

Racism-related developmental origins of mental health: a conceptual model and scoping review

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Mental health disparities faced by racially and ethnically minoritized populations in the USA are persistent and widespread. This analysis presents a conceptual model and scoping review of an overlooked pathway to racial/ethnic mental health disparities: racism-related developmental origins of mental health. The model links historical and structural racism to an increased likelihood of exposure to disproportionate and unique stressors among racially and ethnically minoritized populations, which in turn confers heightened vulnerability to mental health disorders in the next generation. Here, through a scoping review, we describe the state of the evidence supporting this model. Our results show how preconception, prenatal and early-life experiences—including exposure to trauma and stressful life events, socioeconomic disadvantage, environmental toxins, sociocultural factors (such as discrimination and acculturative stress), and aspects of maternal mental, behavioral and physical health—are linked to an increased risk of poor mental health. Areas where more research is needed are discussed, including further investigation of sociocultural stressors and potential protective factors.

Compared with their white counterparts, racially and ethnically minoritized (REMD) individuals experience more severe symptoms and persistent courses of mental illness^{1–3}. There are also higher rates of mental health disorders such as post-traumatic stress disorder⁴, alcohol use disorder⁵, schizophrenia⁶, attention deficit hyperactivity disorder (ADHD)^{7,8}, conduct disorder⁷ and major depression⁹ among many REMD populations compared with white populations. Despite such stark disparities, gaps remain in understanding the contributors to these differences, spurring calls to identify and address reasons for this disproportionate mental illness burden¹. Importantly, there is a growing understanding of the imperative for such efforts to be grounded in the recognition that race itself is not a biological risk factor for poor health outcomes. Rather, the task is to identify and confront the deeper reasons behind persistent racial disparities in health^{10–12}.

So far, the growing body of research in pursuit of this goal has primarily focused on two complementary strategies. The first strategy involves documenting how REMD individuals face disproportionate and unique stressors that contribute to mental health risk and diagnoses, stemming from experiences of racism, discrimination and trauma^{13–15}. The second examines how these vulnerabilities are exacerbated by barriers to treatment faced by REMD populations, including lack of access to mental health services and/or culturally competent providers, differences in insurance type, mental health stigma and medical mistrust stemming from past experiences of mistreatment^{8,16–19}. Here we argue that there is a third critical mechanism that has often been overlooked and understudied: the chronic stressors to which REMD populations are disproportionately exposed alter maternal physiology and, consistent with the developmental origins of health and disease (DOHaD) model, transmit mental health vulnerabilities across generations^{20–22}.

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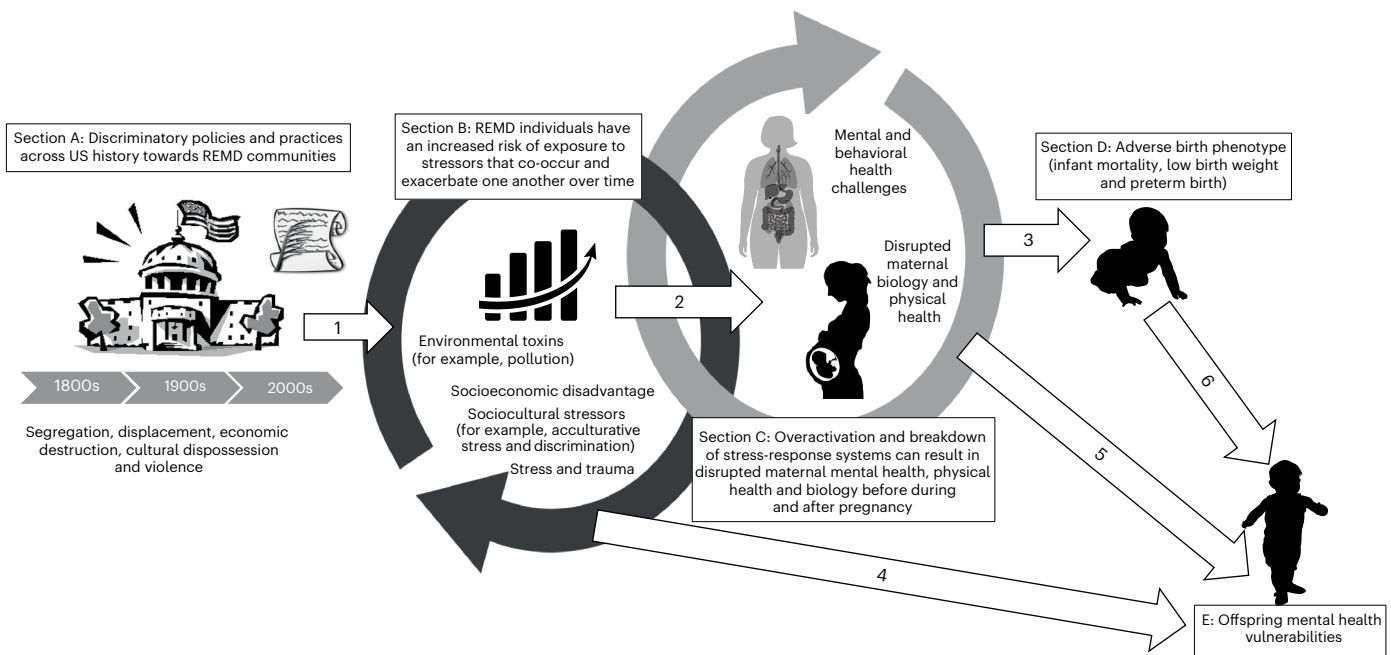


Fig. 1 | An integrated conceptual model of the racism-related developmental origins of mental health. Figure adapted with permission from ref. 22, Elsevier.

The DOHaD model posits that preconception, prenatal and early-life experiences impact later mental and physical health through environmental influences during sensitive windows of fetal development and early life²³. Originally based on studies of nutritional deprivation in utero, evidence has grown to show that adverse experiences such as exposure to stress and trauma early in life, including prenatally, can lead to an increased vulnerability to health challenges^{24–26}. For example, prenatal maternal stress and adversity have been linked to internalizing and externalizing behaviors, depression, anxiety and ADHD among offspring^{27–33}. In addition, research has shown that maternal adversity exposures even before pregnancy are associated with child physical and mental health outcomes, including childhood respiratory risk, increased body mass index, hospitalization for severe infections, other illnesses and antibiotic use, poorer overall health status, emotional reactivity in infancy, and child internalizing and externalizing disorders^{34–42}. Last, exposures to stress in infancy have been tied to accelerated epigenetic age and worse metabolic health, increased somatic symptoms, excessive inflammation, child internalizing and externalizing symptoms, adolescent and young adult increased risk for depression and suicidality, and an overall greater likelihood of developing psychiatric or psychological disorders^{43–53}.

The DOHaD framework is particularly relevant to REMD populations given the state of maternal and child health in the USA. Currently, the USA ranks first among high-income countries for its maternal mortality rate⁵⁴. A major factor driving these outcomes is the persistent racial and ethnic disparities in maternal health. For instance, Black women are more than twice as likely to die from pregnancy-related complications than white women, followed by American Indian and Alaska Native women⁵⁵. Latinx/Hispanic, Asian American/Pacific Islander and multiethnic women also experience disproportionately higher rates of severe maternal morbidity compared with white women⁵⁶. A growing body of research has shown that structural racism, implicit bias and inequitable access to high-quality care are central drivers of these disparities^{22,57,58}.

Despite the robust body of evidence linking early-life stress exposure to mental health risk, the growing understanding of the disproportionate and unique racism-related stressors faced by REMD populations, and the documented presence of REMD physical and mental health

disparities^{20–22}, the field is missing a conceptual and empirical synthesis of the links between these concepts to show how REMD populations' vulnerability to mental illness may begin even before conception. In this analysis, we present an integrated conceptual model of racism-related developmental origins of mental health and a scoping review of supporting evidence. The model, illustrated in Fig. 1, posits that the legacy of hundreds of years of discriminatory policies in the USA, such as slavery, internment camps and redlining, has created enduring societal structures and norms that continue to marginalize and oppress REMD populations (Fig. 1, section A). These structures manifest in present-day conditions such as mass incarceration, under-resourced and polluted neighborhoods, economic hardship and community violence^{59–65}. The presence of these adverse structures and norms increases the likelihood of exposure to stress and adversity among REMD populations (Fig. 1, pathway 1). Initial stressors beget new stressors in a proliferation process⁶⁰ and, over time, get 'under the skin' (for example, weathering, toxic stress, wear and tear) through disruption of stress-response systems, including neuroendocrine, autonomic nervous and immune systems, which in turn contributes to stress-related disease⁶⁶ (Fig. 1, pathway 2). In the context of pregnancy, these biological disruptions can alter fetal development, timing of parturition and increase the risk of adverse birth outcomes²² (Fig. 1, pathway 3). Finally, these factors—parent and child exposure to stressful experiences and environments, pre- and postnatal maternal health challenges (physical, mental and behavioral) and adverse birth outcomes—can all increase child vulnerability to mental illness^{46,51,52,67–71} (Fig. 1, pathways 4–6).

Existing reviews provide overviews of the supporting evidence for pathways 1, 2 and 3 in Fig. 1. Bailey and colleagues⁷² present a narrative review explaining how the system of structural racism creates conditions that disproportionately expose REMD populations to chronic stressors and conditions of adversity (pathway 1), which in turn contributes to poor health (pathway 2). Specifically, interconnected institutions, including housing, schools, the labor market, healthcare and the criminal justice system, contain embedded policies and practices that disadvantage certain groups compared with others; these institutions often reinforce one another, compounding their detrimental effects. For example, Black Americans have historically been segregated into neighborhoods in closer proximity to environmental hazards, such as high-traffic roadways,

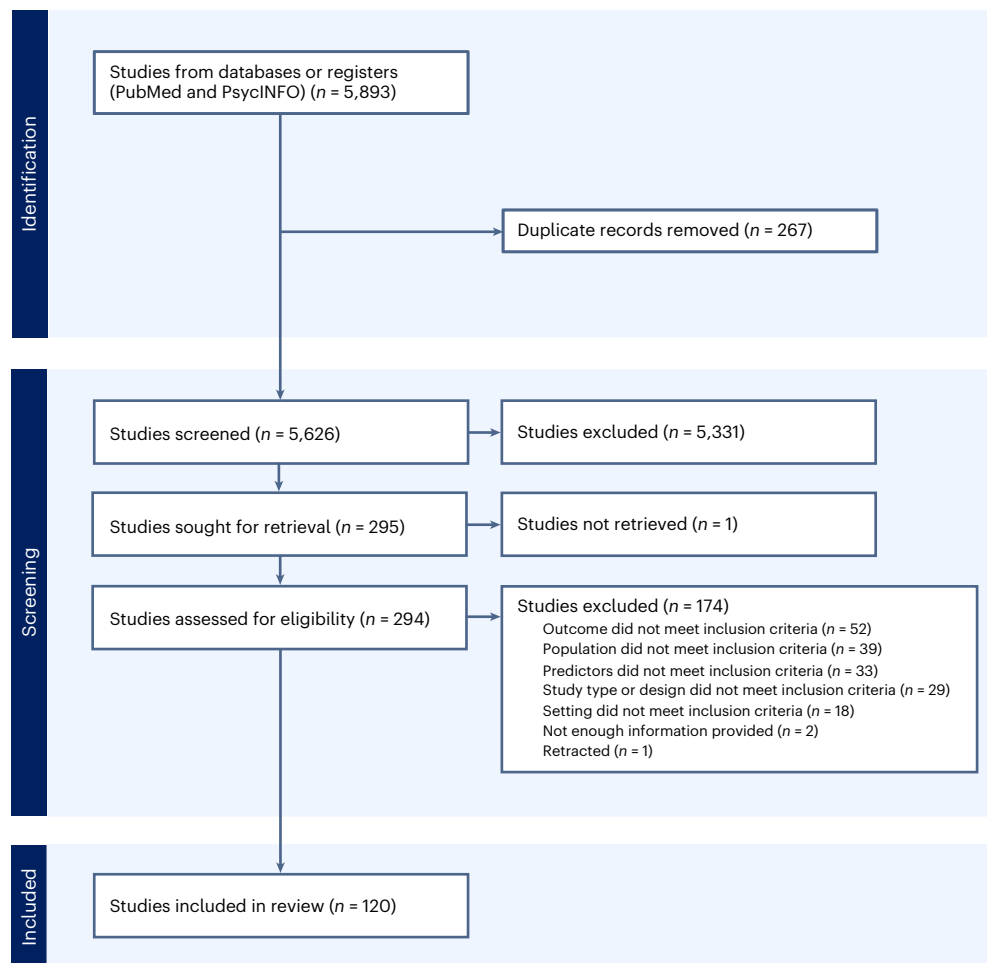


Fig. 2 | PRISMA flow diagram of the literature search and selection process.

industrial facilities and other sources of pollution (for example, the Flint water crisis), resulting in increased exposure to toxins. Through similar pathways, residential segregation has also been associated with elevated exposure to crime and reduced healthcare access and utilization. As another example, exclusionary laws and policies in employment (such as the Social Security Act of 1935, which denied majority Black domestic and agricultural workers access to key benefits) have perpetuated inter-generational poverty, a key determinant of poor health.

In further support of the proposed model, the American Psychological Association⁶⁰ presented a state-of-the-science report on stress as a driver of health disparities. The report summarizes research documenting notable disparities by both socioeconomic status and race in exposure to stressors such as discrimination, violence, financial insecurity and neighborhood disadvantage, and illustrates how these disparities can be traced to past and present discriminatory actions and policies (Fig. 1, pathway 1). The authors review the research linking exposure to these stressors to poor health through their influence on neurobiological, physiological and behavioral functioning (Fig. 1, pathway 2). The report emphasizes the bidirectional relationship between stress and health, with each perpetuating the other across generations. Specific to adverse pregnancy outcomes (Fig. 1, section D), our 2022 narrative review²² synthesizes evidence linking racism-related stress to disruptions in stress-response systems and, in turn, to adverse pregnancy outcomes (Fig. 1, pathways 2 and 3). We highlight how repeated activation of the hypothalamic–pituitary–adrenal axis, autonomic nervous system and immune system contribute to ‘toxic stress’ and biological wear and tear. This dysregulation increases vulnerability to hypertension, infection and other stress-related conditions that compromise pregnancy health²².

Taken together, existing reviews^{22,60,72} provide empirical support for part of the model in Fig. 1; specifically, how structural racism drives disproportionate stress exposure, how that stress gets under the skin through biological disruption, and why these mechanisms contribute to persistent racial disparities in maternal health. So far, the literature is missing a comprehensive review examining the remaining part of the model (Fig. 1): the associations between maternal exposure to stressful experiences and environments; maternal physical, mental and behavioral health challenges; and adverse birth outcomes to offspring mental health among REMD populations (Fig. 1, pathways 4, 5 and 6). Therefore, the goal of this scoping review is to provide an overview of the state of the evidence on associations between different types of stressors in the prenatal, preconception and postpartum periods and offspring mental health (pathway 4); maternal mental, behavioral and physical health and offspring mental health (pathway 5); and birth phenotype and offspring mental health (pathway 6), specifically among REMD populations. Importantly, our model highlights that structural racism amplifies exposure to a wide spectrum of adversities (Fig. 1, section B), including many not typically recognized as racism related at face value. For this reason, our analysis takes a broad approach, examining the full range of stressors and adversities shown to impact REMD populations’ health and well-being.

Methods

Search strategy

The scoping review conducted for this analysis followed the Preferred Reporting Items of Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) guidelines⁷³. The review was not

preregistered as PROSPERO does not accept scoping reviews. However, the methods were specified a priori and are reported here in full for transparency. Together, the first and last author of this scoping review developed the search strategy based on key terms in the literature on REMD populations in the USA and the developmental origins of psychopathology. Systematic searches were executed on 2 September 2021 and again on 7 June 2023. The full list of search terms is provided in Supplemental Appendix 1. Two databases (PubMed and PsycINFO) were selected for their comprehensive coverage of biomedical, psychological and social science literature. Searches in PubMed used both keywords and Medical Subject Headings (MeSH) to maximize capture of relevant articles. The search was limited to studies that were peer reviewed, in English, conducted with humans and quantitative.

Searches yielded a total of 5,893 studies, which were uploaded into Covidence systematic review software. After removing duplicates, three research team members reviewed the titles and abstracts of the remaining studies to evaluate whether they met inclusion criteria. Criteria for inclusion were as follows: (1) be an empirical study conducted in the USA; (2) participants were either solely or majority members of a racially or ethnically minoritized group, including Asian/Asian American, Hispanic/Latinx, African American/Black, American Indian/Native American, Alaska Native, Native Hawaiian, Pacific Islander or multiethnic/multiracial; and (3) studies examined the association between at least 1 measure of the preconception, prenatal or early postnatal environment (within the first 12 months of life) and at least 1 measure of externalizing or internalizing symptoms or behaviors, or early indicators of risk such as temperament, fear inhibition, prosocial behavior and insecure attachment^{74–77}. This review did not include studies focused only on neurodevelopmental disorders such as autism or cognitive delays.

To ensure screening reliability, all coders screened the first 50 article abstracts. Initial agreement was 92% between all coders, who then met to further refine screening rules and reach consensus on articles with disagreements. Coders then individually screened the remaining article abstracts for whether they met inclusion criteria. To continuously check reliability, the coding team screened 20 of the same abstracts each week of active coding (the average agreement between the coding team members and team lead was 98%). After abstract screening was completed, each full-text review was completed by two reviewers. For studies meeting the inclusion criteria, the following data were extracted: sample size, mothers' and children's race/ethnicity, predictor construct and measurement tool, developmental stage when the predictor was measured, outcome construct and measurement tool, child age when the outcome was measured and a summary of findings. Two reviewers completed extraction for each study. All coding discrepancies at each stage were discussed and resolved by team members. We extracted key study characteristics (for example, sample demographics, predictors, outcomes, measures and findings) into a summary table (Supplementary Data Table 1) to facilitate comparison across studies.

Results

After removal of duplicates, our search yielded 5,626 studies for screening, 120 of which were determined to meet inclusion criteria. The PRISMA flow diagram showing the number of studies included and excluded at each search stage is shown in Fig. 2. We present our results in accordance with our model, grouping together studies that examine the different model pathways 4, 5 and 6. A detailed summary of the included studies, including sample composition and key findings, is presented in Supplementary Data Table 1. Summary counts of significant, null and mixed findings are presented in Supplementary Table 1. However, given the substantial heterogeneity in exposures, outcomes and analytic approaches across studies, these values should be interpreted cautiously and are best viewed as descriptive context for the detailed narrative results that follow. In our research summary, we use the racial and ethnic terminology used in each study, recognizing

that descriptions and terms for different races and ethnicities have not remained consistent over time and continue to evolve. In addition, we acknowledge that not all birthing people identify as mothers. Throughout this analysis, we use the terminology of the included studies, while recognizing the importance of inclusive language in future research. Importantly, it is established in DOHaD research that some offspring vulnerabilities to preconception, prenatal and postpartum stress are sex differentiated^{32,78–83}. Therefore, any findings related to sex differences are explicitly noted in Supplementary Data Table 1.

Pathway 4

We begin by synthesizing research investigating pathway 4, which is the link between exposure to specific stressors in preconception, prenatal and early-life periods and child mental health outcomes among REMD populations.

Environmental toxins. Multiple studies examined the impact of early-life exposure to environmental toxins on mental health, with a particular focus on the chemicals present in plastics (bisphenol A (BPA) and phthalates), chemicals stemming from the combustion of fossil fuels (polycyclic aromatic hydrocarbons (PAH), nitrogen dioxide and particulate matter) and pesticides (organophosphates). Many of these studies^{84–91} were conducted with the Columbia Center for Children's Environmental Health cohort, which comprised low-income African American and Latino mothers and children from New York City. Existing research on chemicals in plastics revealed associations between prenatal BPA and phthalate exposure and behavioral and mental health in early and middle childhood (3–12 years old). Generally, greater exposure is tied to more behavioral and emotional problems among boys, and fewer behavioral and emotional problems among girls^{84,87,89,91}.

Prenatal exposure to chemicals from fossil fuel combustion has shown varying impacts on child development. While several studies^{86,92} on prenatal PAH exposure found it was not associated with behavioral problems in young children, another study⁸⁵ identified links to delayed self-regulatory capacity and ADHD symptoms in older youth, exacerbated by material hardship^{88,90}. Higher prenatal nitrogen dioxide levels were associated with internalizing and externalizing behaviors, with increased susceptibility among African American individuals and participants facing socioeconomic adversity⁹³. Particulate matter exposure was also correlated with increased internalizing behaviors in children⁹⁴.

So far, research between prenatal organophosphate exposure and child development has focused only on attention problems in young children, and the results are inconclusive. One study⁹⁵ did not find evidence of an association, while another study⁹⁶ identified a significant link. An additional study⁹⁷ did not find associations between prenatal organophosphate exposure and risk-taking behavior at 18 years old.

Socioeconomic disadvantage. Several studies examined the effects of socioeconomic disadvantage, broadly defined, in the prenatal and postpartum periods. Greater household density in the prenatal period was related to higher levels of youth depression and anxiety⁹⁸. Food insecurity, neighborhood stress and material hardship (for example, housing disrepair and difficulty paying bills) in late pregnancy predicted infant temperament (specifically lower orienting and regulatory capacity)⁹⁹. Socioeconomic risk was positively associated with child socioemotional behavioral problems at age one; however, maternal parenting knowledge mitigated risk¹⁰⁰. Finally, inadequate family income at birth and lower maternal education were both associated with emotional-behavioral problems among young children^{101–103} and child ADHD¹⁰⁴.

Stressful life events. Three studies^{105–107} identified associations between stressful life events (SLEs) during pregnancy and internalizing and externalizing symptoms, in contrast to one study¹⁰⁸ that did

not document a link between prenatal SLEs and infant temperament. Interestingly, Boyd and colleagues¹⁰⁹ examined the associations of both negative and positive life events experienced in the past year, assessed at pregnancy and 3 months postpartum, with infant behavior. When including both negative and positive life events in a regression model, only positive life events positively predicted more infant interaction with their mother¹⁰⁹. With the exception of Lin and colleagues' study¹⁰⁵, which comprised a 100% Mexican American sample, all studies discussed in this section were conducted with diverse, majority racially/ethnically minoritized, low-income, urban samples.

Trauma exposure. A large portion of studies identified in our review strategy focused on the association of maternal exposure to trauma and child socioemotional and/or behavioral development. Measures of trauma included intimate partner violence, adverse childhood experiences (ACEs), child maltreatment and childhood sexual abuse. Most studies^{100,110–116} but not all^{106,117–120} documented associations between maternal trauma in the preconception and prenatal periods and offspring mental and behavioral health or early indicators of mental health.

Many of these studies^{110,111,115,121–125} identified mediators or exacerbating moderators influencing the relationship between maternal trauma exposure and child outcomes; specifically child hypothalamic–pituitary–adrenal axis and sympathetic nervous system functioning, child trauma and maltreatment exposure, poorer maternal mental health and mental state, child emotion dysregulation and poorer parent–child communication. Family social support mitigated the effects of maternal ACEs on children's externalizing behaviors in a sample of African American mothers¹²⁶.

Sociocultural factors. Several studies investigated the influence of sociocultural factors, such as discrimination, acculturation, acculturative stress and neighborhood ethnic concentration. Multiple studies of mothers identifying as Black or Latina documented associations between more maternal experiences of discrimination and negative emotionality^{21,127} and lower infant attention in the context of a stressor¹¹⁸; in some cases these relations were mediated or explained by the association of discrimination with maternal mental health and distress in pregnancy^{118,127}. Acculturative stress during pregnancy also predicted negative infant emotionality in a sample of Black and Latina women²¹. Examination of temperament profiles among infants of Mexican American mothers in conjunction with behavioral dysregulation and mother's acculturation levels revealed that one specific profile (negative reactive, low regulated) was related to more behavioral dysregulation, only for infants of mothers low in Anglo orientation or high in Mexican orientation¹²⁸. Finally, higher neighborhood Latinx concentration was associated with Mexican American mothers' stronger beliefs that they would find fulfillment in their role as a mother and fewer postpartum depression symptoms, which in turn predicted fewer child behavior problems¹²⁹.

Pathway 5

The next section of this scoping review focuses on research examining pathway 5 of Fig. 1, that is, evidence that connects maternal psychological and behavioral characteristics, along with physical health and biological mechanisms, to child mental health.

Maternal psychological and behavioral characteristics. Most but not all¹⁰⁹ studies document support for associations between maternal pre- or postpartum depression and challenges in socioemotional development among infants¹³⁰, toddlers^{131,132} and older children^{98,133,134}. Edwards and Hans¹³⁵ examined possible mediators of this association, finding that lower postpartum maternal sensitivity (that is, responding appropriately to infant cues and distress) and more maternal depressive symptoms explained the association between prenatal depression and toddler behavioral problems in their sample.

Beyond depression and maternal sensitivity, several studies focused on a broader single construct or multiple constructs of maternal psychological distress. Prenatally, higher maternal cyclothymic personality scores, lower maternal–fetal attachment, more stress and psychological distress, and poorer maternal mental health predicted socioemotional problems in children^{101,102,133,136–138}, lower surgency and self-regulation among infants¹⁰⁸, and poorer infant attention in the context of a stressor¹¹⁸. In a sample of Latina women, pregnancy anxiety was linked to higher child negative affect, particularly among Latina women who preferred Spanish over English¹³⁹.

In the postpartum period, parenting stress, depression and psychological distress were linked to poorer child mental health^{101–103,107,130,134,138}.

In a sample of Black women, no association was found between the level of pregnancy wantedness assessed during pregnancy and the infant's maternal attachment security¹⁴⁰. Last, two studies examined maternal post-traumatic stress symptoms in racially/ethnically diverse samples, with one study¹³⁰ finding that prenatal and postpartum post-traumatic stress symptoms did not predict infant socioemotional development, and another study¹¹⁷ showing that maternal post-traumatic stress symptoms assessed postpartum increased the risk for infants' insecure attachment at 13 months¹¹⁷.

Maternal substance use. A large number of studies examined the impact of maternal substance use on offspring socioemotional and behavioral development. Studies overwhelmingly found associations between prenatal cocaine^{141–149} and tobacco and/or nicotine use^{114,136,137,147,150–160} and child outcomes, including psychopathology, behavioral regulation problems, aggression and increased substance use. A much smaller number of studies^{161–165} found no relation between prenatal cocaine exposure and later child behavioral and mental health. There were almost no studies on prenatal methamphetamine^{166,167} or heroin¹⁶⁸ exposure.

The research on prenatal alcohol use was mixed, with only about 50% of studies finding evidence that prenatal alcohol use predicted offspring behavioral and child mental health, including greater infant negative affect¹⁶⁹, persistent drinking later in life¹⁷⁰, and more externalizing and internalizing problems^{142,171–175}.

A subset of studies^{141,176–182} examined links between prenatal drug use and developmental outcomes in children but did not specify any one particular substance, making it difficult to draw any specific conclusions regarding their findings.

Maternal physical health and biological mechanisms. The research on maternal physical health includes examinations of metabolic disorders, infection, inflammation, vitamin D levels and age at childbirth. First, related to infections, in a sample of white and African American participants, herpes simplex virus 2 infection during pregnancy predicted an increased risk of offspring psychoses in adulthood¹⁸³, while prenatal human immunodeficiency virus exposure was related to a higher likelihood of attempting suicide in an urban sample of Black, Latinx and mixed-race youth¹⁸⁴. Camerota et al.¹⁸⁵ examined prenatal inflammation, as measured by plasma levels of C-reactive protein and pro-inflammatory cytokines (for example, interleukin-6 and tumor necrosis factor), among African American women and found a positive association between tumor necrosis factor and infant negative affect.

Of the research examining prenatal vitamin D levels, studies showed that lower prenatal vitamin D levels predicted higher levels of externalizing behavior¹⁸⁶ and an increased risk of ADHD, particularly among males¹⁸⁷. Chawla and colleagues¹⁸⁸ found that lower prenatal maternal vitamin D levels were linked to more internalizing symptoms among white children and fewer among Black and Hispanic children. Lower prenatal maternal vitamin D was also associated with higher dysregulation scores in Hispanic children¹⁸⁸. It is important to note that these race/ethnic group differences in associations are difficult to interpret because Black women exhibited strikingly lower levels of

vitamin D than Hispanic and white women, and Hispanic women had slightly lower levels than white women.

Shifting to markers of metabolic disorders, two studies^{189,190} showed evidence for higher prepregnancy weight and offspring behavioral problems. However, Tanda and Salsberry¹⁹⁰ only observed this relation for children of white women and not for children of African American women. Last, an examination of associations between pregestational and gestational diabetes and child internalizing and externalizing problems observed no link¹⁹¹.

Regarding age at childbirth, one study¹⁹² of a racially/ethnically diverse sample identified an increased risk of learning disability for children whose mothers were aged 18–24 years old or 35–39 years old when they gave birth (a U-shaped association). Children of mothers who were between 18 and 24 years old when they gave birth were also at increased risk of ADHD; however, children whose mothers were between 35 and 39 years old had decreased risk¹⁹². Russotti and colleagues¹²⁵ examined age at childbirth in relation to child internalizing symptoms in a predominantly Black sample, finding that adolescent childbirth heightened risk. Notably, this relation was mediated by chronic childhood maltreatment¹²⁵. Another study¹⁴⁵, also with a predominantly Black sample, found no relation between maternal age at birth and developmental trajectories of externalizing behavior.

Finally, in an effort to further understand the molecular mechanisms linking maternal prenatal stress to fetal neurodevelopment, Aushev and colleagues¹⁹³ examined transcriptome-wide profiles of the placenta in relation to psychosocial stress during pregnancy and infant temperament in a racially/ethnically diverse sample. They identified multiple placental co-expression modules associated with both maternal stress and infant regulatory capacity, supporting the possibility that maternal prenatal stress may influence fetal neurodevelopment through transcriptional regulation of gene expression¹⁹³.

Pathway 6

Among studies investigating whether birth phenotype predicted child emotional and behavioral characteristics in REMD populations, two studies with racially/ethnically diverse samples documented that preterm birth was associated with more infant negative affect (mediated by elevated maternal postpartum depressive symptoms)¹⁹⁴ and toddler behavioral dysregulation¹⁷⁸. Several other studies, also with racially/ethnically diverse samples, did not identify any relation between birth weight or gestational age and infant temperament¹⁹⁵ or child emotional and behavioral problems^{98,103,145}.

Discussion

The goal of this scoping review was to present an integrated conceptual model of racism-related developmental origins of mental health and an overview of the evidence supporting it. The model (Fig. 1) highlights how centuries of discriminatory policies in the USA contributed to the creation of societal structures and norms that continue to marginalize and oppress REMD populations in the present day (pathway 1). In turn, these societal conditions increase the likelihood of exposure to stress and adversity among REMD populations, disrupting stress-response systems and elevating risk for stress-related diseases (pathway 2) and adverse birth outcomes (pathway 3). Finally, the combination of parental and offspring exposure to stressful experiences, pre- and postnatal maternal health challenges and adverse birth outcomes can increase the vulnerability of offspring to mental illness (pathways 4–6). Model pathways 1, 2 and 3 are supported by existing reviews^{22,60,72}. Therefore, this analysis focused on literature examining pathways 4, 5 and 6—specifically, the associations of maternal exposure to stressful experiences and environments, maternal health challenges (physical, mental and behavioral), and adverse birth outcomes with offspring mental health among REMD populations.

Our model and review results underscore the significance of traumatic life events and SLEs as a key exposure category. Structural racism contributes to conditions that elevate the likelihood of REMD

populations encountering a diverse range of stressors, including those that may not immediately appear related to racism. For instance, adverse childhood experiences, or ACEs, historically conceptualized as ten potentially traumatic childhood events, have consistently been linked to long-term physical and mental health outcomes in adulthood and with early mortality^{196–198}. One ACE is incarceration of a family member. This is an example of how structural racism disproportionately burdens racially/ethnically minoritized communities with ACEs^{13–15,199–202}, given that Black and Latinx individuals are at greater risk of having a relative incarcerated due to discriminatory legal and law enforcement policies and practices^{59,61}.

In this review, a large number of studies examined preconception or prenatal exposure to traumatic experiences (such as intimate partner violence, ACEs, child maltreatment and childhood sexual abuse) and stressful life events, or SLEs (such as major personal illness, troubles at work, relationship problems, housing difficulties, legal issues or financial problems). These studies^{100,105–107,110–116} overwhelmingly found evidence for associations to child mental health. There was evidence linking SLEs during pregnancy to internalizing and externalizing symptoms in offspring^{105–107}, but no studies in this review examined preconception exposure to SLEs. Notably, this was not the case for maternal trauma experiences, as several studies^{100,111–116} documented associations between maternal exposure to traumatic events in their childhood and offspring outcomes. This finding suggests that timing is an important factor and pregnancy may be a susceptible time for the influence of SLEs on offspring outcomes. Also important to acknowledge is that many widely used measures of stress and trauma, such as the ACEs scale, may not adequately capture the unique adversities disproportionately experienced by REMD groups^{200,202–204}. The development of more inclusive and culturally responsive measures will be essential for advancing research in this area.

Racially/ethnically minoritized individuals are also disproportionately exposed to environmental toxins and socioeconomic hardship, shaped by factors like discrimination and historical segregation^{60,63,64,72}. The findings of this review show that socioeconomic disadvantage, conceptualized by different studies as household density, food insecurity, neighborhood stress, material hardship, inadequate family income and lower maternal education, was linked to an increased risk of mental health problems in children^{98–104}. Prenatal exposure to toxins, such as BPA, phthalates, PAHs, nitrogen dioxide and particulate matter, among REMD populations is also associated with an increased risk of mental health problems in children^{84,85,87–91,94}; in particular, the effects of BPA and phthalates exposure may be sex differentiated^{84,87,89,91}.

Only in recent years (2018 and later) have researchers begun to examine another category of stressors highlighted in our model: maternal exposure to sociocultural factors, such as discrimination, acculturation, acculturative stress and neighborhood ethnic concentration, in relation to child mental health. Historically, acculturation and acculturative stress have largely been researched in immigrant populations; however, a smaller body of research shows that US-born REMD groups also encounter these processes as they navigate and adapt to dominant Euro-American cultural norms^{205,206}. In the existing studies^{21,118,127} covered by this review, discrimination and acculturative stress emerged as key maternal risk factors for child mental health. Nascent research also suggests acculturation and neighborhood ethnic concentration are important considerations, with Lin and colleagues¹²⁸ finding that, for Mexican American mothers and their infants, mothers' Mexican orientation appeared to confer infant temperament risk, while Anglo orientation mitigated risk. In addition, Curci et al.¹²⁹ found higher neighborhood concentration of participants' own ethnicity was linked to fewer child behavioral problems. Going forward, researchers must continue to expand their study of sociocultural stressors, including a focus on other forms of racism-related stress and adversity that are emerging as critical to health, such as vicarious racism and police violence²².

Pathway 5 of our model proposes that maternal mental, behavioral and physical health consequences of exposure to stress and adversity are additional factors influencing offspring vulnerability to mental illness^{69,71}. The results of our review consistently linked maternal mental health, especially depression, to child mental health^{98,101–103,107,117,130–138,207}. However, most research studies examined depression in the postpartum period, and more research is needed on preconception and prenatal depression.

Studies examining maternal substance use largely focused on cocaine, alcohol, marijuana and tobacco. Consistently, prenatal cocaine, marijuana and tobacco use were associated with child mental health challenges^{98,114,136,137,141–160,208–213}. These findings are generally in line with literature examining the effects of prenatal substance use on child developmental outcomes in broader populations²¹⁴. Notably, the proportion of studies in this scoping review focused on substance use (about 44%) as opposed to other risk factors, such as the percentage of studies examining prenatal mental health (about 18%), is striking. Historically, REMD groups in the USA have been targeted and stereotyped by media coverage, rhetoric and policies as the primary users of illegal substances²¹⁵, and the large proportion of studies focused on prenatal substance use in REMD populations highlights a potential research bias and an example of how racism and stereotyping can become systemically embedded. Importantly, some REMD groups do face disparities in substance use, which has been tied to structural racism and historical trauma^{215,216} that has resulted in challenges such as limited access to substance use prevention and treatment resources. Further, research has shown that chronic stress from discrimination may increase the likelihood of substance use as a coping mechanism²¹⁵.

An important consideration recently highlighted by researchers is the need to consider certain common co-occurring risk factors in the study of prenatal substance use, many of which disproportionately impact REMD populations, including higher risk for exposure to family and community violence, caregiver psychological distress and trauma experiences, and disparities related to income, race and geographical location²¹⁴. A recent study²¹⁷ of 9,838 children in the Adolescent Brain Cognitive Development cohort found that when adjusting for environmental (birth, familial and societal risk factors) and genetic contexts (family histories and polygenic risk scores of mental disorder), associations between prenatal tobacco, marijuana and alcohol use and child outcomes were all reduced.

A limited number of studies examined maternal physical characteristics, health and biology with child mental health outcomes. A few preliminary studies^{183–185} suggested that prenatal infection is linked to child temperament or later mental health. In addition, the majority of research looking at prenatal vitamin D levels and higher prepregnancy weight identified associations with child internalizing and externalizing symptoms^{186–188}. However, one of these studies¹⁸⁸ found racial/ethnic differences, with racial/ethnically minoritized youth not having the same negative associations, highlighting an important area for further research. One unique study¹⁹³ identified one placental gene co-expression module that was positively associated with maternal stress and negatively associated with infant regulatory capacity, bringing attention to a potential mechanism for how maternal stress directly impacts infant development. More studies are needed in this area, focusing on mechanisms through which maternal exposure to stressors shapes fetal development.

Finally, there were only a small number of studies^{22,68,218} that included birth phenotype, which is presented in pathway 6 of our model as an additional pathway linking disproportionate maternal stress exposure and child mental health outcomes. These studies^{98,103,145,178,194,195} reported contradictory findings, highlighting another area where more research is needed.

In sum, this scoping review provided supporting evidence for each area of our integrated conceptual model of racism-related developmental origins of mental health that had not already benefited

from recent review^{22,60,72}. Some components of the model have more empirical support than others; for example, there is an overwhelming amount of research on prenatal substance use. Areas where more research is needed include sociocultural stressors and maternal physical health, and research focused on social, behavioral and biological mechanisms of intergenerational transmission. Although some studies did identify mediators and moderators of associations, only a small number identified biological mediators, including placental gene co-expression modules, the hypothalamic–pituitary–adrenal axis and sympathetic nervous system function^{122,193}. Studies that identified moderators illustrate how associations between stressors in the model are not linear; rather, stressors tend to co-occur and exacerbate one another. For example, Perera and colleagues^{88,90} found that material hardship exacerbated associations between prenatal PAH exposure and later ADHD symptoms. Some studies examined and identified race as a moderator⁹³. However, it is crucial to highlight the strong and growing scholarly stance not to examine race as a predictor of health outcomes, given that it is a social construct merely serving as a proxy for other factors predictive of health, such as discrimination. Using race as a predictor of health diverts research attention from the underlying issues and alternative explanations for observed findings^{10,219,220}.

Other areas highlighted by the existing research as requiring further study include timing of exposure, timing of mental health assessment and sex differences. Most studies in this review examined prenatal or early postpartum risk factors, with fewer looking at exposures in preconception. However, studies that did examine preconception risk factors found important links to offspring outcomes. In addition, although many studies found that associations between maternal risk factors and child mental health outcomes were dependent on child age, no clear single pattern emerged. This was the same case for sex differences, as they were identified in multiple studies but without a consistent trend. For example, several studies^{84,87,89,91} found that, in boys, higher prenatal BPA exposure was linked to increased externalizing and internalizing symptoms, with fewer behavioral and emotional problems among girls. Other studies^{147,148} found that girls with prenatal cocaine exposure were more susceptible to behavioral problems than boys. Many studies did not test for sex-differentiated associations.

Finally, very few studies in this review examined protective factors. Hatch and colleagues¹²⁶ report that family social support buffered the effect of maternal ACEs on children's externalizing behaviors, while we²¹ identified parenting self-efficacy as a buffer of associations between maternal acculturative stress and infant negative temperament. Ahmad and colleagues¹⁰⁰ found that maternal parenting knowledge mitigated that relation between family socioeconomic disadvantage and child socioemotional behavioral problems. These findings all point to potential targets for intervention and prevention and highlight the need for further research on protective factors.

Limitations

This review has several limitations. First, the search strategy was restricted to two databases (PubMed and PsycINFO). As the initial searches yielded a high number of eligible studies, we did not broaden our search to additional databases, which may have excluded relevant studies indexed elsewhere or in the gray literature. Second, although we sought to capture studies involving any racially or ethnically minoritized populations through the use of broad descriptors and commonly reported categories, we recognize that not every specific racial or ethnic group was explicitly listed. As such, there is a possibility that some relevant studies may not have been retrieved by our search terms. Finally, although some experiences of racism are shared across REMD populations, others may be unique to specific groups. However, the available evidence did not allow us to draw group-specific or comparative conclusions, as relatively few studies focused exclusively on a single racial/ethnic group or provided systematic comparisons across groups. Rather, this review represents a first step in acknowledging that

REMD populations as a whole are uniquely exposed to racism-related stressors and in mapping the evidence to date. As the field advances and the literature grows, future research will be able to examine the unique experiences of different racial/ethnic groups.

Conclusion

This scoping review provides the most comprehensive synthesis so far of evidence linking racism-related exposures in the preconception, prenatal and postpartum periods to offspring mental health outcomes across developmental stages. Most existing research has attributed the mental health disparities faced by REMD individuals to the intersection of unique stressors arising from racism, discrimination, trauma and barriers to treatment. The first goal of the model and supporting scoping review presented in this analysis is to highlight another potential driver of these disparities that begins before birth: racism-related developmental origins of psychopathology. Studies covered here show how preconception, prenatal and early-life experiences, particularly disproportionate exposure to stress and trauma, can impact the mental health vulnerability of REMD individuals. Recognizing these developmental origins adds critical context as to how disparities emerge. The results also underscore the need for further research on sociocultural stressors, timing of exposures and outcomes, sex differences, and the mediators and moderators of racism-related developmental origins of psychopathology. This is a tall order as structural inequities impede research progress in this area of study. Scholars studying REMD populations face disparities in funding and higher rates of manuscript rejection, with their work often relegated to ‘specialty’ journals. In contrast, research on predominantly white populations is more likely to be published in higher-impact outlets²²¹. Addressing these barriers is essential to ensure that future scholarship fully reflects the lived realities of all populations and contributes to reducing persistent mental health disparities.

Data availability

All data supporting the findings are derived from previously published studies and are cited within the article. Extracted data supporting the synthesis are included in the article and its Supplementary Information.

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Additional information

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