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Toward birthing liberation: An analysis of structural racism and its implications for perinatal mental health

By

Mahader Tamene

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Epidemiology

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

Professor Mahasin Mujahid, Chair
Professor Jennifer Ahern
Associate Professor Patrick Bradshaw
Professor David Harding

Summer 2024

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by

Mahader Tamene

Abstract

Toward birthing liberation: An analysis of structural racism and its implications for perinatal mental health

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Mahader Tamene

Doctor of Philosophy in Epidemiology

University of California, Berkeley

Professor Mahasin Mujahid, Chair

Background: Perinatal mental disorders (PMD), including perinatal mood and anxiety disorders (PMAD) and serious mental illness (SMI) are a major public health issue in the United States. Studies find racial and ethnic disparities in these disorders, including in diagnosis and access to adequate and quality mental healthcare. These disorders are linked to adverse pregnancy outcomes and impaired child development. While racial and ethnic disparities in PMD and access to quality mental healthcare have been documented, evidence on the prevalence of PMD by race is mixed, highlighting the need for further research. Moreover, limited research has empirically investigated the association of structural racism on these disparities.

Methods: This dissertation aims to evaluate the trends and racial and ethnic disparities in PMD and examine and the relationship between structural racism and these outcomes among a diverse cohort in California. The first paper investigates trends and racial disparities in PMD. The second paper incorporates a systematic review of empirical studies on structural racism and mental health/substance use outcomes. The final paper empirically examines the association between structural racism, measured by the index of concentration at the extremes (ICE), and PMD.

Conclusion: Leveraging California state-wide data on over 10 million births spanning 20 years (1997-2018), this dissertation seeks to advance knowledge on the racial disparities and drivers of PMD. Understanding the impact of structural racism on perinatal mental health is crucial for identifying underlying causes and informing effective, multi-sectoral public health interventions and population-level policies aimed at eliminating these inequities and ultimately improving the mental health of birthing populations in this country.

Dedication

To the original Dr. Tamene. Thank you for inspiring me in all the ways. I miss you tremendously. I hope I continue making you proud. I love you more than I can say.

Table of Contents

List of Tables and Figures iii

Acknowledgements v

Introduction 1

Chapter 1. Trends and racial disparities in hospital-diagnosed perinatal mental disorders in California 4

- Abstract 4
- Methods 6
- Results 7
- Discussion 8
- Tables and Figures 13

Chapter 2. Toward quantifying and measuring the impact of structural racism on mental health inequities: a systematic review of the public health literature 21

- Abstract 21
- Introduction 22
- Methods 24
- Results 25
- Discussion 28
- Conclusion 32
- Tables and Figures 33

Chapter 3. Structural racism and perinatal mental health – The role of racialized economic segregation 43

- Abstract 43
- Introduction 44
- Methods 45
- Results 47
- Discussion 49
- Limitations 51
- Conclusion 51
- Tables and Figures 52

Conclusion 60

References 61

List of Tables and Figures

List of Tables

Chapter 1

Table 1. Prevalence (per 1,000 births) in PMD[a] (Any, PMAD[b] and SMI[c]), California, 1997-2018 14
Table 2. Racial differences in PMD[a] (Any, PMAD[b], and SMI[c]), California, 1997-2018 15
Table 3. Prevalence of PMD[a] (Any, PMAD[b], and SMI[c]) in 1997 and 2018 and percent change from 1997-2018, California 16

Supplement Table 1. ICD-9-CM and ICD-10-CM codes for perinatal mood and anxiety disorders (PMAD) and serious mental illness (SMI) 19
Supplement Table 2. Sensitivity Analysis comparing prevalence of PMD (Any, PMAD, SMI), California, 1997-2018 vs 1997 – 2015 20

Chapter 2

Table 1. Summary of included studies 34
Table 2. Conceptualization and measurement of structural racism 35
Table 3. Results of individual studies 37

Supplement Table 1. Definitions of structural racism 41
Supplement Table 2. Summary of study limitations 42

Chapter 3

Table 1. Distribution of population characteristics among racial/ethnic groups by any PMD in California, 1997-2018 52
Table 2. Associations between ICE race-income and overall perinatal mental health status, California, 1997-2018 53

Supplement Table 1. ICD-9-CM and ICD-10-CM codes for PMAD and SMI 55
Supplement Table 2. Distribution of population characteristics among racial and ethnic groups by PMAD status, California, 1997-2018 56
Supplement Table 3. Distribution of population characteristics among racial and ethnic groups by SMI status, California, 1997-2018 57
Supplement Table 4. Associations between ICE income and overall perinatal mental health status, 1997-2018 58
Supplement Table 5. Associations between ICE race-ethnicity and overall perinatal mental status, 1997-2018 59

List of Figures

Chapter 1

Figure 1. Analytical sample selection, California, 1997-2018 13
Figure 2. A-C: Prevalence of perinatal mental disorders, 1997 - 2018, overall (PMD) (Fig. 2A) and by category (PMAD, SMI) (Fig. 2B, 2C) across racial and ethnic categories 17
Figure 3. A-C: Trends in racial and ethnic disparities of perinatal mental disorders, overall (Fig. 3A) and by category (PMAD, SMI) (Fig. 3B, 3C) 18

Chapter 2

Figure 1. PRISMA Flow Diagram 33

Chapter 3

Figure 1. Analytical sample selection, California, 1997-2018 52

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"Rarely, if ever, are any of us healed in isolation. Healing is an act of communion." – bell hooks

To name the expansiveness of everyone that sits here would take longer than this dissertation but suffice it to say I am ONLY here because of the love, compassion and grace I've received from my village. There are no words adequate to describe my gratitude for the ways I have been held, loved, nurtured, heard and seen throughout this journey.

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Introduction

Perinatal mental disorders (PMD), including perinatal mood and anxiety disorders (PMAD) and serious mental illness (SMI), represent a pressing public health issue in the United States. According to CDC estimates, these conditions are a leading cause of preventable pregnancy-related deaths through pregnancy and in the first year postpartum (Trost et al., 2022). PMAD, which include depression and anxiety, are some of the most common morbidities in the perinatal period, with estimates ranging from 5 to 25% (Gavin et al., 2005). SMI, encompassing bipolar and psychotic disorders substantially impairs functioning for birthing persons (Kalra et al., 2022).

These disorders have deleterious short and long-term consequences for birthing persons, leading to adverse pregnancy outcomes such as miscarriage, preterm birth, and low-birth weight (Fawcett et al., 2019; Grote et al., 2010; Lopez et al., 2019). The effects of these conditions can have devastating consequences not only for the birthing person, but also for their offspring. For example, postnatal depression among birthing people has been associated with adverse childhood outcomes including impaired growth, behavior, and cognitive development (Grace et al., 2003; Lopez et al., 2019). The economic cost on the healthcare system is also significant, with higher healthcare expenditures for delivery hospitalizations among birthing persons with mental health disorders, amounting to an estimated $102 million annually (T. H. Brown & Homan, 2023; Luca et al., 2020).

While evidence on the prevalence of PMD by race is mixed, several studies have reported racial disparities. Studies show that minoritized birthing persons, including Black, Hispanic, American Indian, and Alaska Native individuals, often exhibit higher prevalences of these disorders compared to their white counterparts (Chan et al., 2021; Conteh, 2022; Howell et al., 2005; Liu & Tronick, 2013; Mukherjee et al., 2016; Sujan et al., 2023). For example, a cohort study of pregnant Bostonians found that prevalence of depressive symptoms was higher among Black and Hispanic pregnant persons compared to non-Hispanic white birthing persons(Rich-Edwards, 2006). Furthermore, Black birthing persons experience higher rates of unmet mental health needs compared to their White peers, with studies finding they are less likely to be screened for postpartum depression (Hall et al., 2023; Sidebottom et al., 2021) and less likely to be offered treatment, including medications and continued care (Karvonen et al., 2022; Kozhimannil et al., 2011). However, some studies report no significant racial differences or even higher prevalence among white birthing persons (Fridman et al., 2014; Kim et al., 2020). More research is needed to assess racial and ethnic differences in perinatal mental health.

Beyond documenting racial disparities in PMD is the need to understand the drivers of these disparities, including structural racism. Racism is an organized system that categorizes and ranks people into racial groups based on an ideology of inferiority that devalues, disempowers and differentially allocates resources and opportunities to racial groups deemed inferior (Williams et al., 2019). It can manifest in multiple forms, including through interpersonal interactions (i.e., negative actions/attitudes or statements toward a different race) and structural forces. Structural racism specifically refers to the macrolevel systems, social forces, policies, and institutional practices that produce and mutually reinforce racial and ethnic inequities in the US (C. P. Jones,

2000; Williams & Mohammed, 2013). Evidence of these persistent inequities rooted in a system of oppression can be found in multiple domains including in housing, employment, education, earnings, benefits, credit, media, health care, criminal justice, etc. (Bailey et al., 2017).

Manifestations of structural racism within each of these domains can directly impact health by acting as a chronic psychosocial stressor that becomes biologically embodied, and can lead to accelerating aging, and adverse mental and physical health outcomes (Levine & Crimmins, 2014; Smith-Bynum et al., 2014; Williams et al., 2003; Williams & Mohammed, 2013). It can also harm health by limiting access to critical resources and power (e.g., employment, safe housing, living environment, quality healthcare, education, etc.). Other pathways include excess exposure to toxins and pathogens; trauma; and health risk behaviors in response to discrimination (Krieger, 2001).

Considering the multifaceted and complex nature of structural racism, it is essential to develop and understand different empirical methods for measuring it. Traditional individual-level measures of racial discrimination are limited in capturing the broader context of structural racism. Improved measurement techniques are essential for accurately assessing the impact of structural racism on health (Adkins-Jackson et al., 2022; Hardeman et al., 2022). While growing literature has documented this structural racism-health association more broadly, limited work to date, has empirically investigated how the manifestations of structural racism may influence PMD.

To address the gaps in literature mentioned above, this dissertation leverages a large population-based dataset of over 11 million live hospital births and hospitals discharge records to evaluate trends and racial disparities in PMD (PMAD, SMI, and their combination, any PMD) and to examine if and how structural racism is associated with these outcomes.

Chapter one examines trends and racial disparities in PMD in California from 1997 – 2018. We used International Classification of Diseases, Ninth & Tenth Revision, Clinical Modification (ICD-9-CM and ICD-10-CM) hospital discharge diagnostic codes from the California Department of Health Care Access and Information (HCAI) that were relevant for PMAD and SMI diagnoses over the perinatal period (i.e. course of pregnancy and 1-year postpartum). We computed prevalence of PMD by race and ethnicity and examined racial and ethnic disparities in outcomes and trends using logistic regression.

Chapter two is a systematic review examining empirical papers on the relationship between structural racism and mental health/substance use outcomes. We updated and expanded on previous findings, examining how recent studies conceptualize and measure structural racism, and whether these decisions were informed by social theory. We also reviewed the various methods used to evaluate the mental health and substance use consequences of structural racism. Finally, we concluded by offering recommendations for future directions, including interventions aimed at reducing racism and its effects.

Chapter three empirically estimates the association between a measure of structural racism and PMD. Using the same data and outcome definitions from Chapter 2, we examined the index of concentration at the extremes (ICE), a measure of spatial social polarization, as a proxy for

structural racism in relation to PMD. We calculated ICE measures using census data at the census tract level and categorized from least to most polarized based on racialized, economic, and a combination of racialized and economic polarization. Race and ethnicity-stratified mixed effects log-binomial models estimated the risk of PMD, adjusting for maternal age, education, and insurance and accounting for clustering by census tract.

This dissertation aims to advance knowledge on the racial disparities and drivers of PMD. Understanding the root causes of longstanding and persistent racial health inequities, such as structural racism, is imperative to informing effective, multi-sectoral public health interventions and population-level policies aimed at eliminating these inequities and ultimately improving the mental health of birthing populations in this country. This work hopes to add to existing knowledge to move to effective and actionable change.

Note about language: This dissertation utilizes both gendered terms (e.g., "maternal" and "women") and gender-inclusive terms (e.g., "birthing people") interchangeably, acknowledging that not all who experience childbirth identify as women. The addition of gender-inclusive language is meant to recognize any conversation and research on reproductive health must also include trans, gender nonconforming, and nonbinary persons (Pezaro et al., 2024).

Chapter 1. Trends and racial disparities in hospital-diagnosed perinatal mental disorders in California

Abstract

Background: Perinatal mental disorders (PMD), including perinatal mood and anxiety disorders (PMAD) and serious mental illness (SMI), pose a critical public health concern. Untreated, these disorders have short and long-term consequences for birthing persons and their children. Evidence of racial disparities in PMD is mixed, and little is known about whether these disparities are changing over time.

Objective: To address these gaps, we evaluated trends and racial and ethnic disparities in the prevalence of hospital-diagnosed PMD (PMAD, SMI, and any PMD as a composite of both).

Methods: We used a population-based cohort of all live singleton and first recorded non-singleton hospital births in California delivered ≥ 20 weeks gestation from 1997 – 2018 (N= 10,509,675). PMD outcomes, including PMAD and SMI were identified using ICD-9-CM and ICD-10-CM codes captured in hospital discharge records through the perinatal period. We computed prevalence of PMAD, SMI and any PMD by race and ethnicity over time and examined racial and ethnic disparities in outcomes and trends using log binomial models.

Results: Hospital-based PMD diagnoses occurred in 18.9 per 1,000 births, with PMAD occurring more frequently than SMI (16.7 and 5.0 per 1,000 births, respectively). The prevalence of hospital-diagnosed PMD increased across all racial and ethnic groups over time, with Hispanic birthing persons experiencing the largest increase. While there was no clear evidence of racial disparities in hospital-diagnosed PMAD, there were indications of such disparities in hospital-diagnosed SMI. Compared to non-Hispanic white birthing persons, Black birthing persons experience elevated risk of hospital-based SMI diagnosis (PR: 1.15, 95% CI: 1.12, 1.18). This relative disparity varied over time, with PR of hospital-based SMI diagnoses increasing from 1997-2001 and 2008-2014 but decreasing from 2001-2008 and 2014-2016.

Conclusion: These findings may be capturing real differences in outcome, ascertainment bias, differential access to adequate mental health care, racial bias in diagnosis, and stigma. Future research should investigate the social and structural drivers of racialized differences in perinatal mental illness diagnoses.

Introduction

Perinatal mental disorders (PMD) pose a critical U.S. public health concern. These disorders, including perinatal mood and anxiety disorders (PMAD) and serious mental illness (SMI), have deleterious short and long-term effects on birthing persons, including adverse pregnancy outcomes such as miscarriage, preterm birth, and low-birth weight (Fawcett et al., 2019; Grote et al., 2010; Lopez et al., 2019). The effects of these conditions can have devastating consequences not only for the birthing person but also for their children. For example, postnatal depression among birthing people has been associated with adverse childhood outcomes, including impaired growth, behavior, and cognitive development (Grace et al., 2003; Lopez et al., 2019).

These disorders are also costly to the healthcare system (C. C. Brown et al., 2021; Luca et al., 2020). One study analyzing hospital discharge data produced by the Agency for Healthcare Research and Quality (AHRQ) found $458 higher costs per delivery hospitalization among birthing persons with mental health disorders relative to their counterparts without mental health disorders, or an estimated $102 million annually (C. C. Brown et al., 2021).

Evidence of U.S. racial disparities in perinatal mental illness is mixed. Several studies have reported higher prevalences among minoritized birthing persons relative to their white counterparts, including Black, American Indian, or Alaska Native (Chan et al., 2021; Conteh, 2022; Howell et al., 2005; Liu & Tronick, 2013; Meredith et al., 2023; Mukherjee et al., 2016; Sujan et al., 2023). For example, a prospective cohort study of pregnant persons in Boston found Black and Hispanic mothers had a higher prevalence of depressive symptoms compared with non-Hispanic white mothers (Rich-Edwards, 2006). Another study conducted by the Centers for Disease Control and Prevention looking at Pregnancy Risk Assessment Monitoring System (PRAMS) data found that non-Hispanic Black, American Indian or Alaska Native, and Asian or Pacific Islander women had a higher risk of postpartum depression symptoms compared to non-Hispanic women (Bauman et al., 2020). Additionally, Black women experienced higher rates of unmet mental health needs, including a longer time to diagnosis (Hall et al., 2023), and were less likely to be offered treatment compared to their white counterparts (Karvonen et al., 2022; Kozhimannil et al., 2011).

Contrary to these findings, other studies report no racial differences in perinatal mental health conditions, and some even find higher risk among white birthing persons. For example, a study in Minnesota looking at adverse childhood experiences of a cohort of pregnant and postpartum women admitted to a dyadic program within a large county hospital found no significant difference in depression and anxiety scores across race/ethnicity (Kim et al., 2020). A California cohort study of maternal comorbidities found that relative to white women, Asian or Pacific Islander women and Hispanic women had lower odds of mental health conditions (Fridman et al., 2014). These findings align with other, more extensive epidemiologic surveys on mental health in the general population that find lower lifetime risk of mood and anxiety disorders among Black persons relative to their white counterparts (Breslau et al., 2005). Variations in methodologies across studies in how perinatal mental illness cases were defined and assessed likely contributes to these varied results (VanderKruik et al., 2017).

Research assessing racial and ethnic differences in PMD, particularly for SMI, remains underexplored. To our knowledge, only one other study examined racial disparities in SMI, finding a higher prevalence among white women compared to their non-white counterparts (McKee et al., 2020). Moreover, temporal changes in racial and ethnic disparities for perinatal mental disorders (including PMAD and SMI) are poorly understood. Thus, to improve understanding of racial and ethnic disparities in maternal mental health, this paper examined racial and ethnic differences in the prevalence of hospital-diagnosed PMD, specifically PMAD and SMI. We also examined changes in these outcomes from 1997 to 2018 overall, and by race and ethnicity, and considered the 20-year trends of racial and ethnic disparities.

Methods

Study population

The study includes the statewide population of all singleton and first recorded hospital live births in California from 1997 to 2018. Data come from the California Department of Health Care Access and Information (HCAI), which linked birth certificate files with hospital discharge records and emergency department visits. We excluded births with implausible gestational age (<20 weeks or >45) (n = 309,986), fetal deaths (n = 31,948) and missing maternal race and ethnicity data (n = 166,171). The final analytic sample was comprised of 10,509,675 births (Figure 1). The State of California Committee for the Protection of Human Subjects and the institutional review boards of Stanford University and the University of California, Berkeley approved the study protocol.

Measures

We defined hospital-diagnosed PMD using the International Classification of Diseases, Ninth & Tenth Revision, Clinical Modification (ICD-9-CM and ICD-10-CM) codes for relevant PMAD and SMI diagnoses occurring at least once any time during the perinatal time period (i.e., pregnancy and up to one-year postpartum). These codes were extracted from the hospital discharge record of each birthing person. Based on the available data, a perinatal hospital diagnosis was defined as happening during either an ER visit, an inpatient hospitalization or an ambulatory surgery hospitalization. Relevant ICD-9-CM and ICD-10-CM codes for PMAD and SMI were based on prior work (McKee et al., 2020) (Supplement Table 1). Experiences of any hospital-diagnosed PMAD, SMI, and any PMD, which was a composite of the previous two diagnoses, were categorized as binary (yes/no).

Self-reported maternal race and ethnicity were collected from the birth certificate and categorized as non-Hispanic white (hereafter, white), non-Hispanic Black (hereafter, Black), non-Hispanic Asian or Pacific Islander (hereafter Asian or Pacific Islander), Hispanic, non-Hispanic American Indian or Alaska Native (hereafter American Indian or Alaska Native), and other (includes multiracial individuals and those that self-identify as other). Due to the limited sample size (N= 6,647 (0.06%)), we only report overall prevalence but do not interpret results or visualize trends for those with a racial and ethnic category of other. We similarly do not visualize trends for American Indian or Alaska Native individuals because of limited cases per year. In line with previous work of our team, we conceptualize race as a proxy for exposure to structural

racism, understanding race as a social construct and acknowledging lack of data on social and structural determinants of health as a limitation of this study (El Ayadi et al., 2024).

Statistical Analysis

We computed the prevalence of PMAD and SMI in the total population and by race and ethnicity from 1997 to 2018. To evaluate disparities in the prevalence of hospital-diagnosed PMD, we estimated the association of race and ethnicity with hospital-diagnosed PMAD, SMI and any PMD using log binomial models, with white birthing persons as the referent group.

We evaluated racial and ethnic differences in the trend of PMD to understand how disparities are changing over time. First, crude annual prevalence of hospital-diagnosed PMAD and SMI were calculated for each racial and ethnic group and plotted from 1997 to 2018. The fold-increase in PMD (any, PMAD and SMI) in 1997 and 2018 was also calculated. To test whether these trends were influenced by the ICD-10 transition, we conducted a sensitivity analysis using data from 1997 to 2015 (transition year) and compared to our main 1997 to 2018 results. Then, prevalence ratios and 95% confidence intervals of hospital-diagnosed PMAD and SMI comparing every racial and ethnic category to white birthing persons were modeled for each year to estimate annual racial and ethnic differences over time. We did not include an adjustment set in our analyses so as not to mask any temporal effects. All analyses were conducted in R 3.8.6 and SAS 9.4.

Results

The 10,509,675 live births comprised 29.4% white, 5.8% Black, 13.4% Asian or Pacific Islander, 50.8% Hispanic, 0.4% American Indian or Alaska Native, and 0.1% other (Table 1). Hospital-based PMD diagnosis occurred in 18.9 per 1,000 births (Table 1). Among the two categories, hospital-diagnosed PMAD had a higher total prevalence at 16.7 per 1,000 births (compared to 5.0 per 1,000 births for SMI). The overall prevalence of hospital-diagnosed PMAD and SMI differed by race/ethnicity (Table 1). American Indian or Alaska Native and white birthing persons had a higher prevalence of hospital-based PMAD diagnosis (29.5 and 28.7 per 1,000 births, respectively), and Hispanic and Asian or Pacific Islander groups had the lowest prevalence of hospital-diagnosed PMAD (11.5 and 8.2 per 1,000 births, respectively). A higher prevalence of hospital-based SMI diagnosis occurred among American Indian or Alaska Native and Black birthing persons (13.0 and 9.7 per 1,000 births, respectively) compared to Hispanic and Asian or Pacific Islander groups that had the lowest prevalence of hospital-based SMI diagnosis (3.2 and 2.2 per 1,000 births, respectively).

Racial and ethnic differences were evident in any hospital-diagnosed PMD, PMAD, and SMI. Black (PR = 0.76, 95% CI = 0.75, 0.78), Asian or Pacific Islander (PR = 0.28, 95% CI = 0.27, 0.28), and Hispanic (PR = 0.39, 95% CI = 0.39, 0.39) birthing people had an associated lower risk of any PMD hospital diagnosis compared to white birthing people (Table 2). Alternatively, American Indian or Alaska Native birthing people had an associated higher risk of any PMD compared to white birthing people (PR = 1.13, 95% CI = 1.08, 1.19). Similar patterns existed for PMAD and SMI, except SMI among Black birthing people, who had an associated 15% elevated risk of SMI compared to their white counterparts (PR = 1.15, 95% CI = 1.12, 1.18) (Table 2).

All hospital-diagnosed PMD prevalence (any, PMAD, SMI) increased over time for all races and ethnicities (Figure 2: A-C). Overall, any PMD prevalence increased by 21-fold, from 2.6 to 55.1 per 1,000 births in 1997 and 2018, respectively (Table 3). While white individuals had the highest prevalence of PMD over time (Figure 2A), the largest fold increase was seen among Hispanic individuals, with a near 35-fold increase from 1997 to 2018 (1.2 to 41.5 per 1,000 births, respectively). PMAD diagnoses also increased over time (27-fold increase), from 1.9 per 1,000 births in 1997 to 51 per 1,000 births in 2018. White birthing persons had the highest prevalence over time (Figure 2B) but the largest fold increase was seen among Hispanic individuals. Increasing SMI prevalence over time was also seen for all racial categories, with a heightened increase starting in 2014 (Figure 2C). Higher prevalence over time was observed among Black birthing persons until 2014, when white birthing persons prevalence increased to that of their Black counterparts. Our sensitivity analysis comparing the fold-increase from 1997 to 2015 to that of our main analysis found that the transition to ICD-10 did not seem to influence the estimated trends (Supplement Table 2).

Results of the relative racial and ethnic differences for any PMD show Asian or Pacific Islander and Hispanic groups exhibit lower prevalence compared to their white counterparts (Figure 3A). There is minimal variation in prevalence ratios over time for Asian and Hispanic birthing persons, with a slight upward trend for Hispanic birthing persons from 2009 onward. Similar patterns are evident in PMAD (Figure 3B). Black birthing people experience greater variability of risk across all outcomes and years (Figures 3A – C).

Discussion

This paper found an overall growing prevalence of hospital-diagnosed PMD, including PMAD and SMI in California from 1997 to 2018. Additionally, we found some evidence of racial and ethnic disparities for both hospital-based PMAD and SMI diagnosis among American Indian or Alaska Native birthing persons compared to white birthing persons. For Black birthing persons, we found some evidence of disparities in hospital-diagnosed SMI, but not for PMAD, although adjustment of sociodemographic variables nullified results; none were evident for PMAD. While there were minimal variations in disparities over time for Asian and Hispanic birthing persons across all hospital-based mental illness diagnostic categories, both increasing and decreasing disparities were seen over time for Black birthing persons compared to their white counterparts.

Our findings of increasing prevalence in hospital-based PMD over time are consistent with other study findings. For example, one study using a nationally representative sample of hospital deliveries found a growing burden of PMAD and SMI in the U.S., with a 120% increase in PMAD and a 93% increase in SMI between 2006-2015 (McKee et al., 2020). Another study examining trends in maternal comorbidities in California reported a 65% increase in the prevalence of mental health conditions in the state between 1999 and 2005 (Fridman et al., 2014). Our study extends these findings by conducting analyses over a longer follow-up period. Our findings were much more pronounced with a 21-fold (2,023%) increase in overall perinatal mental disorders, and 28-fold and 31-fold increase for PMAD and SMI, respectively. Furthermore, previous papers lump perinatal mental health as part of a broader analysis of

maternal morbidity rather than investigating them separately. Given that perinatal mental health is an important contributor to adverse perinatal outcomes, particularly deaths from self-harm, it is imperative to consider its burden independently. According to the CDC, mental health conditions comprise the leading cause of pregnancy-related mortality (Trost et al., 2022). Thus, this paper offers a more comprehensive and focused perspective on trends in the prevalence and disparities of perinatal mental health.

Heightened societal awareness and attention around mental health may contribute to this observed rise (Howard & Khalifeh, 2020). Innovative approaches, including psychiatry referral programs and integration of mental health care services into primary care, have increasingly been deployed to improve the prevention, diagnosis, and treatment of mental health conditions (Griffen et al., 2021). Policies such as the Mental Health Parity and Addiction Equity Act of 2008 (MHPAEA) have funded some of the most significant expansions in mental health coverage (Beronio et al., 2014). Professional and medical organizations have also issued perinatal mental health screening recommendations. For example, the United States Preventive Service Task Force (USPSTF) recommends that all adults, including pregnant and postpartum women, be screened for depression (Siu & (USPSTF), 2016). Similarly, the American Academy of Pediatrics (AAP) recommends incorporating routine postpartum depression screening into early well-child visits (Earls et al., 2019). The American College of Obstetricians and Gynecologists (ACOG) extends these recommendations, suggesting screening for both depression and anxiety be conducted at least once during pregnancy and followed up again postpartum ("Committee Opinion No. 630: Screening for Perinatal Depression," 2015). The increased normalization and affordability of mental health may also enable birthing persons to feel more comfortable endorsing symptoms and seeking care for mental health struggles.

We also extend previous findings through our examination of racial and ethnic disparities over 20 years. Our results found a higher prevalence of hospital-diagnosed PMAD among white birthing persons compared to almost all racial and ethnic individuals, which is consistent with other research (Hall et al., 2023; Kozhimannil et al., 2011). These findings may be capturing real racial and ethnic differences in perinatal mental illness burden. As previous literature has documented, minoritized populations report similar or lower rates of mental disorders compared to their white counterparts, despite facing greater social and economic challenges (McGuire & Miranda, 2008; Thomas Tobin et al., 2022; Williams, 2018). Potential explanations for these findings include stronger social support and self-esteem among minoritized populations, which can mitigate stress (Turner, 2013). As posited by the minority stress model, individual and group level coping mechanisms, including strong racial identity community connectedness can ameliorate the impacts of racialized stressors, such as racism (Frost & Meyer, 2023; LaMotte et al., 2023). Another framework that may explain the 'mental health paradox' is John Henryism, a high-effort coping strategy employed by minoritized populations to combat prolonged exposure to racialized social and environmental stressors. John Henryism has been attributed to lower mental health risk (e.g. lower depressive symptoms) but at the expense of physical health (e.g. increased allostatic load), potentially explaining the often-contradictory findings of lower mental illness among minoritized populations (Robinson & Thomas Tobin, 2021; Williams, 2018).

Given that our outcomes are based on hospital diagnoses, these findings are more likely influenced by factors that contribute to ascertainment bias or differential detection of perinatal

mental disorders rather than actual differences in burden. Perinatal individuals of color face greater structural and social barriers to accessing care compared with their white counterparts (Hall et al., 2023; Karvonen et al., 2022; McKee et al., 2020; Mensah et al., 2021). Even upon accessing care, birthing individuals of color, particularly Black women, are less likely to be screened for perinatal mental illness (Conteh, 2022; Sidebottom et al., 2021). A history of medical discrimination and exploitation has also contributed to a lack of trust and stigma in mental healthcare (Gaston et al., 2016; Hall et al., 2023). For example, one New Jersey cohort study of postpartum patients reported that African-American and Hispanic women were less likely to completely trust their healthcare providers (Howell et al., 2005). Another study exploring the perceived need for mental healthcare among low-income Black and Hispanic pregnant and postpartum women found stigma-related concerns greatly impacted participants' endorsement of need (Nadeem et al., 2009). Stigma may lead to reluctance to discuss symptoms among minoritized perinatal patients, hindering conversations with providers and, in turn, impacting timely diagnosis. Culturally sensitive care, such as using non-stigmatizing language and provider-patient identity concordance, have been suggested as potential strategies to improve medical trust and combat stigma (Hall et al., 2023). Racial differences in severity and length might additionally provide a more comprehensive understanding of the burden of perinatal mental disorders, as previous research has shown severity and chronicity to be higher in Black populations (Sujan et al., 2023; Williams et al., 2007). Thus, even though our study found a higher prevalence of PMAD among White birthing individuals, this may be moderated by symptom severity and study. This should be explored in future research.

Interestingly, and contrary to other minoritized birthing people, the study found American Indian or Alaska Native birthing people had a higher risk of all hospital-diagnosed PMD compared to their white counterparts. Other research has found an increased PMD risk among American Indian or Alaska Native birthing people (Bauman et al., 2020; Meredith et al., 2023). This merits further exploration to understand what distinct experiences of mental health access, perceptions of illness in the medical field, and stigma look like among this population, as this may explain some of these contradictory findings. In this study, Black birthing persons had a higher risk of hospital-based SMI than their white counterparts in unadjusted models. Less research has been conducted on SMI among birthing persons, but findings in other populations reveal an average three-to-four-fold greater risk of SMI among Black persons relative to their white counterparts (R. C. Schwartz, 2014). The increased prevalence of SMI among Black birthing persons within a hospital inpatient setting might be patterning differential access to psychiatric care in unique ways than PMAD, further evidenced by sociodemographic factors such as education and insurance nullifying the increased risk. Inpatient care indicates a high level of acuity of care and should be the last resort for seeking care. Black women are less likely to have adequate access to outpatient community-based healthcare, delaying timely appropriate care (Chan et al., 2021). Thus, while stigma and medical mistrust in mental health care may contribute to lower endorsement of PMAD symptoms, some of the symptoms of delayed and untreated SMI, including psychosis and delusion, may necessitate more immediate care. Given the undoubted importance of perinatal SMI, more research is vital to identifying at-risk women and improving treatments for those affected during this time (I. Jones et al., 2014).

Some evidence also suggests that clinician tendency to over-diagnose schizophrenia and under-diagnose mood disorders in Black populations may be responsible for the racialized differences

in hospital-based SMI diagnosis (Neighbors et al., 2003; Strakowski et al., 1996). This may be due to the inaccurate classification of normative levels of paranoia among Black individuals as indicative of psychopathological risk. Given the long-standing anti-Black racism (e.g. slavery, segregation, racially biased policing, and excessive use of force, etc.) in the United States, Black individuals develop a heightened awareness and suspiciousness of others, independent of mental or physical health status. This "adaptive paranoia" is an appropriate response to individual and structural racism (Mosley et al., 2017). The lack of assessment tools for psychotic disorders to differentiate normative endorsements of paranoia may contribute to these increased diagnostic rates of psychotic disorders (Wolny et al., 2023). Recommendations for future directions include ensuring a more racially diverse sample in measurement testing and integrating measures of perceived discrimination.

Given the impact of PMAD and SMI on the rising chronic conditions among birthing persons (C. C. Brown et al., 2021), quantifying the burden of these illnesses is imperative to informing targeted interventions and policies to improve maternal health outcomes. Developing targeted strategies and interventions is imperative to addressing the burden of perinatal mental illness. Improved screening is essential to the early identification of perinatal mental disorders and has increasingly been adopted not only at the health system level but systematically through state policy. For example, California's AB 2193, the Maternal Mental Health Screening law, which took effect in July 2019, requires obstetric providers to screen for perinatal depression at least once during pregnancy or the postpartum period. These initiatives should expand to include screening for other mental health disorders, including suicidality (C. C. Brown et al., 2021; Orsolini et al., 2016).

Even with policy screening mandates, providers may still be reticent to screen when uncertain about the next steps (Avalos et al., 2022). As such, interventions and policies should go beyond mandating screening to explicitly include processes for early intervention (Yu & Sampson, 2016). Integrated care serves as a promising intervention to improve access to mental health care by bringing services within primary care settings (Griffen et al., 2021). One health system in Denver, serving a quarter of the city, found increased screening and treatment coverage after implementation of its integrated behavioral health program (Lomonaco-Haycraft et al., 2019). Integration can help reduce societal and structural stigma by bringing perinatal mental services into standard care practices.

Limitations

The study has several limitations that should be considered when interpreting results. First, our outcome variables likely underestimated PMD prevalence. We relied on hospital discharge diagnoses during the perinatal period (pregnancy to one year postpartum), missing diagnoses made in primary care or community clinics (Cox et al., 2016; Munk-Olsen et al., 2006). Future research should employ population-based surveys to more closely capture true PMD burden. It is likely, however that our measure of hospital-diagnosed SMI most closely captures true burden, as severity of these diagnoses makes it more likely to be diagnosed within a hospital setting (VanderKruik et al., 2017). One meta-analysis examining the diagnosis of PMAD found that just under a third of birthing persons were accurately diagnosed with postpartum depression. Second,

this analysis did not look at outcomes based on different times of the perinatal period (i.e. during pregnancy and postpartum). Research has found that provider-initiated screening of mental health is more likely to occur postpartum as this period is considered the highest risk for symptoms, including psychosis and mania (Bauman et al., 2020; Howard & Khalifeh, 2020). This occurs despite studies finding that for many birthing persons, onset occurs prior to this period (Wisner et al., 2013). Future research should investigate the timing of the diagnosis to better elucidate the role of early recognition, as this is crucial to preventing future adverse maternal and child outcomes. Finally, our study was restricted to California, potentially limiting the generalizability of our findings. Nevertheless, California represents 1 in 8 births in the U.S. and is one of the largest states in the country; findings from this study can provide broader insight beyond the state.

Conclusion

Our study findings suggest a growing burden of PMD. Trends may be influenced by a variety of factors, including improved diagnostic criteria and increased attention to mental health and wellbeing. Findings of racial differences may be capturing real differences in burden, ascertainment bias, differential access to mental healthcare, racial bias in diagnosis, and stigma.

Future research should look at how structural and institutional factors, such as racism, may play a role in the patterning of perinatal mental disorder diagnosis. Uncovering these drivers of racialized differences in risk may help inform targeted strategies and interventions to address the burden of perinatal mental illness. Interventions aimed at addressing the structural drivers of perinatal mental health are also of critical importance. Pushing for policies such as free childcare, paid parental leave, universal basic income, and transportation assistance seeks to improve the structural conditions of inequity racially minoritized birthing persons must navigate (Foster et al., 2021; Shim & Compton, 2018). Ultimately, to comprehensively address perinatal wellbeing, it is imperative to shift focus toward policy initiatives actively dismantling the structural inequities that drive inequitable outcomes in perinatal mental health.

Tables and Figures

Figure 1. Analytical sample selection, California, 1997-2018 (n=10,509,675)

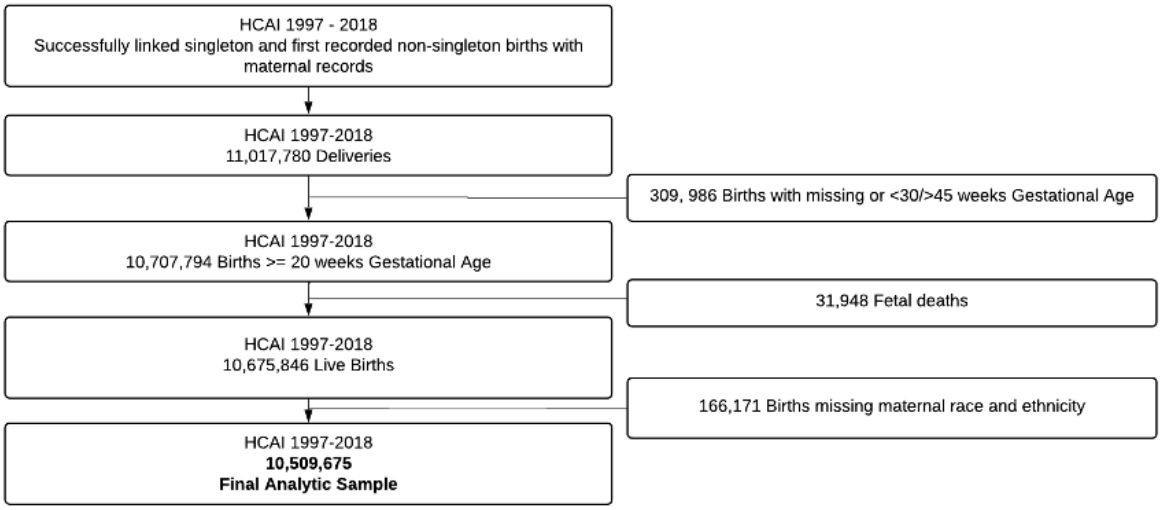


Table 1. Prevalence (per 1,000 births) in PMD[a] (Any, PMAD[b] and SMI[c]), California, 1997-2018

PMD category	White N = 3,090,865 (29.4%)	Black N= 610,304 (5.8%)	Asian or Pacific Islander N = 1,412,557 (13.4%)	Hispanic N = 5,343,414 (50.8%)	American Indian or Alaska Native N = 45,704 (0.4%)	Other[d] N = 6,831 (0.1%)	TOTAL N = 10,509,675
Any PMD	32.7	25.0	9.1	12.7	37.0	23.3	18.9
PMAD	28.7	19.2	8.2	11.5	29.5	18.9	16.7
SMI	8.4	9.7	2.2	3.2	13.0	10.2	5.0

[a] PMD = perinatal mood disorders
[b] PMAD = perinatal mood and anxiety disorders
[c] SMI = serious mental illness
[d] Other category includes multiracial individuals and those that self-identify as other race or ethnicity.

Table 2. Racial differences in PMD[a] (Any, PMAD[b], and SMI[c]), California, 1997-2018 (n = 10,509,675)

PMD category	White N = 3,090,865 (29.4%)	Black N= 610,304 (5.8%)	Asian or Pacific Islander N = 1,412,557 (13.4%)	Hispanic N = 5,343,414 (50.8%)	American Indian or Alaska Native N = 45,704 (0.4%)
	PR (95% CI)	**PR (95% CI)**	**PR (95% CI)**	**PR (95% CI)**	**PR (95% CI)**
Any PMD	ref	0.76 (0.75, 0.78)	0.28 (0.27, 0.28)	0.39 (0.39, 0.39)	1.13 (1.08, 1.19)
PMAD	ref	0.67 (0.66, 0.68)	0.29 (0.28, 0.29)	0.40 (0.40, 0.41)	1.03 (0.97, 1.08)
SMI	ref	1.15 (1.12, 1.18)	0.26 (0.25, 0.27)	0.38 (0.37, 0.39)	1.55 (1.43, 1.68)

[a] PMD = perinatal mood disorders
[b] PMAD = perinatal mood and anxiety disorders
[c] SMI = serious mental illness

Table 3. Prevalence of PMD[a] (Any, PMAD[b], and SMI[c]) in 1997 and 2018 and percent change from 1997-2018, California (n = 10,509,675)

		Perinatal mental disorders per 1,000 births				
PMD category		**White**	**Black**	**Asian or Pacific Islander**	**Hispanic**	**Total[d]**
Any PMD	1997	4.9	3.8	1.1	1.2	2.6
	2018	91.7	73.5	25.8	41.5	55.1
	Fold increase	18.7	19.3	23.5	34.6	21.2
PMAD	1997	3.4	2.3	0.8	0.9	1.9
	2018	85.5	62.0	24.2	38.7	51.0
	Fold increase	25.1	27.0	30.3	43.0	26.8
SMI	1997	1.5	1.6	0.3	0.3	0.8
	2018	40.3	40.8	10.6	19.5	25.2
	Fold increase	26.9	25.5	35.3	65.0	31.5

[a] PMD = perinatal mood disorders
[b] PMAD = perinatal mood and anxiety disorders
[c] SMI = serious mental illness
[d] American Indian or Alaska Native, and other race and ethnicity included

Figure 2. A-C: Prevalence of perinatal mental disorders (PMD), 1997 - 2018, overall (any PMD) (Fig. 2A) and by category (PMAD, SMI) (Fig. 2B, 2C) across racial and ethnic categories (n = 10,509,675)

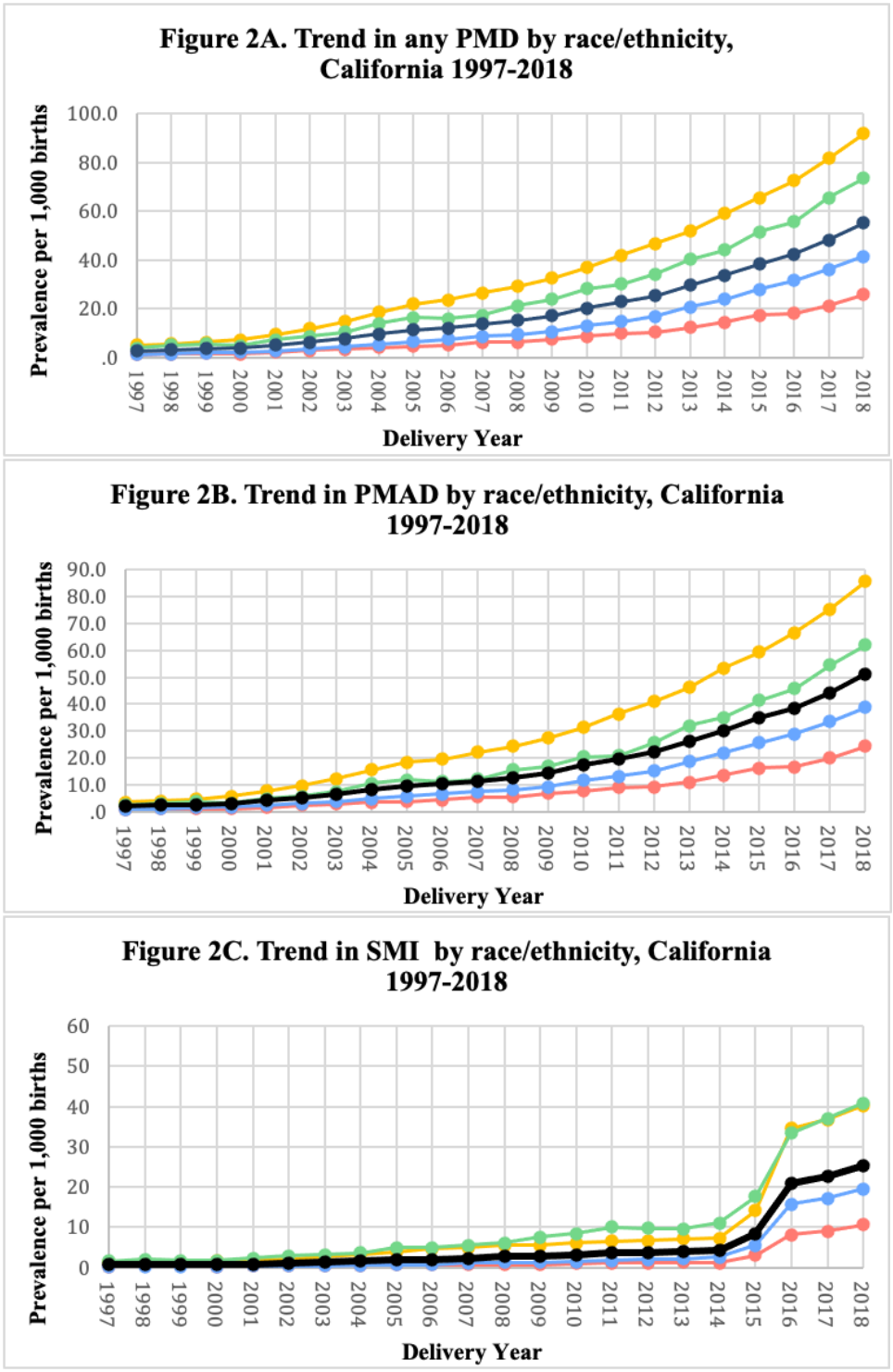


Figure 3. A-C: Trends in racial and ethnic disparities of perinatal mental disorders (PMD), overall (any PMD) (Fig. 3A) and by category (PMAD, SMI) (Fig. 3B, 3C) (n=10,509,675)

Figure 3A.

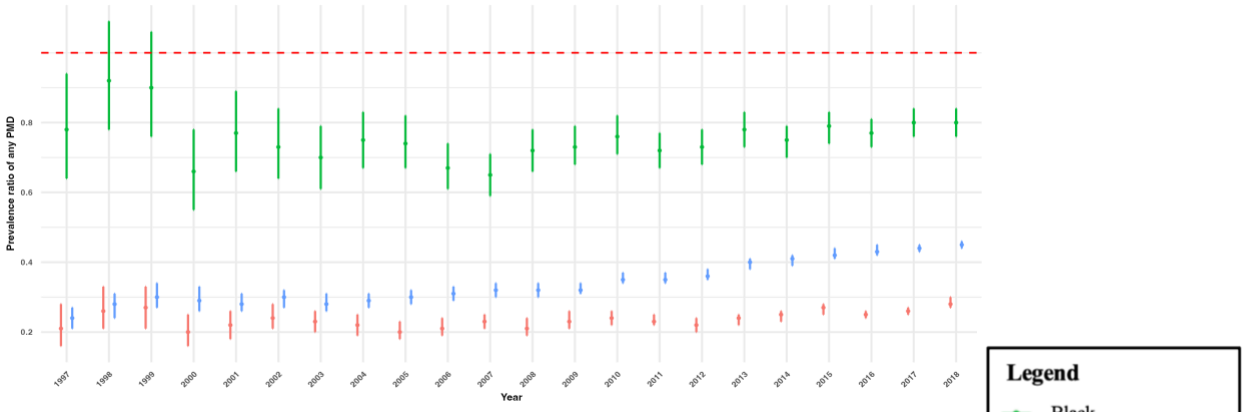


Figure 3B.

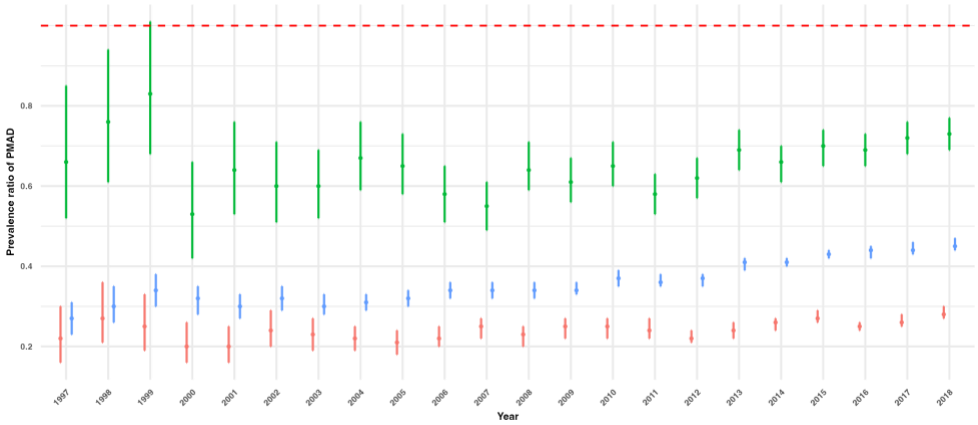
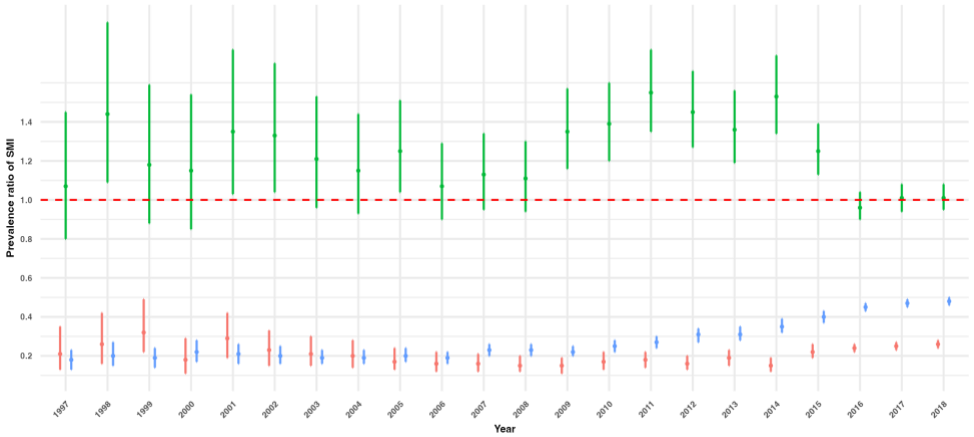


Figure 3C.



Supplementary materials

Supplement Table 1. ICD-9-CM and ICD-10-CM codes[a] for PMAD[b] and SMI[c]

PMD[d] category	Diagnoses	ICD-9	ICD-10 codes
PMAD	Depression: major depressive affective disorder, depressive disorder Anxiety: anxiety disorder, phobia, obsessive-compulsive disorder, acute stress reaction, posttraumatic stress disorder, overanxious disorder	296.20, 296.21, 296.22, 296.23, 296.24, 296.25, 296.26, 296.30, 296.31, 296.32, 296.33, 296.34, 296.35, 296.36, 300.4, 311, 293.84, 300.00, 300.01, 300.02, 300.09, 300.10, 300.20, 300.21, 300.22, 300.23, 300.29, 300.3, 300.5, 300.89, 300.9, 308.0, 308.1, 308.2, 308.3, 308.4, 308.9, 309.81, 313.0, 313.1, 313.21, 313.22, 313.30, 313.82, 313.83,	F329, F320, F321, F322, F323, F324, F325, F339, F330, F331, F332, F333, F3341, F3342, F4341, F329, F064, F419, F410, F411, F418, F449, F409, F4001, F4002, F4011, F40218, F42, F488, F458, F489, F430, F4310, F938, F328, F3340, F064, F413, F4000, F4010, F40210, R4681, Q871, F609, F068, F410, R451, F438, F439, F4311, F40298, F329, F948, F845, Z62891, Z553, F419, F40220, R457, F4312, F40228, F40230, F40231, F40232, F40233, F40240, F40241, Z733, F40242, F40243, F40248, F40290, F40291, F408, F5105, F5113, F99, R452
SMI	Bipolar disorder: Bipolar 1 disorder, manic disorder, other unspecified bipolar disorders and episodic mood disorders Psychotic disorders: psychotic disorder, schizophrenia, paranoid state, depressive psychosis	296.00, 296.01, 296.02, 296.03, 296.04, 296.05, 296.06, 296.40, 296.41, 296.42, 296.43, 296.44, 296.45, 296.46, 229.650, 296.51, 296.52, 296.53, 296.54, 296.55, 296.56, 296.60, 296.61, 296.62, 296.63, 296.64, 296.65, 296.66, 296.7, 296.10, 296.11, 296.12, 296.13, 296.14, 296.15, 296.16, 296.80, 296.81, 296.82, 296.89, 296.90, 296.99, 293.81, 293.82, 295.00, 295.01, 295.02, 295.03, 295.04, 295.05, 295.10, 295.11, 295.12, 295.13, 295.14, 295.15, 295.20, 295.21, 295.22, 295.23, 295.24, 295.25, 295.30, 295.31, 295.32, 295.33, 295.34, 295.35, 295.40, 295.41, 295.42, 295.43, 295.44, 295.45, 295.50, 295.51, 295.52, 295.53, 295.54, 295.55, 295.60, 295.61, 295.62, 295.63, 295.64, 295.65, 295.70, 295.71, 295.72, 295.73, 295.74, 295.75, 295.80, 295.81, 295.82, 295.83, 295.84, 295.85, 295.90, 295.91, 295.92, 295.93, 295.94, 295.95, 297.0, 297.1, 297.2, 297.3, 297.8, 297.9, 298.0, 298.1, 298.2, 298.3, 298.4, 298.8, 298.9,	F329, F320, F322, F3013, F302, F303, F304, F3110, F3111, F3112, F3113, F312, F3173, F3174, F3130, F3131, F3132, F314, F315, F3175, F3176, F3160, F3161, F3162, F3163, F3164, F3177, F3178, F319, F3010, F3011, F3012, F308, F328, F3181, F39, F348, F062, F060, F2089, F201, F202, F200, F2081, F205, F259, F209, F22, F24, F23, F323, F28, F4489, F29, F309, F3189, F3170, F3172, F3171, F349, F338, F0630, R401, F061, F21, F251, F250, F258, F203, F528, F09, R140

[a] based on prior work by McKee et al. 2020
[b] PMAD = perinatal mood and anxiety disorders
[c] SMI = serious mental illness
[d] PMD = perinatal mood disorders

Supplement Table 2. Sensitivity Analysis comparing prevalence of PMD (Any, PMAD, SMI), California, 1997-2018 vs 1997 – 2015 (n=10,509,675)

PMD Category		White	Black	Asian or Pacific Islander	Hispanic	Total[b]
Any PMD	1997	4.9	3.8	1.1	1.2	2.6
	2015	65.5	51.4	17.4	27.8	38.3
	2018	91.7	73.5	25.8	41.5	55.1
	1997 – 2015 Fold increase	13.4	13.5	15.8	23.2	14.7
	1997 – 2018 Fold increase	18.7	19.3	23.5	34.6	21.2
PMAD[c]	1997	3.4	2.3	0.8	0.9	1.9
	2015	59.4	41.3	16.3	25.5	34.7
	2018	85.5	62.0	24.2	38.7	51.0
	1997 – 2015 Fold increase	17.5	18.0	20.4	28.3	18.3
	1997 – 2018-Fold increase	25.1	27.0	30.3	43.0	26.8
SMI[d]	1997	1.5	1.6	0.3	0.3	0.8
	2015	14.1	17.6	3.1	5.6	8.4
	2018	40.3	40.8	10.6	19.5	25.2
	1997 – 2015 Fold increase	9.4	11.0	10.3	18.7	10.5
	1997 – 2018 Fold increase	26.9	25.5	35.3	65.0	31.5

[a] Sensitivity analyses present prevalence between two different year spans: 1997-2018 which has the most up to date data yet includes the transition to ICD-10, and 1997-2015 before the transition to ICD-10
[b] American Indian and Alaska Native and other race/ethnicity included
[c] PMAD = perinatal mood and anxiety disorders
[d] SMI = serious mental illness

Chapter 2. Toward quantifying and measuring the impact of structural racism on mental health inequities: a systematic review of the public health literature

Abstract

Background: Evidence finds structural racism is a chronic stressor that becomes biologically embodied through various mechanisms, leading to adverse health outcomes and perpetuating predictable racial health inequities across societal structures. While existing literature has primarily focused on individual forms of discrimination, growing research examines the health effects of structural racism as a coordinated, cross-institutional system. This systematic review updated previous findings from a 2018 review, and examined how recent studies conceptualize and measure structural racism, particularly its impact on mental health and substance use.

Methods: Following Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, we identified articles published from 2018-2022 through electronic database searches (PubMed, EMBASE) and manual searches of reference lists. We included English language manuscripts published in the US that reported original quantitative data analysis, focused on "structural racism" as a primary exposure, and examined mental health and substance use outcomes.

Results: 7 studies were included, 4 (57.1%) of which were cross-sectional. Over half of the studies (57.1%) included between 1,000 and 10,000 participants, primarily sampling Black or Black and White individuals. The majority (85.7%) of studies found that structural racism is significantly associated with adverse mental health and substance use outcomes, particularly among Black and other racialized populations. These studies employed multidimensional measures and interaction analyses, guided by theories such as intersectionality and critical race theory, highlighting the importance of theory-driven research.

Conclusion: Future research should incorporate qualitative methods, advanced causal mediation analyses, and policy-focused indicators to deepen the understanding of structural racism and inform effective structural interventions. Addressing structural racism requires comprehensive solutions, such as reparations, to mitigate health disparities and achieve health equity.

Introduction

The study of racism and the multifaceted ways it shapes racial and ethnic health inequities has gained steady recognition in public health research. Racism is a structured system that classifies people into racial groups in order to impose an ideology of inferiority that diminishes, disempowers, and unevenly distributes resources and opportunities to those socially constructed as inferior (Williams & Mohammed, 2009). Evidence documents the ways in which racism is both an acute and chronic stressor that becomes biologically embodied via direct and indirect mechanisms, including psychosocial, physical, and material pathways, resulting in adverse physical and mental health (Levine & Crimmins, 2014; Smith-Bynum et al., 2014; Williams et al., 2003). Given that it can manifest in multiple forms, including through individual and interpersonal interactions (i.e. negative actions/attitudes or statements toward a different race) as well as through institutional or structural forces, this ultimately creates and perpetuates predictable population patterns of racial health inequity (C. P. Jones, 2000; Williams & Mohammed, 2013).

To-date, the literature on racism and health has predominantly focused on individual-level exposures (Gee & Ford, 2011; C. P. Jones, 2000). These studies typically utilize measures such as the Everyday Discrimination Scale and the Experiences of Discrimination Scale, which ask respondents how often they have been treated unfairly due to their race, ethnicity, age, or other characteristics in a variety of circumstances (e.g. being followed in a store, called names) or life domains (e.g. education, healthcare, employment) (Williams & Mohammed, 2009). While these measures have contributed significantly to our understanding of health disparities, they were not designed to consider the broader context of structural racism entrenched in societal institutions. Given that structural racism captures the aspects of racism embedded in social, legal and economic institutions, has implications for individual-level experiences of discrimination, and consequently exerts widespread influence on vulnerable communities, it may have a more substantial impact on population health outcomes (Gee & Ford, 2011).

While much of the existing health literature has focused on individual forms of racial discrimination, a growing body of work has begun to examine the health effects of structural racism as a consciously and unconsciously coordinated, cross-institutional system, distinct from institutional racism, which focuses on single institutions rather than the connection across multiple institutions (Gee & Hicken, 2021). In fact, the field of public health has witnessed a rapid growth in scientific interest around structural racism, resulting in the proliferation of many publications over the last few years (Bailey et al., 2017; Dean & Thorpe, 2022).

With this shift toward analyzing structural drivers of health inequities, is a growing recognition of the need for improved measurement techniques to better capture structural racism and its impact on health (Adkins-Jackson et al., 2022; T. H. Brown & Homan, 2023; Hardeman et al., 2022). A prior systematic review on measures of structural racism was published in 2018, covering articles published between 2007-2017. However, the paper did not: (1) outline the various analytic approaches and study designs employed; (2) discuss the extent to which conceptualization of structural racism matched study operationalization; and (3) examine whether studies were grounded in and informed by social theory.

Further, since the publication of the 2018 review, there has been widespread discussion and scholarship on structural racism in the current public health landscape. With the gruesome murder of George Floyd and the elevated rates of COVID-19 infection and death among minoritized populations, 2020 was marked by a national racial reckoning that shone a light on the pervasive nature of structural racism and its manifestation in inequities of today (Tamene et al., 2020). Prominent health professional associations, including the American Public Health Association and the American Medical Association issued urgent calls to recognize and address the health impacts of structural racism (Mendez et al., 2021). The National Institute on Minority Health and Health Disparities released a request for funding announcement (RFA) to support observational or intervention research on the impact of structural racism on health in order to improve health equity and inform policies (Blachman-Demner & Tyus, 2022).

Despite this rapid growth and increased focus on the structural racism-health association, research examining its impact on mental health and substance use outcomes remains limited. This may be because evidence on racial disparities in mental health and substance use is complex. Studies have shown that minoritized populations have either lower or similar prevalence of mental disorders compared to whites, despite greater exposure to stressors (McGuire & Miranda, 2008; Thomas Tobin et al., 2022). Nevertheless, some research has uncovered racial disparities. For example, a 2020 CDC Morbidity and Mortality Weekly Report (MMWR) report, for example, found higher prevalence of depression, suicidal ideation and substance use among Black and Hispanic adults relative to their white counterparts (McKnight-Eily, 2021). Mental illness also appears to be more severe, persistent and debilitating for minoritized populations compared to their white counterparts (Breslau et al., 2005; Williams et al., 2007). Moreover, minoritized populations experience higher rates of unmet mental health needs, including underdiagnosis and delayed diagnosis, as well as being less likely to be offered treatment (Cook et al., 2017; Hall et al., 2023; Kozhimannil et al., 2011; Marrast et al., 2016).

As the recent COVID-19 pandemic and publicized police killings of Black people have also reinforced, it is widely agreed that societal stressors can have adverse mental health impacts on ethnically minoritized populations (Shim, 2021; Williams, 2018). One study examining the impacts of George Floyd's murder found significantly larger increases in depression and anxiety symptoms among Black people following George Floyd's death (Eichstaedt et al., 2021). Beyond exposure to state sanctioned violence, other race-related stressors can affect mental health of minoritized populations, including, but not limited to, inequities in living and working conditions, exposure to community violence, and limited access to healthcare and other resources (Williams, 2018). Understanding this relationship is crucial to identifying its underlying causes and informing development of interventions. With a focus on mental health and substance use outcomes, this paper seeks to capture the latest research exploring papers from 2018 - 2022. We updated and expanded findings of the previous systematic review by examining how studies conceptualized and operationalized structural racism and whether these decisions were informed by social theory, as well as the various methods used to evaluate the mental health and substance use consequences of structural racism. We concluded by offering recommendations for future directions, including interventions aimed at reducing racism and its effects.

Methods

Our search strategy followed Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Liberati et al., 2009).

Eligibility Criteria

We conducted a systematic review of articles published between 2018 - 2022 as an update to and in accordance with procedures followed by Groos and colleagues (Groos et al., 2018).

Information Sources

Articles were identified through electronic PubMed and EMBASE database searches.

Search Strategy

Following the search strategy outlined by Groos and colleagues, the keywords "structural racism," "systemic racism," "institutional racism" and "institutionalized racism" were utilized to identify relevant articles (Groos et al., 2018). Institutional and institutionalized racism were included in this search given the interchangeable use of the terms institutional and structural racism in public health literature (Bailey et al., 2017; Bonilla-Silva, 1997; Williams et al., 2019).

Selection Process

English language manuscripts were included if they: (1) were published in the US; (2) reported on original quantitative data analysis; (3) situated "structural racism" as a primary exposure, including measuring its direct effects; and (4) examined health-related outcomes. All retrieved articles were uploaded to Covidence systematic review software, where duplicates were automatically removed. The first stage of the review involved a detailed screening of titles and abstracts. This screening was independently performed by at least two reviewers. Articles that met the predefined inclusion criteria based on the title and abstract screening were advanced to the next stage of review. Any discordance between reviewers or articles for which it was unclear if they met inclusion criteria based on title and abstract was discussed by all reviewers to reach consensus. Those articles that passed the initial screening phase were rescreened with full-text review. Each article was again assessed by two reviewers, with any discrepancies resolved through discussion.

Data Collection Process

In the final phase of the review process, remaining articles were extracted using a standardized template developed a priori and programmed into Covidence. Two reviewers independently extracted data from each article using this template. To guarantee the accuracy and completeness of the extracted data, reviewers met regularly to discuss and resolve any discrepancies, reaching a consensus on all data extractions. The first author, M. Tamene reviewed every article through all stages of the review process.

Data Items

In addition to data items established by PRISMA guidelines, the *a priori* extraction template featured extraction items specific to the study of structural racism informed by interests within and beyond the research community. These included: how researchers (1) utilize theories to

conceptualize structural racism, (2) operationalize structural racism; and (3) employ various methods to evaluate its impact on health inequities.

Study Selection

The initial yield comprised 3,221 publications, with 1,251 excluded as duplicates. Of the 1,970 remaining, 1,774 were excluded in the initial screen for not meeting inclusion criteria, with the resulting 183 articles undergoing full-text review. Through full-text review, another 89 articles were excluded, leaving 94 total articles that empirically examined structural racism and health more broadly. While analysis of the broader review is currently underway, the team included an additional screening filter to only select mental health and substance use-related outcomes. This yielded 7 articles, which serve as the focus of this paper's analysis (Figure 1).

Synthesis Methods

The first author, M. Tamene, summarized key characteristics across each study, including sample description, whether theory was examined, measure of structural racism used, etc. (Tables 1-3); notable patterns and themes across studies were then synthesized. We did not conduct a meta-analysis due to the heterogeneity of measures of structural racism, health outcomes, adjustment sets, and study designs.

Results

Study characteristics

Of the 7 studies included in final analysis, the majority (71.4%) were national samples (Table 1). 4 (57.1%) were cross-sectional studies and the other 3 (42.9%) were cohort studies. Sample sizes ranged from 151 to 331,171, with over half (57.1%) having between 1,000 to 10,000 study participants. There was some range in age composition across the study samples, with 1 (14.3%) focused on early to middle adolescents, 3 (42.9%) examining late adolescence to young adulthood and 2 (28.6%) sampling adults (Table 1). 1 (14.3%) study did not explicitly state the age distribution of its sample (Das et al., 2021). Almost all the studies were restricted to participants that identified as Black only (42.9%) or Black and white (42.9%), with only one of the studies including other racialized persons (i.e. Asian, Black, Latinx, or multiracial U.S.) (Lett, Asabor, Tran, et al., 2022). Nearly all the studies were also conducted in secondary datasets, utilizing nationally representative survey datasets (e.g. Panel Study of Income Dynamics, National Longitudinal Survey of Adolescent to Adult Health (Add Health). Finally, the studies utilized a range of geographic units of analysis, including state (n = 3), county (n =1), census tract (n =1) and other (2). Two studies used schools as their geographic unit of analysis, and a final study did not explicitly state the geographic unit of analysis, but it appeared to be at the individual level.

Conceptualization and measurement of structural racism

All studies include explicit definitions of structural racism (Supplement Table 1). All definitions highlight that structural racism is a fundamental cause of racial health inequities (Phelan et al., 2010; Phelan & Link, 2015). Many speak to structural racism as embedded in interconnected inequitable systems (social, legal, economic, political, etc.) that go beyond individual-level discrimination or racial prejudice. These systems are made inequitable by differentially

distributing power, opportunities and resources to benefit racially dominant groups and marginalize racially minoritized groups (Bonilla-Silva, 1997; Braveman et al., 2022; Feagin, 2013; Krieger, 2012; Williams & Mohammed, 2013). To understand what assumptions guided conceptualization and selection of indicators for measuring structural racism, we assessed whether articles used a guided theory. 6(85.7%) of the 7 articles use an explicit guiding theory to frame their empirical analysis of structural racism. 3 (42.9%) used intersectionality theory, 1 (14.3%) used critical race theory, and 1 (14.3%) employed a 'syndemic framework' (Table 2).

More than half (5 of 7 or 71.4%) of the studies employed multidimensional measures of structural racism. Of the 7 articles, 3 (42.9%) used the state racism index (SRI) (English et al., 2021, 2022; Lett, Asabor, Beltrán, et al., 2022) (Table 2). The SRI was originally developed by Mesic et al. (2018) to examine the relationship of state-level structural racism and Black-white disparities in fatal police shootings of unarmed people (Mesic et al., 2018). Ranging on a scale from 0 to 100, it quantifies social inequity based on residential segregation and Black-White disparities in incarceration rates, educational attainment, poverty levels, median household income, home ownership, and employment status. A single overall SRI is developed for each state by averaging the scores for each of the five dimensions. Njoroge and colleagues utilized explanatory factor analysis to create a factor score from several unidimensional dimensions, using the median cut off to categorize into high vs. low levels of structural racism (Table 2) (Njoroge et al., 2022). Polos et al. (2022) introduce two measures to capture structural racism in schools: the School Contextual Disadvantage Index (CDI), reflecting differences in resources and opportunities across schools due to historical racism, and the School Structural Racism Index (school SRI), indicating differences in resources between Black and white students within the same schools (Polos et al., 2022). The CDI measures school mean values of indicators across six domains as a geometric mean, resulting in scores that range from 0 to 4. Similarly, the school SRI captures Black-white inequities in school-based contextual disadvantage using a geometric mean of the school Black-white ratio for the variables within the same six domains (Table 2).

2 (28.6%) of the studies captured unidimensional measures of structural racism in their analysis (Table 2). Das and colleagues used data on police killings on unarmed African Americans from the Mapping Police Violence (MPV) database (Das et al., 2021). The MPV database collects data on officer-involved instances of violence through crowdsourcing and is validated through multiple sources (e.g., news reports, social media references). Finally, Schwarz et al. look at residential racial segregation, using Local Getis-Ord G* statistics. The G* statistics are Z-scores that measure residential segregation by comparing the proportion of Black residents in a tract and neighboring tract to the average proportion in a larger area (Core-Based Statistical Area (CBSA) or county) (Getis & Ord, 1992; G. L. Schwartz et al., 2022).

Almost all of the studies (85.7%) employed stratification or interaction analyses to investigate if the relationship between structural racism and mental health or substance use was modified by another variable. Effect measure modifiers included race and ethnicity (n = 3), anti-LGBTQ policy (n = 2), interpersonal racism (n = 1), negative COVID-19 experiences (n = 1), biological sex (n = 1), and developmental timing of residential segregation (n=1). These analytic approaches underscore authors' attempts to match quantitative operationalization with their definition and conceptualization of structural racism. For example, Lett et al. (2022) introduce the term "multilevel" racism to specifically capture the joint associations of interpersonal and

structural racism with their health outcomes of interest, which they then examine analytically through interaction analyses (Lett, Asabor, Beltrán, et al., 2022). Similarly, Njoroge et al. propose a 'syndemic' framework – defined as two or more interrelated conditions combining to adversely impact health – to explore how the intersection of racism and COVID-19 impact the postpartum mental health of Black birthing individuals (Njoroge et al., 2022; Singer, 2000). They operationalize this by introducing an interaction term, negative COVID-19 experiences, to their analysis (Njoroge et al., 2022). We present stratified and interaction analyses (Table 3).

Results of Individual Studies

Across our 7 studies, 6 found that structural racism measures were associated with at least one adverse mental health and/or substance use outcome (Table 3). Among the 6 studies that found a significant association between structural racism and adverse mental health and substance use outcomes, 3 conducted studies solely in racialized populations (2 among Black populations; 1 among Asian, Black, Latinx, or multiracial U.S.) (Das et al., 2021; Lett, Asabor, Tran, et al., 2022; G. L. Schwartz et al., 2022), while the other 3 included Black and white participants in their sample (English et al., 2021, 2022; Polos et al., 2022). One study examining the relationship between police killings of unarmed Black people and psychiatric ED visits among Black-identifying individuals, found an 11% increase in ED visits for depression among African Americans in 0 and 3 months following a police killing (SE: 0.05; $p < 0.05$ and SE: 0.04; $p < 0.05$, respectively) (Das et al., 2021). Another study sampling only Black populations found that Black young adults who grew up in lower-segregation neighborhoods had lower odds of drinking more than once a week compared to those from high-segregation neighborhoods (OR = 0.65, 95%CI: 0.480, 0.877; $p = 0.005$) (G. L. Schwartz et al., 2022). Lett and colleagues, who sampled Asian, Black Latinx or multiracial transgender and gender-expansive young adults of color (TYAOC), found a significant association between structural racism and the use of alcohol or drugs before sex within this population (OR = 1.57, 95% CI: 1.11, 2.23) (Lett, Asabor, Tran, et al., 2022).

The 3 papers that sampled both Black and white participants found different results across the two subgroups. The 2 papers by English and colleagues both examined the potential adverse health impacts of structural racism for non-Hispanic Black and non-Hispanic White sexual minority men (SMM) (English et al., 2021, 2022). In these studies, they found that structural racism was positively associated with various mental health and substance use outcomes (e.g. anxiety and depressive symptoms, heavy drinking, self-harm, suicide attempt) for Black SMM but not for white SMM (Table 3). The other study used two measures of structural racism in schools, the CDI and school SRI, to understand their relationship to depressive symptoms among Black and white students (Polos et al., 2022). When using the CDI measure, the authors found that although higher CDI levels were associated with more depressive symptoms for both Black and white students, Black students were twice as likely to attend high CDI schools. The magnitude of association was greatest for Black girls ($\beta = 0.422$, SE = 0.225, $p<0.1$) (Table 3). SRI measures similarly yielded differential outcomes by race. While statistically imprecise, their results suggested a positive association between school SRI and depressive symptoms in Black students, a slightly negative association for white girls, and no effect for white boys (Table 3). For example, assessing depressive symptoms across school SRI levels by race and gender, the study found that if Black girls hypothetically moved from a school with no structural racism to

one with the highest SRI (around 40), their depressive symptoms would increase by nearly 1 point on average (Polos et al., 2022).

Of note, all 7 studies included an adjustment set. Age was the most commonly adjusted covariate, included in 5 (71.4%) of the studies. Many also adjust for other sociodemographic variables, including income, education and employment status. Neighborhood contexts (e.g., urban-rural classification, violent crime rate) are also commonly adjusted for across some of the studies, as are area-level fixed effects (Table 3). 5 (71.4%) of the 7 studies also explicitly discuss why they selected their particular covariates (Das et al., 2021; English et al., 2021, 2022; Lett, Asabor, Tran, et al., 2022; G. L. Schwartz et al., 2022). Only one study discussed concerns about over-adjustment (Lett, Asabor, Tran, et al., 2022). Another noted the potential for neighborhood poverty to be both a confounder (i.e., in shaping the racial make-up of in-movers) and a mediator (i.e., residential segregation reducing economic opportunity), included in their model as a confounder, to also be a mediator between their structural racism-health association (G. L. Schwartz et al., 2022). As such, the paper presented results including and excluding neighborhood poverty from its analysis.

Discussion

We conducted a systematic review on the structural racism-mental health and substance use association in the public health literature. Our search yielded 7 studies. The majority of the studies found significant associations between structural racism and adverse mental health and substance use outcomes, particularly among Black and other racialized populations. Higher levels of structural racism were linked to increased psychiatric visits, depressive and anxiety symptoms, as well as adverse substance use related outcomes, including heavy drinking and consuming alcohol or drugs prior to sex. These findings suggest that structural racism may play a significant role in patterning mental health and substance use inequities across the life course.

That these studies found a greater risk of adverse mental health and substance use outcomes among Black and minoritized populations is consistent with broader public health literature. One proposed pathway by which structural racism harms health is by limiting access to power, resources and opportunities (e.g., employment, safe housing, living environment, quality healthcare, education, etc.) for minoritized populations while simultaneously privileging dominant groups (Krieger, 2012; Phelan & Link, 2015). Racism also acts as a chronic stressor that over-activates the body's stress response, causing accelerated aging and leading to unhealthy coping behaviors (Geronimus, 1992; Pascoe & Smart Richman, 2009; Williams & Mohammed, 2009). The sustained economic disadvantage and racism-related stress may accumulate over the life course to erode the health of minoritized populations, resulting in poor health outcomes (Krieger, 2001, 2014a).

The careful conceptualizations and operationalization of measures across many of these studies reflect the best practices recommended by structural racism scholars. In their recent commentary, Drs. Dean and Thorpe highlight the limitations of current structural racism measures, including heterogeneity in definitions, inappropriate labeling of institutional racism as structural racism, and the use of single indicator models that fail to capture the complex, interactive nature of

social structures (Dean & Thorpe, 2022). They argue that accurate measurement of structural racism must capture its interactive effects across multiple institutions. Together with other scholars in the field, they suggest the use of indices, scales, or interaction analyses to better capture multiple dimensions of structural racism (Adkins-Jackson et al., 2022; Jahn, 2022). Additionally, researchers are encouraged to consider within-group analyses to uncover differential manifestations of structural racism (Wien et al., 2023). The studies in this review highlight the empirical advancements in the quantification of structural racism, as more than half used a multidimensional measure, nearly all employed interaction analyses, and many conducted within-group analyses. This offers a hopeful direction for future research in understanding and addressing health inequities rooted in structural racism.

The insightful conceptualization and operationalization of many of these studies is informed by their foundational grounding in social theory, highlighting the importance of theory-driven research. Theories can help provide a foundational understanding of how social structures and systems influence health (Diez Roux, 2022). This understanding is crucial for framing research questions, guiding data collection, interpreting findings, and identifying effective interventions (Jahn, 2022; Krieger, 2014b). Promisingly, many of the theories cited by the papers in this review align with those frequently used to examine the role of structural racism and health, including ecosocial theory, intersectionality, and critical race theory (Wien et al., 2023). These shared theoretical frameworks may confer benefits in structural racism research. This includes enhancing collaboration by providing a common language and understanding, enabling researchers to build from previous work more effectively, and offering common principles and methods for easier comparison of across studies. By integrating theoretical frameworks from various disciplines and using comprehensive, multidimensional models, researchers can better understand and mitigate the health inequities caused by structural racism (Adkins-Jackson et al., 2022).

Building on the importance of theory-driven research, selecting an appropriate adjustment set becomes a critical next step to accurately analyze the effects of structural racism on health outcomes. Researchers are tasked with identifying and adjusting all potential confounders, while simultaneously trying to avoid adjusting on a mediator variable and introducing overadjustment bias (van Zwieten et al., 2022). Our systematic review highlights similar concerns, with studies like Schwartz et al. (2022) addressing the dual role of neighborhood poverty as both a mediator and confounder, thus presenting models with and without this variable to offer scientifically justifiable alternatives (G. L. Schwartz et al., 2022). Additionally, Lett et al. (2022) intentionally do not adjust for gender or ethnoracial identity in their analyses as they consider these variables to be correlated proxies of their primary exposure variables. Adjusting, would thus, lead to over adjustment, biasing results toward the null (Lett, Asabor, Tran, et al., 2022). To choose an appropriate adjustment set, future researchers should clearly define their research question, construct and justify causal diagrams to distinguish confounders from mediators, and select variables that maximize adjustment for confounding while minimizing adjustment for mediators (Lett, Asabor, Tran, et al., 2022; van Zwieten et al., 2022).

Finally, recent scholarship has called for more focus on selecting theoretically meaningful geographic units of analysis (Riley, 2018). Structural racism operates differently by geographic unit (e.g., census tracts, county, state), with each spatial level of analysis representing distinct

patterns of social contexts and policies (Chambers et al., 2018). Much neighborhood-level research employs census tract-level analyses, because public data tend to be readily available at this level and because this is also thought to capture the heterogeneity of racial inequities, which may be otherwise diluted at larger area-levels. However, other units of analysis may better capture the institutional and policy-driven aspects of structural racism. For example, state-level analyses, which made up nearly half (42.9%) of the review studies, have important implications for consideration. One, this may sometimes be the smallest geographic unit for which data is available (Bor et al., 2018). There may also be some exposures that show meaningful variation across states (e.g., number of fatal police shootings) (Mesic et al., 2018). Additionally, historical, and contemporary laws, policies and institutional practices happen at the state-level, serving as a unique site for policy interventions that may ameliorate some of the effects of structural racism (Lukachko et al., 2014). In other contexts, counties may provide a more key unit of interest given their role in allocating resources through laws and regulations (Bell et al., 2019). For example, in the state of California, counties had governance power to expand Medicaid coverage to an income level up to 200 percent of the federal poverty line (FPL) (from the Medicaid standard cutoff of 133 percent of FPL) (Sommers et al., 2016). Ultimately, consideration of geographic units of analysis should ensure theoretically meaningful units of analysis and leverage data that demonstrates variation in racial and ethnic disparities.

Limitations of reviewed studies

Findings of these studies should be interpreted in context of several limitations. Several common limitations arose across the studies (Supplement Table 2). First, multiple studies noted small sample sizes impacting the power to detect associations and potentially leading to underestimated or null point estimates, particularly for stratified and interaction analyses. Limited generalizability and potential bias in self-reported data also came up across several studies. Finally, 4 (57.1%) of the studies were cross-sectional, limiting the ability to establish causal relationships. These shared limitations emphasize the need for larger datasets that particularly oversample minoritized populations, precise and comprehensive measurement tools, and longitudinal studies to enhance the validity and generalizability of research findings on structural racism and health.

Limitations of our review

There were a few limitations in this review. First, only 7 papers were included in the final analysis, 2 of which come from the same author. These studies may not be representative of the broader structural racism-health literature. Future reviews examining all health outcomes may reveal different trends worth investigating. Second, we followed the search strategy of the previous systematic review, and it is possible that our search terms might have missed some studies. For example, articles that quantitatively measured structural racism and its association to health but did not use our terms would not be included. Finally, our review may be susceptible to publication bias, given the tendency for positive results to be more likely published than negative or null results.

Future Directions

Future research should continue to expound on the work of these current studies to better elucidate how structural racism operates differently within different domains and across intersecting axes of social position. Utilizing policy as an indicator of structural racism can be

one such approach as policies can reinforce structural racism across various social systems and institutions and play a role in patterning population health outcomes (Raifman et al., 2017; Sudhinaraset et al., 2021). Policies also have direct implications for intervention and measurement of mechanisms of structural racism's effects on health (Jahn, 2022). Agénor and colleagues have begun work to support this endeavor, by developing a database of structural racism-related state laws (Agénor et al., 2021). This database can serve as a valuable resource for researchers as well as policymakers and advocates to both evaluate and address the impact of racist state policies on racial and ethnic health inequities. Of note, we do not advocate for one singular approach to measuring a complex phenomenon. For example, while multidimensional measures have been highlighted as more accurately measuring the underlying construct and conceptualization of structural racism, they may have less direct implications for interventions. Researchers should ensure that measures of structural racism are interpretable and useful to drive actionable change.

Qualitative methods can also offer valuable insights into understanding and addressing structural racism's impact on health. For example, in one paper, Chambers and colleagues conducted focus groups with Black women across the reproductive lifespan asking about their definition of structural racism, how it showed up in their communities, and its impact on adverse maternal and infant health outcomes. These results were used to inform identification of unique domains of structural racism, including policing of Black families and hidden resources (Chambers et al., 2021). Qualitative methods can also inform policy by capturing rich, contextual insights into lived experiences, thereby revealing the nuances and complexities that quantitative analysis alone may be unable to glean (Adkins-Jackson et al., 2022). Future research should explore incorporating qualitative and mixed methods approaches to deepen the field's understanding of structural racism.

Pathways through which structural racism influences health remains understudied. Advanced casual mediation analyses can be employed to more accurately model the dynamic and relational processes by which structural racism can produce health disparities (Graetz et al., 2022). This requires access to rich longitudinal data that can capture some of the contextual factors that may be mediators (e.g., social networks, limited access to health foods, exposure to toxins, etc.). Future research should focus on identifying and collecting the necessary data to explore these mediation pathways comprehensively. Researchers should also incorporate and engage with critical race theories, including intersectionality and ecosocial theory, which offer frameworks to conceptualize the numerous pathways through which structural racism likely impacts health (Agénor, 2020; Krieger, 2014b; Phelan & Link, 2015).

Finally, structural racism requires structural solutions (Hardeman et al., 2022; Williams & Etkins, 2021). While many studies, including those in our review, acknowledge the need for structural solutions (e.g., promoting policies to reduce racial inequities in housing, education, incarceration), not all provide clear recommendations. For example, a systematic review on the effects of structural interventions to address health inequities found that of the 29 articles reviewed, only 18 address racial and ethnic health inequities and none looked at policies to mitigate anti-Black racism (Clark et al., 2022). One structural solution that has been proposed in the public health literature is reparations. Black reparations are a crucial structural solution to addressing persistent Black–White health disparities in the United States, by expanding

resources, reducing stress, and providing intergenerational benefits that ultimately improve health outcomes for Black Americans (T & Sandro, 2020). Guaranteed basic income is yet another solution and innovative programs across the U.S. have seen some success. For example, the Magnolia Mother's Trust Project in Jackson, Mississippi offered 110 single black mothers $12,000 for one year. Preliminary findings found improved mental and physical health outcomes among these mothers relative to comparable mothers not in the program (Onifade et al., 2023). This economic boost eases some of the financial burden disproportionately faced by Black and Brown parents. Structural place-based interventions, including providing affordable housing and investing in revitalization of disadvantaged neighborhoods (e.g., construction of safe walkways, streets, improving quality of public housing infrastructure) have also led to promising health improvements for minoritized populations (Bailey et al., 2017; Gaumer et al., 2015). One such intervention, conducted in Philadelphia, found that full remediation (i.e., installation of new windows and doors, cleaning trash, weeding) of abandoned houses led to significant reductions in weapons violations and gun assaults (South et al., 2023). Therefore, in addition to documenting and quantifying the complex relationship between structural racism and health, future research should propose and study structural interventions that minimize and combat exposure to structural racism. These key interventions emphasize the importance of addressing structural racism through coordinated, multisectoral approaches, policy reform, education, and community-based initiatives to advance health equity and improve mental and physical health outcomes.

Conclusion

Our systematic review of seven studies highlights the significant association between structural racism and adverse mental health and substance use outcomes, particularly among Black and racialized populations. Higher levels of structural racism were linked to increased psychiatric visits, depressive and anxiety symptoms, and negative substance use behaviors. The studies utilized explicit definitions of structural racism, guided theories such as intersectionality and critical race theory, and employed multidimensional measures and interaction analyses. This theoretical and methodological rigor offers promising signs for future directions in the research of structural racism and health inequities. To measure structural racism is to make the invisible visible. While we know no measure is perfect, scholarship looking to better understand its phenomena, operationalize it into a quantifiable measurement, and elucidate mechanisms of impact are imperative to achieving health equity.

Tables and Figures

Figure 1. PRISMA Flow Diagram

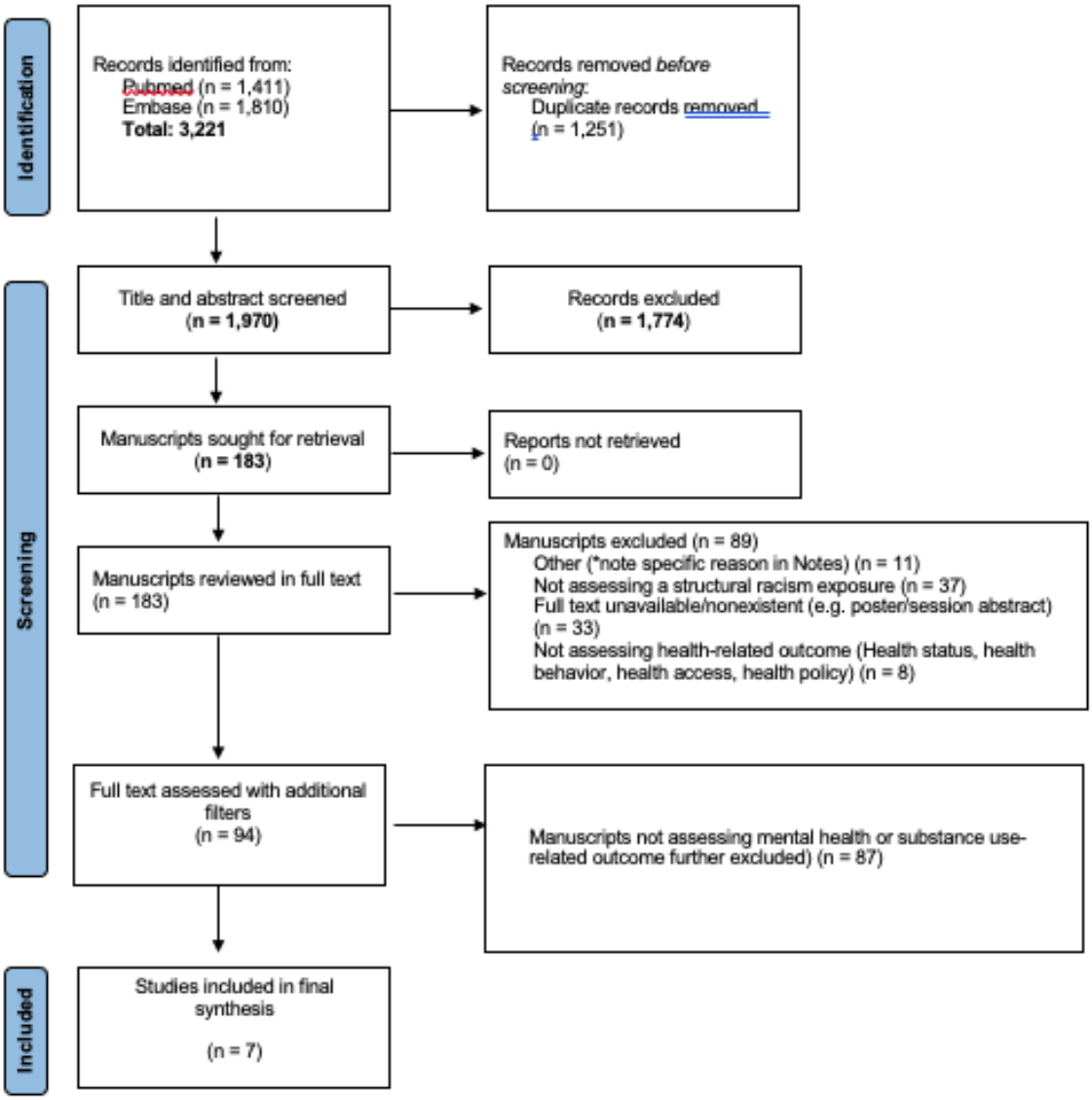


Table 1. Summary of included studies (n = 7)

Study characteristic	N (%)	References
Study design		
Cross-sectional	4 (57.1%)	(English D et al., 2021, 2022; Lett E et al., 2022; Polos et al., 2022)
Cohort	3 (42.9%)	(Das A et al., 2021; Njoroge WFM et al., 2022; Schwartz GL et al., 2022)
Study period		
Min (years)	1	(English D et al., 2021, 2022; Lett E et al., 2022; Njoroge WFM et al., 2022; Polos et al., 2022)
Max (years)	12	(Schwartz GL et al., 2022)
Median (years)	1	
Sample size		
<1,000	2 (28.6%)	(Lett E et al., 2022; Njoroge WFM et al., 2022)
1,000 – 10,000	4 (57.1%)	(English D et al., 2021, 2022; Polos et al., 2022; Schwartz GL et al., 2022)
>10,000	1 (14.3%)	(Das A et al., 2021)
Age composition		
Early to middle adolescence	1 (14.3%)	(Polos et al., 2022)
Late adolescence to young adulthood	3 (42.9%)	(English D et al., 2022; Lett et al., 2022; Schwartz GL et al., 2022)
Adulthood	2 (28.6%)	(English D et al., 2021; Njoroge WFM et al., 2022)
Other	1 (14.3%)	(Das A et al., 2021)
Racial composition		
Black only	3 (42.9%)	(Das A et al., 2021; Njoroge WFM et al., 2022; Schwartz GL et al., 2022)
Black and white	3 (42.9%)	(English D et al., 2021, 2022; Polos et al., 2022)
Black, Asian, Latinx, multiracial	1 (14.3%)	(Lett E et al., 2022)
Location		
National	5 (71.4%)	(English D et al., 2021, 2022; Lett E et al., 2022; Polos et al., 2022; Schwartz GL et al., 2022)
State	1 (14.3)	(Das A et al., 2021)
Local	1 (14.3%)	(Njoroge WFM et al., 2022)
Geographic level		
Census-tract	1 (14.3%)	(Schwartz GL et al., 2022)
County	1 (14.3%)	(Das A et al., 2021)
State	3 (42.9%)	(English D et al., 2021, 2022; Lett E et al., 2022)
Other	2 (28.6%)	(Njoroge WFM et al., 2022; Polos et al., 2022)

Table 2. Conceptualization and measurement of structural racism

Reference	Guiding theory	Structural racism domain	Measure of structural racism	Exposure Data Source
(Das A et al., 2021)	Critical race theory Theory of race-based traumatic stress	Policing	Police killings of unarmed African Americans	Mapping Police Violence (MPV)
(English D et al., 2021)	Intersectionality	Multidimensional measure comprising 5 dimensions: • residential segregation • incarceration • educational attainment • employment status	State racism index	(Mesic A et al., 2018) developed measures Original sources: • US Census Data (2000, 2010, 2013 – 2015) • US Bureau of Justice Statistics (2000, 2010, 2015)
(English D et al., 2022)	Intersectionality	Multidimensional measure comprising 5 dimensions: • residential segregation • incarceration • educational attainment • employment status	State racism index	(Mesic A et al., 2018) developed measures Original sources: • US Census Data (2000, 2010, 2013 – 2015) • US Bureau of Justice Statistics (2000, 2010, 2015)
(Lett E et al., 2022)	Intersectionality	Multidimensional measure comprising 5 dimensions: • residential segregation • incarceration • educational attainment • employment status *Note:* multilevel racism combines structural racism and interpersonal racism	State racism index multilevel racism	(Mesic A et al., 2018) developed measures Original sources: • US Census Data (2000, 2010, 2013 – 2015) • US Bureau of Justice Statistics (2000, 2010, 2015)
(Njoroge WFM et al., 2022)	Syndemic framework	Multidimensional measure comprising following dimensions: • income (below vs above $20,000 annually) • educational attainment (no vs partial college graduate degree) • insurance (private vs public/ Medicaid) • relationship status (married/living with partner vs other) • house ownership (own vs other) • HOLC grade (A to D) • neighborhood socioeconomic status	Uses explanatory factor analysis to derive factor scores; median cutoff used to split into high vs low levels of racism	Electronic medical records University of Richmond HOLC maps

Reference	Guiding theory	Structural racism domain	Measure of structural racism	Exposure Data Source
(Polos et al., 2022)	Ecosocial theory	Multidimensional measure, capturing joint effects of: • student body background characteristics, • school connectedness, • perceived life chances, • teacher-student racial composition (SRI only), • disciplinary atmosphere, • school attendance, and • school quality characteristics (CDI only)	school contextual disadvantage index (CDI) (binary: median cutoff used to split into low and high disadvantage) school structural racism index (school SRI) (continuous score: 0-100)	National Longitudinal Survey of Adolescent to Adult Health (Add Health Survey)
(Schwartz GL et al., 2022)	--	Residential segregation	residential racial segregation during childhood. using Local Getis-Ord G* statistics (categorical: Always segregated; moved lower to high segregation; moved high to lower segregation; always low segregation; others)	US Census Data (1990, 2000, 2010) American Community Survey's 5-year estimates (2010-2014, 2014-2018)

Table 3. Results of individual studies (n = 7)

Reference	Sample description	Structural racism measure	Outcome[a]	Effect measure modifiers	Covariates	Summary measure of association[b]
(Das A et al., 2021)	Black-identifying individuals visiting ED for depression in AZ, KY, NC, NJ, NY,	Police killings of unarmed African Americans	psychiatric ED visits for depression	--	• number of EDs/hospitals • violent crime arrest rate • county, month, year fixed effects	• Police killings month lag0: RD = 0.11 (SE: 0.05; p < 0.05) • Police killings month lag 1: RD = 0.05 (SE: 0.05, p > 0.05) • Police killings month lag 2: RD = 0.09 (SE: 0.05, p > 0.05) • Police killings month lag 3: RD = 0.11 (SE: 0.04; p < 0.05)
(English D et al., 2021)	Non-Hispanic Black and non-Hispanic White sexual minority men (SMM), excluding those who were multiracial, living with HIV, residing in Washington, D.C. or Puerto Rico, or had missing covariate data.	State racism index	• anxiety symptoms, • depressive symptoms • perceived burdensomeness (psychological precursor to suicidality), • heavy drinking	• race • anti-LGBTQ policy	• rural-urban classification • subjective SES • income • education • formal employment status • insurance status • housing instability • age • sexual identity • gender identity • relationship status • sexual activity • sexual identity outness • sexual positioning	*Stratified analyses:* Black SMM • anxiety symptoms: β = 0.20 (SE = 0.10, p = 0.04) • perceived burdensomeness: β = 0.42 (SE = 0.09, p<0.001) • heavy drinking: β = 0.23 (SE=0.10, p = 0.01) *Interaction analyses:* High anti-LGBTQ policies for Black SMM • perceived burdensomeness: β = 0.38 (SE = 0.08, p<0.001) • heavy drinking: β = 0.22 (SE=0.07, p = 0.003) Note: Authors did not report estimates for structural racism*low anti-LGBTQ policies; Note: All findings were nonsignificant for White SMM; authors did not report estimates
(English D et al., 2022)	non-Hispanic Black and non-Hispanic white sexual SMM excluding those who were multiracial, aged <16 OR >25, residing in Washington, D.C. or Puerto Rico, or had missing covariate data.	State racism index	<u>Individual suicide risk factors</u> • depressive symptoms • heavy drinking <u>Interpersonal suicide risk factors</u> • perceived burdensome-ness • thwarted belonging-ness suicidality • suicidal ideation • self-harm • suicide attempt	• race • anti-LGBTQ policy	• rural-urban classification • subjective SES • income • formal employment status • housing instability • age • sexual identity • relationship status • living situation (living alone/with others • whether participants have living parents/stepparents and are contact with them • insurance	*Stratified analyses:* Black SMM • depressive symptoms: β = 0.68 (SE = 0.33, p = 0.04) • perceived burdensomeness: β = 0.73 (S.E.=0.22, p = 0.001) • heavy drinking: β = 0.41 (S.E.=0.19, p=0.03) • thwarted belonginess: β = 0.85 (S.E.=0.11, p<0.001) • self-harm: β = 0.75 (S.E.=0.37, p=0.04) • suicide attempt: β = 0.99 (S.E.=0.51, p=0.05) *Interaction analyses:* High anti-LGBTQ policies for Black SMM • perceived burdensomeness: β = 0.08 (S.E.=0.02, p=0.001) • heavy drinking: β = 0.12 (SE=0.06, p = 0.04) • thwarted belonginess: β = 0.09 (S.E.=0.01, p<0.001) • suicide attempt: β = 0.10 (S.E.=0.05, p=0.059)

Reference	Sample description	Structural racism measure	Outcome[a]	Effect measure modifiers	Covariates	Summary measure of association[b]
					coverage • military experience	Note: Authors did not report estimates for structural racism*low anti-LGBTQ policies; Note: All findings were not significant for White SMM; authors did not report estimates
(Lett E et al., 2022)	Asian, Black, Latinx, or multiracial U.S. transgender and gender-expansive young adults of color (TYAOC0, (ages 18–30)	State racism index multilevel racism	history of consuming alcohol or drugs prior to sex	Interpersonal racism	• age • immigrant status • education	*Main analysis* OR = 1.57 (95% CI: 1.11, 2.23) *Interaction* (i.e. multilevel racism = interpersonal + structural) OR = 2.15 (95% CI: 1.27, 3.65)
(Njoroge WFM et al., 2022)	Black participants from a COVID-19 longitudinal cohort study, receiving prenatal care at two UPenn Health System hospitals in Philadelphia	Uses explanatory factor analysis to derive factor scores; median cutoff used to split into high vs low levels of racism	• postpartum depression • postpartum anxiety	Negative COVID-19 experiences	• maternal age • parity • gestational weeks at prenatal assessment • weeks postpartum at the postpartum survey	*Main analysis:* • postpartum depression: OR = 0.79 (95% CI: 0.37. - 1.69) • postpartum anxiety: OR = 0.59 (95% CI: 0.21 - 1.51) *Interaction analysis: postpartum depression* • Negative COVID-19 experience* high levels of system racism (structural + interpersonal) OR = 2.52 (95 % CI: 1.38 - 4.60) • Negative COVID-19 experience * low levels of system racism OR: 1.03 (95% CI: 0.59 - 1.78) *Note:* systemic racism defined as high levels of structural racism + interpersonal racism *Note:* no significant interaction found for postpartum anxiety; authors did not report estimates
(Polos et al., 2022)	non-Hispanic Black students and non-Hispanic white students	school contextual disadvantage index (CDI) (binary: median cutoff used to split into low and high disadvantage) school structural racism index (school SRI) (continuous score: 0-100)	depressive symptoms	• race • biological sex • CDI	• Student age, • grade level, • school percentage of Black students	CDI White girls: β = 0.285 (SE = 0.098, p<0.01) White boys: β = 0.270 (SE = 0.052, p<0.01) Black girls: β = 0.422 (SE = 0.225, p<0.1) Black boys: β = 0.204 (SE = 0.115, p<0.1) School SRI White girls: β = -0.006 (SE = 0.009, p>0.1) White boys: β = 0.000 (SE = 0.005, p>0.1) Black girls: β = 0.022 (SE = 0.019, p>0.1)

Reference	Sample description	Structural racism measure	Outcome[a]	Effect measure modifiers	Covariates	Summary measure of association[b]
						Black boys: β = 0.017 (SE = 0.015, p>0.1) *Interaction analysis* White girls, low school disadvantage: β = -0.001 (SE = 0.009, p>0.01) White girls, high school disadvantage: β = -0.020 (SE = 0.016, p>0.01) White boys, low school disadvantage: β = 0.006 (SE = 0.007, p>0.01) White boys, high school disadvantage: β = -0.015 (SE = 0.008, p<0.01) Black girls, low school disadvantage: β = 0.071 (SE = 0.034, p<0.05) Black girls, high school disadvantage: β = -0.006 (SE = 0.019, p>0.01) Black boys, low school disadvantage: β = 0.042 (SE = 0.023, p<0.01) Black boys, high school disadvantage: β = 0.001 (SE = 0.018, p>0.01)
(Schwartz GL et al., 2022)	Black Transition to Adulthood study participants reporting at least every health outcome at least once and having at least two observations during childhood	residential racial segregation during childhood. using Local Getis-Ord G* statistics (categorical: Always segregated; moved lower to high segregation; moved high to lower segregation; always low segregation; others)	psychological distress Alcohol consumption: • drunk more than once per week • average number of binge drinking days	developmen-tal timing of residential segregation	• family income • parental education • parents married • neighborhood poverty • neighborhood population • state fixed effects	*Main effects* • Psychological distress: β = 0.28 (95% CI: -0.189, 0.742; p = 0.244) • Drink >1x per week: OR = 0.65 (95% CI: 0.480, 0.877; p = 0.005) • # of days binge drinking: β = -2.51 (95% CI: -6.036, 1.013; p = 0.162) *Note: estimates presented compare always low to always high segregation trajectory* *Interaction analysis:* Moved high to lower (vs always high) * early childhood: • Psychological distress: β = 0.03 (95% CI: -0.67, 0.72; p = 0.941) • Drink >1x per week: OR = 1.04 (95% CI: 0.66, 1.63) • # of days binge drinking: β = 3.99 (95% CI: -3.73, 11.71)

Reference	Sample description	Structural racism measure	Outcome[a]	Effect measure modifiers	Covariates	Summary measure of association[b]
						Moved high to lower (vs always high) * adolescence: • Psychological distress: β = 0.78 (95% CI: -0.12, 1.68; p = 0.089) • Drink >1x per week: OR = 0.75 (95% CI: 0.44, 1.28) • # of days binge drinking: β = -1.71 (95% CI: -7.02, 3.60)
						Moved lower to high segregation (vs always high)* early childhood: • Psychological distress: β = 0.78 (95% CI: -0.12, 1.68) • Drink >1x per week: OR = 0.87 (95% CI: 0.52, 1.44) • # of days binge drinking: β = -0.90 (95% CI: -5.33, 3.52)
						Moved lower to high segregation (vs always high)* adolescence: • Psychological distress: nonsignificant; estimates not reported • Drink >1x per week: OR = 0.51 (95% CI: 0.19, 1.34) • # of days binge drinking: β =-3.58 (95% CI: -8.40, 1.24)

[a] *Note:* while some authors look at other health outcomes, we only report estimates for mental health and substance use outcomes for this review

[b] All summary measures shared are of fully adjusted point estimates

Supplementary materials

Supplement Table 1. Definitions of structural racism (n = 7)

Reference	Definition	Definition references
(Das A et al., 2021)	"Structural racism, however, captures dimensions of racism embedded in social, legal, and economic institutions" "Although examination of individual-level factors has advanced knowledge of health inequities, it ignores the broader ecological context of racism embedded in societal institutions (Additionally, facets of structural racism may have a stronger relation to population health given its widespread influence on vulnerable communities"	Jones, 2000; Gee and Ford, 2011
(English D et al., 2021)	"Structural racist and anti-LGBTQ oppression have their foundation in historically rooted cultural ideologies and interconnected institutions (e.g., law enforcement, local governments) that systematically label and marginalize Black sexual minority communities. Structural oppression is unique from individual oppression because it is enacted through systems rather than just individuals with power and prejudice"	Delgado, 2017; Bowleg, 2013; Link and Phelan, 2001; Williams, 2000; Carmichael et al., 1992; Feagin, 2013
(English D et al., 2022)	"Structural oppression is enacted through systems (e.g., interconnected laws and policies) on an aggregate level, rather than solely through the actions of individuals who possess power and prejudice"	Feagin, 2013
(Lett E et al., 2022)	"a fundamental "social-structural" determinant of HIV inequities that must be addressed to end the HIV epidemic. The authors explicate how many of the remaining barriers to HIV prevention and treatment are manifestations of structural racism, including carceral and police violence and discrimination, neighborhood disadvantage, and limited access to social services among others"; "policies, practices, and norms that constitute structural racism and transphobia operate on national, state, community, and organizational levels."	Bowleg et al., 2022
(Njoroge WFM et al., 2022)	"Structural racism, defined as "practices that maintain or exacerbate unfair inequalities in power, resources or underlying systemic racist conditions,"	Flanagin, Frey, and Christiansen, 2021
(Polos et al., 2022)	"embedded in the interconnected policies, practices, and norms that enable the operation of systemic racism across political, legal, economic, school, and other societal systems This suggests structural racism can be measured as variation or inequities in structures, systems, and institutions, or their manifestations, that produce racially disparate outcomes." "social systems are hierarchically structured by race and differentially distribute rewards accordingly. Moreover, the structure is comprised of the aggregate of social relations and practices based on racial distinctions. This perspective more explicitly links macro-structures to individuals and emphasizes social contexts as important places in which structural racism is reified"	Bonilla-Silva, 1997; Braveman et al., 2022
(Schwartz GL et al., 2022)	"fundamental determinant" of health—a structural factor shaping more proximate social and environmental determinants of health and of racial health inequities"	Williams and Collins, 2001; Gordon, 2020; Keene and Padilla, 2010; Massey and Denton, 1993

Supplement Table 2. Summary of study limitations (n = 7)

Reference	Limitations
(Das A et al., 2021)	· Difficulty differentiating ED visit types (i.e. emergent vs non-emergent) · Potential under-reporting of police killings data may affect results · Potential for unmeasured confounding
(English D et al., 2021)	· Limited generalizability due to younger, more educated sample than general SM population · Anti-LQBTQ policy measure did not differentiate policies targeting specific LGBTQ+ groups not part of study population (e.g. anti-trans bathroom law) · SRI and anti-LQBTQ policy not directly comparable, (i.e. one based on specific policies while another proxy/proximal indicator of policies
(English D et al., 2022)	· Limited generalizability due to higher sexual risk in sample than general SM population · Anti-LQBTQ policy measure did not differentiate policies targeting specific LGBTQ+ groups not part of study population (e.g. anti-trans bathroom law)
(Lett E et al., 2022)	· Small sample size not designed to power analyses of structural measures (i.e. stratified analysis) · Structural racism measure only captures part of the multilevel impact of structural racism
(Njoroge WFM et al., 2022)	· Small sample size and attrition likely underestimating risk and underpowered to detect association · Postpartum mental health outcomes self-reported, may be subject to social desirability bias · Race/ethnicity identification based on EMR, less accurate than patient self-report
(Polos et al., 2022)	· Limited generalizability due to focus on adolescence, missing earlier sensitive periods · Selected structural racism not likely to capture all operating domains · Depressive symptoms measure may under-identify depression in Black adolescents
(Schwartz GL et al., 2022)	· Limited generalizability with PSID data not reflecting experiences of recent Black immigrants · Outcomes and covariates self-report may be subject to reporting bias · Limited information on reasons for childhood moves (e.g. eviction or family home purchase). · Some residential histories may be incomplete. · Census tracts may not align with residents' neighborhood boundaries · Potential for unmeasured confounding.

Chapter 3. Structural racism and perinatal mental health – The role of racialized economic segregation

Abstract

Background*:* Perinatal mental disorders (PMD), encompassing perinatal mood and anxiety disorders (PMAD) and serious mental illness (SMI), are important complications affecting birthing persons during pregnancy and postpartum. Some racial and ethnic differences have been documented. Limited research to date, however, has empirically investigated how the manifestations of structural racism influence racial differences in perinatal mental health outcomes.

Objective: To examine associations between the index of concentration at the extremes (ICE), a measure of spatial social segregation, as a proxy for structural racism in relation to hospital-diagnosed PMD among a racially and ethnically diverse cohort in California.

Methods: We used data from a population-based cohort of all live hospital births between 1997 and 2018 in California. PMD outcomes, including PMAD and SMI, were identified using ICD-9-CM and ICD-10-CM codes captured in hospital discharge records through the perinatal period. The ICE race-income metric, measuring the area-level difference between high income whites (most privileged) and low-income Blacks (least privileged), was calculated using census data at the census tract level and categorized into tertiles from least privileged to most privileged based on racial and economic spatial segregation. Race and ethnicity-stratified mixed effects log-binomial models estimated the risk of hospital-diagnosed PMD, adjusting for maternal age, education, and insurance and accounting for clustering by census tract. Sensitivity analyses explored whether results differed using the individual components of ICE race-income (i.e. ICE race-ethnicity and ICE income).

Results: For Black, Asian or Pacific Islander, and Hispanic birthing persons, living in the least privileged neighborhoods was associated with a reduced risk of hospital-diagnosed PMD. Conversely, for white birthing persons, living in the least privileged neighborhoods was associated with an increased risk of hospital-diagnosed PMD. Sensitivity analyses confirmed these patterns.

Conclusions: Our findings reveal a complex association between structural racism and hospital-diagnosed PMD. For minoritized populations, living in less privileged neighborhoods might offer protection or result in underdiagnosis due to inadequate healthcare, while their white counterparts can more readily access healthcare outside their neighborhoods. Advancements in research can help inform place-based and structural interventions aimed at improving perinatal mental health outcomes.

Introduction

Perinatal mental disorders (PMD) are serious complications that can affect birthing persons during pregnancy and through the first year postpartum. These disorders encompass perinatal mood and anxiety disorders (PMAD) and serious mental illness (SMI), including psychosis and bipolar disorder (O'Hara & Wisner, 2014). There are substantial impacts on the functioning of both the birthing person and their offspring associated with PMD, including preterm birth, low birth weight, and impaired child development (Gavin et al., 2005; Grace et al., 2003; Grote et al., 2010; Lopez et al., 2019).

Racial and ethnic differences in maternal mental illness have also been documented. Several studies have found that minoritized populations, including Black, Hispanic, American Indian, or Alaska Native birthing persons, experience a higher prevalence of perinatal mental illness, particularly depression and anxiety, compared with their white counterparts (Bauman et al., 2020; Matthews et al., 2021; Rich-Edwards, 2006; Shorter et al., 2021). Conversely, other studies find no significant racial differences or even higher prevalence among white birthing persons, highlighting the complexity and variability of perinatal mental health across different racial and ethnic groups (Fridman et al., 2014; Kim et al., 2020; Sujan et al., 2023). Variability in the definitions and study design of maternal mental health, including what diagnoses to count, and time of measurement (i.e., prenatal, postnatal or both) may likely account for the variation in study findings (Wilcox et al., 2021). However, research consistently highlights racial inequities in access to adequate and quality mental healthcare. Black birthing persons experience higher rates of unmet mental health needs compared to their white peers, with studies finding they are less likely to be screened for postpartum depression, to receive outpatient mental healthcare treatment, to receive continuity of care, and to fill prescriptions for antidepressant medication (Hall et al., 2023; Karvonen et al., 2022; Kozhimannil et al., 2011). The high burden of perinatal mental health conditions, as well as the reported inequities in access to quality healthcare, underscore the critical need to understand the upstream drivers that underlie racial and ethnic disparities.

Structural racism is increasingly being recognized as a fundamental driver and cause of poor health outcomes. As outlined by Dr. Zinzi Bailey and her colleagues, structural racism refers to the "totality of ways in which societies foster racial discrimination, through mutually reinforcing inequitable systems (in housing, education, employment, earnings, benefits, credit, media, health care, criminal justice, and so on) that in turn reinforce discriminatory beliefs, values, and distribution of resources, which together affect the risk of adverse health outcome" (Bailey et al., 2017, p 1455). Ecosocial theory further offers a framework to understanding the pathways by which racism within each of these systems can lead to health inequities (Krieger, 2001). Structural racism can act as a chronic psychosocial stressor that becomes biologically embodied and can lead to accelerating aging and adverse mental and physical health outcomes (Levine & Crimmins, 2014; Smith-Bynum et al., 2014; Williams et al., 2003; Williams & Mohammed, 2013). It can also harm health by limiting access to critical resources and power (e.g., employment, safe housing, living environment, quality healthcare, education, etc.). Other pathways include excess exposure to toxins and pathogens; trauma; and health risk behaviors in response to discrimination (Krieger, 2012). While growing literature has documented this structural racism-health association more broadly, limited work to date, has empirically

investigated how the manifestations of structural racism influence perinatal mental health outcomes.

Given the multidimensional nature of structural racism, it is imperative for measures of structural racism to capture the intersecting and mutually reinforcing pathways by which this driver may impact health outcomes (Chambers et al., 2019; Groos et al., 2018). One measure increasingly employed in public health research is the Index of Concentration at the Extremes (ICE), which quantifies spatial social segregation by race, income, and its combination (Krieger et al., 2016). More specifically, this measure is operationalized by examining racial and economic privilege versus disadvantage within a concentrated area through three different measures, ICE-race, ICE-income, and ICE race-income. The novel application of combining race and ethnicity and income into a single measure, through the ICE race-income metric, is gaining momentum in public health literature as it begins to address the need for measurements that capture the multidimensionality of structural racism (Larrabee Sonderlund et al., 2022).

Previous applications of ICE in public health research have been used to explain racial health inequities in various outcomes, including adverse birth outcomes, cancer, premature and all-cause mortality, and communicable diseases (Larrabee Sonderlund et al., 2022). However, only one study to date has examined the relationship between ICE and perinatal mental health but was limited by a small sample size with low racial diversity and only looked at prenatal depression (Haight et al., 2023). This study extends these findings and adds to the greater literature looking at the relationship between various measures of area-level inequity and health by leveraging a large population-based dataset of more than 10 million birth and hospital records spanning over 20 years, looking at a more comprehensive set of mental health outcomes across the perinatal period and examining this relationship across more racial and ethnic groups.

In this cross-sectional study, we utilized census tract-level difference between high income whites and low-income Blacks (i.e., ICE race-income) as a proxy for structural racism to examine associations with hospital-diagnosed perinatal mental health outcomes, PMAD, SMI, and their composite (any PMD) in California from 1997 to 2018. We hypothesized that residing in the least privileged neighborhoods will be associated with an increased risk of hospital-diagnosed PMD for minoritized populations but will have no associated risk for white birthing persons.

Methods

Study population

We obtained state-wide data on all singleton and first non-singleton recorded hospital live births in California from 1997 to 2018 from the California Department of Health Care Access and Information (HCAI), which linked birth certificate files with hospital discharge records from pregnancy through one-year postpartum. We excluded births with implausible gestational age (<20 weeks or >45) (n = 309,986), fetal deaths (n = 31,948), and missing maternal race and ethnicity, age, education, payer, as well as those missing census-tract data for the creation of ICE measures (n = 520,783). The final analytic sample comprised 10,155,036 births (Figure 1). Approval of the study protocol came from the State of California Committee for the Protection

of Human Subjects and the institutional review boards of Stanford University and the University of California, Berkeley.

Outcome

We defined hospital-diagnosed PMD using the International Classification of Diseases, Ninth & Tenth Revision, Clinical Modification (ICD-9-CM and ICD-10-CM) codes for relevant PMAD and SMI diagnoses occurring at least once any time during the perinatal time period (i.e., pregnancy and up to one-year postpartum). These codes were extracted from the hospital discharge record of each birthing person. Based on the available data, a perinatal hospital diagnosis was defined as happening during either an ER visit, an inpatient hospitalization or an ambulatory surgery hospitalization. Relevant ICD-9-CM and ICD-10-CM codes for PMAD and SMI were based on prior work (McKee et al., 2020) (Supplement Table 1). Experiences of any PMAD, SMI, and any PMD, which was a composite of the previous two diagnoses, were categorized as binary (yes/no).

Exposure

We calculated ICE race-income, a measure of spatial social segregation that quantifies residents at the extremes of racial and economic deprivation and privilege, using Census 2000 data, as well as American Community Survey 2006 – 2010, 2011 – 2015, and 2015 – 2019 5-year estimates. We use measures of black-white inequity to construct our ICE measures because we conceptualize this to be the clearest indicator of anti-black structural racism. In other words, the policies and practices of racial discrimination, including historical redlining and subsequent disinvestment, occur most systematically in neighborhoods with high Black concentration, structuring opportunities and resources differentially and consequently patterning population health disparities across all racial and ethnic groups (Krieger et al., 2018; Massey & Brodmann, 2014).

In response to calls to better capture the multidimensionality of structural racism, we use ICE race-income as our primary ICE exposure. The measure was calculated using the formula: $ICE_i = (A_i - P_i) / T_i$, where for every census tract i, A represents the number of people in the most privileged group, P is the total number of people in the least privileged or disadvantaged group and T is the total census-tract population (Chambers et al., 2019; Dyer et al., 2022; Krieger et al., 2016, 2017, 2018). ICE race-income measure was derived by taking the difference between the number of non-Hispanic white persons making $100,000 or more (i.e., most privileged group) minus non-Hispanic Black persons making less than $25,000 annually (i.e., least privileged group). Scores range from -1 to 1, with negative values indicating least privileged neighborhoods (i.e., greater concentration of low-income Black persons), positive numbers indicating most privileged neighborhoods (i.e. greater concentration of high-income white persons), and 0 meaning either an absence of residents from both groups or an equal distribution. ICE scores were categorized into tertiles for interpretability, whereby Tertile 1 represents communities that have experienced the most structural divestment as a function of anti-Black racism and economic disenfranchisement and are thus the least privileged census-tracts, Tertile 2 being middle, and Tertile 3 (reference group) representing communities that have conversely experienced the most structural investment as a function of anti-Black racism and economic disenfranchisement and thus denoted as the most privileged census-tracts (Dyer et al., 2022; Eichenbaum & Tate, 2022).

Effect Modifier

Race and ethnicity of the study's participants were self-reported on birth certificates. This includes non-Hispanic white (hereafter, white), non-Hispanic Black (hereafter, Black), non-Hispanic Asian or Pacific Islander (hereafter Asian or Pacific Islander), Hispanic, non-Hispanic American Indian or Alaska Native (hereafter American Indian or Alaska Native), and other (includes multiracial individuals and those that self-identify as other). Due to the limited sample size of the other group (N=6,466), we only present overall prevalence but do not include it in distribution tables or analytic models. Based on previous literature and a priori knowledge, we adjusted for two different sets of covariates.

Covariates

Model 1, our basic confounder set, controlled for maternal age (<20, 20-24, ≥35 years). In Model 2, we additionally controlled for other sociodemographic variables that may be considered risk factors for PMD, including education (high school or less, some college, undergraduate or postgraduate degree), and payer (private, public, uninsured or other) (Haight et al., 2023; McKee et al., 2020). While these may be arguably on the causal pathway, given the cross-sectional nature of this study, we cannot rule out the possibility that these variables may confound the relationship between ICE race-income and PMD.

Statistical Analysis

In descriptive analyses, we examined the distribution of population characteristics by race and ethnicity, ICE tertiles, and other study covariates. To assess the relationship between the ICE race-income and hospital-diagnosed perinatal mental health outcomes, we estimated race-stratified (i.e., separate models by race) risk ratios and 95% confidence intervals using log-binomial models that accounted for clustering by census tract. Adjusted risk ratios (aRR) are presented, adjusting for maternal age (Model 1), and maternal age, education, and payer (Model 2). Given the small sample size, we did not include those with the racial and ethnic category of 'other' in our log-binomial models. All analyses were conducted in SAS 9.4 and Stata/IC, version 15.1.

Robustness Checks

In sensitivity analyses, we separately explored ICE-race and ICE-income measures to assess how these unidimensional metrics compare to their combined multidimensional counterpart, ICE race-income. Following the same formula as ICE race-income, ICE race-ethnicity was calculated by comparing non-Hispanic Black vs. non-Hispanic white whereas ICE-income compared those making less than $25,000 vs those making $100,000 or more. These metrics were also similarly categorized into tertiles for interpretability, with Tertile 3 used as reference.

Results

Descriptive statistics

The final analytic sample included 10,155,036 live births, with the majority of participants (i.e., birthing parents) identifying as Hispanic (50.8%), followed by white (29.5%), Asian or Pacific Islander (13.4%), Black (5.8%), American Indian or Alaska Native (0.4%) and Other (0.1%) (Table 1). American Indian or Alaska Native birthing people had the highest prevalence of

hospital-diagnosed PMD (3.7%), while Asian or Pacific Islander birthing people had the lowest (0.9%). The highest prevalence of hospital-diagnosed PMAD and SMI was also among American Indian or Alaska Native birthing people (2.9% and 1.3%, respectively) and the lowest among Asian or Pacific Islanders (0.8% for PMAD and 0.2% for SMI) (Supplement Tables 2 and 3). White birthing persons were more likely to reside in the most structurally invested census tracts compared to structurally disinvested census tracts, while the opposite was true for Black, Hispanic and American Indian or Alaska Native birthing people (Table 1). Those with a hospital-based PMD diagnosis were more likely to be older and have some college education, and those who identified as Black, Asian or Pacific Islander, and Hispanic among PMD diagnosed participants were more likely to reside in the most structurally invested neighborhoods. Asian or Pacific Islander and Hispanic participants who received a PMD diagnosis were more likely to have private insurance.

Any PMD

Models stratified by race and ethnicity suggest that among Black (aRR Tertile 1 vs. Tertile 3 = 0.75, 95% CI = 0.70, 0.81), Asian or Pacific Islander (aRR Tertile 1 vs. Tertile 3 = 0.78, 95% CI = 0.72, 0.84) and Hispanic (aRR Tertile 1 vs. Tertile 3 = 0.65, 95% CI = 0.62, 0.68) birthing persons, living in the least privileged neighborhoods is associated with a lower risk of hospital-based PMD diagnosis relative to their counterparts living in more privileged neighborhoods (Table 2). Conversely, among white birthing persons, living in the least privileged neighborhoods was associated with a higher risk of hospital-based PMD diagnosis compared to their counterparts residing in the most privileged neighborhoods (aRR Tertile 1 vs Tertile 3 = 1.09, 95% CI = 1.05, 1.13) (Table 2). Moreover, results for white participants suggested a dose-response pattern wherein risk for hospital-based PMD diagnosis was lower with each tertile increase in neighborhood privilege (aRR Tertile 2 vs 3 = 1.04. 95% CI = 1.01, 1.08). We did not observe statistically significant results among participants who identified as American Indian and Alaska Native.

PMAD

Results for hospital-diagnosed PMAD largely mirrored those observed for hospital-diagnosed any PMD. That is, among Black (aRR Tertile 1 vs. Tertile 3 = 0.72, 95% CI = 0.67, 0.78), Asian or Pacific Islander (aRR Tertile 1 vs. Tertile 3 = 0.77, 95% CI = 0.72, 0.83), and Hispanic (aRR Tertile 1 vs. Tertile 3 = 0.65, 95% CI = 0.62, 0.68) participants, residing in the least privileged neighborhoods was associated with a lower risk of hospital-diagnosed PMAD (Table 2). However, among both white and American Indian and Alaska Native participants, PMAD-specific results were not statistically significant.

SMI

Results for hospital-diagnosed SMI also paralleled those observed for hospital-diagnosed any PMD. In fully adjusted models, among Black and Asian or Pacific Islander birthing people, living in the least privileged neighborhood was associated with an approximately 20% lower risk of hospital-based SMI diagnosis relative to their privileged counterparts. Among Hispanic populations, the magnitude of this association was even greater: residing in the least privileged neighborhood was associated with a 33% lower risk of hospital-based SMI diagnosis (Table 2, Model 2). Alternatively, among White birthing persons, living in the least privileged neighborhood was associated with a 30% higher risk of hospital-diagnosed SMI relative to their counterparts residing in the most privileged neighborhoods.

Robustness Checks

When examining the association between ICE income alone and hospital-diagnosed PMD, PMAD and SMI, patterns were largely consistent with those observed for ICE race-income together. ICE race-ethnicity and hospital-based PMD diagnosis associations, however, differed among White and American Indian or Alaska Native birthing persons. For example, after adjustment, among white birthing persons, residing in T2 was associated with a lower risk of hospital-diagnosed PMD (aRR Tertile 2 vs. Tertile 3 = 0.95, 95%CI = 0.92, 0.98) (Supplement Table 5). Further, whereas results were not statistically significant when examining American Indian or Alaska Native persons using ICE race-income (aRR Tertile 1 vs. Tertile 3 = 1.03, 95% CI = 0.87, 1.21), when using ICE-race, living in the least privileged neighborhoods (T1 & 2) was associated with a lower risk of hospital-diagnosed PMD (aRR Tertile 1 vs Tertile 3 = 0.79, 95% CI = 0.68, 0.91; aRR Tertile 2 vs 3 = 0.79, 95% CI = 0.68, 0.91) (Supplement Tables 5).This divergence was similar across all outcomes.

Discussion

In this study, we utilized census tract-level Black-White racial and economic segregation as a measure of anti-Black structural racism to examine its association with hospital-diagnosed PMAD, SMI, and their combination (any PMD) in California from 1997 to 2018. We found that the relationship between structural racism and hospital-diagnosed perinatal mental illness varied by race and ethnicity. Contrary to our original hypothesis, among Black, and Asian or Pacific Islander, and Hispanic birthing persons, living in the least privileged neighborhoods – i.e., neighborhoods that have a greater proportion of low-income Black individuals – was associated with a lower risk of hospital-diagnosed PMAD, SMI, and any PMD when compared to their counterparts in the most privileged neighborhoods. By contrast, among White birthing persons, living in the least privileged neighborhoods was associated with a higher risk of hospital-based diagnosis of perinatal mental health outcomes when compared to their counterparts living in the most privileged neighborhoods. In sensitivity analyses examining racial and economic privilege separately, we found that for all racial groups, living in the least privileged neighborhood – i.e., living in majority-Black neighborhoods – was associated with a lower risk of hospital-based diagnosis for all the hospital-diagnosed mental health outcomes. This persisted across both tertiles of racialized segregation for all minoritized racial groups and was evident among white birthing persons living in T2 neighborhoods.

Our findings that for racially minoritized birthing persons, living in the least privileged neighborhoods was associated with lower risk of hospital-based perinatal mental illness diagnosis is consistent with the only other paper to investigate the relationship between structural racism and perinatal mental illness (Haight et al., 2023). This North Carolina study of a pregnancy cohort found that among Black participants, living in the least privileged neighborhoods (i.e., majority Black (ICE-race), majority low income (ICE-income), or majority low-income Black (ICE race-income)) was associated with a lower prevalence of prenatal depression. One potential explanation for these findings is that racially minoritized populations living in predominantly white or affluent neighborhoods may experience higher levels of race-related stress, discrimination and social exclusion, contributing to increased risk of depressive symptoms (Alegría et al., 2014; Evans, 2003; Harrell, 2000). Thus, living in a predominantly

Black and less affluent neighborhood may serve as a buffer against these stressors and offer more culturally competent and familiar resources, reducing the likelihood of mental illness (Haight et al., 2023). Future research should examine the role neighborhood supportive institutions like community centers and locally owned businesses can play in buffering the negative health impacts of concentrated area-level poverty and segregation.

Notably, in the context of hospital-based perinatal mental health, the differences in diagnosis among racially minoritized populations may also reflect access barriers rather than actual burden. Long-standing policies and practices of disinvestment in racially and economically disadvantaged neighborhoods (i.e., least privileged) have led to limited access to health-promoting resources including quality health care (Barber, 2020; Kershaw & Albrecht, 2015; Williams & Collins, 2001). For example, one study examining racial and ethnic disparities in access to community-based perinatal mental health programs in New Jersey found that geographic accessibility issues limited program reach for Black, Hispanic and Asian birthing persons (Rokicki et al., 2024). Another looking at the association of racial residential segregation and closure of hospitals found that high levels of residential segregation, combined with a high percentage of poor residents, increase the likelihood of public hospital closure (Ko et al., 2014). The lack of medical care in the least privileged neighborhoods, due to structural disinvestment, may help explain some of these findings. Given that our outcomes were limited to hospital-diagnosed PMD, future research should employ population-based surveys to better understand how these associations hold when looking to capture true burden.

Poor quality care in the least privileged neighborhoods may also contribute to underdiagnosis, with studies showing that even when accessing care, birthing persons of color, particularly Black birthing persons, are less likely to be screened for perinatal mental illness (Conteh, 2022; Sidebottom et al., 2021). One study conducted in New York City, found that neighborhoods with extreme racial and economic segregation (i.e., least privileged) had the highest risk of severe maternal morbidity, with over a third of this risk attributable to the quality of the delivery hospital (Janevic et al., 2021). Given that improving hospital quality may be one downstream intervenable factor to mitigating the impact of structural racism, future research should similarly investigate potential pathways, such as hospital quality, between structural racism and perinatal mental illness.

Our findings of a higher associated risk of PMD among white birthing persons living in the least privileged neighborhoods may point to their greater privilege and ability to access resources outside of their neighborhood. One study looking at racial and economic differences in mobility patterns across the US found that white people living in poor neighborhoods are more likely to visit affluent neighborhoods compared to their Black and Hispanic counterparts (Wang et al., 2018). This greater mobility privileges them with relatively better access to resources found in more affluent neighborhoods, including quality healthcare. These findings suggest that the health impacts of structural racism likely operate differentially across racial groups, by restricting opportunities and access to quality healthcare for Black and Hispanic residents, while poor white residents face fewer barriers (Massey & Fischer, 2000).

Finally, our examination of anti-black structural racism was operationalized by looking at black-white racial and economic inequity within neighborhoods, because we understood this to best

capture the historical processes by which neighborhoods are made to be structurally disadvantaged. The fact that we also found a lower associated risk of hospital-based PMD diagnosis for Hispanic and Asian or Pacific Islander birthing people living in these least privileged neighborhoods suggests that neighborhood structural factors impact health outcomes beyond race alone.

Limitations

Our study is not without limitations. First, our outcome variables likely underestimate the prevalence of PMD. Our outcome data were ascertained from hospital discharge diagnosis through the perinatal period (i.e., any time during pregnancy and up to one year postpartum) and thus require both a medical diagnosis and an event severe enough to precipitate care-seeking in the hospital setting during this timeframe. Diagnoses that occur outside of the hospital setting, including in primary care or community-based clinics are thus missing from our data. Second, our use of ICE as a proxy measure for structural racism did not fully capture the multidimensional nature of the construct. Future research should consider employing multidimensional approaches (e.g., area-level black-white inequity in education and incarceration) to measuring the influence of structural racism on perinatal mental health outcomes (Adkins-Jackson et al., 2022; Jahn, 2022). The cross-sectional design of this study limits our ability to establish causation. Future research should consider collecting and analyzing qualitative and longitudinal data to better understand the mechanisms underlying the relationship between structural racism and perinatal mental illness. For example, given that hospital-level factors can serve as a critical lever for intervention, studying their potential mediating and modifying role in the relationship between structural racism and maternal mental health is timely and important. Our analysis was restricted to California, potentially limiting the generalizability of our findings. However, California represents 1 in 8 of all births in the United States and as one of the largest states in the country, findings from this study can provide broader insight beyond the state. Finally, there may be unmeasured confounding from variables not included in our analysis that may be a common cause of selection into a neighborhood and perinatal mental health (e.g. residential mobility, housing policies).

Conclusion

This study examines the influence of structural racism, using measures that capture racialized economic segregation on perinatal mental health in California. Our findings reveal a complex association between structural racism, as measured by spatial racial and economic segregation, and hospital-diagnosed PMD. Future research should conduct longitudinal analyses, utilize survey data to better capture PMD burden, and investigate potential mechanisms (e.g., hospital quality) of the association. Advancements in research can help inform place-based and structural interventions aimed at improving perinatal mental health outcomes.

Tables and Figures

Figure 1. Analytical sample selection, California, 1997-2018 (n=10,155,036)

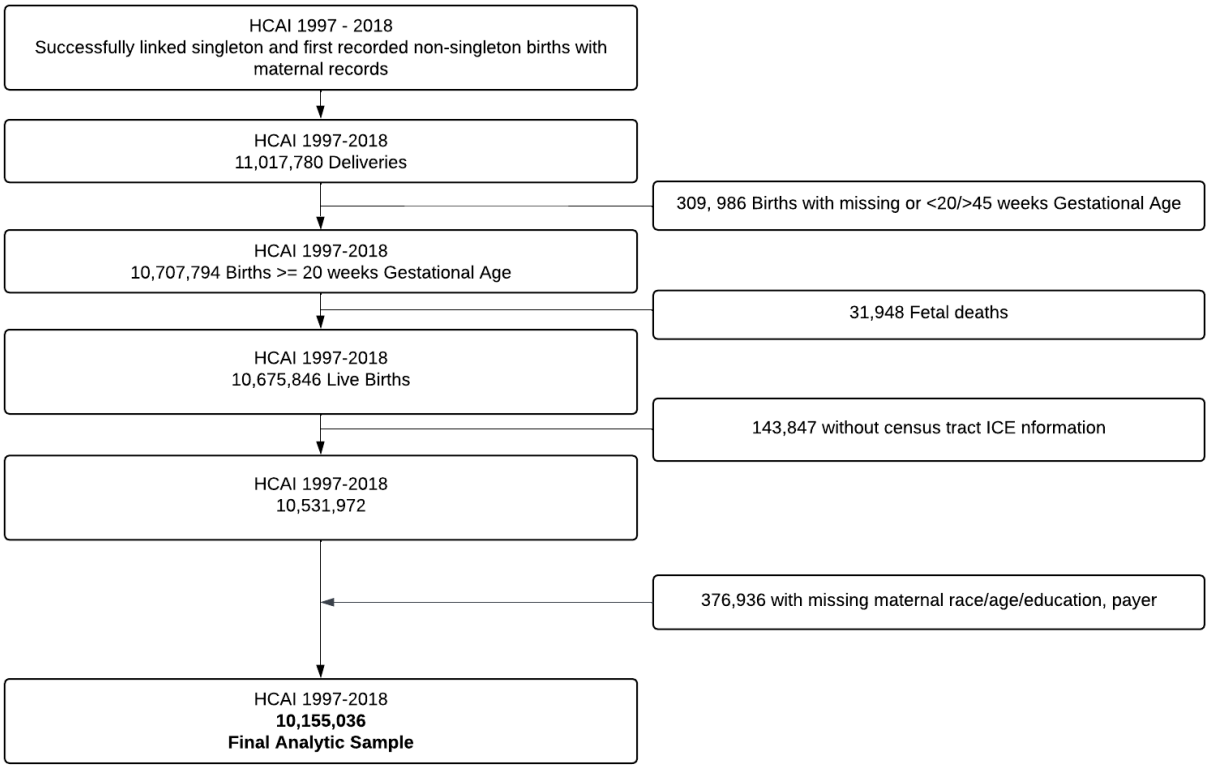


Table 3. Distribution of population characteristics among racial/ethnic groups by any PMD[a] in California, 1997-2018 (n = 10,155,036)

	White			Black			Asian or Pacific Islander			Hispanic			American Indian or Alaska Native		
	Overall (N (%))	No PMD (%)	PMD (%)	Overall (N (%))	No PMD (%)	PMD (%)	Overall (N (%))	No PMD (%)	PMD (%)	Overall (N (%))	No PMD (%)	PMD (%)	Overall (N (%))	No PMD (%)	PMD (%)
Total	2,997,229 (29.5%)	96.8	3.2	585,208 (5.8%)	97.6	2.4	1,362,859 (13.4%)	99.1	0.9	5,160,341 (50.8%)	98.7	1.3	42,933 (0.4%)	96.3	3.7
Maternal age															
<20	128,835 (4.3%)	4.3	3.4	73,848 (12.6%)	12.8	7.1	29,708 (2.2%)	2.2	1.6	615,559 (11.9%)	12.0	6.9	5,321 (12.4%)	12.6	7.8
20-34	2,169,855 (72.4%)	72.5	69.1	429,092 (73.3%)	73.3	73.6	968,644 (70.8%)	71.2	63.0	3,871,176 (75.0%)	75.1	72.3	32,237 (74.9%)	75.1	74.9
≥35	698,539 (23.3%)	23.2	27.5	82,268 (14.1%)	13.9	19.3	364,507 (26.7%)	26.7	35.5	673,606 (13.1%)	13.0	20.8	5,375 (12.4%)	12.3	17.3
Maternal education															
High school or less	870,783 (29.1%)	29.1	28.7	304,329 (52.0%)	52.1	48.3	302,985 (22.1%)	22.3	17.0	3,754,170 (72.8%)	73.0	53.5	26,042 (60.5%)	60.8	55.9
Some college	815,529 (27.2%)	27.1	31.6	191,192 (32.7%)	32.5	38.0	303,064 (22.2%)	22.2	26.5	985,257 (19.1%)	18.9	31.9	12,058 (28.1%)	27.9	33.1
College graduate	1,310,917 (43.7%)	43.9	39.7	89,687 (15.3%)	15.4	13.7	756,810 (55.7%)	55.5	56.5	420,914 (8.2%)	8.1	14.6	4,833 (11.4%)	11.3	11.0
Insurance payer															
Private	2,250,735 (75.1%)	75.2	71.0	242,288 (41.4%)	41.4	43.2	954,101 (70.1%)	69.9	79.5	1,656,694 (32.1%)	31.8	52.4	17,148 (40.0%)	40.1	35.0
Public or government	689,709 (23.0%)	22.9	27.8	328,736 (56.1%)	56.2	55.7	304,212 (22.3%)	22.4	18.7	3,363,293 (65.1%)	65.4	46.5	24,695 (57.5%)	57.3	63.4
Uninsured or other	56,785 (1.9%)	1.9	1.2	14,184 (2.4%)	2.5	1.2	104,546 (7.6%)	7.7	1.8	141,354 (2.7%)	2.8	1.2	1,090 (2.5%)	2.6	1.6
ICE race-income															
T1 (least privileged)	557,980 (18.6%)	18.6	20.6	376,316 (64.3%)	64.5	57.5	356,910 (26.2%)	26.2	20.3	2,885,927 (55.9%)	56.1	42.1	18,356 (42.8%)	42.7	45.6
T2	1,032,769 (34.5%)	34.4	34.9	143,904 (24.6%)	24.5	29.3	538,826 (39.5%)	39.5	40.1	1,653,347 (32.0%)	32.0	37.5	16,144 (37.6%)	37.7	34.8
T3 (most privileged)	1,406,480 (46.9%)	47.0	44.5	64,988 (11.1%)	11.1	13.2	467,123 (34.3%)	34.2	39.6	621,067 (12.0%)	11.9	20.5	8,433 (19.6%)	19.6	19.7

[a] PMD = perinatal mental disorders

Table 4. Associations between ICE race-income and overall perinatal mental health status, California, 1997-2018 (n = 10,155,036)

	PMAD[a]		SMI[b]		Any PMD[c]	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
			White			
T1 (least privileged)	1.08 (1.04, 1.13)	1.04 (1.00, 1.08)	1.95 (1.85, 2.06)	1.30 (1.23, 1.37)	1.16 (1.12, 1.21)	1.24 (1.19, 1.29)
T2	1.06 (1.03, 1.10)	1.04 (1.01, 1.08)	1.30 (1.24, 1.36)	1.06 (1.01, 1.11)	1.07 (1.03, 1.10)	1.11 (1.07, 1.15)
T3 (most privileged)	Ref	ref	ref	ref	ref	ref
			Black			
T1 (least privileged)	0.71 (0.66, 0.77)	0.72 (0.67, 0.78)	1.06 (0.95, 1.18)	0.80 (0.72, 0.90)	0.81 (0.76, 0.87)	0.75 (0.70, 0.81)
T2	1.01 (0.95, 1.10)	1.01 (0.94, 1.10)	1.12 (1.0, 1.26)	0.97 (0.86, 1.09)	1.05 (0.97, 1.13)	1.00 (0.93, 1.07)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Asian or Pacific Islander			
T1 (least privileged)	0.66 (0.61, 0.72)	0.77 (0.72, 0.83)	0.89 (0.78, 1.0)	0.82 (0.73, 0.93)	0.70 (0.64, 0.76)	0.78 (0.72 0.84)
T2	0.89 (0.84, 0.94)	0.94 (0.89, 1.00)	0.99 (0.89, 1.09)	0.97 (0.88, 1.08)	0.91 (0.85, 0.96)	0.95 (0.89, 1.00)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Hispanic			
T1 (least privileged)	0.45 (0.44, 0.47)	0.65 (0.62, 0.68)	0.55 (0.52, 0.58)	0.67 (0.63, 0.71)	0.47 (0.45, 0.49)	0.65 (0.62, 0.68)
T2	0.71 (0.68, 0.74)	0.85 (0.82, 0.89)	0.76 (0.71, 0.80)	0.83 (0.78, 0.89)	0.72 (0.69, 0.75)	0.85 (0.81, 0.88)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			American Indian and Alaska Native			
T1 (least privileged)	0.99 (0.84, 1.19)	0.97 (0.81, 1.16)	1.67 (1.28, 2.19)	1.21 (0.91, 1.61)	1.12 (0.95, 1.32)	1.03 (0.87, 1.21)
T2	0.92 (0.77, 1.09)	0.90 (0.76, 1.08)	1.23 (0.93, 1.63)	1.00 (0.75, 1.34)	0.96 (0.81, 1.14)	0.91 (0.77, 1.08)
T3 (most privileged)	ref	ref	ref	ref	ref	ref

[a] PMAD = perinatal mood and anxiety disorders
[b] SMI = serious mental illness
[c] PMD = perinatal mental disorders
[c] Model 1 was adjusted for maternal age
[d] Model 2 was adjusted for maternal age, education, and insurance payer

Supplementary materials

Supplement Table 1. ICD-9-CM and ICD-10-CM codes[a] for PMAD[b] and SMI[c]

PMD[d] Category	Diagnoses	ICD-9	ICD-10 codes
PMAD	Depression: major depressive affective disorder, depressive disorder Anxiety: anxiety disorder, phobia, obsessive-compulsive disorder, acute stress reaction, posttraumatic stress disorder, overanxious disorder	296.20, 296.21, 296.22, 296.23, 296.24, 296.25, 296.26, 296.30, 296.31, 296.32, 296.33, 296.34, 296.35, 296.36, 300.4, 311, 293.84, 300.00, 300.01, 300.02, 300.09, 300.10, 300.20, 300.21, 300.22, 300.23, 300.29, 300.3, 300.5, 300.89, 300.9, 308.0, 308.1, 308.2, 308.3, 308.4, 308.9, 309.81, 313.0, 313.1, 313.21, 313.22, 313.30, 313.82, 313.83	F329, F320, F321, F322, F323, F324, F325, F339, F330, F331, F332, F333, F3341, F3342, F4341, F329, F064, F419, F410, F411, F418, F449, F409, F4001, F4002, F4011, F40218, F42, F488, F458, F489, F430, F4310, F938, F328, F3340, F064, F413, F4000, F4010, F40210, R4681, Q871, F609, F068, F410, R451, F438, F439, F4311, F40298, F329, F948, F845, Z62891, Z553, F419, F40220, R457, F4312, F40228, F40230, F40231, F40232, F40233, F40240, F40241, Z733, F40242, F40243, F40248, F40290, F40291, F408, F5105, F5113, F99, R452
SMI	Bipolar disorder: Bipolar 1 disorder, manic disorder, other unspecified bipolar disorders and episodic mood disorders Psychotic disorders: psychotic disorder, schizophrenia, paranoid state, depressive psychosis	296.00, 296.01, 296.02, 296.03, 296.04, 296.05, 296.06, 296.40, 296.41, 296.42, 296.43, 296.44, 296.45, 296.46, 229.650, 296.51, 296.52, 296.53, 296.54, 296.55, 296.56, 296.60, 296.61, 296.62, 296.63, 296.64, 296.65, 296.66, 296.7, 296.10, 296.11, 296.12, 296.13, 296.14, 296.15, 296.16, 296.80, 296.81, 296.82, 296.89, 296.90, 296.99, 293.81, 293.82, 295.00, 295.01, 295.02, 295.03, 295.04, 295.05, 295.10, 295.11, 295.12, 295.13, 295.14, 295.15, 295.20, 295.21, 295.22, 295.23, 295.24, 295.25, 295.30, 295.31, 295.32, 295.33, 295.34, 295.35, 295.40, 295.41, 295.42, 295.43, 295.44, 295.45, 295.50, 295.51, 295.52, 295.53, 295.54, 295.55, 295.60, 295.61, 295.62, 295.63, 295.64, 295.65, 295.70, 295.71, 295.72, 295.73, 295.74, 295.75, 295.80, 295.81, 295.82, 295.83, 295.84, 295.85, 295.90, 295.91, 295.92, 295.93, 295.94, 295.95, 297.0, 297.1, 297.2, 297.3, 297.8, 297.9, 298.0, 298.1, 298.2, 298.3, 298.4, 298.8, 298.9	F329, F320, F322, F3013, F302, F303, F304, F3110, F3111, F3112, F3113, F312, F3173, F3174, F3130, F3131, F3132, F314, F315, F3175, F3176, F3160, F3161, F3162, F3163, F3164, F3177, F3178, F319, F3010, F3011, F3012, F308, F328, F3181, F39, F348, F062, F060, F2089, F201, F202, F200, F2081, F205, F259, F209, F22, F24, F23, F323, F28, F4489, F29, F309, F3189, F3170, F3172, F3171, F349, F338, F0630, R401, F061, F21, F251, F250, F258, F203, F528, F09, R140

[a.] based on prior work by McKee et al. 2020
[b] PMAD = perinatal mood and anxiety disorders
[c] SMI = serious mental illness
[d] PMD = perinatal mood disorders

Supplement Table 2. Distribution of population characteristics among racial and ethnic groups by PMAD[a] status, California, 1997-2018 (n = 10,155,036)

	White			Black			Asian or Pacific Islander			Hispanic			American Indian or Alaska Native		
	Overall (N (%))	No PMAD (%)	PMAD (%)	Overall (N (%))	No PMAD (%)	PMAD (%)	Overall (N (%))	No PMAD (%)	PMAD (%)	Overall (N (%))	No PMAD (%)	PMAD (%)	Overall (N (%))	No PMAD (%)	PMAD (%)
Total	2,997,229 (29.5%)	97.2	2.8	585,208 (5.8%)	98.1	1.9	1,362,859 (13.4%)	99.2	0.8	5,160,341 (50.8%)	98.9	1.1	42,933 (0.4%)	97.1	2.9
Maternal age															
<20	128,835 (4.3%)	4.3	2.8	73,848 (12.6%)	12.7	6.8	29,708 (2.2%)	2.2	1.4	615,559 (11.9%)	12.0	6.4	5,321 (12.4%)	12.6	7.0
20-34	2,169,855 (72.4%)	72.5	68.7	429,092 (73.3%)	73.3	73.9	968,644 (70.8%)	71.1	63.0	3,871,176 (75.0%)	75.1	72.3	32,237 (74.9%)	75.1	75.8
≥35	698,539 (23.3%)	23.2	28.4	82,268 (14.1%)	14.0	19.3	364,507 (26.7%)	26.7	35.6	673,606 (13.1%)	13.0	21.3	5,375 (12.4%)	12.4	17.2
Maternal education															
High school or less	870,783 (29.1%)	29.2	25.8	304,329 (52.0%)	52.2	44.0	302,985 (22.1%)	22.3	15.8	3,754,170 (72.8%)	73.0	52.4	26,042 (60.5%)	60.9	55.4
Some college	815,529 (27.2%)	27.1	31.4	191,192 (32.7%)	32.5	40.0	303,064 (22.2%)	22.2	26.1	985,257 (19.1%)	18.9	32.3	12,058 (28.1%)	27.9	34.1
College graduate	1,310,917 (43.7%)	43.8	42.8	89,687 (15.3%)	15.3	16.0	756,810 (55.7%)	55.5	58.0	420,914 (8.2%)	8.1	15.3	4,833 (11.4%)	11.2	12.5
Insurance payer															
Private	2,250,735 (75.1%)	75.1	75.0	242,288 (41.4%)	41.2	49.7	954,101 (70.1%)	69.9	81.7	1,656,694 (32.1%)	31.8	54.6	17,148 (40.0%)	40.0	38.8
Public or government	689,709 (23.0%)	23.0	23.9	328,736 (56.1%)	56.3	49.4	304,212 (22.3%)	22.4	16.4	3,363,293 (65.1%)	65.4	44.3	24,695 (57.5%)	57.5	59.6
Uninsured or other	56,785 (1.9%)	1.9	1.2	14,184 (2.4%)	2.5	1.0	104,546 (7.6%)	7.7	1.8	141,354 (2.7%)	2.8	1.1	1,090 (2.5%)	2.6	1.6
ICE race-income															
T1 (least privileged)	557,980 (18.6%)	18.6	18.6	376,316 (64.3%)	64.5	54.6	356,910 (26.2%)	26.2	19.4	2,885,927 (55.9%)	56.1	41.2	18,356 (42.8%)	42.7	43.4
T2	1,032,769 (34.5%)	34.5	34.8	143,904 (24.6%)	24.5	31.1	538,826 (39.5%)	39.5	40.2	1,653,347 (32.0%)	32.0	37.9	16,144 (37.6%)	37.7	35.5
T3 (most privileged)	1,406,480 (46.9%)	47.0	46.6	64,988 (11.1%)	11.0	14.3	467,123 (34.3%)	34.2	40.4	621,067 (12.0%)	11.9	21.0	8,433 (19.6%)	19.6	21.1

[a] PMAD = perinatal mood and anxiety disorders

Supplement Table 3. Distribution of population characteristics among racial and ethnic groups by SMI[a] status, California, 1997-2018 (n = 10,155,036)

	White			Black			Asian or Pacific Islander			Hispanic			American Indian or Alaska Native		
	Overall (N (%))	No SMI (%)	SMI (%)	Overall (N (%))	No SMI (%)	SMI (%)	Overall (N (%))	No SMI (%)	SMI (%)	Overall (N (%))	No SMI (%)	SMI (%)	Overall (N (%))	No SMI (%)	SMI (%)
Total	2,997,229 (29.5%)	99.2	0.8	585,208 (5.8%)	99.1	0.9	1,362,859 (13.4%)	99.8	0.2	5,160,341 (50.8%)	99.7	0.3	42,933 (0.4%)	98.7	1.3
Maternal age															
<20	128,835 (4.3%)	4.3	5.0	73,848 (12.6%)	12.7	7.7	29,708 (2.2%)	2.2	2.5	615,559 (11.9%)	11.9	8.5	5,321 (12.4%)	12.4	9.0
20-34	2,169,855 (72.4%)	72.4	69.9	429,092 (73.3%)	73.3	72.7	968,644 (70.8%)	71.1	60.8	3,871,176 (75.0%)	75.0	71.9	32,237 (74.9%)	75.1	75.2
≥35	698,539 (23.3%)	23.3	25.1	82,268 (14.1%)	14.0	19.6	364,507 (26.7%)	26.7	36.7	673,606 (13.1%)	13.0	19.7	5,375 (12.4%)	12.4	18.8
Maternal education															
High school or less	870,783 (29.1%)	29.0	37.1	304,329 (52.0%)	52.0	55.9	302,985 (22.1%)	22.3	20.6	3,754,170 (72.8%)	72.8	55.6	26,042 (60.5%)	60.7	61.4
Some college	815,529 (27.2%)	27.2	32.9	191,192 (32.7%)	32.7	34.7	303,064 (22.2%)	22.2	27.5	985,257 (19.1%)	19.1	32.4	12,058 (28.1%)	28.0	32.2
College graduate	1,310,917 (43.7%)	43.9	30.1	89,687 (15.3%)	15.4	9.4	756,810 (55.7%)	55.5	52.0	420,914 (8.2%)	8.1	12.0	4,833 (11.4%)	11.3	6.4
Insurance payer															
Private	2,250,735 (75.1%)	75.3	56.0	242,288 (41.4%)	41.5	30.4	954,101 (70.1%)	70.0	69.5	1,656,694 (32.1%)	32.1	42.7	17,148 (40.0%)	40.2	22.2
Public or government	689,709 (23.0%)	22.9	42.8	328,736 (56.1%)	56.1	68.3	304,212 (22.3%)	22.3	28.9	3,363,293 (65.1%)	65.2	56.1	24,695 (57.5%)	57.3	76.3
Uninsured or other	56,785 (1.9%)	1.9	1.2	14,184 (2.4%)	2.4	1.4	104,546 (7.6%)	7.7	1.7	141,354 (2.7%)	2.7	1.1	1,090 (2.5%)	2.6	1.5
ICE race-income															
T1 (least privileged)	557,980 (18.6%)	18.5	27.9	376,316 (64.3%)	64.3	62.7	356,910 (26.2%)	26.2	24.3	2,885,927 (55.9%)	56.0	45.3	18,356 (42.8%)	42.7	51.3
T2	1,032,769 (34.5%)	34.5	34.8	143,904 (24.6%)	24.6	26.3	538,826 (39.5%)	39.5	39.7	1,653,347 (32.0%)	32.0	36.1	16,144 (37.6%)	37.7	33.6
T3 (most privileged)	1,406,480 (46.9%)	47.0	37.3	64,988 (11.1%)	11.1	11.1	467,123 (34.3%)	34.3	36.0	621,067 (12.0%)	12.0	18.6	8,433 (19.6%)	19.7	15.1

[a] SMI = serious mental illness

Supplement Table 4. Associations between ICE income and overall perinatal mental health status, 1997-2018 (n = 10,155,036)

	PMAD[a]		SMI[b]		Any PMD[c]	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
			White			
T1 (least privileged)	1.09 (1.05, 1.14)	1.05 (1.00, 1.09)	1.95 (1.85, 2.1)	0.92 (0.83, 1.03)	1.24 (1.19, 1.29)	1.09 (1.05, 1.14)
T2	1.10 (1.06, 1.14)	1.08 (1.04, 1.11)	1.31 (1.24, 1.36)	0.96 (0.86, 1.08)	1.14 (1.10, 1.18)	1.08 (1.04, 1.11)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Black			
T1 (least privileged)	0.80 (0.73, 0.86)	0.83 (0.76, 0.90)	1.23 (1.11, 1.37)	0.92 (0.83, 1.03)	0.92 (0.86, 0.99)	0.87 (0.80, 0.94)
T2	0.98 (0.90, 1.06)	0.98 (0.91, 1.07)	1.12 (1.00, 1.26)	0.96 (0.86, 1.08)	1.01 (0.94, 1.10)	0.98 (0.90, 1.05)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Asian or Pacific Islander			
T1 (least privileged)	0.73 (0.67, 0.80)	0.86 (0.80, 0.93)	1.01 (0.89, 1.15)	0.94 (0.82, 1.06)	0.78 (0.72, 0.85)	0.88 (0.81, 0.94)
T2	0.93 (0.87, 0.99)	0.99 (0.93, 1.05)	1.05 (0.95, 1.16)	1.00 (0.91, 1.12)	0.95 (0.89, 1.0)	0.99 (0.93, 1.05)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Hispanic			
T1 (least privileged)	0.48 (0.46, 0.51)	0.70 (0.67, 0.73)	0.59 (0.56, 0.63)	0.73 (0.68, 0.77)	0.51 (0.48, 0.52)	0.70 (0.67, 0.73)
T2	0.70 (0.67, 0.73)	0.84 (0.81, 0.88)	0.77 (0.72, 0.82)	0.86 (0.80, 0.91)	0.70 (0.67, 0.73)	0.84 (0.80, 0.87)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			American Indian and Alaska Native			
T1 (least privileged)	0.98 (0.83, 1.16)	0.95 (0.80, 1.13)	1.66 (1.25, 2.20)	1.15 (0.85, 1.56)	1.07 (0.92, 1.26)	0.97 (0.82, 1.14)
T2	0.96 (0.80, 1.14)	0.95 (0.79, 1.13)	1.37 (1.01, 1.84)	1.11 (0.82, 1.52)	0.99 (0.84, 1.17)	0.94 (0.79, 1.11)
T3 (most privileged)	ref	ref	ref	ref	ref	ref

[a] PMAD = perinatal mood and anxiety disorders
[b] SMI = serious mental illness
[c] PMD = perinatal mental disorders
[c] Model 1 was adjusted for maternal age
[d] Model 2 was adjusted for maternal age, education, and insurance payer

Supplement Table 5. Associations between ICE race-ethnicity and overall perinatal mental status, 1997-2018 (n = 10,155,036)

	PMAD[a]		SMI[b]		Any PMD[c]	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
			White			
T1 (least privileged)	1.09 (1.05, 1.14)	0.96 (0.92, 1.00)	1.39 (1.32, 1.47)	1.10 (1.05, 1.17)	1.07 (1.02, 1.11)	1.00 (0.96, 1.04)
T2	1.10 (1.1, 1.4)	0.95 (0.92, 0.98)	1.02 (0.97, 1.07)	0.95 (0.91, 0.99)	0.97 (0.94, 1.00)	0.95 (0.92, 0.98)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Black			
T1 (least privileged)	0.68 (0.63, 0.74)	0.69 (0.63, 0.75)	0.79 (0.71, 0.89)	0.67 (0.60, 0.74)	0.73 (0.68, 0.78)	0.69 (0.64, 0.75)
T2	0.82 (0.75, 0.89)	0.82 (0.75, 0.89)	0.82 (0.73, 0.92)	0.74 (0.66, 0.84)	0.83 (0.77, 0.90)	0.81 (0.75, 0.87)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Asian or Pacific Islander			
T1 (least privileged)	0.59 (0.55, 0.64)	0.67, (0.62, 0.71)	0.68 (0.60, 0.76)	0.66 (0.58, 0.74)	0.61 (0.57, 0.66)	0.67 (0.62, 0.72)
T2	0.76 (0.72, 0.81)	0.80, (0.75, 0.85)	0.80 (0.72, 0.90)	0.81 (0.72, 0.90)	0.77 (0.73, 0.82)	0.80 (0.75, 0.85)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			Hispanic			
T1 (least privileged)	0.47 (0.44, 0.49)	0.61 (0.59, 0.64)	0.49 (0.46, 0.52)	0.57 (0.54, 0.61)	0.47 (0.45, 0.49)	0.60 (0.57, 0.63)
T2	0.71 (0.67, 0.74)	0.79 (0.76, 0.83)	0.73 (0.68, 0.78)	0.78 (0.73, 0.82)	0.70 (0.67, 0.73)	0.78 (0.75, 0.81)
T3 (most privileged)	ref	ref	ref	ref	ref	ref
			American Indian and Alaska Native			
T1 (least privileged)	0.71 (0.60, 0.83)	0.70 (0.60, 0.83)	0.97 (0.77, 1.23)	0.91 (0.72, 1.14)	0.80 (0.69, 0.93)	0.79 (0.68, 0.91)
T2	0.76 (0.66, 0.89)	0.77 (0.66, 0.89)	0.84 (0.68, 1.03)	0.86 (0.70, 1.06)	0.78 (0.68, 0.90)	0.79 (0.68, 0.91)
T3 (most privileged)	ref	ref	ref	ref	ref	ref

[a] PMAD = perinatal mood and anxiety disorders
[b] SMI = serious mental illness
[c] PMD = perinatal mental disorders
[c] Model 1 was adjusted for maternal age
[d] Model 2 was adjusted for maternal age, education, and insurance payer

Conclusion

This dissertation aims to evaluate the trends and racial and ethnic disparities in PMD and examine and the relationship between structural racism and these outcomes among a diverse cohort in California.

In chapter one, we examined trends and racial and ethnic disparities in perinatal mental disorders in California from 1997 to 2018. We found an overall growing prevalence of perinatal mental disorders, including perinatal mood and anxiety disorders and serious mental illness in California from 1997 to 2018. Additionally, we found some evidence of racial and ethnic disparities for both PMAD and SMI among American Indian and Alaska Native birthing persons compared to White birthing persons. For Black birthing persons, we found some evidence of disparities in SMI, but not for PMAD, although adjustment of sociodemographic variables nullified results; none were evident for PMAD. While there were minimal variations in disparities over time for Asian and Hispanic birthing persons across all mental illness diagnostic categories, both increasing and decreasing disparities were seen over time for Black birthing persons compared to their White counterparts.

In chapter two, our systematic review on the structural racism-mental health and substance use association yielded 7 studies. Most of the studies found significant associations between structural racism and adverse mental health and substance use outcomes, particularly among Black and other racialized populations. Higher levels of structural racism were linked to increased psychiatric visits, depressive and anxiety symptoms, as well as adverse substance use related outcomes, including heavy drinking and consuming alcohol or drugs prior to sex. These findings suggest that structural racism may play a significant role in patterning mental health and substance use inequities across the lifecourse.

In chapter three, we utilized census tract-level racial and economic polarization as a measure of structural racism to examine its association with PMAD, SMI, and their combination (overall PMD) in California from 1997 to 2018. We found that the relationship between structural racism and perinatal mental illness varied by race and ethnicity. Black and Asian or Pacific Islander birthing persons that lived in the most polarized neighborhoods had a reduced risk of PMAD, SMI and overall PMD. Conversely, for white birthing persons, we found that living in the most polarized neighborhoods – those that have a greater proportion of low-income black individuals – increased their risk of all perinatal mental illness diagnoses. In our sensitivity analyses, we found that when using ICE-race, living in the most racially segregated neighborhood (i.e., living in majority-black neighborhoods) decreased the risk of diagnosis of all mental health outcomes for all racial groups. These findings suggest that polarized neighborhoods play a role in barriers to adequate mental health diagnosis and care for minoritized birthing persons.

This dissertation seeks to advance knowledge on the racial disparities and drivers of PMD. Understanding the impact of structural racism on perinatal mental health is crucial for not only identifying underlying causes, but also for informing effective, multi-sectoral public health interventions and population-level policies aimed at eliminating these inequities, and ultimately improving the mental health of birthing populations in this country.

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