anxiety, and schizophrenia ranked third, ninth, and twelfth, respectively [5]. Such severe morbidity is partly explained by the young age at onset for such disorders, which typically begin to emerge in childhood and adolescence, and may be associated with ~~both~~ lifelong episodes of mental ill health. In turn, several mental health disorders are now associated with reduced life expectancy as a result of both excess suicide rates in people with mental health problems, as well as worse physical health, health care, and lifestyle choices. Most strikingly, people with schizophrenia may have a reduced life expectancy of between 10 and 25 years compared with the general population [6].

It is clear that this burden of psychiatric morbidity and mortality presents an imperative issue for public mental health. Beyond this, improving and ameliorating poor mental health will have a corresponding effect on physical health, well-being, and quality of life. But before we can move to such a point, we require a firm understanding of the burden of psychiatric disorders in the population. This is important for two reasons. Firstly, quantifying this burden—here, either in terms of *incidence* or *prevalence*—will allow mental health service planners, commissioners, and those designing interventions to improve psychiatric health, to make informed decisions about how to allocate finite resources most efficiently within a healthcare system. One such example from England is the development of a population-level prediction tool, which applies empirical epidemiological data on the risk of psychotic disorders to regional population demographics, providing accurate data about the annual *incidence* of schizophrenia and other psychoses in different communities [7]. Secondly, an understanding of any variance (or

homogeneity) in *incidence* or *prevalence* of psychiatric disorders may inform or generate hypotheses about the possible causes of disorder.

In this chapter we provide an overview of the major epidemiological evidence describing the burden of three major psychiatric outcomes; common mental disorders (depression and anxiety), psychotic disorders (schizophrenia and other psychotic disorders), and suicide. Where major patterns of variation exist—by person or place—we also highlight these, with a special focus on the role of ethnicity and its implications for understanding the social and economic determinants of health. Because there is a substantial literature on these topics already, we have chosen to be selective rather than comprehensive in our treatment of the literature. We will refer to the major epidemiological studies conducted in psychiatric epidemiology over the past 30 years—typically in North America and Europe—as well as important systematic reviews and landmark studies. Finally, in this chapter, we briefly consider how this epidemiological data may inform possible interventions for public mental health.

Common mental disorders

A recent major systematic review of common mental disorders (CMD), which included both mood and anxiety disorders, placed the annual *prevalence* as 15.4% (95% confidence interval (CI) 12.8–18.6%) [8]. Remarkably, the pooled lifetime *prevalence* of disorders reported in this review rose to nearly one in three people (29.2%; 95% CI 25.9–32.6%). The annual *prevalence* of CMD appears to be almost twice as common in women as men, a pattern that holds for both mood (women: 7.3% (95% CI 6.5–8.1%); men: 4.0% (95% CI: 3.5–4.6%)) and anxiety disorders (women: 8.7% (95% CI 7.7–9.8%); men: 4.3% (95% CI 3.7–4.9%)). The same review found some global variation in these patterns, most notably with lower estimates from North and South East Asia, and Sub-Saharan Africa, with higher rates in English-speaking populations. It is unclear whether such differences reflect genuine ethnic, social, and cultural variation in the manifestation of mental health symptomatology, or may arise for other reasons, including possible biases in the cultural sensitivity of diagnostic tools to detect mental health symptoms in different settings.

Epidemiological studies of CMD are most typically conducted using cross-sectional designs of the general population to estimate past symptomatology (i.e. in the past week, year, or lifetime) meeting diagnostic criteria for a disorder. While such studies may be somewhat prone to recall, they permit estimation of *prevalence* for a set of disorders, which may be under-reported in studies solely reliant on hospital records or routine databases, as many people meeting diagnostic criteria for a CMD may never present to mental health services. *Incidence* studies of CMD are more rarely conducted, given this issue, and given that it may be particularly tricky to determine whether an episode of depression or anxiety is truly the first someone may have experienced. Furthermore, while CMDs are—vis-à-vis other psychiatric disorders—just that, relatively common, the absolute occurrence of episodes may be infrequent and require large sample sizes in order to provide precise *prevalence* estimates, or detect statistically robust differences in burden between different population subgroups. For these reasons, large, high-quality epidemiological studies of CMD are relatively infrequent.

Three of the largest and methodologically robust examples of their kind are the Epidemiologic Catchment Area (ECA) study [9], the National Comorbidity Surveys (NCS; I and II) [10]—both from the USA—and the Adult Psychiatric Morbidity Surveys (APMS) in the UK. The earliest of these three was the National Institute for Mental Health-funded ECA study, conducted in five sites to establish the 1-month and lifetime *prevalence* of psychiatric morbidity in the general population. The study represented a major advance in epidemiological enquiry of mental health disorders, using a standardized survey design across all five sites to establish the *prevalence* of mental health disorders according to validated diagnostic criteria (according to the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM-III)), obtained from standardized diagnostic interviews. It is difficult to overestimate the magnitude of this advance; the ECA study—almost for the first time—sought to differentiate 'global impairment' into specific, operationalized, and validated diagnostic categories. This approach recognized that differences in presentation may reflect underlying variation in aetiology, treatment, and care, in accordance with observations from other medical disciplines:

> We know from clinical information that persons with different mental disorders … have different demographic characteristics...family histories, life events, and neurobiologic correlates. They also have different responses to specific treatments. Such variations in correlates of other medical conditions are generally indicative of different diagnostic categories, etiologies, and need for care (Regier et al. 1984, pp.937-8 [9]).

Studies of specific psychiatric disorders required larger sample sizes, and this was recognized in the ECA study design, which sought to interview almost 20,000 people from its five sites. Subsequently, the study was able to provide precise estimates of the 1-month *prevalence* of all major psychiatric disorders in the adult population together (15.4%), as well as disorder-specific estimates [11]. The 1-month *prevalence* of affective disorders were higher in women (6.6%) than men (3.5%), a pattern that held for major depressive disorder, dysthymia, and anxiety disorders separately [12]. Further analysis of the ECA data suggested that *prevalence* estimates of these disorders (most strongly for major depressive disorders and anxiety disorders) tended to be higher among people from lower socio-economic backgrounds, and those who were separated or divorced, although—like with any cross-sectional study—reverse causation could explain such correlations. The study found little variation in CMDs by ethnicity, but both neighbourhood disadvantage and residential instability were found to be associated with a higher 12-month *prevalence* of major depressive disorder [13].

Both the ECA study and the NCS study which followed a decade later, reported 12-month *prevalence* of any mental health disorders to be nearly one in three of the US population. Startlingly, both studies found that at least one in five of this group (rising to one in four in the NCS study) had not received treatment for their disorder, whereas half of those receiving treatment in the NCS study, did not meet DSM-III revised diagnostic criteria for a mental health disorder. Such studies are thus vital for taking the temperature of psychiatric morbidity in the population, as well as the level of untreated or over-treated need requiring redress through service reorganization. As mental health and well-being are not stochastically, or even solely genetically, determined, patterns of need at the population level will be influenced by changing sociodemographic, economic, and other (social or physical) environmental dynamics

over time. Furthermore, patterns of care are also subject to changing social, economic, and political landscapes over time, and, as such, single, isolated measurements of a nation's mental health 'temperature' provided by single cross-sectional surveys may not detect broader climatic shifts in psychiatric morbidity over the longer term.

Such issues led the original authors of the NCS study to initiate a replication study (NCS-R) between 2001 and 2003, a decade after the first [10, 14]. While the NCS-R study found similar 12-month *prevalence* estimates of anxiety, mood, and substance abuse disorders to the earlier NCS study (of around one in three respondents), one major difference between the surveys was the increase in treatment for such disorders, rising from 24.3% to 40.4% of respondents with a diagnosable (DSM, fourth edition (DSM-IV)) mental health condition. Nonetheless, the majority of people who met criteria for psychiatric disorder in the year prior to each survey still received no treatment for their care, whereas there were substantial increases in treatment among people who did not meet criteria for a DSM-IV mental health disorder, or whose disorder was in the mild range. The authors noted various possible reasons for such changes, including more direct-to-consumer marketing strategies by the pharmaceutical industry (not permitted in other countries, including, e.g., the UK), better mental health awareness, increased insurance coverage, and better access to community services. Finally, the NCS-R highlighted the treatment gap between various groups; women, those aged between 35 and 54 years, and white groups (vis-à-vis non-Hispanic black and Hispanic populations) were more likely to receive treatment.

One further notable epidemiological study of CMD outside of the USA deserves attention; the APMS in the UK, a repeated cross-sectional household survey conducted over four successive waves in 1993 [15], 2000 [16], 2007 [17], and 2014 [18]. Compared with the US studies, the estimated 12-month *prevalence* of CMDs was lower in the UK, with around one in six people meeting diagnostic criteria in the last wave of the APMS study, albeit using a different diagnostic instrument (Clinical Interview Schedule–Revised). Nonetheless, trends between the US and UK surveys in treatment patterns exist. Like the ECA and NCS studies, the APMS surveys have found substantial levels of untreated mental health need in the community, with only one in three of the 2014 wave of the APMS reporting having sought treatment for their CMD [18]. As in the USA, treatment rates have risen over time, which the APMS authors attributed to greater use of psychotropic medications and psychological therapies. Finally, inequalities in receipt of treatment broadly echoed the findings of the NCS-R study, with older, female, and white participants all more likely to receive treatment for their mental health disorder.

Schizophrenia and other psychotic disorders

Cross-sectional surveys of psychotic disorders generally have more limited utility than cohort-based study designs. This arises for three primary reasons. Firstly, psychotic disorders occur less frequently than CMD (see previous section). Typically, the annual *prevalence* of schizophrenia has been estimated to be around four in 1000 [19], although heterogeneity may exist between populations. Therefore, cross-sectional surveys of psychotic disorders need to be very large to obtain precise *prevalence* estimates, and such studies tend to be less frequently conducted as a result. This reason alone, however, is insufficient to favour other designs over cross-sectional surveys,

as the same sample size requirements would apply to, for example, cohort study designs. The second reason why cross-sectional surveys tend to be less frequently adopted to study schizophrenia and related psychotic disorders concerns the hunt for aetiological risk factors for psychosis, which has received substantial research attention. As cross-sectional surveys cannot establish direction of causation between a putative exposure and the outcome, they are of more limited use in this regard. Instead, cohort-based study designs often allow for temporal separation of exposure and outcome, to ensure the former precede the latter. This is particularly important in mental health research, where patterns of exposure (e.g. by sociodemographic markers, deprivation, or urban living) may also mirror patterns of effect secondary to the onset of disorder (i.e. downward social drift following the onset of schizophrenia). The use of longitudinal designs, and focus on *incidence* (new cases) rather than *prevalence* (new and existing cases) minimizes (but may not altogether exclude) issues of reverse causality. In addition, in their seminal monograph on international variation in the *incidence* of schizophrenia (also discussed in more detail later) Jablensky et al. [20] also note that:

> [i]ncidence rates are better than *prevalence* rates [*sic*] for comparisons between different populations, because they are less affected by differential mortality, migration, and other demographic factors. The study of series of patients of recent onset is important also in view of the possibility that pathogenetic or triggering factors which are active in the period preceding the first manifestations of the disorder may cease to operate at later stages of its evolution (Jablensky et al. [20], pp. 43).

The final reason why cross-sectional surveys are less commonly used than cohort-based designs leverages a feature of psychotic disorders that differs notably from CMD: presentation to services. Because the onset of psychotic disorders is often marked by substantial, distressing, and overt symptomatology, including florid psychotic states, bizarre behaviour, social withdrawal, and cognitive impairment, people with psychotic disorder tend to present to mental health services. That said, the duration of untreated psychosis may be long for some individuals [21], and is strongly associated with worse outcomes [22]. Thus, unlike CMDs—where a substantial proportion of people may be untreated (and undetected)—people with psychotic disorders are more routinely picked up in hospital records, healthcare registers, and other routine databases. As a result, while cohort-based study designs of the *incidence* of psychotic disorders may need to be extremely large, they can achieve such sample size requirements in a cost-efficient manner by leveraging use of reliable healthcare databases. In this section, we briefly review some selected major epidemiological studies of psychosis *incidence* in the past 30 years, and highlight the main findings from these studies.

A landmark study in the understanding of the epidemiology of schizophrenia and other psychotic disorders was the World Health Organization's (WHO) Determinants of Outcomes of Severe Mental Disorders study, colloquially known as the 'ten-country' study [20]. The study was conducted between 1978 and 1981 in 12 international settings in ten countries, designed to apply a systematic methodology to—amongst other aspects—the *incidence* of disorder. The study employed a robust case-finding approach to identify all new cases in defined catchment areas over a 2-year period, which met International Classification of Diseases, Ninth Revision (ICD-9) criteria for non-organic psychotic disorders. Importantly, the study established that schizophrenia could be reliably identified as

a feature of all populations where it was studied, from Nigeria to India to Denmark to Japan. Furthermore, its manifestations across these settings were marked more by their similarities than their differences, suggesting broadly consistent cultural validity to the nosological ~~identity~~ defined in ICD-9 as 'schizophrenia'. *Incidence* data of sufficient epidemiological quality were eventually available from eight (including rural and urban Chandigarh as two separate sites) of the 12 centres, which were considered to have 'fairly complete coverage … of the various "helping agencies" that were likely to serve as first-contact sites for psychotic patients' [20]. Importantly, the study identified a two- to threefold variation in the *incidence* of narrowly and broadly defined schizophrenia, respectively, across these international settings. *Incidence* rates thus ranged from 7 to 14 per 100,000 for narrowly defined schizophrenia, and from 15 to 42 per 100,000 for its broadly defined counterpart, typically referred to today as schizophrenia spectrum disorders (SSD). Such variation may allude to important underlying risk factors for disorder (e.g. ethnicity, see 'How patterns of *incidence* and *prevalence* of mental health disorders vary by ethnicity'). Nonetheless, despite this apparent variation, and its potential importance for advancing our understanding of the causes of schizophrenia, an unfortunate legacy of the WHO 'ten-country' was a general misinterpretation of its principal findings (it should be noted, not by the original authors) that the study showed no international variation in risk. This dogma was solely based on the findings for narrowly defined schizophrenia, which were underpowered to detect a statistically significant variation in *incidence* (but not broadly defined schizophrenia, which showed statistically significant variation ($P <$ 0.05)). The view that psychotic disorders were invariant to place effects dominated much of the psychiatric literature for the next 20 years, and was used to advance exploration of the possibility that schizophrenia was almost entirely genetic in origin [23, 24].

A series of important studies conducted since the WHO ten-country study [20] have added a strong evidence base to show that schizophrenia and other psychotic disorders vary by robust, replicable factors, including age (higher during late adolescence and the early 20s), sex (higher among men), ethnicity (higher among minority groups, see 'How patterns of *incidence* and *prevalence* of mental health disorders vary by ethnicity'), and place (higher in people exposed to more urban, deprived environments). For comprehensive systematic reviews on these topics, see McGrath et al. (all aspects) [25], Kirkbride et al. (all aspects) [26], March et al. (variation by place) [27], Bourque et al. (variation by migration and generation status) [28], and Cantor-Graae and Selten (variation by ethnicity and migration) [29]. One important example, conducted in the UK, was the Aetiology and Ethnicity in Schizophrenia and Other Psychoses (AESOP) study [30]. That study used a broadly comparable design to the WHO study, and sought to ascertain the *incidence* of first-episode psychotic disorders in community settings in three defined catchment areas in the UK, South East London, Nottinghamshire, and Bristol, comprising a mix of urban, suburban, and rural environments. In that study, Kirkbride et al. [30] found that the *incidence* of non-affective psychotic disorders ~~(SSD)~~ varied from 13.9 in Nottinghamshire to 40.5 per 100,000 in South East London, consistent with international variance in rates observed by Jablensky et al. [20], albeit on a national scale. Register-based *incidence* studies using prospectively designed national cohorts have also provided valuable epidemiological evidence about the *incidence* of disorders, most notably from Sweden

and Denmark [31–33]. Linking entire population cohorts to hospital registers, such studies provide a powerful tool for the analysis of prospectively collected risk factors in relation to later mental health outcomes. Age-adjusted *incidence* rates of SSD from Sweden suggest that the rate in the background Swedish population is around 31 per 100,000 in women and 49 per 100,000 among men [31], reflecting known sex differences in the risk of psychotic disorders. *Incidence* rates of a similar or greater magnitude have been found for SSD in Danish registers [33], which were also able to estimate lifetime risk (similar to lifetime *prevalence*) at around 3.7% (similar for men and women).

Readers will note that the *incidence* and *prevalence* estimates obtained from these register-based cohort designs appear to be higher than comparable estimates of *incidence* from the first-contact studies described earlier (i.e. the WHO ten-country study, the AESOP study), or lifetime *prevalence* estimates from cross-sectional surveys. This pattern has been recently noted by Hogerzeil et al. [34, 35], using a dual first-contact and register-based design in the same population. Several reasons for this discrepancy may exist, but register-based designs may be more comprehensive in case identification, as 'hospital' registers will typically identify all people diagnosed with a disorder in a given healthcare system, including both in- and outpatient facilities. By comparison, first-contact designs, which rely on regular contact with a variety of secondary and tertiary care providers allied to mental health, may still be more likely to miss cases presenting to other parts of a healthcare system, unless these are also monitored. It is also possible that first-contact designs ~~over~~estimate the population at risk (i.e. the denominator), because they typically take a static estimate of the population at risk at a single point in time (i.e. from a census or similar) and multiply this by the length of case ascertainment to approximate total person-years at risk [30]. This method thus ignores changes to the population at risk over time caused by entry to (owing to immigration, changes in birth and infant mortality rates) and exit from (emigration, adult mortality) the catchment area population. While such bias may be small over shorter periods of case ascertainment, they could be amplified over longer periods, or ~~during~~ periods of rapid change~~s~~ in migration or mortality in the population. ~~These issues could lead to under- or overestimates of the true population at risk.~~ By contrast, via linkage to migration and death registers, register-based cohort studies can typically estimate the exact person-years at risk with a higher degree of precision. While these two reasons (better case finding and more precise denominator estimation) suggest that the higher rates observed in register-based cohort designs may be more reliable, we also note that first-contact designs can offer better validation of psychiatric diagnoses than register-based designs. First-contact studies, such as the WHO or AESOP studies, use standardized diagnostic assessments to validate any psychiatric diagnoses initially made in clinical settings to identify cases. By contrast, psychiatric case registers usually rely on clinical diagnoses made by mental health practitioners working in a variety of mental health settings, and may be more subject to inter-rater differences, and the vagaries of shifting diagnostic practices or sociocultural attitudes to mental health over time. While studies of register-based diagnoses for schizophrenia suggest they are valid for research purposes [36, 37], single-registry snapshots do not guarantee their validity for all psychotic (or psychiatric) disorders, or across all time periods, healthcare settings, or geographical locations. Allebeck (p. 390)

suggests that these 'issues of validity and generalizability needs to be addressed for each specific study purpose' [38].

Suicide

Over 800,000 people die by suicide each year, making it an immense issue for global and public health. While we recognize the important of understanding other suicidal outcomes, including suicidal thoughts, self-harm, and suicide attempts, we restrict this subsection to major studies of rates of suicide. Our focus here, on suicide, is, in part, because it represents the most severe suicidal outcome and, in part, because the available literature may be less subject to (although not free from [39]) under-reporting and detection biases than other suicidal outcomes. A larger literature on other important suicidal outcomes demonstrates how such outcomes contribute substantially to psychiatric morbidity in the general population, and particularly among young people [18, 40]. For *prevalence* examples, see the UK APMS (i.e. McManus et al. [18]), and the Australian population surveys [41], and for good reviews on this topic see Pitman et al. [40] and Evans et al. [42]). Such outcomes contribute.

Quantifying the overall burden of suicide is difficult, because rates vary by age, sex, ethnic group, and socio-economic position. Rates also vary cross-culturally and therefore by country, as well as over time. Nonetheless, since the 1960s and 1970s several studies, in several settings, have noted a decline in suicide among older people, with some more recent increases among young men, including in the UK, Japan, Australia, and New Zealand [43]. In the USA, although rates increased among young men between 1964 and 2013, the overall age pattern remains one of upward risk by age, with substantial peaks in men after 75 years of age. By contrast, rates for women are more uniform over age in the USA, as well as elsewhere [43]. Typically, suicide rates are about three times greater in men than women. This pattern is observed consistently in the UK, across all ages, where overall risks were estimated to be 16.6 in men and 5.4 in women per 100,000 of the population in 2015 [44]. Using the most recently available global estimates of suicide from WHO (2012) [45], age-standardized rates vary substantially worldwide, from fewer than 5 per 100,000 in much of North Africa, Southern Africa, Mexico, the Middle East, the Philippines, and Indonesia, to over 15 per 100,000 in parts of Russia and many countries in the former Soviet Bloc, East Africa, India, and Japan. For men, rates ranged from 0.6 per 100,000 in Saudi Arabia to 70.8 per 100,000 in Guyana; for women, rates ranged from 0.2 per 100,000 in Syria to 35.1 in the Democratic People's Republic of Korea [43]. Owing to these high rates, combined with large population bases, suicide in low- and middle-income countries are thought to account for around 75% of all suicides worldwide [45], making them a priority for global and public mental health.

Variations in suicide rates by person, place, and time are likely to occur for a variety of reasons, including changes in the availability of means, policy interventions, socio-economic factors, and other psychosocial stressors, as well as the influence of socio-cultural customs, values, and norms. The occurrence of some of these factors—most notably, availability of means and socio-economic stressors—may partially explain the higher rates of suicide observed in rural compared with urban populations [46–49], a pattern particularly pronounced amongst men [46, 47]. For example, higher suicide rates in rural parts of the USA, Canada, the UK, and Australia have been linked to the greater availability of firearms [47, 50–53], whereas pesticide poisonings have been observed to be more common in rural parts of Taiwan and South Korea [54, 55]. Socio-economic drivers of suicide rates are also important. There is accumulating evidence, for example, that economic recessions may impact on suicide rates through factors such as unemployment (see, e.g., Frasquihlo et al. [56]). In a further review of such evidence, the European Psychiatric Association [57] has suggested that various factors related to economic recession—including unemployment, indebtedness, precarious working conditions, inequality, housing security, and a loss of social cohesion (first noted in relation to suicide risk by Durkheim [58] in the nineteenth century)—are related to suicidal behaviours and a range of other mental health problems. Exposure to such factors is rarely distributed equitably throughout the population, and the authors suggest that certain high-risk groups, including working-age men and those of low socio-economic position, may bear a disproportionate burden of increased risk attributable to these effects, exacerbated during periods of economic recession. The European Psychiatric Association also noted that [57]:

> the existence of well-developed social protection and health services is also relevant. In this way, countries with a consolidated welfare state appears [*sic*] to be less exposed to adverse health outcomes related to economic decline (Martin-Carrasco et al. [57] pp. 105).

Nonetheless, they also note that the direction of causality in the association between suicide, economic hardship, and previous psychiatric morbidity have yet to be fully established. While further research is clearly required here, if true, such findings suggest that during periods of economic recession policy-level decisions that seek to reduce government expenditure through cuts to mental health services may increase suicide deaths and other mental health problems. To combat the economic impact of recession on suicide and other areas of psychiatric morbidity, the European Psychiatric Association concludes by suggesting several areas of policy intervention, including initiatives that maintain income support, create jobs and more stable working environments, and tackle housing instability and structural inequalities present in society (for a detailed explanation of possible interventions see Martin-Carrasco et al. [57]). We note that many of the drivers of, and interventions against, increased suicide rates during periods of economic recession will also apply to subgroups of the population at all points in time. Given the heterogeneity in suicide rates between person and place over time, one area of policy intervention to provide appropriate treatment response and service provision for affected individuals and groups is accurate population surveillance, using consistent definitions of suicide and accurate recording via routine health observatories. Such accurate surveillance systems—although more challenging to implement in some settings—would allow early detection of emerging high risk groups for suicide, thus more effectively informing mental health service provision.

How patterns of *incidence* and *prevalence* of mental health disorders vary by ethnicity

The overall *incidence* and *prevalence* estimates highlighted in the first section may mask important heterogeneity within subgroups of the population. Closer investigation of groups that may be at increased risk is therefore important for public mental health and health service planning, and may also enrich our understanding of the aetiology of disorders. Ethnic variation in the *incidence* and

prevalence of many health conditions has been widely documented [59], including psychiatric disorders. Early reports of high rates of hospitalization for schizophrenia, for example amongst Norwegians emigrating to the USA, date as far back as the 1920s [60]. Ethnic variation in mental illness was also identified in the aforementioned ECA study (see 'The burden of major psychiatric disorders'), which found that both the lifetime and 12-month *prevalence* of major mental disorders was higher among black respondents than those of white or Hispanic origin [61]. However, the study also noted that these ethnic groups tended to differ on important demographic characteristics (i.e. confounding), with black respondents more likely to be younger, poorer, and have less education than their white counterparts. In this section we briefly highlight the major epidemiological evidence describing any variation in the burden (*incidence* or *prevalence*) of CMD, psychotic disorders, and suicide by ethnicity and migration status.

Common mental disorders

Although a considerable literature exists on the overall *prevalence* of CMDs (see 'Schizophrenia and other psychotic disorders'), until recently there has been little population-based investigation into ethnic variations in CMD [62]. Interestingly, the overall balance of evidence in regard to CMD does not provide consistent or conclusive evidence of ethnic variation.

Research from the USA initially suggested some ethnic variation in affective disorders existed. For example, the ECA study observed that rates of depression and dysthymia were higher in white and Hispanic groups than in black individuals [63]. However, this study did not find evidence for ethnic variation in bipolar disorder [63]. By contrast, the *prevalence* of depression across 23 countries in the European Social Survey was reported to be higher among ethnic minority groups (7.1%) than the majority-Caucasian population (5.9%) [64]. Meanwhile, research from another cross-sectional survey in England, known as EMPIRIC, noted some ethnic variation in the *prevalence* of CMD, although this was modest [62]. The APMS in England found that the higher CMD annual *prevalence* in ethnic minority groups was largely explained by sociodemographic confounders [65, 66]. In a meta-analysis on the relationship between mood disorders and migration, Swinnen and Selten concluded that, if anything, there was only a marginal increase in mood disorders amongst migrants overall (relative risk (RR) 1.38, 95% CI 1.17–1.62) [67].

Notwithstanding this mixed pattern, some authors have suggested that the experience of interpersonal racism and perceived racial discrimination in society are associated with CMD risk [68, 69]. UK-based research showed that the weekly *prevalence* of CMD increased significantly for Caribbean, Indian, Irish, and Pakistani ethnic groups who had experienced interpersonal racism compared with those reporting no harassment. This effect was particularly pronounced among ethnic minority women [68]. The study also found that the experience of employment-related discrimination increased the *prevalence* of CMD among many of these groups, and, further, that the effect appeared more pronounced amongst Similarly, Bhui et al. found that CMD risk was highest among ethnic minorities who reported unfair treatment or racial insults [69]. This relationship was also demonstrated in the Netherlands, where perceived discrimination accounted for an estimated 25% of the depression risk for Turks and South-Asian Surinamese living in the Netherlands [70].

In addition to differences in the prevalence of CMD between ethnic groups, there were also variations in terms of mental health service use. A detailed report using National Survey on Drug and Health Data revealed that access to care and quality of care varied according to ethnicity [71]. For example, white adults and those reporting mixed ethnicity were more likely to report using mental health services in the past year, to have a prescription for psychiatric medication, or to receive outpatient services than black adults [71]. Other research on health service usage for CMD from the UK found that black and South Asian ethnic groups were less likely to have seen their doctor in the past year than white individuals [65]. Furthermore, even after controlling for symptom severity, black individuals were less likely to receive antidepressants than white individuals [65]. For people with depression in the past year in the USA, Latinos, Asians, and African Americans were less likely than non-Latino whites to have access to mental health treatment [72]. Also, these minority groups were less likely to have received adequate treatment for acute depressive episodes [72]. In general, ethnic minorities are less likely to receive care when they need it and are more likely to receive poor-quality care when they are treated [73]. These disparities in mental health care between ethnic groups could be driving ethnic differences in rates of mental health and mental illness, and, as such, public health policy must be developed to improve access to and quality of mental health care for ethnic minority groups.

Although the *prevalence* of CMD between different ethnic groups appears small, there is some evidence that first generation migrants (i.e. born abroad) may be at higher risk. For example, research from Sweden showed that migrants have increased CMD risk than native Swedes, although there was considerable between-group heterogeneity [74], with those from Finland and the Middle East at particularly elevated risk. Similarly, the Israel World Mental Health Survey found that the 12-month CMD *prevalence* was approximately double for migrants of North African or Asian origin than for European or American migrants, even after adjustment for socio-economic factors [75]. Other social determinants of health may underlie these differences, including the experience structural or interpersonal discrimination. Furthermore, migration is often accompanied by a change in socio-economic position, and Das-Munshi et al. have proposed that downward intragenerational mobility (i.e. movement to a lower socio-economic position, including lower occupational or income status, during an individual's lifetime) is associated with international migration and increased vulnerability to CMD [76]. Other predisposing factors related to migration, including the economic circumstances in a migrant's country of origin, reasons for migration, and experiences in the host country may also play a role in differing CMD risk across migrant groups [77, 78].

Psychotic disorders

The elevated rates of psychotic disorders among ethnic minorities is one of the most replicated areas of psychiatric epidemiological research, providing clear and persuasive evidence that *incidence* rates are elevated in ethnic minority groups [79–81]. Some of the earliest evidence for this came from the seminal work of Ørnulv Ødegaard, who showed that migrant status was a risk factor for psychosis among Norwegians emigrating to Minnesota in the USA in the 1930s [60]. This finding has since been replicated, and extended to the descendants of migrants, in numerous settings [28, 29, 60,

82–87], including the UK [26, 88, 89], Sweden [90–92], Denmark [82, 84], the Netherlands [93], the USA [94], Canada [86, 95], and Israel [96]. The AESOP study in the UK found that ethnic minority groups were at increased risk for all psychotic disorders, with black Caribbean and African groups at highest risk [97]. A replication of this finding in a separate sample in East London found these excess *incidence* rates persisted after adjustment for socio-economic status [98]. A recent systematic review from England [26], where this issue has arguably been studied most often, suggests that the *incidence* of schizophrenia is around five times greater for people of black Caribbean (pooled RR 5.6, 95% CI 3.4–9.2) and African (pooled RR 4.7, 95% CI 3.3–6.8) backgrounds than white British people, with South Asia at around double the risk (pooled RR 2.4, 95% CI 1.3–4.5).

Research from the APMS study has shown that the *prevalence* of psychosis is also higher among black minority groups in the UK [66]. However, this study also suggested that the excess psychosis risk among some ethnic minority groups may be partially explained by socio-economic disadvantage [66], a finding only partially supported by comparable *incidence* studies [99].

The exact reasons for elevated risk of psychotic disorders among migrants and ethnic minority groups is still unknown (for good reviews of plausible hypotheses, see Bhugra [100], Fung et al. [101], and Morgan et al. [102]. The heterogeneity in risk between minority and migrant groups suggests that factors such as visible minority status, discrimination, or psychosocial adversity may be causally relevant to these differences. A meta-analysis found that the effect size was greater for migrants from developing versus developing countries (RR 3.3, 95% CI 2.8–3.9) [29]. One suggested explanation for higher rates among migrant groups is exposure to adversity in the country of origin, not limited to poverty, trauma, or political unrest. In support of this hypothesis, recent data from Hollander et al. demonstrated that refugees in Sweden are at even greater risk of schizophrenia than other non-refugee migrants from the same regions of origin [91]. Visible minority status may also contribute to such experiences by migrants and their descendants in their destination country following immigration [96, 103]. There are several other plausible mechanisms that may underpin these associations, including the suggestion that ethnic minority groups may be more likely to be misdiagnosed with a psychotic disorder than non-migrant groups. Although a body of indirect research does not support this idea (including the absence of differences in psychosis rates between people living in the Caribbean and the white British group in the UK [104–106], and similar symptomatic profiles by ethnicity at first presentation [107]), further research is required to examine all putative drivers of this public mental health tragedy [108].

Suicide

Despite the wide variation between countries in terms of overall suicide rate (see earlier 'Suicide' subsection), these aggregate estimates hide heterogeneity between groups within each country, including by ethnicity. In the UK, high rates of suicide have been demonstrated among black populations, as well as older South Asian women compared with the white British majority group [109]. Among past-year mental health service users, rates of suicide were lower among South Asians men but elevated for older South Asian women versus white individuals [110]. When stratified by age and sex, the research found that suicide rates were elevated among black Caribbean and black African men, as well as young women of South Asian origin [110]. In Sweden suicide has been shown to be elevated among migrants and their children of Finnish (odds ratio (OR)$_{\text{migrants}}$ 1.4, OR$_{\text{children of migrants}}$ 1.7) or Western (including Norway, Denmark, Iceland, Germany, UK, USA, Canada) origin (OR$_{\text{migrants}}$ 1.2, OR$_{\text{children of migrants}}$ 1.7) [111].

Despite this variation, researchers since the 1920s have noted that suicide rates among immigrants were correlated with rates in their countries of origin [112]. This led researchers to hypothesize that migrants may 'import' their suicide risk [113, 114], as well as typical methods of suicide commonly found in their country of origin [114]. This evidence of the portability of suicide risk, coupled with evidence for the geographical differences in suicide rates between countries (see 'The burden of major psychiatric disorders') may strengthen genetic explanations of suicide risk, which posit that variation of rates between subgroups could be explained by genetic factors alternatively, important socio-environmental factors that differ between the country of origin and host country may explain these findings [114]. Some studies have also suggested convergence of migrant suicide risk to the host country rate over time following immigration perhaps owing to adopting new behavioural norms or sociocultural attitudes, which point to contextual and environmental risks for suicide [113, 115], others have suggested this may simply be explained by regression to the mean (the phenomenon where repeated measures vary non-systematically around the true mean, so usually high or low measurements tend to be followed by measurements that are closer to the mean) [114, 116].

Globally, some of the highest rates of suicide are among Indigenous people in Australia, Canada, New Zealand, Greenland, and the USA [117, 118]. Estimates from Australia showed that the rates among Indigenous peoples was more than twice as high as those for non-Indigenous Australians [119]. For Inuit peoples, a group of Indigenous peoples in Canada, the suicide rate is 11 times higher than the Canadian average [120].

How the epidemiological burden of major psychiatric disorders translates into the potential for intervention

In section 'The burden of major psychiatric disorders' we provided an overview of the burden of major psychiatric disorders worldwide, and how these varied by the tenets of person and place. In the section, we gave greater detail on how these psychiatric disorders varied by migration and ethnicity, two factors along which there is considerable inequality in how the mental health burden is shared within populations. While a major of psychiatric epidemiology is to elucidate the underlying risk factors that cause these inequalities, it is also important to consider what impact the prevention of excesses risk may have on the global burden of psychiatric disorders. By doing so, psychiatric epidemiology can inform public mental health, policy interventions, and health service planning. In this section we consider, briefly, a thought experiment, by asking *if a risk factor for psychiatric disorder could be removed from the population, assuming causality, what proportion of cases could be prevented?* This question lies at the heart of the formula for the *population attributable risk* (PAR), which jointly considers the *measure of association* (i.e. a risk ratio), as well as the

level (*prevalence*) of the exposure in the population [3]. Specifically, the PAR estimates the reduction in *incidence* of an illness that would be achieved if the population was not exposed to the risk factor [3]. While the PAR is useful for translating epidemiological risks into *measures of impact* for public health, it is based on a number of assumptions, including that there is a causal relationship between exposure and outcome, that removal of the exposure has a direct reduction on the outcome, and that the risk factor itself is modifiable (see Greenland and Rothman [3] and Rockhill et al. [121] for more coverage of these issues). Clearly, the latter is—depending on interpretation—both impossible and troubling with respect to the issues of migration and ethnicity discussed in the previous section. This makes the search for the drivers of the increased risks for some migrant and ethnic minority groups an imperative issue for contemporary psychiatric epidemiology. If we can move closer to the identification of these risk factors, we can get a better handle on their impact in terms of public mental health. Nonetheless, PARs for the respective roles of migration and ethnicity in relation to psychiatric disorders (see later) may still be ideologically useful, as beacons for the potentially preventable burden of disorders that could be prevented in the population if all underlying factors could be identified and prevented.

One challenge with applying research to public health interventions is that often the risk factors with the highest predictive power at the individual level (i.e. a large risk ratio) have a small population impact (i.e. because they are rare). Thus, epidemiological evidence that takes into account the magnitude of risk introduced by a risk factor, as well as the exposure patterns in the population is a powerful tool for public health intervention. Furthermore, PARs can be used to indicate the type of prevention strategies that may be most amenable to a given intervention. For example, as Geoffrey Rose made clear in his seminal 1984 lecture [122], population-level strategies may take priority over individual high-risk prevention approaches, when 'a large number of people at a small risk may give rise to more cases of disease than the small number who are at high risk'. This *prevention paradox* speaks to why the use of PAR can be important for public health interventions, as it provides a metric of which factors could result in the greatest potential impact.

Common mental disorders

Considering the wide range of risk factors for CMD, knowledge of PAR can potentially be used to help prioritize interventions and make informed use of limited public health resources. The multifactorial causes of CMD mean efforts to target high-risk individuals may be less effective from a public health perspective than population-based approaches. However, one major issue in designing such interventions is the level of unmet psychiatric need in the community which may never present to mental health services. Recognizing that previous episodes of depression and anxiety are one of the strongest predictors of future risk, the National Health Service in England launched a new model for treating psychological distress in the population in 2008, known as adult Improving Access to Psychological Therapies (IAPT). These services are designed to offer a stepped-care, evidence-based approach to treatment appropriate to the presenting psychological distress, including cognitive behavioural therapy and other treatments for which there is sufficient evidence as recommended

by the National Institute for Health and Care Excellence [123]. These therapies are delivered by trained, accredited practitioners. Importantly, one major focus of IAPT provision is ease of access and use of the service. The national IAPT service model is designed to be highly accessible, with low barriers to entry (both self-referrals and referral via primary care are accepted), with services delivered by guided self-help, via telephone or in person, depending on the severity of the presenting symptoms. IAPT services also offer access to an employment adviser to reduce unemployment and lost work days, through both absenteeism and presenteeism. The IAPT intervention is designed to foster greater adherence to the intervention, be cost-effective (although whether it achieves this is unclear [124]), reduce stigma, and, most importantly, uses psychological approaches that have been shown to improve mental health outcomes for a larger proportion of society than would previously have had access to services [125, 126].

Elsewhere, other approaches to CMD prevention have identified targets for prevention. Research on adolescents in the USA, for example, has shown significant PARs across many areas, including interpersonal relationships (e.g. low family connectedness, including low understanding (PAR 31%), low attention (PAR 29%), and low paternal warmth PAR (23%)); affect regulation and cognition (e.g. baseline depressed mood (PAR 39%)); delinquent/near-delinquent activities (e.g. early sexual relationships (PAR 41%)); and low levels of constructive community involvement conferred significant PAR to adolescent depression (e.g. not attending youth group (PAR 36%)) [127]. The PAR estimates from this study demonstrate that there are many risk factors from multiple domains that increase the risk of CMD, and, further, can guide the focus of preventative interventions to areas such as family connectedness or constructive community engagement. Similarly, Goodman et al. [128] demonstrated that socio-economic factors contributed a significant PAR to adolescent depression: 40% and 26% for education and income, respectively [128]. If these socio-economic factors are causal, we would expect substantial reductions in the *incidence* of CMD if effective interventions were put in place to remove the damaging effect of these risk factors, including approaches highlighted in the WHO report on effective interventions and policy options for preventing mental disorders. The WHO has compiled a report highlighting the evidence for effective interventions and policy interventions on housing, education, or economic insecurity that have been shown to reduce the burden of CMD [129, 130].

Schizophrenia and other psychotic disorders

In the case of psychotic illnesses, both ethnic minority status and urbanicity have been identified as important risk factors for development of schizophrenia and other psychotic disorders. The PAR for these two risk factors have been estimated in the UK [131]. In terms of ethnicity, the paper suggested that up to 22% of cases of psychotic disorders could be prevented if we were able to identify and remove all exposures that underlie the elevated risk of psychosis in ethnic minority populations. Furthermore, if it were possible to identify and remove all factors associated with the elevated risk among those living in urban environments, 27% of cases of non-affective psychosis could be prevented [131]. When considered together, the joint PAR for ethnicity and urbanicity in relation to non-affective psychosis was over 60% [131], reflecting possible synergistic effects between these two exposures. However, while these

high PAR estimates suggest that urbanicity and ethnicity may be important targets for population strategies, the authors point out that it is the underlying drivers of risk that these markers represent which need to be identified, tested, and established as causal mechanisms for psychosis. Furthermore, any prevention strategy targeting a non-specific exposure such as 'urban living' is unlikely to be practical or cost-effective given the absolute *incidence* of psychotic disorders [131].

Suicide

The *prevention paradox* is clearly demonstrated in suicide prevention efforts. The robust association between psychiatric disorders and suicide has led many suicide prevention efforts to focus on individuals with mental illness, as they have high risk of suicide, despite the relatively low population *prevalence* of mental illnesses. By contrast, socio-economic factors, like education, income, social exclusion, or deprivation, are more distally associated with suicide risk, but because they are more commonly distributed in the population, efforts to make even small improvements in socio-economic conditions have the potential to substantially reduce suicide risk, if causal.

In their systematic review, Li et al. estimated the *population attributable risk* associated with psychotic disorders and socio-economic factors were of similar magnitude [132]. For example, the PAR in males for low educational achievement was 41% and low occupational status was 33%, whereas the PAR for affective disorders was 26% and substance use disorders was 9% [132]. Similar findings were also observed in females, suggesting that prevention strategies focusing on either would produce similar population-level effects on suicide [132]. This finding was replicated in Denmark using population registers, and the authors indicated the evidence highlights the need to combine suicide prevention programs that focused on both high risk groups (i.e. with mental illness), and on population interventions targeting unemployment and improving social cohesion [133].

Public mental health and variation in rates by ethnicity

In this chapter (‘How patterns of *incidence* and *prevalence* of mental health disorders vary by ethnicity’), we have paid particular attention to how the rates of schizophrenia and other psychotic disorders, CMD, and suicide vary by ethnicity, most notably for psychotic disorders. This issue has a particular public mental health challenge, given the inequality in risk of psychotic disorders faced by some ethnic minority groups. Ethnicity is not, of course, a modifiable risk factor, making it vital to consider why the health of minority populations is negatively influenced when thinking about putative intervention strategies to reduce harm. Mental health promotion and illness prevention efforts need to first identify the mechanisms that drive the increased rates in ethnic minority groups, and then find effective interventions to mitigate this risk. To use the well-known analogy in public health, we can design culturally sensitive and appropriate interventions that pull people out of the stream; however, it is important for us to consider what is causing so many to fall into the stream in the first place. There is an ethical imperative in public health to pay attention to large systemic shifts that are required to remove the excess burden of mental illnesses among ethnic minority populations. This requires cross-sectoral, intersectional efforts, as the reasons certain population groups are more likely to end up in the 'stream' of poor mental health have to do with systems of power, privilege, advantage, and systemic racism that are rooted in legacies of discrimination and disadvantage. The intersectionality lens is an important consideration as risks, like ethnic minority status, urban living, and low socio-economic status, tend to cluster, exacerbating the increased risks. At minimum, 'preventive interventions must not directly affirm or contribute to inequality or injustice' [134]. However, an ethical approach insists that we go beyond this to actively promote equity and justice. While there does not seem to be any simple answers for how to address these systemic issues of power and injustice that may be contributing to increased risks for marginalized populations, researchers and public health professionals should attend to these issues in order to attempt to address these critical issues.

References

1. Porta M (ed.). *A Dictionary of Epidemiology*. 6th ed. Oxford: Oxford University Press, 2016.
2. Tripepi G, Jager KJ, Dekker FW, Wanner C, Zoccali C. Measures of effect: relative risks, odds ratios, risk difference, and "number needed to treat." *Kidney Int* 2007; 72: 789–791.
3. Greenland S, Rothman KJ. Measures of effect and measures of association. In: Rothman KJ, Greenland S (eds). *Modern Epidemiology*. 2nd ed. Philadelphia, PA: Lippincott Willams and Wilkins, 1998, pp. 47–66.
4. Perez L, Künzli N. From measures of effects to measures of potential impact. *Int J Public Health* 2009; 54: 45–48.
5. Vos T, Allen C, Arora M, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* 2016; 388: 1545–1602.
6. Laursen TM, Munk-Olsen T, Vestergaard M. Life expectancy and cardiovascular mortality in persons with schizophrenia. *Curr Opin Psychiatry* 2012; 25: 83–88.
7. Kirkbride JB, Jackson D, Perez J, et al. A population-level prediction tool for the incidence of first-episode psychosis: translational epidemiology based on cross-sectional data. *BMJ Open* 2013; 3: 1–14.
8. Steel Z, Marnane C, Iranpour C, et al. The global prevalence of common mental disorders: a systematic review and meta-analysis 1980-2013. *Int J Epidemiol* 2014; 43: 476–493.
9. Regier DA, Myers JK, Kramer M, et al. The NIMH Epidemiologic Catchment Area program. Historical context, major objectives, and study population characteristics. *Arch Gen Psychiatry* 1984; 41: 934–941.
10. Kessler RC, Demler O, Frank RG, et al. Prevalence and treatment of mental disorders, 1990 to 2003. *N Engl J Med* 2005; 352: 2515–2523.
11. Regier DA, Boyd JH, Burke JD, Jr, et al. One-month prevalence of mental disorders in the United States. Based on five Epidemiologic Catchment Area sites. *Arch Gen Psychiatry* 1988; 45: 977–986.
12. Regier DA, Farmer ME, Rae DS, et al. One-month prevalence of mental disorders in the United States and sociodemographic characteristics: the Epidemiologic Catchment Area study. *Acta Psychiatr Scand* 1993; 88: 35–47.
13. Silver E, Mulvey EP, Swanson JW. Neighborhood structural characteristics and mental disorder: Faris and Dunham revisited. *Soc Sci Med* 2002; 55: 1457–1470.
14. Kessler RC, McGonagle KA, Zhao S, et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. *Arch Gen Psychiatry* 1994; 51: 8–19.

15. Jenkins R, Lewis G, Bebbington P, et al. The National Psychiatric Morbidity surveys of Great Britain—initial findings from the household survey. *Psychol Med* 1997; 27: 775–789.

16. Singleton N, Bumpstead R, O'Brien M, Lee A, Meltzer H. *Psychiatric Morbidity Among Adults Living in Private Households, 2000*. London: Her Majesty's Stationary Office, 2001.

17. McManus S, Meltzer H, Brugha T, Bebbington P, Jenkins R. *Adult Psychiatric Morbidity in England, 2007: Results of a Household Survey*. London: The NHS Information Centre for Health and Social Care, 2009.

18. McManus S, Bebbington PE, Jenkins R, Brugha T. Mental health and wellbeing in England: Adult Psychiatric Morbidity Survey 2014 Executive Summary. Available at: http://content.digital.nhs.uk/catalogue/PUB21748/apms-2014-exec-summary.pdf (2016, accessed 25 February 2018).

19. Saha S, Chant D, Welham J, McGrath J. A systematic review of the prevalence of schizophrenia. *PLOS MED* 2005; 2: e141.

20. Jablensky A, Sartorius N, Ernberg G, et al. Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organization ten-country study. *Psychol Med* 1997; 20: 1–97.

21. Morgan C, Fearon P, Hutchinson G, et al. Duration of untreated psychosis and ethnicity in the AESOP first-onset psychosis study. *Psychol Med* 2006; 36: 239–247.

22. Melle I, Larsen TK, Haahr U, et al. Reducing the duration of untreated first-episode psychosis: effects on clinical presentation. *Arch Gen Psychiatry* 2004; 61: 143–150.

23. Crow TJ. Schizophrenia as the price that homo sapiens pays for language: a resolution of the central paradox in the origin of the species. *Brain Res Rev* 2000; 31: 118–129.

24. McGrath JJ. Myths and plain truths about schizophrenia epidemiology—the NAPE lecture 2004. *Acta Psychiatr Scand* 2005; 111:4–11.

25. McGrath J, Saha S, Welham J, El Saadi O, MacCauley C, Chant D. A systematic review of the incidence of schizophrenia: the distribution of rates and the influence of sex, urbanicity, migrant status and methodology. *BMC Med* 2004; 2: 1–22.

26. Kirkbride JB, Errazuriz A, Croudace TJ, et al. Incidence of schizophrenia and other psychoses in England, 1950-2009: A systematic review and meta-analyses. *PLOS ONE* 2012; 7: e31660.

27. March D, Hatch SL, Morgan C, et al. Psychosis and place. *Epidemiol Rev* 2008; 30: 84–100.

28. Bourque F, van der Ven E, Malla A. A meta-analysis of the risk for psychotic disorders among first- and second-generation immigrants. *Psychol Med* 2011; 41: 897–910.

29. Cantor-Graae E, Selten J-PP. Schizophrenia and migration: a meta-analysis and review. *Am J Psychiatry* 2005; 162: 12–24.

30. Kirkbride JB, Fearon P, Morgan C, et al. Heterogeneity in incidence rates of schizophrenia and other psychotic syndromes: findings from the 3-center AeSOP study. *Arch Gen Psychiatry* 2006; 63: 250–268.

31. Leao TS, Sundquist J, Frank G, Johansson LM, Johansson SE, Sundquist K. Incidence of schizophrenia or other psychoses in first- and second-generation immigrants: a national cohort study. *J Nerv Ment Dis* 2006; 194: 27–33.

32. Mortensen PB, Pedersen CB, Westergaard T, et al. Effects of family history and place and season of birth on the risk of schizophrenia. *N Engl J Med* 1999; 340: 603–608.

33. Pedersen CB, Mors O, Bertelsen A, et al. A comprehensive nationwide study of the incidence rate and lifetime risk for treated mental disorders. JAMA Psychiatry 2014; 71: 573–581.

34. Hogerzeil SJ, van Hemert AM, Rosendaal FR, Susser E, Hoek HW. Direct comparison of first-contact versus longitudinal register-based case finding in the same population: early evidence that the incidence of schizophrenia may be three times higher than commonly reported. *Psychol Med* 2014; 44: 3481–3490.

35. Hogerzeil SJ, van Hemert AM, Veling W, Hoek HW. Incidence of schizophrenia among migrants in the Netherlands: a direct comparison

36. Dalman C, Broms J, Cullberg J, Allebeck P. Young cases of schizophrenia identified in a national inpatient register. *Soc Psychiatry Psychiatr Epidemiol* 2002; 37: 527–531.

37. Löffler W, Häfner H, Fätkenheuer B, et al. Validation of Danish case register diagnosis for schizophrenia. *Acta Psychiatr Scand* 1994; 90: 196–203.

38. Allebeck P. The use of population based registers in psychiatric research. *Acta Psychiatr Scand* 2009; 120: 386–391.

39. Claassen CA, Yip PS, Corcoran P, Bossarte RM, Lawrence BA, Currier GW. National suicide rates a century after Durkheim: do we know enough to estimate error? *Suicide Life Threat Behav* 2010; 40: 193–223.

40. Pitman A, Krysinska K, Osborn D, et al. Suicide in young men. Lancet 2012; 379: 2383–2392.

41. Fairweather-Schmidt AK, Anstey KJ. Prevalence of suicidal behaviours in two Australian general population surveys: methodological considerations when comparing across studies. *Soc Psychiatry Psychiatr Epidemiol* 2012; 47: 515–522.

42. Evans E, Hawton K, Rodham K, Deeks J. The prevalence of suicidal phenomena in adolescents: a systematic review of population-based studies. *Suicide Life Threat Behav* 2005; 35: 239–250.

43. Snowdon J, Phillips J, Zhong B, Yamauchi T, Chiu HFK, Conwell Y. Changes in age patterns of suicide in Australia, the United States, Japan and Hong Kong. *J Affect Disord* 2017; 211: 12–19.

44. Office for National Statistics. Suicides in the UK: 2015 registrations. Available at: https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/bulletins/suicidesintheunitedkingdom/2015registrations (2016, accessed 18 February 2018).

45. World Health Organization. Suicide data. Available at: http://www.who.int/mental_health/prevention/suicide/suicideprevent/en/ (2016, accessed 15 January 2017).

46. Yip PSF, Liu KY, Hu J, Song XM. Suicide rates in China during a decade of rapid social changes. *Soc Psychiatry Psychiatr Epidemiol* 2005; 40: 792–798.

47. Fontanella CA, Hiance-Steelesmith DL, Phillips GS, et al. Widening rural–urban disparities in youth suicides, United States, 1996–2010. *JAMA Pediatr* 2015; 169: 466.

48. Patel V, Ramasundarahettige C, Vijayakumar L, et al. Suicide mortality in India: a nationally representative survey. Lancet 2012; 379: 2343–2351.

49. Levin KA, Leyland AH. Urban/rural inequalities in suicide in Scotland, 1981–1999. *Soc Sci Med* 2005; 60: 2877–2890.

50. Ngamini Ngui A, Apparicio P, Moltchanova E, Vasiliadis H-M. Spatial analysis of suicide mortality in Québec: spatial clustering and area factor correlates. *Psychiatry Res* 2014; 220: 20–30.

51. Kapusta ND, Zorman A, Etzersdorfer E, Ponocny-Seliger E, Jandl-Jager E, Sonneck G. Rural–urban differences in Austrian suicides. *Soc Psychiatry Psychiatr Epidemiol* 2008; 43: 311–318.

52. Searles VB, Valley MA, Hedegaard H, Betz ME. Suicides in urban and rural counties in the United States, 2006–2008. *Crisis* 2014; 35: 18–26.

53. Qi X, Hu W, Page A, Tong S. Dynamic pattern of suicide in Australia, 1986–2005: a descriptive-analytic study. *BMJ Open* 2014; 4: e005311.

54. Chang SS, Sterne JA, Wheeler BW, Lu TH, Lin JJ, Gunnell D. Geography of suicide in Taiwan: spatial patterning and socioeconomic correlates. *Health Place* 2011; 17: 641–650.

55. Park B, Lester D. Rural and urban suicide in South Korea. *Psychol Rep*. 2012; 111: 495–497.

56. Frasquilho D, Matos MG, Salonna F, et al. Mental health outcomes in times of economic recession: a systematic literature review. *BMC Public Health* 2016; 16: 115.

57. Martin-Carrasco M, Evans-Lacko S, Dom G, et al. EPA guidance on mental health and economic crises in Europe. *Eur Arch Psychiatry Clin Neurosci* 2016; 266: 89–124.

58. Durkheim E, Spaulding JA, Simpson G. *Suicide: A Study in Sociology*. London: Routledge & Kegan Paul, 1952.

of first contact longitudinal register approaches. *Soc Psychiatry Psychiatr Epidemiol* 2017; 52: 147–154.

59. Nazroo JY. The structuring of ethnic inequalities in health: Economic position, racial discrimination, and racism. *Am J Public Health* 2003; 93: 277–284.

60. Ødegaard Ø. Emigration and insanity. *Acta Psychiatr Neurol* 1932; 4 (Suppl.): 1–206.

61. Robins L, Locke B, Regier D. An overview of psychiatric disorders in America. In: Robins LN, Regier DA (eds). *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study*. Free Press: New York, 1991, pp. 228–267.

62. Weich S, Nazroo J, Sproston K, et al. Common mental disorders and ethnicity in England: the EMPIRIC study. *Psychol Med* 2004; 34: 1543–1551.

63. Weissman MM, Bruce ML, Leaf PJ, Florio LP, Holzer C, III. Affective disorders. In: Robins L, Regier DA (eds). *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study*. Free Press: New York, 1991, pp. 53–80.

64. Missinne S, Bracke P. Depressive symptoms among immigrants and ethnic minorities: a population based study in 23 European countries. *Soc Psychiatry Psychiatr Epidemiol* 2012; 47: 97–109.

65. Cooper C, Spiers N, Livingston G, et al. Ethnic inequalities in the use of health services for common mental disorders in England. *Soc Psychiatry Psychiatr Epidemiol* 2013; 48: 685–692.

66. Brugha T, Jenkins R, Bebbington P, Meltzer H, Lewis G, Farrell M. Risk factors and the prevalence of neurosis and psychosis in ethnic groups in Great Britain. *Soc Psychiatry Psychiatr Epidemiol* 2004; 39: 939–946.

67. Swinnen SGHA, Selten JP. Mood disorders and migration: meta-analysis. *Br J Psychiatry* 2007; 190: 6–10.

68. Karlsen S, Nazroo JY, McKenzie K, Bhui K, Weich S. Racism, psychosis and common mental disorder among ethnic minority groups in England. *Psychol Med* 2005; 35: 1795–1803.

69. Bhui K, Stansfeld S, McKenzie K, Karlsen S, Nazroo J, Weich S. Racial/ethnic discrimination and common mental disorders among workers: findings from the EMPIRIC study of ethnic minority groups in the United Kingdom. *Am J Public Health* 2005; 95: 496–501.

70. Ikram UZ, Snijder MB, Fassaert TJL, Schene AH, Kunst AE, Stronks K. The contribution of perceived ethnic discrimination to the prevalence of depression. *Eur J Public Health* 2015; 25: 243–248.

71. Substance Abuse and Mental Health Services Administration(SAMHSA). *Racial/Ethnic Differences in Mental Health Service Use Among Adults*. HKHS Publication No. SMA-15-4906. Rockville, MD: SAMHSA, 2015.

72. Algeeria M, Chatterji P, Well SK, et al. Disparity in depression among racial and ethnic minority populations in the United States. *Psychiatr Serv* 2008; 59: 1264–1272.

73. McGuire T, Miranda J. Racial and ethnic disparities in mental health care: evidence and policy implications. *Health Aff (Millwood)* 2008; 27: 393–403.

74. Gilliver SC, Sundquist J, Li X, Sundquist K. Recent research on the mental health of immigrants to Sweden: a literature review. *Eur J Public Health* 2014; 24(Suppl. 1): 72–79.

75. Nakash O, Levav I, Gal G. Common mental disorders in immigrant and second-generation respondents: results from the Israel-based World Mental Health Survey. *Int J Soc Psychiatry* 2013; 59: 508–515.

76. Das-Munshi J, Leavey G, Stansfeld SA, Prince MJ. Migration, social mobility and common mental disorders: critical review of the literature and meta-analysis. *Ethn Health* 2012; 17: 17–53.

77. Lindert J, Ehrenstein OS von, Priebe S, Mielck A, Brähler E. Depression and anxiety in labor migrants and refugees—a systematic review and meta-analysis. *Soc Sci Med* 2009; 69: 246–257.

78. Porter M, Haslam N. Predisplacement and postdisplacement of refugees and internally displaced persons. *JAMA* 2005; 294: 610–612.

79. Schwartz RC, Blankenship DM. Racial disparities in psychotic disorder diagnosis: a review of empirical literature. *World J Psychiatry* 2014; 4: 133–140.

80. Kirkbride J, Errazuriz A, Croudace T, et al. *Systematic Review of the Incidence and Prevalence of Schizophrenia and Other Psychoses*

in England. London: Department of Health Policy Research Programme, 2012.

81. King M, Coker E, Leavey G, Hoare A, Johnson-Sabine E. Incidence of psychotic illness in London: comparison of ethnic groups. *BMJ* 1994; 309: 1115–1119.

82. Cantor-Graae E, Pedersen CB. Full spectrum of psychiatric disorders related to foreign migration. *JAMA Psychiatry* 2013; 70: 427.

83. Cantor-Graae E, Pedersen CB. Risk of schizophrenia in second-generation immigrants: a Danish population-based cohort study. *Psychol Med* 2007; 37: 485–494.

84. Pedersen CB, Demontis D, Pedersen MS, et al. Risk of schizophrenia in relation to parental origin and genome-wide divergence. *Psychol Med* 2012; 42: 1515–1521.

85. Amad A, Guardia D, Salleron J, Thomas P, Roelandt JL, Vaiva G. Increased prevalence of psychotic disorders among third-generation migrants: Results from the French Mental Health in General Population survey. *Schizophr Res* 2013; 147: 193–195.

86. Kirkbride JB, Hollander AC. Migration and risk of psychosis in the Canadian context. *CMAJ* 2015; 187: 637–638.

87. Selten JP, Cantor-Graae E, Kahn RS. Migration and schizophrenia. *Curr Opin Psychiatry* 2007; 20: 111–115.

88. Coid JW, Kirkbride JB, Barker D, et al. Raised incidence rates of all psychoses among migrant groups: findings from the East London first episode psychosis study. *Arch Gen Psychiatry* 2008; 65: 1250–1258.

89. Tortelli A, Errazuriz A, Croudace T, et al. Schizophrenia and other psychotic disorders in caribbean-born migrants and their descendants in england: systematic review and meta-analysis of incidence rates, 1950–2013. *Soc Psychiatry Psychiatr Epidemiol* 2015; 50: 1039–1055.

90. Cantor-Graae E, Zolkowska K, McNeil TF. Increased risk of psychotic disorder among immigrants in Malmö: a 3-year first-contact study. *Psychol Med* 2005; 35: 1155–1163.

91. Hollander A-C, Dal H, Lewis G, Magnusson C, Kirkbride JB, Dalman C. Refugee migration and risk of schizophrenia and other non-affective psychoses: cohort study of 1.3 million people in Sweden. BMJ 2016; 352: i1030.

92. Zolkowska K, Cantor-Graae E, McNeil TF. Increased rates of psychosis among immigrants to Sweden: is migration a risk factor for psychosis? *Psychol Med* 2001; 31: 669–678.

93. Veling W. Ethnic minority position and risk for psychotic disorders. *Curr Opin Psychiatry* 2013; 26: 166–171.

94. Bresnahan M, Begg MD, Brown A, et al. Race and risk of schizophrenia in a US birth cohort: another example of health disparity? *Int J Epidemiol* 2007; 36: 751–758.

95. Smith GN, Boydell J, Murray RM, et al. The incidence of schizophrenia in European immigrants to Canada. *Schizophr Res* 2006; 87: 205–211.

96. Weiser M, Werbeloff N, Vishna T, et al. Elaboration on immigration and risk for schizophrenia. *Psychol Med* 2008; 38: 1113–1119.

97. Fearon P, Kirkbride JB, Morgan C, et al. Incidence of schizophrenia and other psychoses in ethnic minority groups: results from the MRC AESOP Study. *Psychol Med* 2006; 36: 1541–1550.

98. Kirkbride JB, Barker D, Cowden F, et al. Psychoses, ethnicity and socio-economic status. *Br J Psychiatry* 2008; 193: 18–24.

99. Kirkbride JB, Barker D, Cowden F, et al. Psychoses, ethnicity and socio-economic status. *Br J Psychiatry* 2008; 193: 18–24.

100. Bhugra D. Migration and schizophrenia. *Acta Psychiatr Scand* 2000; 102: 68–73.

101. Fung WLA, Jones PB, Bhugra D. Ethnicity and mental health: the example of schizophrenia and related psychoses in migrant populations in the Western world. *Psychiatry* 2009; 8: 335–341.

102. Morgan C, Charalambides M, Hutchinson G, Murray RM. Migration, ethnicity, and psychosis: toward a sociodevelopmental model. *Schizophr Bull* 2010; 36: 655–664.

103. Littlewood R, Lipsedge M. Migration, ethnicity and diagnosis. *Psychiatr Clin (Basel)* 1978; 11: 15–22.

104. Hickling FW, Rodgers-Johnson P. The incidence of first contact schizophrenia in Jamaica. *Br J Psychiatry* 1995; 167: 193–196.

105. Bhugra D, Hilwig M, Hossein B, et al. First-contact incidence rates of schizophrenia in Trinidad and one-year follow-up. *Br J Psychiatry* 1996; 169: 587–592.

106. Mahy GE, Mallett R, Leff J, Bhugra D. First-contact incidence rate of schizophrenia on Barbados. *Br J Psychiatry* 1999; 175: 28–33.

107. Demjaha A, Morgan K, Morgan C, et al. Combining dimensional and categorical representation of psychosis: the way forward for DSM-V and ICD-11? *Psychol Med* 2009; 39: 1943–1955.

108. Morgan C, Hutchinson G. The social determinants of psychosis in migrant and ethnic minority populations: a public health tragedy. *Psychol Med* 2009 (Epub ahead of print).

109. McKenzie K, Bhui K, Nanchahal K, Blizard B. Suicide rates in people of South Asian origin in England and Wales: 1993–2003. *Br J Psychiatry* 2008; 193: 406–409.

110. Bhui KS, McKenzie K. Rates and risk factors by ethnic group for suicides within a year of contact with mental health services in England and Wales. *Psychiatr Serv* 2008; 59: 414–420.

111. Hjern A, Allebeck P. Suicide in first- and second-generation immigrants in Sweden. A comparative study. *Soc Psychiatry Psychiatr Epidemiol* 2002; 37: 423–429.

112. Cavan R. *Suicide.* Chicago, IL: University of Chicago Press, 1928.

113. Spallek J, Reeske A, Norredam M, Nielsen SS, Lehnhardt J, Razum O. Suicide among immigrants in Europe—a systematic literature review. *Eur J Public Health* 2015; 25: 63–71.

114. Voracek M, Loibl LM. Consistency of immigrant and country-of-birth suicide rates: a meta-analysis. *Acta Psychiatr Scand* 2008; 118: 259–271.

115. Kliewer E. Immigrant suicide in Australia, Canada, England and Wales, and the United States. *J Aust Popul Assoc* 1991; 8: 111–128.

116. Barnett AG, van der Pols JC, Dobson AJ. Regression to the mean: what it is and how to deal with it. *Int J Epidemiol* 2005; 34: 215–220.

117. Leenaars A a, Echohawk M, Lester D, Leenaars L. What suicide among indigenous peoples: what does the international knowledge tell us? *Can J Native Stud* 2007; 2: 479–501.

118. McKenzie K, Serfaty M, Crawford M. Suicide in ethnic minority groups. *Br J Psychiatry* 2003; 183: 100–101.

119. Ferguson M, Baker A, Young S, Procter N. Understanding suicide among aboriginal communities. *Aust Nurs Midwifery J* 2016; 23: 36.

120. Crawford A. Inuit take action towards suicide prevention. *Lancet* 2016; 388: 1036–1038.

121. Rockhill B, Newman B, Weinberg C. Use and misuse of population attributable fractions. *Am J Public Health* 1998; 88: 15–19.

122. Rose G. Sick individuals and sick populations. *Int J Epidemiol* 1985; 14: 32–38.

123. Clark DM. Implementing NICE guidelines for the psychological treatment of depression and anxiety disorders: the IAPT experience. *Int Rev Psychiatry* 2011; 23: 318–327.

124. Mukuria C, Brazier J, Barkham M, et al. Cost-effectiveness of an Improving Access to Psychological Therapies service. *Br J Psychiatry* 2013; 202: 220–227.

125. Hammond GC, Croudace TJ, Radhakrishnan M, et al. Comparative effectiveness of cognitive therapies delivered face-to-face or over the telephone: an observational study using propensity methods. *PLOS ONE* 2012; 7: e42916.

126. Richards DA, Suckling R. Improving access to psychological therapies: phase IV prospective cohort study. *Br J Clin Psychol* 2009; 48: 377–396.

127. Booth KVP, Paunesku D, Msall M, Fogel J, Van Voorhees BW. Using population attributable risk to help target preventive interventions for adolescent depression. *Int J Adolesc Med Health* 2008; 20: 307–319.

128. Goodman E, Slap GB, Huang B. The public health impact of socioeconomic status on adolescent depression and obesity. *Am J Public Health* 2003; 93: 1844–1850.

129. Saxena S, Jané-Llopis E, Hosman C. Prevention of mental and behavioural disorders: implications for policy and practice. *World Psychiatry* 2006; 5: 5–14.

130. World Health Organization (WHO). *Prevention of Mental Disorders: Effective Interventions and Policy Options.* Geneva: WHO, 2004.

131. Kirkbride J, Coid JW, Morgan C, et al. Translating the epidemiology of psychosis into public mental health: evidence, challenges and future prospects. *J Public Ment Health* 2010; 9: 4–14.

132. Li Z, Page A, Martin G, Taylor R. Attributable risk of psychiatric and socio-economic factors for suicide from individual-level, population-based studies: a systematic review. *Soc Sci Med* 2011; 72: 608–616.

133. Qin P, Agerbo E, Mortensen PB. Suicide risk in relation to socioeconomic, demographic, psychiatric, and familial factors: a national register-based study of all suicides in Denmark, 1981–1997. *Am J Psychiatry* 2003; 160: 765–772.

134. Mrazek PJ, Haggerty RJ. *Reducing Risks for Mental Disorders: Frontiers for Preventive Intervention Research.* Washington, DC: National Academy Press, 1994.