# Mechanisms behind the higher psychosis risks in ethnic minority groups: a scoping review

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# Contents

# Abstract

It's well-established that certain ethnic minority groups in Western countries have a higher risk of psychosis than the general population, and that this effect doesn't stem from these ethnic groups being more genetically disposed towards psychosis. However, the actual mechanisms behind the effect are still unclear. In this thesis paper, I conduct a scoping review of scientific literature published since Morgan et al.'s review on the topic in 2019, in order to map mechanisms posited to explain the connections between psychosocial factors and the raised psychosis risks in ethnic minorities. Additionally, I discuss the strengths and weaknesses of these mechanisms, examine how they can (or can't) work together, and examine how they conceptualise ethnicity and/or race.

APA PsycNet and PubMed were searched, and 35 eligible publications presenting argumentation for mechanisms were identified. The mechanisms suggested by the included articles can be divided into four categories, consisting of primarily biological mechanisms, primarily psychological mechanisms, mechanisms relating to diagnostic processes, and one selective migration mechanism.

All identified mechanisms have their strengths and weaknesses and are generally in need of further research. They are vastly different mechanisms, whereof some could feasibly be parts of the same, more complex mechanisms, some could be working in parallel to each cause some of the effect, and some (mostly the ones with a psychological focus) seem mutually incompatible due to disagreements on what constitutes the core aspects of psychosis to be explained. However, most articles (30 out of 35) point to racism and discrimination as important risk factors leading to the raised psychosis risks, whether this is racism on a structural, institutional, or everyday interpersonal level. Relatedly, both race and ethnicity are mostly conceptualised as social constructs and in terms of how people perceive themselves and others as parts of certain groups and identities, and how those perceived as differing from the (White) majority are discriminated against.

While many of these findings are in line with those of Morgan et al., I found many more mechanisms than they address, and especially psychological mechanisms and ones proposing that some of the effect stems from misdiagnosis receive much more attention in the included literature from the years since 2019 than they do in Morgan et al.'s review.

# Part 1: Introductory framework

The purpose of this master's thesis is to discuss the question of why risks of psychosis are higher among ethnic minority groups than in the general population in Western countries. Overall incidence rates of psychotic disorders for ethnic minorities are reported to be about 1.5-3.0 times higher in these groups combined, but the magnitude depends heavily on the minority group and place being studied (Morgan et al., 2019, p. 247). For example, Tortelli et al. (2015, p. 1050f) found in a meta-analysis that rates of schizophrenia among people of Black Caribbean descent were almost five times higher than in White or general reference populations in the UK. Meanwhile, Kirkbride et al. (2017, p. 1254f) similarly found raised psychosis rates for Black, Pakistani, and Bangladeshi groups in the UK, but not for non-British White groups, and other studies show that rates for migrants in Canada or Israel may not be significantly higher than for the general population (Morgan et al., 2019, p. 247). The raised psychosis rates obviously have certain societal costs but may also be contributing to social inequality via societal stigmatisation of psychosis as well as the considerable effects of psychosis on health and quality of life, in the worst cases leading to a life expectancy shortened by 15-20 years (Nordentoft & Vandborg, 2017, pp. 298; 312).

While the fact that there *is* a problem is rather well-established, it's difficult to design interventions without explanations for *why* there is a problem. For example, if the issue stems from our diagnostic approaches being too 'Westernised', leading to an overdiagnosis of psychosis in patient groups whose culture Western psychiatrists don't understand or approve of, then the solution may lie in reworking diagnostic approaches (Fernando, 1991, pp. 143-145). However, if the issue is caused by psychosocial pressures that disproportionately affect ethnic minorities and raise psychosis risks, the solution may instead be found in political and societal efforts to alleviate these pressures (Anglin et al., 2021, p. 604). There is now strong evidence that the effect doesn't stem from genetic differences between ethnical groups, as psychosis risks among people originating from the same countries vary widely depending on where they live (Jongsma et al., 2021, p. 1914). What *does* cause the effect is still up for debate. Some tentative explanations from the scientific literature were examined by Morgan et al. in 2019, and the main explanatory factors found were psychosocial ones such as the stress of migration, cultural marginalisation, and experiences of discrimination. However, their review is firstly several years old, and secondly didn't find much in the way of

mechanisms behind how these psychosocial factors cause psychosis. Therefore, the problem formulation of this thesis is as follows:

*How does the contemporary scientific literature explain the connection between psychosocial risk factors and raised psychosis risks among ethnic minority groups?*

To study this, I am conducting a scoping review looking for explanations in the scientific literature published since Morgan et al.'s review. Specifically, I am looking for proposed theoretical *mechanisms* behind the raised risks, i.e., explanations that don't just show an empirical connection between e.g. ethnic minorities, urban living and discrimination (all suggested to be risk factors for psychosis; Kelly et al., 2010, p. 76, Pearce et al., 2019, p. 1038f), but ones that try to explain theoretically *how* psychosocial factors like urban living or discrimination cause higher psychosis risks among ethnic minorities. Furthermore, in order to examine the posited explanations, I am asking the following questions:

- What are the theoretical and empirical rationales of the mechanisms, and how do they hold up to critical scrutiny?
- How are psychosis and ethnicity and/or race conceptualised, and how does this affect the conclusions that are reached?
- What happens when we put the different mechanisms together – can some of them complement each other, or are they mutually exclusive?

My attempt to answer these questions is structured in three parts. This introductory framework includes clarification of some terms that are central to the topic, an introduction to the scoping review as a method, and a historical look at the research question and why it is (or at least has been) a controversial one. The second part is the scoping review itself, written in an article format as if intended for publication in a scientific journal. The closing framework contains discussions of the review findings that didn't fit into the article format, including discussion of some of the additional questions asked above.

# Clarification of central terms

A key concept in my problem formulation is psychosis, which is a condition characterised by a loss of contact with reality. The sense of reality and/or ability to 'reality test', i.e. to tell whether your experience is based on reality, is dramatically reduced in a full-blown psychosis (Nordentoft & Vandborg, 2017, p. 300). This may cause delusional thinking, hallucinations, and thought disturbances (Simonsen & Haahr, 2017, p. 325). Of all the psychotic disorders, schizophrenia is considered to be the most invalidating one (at least in its most serious forms), with its heavy and in some cases lifelong effects on quality of life (Videbech et al., 2018, p. 79f). However, there are many ways to experience psychosis that don't necessarily imply schizophrenia. The ICD-10 includes several other diagnoses in its section on psychotic disorders, such as delusional disorders that involve persistent delusions but no hallucinations, or brief psychotic disorders that develop rapidly, but also disappear rapidly, often without returning later (World Health Organization, 1994, pp. 65-79). While the initial studies showing higher psychosis rates in ethnic minorities were focusing on schizophrenia, it is important to also consider other psychoses in the context of this thesis, as more recent studies tend to show higher rates among ethnic minorities for all psychotic disorders, not just schizophrenia (Morgan et al., 2019, p. 250). Up to 10% of the general population will experience psychosis-like symptoms at some point in their lives, and psychosis is increasingly considered a spectrum rather than an 'either/or' category (Simonsen & Haahr, 2017, p. 325). Therefore, in this thesis, I have chosen not to focus on any one specific psychotic disorder, but on the full spectrum of them.

Another central concept to my research question is ethnicity. However, this is also a concept that can be very difficult to define. As Jongsma et al. (2021) discuss, ethnicity can be conceptualised in many ways, e.g. based on migratory history, social structures, and identity, each with different implications for how to look at the raised psychosis risks in ethnic minorities. I am specifically not going to settle on any one conceptualisation of ethnicity to focus on beforehand, as I am interested in uncovering the kinds of explanations posited in the scientific literature, and these may vary in their ethnicity conceptualisations. I therefore find it more interesting to look at what kinds of ideas of ethnicity are used in explanations and in what ways, rather than to close myself off to certain explanations in advance because they differ from any ethnicity definition that I would personally find the most useful.

# The scoping review as a method

Scoping reviews are a variant of the more traditional systematic review. Both approaches have in common that rather than producing new empirical material, they attempt to systematically sift through the already-available literature relevant to the research question in search of an answer. This is done to help make sense of a large amount of research, as well as with the reasoning that an answer based on many sources of evidence is more likely to be accurate than one based on a single study (Petticrew & Roberts, 2006, p. 2f; Pollock et al., 2024, p. 2). But where the systematic review would typically be an attempt to synthesise the best available evidence to answer a somewhat narrow question like the efficacy of a certain intervention, a scoping review is typically used for broader questions about what kinds of research is being conducted on a topic, or where the gaps are in the existing research (Arksey & O'Malley, 2005, pp. 20-22). Rather than synthesising evidence, the aim is therefore to 'map' and provide an overview of the field such as what is known about a topic, what type of research is being conducted, or what the key concepts related to the topic are (Pollock et al., 2024, p. 4f). This also means that while it's possible to only focus on specific types of research studies in a scoping review as one might do in a systematic review, it's often not a necessity since the evidence doesn't need to be synthesised in the same way. Likewise, quality assessment of the included studies is less of a requirement than it is in the systematic review, since the goal rarely is to provide a definitive answer to which parts of the available research hold the 'correct' answer (Arksey & O'Malley, 2005, p. 27; Pollock et al., 2024, p. 4).

Since my problem formulation has to do with characterising certain aspects of the scientific literature regarding ethnic minorities and psychosis risk, the above makes the scoping review an ideal method for my purposes. I am not attempting to synthesise evidence on one possible mechanism for the connection between psychosocial risk factors and psychosis, or to answer how much impact certain psychosocial factors have on psychosis risk. Rather, I am attempting to map and describe what mechanisms are being proposed and what reasoning is being used to support them, for which reason I have chosen to conduct a scoping review. However, my aim is also to critically examine and discuss my findings, which is not necessarily an inherent part of a scoping review. This discussion therefore mainly takes place in the closing framework.

# Psychosis historically

The term ‘psychosis’ was first used in psychiatric literature in 1841 by Karl Friedrich Canstatt as a synonym for ‘psychic neurosis’. ‘Neurosis’ was at the time used as a term for diseases of the nervous system, and a psychic neurosis was thus a disease of the nervous system with psychological manifestations (Bürgy, 2008, p. 1200f). However, Ernst von Feuchtersleben is widely credited as the person who coined the term ‘psychosis’ in 1845, using it for diseases that according to him affected the entire personality and human experience, emphasising the interplay between body and mind and thereby breaking with earlier understandings of mental illness as rooted in either the soul or the body (Beer, 1996, p. 274f; Moskowitz et al., 2019, p. 10). As the psychosis concept developed, focus was increasingly directed towards biological causes like diseases of the brain, even if the exact disease cause had not been found yet. The psychosis concept of the time was still not the one we know today, as, depending on who you asked, it could include not just what we would think of as schizophrenia and psychosis, but also e.g. mood disorders and personality disorders (Beer, 1996, pp. 276-278).

Emil Kraepelin is credited as the first person to describe what we today would consider schizophrenia with his work in the late 1800s on ‘dementia praecox’; a condition that he considered to be a chronic and degenerative disease different from manic depressive conditions. Moreover, he started using the psychosis term for specific illnesses rather than a broad catalogue of mental illness (Beer, 1996, p. 278f; Nordentoft & Vandborg, 2017, p. 298). The word ‘schizophrenia’ was first used by Paul Eugen Bleuler in 1908 as a redefinition of Kraepelin’s ‘dementia praecox’. He moved the emphasis away from a degenerative course, which he didn’t believe was always the case, to a view that ‘splitting’ of psychic functions was the central characteristic of the condition – hence the word schizophrenia, meaning ‘split mind’. Importantly, Bleuler did not mean the kind of ‘Jekyll and Hyde’-style split personality often mistakenly associated with schizophrenia (McNally, 2016, pp. 21; 25). Rather, the splitting in question was one that tore apart the psychic functions, causing the split functions or aspects of personality to be experienced as foreign objects or enemies in the body, and causing an alteration in the patient’s way of thinking, way of feeling, and relation to the world that Bleuler believed was unique to schizophrenia (McNally, 2016, pp. 24; 41f). Sigmund Freud also chimed in in the early 1900s, viewing psychosis as a

disturbed connection to reality, and viewing schizophrenia as a combination of this and megalomania (McNally, 2016, p. 45; Moskowitz et al., 2019, p. 10).

While much confusion and discussion about the nature of psychosis and schizophrenia continued in the 20th century, this understanding of psychosis as a disturbed contact to reality persisted and was seen in diagnostic manuals in both the US and internationally by the 1980s (Bürgy, 2008, p. 1203; Moskowitz et al., 2019, p. 10f). What also persisted was the view that schizophrenia was a brain disease, and moreover, one that was genetically caused. Despite stress already being proposed as a contributing factor in the 1960s (Rosenthal, 1966), the prevailing consensus in the psychiatric system well into the late 1900s remained that schizophrenia was primarily genetically caused.

It was at a time when schizophrenia was thus viewed as an entirely genetically and biologically caused brain disease that it was first found that some ethnic minorities had a higher risk of being diagnosed with schizophrenia, making it a controversial finding (Morgan et al., 2019, p. 250). With schizophrenia viewed like this, the finding seemed at first glance to have only two possible explanations: that ethnic minorities had a higher genetic risk of schizophrenia, or that the excess schizophrenia cases were not actual schizophrenia cases at all, but cases of misdiagnosis. The former explanation understandably caused strong reactions, as it fuelled racist arguments about the superiority of 'White' genes (Fernando, 1991, p. 143f). It also has rather strong arguments against it. Already in a classic study from 1932, Ødegaard (1932, pp. 70f; 99f) found that psychosis rates among Norwegian migrants in Minnesota were higher not just than rates in the general population of Minnesota, but also than rates in the general population of Norway. Since then, it has many times been found that psychosis risks for emigrants from the same place vary with the place they migrate to (Jongsma et al., 2021, p. 1914), and that psychosis rates within the same country vary with the area you look at (March et al., 2008).

Meanwhile, the other explanation has lived on for longer and is based on the argument that racism towards minorities is the cause of mis- or overdiagnosis of schizophrenia and psychosis, whether due to everyday racism from individual psychiatrists or from institutional racism built into psychiatric systems and their practices (Fernando, 1991, p. 143; Littlewood & Lipsedge, 1997, p. 104f; Metzl, 2010, p. 108). After all, if schizophrenia "is an illness that should occur in *1 percent* of any given population […] regardless of where they live, how they dress, who they know, or what type

of music they happen to prefer" (Metzl, 2010, p. xf), then surely, finding a higher rate in any population must be a sign of psychiatric discrimination against that population.

However, the case for environmental factors contributing to psychosis risk has been building over the past decades, and in our current understanding of psychosis and schizophrenia, it's not necessarily the case that all populations must have the same relative rates of psychosis or schizophrenia, as these are no longer considered purely genetically based. While there is some evidence of a certain heritable element in especially the schizophrenia spectrum, no genes have been found whose presence or absence guarantee the presence or absence of psychosis (Nordentoft & Vandborg, 2017, p. 308; Simonsen & Haahr, 2017, p. 332). Rather, the development of psychosis is thought to result from an interplay of heritable and environmental factors, including psychosocial ones, in a diathesis stress model (Nordentoft & Vandborg, 2017, p. 308f; Zwicker et al., 2018). Of course, this doesn't exclude the possibility that at least some of the raised psychosis risk among ethnic minorities is caused by mis- or overdiagnosis. A central part of the history of psychosis and schizophrenia is that, amid the confusion of what to define as psychosis, the schizophrenia diagnosis *has* been used in contexts that pathologised practices or traits that we would not today deem pathological, but which were frowned upon at the time. Examples are masturbation, LGBTQ+ identities, political dissension, and, indeed, being of a certain race (Fernando, 1991, pp. 120-122; McNally, 2016, pp. 131-134; 137-139; 141-143). However, the idea that the *entirety* of the raised psychosis rates can be explained by misdiagnosis ironically suffers from one of the same problems as the idea that the higher rates stem from 'inferior' genes; it bases its argument on the idea that schizophrenia is a biological disease alone. What the diathesis stress model implies, on the other hand, is that many other factors than genetic or biological ones might be especially affecting ethnic minorities.

Importantly, the inclusion of environmental factors does not exclude the possibility that racism is part of the explanation – in fact, it opens up the possibility of looking at other ways in which racism may drive stigmatised groups towards psychosis. Nor does it exclude the danger of arriving at conclusions that are just as racist as the idea of inferior genes. This was, for instance, demonstrated in the US in the 1960s and 1970s, where mainstream psychiatric research articles included ideas that participation in Black liberation movements and 'antiwhite' attitudes caused schizophrenia among Black men, while their experiences of racism and discrimination were framed as delusions (Metzl, 2010, pp. 100-102). In any explanation, therefore, it is important

to critically reflect on its theoretical and empirical basis, including any biases that may be hidden (or not so hidden) in it.

In summary, this topic is one with a history of controversy, and it's perhaps not surprising that there is a lack of consensus on the cause of certain ethnic minorities' higher psychosis risks when there has historically been such a lack of consensus on what psychosis even is and what causes it. Moreover, there is certainly a risk of arriving at prejudiced, insensitive, or overly simplistic conclusions about the causes of ethnic minority groups' higher psychosis risks – something that I, as a person who is *not* part of an ethnic minority, should perhaps attempt to be particularly conscious of. However, this makes it all the more important to properly review the existent explanatory hypotheses and models, so as to avoid resorting to interventions based on reductionist and prejudiced understandings.

# Part 2: The article

## Introduction

The purpose of this scoping review is to uncover explanations posited in the scientific literature for why ethnic minorities have a higher risk of developing psychotic symptoms and/or receiving a psychosis diagnosis. It is by now fairly well-established that rates of psychosis are higher among certain ethnic minority groups than in the general population, with the overall incidence rates reported to be about 1.5-3.0 times higher in these groups combined, but depending heavily on the minority group and the place (Morgan et al., 2019, p. 247). This excess of psychosis in certain groups is troubling, as some forms of psychosis and especially schizophrenia in the worst cases can lead to significantly reduced quality of life and even a life expectancy shortened by 15-20 years for the individual, as well as high treatment costs for society (Nordentoft & Vandborg, 2017, p. 312; Simonsen & Haahr, 2017, p. 325; Videbech et al., 2018, p. 79f). While these worst-case outcomes are certainly not the case for all psychosis patients, there is also some evidence that outcomes may on average be worse for some ethnic minorities. For instance, there seem to be lower recovery rates and more continuous courses of psychosis among Black Caribbean patients in the UK (Morgan et al., 2017, p. 89). However, in order to design interventions to combat this issue, we need to understand what causes it. This raises the question: How can we explain the raised psychosis risks among migrants and ethnic minorities?

Morgan et al. (2019) conducted a review trying to answer just that by examining evidence of the varying psychosis rates as well as different proposed explanations for them, including several environmental risk factors associated with both ethnic minority status and psychosis, such as living in urban areas, social fragmentation, and experiencing discrimination. They propose a socio-developmental model, wherein social adversity interacts with genetic risk factors and affects neurobiological development, causing a lasting raised risk of psychosis. This is especially the case if the social adversity in question involves interpersonal hostility and violence. According to this model, the reason why psychosis rates are higher among ethnic minorities and migrants is then that these groups are subjected to more social adversity during childhood and/or before and during migration (Morgan et al., 2019, p. 254f). However, as Morgan et al. point out themselves, this model still needs researching and testing, and crucially,

it is still unclear from this model *how* this social adversity becomes translated into psychosis risk. What are the mechanisms behind it? Morgan et al. (2019, pp. 253f; 256) found few studies on such mechanisms and only few rather limited attempts at explanations that mainly point to associations between different social factors and psychosis risk without attempting to explain where this association is coming from. This calls for further examination of the literature published since then, and a further focus on how this literature explains and conceptualises reasons for the association between the social adversity connected to ethnic minority status and psychosis risk.

This paper takes Morgan et al.'s review from 2019 as a starting point to dive further into the association between psychosocial factors connected to ethnic minority status and psychosis risk. I am attempting to uncover what mechanisms have been proposed to explain the associations of different psychosocial factors and the higher risk of psychosis among ethnic minorities since Morgan et al. wrote their article, as well as undertaking a critical discussion of the literature on this topic. By 'mechanisms', I mean models and hypotheses that posit causal explanations, i.e., not just correlations between different social risk factors and psychosis, but suggestions as to *why* and *how* these are connected. Thus, while I am in some ways extending the work of Morgan et al., I also have a different focus by concentrating more on the mechanisms behind psychosocial risk factors posited in the scientific literature, as well as critical examination of these.

To do this, I need to identify and map the different types of mechanisms proposed in the literature; a purpose for which the scoping review is particularly well suited (Arksey & O'Malley, 2005, pp. 20-22; Pollock et al., 2024, p. 4f). Like a systematic review, the scoping review seeks to systematically find all articles relevant to a research question. However, where the systematic review typically gathers and synthesises evidence on a relatively narrow question like the efficacy of a certain intervention, the scoping review lends itself to broader questions by providing an overview of the kinds of research being undertaken and/or the key factors and concepts relevant to a topic (Munn et al., 2018, p. 2). Thus, to get an overview of the explanations posited in the literature, a scoping review seems a fitting method. By using this method, I am also taking a different approach to the topic than for example Jongsma et al. (2021), who also reviewed this topic, but specifically from the angle of how different conceptualisations of ethnicity lead to different explanations for the high psychosis rates, and without an explicit systematic approach.

# Methods

In reporting the scoping review, I am following the Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR), a checklist to help ensure that the essential points for research transparency and rigour are reported in scoping reviews (Tricco et al., 2018). A copy of this checklist filled out with the relevant page numbers of this project is attached in appendix A. Part of what is asked for in PRISMA-ScR is to indicate whether a review protocol exists and is registered in advance of conducting the scoping review, which is recommended in order to secure transparency and rigour in research methods (Pollock et al., 2024, p. 6). As this is a student project not intended for publication, this was not feasible.

## *Search strategy*

The search for articles to include in the review was conducted using APA PsycNet and PubMed. These are databases for psychological and medical literature, respectively. I used two blocks of search terms: one covering synonyms for ethnic minorities and one covering psychosis synonyms. The search terms used are listed in table 1.

***Table 1. Search terms used***

| | |
|---|---|
| Block 1: migrants and ethnic minorities | Migrant* OR migrat* OR immigra* OR refugee* OR "ethnic minorit*" OR "minority ethnic*" OR "minoritised ethnic*" OR "minoritized ethnic*" OR "ethnic group*" OR "ethnic difference*" OR "ethnic disparit*" OR "racial minorit*" OR "racially minoritised" OR "racially minoritized" OR "racial group*" OR "racial difference*" OR "racial disparit*" |
| | AND |
| Block 2: psychotic disorders | Psychosis OR psychoses OR psychotic OR schizophreni* |

In addition, I used index and MeSH terms in order to find relevant articles that didn't include the other search terms in their title or abstract. These are listed in table 2. A search filter was used in both databases to show results from 2019 or later, in order to find literature published after Morgan et al.'s (2019) review. Before the final search, I consulted information specialists from Aalborg University Library in order to optimise the search string. The final search was conducted on the 11th of March 2025.

***Table 2. Index and MeSH terms used.***

| **Index terms used on PsycNet** | |
|---|---|
| Block 1: migrants and ethnic minorities | "Minority groups" OR "racial and ethnic groups" OR "racial and ethnic differences" |
| Block 2: psychotic disorders | Psychosis OR schizophrenia |
| **MeSH terms used on PubMed** | |
| Block 1: migrants and ethnic minorities | "Ethnic and racial minorities" OR "racial groups" |
| Block 2: psychotic disorders | "Schizophrenia spectrum and other psychotic disorders" |

## *Selection of literature*

Search results were screened for relevance in two phases, title/abstract screening and full-text screening, using the online review tool Rayyan. Ideally, screening should be completed by multiple reviewers in order to minimise error and bias in selection of sources (Petticrew & Roberts, 2006, p. 119f). As I worked alone, this was not possible. However, I conducted the title/abstract screening of all articles twice, 'blinding' myself to the results of the first screening when conducting the second one, and with about a week between the two different screenings of each result. The rationale behind this was that I would not be able to remember my decisions about hundreds of titles and abstracts a week later. I could therefore make a relatively independent new screening before resolving any disagreements between my first and second screening, thus hopefully minimising the effects of any errors made. This was not feasible for the full-text screening, as the smaller amount of literature and more thorough screening made it easier to remember my decisions, and full-text screening was therefore only conducted once. The eligibility criteria used to select articles were as follows:

- Articles must posit at least one mechanism to explain the connection between one or more psychosocial risk factors and raised psychosis risks in one or more ethnic minority groups – i.e., an explanation for *how* said psychosocial risk factor(s) would raise the risk of psychosis.
- Providing argumentation for such a mechanism must be an aim of the article.
- The posited mechanism(s) must not simply explain the raised psychosis risk as part of general worsened mental health among ethnic minorities but must explain why psychosis risk specifically is higher.
- Articles must be written in English.
- Articles must be peer-reviewed.
- Articles must be published in 2019 or later.
- Articles must not already be included in Morgan et al.'s (2019) review.

These criteria were chosen in order to find mechanisms proposed in contemporary scientific literature of a certain standard, while avoiding repetition of work already done by Morgan et al. By saying that argumentation for said mechanism must be an aim of the article, I mean that it is not enough to e.g. mention in passing some possible mechanisms that have been suggested by others or to test whether a psychosocial factor is associated with psychosis risk and then speculate on why this could be. Rather, articles must set out to e.g., test a hypothesised mechanism empirically, review literature on (at least among other things) mechanisms, or propose a mechanism based on empirical or theoretical reasoning. While mapping every possible mechanism even mentioned in the scientific literature would be an interesting project, it would be too large of an undertaking for one person as part of a student project.

### *Analysis approach*

An extraction form was used for all articles to give an overview of their characteristics and qualities of interest besides mechanisms. This included data on authors, year of publication, title, study methods (shortened into a few words like "qualitative interview study"), ethnic minority groups under study, psychosocial risk factors involved in the proposed mechanisms, directly mentioned implications for intervention to reduce psychosis risk for ethnic minorities (if any), conceptualisations of psychosis, and conceptualisations of ethnicity and/or race. Collecting most of these data involved an element of analysis and interpretation in order to pick out the most important

characteristics of e.g. the study methods or mechanisms described. Especially the conceptualisations of psychosis and ethnicity/race often had to be inferred from the ways these were operationalised in studies, as few studies directly defined the terms. The data extraction form can be found in appendix B.

To extract and analyse the mechanisms presented by the included articles, I used an approach inspired by Thomas and Harden's (2008) thematic synthesis, developed for the synthesis of qualitative research in systematic reviews. Of course, there is nothing in my eligibility criteria indicating that included research must be qualitative only. However, as my focus is on mapping the mechanisms included in the articles rather than on synthesising the data they are based on, it made sense to mainly analyse text passages describing mechanisms and the reasoning behind them. Thus, even if an article in itself was about a quantitative research project, the 'data' I analysed from it was qualitative.

In the first stage, parallel with extracting the data for the extraction form, I identified passages in each article describing mechanisms and coded these, using the coding software NVivo 15, in a way that summarised the mechanisms without attempting to go beyond the written words or make interpretations. This differs somewhat from Thomas and Harden's line-by-line coding of the entire material, as I was trying to characterise specifically the mechanisms presented rather than the entire evidence base. In the second stage, I identified categories that Thomas and Harden might call 'descriptive themes': higher-order codes under which the initial codes can be categorised, but which still stay close to the original data under analysis and describe rather than interpret. Once again differing from Thomas and Harden, I didn't attempt to develop a third level of codes called 'analytical themes', whose aim usually is to go beyond what the original articles infer from their data to make your own interpretations and inferences from the descriptive themes developed in the second phase (Thomas & Harden, 2008, pp. 4-7). The reason I didn't do this is that Thomas and Harden's approach is one aimed at qualitative systematic reviews rather than scoping reviews; they are attempting to *synthesise* qualitative evidence, while I attempted to *map and categorise* explanations.

# Results

The search yielded 1561 results, reduced to 1096 after removing 465 duplicates. 982 results were excluded during title/abstract screening, leaving 114 articles for full-text screening, of which I was unable to procure one (namely, Plowden, 2019). After screening the rest according to the eligibility criteria described above, 35 articles were included in the analysis. These are listed in table 3 along with their methods, the ethnic minority groups whose heightened psychosis risks are being studied or discussed in the articles, and the psychosocial risk factors that are involved in the mechanisms presented in the articles. Other collected data such as implications for intervention and conceptualisations of race or ethnicity and psychosis are presented in appendix C and are discussed either in the closing framework or my exam presentation. It should be noted that the risk factors in table 3 are not an exhaustive list of all psychosis risk factors mentioned in the included articles, but a list of psychosocial risk factors that the included articles incorporated into mechanisms describing *how* they cause psychosis risks.

**Figure 1. PRISMA Flowchart**

*Source of template: Page et al. (2021).*

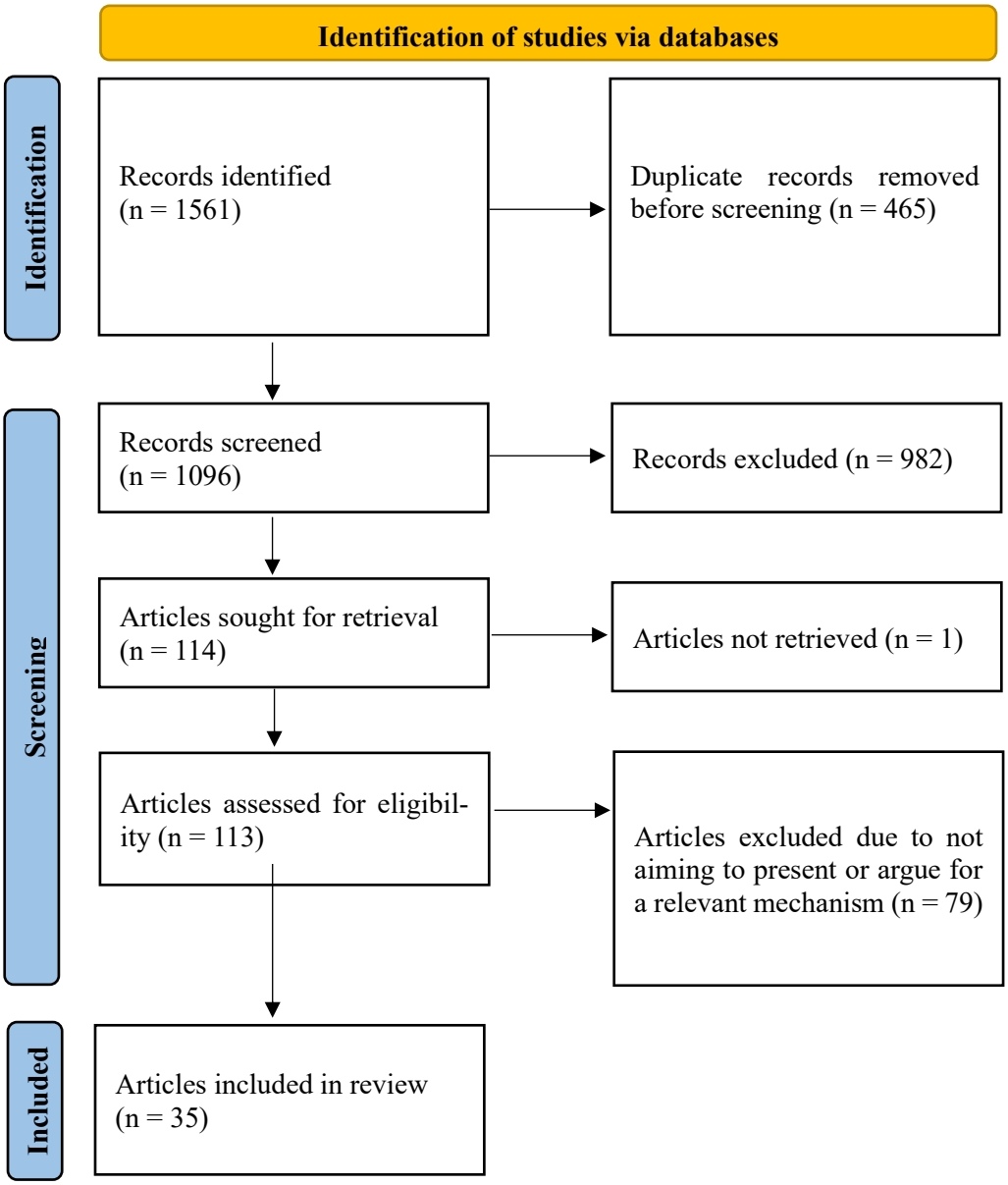

**Table 3. Overview of included articles**

| # | Authors and year of publication | Title | Study methods | Ethnic minority group(s) | Psychosocial psychosis risk factor(s) involved in proposed mechanism(s) |
|---|---|---|---|---|---|
| 1 | Alherz, Almusawi, & Alsayegh (2022) | Diglossia Correlates With Prodromal Symptoms of Psychosis Among First-Generation Migrants | Questionnaire study | First-generation migrants in English-majority speaking OECD countries | Diglossia and migration |
| 2 | Alherz, Almusawi, & Barry (2019) | Diglossia in the Etiology of Schizophrenia: A Hypothesis | Theoretical argument | Migrants and minorities experiencing diglossia | Diglossia and migration |
| 3 | Andersen, Al-Shawaf, & Bearden (2021) | Positive schizotypy predicts migration intentions and desires | Literature review and questionnaire study | Migrants (though study participants are *non*-migrant Americans) | Migration (or schizotypy as explaining migration) |
| 4 | Anglin (2023) | Racism and Social Determinants of Psychosis | Literature review | Non-White (especially Latinx and Black) Americans | Structural racism and its consequences, including discrimination, disadvantaged neighbourhoods, and segregation |
| 5 | Anglin et al. (2021) | From Womb to Neighborhood: A Racial Analysis of Social Determinants of Psychosis in the United States | Narrative review | Racial and ethnic minorities in the US | Structural racism including segregation, discrimination, stressful racial dynamics, and trauma from e.g. police victimisation |

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| 6 | Anglin & Lui (2023) | Racial microaggressions and major discriminatory events explain ethnoracial differences in psychotic experiences | Questionnaire study | Black/African Americans and first- or second-generation immigrants in the Northeastern US | Racial microaggressions |
| 7 | Bhui, Halvorsrud, Mooney, & Hosang (2021) | Is psychosis a syndemic manifestation of historical and contemporary adversity? Findings from UK Biobank | Statistical analysis of data from the UK Biobank cohort | Ethnic minorities in the UK | Childhood and current adversity, especially feeling hated in childhood, sexual assault, and low household income |
| 8 | Cai, Wang, Yan, Conwell & Temkin-Greener (2022)[1] | The diagnosis of schizophrenia among nursing home residents with ADRD: Does race matter? | Statistical analysis of data from American nursing homes | Black nursing home residents with Alzheimer's Disease and Related Dementias (ADRD) in the US | Structural and institutional racism |
| 9 | Caroppo, Lanzotti, & Janiri (2020) | Psychopathology in refugees subjected to the Dublin Regulation: an Italian study | Semi-structured interview study | Refugees and asylum seekers in Italy | Migration and asylum-seeking processes |
| 10 | Elahi et al. (2022) | Symptoms of Paranoia Experienced by Students of Pakistani Heritage in England | Questionnaire study | People of Pakistani heritage born in England | Discrimination |

[1] Cai et al. (2022) was analysed using the authors' manuscript, as I didn't have access to the published version. Page numbers in later references refer to the authors' manuscript.

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| 11 | Faber, Roy, Michaels, & Williams (2023) | The weaponization of medicine: Early psychosis in the Black community and the need for racially informed mental healthcare | Literature review | Black Americans and Canadians | Clinician racial bias |
| 12 | Fashaw-Walters, McCreedy, Bynum, Thomas, & Shireman (2021) | Disproportionate increases in schizophrenia diagnoses among Black nursing home residents with ADRD | Statistical analysis of data from American nursing homes | Black nursing home residents with ADRD in the US | Structural and institutional racism |
| 13 | Gara et al. (2019) | A Naturalistic Study of Racial Disparities in Diagnoses at an Outpatient Behavioral Health Clinic | Statistical analysis of medical records | African Americans in New Jersey, US | Clinician racial bias |
| 14 | Henssler et al. (2020) | Migration and schizophrenia: meta-analysis and explanatory framework | Systematic review, meta-analysis, and theoretical argument | Migrants | Social defeat and -exclusion, discrimination, and low ethnic density |
| 15 | Hunter et al. (2021) | Black American Maternal Prenatal Choline, Offspring Gestational Age at Birth, and Developmental Predisposition to Mental Illness | Comparison of maternal choline levels | Black Americans | Stress from systemic racism and discrimination |

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| 16 | Huque et al. (2024) | Mechanisms mediating ethnoracial discrimination and suspiciousness in Asian, Black, and Hispanic United States college students | Questionnaire study | Asian, Black, and Hispanic Americans | Discrimination |
| 17 | Jegarl et al. (2023) | Psychotic Misdiagnosis of Racially Minoritized Patients: A Case-Based Ethics, Equity, and Educational Exploration | Case study and expert discussion | Racially minoritised people in the US, with a Togolese immigrant as a case example | Clinician racial bias |
| 18 | Jeste et al. (2023) | Review of Major Social Determinants of Health in Schizophrenia-Spectrum Psychotic Disorders: III. Biology | Literature review | Black and Latinx Americans, migrants and refugees | Racism, marginalisation, and migration-related stressors |
| 19 | Jongsma, Karlsen, Kirkbride, & Jones (2021) | Understanding the excess psychosis risk in ethnic minorities: the impact of structure and identity | Theoretical argument and literature review | Ethnic minority groups in Western countries | Stereotyping, discrimination, and socioeconomic disadvantage |
| 20 | Kircher, Krautheim, & Straube (2020) | "Automatic outgroup categorisation" and limbic brain activation: A mechanism underlying psychosis risk in migrants and city dwellers | Theoretical argument | Migrants and ethnic minority members | Urban living, low ethnic density, and frequent meetings with people perceived as outgroup members |
| 21 | Knight, Yang & Jarvis (2024) | "Dem sey mi mad": a scoping review of the attitudes and beliefs of English-speaking Afro-Caribbeans about psychosis | Scoping review | English-speaking Afro-Caribbeans | Racism, discrimination, and clinician misunderstandings resulting from these and cultural differences |

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| 22 | Ku et al. (2023) | Associations between childhood ethnoracial minority density, cortical thickness, and social engagement among minority youth at clinical high-risk for psychosis | Comparison of brain scans | Primarily Hispanic, Asian, and Black Americans | Low ethnic density and its consequences like discrimination, less robust social networks, and feelings of non-belonging and being different |
| 23 | Lazaridou et al. (2023) | Racism and psychosis: an umbrella review and qualitative analysis of the mental health consequences of racism | Umbrella review and qualitative interview study | African migrants in Germany | Racism |
| 24 | Lincoln, Johnson, Laquidara, Wilt, & Obeid (2022) | Increased rates of social defeat and schizotypy in racial minorities | Questionnaire study | Racial/ethnic minorities (implied in the US, but never specified) | Social defeat caused by e.g. discrimination |
| 25 | McIntyre, Elahi, Barlow, White, & Bentall (2021) | The relationship between ingroup identity and Paranoid ideation among people from African and African Caribbean backgrounds | Questionnaire study | People from African and African Caribbean backgrounds in the UK (but also argues its relevance for migrants and refugees) | Racism and discrimination |
| 26 | Nazroo, Bhui, & Rhodes (2020) | Where next for understanding race/ethnic inequalities in severe mental illness? Structural, interpersonal and institutional racism | Theoretical argument | Ethnic minorities in the UK, mainly Black Caribbean and Black African groups | Everyday, institutional, and structural racism |

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| 27 | Pozza (2019) | The role of aberrant salience and alexithymia in psychotic experiences of non-treatment-seeking adolescent immigrants compared with natives | Questionnaire study | Immigrants in Italy | Immigrant status |
| 28 | Rabin & Palaniyappan (2023) | Brain health in ethnically minority youth at risk for psychosis | Comment on Ku et al. (2023) | Primarily Hispanic, Asian, and Black Americans | Low ethnic density and lack of positive social engagement |
| 29 | Saxena, Liu, Handley, & Dodell-Feder (2024) | Social victimization, default mode network connectivity, and psychotic-like experiences in adolescents | Comparison of brain scans and questionnaire study | Black and Hispanic Americans | Chronic social victimisation including racism from peers |
| 30 | Schwartz et al. (2019) | Exploring the Racial Diagnostic Bias of Schizophrenia Using Behavioral and Clinical-Based Measures | Comparison of symptom ratings, diagnoses, and speech samples | African Americans | Clinician racial bias |
| 31 | Simmons, Arbabi, Felsky, Wainberg & Tripathy (2024) | Reported race-associated differences in control and schizophrenia post-mortem brain transcriptomes implicate stress-related and neuroimmune pathways | Differential gene expression analyses of postmortem brain tissue | Black Americans | Social stressors experienced by Black Americans |
| 32 | Tobon et al. (2021) | Racial Implicit Associations in Psychiatric Diagnosis, Treatment, and Compliance Expectations | Survey study | Black Americans | Clinician racial bias |

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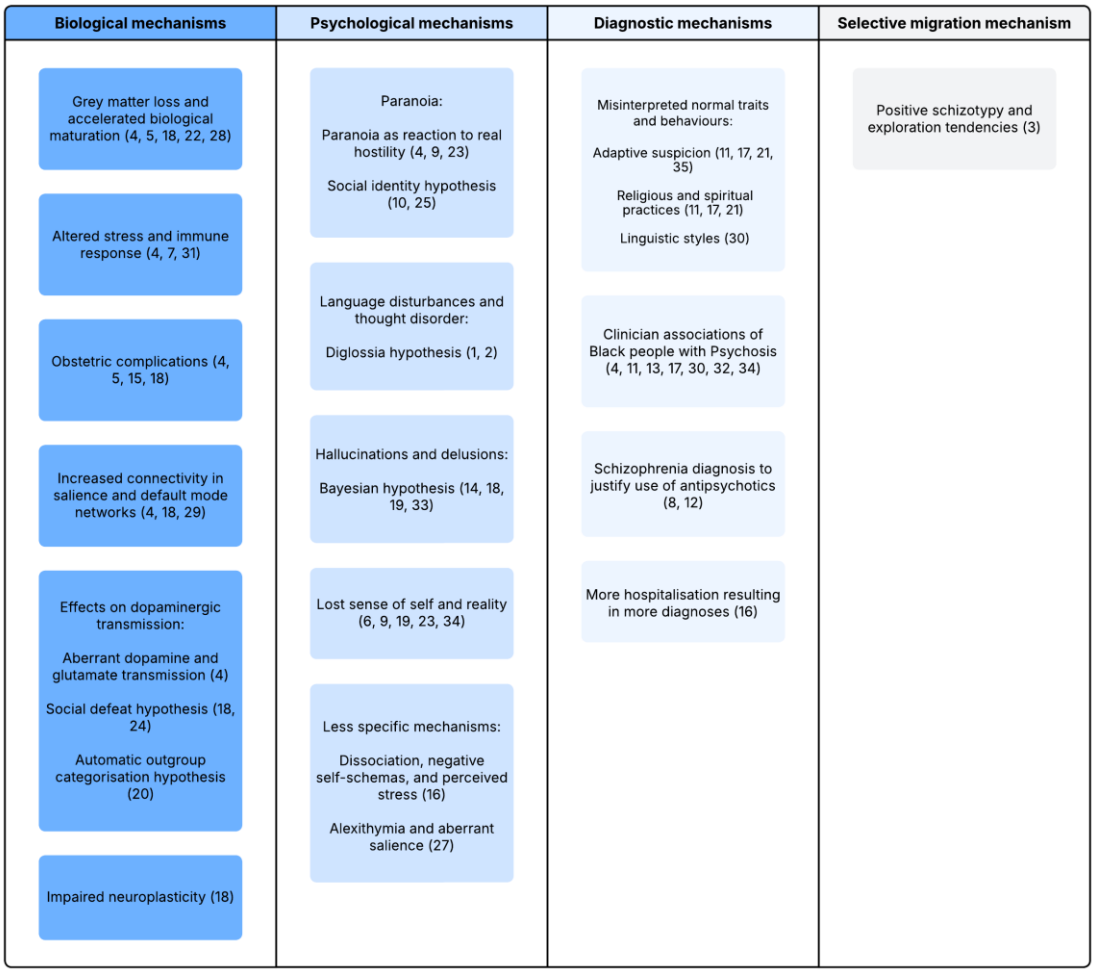
| 33 | Varchmin, Montag, Treusch, Kaminski & Heinz (2021) | Traumatic Events, Social Adversity and Discrimination as Risk Factors for Psychosis – An Umbrella Review | Umbrella review of meta-analyses | Migrants, refugees, and Black people | Trauma, social adversity, and discrimination |
| 34 | Wallerstein (2020) | Hunting the Real: Psychosis and Race in the American Hospital | Theoretical argument and case studies | Black Americans | Historical and current racism and its erasure in societal conversation and consciousness |
| 35 | Wolny et al. (2023) | Race and self-reported paranoia: Increased item endorsement on subscales of the SPQ | Investigation of item and subscale functioning in the Schizotypal Personality Questionnaire (SPQ) | Black/African Americans | Racism and lack of understanding of its consequences in clinical contexts and development of assessment tools |

As can be seen in table 3, study methodology varies greatly. The most used method is quantitative questionnaire or survey studies, which eleven articles use (one of which also analyses speech samples), followed by ten review studies, six articles presenting (without testing) theoretical arguments, four articles doing statistical analyses of data collected by others, four articles comparing biological measures of participants, three articles presenting case studies, two interview studies, one expert discussion, one comment on the results of another study, and one investigation of item- and subscale-level functioning of a screening tool. Thus, methods of the included articles vary but are mostly quantitative and/or review methods. Some studies use multiple methods, which is why the numbers above add up to more than 35. It can also be seen that a majority (30 out of 35) of the included articles explain the high psychosis risks as a consequence of racism, discrimination and/or stereotyping; however, it varies whether the racism in question is on a structural, institutional, and/or interpersonal level, as well as *how* racism is thought to cause psychosis risks.

In coding the proposed mechanisms, four descriptive themes arose: primarily biological mechanisms, primarily psychological mechanisms, mechanisms relating to diagnostic processes, and selective migration mechanisms, each with one to six subcategories of mechanisms contained within them. These are presented in the following, and an overview is depicted in figure 2. As with risk factors in table 3, some articles mention more mechanisms than they are depicted as mentioning in figure 2. This is because I have only depicted mechanisms that articles actually present an argument for and suggest as plausible; if a mechanism is only mentioned in an article but not described, or is mentioned as an unlikely one, this is not shown in figure 2. It should also be noted that since I have included literature reviews, some research studies can be represented multiple times, and the number of articles presenting a mechanism therefore doesn't necessarily reflect the number of research studies conducted on said mechanism during the period included in the search.

**Figure 2. Overview of mechanisms**

*The numbers in parentheses refer to the articles that present the mechanisms, represented by their number in table 3.*




## *Biological mechanisms*

11 articles present mechanisms that explain psychosis risks in terms of biology. These are mainly based on studies of minority groups in the US, except for one article focusing on the UK (Bhui et al., 2021) and one theoretical argument based on migrants and ethnic minorities in general (Kircher et al. 2020). Overall, these mechanisms point to how the various social stressors experienced by ethnic and racial minority groups cause physiological changes in the body.

Ku et al. (2023, p. 1712f) present the hypothesis that ethnic minority youth living in areas with low ethnic density (i.e., a low percentage of residents from ethnic minority groups) may experience greater social stress, resulting in grey matter loss in social brain regions such as the fusiform gyrus and right insula – an effect that may be

buffered by greater social engagement. In a response to this article, Rabin & Palaniyappan (2023, p. 1701f) hypothesise that this may also be a sign of social stress causing accelerated biological maturation, which generally results in reduced brain volume and increased grey matter density; a mechanism also suggested by others to be a psychosis risk resulting from traumatic stressful events and low socioeconomic status (Anglin, 2023, p. 290; Anglin et al., 2021, p. 604) as well as general experiences of adversity like marginalisation and racism (Jeste et al., 2023, p. 869).

Four articles point to how chronic social stressors from discrimination and unfavourable socioeconomic and geographical circumstances experienced by minorities may alter the body's stress and immune response. This is suggested to happen via epigenetic changes in the expression of certain gene sets (Simmons et al., 2024, p. 8), by blunting the neurological stress response to new stressors and impeding recovery from stress, and by causing a chronic inflammatory response (Anglin, 2023, p. 289; Bhui et al., 2021, p. 686; Jeste et al., 2023, p. 870). According to four articles, chronic stressors may even have effects across generations, as the stress of social disadvantage and discrimination in pregnant minority individuals may cause obstetric complications and high stress biomarkers including altered choline and cortisol levels, which are associated with psychosis risk in children of affected individuals (Anglin, 2023, p. 290f; Anglin et al., 2021, p. 603; Hunter et al., 2021, p. 897; Jeste et al., 2023, p. 869f).

Increased connectivity in the brain's salience and default mode networks, implicated in "the detection and integration of emotional and sensory information" and "a variety of self-directed, social, and stimulus-independent processes", respectively (Saxena et al., 2024, p. 463), has also been posited by three articles as a mechanism by which discrimination causes higher psychosis risk (Anglin, 2023, p. 289f; Jeste et al., 2023, p. 871f; Saxena et al., 2024, p. 463).

Four articles mention effects on dopaminergic transmission as a mechanism. While Anglin (2023, p. 288f) mentions aberrant dopamine and glutamate transmission in general as effects caused by chronic stress from e.g. discrimination, others emphasise the effects on the mesolimbic dopamine system. Described in Jeste et al. (2023, p. 874)'s review and tested by Lincoln et al. (2022, p. 1f), the social defeat hypothesis of psychosis suggests that chronic experiences of social exclusion sensitise the mesolimbic dopamine system, leading to a higher risk of psychotic disorders. This social exclusion can come from racism, discrimination, and other factors making you feel like an outsider, making it relevant to ethnic minority groups. Kircher et al. (2020, p.

541f), meanwhile, hypothesise that not even social defeat, but simply *meeting* a lot of people that you perceive as having characteristics that set them significantly apart from yourself will result in dysactivation of the ventral anterior cingulate cortex, which is heavily connected to the mesolimbic dopamine system. They call this hypothesis the automatic outgroup categorisation hypothesis. According to them, this helps explain the higher psychosis risk of both migrants and ethnic minorities living in areas with low ethnic density.

Finally, a mechanism by which more structural aspects of racism may be implicated in psychosis is presented by one article, namely that the resulting "poor education, impoverished environments, limited access to care and recovery programs, and comorbidities" may lead to impaired neuroplasticity (Jeste et al., 2023, p. 873).

## *Psychological mechanisms*

16 articles present mechanisms arguing that some of the psychological consequences of migration or being part of a minority group may lead to higher psychosis risks. Some of these mechanisms also contain a biological element, but I have put them in this category rather than the biological one because their primary focus is on the psychological aspects. These mechanisms are based on studies spanning more broadly in the minority groups they focus on, including a wide range of non-White and migrant groups in both the US and Europe. They also vary quite a lot in their approach to the question, especially in terms of what psychosis symptoms they focus on, which is why I have further divided this category into subcategories based on this.

### Paranoia

Three articles argue that paranoia risk may result from constant meetings with hostility and discrimination from others (Anglin, 2023, p. 289; Lazaridou et al., 2023, p. 1017) and from the threatening environment of migration and asylum-seeking processes (Caroppo et al., 2020, p. 81). In reaction to these threats, a person belonging to a minority group may develop expectations of hostility from others as a self-defence from actual hostility, potentially resulting in clinical paranoia if it goes overboard.

Two articles posit an explanation of paranoia based on social identity theory. McIntyre et al. (2019, p. 19f) argue that people gain feelings of self-worth and personal control from groups they identify with, but only if contact with these groups is positive.

If contact with the ingroup is hostile, it instead becomes a source of distress, and feelings of self-worth and personal control are reduced, leading to a risk of paranoid ideation. Therefore, they argue that identifying with the majority culture but being discriminated against by that same majority group leads to higher risk of paranoia via negative effects on self-worth and personal control. In a test of parts of this hypothesis, Elahi et al. (2022, p. 683f) likewise argue that while identification with a minority culture rather than the majority culture may be a risk factor of paranoia in itself, it also may protect against the effects of discrimination from the majority group.

**Language disturbances and thought disorder**

An explanation less directly tied to acts of discrimination and hostility is found in two articles about diglossia. This is a term for situations where someone speaks two languages (or variants of the same language) and one, the H language, is perceived as having higher social standing than the other, the L language, which is often the 'mother tongue' spoken in the home (Alherz et al., 2019, p. 988). This may lead to higher risk of schizophrenia via problems with language lateralisation in the brain and with developing language skills in children, leading to language changes and thought disorder, as well as via paths made clearer by viewing the languages involved as a way to navigate social hierarchies: The higher status but relative unfamiliarity of the H language compared to the L language may be experienced as the H language influencing thoughts from the outside, and navigation of social hierarchies is connected to dopamine transmission (Alherz et al., 2019, p. 988f; 2022, p. 8).

**Hallucinations and delusions**

Four articles present an explanation from an approach inspired by Bayesian statistics, where psychosis is the result of predictive processing gone wrong. In this understanding, everyone automatically makes predictions about what will happen based on prior knowledge. When this prior knowledge turns out to be inaccurate and prediction errors occur, the prior knowledge is updated. However, if more prediction errors occur due to faulty prior knowledge from e.g. trauma or misunderstandings caused by linguistic or cultural barriers, or if these prediction errors occur in particularly hostile or ambiguous environments where it's harder to assess the size and importance of the prediction error, dopaminergic transmission related to this processing may increase. Salience is

then attributed to otherwise irrelevant stimuli and more false inferences are made, manifesting as delusions and/or hallucinations (Henssler et al., 2020, pp. 326; 331f; Jeste et al., 2023, p. 873; Jongsma et al., 2021, p. 1917f; Varchmin et al., 2021, p. 1).

**Lost sense of self and reality**

Five articles focus on factors that can lead to insecurity about, questioning of, or even loss of the self, as well as questioning your own perceptions and reality sense. Jongsma et al. (2021, p. 1917) suggest that discrimination and stereotyping lead to feelings of disempowerment, in turn leading to a disrupted sense of self and an external locus of control, heightening psychosis risk. These effects are possibly also caused by disempowerment feelings causing a chronic 'flight or fight' response, which is adaptive in the short term but damaging to the brain and body when kept active in the long term. Anglin and Lui (2023, p. 6) examine the effects of racial microaggressions, i.e., smaller everyday acts of racist hostility where it is unclear to the victim whether the hostility should be attributed to racism. They argue that because of this ambiguity, the victims need to use more cognitive resources to process the experiences, ultimately creating insecurity in their own perceptions and sensibilities. Lazaridou et al. (2023, p. 1016f) likewise argue that a sense of differentness and negative self-awareness caused by experiencing racism may lead to questioning the self and aspects of reality. Caroppo et al. (2020, p. 77f) describe the connection between psychosis and asylum-seeking processes, especially the limbo space of the 'Dublin phase', where an asylum application may be suspended while it's decided which member state of the EU is responsible for handling the claim. They argue that this leaves the affected asylum seekers suspended without a clear defining context or legal status, having lost their migratory project as well as their social and cultural meanings and connections. This alienation, in turn, causes problems with self-consciousness and personal identity that may, in the worst cases, lead to psychotic dissociation as well as errors of logic inference and failures in reality testing, also raising the risk of paranoid symptoms (Caroppo et al., 2020, p. 81). Lastly, Wallerstein (2020, p. 260) approaches the question from a psychoanalytic angle, conceptualising psychosis as a way of making sense of aspects of reality that have been 'erased' from societal conversation. In this case, it is the historical and current racially motivated violence against Black people in the US that is denied and erased from public consciousness, leading Black people still affected by it to lose their sense

of what constitutes themselves and reality, driving them to express and make sense of the 'forgotten horror' in destructiveness, delusions and hallucinations (Wallerstein, 2020, pp. 262f; 265-267).

**Less specific mechanisms**

Finally, two articles present mediating psychological factors as mechanisms for psychosis risk but don't elaborate further on why these factors are tied to psychosis, or why they're tied to minority status, respectively. One argues that discrimination leads to dissociation, negative self-schemas, and perceived stress, which in turn lead to psychosis (Huque et al., 2024, p. 62f). The other finds an association between immigrant status and alexithymia (problems with emotional awareness) and aberrant salience (feelings of increased significance of irrelevant stimuli), which in turn are associated with psychotic experiences (Pozza, 2019, pp. 2057-2059).

### *Mechanisms relating to diagnostic processes*

11 articles present mechanisms that would mean that the higher rates of psychotic diagnoses among ethnic minorities are not (or at least not solely) a reflection of actual higher psychosis risk, but (also) of institutional practices making minority groups more likely to receive psychotic diagnoses, regardless of their actual symptoms. These explanations mainly focus on Black people of African or Caribbean descent in the UK, USA, and Canada.

Five of these articles present ways that clinicians belonging to White majority populations may misinterpret normal practices and behaviours due to cultural and experiential differences. For example, continual experiences of racism may cause people from minority groups to develop a level of adaptive mistrust towards other people, not because they are pathologically paranoid, but because people often *are* hostile towards them. A clinician without the same experiences, however, may interpret this as clinical paranoia (Faber et al., 2023, p. 5; Jegarl et al., 2023, p. 31; Knight et al., 2024, p. 8). This may even be reflected in the construction of diagnostic assessment tools, as Wolny et al. (2023, p. 35) show that Black Americans tend to score higher on measures of paranoia on the Schizotypal Personality Questionnaire (SPQ) even when adjusting for overall schizotypal traits. Further, spiritual and religious practices that are considered normative in minority communities may be misinterpreted as religious delusions

(Faber et al., 2023, p. 5; Jegarl et al., 2023, p. 31; Knight et al., 2024, p. 10). Even linguistic styles that differ from the clinician's own may be misinterpreted as disorganised speech, a sign of psychosis (Schwartz et al., 2019, p. 264).

In cases where there *is* psychopathology present, just not necessarily of a psychotic nature, seven articles argue that especially White clinicians tend to associate psychosis with Black people and mood disorders with White people (Gara et al., 2019, p. 130f; Jegarl et al., 2023, p. 31; Schwartz et al., 2019, p. 264; Tobon et al., 2021, p. 24; Wallerstein et al., 2020, p. 257f). According to Faber et al. (2023, p. 4), this is partially due to humans feeling less empathy for people of a different skin colour. This results in both an underemphasis on affective symptoms and an overemphasis on psychotic symptoms found in Black people (Anglin, 2023, p. 282f; Gara et al., 2019, p. 130f; Jegarl et al., 2023, p. 31; Wallerstein, 2020, p. 257f). According to Jegarl et al. (2023, pp. 31; 34), this bias also results in mislabelling trauma reactions as psychotic, mislabelling other symptoms as a result of substance-induced psychosis the moment substance use is involved, and even in mislabelling actual experiences of victimisation as delusional.

A more 'local' mechanism is presented by Cai et al. (2022) and Fashaw-Walters et al. (2021), who argue that rates of schizophrenia diagnoses among Black patients with Alzheimer's Disease and Related Dementias (ADRD) in US nursing homes have risen as an unintended consequence of an initiative by Centers for Medicare and Medicaid Services (CMS) in 2012. This initiative required that use of antipsychotics in nursing homes must be publicly reported unless the patients in question are diagnosed with schizophrenia, in order to reduce the use of antipsychotics for behavioural issues related to ADRD. The authors argue that higher likelihood of behavioural issues and lower quality of care provided to Black patients with ADRD may have led to lower quality of diagnosis and an overuse of the schizophrenia diagnosis to avoid having to report using antipsychotics for behavioural issues in this population.

Finally, Nazroo et al. (2020, p. 269f) present a hypothesis that because of institutional racism in criminal justice, social work, and healthcare systems, Black people in the UK are more likely than other groups to be admitted to psychiatric institutions and actually receive a diagnosis for any psychotic disorders they may have. In contrast to others in this category, this hypothesis doesn't necessarily imply misdiagnosis as much as actual psychotic disorders being more likely to be diagnosed in minorities.

### *The selective migration hypothesis*

Only one article (Andersen et al., 2021) presents a mechanism built on the idea of selective migration, i.e., that individuals with a higher psychosis risk are more likely to migrate, thus contributing to the higher psychosis risk among especially migrants (though this is also mentioned briefly by Jongsma et al., 2021, p. 1915, as a hypothesis that is poorly supported). The argument is that individuals with positive schizotypal traits (i.e., unusual experiences, magical thinking, and ideas of reference) have tendencies towards exploration, making them more likely to want to migrate (Andersen et al., 2021, pp. 1-3).

## Discussion

This review identified 35 articles presenting mechanisms for the connection between psychosocial risk factors and higher psychosis rates among ethnic minority groups. These can be categorised into biological mechanisms, psychological mechanisms, diagnostic mechanisms, and one selective migration mechanism. However, this does not mean that mechanisms are anywhere close to identical to other mechanisms in the same category. Especially the psychological mechanisms vary widely, perhaps partially due to variations in what psychosis symptoms they are focusing on, and partially due to their very different theoretical standpoints. It's not surprising that someone approaching the problem from a standpoint based in Bayesian statistics reaches different conclusions than Wallerstein (2020) approaching it from a psychoanalytical angle. What is perhaps also not downright surprising, but very interesting, is that the included articles seemingly also reach very different conclusions when focusing on delusions than when focusing on language disturbances and thought disorder, despite both supposedly being symptoms of the same spectrum of psychotic disorders. Granted, it is not unlikely that there would be different mechanisms behind symptoms of e.g. affective psychoses and schizophreniform psychoses, but delusions and thought disorder are both considered symptoms of schizophrenia (World Health Organization, 1994, p. 66). Are the different mechanisms proposed for different symptoms then signs that they are mechanisms for different psychotic disorders, signs that our diagnostic categories like schizophrenia actually cover several entirely different disorders, or simply

signs that only some of the mechanisms are correct? Further research on both the proposed mechanisms and on which psychotic disorders and symptoms we are actually trying to explain seems warranted.

An interesting finding when comparing this review to that of Morgan et al. (2019) is that using systematic methods, different mechanisms in general and especially those relating to misdiagnosis take up vastly more space in this review than Morgan et al.'s. Of course, this can have several different causes. Firstly, I am looking at literature specifically published from 2019 and forward, meaning that some of the different balance could be caused by a shift in the relative number of articles being published on the topic. Secondly, I am only focusing on mechanisms and not general risk factors, meaning that the relative balance shift may be caused by it simply being easier to explain mechanisms of misdiagnosis than mechanisms of actual psychosis. But thirdly, it could also be a sign that we should perhaps not dismiss the possible effect of misdiagnosis so easily – not to say that Morgan et al. (2019, p. 250) dismiss it entirely, but it is suggested to not necessarily be a big contributor to the higher psychosis risks in minority groups. Of course, it is important to keep in mind that the current review is a scoping review, not a systematic synthesis of evidence: I cannot say how strong the evidence in favour of misdiagnosis is, only that it is by no means a dead topic in the literature on reasons for the high psychosis risks, especially when it comes to Black minority groups in the US, UK, and Canada. For a more in-depth discussion of the individual categories of mechanisms and how they might work together, see the closing framework.

### *Limitations of this review*

A central limitation of this review is that I have conducted it on my own, something that Pollock et al. (2024, p. 2) argue cannot be done, since a scoping review should ideally be done by a team of experts on both the content of the review, scoping methods, and information science. Especially the reliability of the review is called into question, as I am the only person who has screened articles, extracted data, and coded. Another reviewer may have made different interpretations of articles or noticed things that I missed. As described under methods, I attempted to compensate for some of this during the title/abstract screening by screening articles twice with enough time between screenings to have forgotten how I sorted articles in the first screening.

However, my memory is first of all not faulty enough to make this method feasible for full-text screening, coding, and data extraction, and second of all, while it may have reduced the risk of overlooking something during title/abstract screening, it doesn't reduce the risk of effects from any systematic biases in my judgments. This is especially relevant since some of my inclusion criteria are inevitably based on somewhat subjective judgments, such as when something constitutes a mechanism rather than just another associated risk factor, or when providing argumentation for a mechanism is an aim of an article. These criteria were included due to restrictions of time and resources, as including every article that mentions a mechanism would have resulted in potentially nearing or over a hundred articles, but they also most likely reduced reliability of the criteria.

It should also be noted that my search terms related to migrants and ethnic minorities were restricted to general synonyms for ethnic/racial minorities and migrants and therefore didn't include terms for specific minority groups. This was largely a decision made for feasibility reasons; it didn't seem realistic to list every ethnic minority group in Western countries, and adding only a few select groups seemed like a rather unsystematic approach. However, this means that I may have missed relevant articles that address the psychosis risk of specific ethnic minority groups without using the more general synonyms I searched for in the title, abstract, or keyword tags.

### *Limitations of the included literature*

The included literature in the review has certain methodological issues, some of which are also addressed in the discussion of individual mechanism categories in the closing framework. It is outside the scope of this review to comment on all issues of every article, but there are some overall issues I would like to address.

Several articles propose mechanisms for why certain risk factors would cause psychosis but then conduct studies with designs that only test the association between psychotic symptoms and the risk factors involved. For example, Anglin and Lui (2023) present a mechanism for why racial microaggressions would have an effect on psychosis risk, but then only test whether racial microaggressions have an effect independently of effects of major discriminatory experiences. Another example is that both Cai et al. (2022) and Fashaw-Waters et al. (2021) only demonstrate that the relative number of schizophrenia diagnoses among Black nursing home residents with ADRD

rose after the CMS initiative, not why it rose. Others present theoretical arguments based on previous results without their hypotheses being tested (e.g., Kircher et al., 2020; Nazroo et al., 2020; Wallerstein, 2020). Thus, while many interesting mechanisms are proposed in the included literature, they are somewhat lacking in terms of actual testing.

Moreover, the only studies that use longitudinal data are Cai et al. (2022) and Fashaw-Walters et al. (2021), i.e., the studies arguing that the CMS initiative to publicly record the use of antipsychotics for non-schizophrenia diagnoses caused a rise in schizophrenia diagnoses among Black nursing home residents with ADRD. All other articles that aren't reviews or theoretical arguments are based purely on cross-sectional comparisons of data from one point in time. Granted, some articles include data on events that happened to participants before they developed psychotic symptoms (e.g., Bhui et al., 2021; Caroppo et al., 2020), but this data is collected retrospectively by asking participants to recall these events, opening it up to recall bias (i.e., systematic error from participants' current experiences affecting what they remember of the past; Porta, 2014). This matters because it makes it difficult to actually establish causality. In the 'Bradford Hill criteria' for causality that are still widely referred to today, Bradford Hill (1965, p. 297f) pointed to the importance of, among other things, temporality and experiment: If we want to be sure that something was the cause of an effect, we need to know that the cause came before the effect and ideally that if we change the proposed cause, the effect also changes. All articles that only use cross-sectional data have trouble showing the former, and even Cai et al. and Fashaw-Walters et al. have difficulty showing the latter, as there is no data on what the rates of schizophrenia diagnoses in nursing homes would have looked like if the CMS initiative had never happened.

In some cases, this calls into question the directionality of the proposed effects. It seems entirely plausible that experiencing more discrimination would lead to paranoid ideation, but it also seems plausible that paranoid ideation would cause you to perceive that you've been the victim of more discrimination in the past. In some cases like that of Pozza (2019), it becomes somewhat questionable if they are even showing anything we didn't already know: if aberrant salience and alexithymia are common during psychotic experiences, and psychosis is more common among migrants (Pozza, 2019, p. 2057f), then we would expect migrants to show higher levels of aberrant salience and alexithymia as well as psychotic experiences. Finding that this is so without

showing that one precedes the other doesn't really say anything except that migrants do, indeed, have higher rates of psychotic experiences.

An approach that would be interesting, but which is almost entirely absent from the included studies, is also qualitatively interviewing psychosis patients from ethnic minority groups about *their* experiences of what led to their psychotic symptoms. Lazaridou et al. (2023, p. 1011f) interview African migrants about their experiences with racism and their subclinical psychosis symptoms, and Caroppo et al. (2020, p. 79) include an element of interviewing their migrant participants (some of which have psychotic disorders) about their past, although it is unclear to which degree this is a qualitative interview versus a quantitative checklist. But no article studies more deeply how the process of being part of an ethnic minority and developing psychosis is experienced. Granted, this would be difficult to do with actively psychotic patients, but well-medicated or recovered patients could participate. This would of course also most likely be a cross-sectional study open to the recall bias I criticised above, as a longitudinal study with guarantee that any participants would develop psychosis would require so many participants that it would not be feasible to interview them all in depth. But advantages would first of all be hearing the perspective of the actual people affected, and second of all opening the understanding of the effect up to more complexity, as the experiences of e.g. racism and discrimination are inevitably simplified when you have to boil them down to a few items on a survey. It could give a deeper phenomenological understanding of the effect, while potentially helping to develop hypotheses about mechanisms that could then be tested in longitudinal designs.

# Conclusion

This scoping review aimed to map mechanisms for how psychosocial risk factors raise psychosis risks among ethnic minority groups, posited in the scientific literature in the time from Morgan et al.'s review in 2019 until March 2025. I found many different mechanisms, sometimes pointing in vastly different directions, including biological mechanisms, some with a psychological focus, some pointing to diagnostic practices, and one suggesting a selective migration effect. My findings have both differences from and similarities to those of Morgan et al. For one thing, though Morgan et al. also reviewed studies on mechanisms for the raised risks, they found little apart from the social defeat hypothesis and reduced grey matter volume resulting from environmental

stress. As discussed above, while acknowledging that some misdiagnosis probably happens, they also seem rather dismissive of the idea that it would be a major contributor (Morgan et al., 2019, pp. 250-252). In this study, on the other hand, I found quite a lot of vastly different proposed mechanisms that weren't limited to biological ones, many of which pointed to biased diagnostic practices as a major issue. We are perhaps left with the question of whether all of these mechanisms happened to not be suggested before 2019, or whether we would also find a lot more mechanisms than Morgan et al. did from before 2019 if a similar review with more systematic methods were conducted reviewing literature from that time.

However, there is also a theme present in this review that aligns with Morgan et al.'s conclusions. Almost (but not quite) every mechanism I found assumes that the higher psychosis risks among ethnic minorities are at least in part caused by racism and discrimination. It just varies *how* this racism and discrimination causes the risks and whether it is racism on a structural or institutional level, or if it's part of everyday interpersonal interactions. Of course, since I have only included articles presenting mechanisms and not just risk factors, this may also reflect that it's easier to come up with mechanisms for how racism could cause psychosis risk compared to other risk factors. But it nonetheless suggests somewhat of a general consensus that racism and discrimination are involved, much in line with Morgan et al.'s (2019, p. 256) conclusions that social adversity involving threat, hostility, and violence are likely to be a major risk factor. Though this is a scoping review and therefore one that can't draw firm conclusions about what actually causes the high psychosis risks among ethnic minority groups, the included literature does point towards the possibility that racism plays a major part in it.

# Part 3: Closing framework

This closing framework contains some more in-depth discussions that didn't fit in the article and its space restrictions. These include a discussion of the strengths and weaknesses of the presented mechanisms (which also touches upon their conceptualisations of psychosis), and a discussion of the included literature's conceptualisations of ethnicity and race and how these affect their conclusions.

## Strengths and weaknesses of the presented mechanisms

Some of the more general methodological limitations of the included literature are discussed in the article, but I would like to address the strengths and weaknesses of the mechanisms and their evidence more in depth in the following. As they vary widely, I will discuss them in the same overall categories that they were presented in in the results section of the article.

### *Biological mechanisms*

The biological mechanisms are interesting in their attempt to bridge the gap between social and biological explanations, something that Meloni argued in 2014 (pp. 593f; 597-603) was perhaps not entirely new, but newly popularised in sociology and biology, which previously had a rather antagonistic relationship. Multiple aspects that Meloni mentions as signs of a more social biology are represented. According to the mechanisms found, bodies and brains don't just work in isolation to cause psychosis but are connected with and affected by others in social relationships, and even genes can be affected by social factors via epigenetic changes regulating their expression (Simmons et al., 2024, p. 8). Where the biological mechanisms stand out in comparison to some of the others is that they include this biological element, arguably grounding psychosis in the body, not just in the mind and/or the social separately from this.

However, the mainly biological mechanisms face a problem that is common in biosocial explanations of psychiatric phenomena, as pointed out by Fletcher and Birk (2022, p. 14), namely that they need the psychological as an interface between the social and the biological to work, but that it's unclear what constitutes this psychological interface. Especially the social defeat hypothesis is criticised for reducing

psychology to rather vague terms of ‘uncertainty’ or ‘experiences of exclusion’ that are then supposed to form a bridge between social defeat and the biological consequences thereof (Fletcher & Birk, 2022, p. 9). How this psychological bridge works, what it involves, or what it’s like to be on it is not elaborated on; we only learn that it exists. Even the social factors involved in the social defeat hypothesis are criticised for presenting a somewhat reductionist conceptualisation of sociality, as the original work on the hypothesis assumes that results from experiments with isolated fighting rats can be transferred to understandings of complex human interactions in wider social contexts (Fletcher & Birk, 2020, p. 8).

Similar criticisms can be presented regarding the other biological mechanisms. They may not base their hypotheses on animal experiments, but we still meet some rather vague and oversimplified ideas of the psychological where it is at worst not addressed at all, at best reduced to an interface ‘experiencing’ social risk factors so that biological changes can take place (e.g., Bhui et al., 2021, p. 686; Saxena et al., 2024, p. 462f; Simmons et al., 2024, p. 2). Even some of the mechanisms I’ve categorised as psychological have this problem. Both Huque (2024) and Pozza (2019) present mechanisms where social conditions (discrimination and minority status, respectively) cause certain psychological factors that are associated with psychosis. However, both the social and the psychological are reduced to a few factors taken out of context in explanations that more or less only consist of, in one case, experiences of discrimination being associated with dissociation, negative self-schemas, and perceived stress, which in turn are associated with psychosis-like experiences (Huque et al., 2024, p. 62f), and in the other case, migrant status being associated with alexithymia and aberrant salience, which in turn are associated with psychotic experiences (Pozza, 2019, p. 2058). Further elaboration on how the social conditions could cause the psychological factors, or on how the psychological factors could cause psychosis, is nowhere to be found in these articles.

So why does it matter that the psychological aspects are missing or reduced significantly? For one thing, developing psychosis is something that happens to *persons* with complex cognitive, emotional, and interpersonal experiences, and any explanation that ignores this aspect is perhaps not directly wrong, but simplified. For another thing, we’re left somewhat at a loss as to why psychosis is the result instead of any other type of disorder. After all, social defeat is also used as a model of depression (Morgan et al., 2019, p. 254), and ‘stress’ is hardly a risk factor limited to

psychosis, but a term widely used for pressures and reactions that raise the risk of different disorders (Videbech et al., 2018, p. 25f). Both social exclusion and social stress are also too common for it to make sense that psychotic disorder rates are as relatively low as they are (Morgan et al., 2019, p. 254). What are the processes that cause *psychosis* to be the outcome? One could argue, based on a diathesis stress model of psychosis being caused by an interplay of heritable and environmental factors (Nordentoft & Vandborg, 2017, p. 308f), that social stressors cause psychosis only among those who are genetically disposed towards developing psychosis. But why, then, is it relatively common for the identical twin of a person with a psychotic disorder to not develop psychosis, despite presumably often being exposed to many of the same overall social stressors (Jeste et al., 2023, p. 867f; Pinel & Barnes, 2018, p. 500)? By reducing psychosis explanations to 'social stress of being an ethnic minority' causing 'biological changes in the body', this is difficult to explain without having to add the complexity of different interpersonal and psychological experiences back in. The biological and more reductionist mechanisms are by no means unimportant or uninteresting from a viewpoint of understanding more aspects of how psychosis happens and perhaps how to treat it somatically. However, in terms of truly understanding what happens and how we can intervene, we're left somewhat at a loss as long as we don't understand what happens 'between' being exposed to social risk factors and biological changes happening in the body.

### *Psychological mechanisms*

Especially when looking at the psychological mechanisms, the proposed explanations are very different from each other. As mentioned in the article, some of this perhaps reflects different fundamental theoretical and methodological standpoints of the researchers. Some of it may also result from different conceptualisations of what psychosis even is and what the central problems of it are (see also appendix C for collected data on psychosis conceptualisations). As shown in the results section, some mechanisms mainly focus on paranoia, some on language disturbances, some on hallucinations and delusions, and some on a lost sense of self and reality. The end result is that it's difficult to discuss the psychological mechanisms as one category, which is why I'm discussing them in subcategories similar to those used in the results section – not including Huque et al. (2024) and Pozza (2019), which I have already discussed above.

### Paranoia

Several of the included articles suggest that psychosis risk follows from paranoia caused by continual meetings with hostility. It seems intuitively plausible that paranoia could be caused by a threatening environment during migration and asylum-seeking processes as well as experiences with discrimination and hostility, perhaps moderated by aspects of social identity (Anglin, 2023, p. 289; Caroppo et al., 2020, p. 81; Elahi et al., 2022, p. 683f; Lazaridou et al., 2023, p. 1017; McIntyre et al., 2019, p. 19f). However, an interesting counterpoint is raised by some of the diagnostic process mechanisms: when is this pathological paranoia, and when is it an adaptive reaction to experiences of real hostility? As demonstrated by Wolny et al. (2023, p. 35), Black Americans tend to score higher on measures of paranoia on the SPQ regardless of their overall score, suggesting that the higher paranoia scores don't necessarily reflect a higher level of pathology. Looking at some of the items of the Suspiciousness subscale of the SPQ, this is perhaps not surprising, as it includes items like "I am sure I am being talked about behind my back", and "Do you often feel that others have it in for you?" (Raine, 1991, p. 558). Agreeing on these items may perhaps reflect pathological paranoia in some, but in someone who is continually subjected to racism and discrimination, it may say less about their supposed symptoms and more about their real-life experiences. It's not difficult to imagine that other assessment tools and diagnostic practices would have similar problems, casting doubt on the results of studies supposedly showing higher psychosis risk among victims of racism by showing higher levels of paranoid ideation among them. A fruitful path of further research might be to look into whether other assessment tools than the SPQ have this problem, and making norm material for them based on different minority groups to find out what is *actually* an abnormal level of suspicion in these groups. This would potentially both help to reduce rates of misdiagnosis, and to make it easier to interpret results of studies examining paranoia levels of minority groups.

### Language disturbance and thought disorder – the diglossia hypothesis

The diglossia hypothesis (Alherz et al., 2019; 2022) stands out as one of the few mechanisms that don't implicate direct pressures of racism in psychosis aetiology, although one could perhaps argue that for one spoken language or dialect to be viewed as having

higher status than another, some level of prejudice is likely taking place, making racism indirectly involved. It also stands out as the only mechanism viewing thought disorder and language disturbance as the 'gateway symptoms' to psychosis. A strength of this hypothesis that perhaps the paranoia mechanisms lack is that it actually somewhat contains an argument for why the symptoms it focuses on would lead to other psychotic symptoms. One path is via (somewhat postulated) experiences of the more prestigious H language exerting influence on individual thought, and one is via language's involvement in navigating social hierarchies, which is in turn related to dopaminergic transmission, possibly tying it to dopamine's involvement in delusions and hallucinations (Alherz et al., 2022, p. 8; Pinel & Barnes, 2018, pp. 501-503). However, in the evidence taken to support the hypothesis, it's perhaps unfortunate that diglossia is operationalised as an experience that "your first language puts you at a disadvantage in terms of life opportunities" (Alherz et al., 2022, p. 3), while no other forms of experienced discrimination are controlled for. This makes it difficult to tell whether the higher scores on psychotic symptoms actually are connected to the phenomenon of diglossia specifically, or more widely to experiences of ethnic discrimination, as other mechanisms in this review would argue. Until further evidence is presented, this makes it somewhat unclear why the diglossia explanation would be a better one than the ones pertaining to the discrimination that presumably is experienced more widely by ethnic minority groups than diglossia is.

**Hallucinations and delusions – the Bayesian hypothesis**

The Bayesian hypothesis of psychosis is the only psychological mechanism that specifically focuses on hallucinations and on delusions in a broader sense than just paranoid ones. It is also an explanation that contains both a psychological level in the shape of predictive processing and prediction errors, a social level in the shape of the discrimination and cultural misunderstandings causing these errors, and a biological level in the shape of dopaminergic transmissions related to the processing and the salience that ends up being attributed wrongly (Henssler et al., 2020, pp. 326; 331f; Jeste et al., 2023, p. 873; Jongsma et al., 2021, p. 1917f; Varchmin et al., 2021, p. 1). There is an attempt to integrate the social, the psychological, and the biological, without reducing the psychological to just a passive interface, as I criticised the biological mechanisms for above. It does, however, lead to some unanswered questions: Can the human mind

really be compared so easily to an input processor 'encoding' information and errors and thinking in terms of statistics? Can the judgment about whether an event was as expected be reduced to a two-dimensional evaluation of how unexpected the outcome was and how important the prediction error was, or is it more complicated than this? Do prediction errors elicit any emotions and action tendencies, and how do these fit into the path to psychosis (if at all)? And perhaps most central to the question at hand: How do other psychotic symptoms like thought disorder fit into the model? While interesting, it is a hypothesis that could perhaps use some more exploration – and testing, as this is rather absent from the included articles.

**Lost sense of self and reality**

The mechanisms that focus on a lost sense of self and reality are perhaps some of the most compelling, as they deal directly with the disconnection from reality that is widely considered one of, if not the most defining characteristic of psychosis (Pinel & Barnes, 2018, p. 501; Simonsen & Haahr, 2017, p. 325). It should perhaps be noted that the Bayesian hypothesis, in addressing both delusions and hallucinations, also deals with this disconnect. But where the Bayesian hypothesis mostly conceives of it as input processing gone wrong, the hypotheses in this subcategory address more of the lived experience of what happens, whether it's through the psychosocial disempowerment and negative self-awareness resulting from ongoing stereotyping and discrimination (Jongsma et al., 2021, p. 1917; Lazaridou et al., 2023, p. 1016f), through the insecurity of ambiguous hostile encounters (Anglin & Lui, 2023, p. 6), through loss of clear meanings, connections, and legal status (Caroppo et al., 2020, p. 81), or through yours and your forefathers' experiences being erased from public discourse (Wallerstein, 2020, p. 260). A strength to be considered here is also that by explaining a disconnect from yourself and reality, other symptoms are almost automatically explained along with it, like dissociation, or delusions resulting from failed reality testing (Caroppo et al., 2020, p. 81).

Interestingly, along with the subcategory positing that real experiences with hostility lead to paranoia, this subcategory also contains the only articles that base their hypotheses on, among other methods, qualitative interviews with people from ethnic minority groups and/or psychosis patients about their experiences (Caroppo et al., 2020; Lazaridou et al., 2023). Wallerstein (2020) also bases her conclusions on a

lengthy period of working with primarily Black psychosis patients and her conversations with them during this period. Could it be that this perceived loss of control over your own life and resulting loss of yourself and your connection to reality is something that resonates well with the actual *experience* of developing psychosis as a minority person? Hard to tell: First of all, there are not a lot of articles to go on. Second, it is very unclear how much especially Caroppo et al. base their interpretations on things actually said by participants in open qualitative interviews, and how much they make their own interpretations based on answers to more closed and structured questions (Caroppo et al., 2020, p. 79). And thirdly, Wallerstein's interpretations seem to on one hand be based on lengthy observations of and conversations with Black psychosis patients, but on the other to be jumping to conclusions at times without checking interpretations with the patients in question, such as when she interprets that 'Loki's' act of bashing his head against the wall while shouting "Look what you are making me do!" is a reference to his father's death in the mass-murder-suicide of Jonestown, Guyana in 1978 (Wallerstein, 2020, p. 263f). Nonetheless, an interesting question is raised about which of these mechanisms would resonate with the actual people affected, and whether it indeed would be the ones of experienced hostility leading to paranoia, and various experiences of disempowerment and insecurity leading to a lost sense of self and reality.

### *Diagnostic practices*

An interesting aspect of the mechanisms relating to diagnostic processes is that, at least in the timeframe covered by this review, articles about these mechanisms seem to almost exclusively focus on Black people in the US, the UK, and Canada. This overrepresentation is present in the included articles in general and makes a certain amount of sense, as these groups are also among ones where the increased psychosis risks are the highest (Jegarl et al., 2023, p. 30; Morgan et al., 2019, p. 247), but it is especially present in articles arguing for mis- or over-diagnosis mechanisms. While more research on other minority groups may be called for in the field in general, it might be an especially interesting question for further research whether other ethnic or racial minorities are subject to the same biases and misunderstandings from clinicians, or if this is mainly an issue affecting Black populations.

Moreover, as mentioned by Morgan et al. (2019, p. 250), it's unlikely that the entirety of the raised psychosis risks can be explained by misdiagnosis. As Anglin (2023, p. 283f) points out, disparities don't just exist in psychotic *diagnoses*, but also in various subclinical psychotic experiences, even when rates are based on self-report surveys that don't include clinical assessment. As these subclinical symptoms include a variety of experiences of "unusual thinking, fixed beliefs, and altered perception" (Anglin, 2023, p. 283), it also seems unlikely that they can all be explained entirely by these surveys failing to account for adaptive reactions like the suspicion discussed above. I noted only one included article seemingly claiming that diagnostic errors and biases are the *only* well-supported explanations both empirically and conceptually, but this article seemingly also considers the only other possibility to be genetic or epigenetic group differences, lumped together as if they were the same purely genetic explanation rather than epigenetics being a mechanism for how external factors can change the body (Schwartz et al., 2019, p. 263f).

On the other hand, with the number of articles arguing that misdiagnosis happens, it also seems unwise to disregard it as a contributing factor as much as Morgan et al. (2019, p. 250) perhaps unintentionally do in the review I took as my starting point for this project, when they only briefly mention that there is some evidence suggesting this may be happening in the US, before listing others studies in more detail suggesting that it's unlikely to contribute too much. But as this is a scoping review that neither synthesises the evidence nor systematically assesses the quality of the included research, I can't make any conclusions about the size of the contribution of misdiagnosis to the high psychosis risks either.

### *Selective migration*

Regarding Andersen et al.'s (2021) argument in favour of selective migration as a contributing mechanism, it does raise an interesting criticism of previous arguments against this in pointing out that these arguments are often based on migrants tending to show less general dysfunction than the general population. As they argue, this overall good health doesn't necessarily mean that no selective migration happens, since an effect of general dysfunction making migration less likely might hide a coexisting selection for more specific psychosis-related factors in the overall statistics, in this case positive schizotypal traits associated with exploration tendencies (Andersen et al.,

2021, pp. 1-3). However, their study design is somewhat unconvincing: They examine the association between positive schizotypal traits and plans to migrate among American youth that have specifically *not* migrated. There is first of all a lack of shown connection between positive schizotypal traits and actual *completed* migration plans, and second of all, as they also admit themselves, it may be difficult to generalise the results from an entirely American population to the usually European, African, or Middle-Eastern migrants that have been the focus of studies showing higher psychosis risks among migrants (Andersen et al., 2021, p. 3f). While they raise an interesting hypothesis, it's one that requires more evidence to be truly convincing.

**Table 4. Overview of strengths and weaknesses of the suggested mechanisms**

*Main strengths and weaknesses that I emphasise in the discussion. Many more could likely be mentioned.*

| Mechanisms | Strengths | Weaknesses |
|---|---|---|
| Biological mechanisms in general | Include biological aspects, grounding psychosis in the body and not just the psychological or social | Reductionist in their approach to the psychological and sometimes the social, resulting in trouble showing why psychosis should be the outcome rather than other disorders |
| Less specific mechanisms (Pozza, 2019, and Huque et al., 2024) | None mentioned in this paper | Problems similar to those of the biological mechanisms |
| Paranoia as a reaction to actual hostility + the social identity hypothesis | Seem intuitively plausible, and include articles where minority persons are actually asked qualitatively about their experiences and how they affect them | Difficulty explaining other psychotic symptoms than paranoia, and unclear how much the paranoia in question is actually pathological versus adaptive suspicion |
| The diglossia hypothesis | Includes explanation of how the 'gateway symptom' (language disorder) would lead to other symptoms | The evidence presented has difficulty showing that the effect is caused by diglossia specifically and not general ethnic discrimination |

| | | |
|---|---|---|
| The Bayesian hypothesis | Integrates a social, a psychological (cognitive), and a biological level, focuses broader than just paranoid delusions, and deals directly with the disconnect from reality widely thought of as central to psychosis | Possibly reductionist view of the human mind and experience, and it's unclear how other symptoms fit into the hypothesis |
| Lost sense of self and reality | Deal directly with the disconnect from reality widely thought of as central to psychosis, provide a pathway to some of the other symptoms, and include articles that actually examine the lived experience of participants qualitatively | It's unclear how much some of the qualitative interpretations actually line up with participants' own understandings of their experiences in the presented evidence |
| Diagnostic mechanisms in general | Raise important questions about the validity of not just diagnoses, but results of research studying e.g. paranoia symptoms | Unclear if they can be generalised wider than Black people in the US, UK, and Canada |
| Selective migration based on positive schizotypy | Raises interesting criticism of widely used arguments against selective migration | The presented empirical evidence is somewhat unconvincing |

# Can the different mechanisms work together?

As reflected in the results and discussion above, the mechanisms found in this review point in many different directions. Some may (upon further studies) turn out to be able to work together as parts of the same explanations. For example, within the psychological mechanisms focusing on paranoia, the idea of paranoia as a pathological development of an initially understandable reaction to real hostility could fit with the social identity hypothesis proposing that this hostility is more problematic when it comes from members of a group you identify with. The Bayesian hypothesis, though stated in somewhat different statistics-related terms, could perhaps supplement this with an understanding of what happens cognitively to eventually make your initially adaptive predictions of hostility become dysfunctional. On a related note, regarding both the Bayesian hypothesis and the diglossia hypothesis, aberrant dopamine transmission is explicitly thought to be involved as a biological component (Alherz et al.,

2022, p. 8; Henssler et al., 2020, p. 332), providing an example of psychological and biological mechanisms that may be part of the same explanations (although perhaps the two psychological mechanisms work less well together internally, see further down).

For that matter, since the biological mechanisms in general don't specify what happens on the psychological level and mostly also vice versa, most of the biological mechanisms could be hypothesised to be the biological level of some of the psychological mechanisms, even though it would require research and more thorough examination of the theoretical underpinnings to confirm or disconfirm this. There are a few exceptions of biological mechanisms that already at a glance work less well with the psychological mechanisms: Obstetric complications don't fit very well, since the psychological mechanisms all hypothesise how certain factors cause psychosis risk directly in the people affected, not how it transfers to their children. Another is that the automatic outgroup categorisation hypothesis implies that there isn't necessarily anything more complex going on psychologically than the perception of people you automatically categorise as outgroup members, which clashes with the psychological mechanisms.

Other combinations may not make sense as part of the *same* explanation but could still complement each other by each explaining some of the effect. Overall, it's easy to imagine that some of the high psychosis risks could come from selective migration, some from different aspects of diagnostic practices leading to mis- or more diagnosis in minority groups, and some from biological and psychological mechanisms causing higher risk of real psychosis. There also doesn't seem to be anything inherently stopping all the diagnostic mechanisms from coexisting alongside each other, and the same could be said about the biological mechanisms.

It becomes more problematic when looking at some of the psychological mechanisms internally. As mentioned before, while few articles directly define what they mean by psychosis, it seems from the different focus points in especially the psychological mechanisms that they vary widely in what they see as the central symptoms and aspects of psychosis (see also appendix C). This results in very varied and perhaps somewhat fragmented explanations that therefore also have a harder time working together. It's difficult to pair the diglossia hypothesis and its focus on language as a primary motor of psychosis with mechanisms positing that paranoia and a lost sense of self and reality are reactions to contexts of hostility and ambiguity. It's likewise

difficult to pair either of these understandings with the idea that alexithymia is the driving factor of psychosis risk.

This issue perhaps also reflects the general state of psychosis conceptualisations. Just within the schizophrenia disorder, this diagnosis can cover a wide range of symptom combinations, such that some diagnosed people experience little to no paranoia, while others experience a lot of paranoia but little to no hallucinations or thought disorders (World Health Organization, 1994, pp. 65-71). It has been argued before that schizophrenia is defined so vaguely that the best path forward may even be to drop the diagnosis and simply focus on individual symptoms separately (Bentall, 2006, pp. 223-226). If so, the different mechanisms may not be contradicting each other but simply be mechanisms for different disorders or symptoms. However, if that is the case, it does seem like quite the coincidence that all these separate phenomena constituting psychotic experiences have some reason to be more common among ethnic minorities. If we want to find out more about which of these mechanisms can truly work together to form more complex explanations, and which are worth pursuing further in research, perhaps a fruitful way forward (that is beyond the scope of this project) is to first find out if it's possible to agree on a unified idea of what psychosis is, or if we *do* need to find different explanations for different symptoms. But as mentioned, this is mostly an issue that arises when comparing the psychological mechanisms with each other. Apart from some of these, the posited mechanisms could generally theoretically either work together as parts of the same, more complex explanations, or in parallel as each explaining a part of the effect.

## Conceptualisations of race and ethnicity

The idea behind collecting data on the conceptualisations of race and ethnicity found in the included literature (presented in appendix C) was to examine whether there are any major differences in how these are defined, and whether any such differences are connected to differences in the mechanisms presented. This was inspired by Jongsma et al.'s (2021) discussion of how different views of ethnicity result in different explanations of ethnic minority psychosis risks. However, overall, there is not much explicit disagreement to be found in the included literature. Few included articles explicitly define what they mean by race or ethnicity. In the five articles that define race (Anglin, 2023, p. 279; Faber et al., 2023, p. 2; Fashaw-Walters et al., 2021, p. 3624; Simmons

et al., 2024, p. 5; Tobon et al., 2021, p. 24), one that defines ethnicity (Jongsma et al., 2021, pp. 1915f; 1919), and three that define both race and ethnicity or ethnoracial identity (Huque et al., 2024, p. 59; Knight et al., 2024, p. 2; Nazroo et al., 2020, p. 264), there is broad agreement that ethnicity and race aren't natural categories, but social constructs. Moreover, Anglin, Faber et al., Huque et al., Nazroo et al., and Jongsma et al. all conceptualise ethnicity and/or race as tied to power struggles and as ways to oppress, discriminate against, and withhold resources from people perceived as being different from the majority (White) population. When looking at the articles that don't explicitly define race or ethnicity but do report how they operationalise them, there are also tendencies towards operationalising them as self-identified or based upon how others perceive you (e.g., Hunter et al., 2021, p. 898; Ku et al., 2023, p. 1708; Rabin & Palaniyappan, 2023, p. 701). The other main tendency to be found is to operationalise race or ethnicity based upon geographical descent (i.e., where your ancestors came from), but this is mostly done when focusing on migrants (e.g., Henssler et al., p. 326; Lazaridou et al., 2023, p. 1012), with the exception of Elahi et al. (2022, p. 681) discussing ethnic minority status in general but focusing on people of Pakistani descent.

The only article that operationalises race based on genetics is strangely one of the articles listed above as explicitly defining race as a social construct. Simmons et al. (2024, p. 2f) base their race variable on the race reported by next-of-kin or lab technicians but then check this for validity by comparing with 'genetically inferred' race. Whether this is a reluctant attempt to compensate for not being able to ask the dead participants in their study what they identified as, or if they believe that the social construct of race in question is a socially constructed division of certain gene groups, or if it simply reveals some confusion about what they actually believe race to be, is unclear. But again, this operationalisation is the exception, not the rule. Most articles that don't define and/or operationalise ethnicity and/or race based on participants' own or others' perceptions of them simply don't report at all how they define or operationalise them (e.g., Bhui et al., 2021; Cai et al., 2022; Lincoln et al., 2022).

So are there any overall connections in ethnicity and race definitions and the conclusions reached? They're difficult to spot, except that Simmons et al. (2024) is the only solely 'biological mechanism'-focused article that explicitly defines race or ethnicity, perhaps suggesting less of an interest from biologically minded researchers in the question of the social construction of such categories. Overall, however, it's

interesting that almost all articles point to the ways we treat people (or in the case of the diglossia hypothesis, languages) that we perceive as different as the central factors causing psychosis risks. Caroppo et al. (2020, p. 81) also point to certain more migration-specific factors such as bureaucratic processes involved in being a refugee. But in general, even the migration-focused articles view racism and tensions between ethnic and racial groups as deciding factors, making how we are perceived by others and which groups we believe ourselves to be part of central. Andersen et al.'s (2021, p. 1) selective migration hypothesis pointing to completely different factors than racism becomes interesting as an exception to the rule, as they also present the one article about migration where they seemingly actually *don't* incorporate into the explanation what the geographical origin of the migrants in question is, how they perceive their own group membership, or how they're perceived and treated by others.

Thus, while there may be some tendencies towards operationalising race or ethnicity as geographical origin, the aspects that tend to shine through in explanations are feelings of group membership as well as the social and structural consequences of being categorised by others as a minority or as different. Much as Jongsma et al. (2021, p. 1919) conclude, the most fruitful ways to view ethnicity (and race) when trying to understand psychosis risks are perhaps the ones conceptualising it based on feelings of identity and relations to others, as well as the social structures keeping certain minority groups in positions where they're discriminated against both interpersonally and on societal levels.

# Conclusion

As reflected in the review and subsequent discussion, the mechanisms posited in contemporary scientific literature for how psychosocial risk factors raise psychosis risks among ethnic minorities point in many different directions. Some may, after more research, turn out to be able to work together as parts of the same, more complex explanations. Others may not be part of the *same* explanation but could still complement each other by each explaining some of the effect. Some, however, do seem mutually contradictory due to their basic disagreements about what psychosis is. The confusion of the field can make it difficult to draw many certain conclusions from this paper. However, there are some points that arise which I would like to underline:

1) Every posited mechanism needs to be researched further before anything can be concluded about how much they contribute to the effect (if at all). Even if I had attempted a systematic review synthesising the evidence on any one of them, the literature included here suggests that there would not be enough evidence to synthesise, at least from recent years. While some mechanisms may stand out as particularly under-supported, there are none that stand out as presenting enough empirical evidence to be more than reasonable possibilities.

2) Many of the proposed mechanisms don't necessarily contradict each other. However, to achieve consensus on especially what happens psychologically to cause the higher psychosis risks among ethnic minorities, it is perhaps necessary to first come closer to agreement on what we actually mean by psychosis. Paranoia? Thought disorder? Hallucinations? A lost sense of self and reality? All of the above in combination as one disorder, or appearing separately as independent symptoms? Until we agree somewhat on what we are trying to explain, explaining it will be difficult. Perhaps a fruitful way forward that would also help to bridge the gaps between the social, psychological, and biological levels, would be more interdisciplinary teams working together on the problem, as well as actually talking to some psychosis patients belonging to ethnic minority groups to hear what they have to say about their own experiences.

3) While a lot of included articles don't explicitly define ethnicity or race, the ones that do mostly agree that they are socially constructed concepts. Moreover, in the way they approach the issue of ethnicity/race and psychosis risk, what becomes central is how we perceive ourselves and others as part of these groupings, and the consequences this has for the way certain groups are minoritised and discriminated against at structural, institutional, and interpersonal levels, leading up to my final point:

4) As pointed out in the conclusion of the article section, the vast majority of included articles suggest that racism and discrimination are a cause of the higher psychosis risks in ethnic minorities, mirroring some of the conclusions from the review by Morgan et al. (2019) that this thesis took as its starting point. Thus, even if the mechanisms don't quite agree on *how* racism and discrimination cause psychosis, these risk factors may be viable targets for research into how to intervene to reduce psychosis risks in minority groups. As I have also collected data on such intervention suggestions in the included articles, I will discuss this question further in my exam presentation.

# Curriculum

## New literature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