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Migration, Ethnicity, and Psychosis: Toward a Sociodevelopmental Model

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There is consistent and strong evidence that the incidence of all psychoses is higher in many migrant and minority ethnic populations in a number of countries. The reasons for this are, however, unclear and a wide range of explanations have been proposed, from genetic to neurodevelopmental to psychosocial. In this article, we describe and evaluate the available evidence for and against each of these. What this shows is that: (1) there are few studies that have directly investigated specific risk factors in migrant and minority ethnic populations, with often only 1 or 2 studies of any relevance to specific explanations and (2) what limited research there has been tends to implicate a diverse range of social factors (including childhood separation from parents, discrimination and, at an area level, ethnic density) as being of potential importance. In an attempt to synthesize these disparate findings and provide a basis for future research, we go on to propose an integrated model—of a sociodevelopmental pathway to psychosis—to account for the reported high rates in migrant and minority ethnic populations. Aspects of this model will be directly tested in a new Europe-wide incidence and case–control study that we will conduct over the next 3 years, as part of the European Network of National Schizophrenia Networks studying Gene–Environment Interactions programme.

Key words: migration/ethnicity/psychosis/social

Introduction

There is consistent evidence that the incidence of schizophrenia and other psychoses is elevated in migrant and minority ethnic populations.[1] This appears to hold for a range of groups in many countries (eg, the United Kingdom, the Netherlands, Denmark, Sweden, Australia, and the United States).[2–6] These findings, however, have proved contentious and, despite much speculation and a number of recent studies, it remains unclear why incidence rates are (seemingly) elevated in many diverse groups.[7] In this article, we (*a*) review the various explanations that have been proposed to account for these high rates and evaluate the evidence for and against each, (*b*) go on to propose an integrated model, of a largely sociodevelopmental pathway to psychosis, to account for the high rates, and (*c*) briefly introduce a new Europe-wide incidence and case–control study of psychosis (funded by the European Community's Seventh Framework Program under grant agreement No. HEALTH-F2-2009-241909 [Project EU-GEI]), one aim of which is to further investigate the factors that might account for the reported high rates.[8] Understanding the origins of the increased incidence in migrant and minority ethnic groups is an essential prerequisite to developing appropriate public health responses[9] and a unique opportunity to shed light on the etiology of psychosis in general.

High Incidence Rates

There have been a number of recent reviews summarizing the literature on migration, ethnicity, and the incidence of psychosis[1,2,10]—a literature that dates back to studies in the 1930s by Ødegaard of Norwegian migrants to the United States.[11] In the most systematic, Cantor-Graae and Selten[1] conducted a meta-analysis of incidence rates from 18 studies in a number of countries. They found an overall weighted relative risk (RR) for schizophrenia of 2.9 (95% CI 2.5–3.4) in first- and second-generation migrants compared with nonmigrants. The RR was greatest in second-generation migrants (RR 4.5; 95% CI 1.5–13.1), in migrants from developing countries (RR 3.3; 95% CI 2.8–3.9), and from countries where the majority of the population is black (RR 4.8; 95% CI 3.7–6.2). What is particularly notable is that the degree of elevated risk appears to

vary between and within migrant and minority ethnic populations. This is borne out in specific studies. For example, the U.K. ÆSOP study found that incidence rate ratios for all psychoses (with white British as the baseline group) ranged between 1.5 (95% CI 0.9–2.4) for the Asian and 6.7 (95% CI 5.4–8.3) for the black Caribbean populations.[3] These variations have not been widely commented on but they hint at the operation of different risk and protective factors within and between diverse groups.

Methodological Artifact and Misdiagnosis

A common question about these findings is whether they are valid or whether they are a function of methodological artifact or misdiagnosis.[12] As studies have become more robust (with comprehensive case finding, accurate population denominator data, and standardized procedures for diagnosis), most practical methodological concerns have been addressed.[2] However, misdiagnosis remains a frequently proposed explanation,[13,14] the essence of the argument being that the apparent high rates are a consequence of researchers and/or clinicians erroneously diagnosing schizophrenia in individuals from migrant and minority ethnic groups who are in fact either (*a*) experiencing a mood or brief reactive disorder or, more broadly, (*b*) expressing culturally appropriate emotional distress in response to difficult life circumstances.[7] Only a small number of studies have directly addressed these possibilities (all in relation to the U.K. black Caribbean population), and none have provided any evidence to suggest misdiagnosis, in any form, can account for the reported high rates.[15] For example, in a study at the Maudsley Hospital in London (where many of the U.K. studies of migration, ethnicity, and psychosis have been conducted), Hickling et al[16] compared diagnoses made independently by British psychiatrists and by a Jamaican psychiatrist in the same group of 66 inpatients. The authors found no difference in the percentage of black inpatients diagnosed with schizophrenia by the British psychiatrists or the Jamaican psychiatrist. In 2 other U.K. studies designed to investigate racial stereotyping in psychiatric assessment using case vignettes, there was no evidence that psychiatrists were more likely to diagnose schizophrenia when the ethnicity of the individual in the vignette was black.[17,18] What is more, researchers have increasingly moved away from a narrow focus on a single diagnosis (schizophrenia) to consider the full spectrum of psychoses, including psychosis-like experiences in community samples,[19–21] and the findings have been broadly the same—the incidence and prevalence of all psychoses (including affective and brief reactive) are more or less elevated in most migrant and minority ethnic populations that have been studied.[3,22,23]

A Note on Symptomatology

There is, nevertheless, evidence that within the broad spectrum of psychoses there are some differences between groups in the constellations of presenting symptoms and experiences. For example, in the United Kingdom, there are a number of studies that suggest black patients tend to present with more reality distortion (delusions and hallucinations) and affective symptoms and with fewer negative symptoms, when compared with white patients.[24–26] As far as we are aware, there are no comparable data from other countries, and the generalizability of these findings is unclear. They do, however, raise the intriguing possibility that such variations may reflect different etiological pathways to psychosis in migrant and minority ethnic groups.

Candidate Explanations

Beyond methodological artifact and misdiagnosis, a number of substantive explanations have been proposed to account for the repeated finding that the incidence of schizophrenia and other psychoses is elevated in many migrant and minority ethnic groups.[27] These tend to draw from what we know about psychosis generally and can be grouped broadly into: (*a*) selective migration, (*b*) genetic, (*c*) neurodevelopmental, (*d*) substance use, and (*e*) (psycho)social.

Selective Migration

Ødegaard argued that the high rates of schizophrenia he observed in Norwegian migrants to the United States could be explained by selective migration, ie, the greater tendency for individuals with an existing (genetic) predisposition or vulnerability for schizophrenia to migrate.[11] This conclusion was based on Ødegaard's observation that many of the migrants who developed schizophrenia had histories of poor social adaptation in Norway.[11] However, when applied to other populations in which high rates have been reported, this explanation seems unlikely. Selten and colleagues[28] conducted an intriguing thought experiment to test this in relation to Surinamese migrants to the Netherlands. They imagined that the entire population of Surinam had migrated to the Netherlands and, using this to inflate denominator data and assuming none of these contributed any further cases of schizophrenia, recalculated incidence rates from an earlier study. Having done this, they found that the risk for Surinamese migrants was still significantly higher than for Dutch individuals (RR 1.46, 95% CI 1.35–1.57). What is more, intuitively it seems reasonable to expect that the cognitive deficits and negative symptoms that are often evident prior to the onset of schizophrenia (and which are assumed to reflect underlying genetic and neurodevelopmental risk) will in fact reduce the likelihood of successful migration. In the only study, we are

aware of that has attempted to investigate the impact of psychosis proneness on likelihood of migration, Lundberg et al[29] evaluated potential future migrants in Kampala (Uganda) and found no differences on measures of psychosis-like experiences and mania between those actively planning to migrate and a comparison group with no intention to migrate.

Genetic

It is not surprising, given that schizophrenia and other psychoses are highly heritable, that some attention has focused on the potential direct role of genetics[27,30]—a difficult and controversial topic when set alongside race and ethnicity.[12] There is, however, no evidence that the high rates are a consequence of greater genetic risk in certain migrant and minority ethnic populations. The very fact that so many diverse groups appear to have higher rates suggests that this is unlikely. More specifically, 2 U.K. studies[31,32] that examined risk in relatives (parents and siblings) of individuals with schizophrenia from white and black Caribbean groups both found no differences in risk of schizophrenia between white and black Caribbean parents—a finding that suggests similar degrees of genetic risk in the 2 groups. Both these studies, however, did find that risk of schizophrenia was elevated in black Caribbean siblings, a finding that hints at a role for environmental factors. Similar findings have been reported for parents of Moroccan-Dutch patients with psychosis in the Netherlands.[33] In addition, if population differences in genetic risk did underpin the reported high rates, it would be expected that rates would be high in the originating countries. There is, however, no evidence that the incidence of schizophrenia or other psychoses is similarly elevated in any relevant country (eg, 3 studies[34–36] in the Caribbean found incidence rates much lower than have been reported for the Caribbean population in the United Kingdom). This does not, of course, rule out a possible role for gene–environment interactions, but if such processes are relevant, it is likely that the critical component is differential exposure to environmental factors operating on a similar overall genetic risk.

Neurodevelopmental

Others have considered the potential role of neurodevelopmental risk factors (ie, maternal viral infections, obstetric complications, vitamin D deficiency). These were highlighted by Eagles[37] in an early review and draw from broader evidence linking these with risk of schizophrenia through their impact on early brain development.

Viral Infection There is, for example, evidence that prenatal infection (specifically rubella, influenza, and toxoplasmosis) is associated with an increased risk of schizophrenia in offspring.[38] This raises the possibility that migrants from countries where such infections are uncommon are more at risk when traveling to new countries due to lower immunity.[37] This appears to have been true for postwar migrants from the Caribbean to the United Kingdom; in the 1950s and 1960s, there was an epidemic of congenital rubella in this population.[39] This is, moreover, consistent with the finding that rates of schizophrenia and other psychoses tend to be higher in second-generation migrants. This is, however, largely conjecture, and there is no direct evidence that this has contributed to increased rates in the Caribbean population in the United Kingdom nor does it explain increased rates in first-generation migrants and their persistence in the third and subsequent generations. It is also less plausible as a contributory factor for migrants from areas where such infections are common (eg, Africa[40]).

Obstetric Complications There is a large literature suggesting obstetric complications are associated with a modest increased risk of schizophrenia in offspring,[41] and there has been speculation that a higher prevalence of such complications in migrant groups, coupled with increased infant survival rates in the new countries, may contribute to the observed increases in incidence.[27,37] However, again the limited available evidence suggests this is unlikely. For example, McKenzie and Murray[42] report that there was no evidence of higher rates of obstetric complications in black Caribbean individuals with psychosis, compared with white individuals, in the Camberwell Functional Psychosis Study.[43] If anything, rates were lower in the black Caribbean group.[42] Similarly, in a study of 103 white cases with psychosis and 61 black Caribbean cases, Hutchinson et al[44] found a trend for pregnancy and birth complications to be more common in the white group—a finding that very tentatively suggests early neurodevelopmental insults may in fact be relatively less important in migrant groups. However, we are not aware of any relevant studies in other populations, which again raises questions about the generalizability of existing data.

Vitamin D Deficiency Along similar lines, McGrath[45] has hypothesized that low prenatal vitamin D may impact on brain development in such a way as to increase risk of schizophrenia in offspring and that black migrants moving to colder climates may experience vitamin D deficiencies as a consequence of reduced exposure to sunlight. This, however, is an extremely difficult hypothesis to test directly. It does seem that migrants from countries where the majority population is black have the highest increased risk of developing schizophrenia and other psychoses.[1] However, this finding can equally plausibly be considered in terms of exposure to racial discrimination—it is individuals from black migrant and minority groups who are most visible in predominantly white societies and who are most likely to

experience discrimination. What is more, the highest rates of vitamin D deficiency disorders in the United Kingdom have paradoxically been in Asian groups who have lower rates of psychosis than black groups.

Data from the ÆSOP study[46] provide additional relevant evidence, mainly further suggesting that neurodevelopmental factors are unlikely to be of primary importance in explaining the high rates of psychosis, at least in the U.K. black Caribbean and black African populations. For example, we found that scores on assessments of markers of abnormal neurodevelopment (ie, minor physical anomalies, neurological soft signs) were similar for white, black Caribbean, and black African cases.[47,48] In analyses of magnetic resonance imaging data, we did find greater differences in brain structure between black cases with a first episode of psychosis and black controls (eg, reduced global gray matter, increased lingual gyrus gray-matter volume) than between white cases and white controls.[49] This could reflect exposure to more early neurological insults, but it is equally plausible that this finding is a consequence of greater exposure to adversity and trauma during childhood in the black group or, perhaps most likely, to differential exposure to antipsychotics.

Substance Use

One of the earliest (and most controversial) explanations for the high rates in the U.K. black Caribbean population implicated cannabis use.[27] Recent work on psychosis in general appears to confirm an association with use of cannabis,[50] particularly forms high in tetrahydrocannabinol (eg, ''skunk,'' sinsemilla).[51] The small number of studies that have investigated this in migrant and minority ethnic populations, however, do not provide strong evidence that this is an important factor. Two United Kingdom studies found no difference in reported rates of cannabis use in white and black individuals with a psychosis.[52,53] More recent statistics from the British Crime Survey suggest that cannabis use among 16–59 year olds from black and white British groups is broadly similar.[54] (However, the failure to distinguish black Caribbean and black African may have affected this finding because there may generally be lower use of cannabis in the black African population.) In the Netherlands, data again suggest that migrants from the Caribbean are not more likely than Dutch individuals to use cannabis.[55] It has also been noted that while use of cannabis is high in the Caribbean, there is no evidence that rates of psychosis are particularly high.[27] We should be cautious, however, before dismissing cannabis as a potential contributory factor in the high rates. The evidence to date is weak, with no studies having taken into account age of first use or duration, amount, and type of cannabis used. Intriguingly, a study conducted in Trinidad found that the incidence of psychosis was around 2 times higher in African-Trinidadians compared with Indian-Trinidadians—a finding, initial analyses suggest, that is at least partly accounted for by greater use of cannabis in the African-Trinidadian population.[56]

(Psycho)social

Most attention has focused on the potential role of (psycho)social factors. Eagles[37] began his early review of biological hypotheses by noting that social explanations for the high rates had been accepted almost uncritically, despite a lack of evidence. In the period since, this has changed and relevant data (mainly from the United Kingdom and the Netherlands) have been reported on a number of possible social risk factors and indicators operating at both individual and area (neighborhood) levels.

Before considering these, it is notable that very little attention has been paid to the potential direct impact of migration.[57] Individuals and their families migrate for many reasons (eg, economic betterment, to flee war and persecution, etc.), and the populations of migrants and minority ethnic groups studied to date have very different migration histories. The U.K. black Caribbean population, for example, migrated largely during the 1940s and 1950s to pursue opportunities for work during the postwar economic expansion in the United Kingdom—a very different set of circumstances to those who migrate to seek refuge from war and persecution. All face, to a greater or lesser degree, the stresses of transition from one country to another—unfamiliar cultural practices and beliefs, different climate and environment, challenging interactions with government institutions, and for some a new language.[57] The possibility that processes linked to migration might be relevant to the high rates of schizophrenia in the U.K. black Caribbean population was noted in early reports.[58] However, the apparent delay between migration and onset of disorder (eg, Hemsi[58] reported that a majority had been in the United Kingdom for 2 years or more) meant a direct impact of migration was dismissed and has been largely ignored since. This is surprising. Incidence rates are elevated in first-generation migrants, and we know that the impact of stressors may persist beyond their immediate occurrence.

Individual-Level Exposures

There are a number of published analyses that have investigated the relationship between individual-level variables signifying exposure to social adversity at different points in the life course and risk of psychosis in migrant and minority ethnic groups. In a case–control study conducted in south London, for example, Mallet et al[59] found that 3 variables in particular differentiated black Caribbean individuals with a first episode of schizophrenia from controls: unemployment, living alone, and separation from a parent during childhood. We sought to replicate and extend these findings using data on a larger

sample (n = 390 cases, 391 controls) from the ÆSOP study.[60,61] We found: (*a*) separation from a parent due to family breakdown in childhood was associated with a 2- to 3-fold increased odds of psychosis[60]; (*b*) this held for all ethnic groups, but separations (over and above those associated with migration) were much more common in the black Caribbean group[60]; (*c*) a series of markers of current and long-standing adult social disadvantage (ie, unemployment, living alone, being single, poor education, and limited social networks) were associated with a linear increase in odds of psychosis[61]; and (*d*) this held for all ethnic groups, but cumulative disadvantage was much more common in the black Caribbean group.[61] Our interpretation of these findings is that, if these variables (separation from parents; markers of disadvantage) index exposure to experiences that increase risk of psychosis, their greater prevalence in the black Caribbean may partly explain the increased rates.[9] There are, however, a number of specific limitations. The variables used are crude, no account is taken of mitigating factors (eg, social supports), and it is not possible to disentangle cause and effect. In short, our interpretation is necessarily speculative and whether these findings hold for groups in other countries is unknown.

Other studies have considered specific experiences that may impact primarily on migrant and minority ethnic populations. In analyses of data from the Fourth National Survey of Ethnic Minorities in the United Kingdom, Karlsen and Nazroo[62] found an association between the estimated annual prevalence of psychosis and reports of exposure to verbal abuse (OR 2.9), racial attacks (OR 4.8), and perceived employer racism (OR 1.6). Gilvarry et al,[63] in a study of the frequency of life events and perceived discrimination in a sample of 147 individuals with a long-standing diagnosis of psychosis, found that black Caribbean participants were more likely to attribute life events to discrimination. However, as both these studies were of prevalence samples it is not clear to what extent such experiences and perceptions can be linked to onset of disorder. They are, nonetheless, broadly in line with our finding in further analyses of ÆSOP data that perceived disadvantage partly explained the association between black ethnicity and psychosis.[64] Related to this, Veling et al[65] used general population data on perceptions of discrimination in the Netherlands to order ethnic groups according to levels of exposure to discrimination. They then linked this to incidence rates for schizophrenia and found clear evidence of a linear relationship, with the highest rates being in those groups with the highest levels of perceived discrimination (ie, Moroccan, incidence rate ratio 4.8). In a further set of analyses, Veling et al[66] examined ethnic identity in a sample of 100 non-Western migrants with a first episode of psychosis, 100 matched controls, and 63 siblings; cases were more likely to have a negative ethnic identity (ie, lack of affinity to own ethnic group) than their matched controls (OR 3.29). The authors speculate that, in not identifying with their own ethnic group, some individuals may be more vulnerable to the impact of discrimination. Along similar lines, in a case–control study of first-episode psychosis in the United Kingdom, Bhugra et al[67] found evidence that black Caribbean cases were less likely than their matched controls to identify with their culture of origin.

Area-Level Exposures

The wider contexts within which individuals live have also been investigated and findings suggest that contextual factors may moderate the impact of exposure to specific stressors on risk of psychosis.[68,69] This is most clearly suggested in the now replicated finding that the RR of schizophrenia in migrant and minority ethnic groups is highest in those areas where they form a smaller proportion of the local population. In other words, the less ethnically dense an area, the higher the RR. This was first reported by Faris and Dunham[70] in the 1930s in their study of hospital admissions in Chicago and, more recently, has been reported in 3 separate studies in the United Kingdom and the Netherlands.[71–73] This is particularly interesting when set alongside individual level data suggesting a link between experiences of discrimination and psychosis. It has been suggested, for example, that living in areas of high ethnic density may have a protective effect, perhaps mitigating the impact of discrimination, isolation, and disadvantage.[72,74] Again, this interpretation is speculative, and the hypothesized processes underpinning this finding have not been directly investigated. What is notable, however, is that it such a patterning of risk defies ready explanation in terms of social drift or known biological risk factors.[9]

More broadly, there is now strong evidence that living in more densely populated urban areas is associated with an increased risk of schizophrenia.[75,76] This has led to some speculation about whether the high rates are largely a consequence of urban living.[77] What limited evidence there is on this, however, suggests that this is unlikely to be the whole explanation. For example, Harrison et al[30] found no evidence that area of residence could explain their finding of high rates in the black Caribbean population in Nottingham, U.K. Similarly, in the ÆSOP study (which included centers with varying degrees of population density), the incidence of all psychoses was similarly elevated for each ethnic group in all centers.[3,78]

Taking Stock

Table 1 summarizes the current evidence for each of the main proposed explanatory factors discussed above.

Two conclusions can be derived from the above discussion: (1) overall, there is a limited amount of research

Table 1. Summary of Evidence on Specific Candidate Explanatory Factors for the High Rates of Psychosis in Migrant and Minority Ethnic Groups

Candidate Explanatory Factors[a]	Number of Studies	Findings	References
Misdiagnosis	3	−, −, −	16–18
Selective migration	2	−, −	28,29
Genetic	3	−, −, −	31–33
Neurodevelopmental			
Viral infection	0	?	—
Obstetric complications	2	−, −	42–44
Vitamin D	0	?	—
Neurological markers	1	−	47,48
Brain structure	1	+	49
Substance use (primarily cannabis)	4	−, −, −, −	52–55
Stressors premigration and during migration	0	?	—
(Psycho)social			
Childhood separation from parents	2	+, +	19,59,60
Adult markers of disadvantage	2	+, +	19,59,61
Discrimination—perceived	3[b]	+, +, +	63–65
Discrimination—reported	1[c]	+	62
Ethnic identity	2	+, +	66,67
Ethnic density	4	+, +, +, +	70–73
Urbanicity	2	−, −	3,30

Note: Studies are not weighted in any way, and no account is taken of study quality. −, negative finding; +, positive finding; ?, no study with direct evidence for or against. A "−" or "+" is given for each study that has directly investigated the candidate explanatory factor.

[a]Studies are only included in this table if they provide direct evidence in relation to a specific explanatory factor. Indirect evidence (eg, absence of high rates in originating countries of migrants suggesting no influence of genetics) is not included.

[b]Includes one study of prevalent cases.

[c]A study of prevalent cases.

that has directly investigated candidate explanatory factors—and most have been focused on a single group, the U.K. black Caribbean population and (2) the limited evidence is most consistent with the high rates being a consequence of greater exposure, in migrant and minority ethnic groups, to various forms of social adversity and problematic social contexts over the life course.

If this latter conclusion is true, the evidence that the degree of elevated risk varies by migrant and ethnic group suggests that social risk and protective factors may cluster differently in these groups. For example, rates appear not to be raised in the U.K. Asian population to the same extent as for the black Caribbean population, despite similar migration histories and, presumably, broadly similar experiences of discrimination.[3] In accounting for this, speculation has tended to focus on the potential buffering effects of familial and social supports, which are assumed to be stronger in Asian populations.[9] What this example suggests is that much might be learned from direct comparisons of migrant and minority ethnic populations.

Furthermore, research has so far proceeded by testing specific individual risk factors, largely in isolation from others, with only limited theorizing about how these might combine or interact to produce high population rates of disorder. Consequently, the (tentative) development of an integrated model based on existing evidence may prove particularly useful as further research is planned and conducted.

A Sociodevelopmental Pathway?

In recent years, there has been a resurgence of interest in the potential role of socioenvironmental factors in the etiology of schizophrenia and other psychoses,[79] partly as a consequence of the findings discussed above. In addition to the literature on migration and ethnicity, there is now robust research linking urbanicity,[75] childhood trauma,[80,81] and social adversity over the life course with the onset of psychosis.[82] What has given further impetus to this work is the elucidation of a number of plausible mechanisms (including gene × environment interaction,[83] sensitization of the mesolimbic dopaminergic system,[84,85] dysregulation of the hypothalamic–pituitary–adrenal axis,[86,87] and development of problematic cognitive schema[88,89]) through which social experiences might impact on individuals to increase risk for psychosis. Selten and Cantor-Graae[90] have already drawn on evidence from animal studies that suggest "social defeat" and subsequent isolation in rodents can produce dopaminergic hyperactivity in the mesocorticolimbic system to develop a theory that the unifying explanation for the reported high rates in migrant groups is prolonged exposure to social defeat, ie, chronic discrimination and isolation. This is an appealing hypothesis and may capture one relevant form of adversity and a potential mechanism through which this and other experiences impact on risk. What the evidence reviewed above (and the wider literature) suggests, however, is that a broader range of social experiences and mechanisms are likely to be relevant. For example, other recent research suggests that repeated exposure to social adversity can link to psychosis through the generation of cognitive biases and affective states that predispose to symptom formation, eg, repeated exposure to threat may link to paranoia and formation of persecutory delusions through increased threat anticipation, anxiety, and a consequent tendency to jump to conclusions.[91,92] This is particularly noteworthy given there is some evidence that persecutory delusions may be more common in migrant and minority ethnic groups (eg, Demjaha et al[25] and Sharpley and Peters[93]). Specific risk and protective factors, moreover, may be relevant in different populations (eg, first- vs second-generation

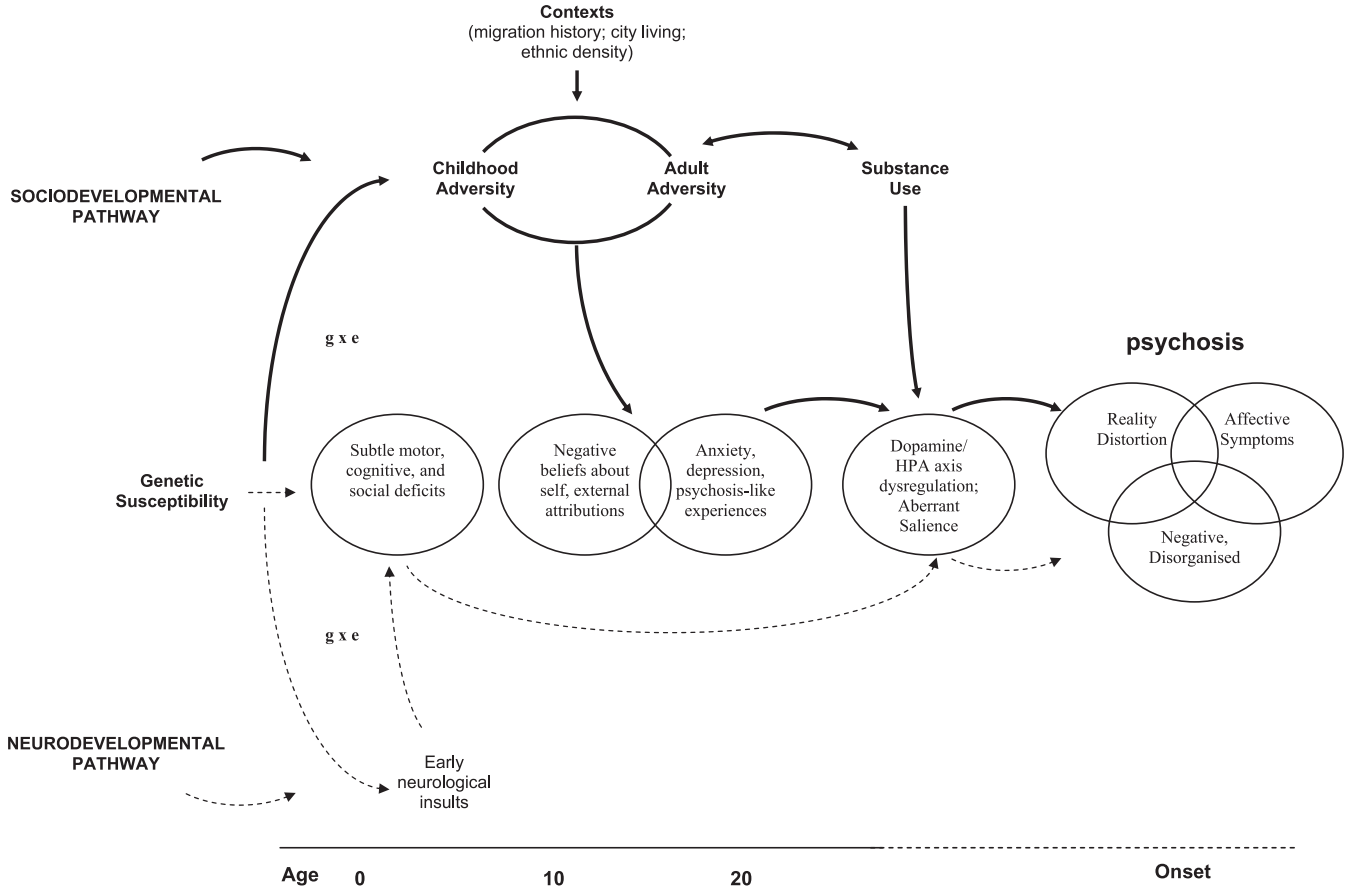

Fig. 1. Hypothesized Sociodevelopmental and Neurodevelopmental Pathways to Psychosis. (Adapted from Murray et al[98]; figure 1, p. s130.)

migrants) at varying stages (eg, immediately premigration and postmigration, during childhood and adulthood).

Taking this broader perspective, the current evidence tentatively raises the question of whether there is a predominantly sociodevelopmental pathway to psychosis that underpins the higher rates of psychosis in migrant and minority ethnic groups.[74] That is, a pathway in which exposure to adversity and trauma[80] (particularly in childhood and/or prior to and during migration) interacts with underlying genetic risk[83] and impacts on brain development[94] (in particular the dopaminergic system[95]) and stress sensitivity[86,87] in such a way as to create an enduring liability to psychosis (reflected in social cognitive biases,[92] the expression of psychosis-like experiences[19], and affective disturbances[91,96]) that becomes manifest (primarily as positive and affective symptoms[25]) in the event of further cumulative stressors[61,97] and/or prolonged substance use, particularly cannabis.[50] We refer to this as sociodevelopmental in the sense that it is social experiences and contexts that make the difference, that, for example, may push more individuals in certain groups along a pathway to psychosis (from a similar base genetic and biological vulnerability), leading to higher population rates of disorder. This is schematically illustrated in figure 1, alongside a more familiar neurodevelopmental pathway.[98]

This is not to imply 2 entirely distinct pathways—there are likely many routes to the development of psychosis, with complex interactions between genetic, neurodevelopmental, psychological, and social factors. What the outline of a specific sociodevelopmental pathway does, however, is highlight the potential centrality for some of problematic social experiences and contexts in the eventual onset of disorder.

Conclusions and Future Directions

There is now very strong evidence that the incidence of schizophrenia and other psychoses is elevated (albeit to varying degrees) in many migrant and minority ethnic populations—a finding that cannot be simply dismissed as methodological artifact. Our review of the various explanations that have been proposed to account for this, and relevant evidence, provisionally suggests that these high rates are largely social in origin. What we have proposed from this—a sociodevelopmental pathway—is, of course, speculative and (as our review suggests) is based on limited data. Recent findings that

the incidence of autism in the United Kingdom is higher in children born to first-generation migrants from the Caribbean[99] reminds us that we should be cautious in dismissing neurodevelopmental explanations, given the apparent overlaps between schizophrenia and autism. As with all models, what we have proposed is primarily a heuristic to guide further research by providing testable hypotheses, and we will have the opportunity to directly test components of this model in a Europe-wide incidence and case–control study of psychosis to be conducted in 12 centers chosen to include areas with large first and subsequent generation migrant populations (part of EU-GEI).[8] Our aim in this is to recruit 1200 cases with a first episode of psychosis, 600 siblings, and 1200 community controls and to collect detailed information on psychopathology and a range of potential risk and protective factors including: social experiences and circumstances (including migration histories, trauma, life events, and social supports); family history of mental disorder; DNA; and neurocognition and social cognition. Data from this study will allow us to directly test our proposed model of a sociodevelopmental pathway and, from this, further contribute to our developing understanding of why there are high rates of psychosis in many migrant and minority ethnic groups.

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