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The Experience of BIPOC Living with Chronic Pain in the USA: Biopsychosocial Factors that Underlie Racial Disparities in Pain Outcomes, Comorbidities, Inequities, and Barriers to Treatment

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Abstract

Purpose of Review—This review synthesizes recent findings related to the biopsychosocial processes that underlie racial disparities in chronic pain, while highlighting opportunities for interventions to reduce disparities in pain treatment among BIPOC.

Recent Findings—Chronic pain is a prevalent and costly public health concern that disproportionately burdens Black, Indigenous, and people of color (BIPOC). This unequal burden arises from an interplay among biological, psychological, and social factors.

Summary—Social determinants of health (e.g., income, education level, and lack of access or inability to utilize healthcare services) are known to affect overall health, including chronic pain, and disproportionately affect BIPOC communities. This burden is exacerbated by exposure to psychosocial stressors (i.e., perceived injustice, discrimination, and race-based traumatic stress) and can affect biological systems that modulate pain (i.e., inflammation and pain epigenetics). Further, there are racial/ethnic disparities in pain treatment, perpetuating the cycle of undermanaged chronic pain among BIPOC.

Keywords

BIPOC; Race; Biopsychosocial; Chronic pain; Disparities

Introduction

Chronic pain affects over 100 million Americans and is recognized as a major public health concern. While chronic pain affects all segments of the population, the burden of pain is unequal across ethnic/racial groups [1]. Health inequities among BIPOC in the USA have developed from interpersonal and structural racism [2, 3]. Decades of discriminatory practices, policies, and events, such as White Flight and residential segregation, have led

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to an unequal social landscape. Discrimination—ranging from subtle microaggressions to institutional or policy-level barriers—adversely affects both mental and physical health [4•, 5].

The biopsychosocial model posits that pain is determined by complex interactions between biological processes, social determinants of health, and various psychological factors. For example, early and prolonged exposure to psychological and environmental risk factors, such as trauma, perceived injustice, poverty, and neighborhood disadvantage, can alter biological systems including pain processing, inflammatory expression, and pain epigenetics [6]. The purpose of this review is to synthesize recent findings related to the biopsychosocial processes that underlie racial disparities in chronic pain and pain treatment (see Fig. 1).

Psychological Factors Associated with Racial Disparities in Chronic Pain

A myriad of psychological factors have been linked to chronic pain. It is well known that BIPOC experience high rates of discrimination specifically related to their racial and ethnic identities that can affect their mental health and perceptions of healthcare. For example, a recent study found that between 50 and 75% of Black, Hispanic, and Asian respondents have experienced racial discrimination in their lifetimes [20]. Moreover, one study showed that over 30% of NHB Americans face discrimination in healthcare encounters and more than 20% avoid seeking healthcare due to anticipated discrimination or poor treatment [21]. Racial discrimination negatively influences mental health resulting in PTSD in Latinx adults [5], depression in African American youth [22], suicidality in people of color [23], and race-based traumatic stress (i.e., emotional or physical pain, including depression, hypervigilance, and headaches, resulting from racism or hostility) across BIPOC [24, 25].

Burgeoning research continues to examine the interplay between perceived racial discrimination and pain outcomes. For example, Bakhshaie et al. explored how anxiety sensitivity, or fear of the negative consequences of anxiety, may play a role in the relationship between perceived discrimination and pain in a sample of Spanish-speaking Latinos seeking health services at a Federally Qualified Health Center [4•]. As expected, greater perceived discrimination was related to greater pain intensity and pain disability. It is notable that anxiety sensitivity partially explained this relationship. Bakhshaie et al. posit that greater exposure to racial discrimination toward Latinos may lead to greater experience of negative emotions (e.g., anxiety and shame), which may result in increased fear and avoidance of such uncomfortable emotional states [4•]. This sensitivity to and inclination to avoid such discomfort may result in worse pain over time. Recent research continues to support the associations between anxiety sensitivity, pain outcomes, and functional impairment in Latinx groups [26, 27].

Aside from discrimination, perceived pain-related injustice, a relatively new psychological construct, has emerged to help explain pain disparities. Perceived pain-related injustice is conceptualized as cognitive appraisals reflecting the externalization of blame and feelings of unfairness about a pain- or injury-related loss, along with the severity and irreparability of the loss [7, 8]. A growing body of research shows that perceived injustice is a risk factor for adverse physical and psychological outcomes [9••], including greater chronicity and severity of pain [8, 9••, 10, 11], functional impairment and disability [12], psychological distress

[10], and posttraumatic stress [13, 14], as well as opioid use and worse treatment outcomes [13, 14].

Perceived injustice may be particularly important when considering the experience of pain among BIPOC, as these individuals may experience broader social inequities in concurrence with their personal appraisals of injustice regarding their pain condition. While the experience of prior racial discrimination may make BIPOC more susceptible to injustice appraisals, research on perceived pain-related injustice has predominately been studied in largely homogeneous, non-Hispanic White (NHW) samples from North America [9••, 15, 16], highlighting a critical gap within the literature and limiting the generalizability of findings. A few recent studies have examined perceived injustice using racially diverse samples and found that participants who identified as non-Hispanic Black (NHB) reported significantly higher levels of pain-related injustice appraisals compared to both NHW and Hispanic participants, although no significant differences between NHW and Hispanic participants were observed [17, 18]. Notably, one study demonstrated that greater injustice appraisals were associated with greater perceived discrimination, and that this association was only significant among NHB and Hispanic, but not White, participants [17]. Moreover, an initial qualitative exploration demonstrated that Arab culture affected how Arab-Americans conceptualize perceived injustice, emphasizing the importance of considering the cultural appropriateness of the Injustice Experience Questionnaire (IEQ), the primary method of assessing pain-related injustice [7, 19]. Overall, these initial findings suggest that there may be differences in pain-related injustice appraisals across racial and cultural groups, potentially due to the broader sociocultural context and other injustice experiences among racial minorities.

Similar to the role of discrimination and perceived injustice, perceived stress also appears to be an important factor in predicting pain in racial and ethnic minorities. In a community sample of NHB and NHW people, Booker and colleagues explored the relationship between perceived stress, movement-evoked pain, and lower extremity function [28]. Results indicated that among NHB participants, there was a relationship between greater perceived stress and greater movement-evoked pain during balance, walking, and chair stand tasks. Furthermore, greater perceived stress was significantly associated with worse physical function performance, slower walking speed, and longer chair stand completion time. However, there were no such relationships for NHW participants. Researchers conclude that perceived stress in BIPOC may be related to biological or psychosocial responses that may influence pain outcomes.

Psychological Factors and Biological Pain Processing

Recent studies employing the use of static and dynamic quantitative sensory testing (QST) suggest potential racial differences in the biological processing of pain or *nociception* [29-31]. Rassu et al. examined the extent to which discrimination, trauma, and social status across the lifespan are associated with temporal summation, a proxy measure of central sensitization (hypersensitivity generated as a consequence of alterations in the central nervous system) [32]. This sample included individuals who identified as NHW and Latinx. As predicted, results revealed that participants who identified as Latinx reported

greater trauma exposure, greater discrimination, lower social status, and greater temporal summation than NHW participants. However, contrary to study hypotheses, greater ethnic discrimination was associated with less temporal summation for the Latinx sample, and trauma experiences were uncorrelated with the pain outcome. Rassu et al. posit that these results may not be consistent with prior findings that support the relationship between greater trauma exposure and discrimination and enhanced central sensitization markers due to nuances in context (e.g., regions and SES), trauma histories (e.g., childhood vs. adult), and culture (e.g., values and beliefs) across study samples and groups [32].

Unlike the methodology described by Rassu et al., other recent studies have adapted their experimental pain models to examine multiple aspects of endogenous pain modulation (i.e., central sensitization and diffuse noxious inhibitory control). It is hypothesized that a pro-nociceptive endogenous pain modulatory balance, characterized by enhanced pain facilitation (e.g., temporal summation) and decreased pain inhibition (conditioned pain modulation), might contribute substantially to worse pain and physical function in patients with chronic pain [33-35]. Indeed, experimental pain studies have consistently reported that compared to NHW individuals, NHB, Asian, and Hispanic individuals present with lower pain tolerances and thresholds as well as less CPM and more temporal summation than their NHW counterparts [36-39].

Although the bulk of QST studies in America have focused primarily on NHB and NHW differences in pain [40], recent studies have focused on mechanisms driving pain disparities among Native Americans. Results of these studies show that Native Americans (NAs) and NHWs are similar in terms of peripheral nociceptive fiber function, endogenous pain inhibition, spinal amplification of pain, and general pain sensitivity to static QST assessments, with the exception of cold pain sensitivity. However pain-related anxiety was found to be associated with pain tolerance across several pain measures as well as CPM-related inhibition among NAs [41, 42•]. More studies with racially diverse samples are needed to better characterize the nociceptive processing of individuals from the BIPOC community as well as to better understand intragroup differences in pain processing.

Social Determinants of Health and Chronic Pain

In addition to psychological factors discussed above, recent studies have begun to examine how social determinants of health influence racial disparities in pain. Social determinants of health can be defined as the conditions in which people are born, grow, live, work, and age. Individual and community health are influenced by everything from the built environment (e.g., housing, neighborhood, and transportation) to the food environment (e.g., food apartheid) to the economic environment (e.g., jobs and wealth) to the education environment (e.g., schools and literacy) to the social environment (e.g., violence and stress) [43]. For example, the lower a person's socioeconomic position, the worse their health status [44]. In fact, zip code has been found to predict health better than an individual's genetics [45]. Inequities across these environments have patterned the health and well-being of BIPOC. A recent systematic review identified educational attainment, socioeconomic status, and occupational factors as being consistently associated with adverse outcomes in adults with chronic low back pain [46••]. Longitudinal studies further support the predictive value

of social determinants. For example, a large cohort study found that low socioeconomic status predicted greater pain interference in older adulthood [47].

Within the context of chronic pain, recent studies and metaanalyses indicate that racial/ethnic disparities in pain are complex, but still, the role of social determinants is generally consistent. A large, nationally representative study characterized pain prevalence among NHW, NHB, Hispanic, Asian American, NA, and multiracial adults [48]. Their findings indicate that pain prevalence is lowest in Asian American adults and highest in NAs and multiracial adults. High pain prevalence in NAs was attributable to their disadvantaged socioeconomic status compared to White adults. Pain prevalence in White, Black, and Hispanic adults fell between that of Asian Americans and NAs, with Hispanics showing the lowest prevalence of these groups. Black and White adults differed relatively little, although Black adults reported slightly higher prevalence of severe pain. Notably, when Black and White adults were compared at the same level of socioeconomic status, Black individuals reported less pain than Whites, including severe pain. Collectively, findings from this study support the inextricable role of social factors such as socioeconomic status in pain disparities.

Lending further support to the role of socioeconomic status to pain, a recent study found that household food insecurity was linked with chronic pain outcomes and prescription opioid use in a sample of Canadians 12 years and older. In this sample, food insecurity was a more powerful predictor than other well-established social determinants such as income and education [49]. A growing body of literature suggests that racial differences in inflammation and chronic pain may be influenced partly by diet and food insecurity. It is suggested that chronic inflammation predisposes an organism to a host of chronic ailments, including chronic pain. Given the predictive value of food insecurity on inflammation and chronic pain outcomes, its potential role in explaining pain disparities in BIPOC should be further examined.

Associations Between Social Determinants of Chronic Pain and Inflammation

Neighborhood disadvantage, which is a novel social determinant of health [50], has been linked to poorer diet quality and is associated the overconsumption of meat and carbohydrates and limited consumption of fresh produce and fish [51]. Carbohydrates and unhealthy fats consumed in excess can increase oxidative stress, which can lead to elevated levels of systemic inflammation [39]. It is well documented that a higher percentage of racial minorities in America live in disadvantaged neighborhoods, in comparison to NHW Americans [50]. BIPOC individuals who reside in lower-income areas likely have limited access to supermarkets compared to those who live in more affluent communities [52]. Thus, variability in diet among racial groups may underlie the observed racial disparities in chronic pain and related outcomes. A recent study demonstrated that a diet-based intervention can reduce pain severity and oxidative stress in chronic pain patients [53], but it is plausible that a reduced carbohydrate intervention might be most advantageous for BIPOC individuals [39].

A recent review provides evidence that systemic levels of inflammatory markers such as cytokines are elevated in individuals living with chronic pain [54]. There are pro-

inflammatory cytokines that promote inflammation as well as anti-inflammatory cytokines that decrease the inflammatory response via antagonist effects and are related to the attenuation of hyperalgesia [55]. It has also been suggested that cytokines have the potential to alter neuronal activity in both the peripheral and central nervous system [56]. Clinical studies have reported that individuals who are more pain sensitive present with a cytokine profile characterized by increased pro-inflammatory and decreased anti-inflammatory markers of inflammation [57, 58]. Such inflammation may contribute to BIPOC disparities in pain. Indeed, recent studies have consistently reported that BIPOC and individuals who are socioeconomically disadvantaged present with substantially greater circulating levels of systemic inflammation than patients who are affluent and or identify as NHW [59, 60••, 61]. Moreover, in a pooled analysis of seven studies [62], racial minorities presented with greater basal levels pro-inflammatory CRP, IL-6, and TNFα as well as lower basal levels of IL-4 and IL-10 throughout their life course. It remains unclear, however, the role of inflammation in BIPOC pain disparities. One potential mechanism is epigenetic changes.

Pain Epigenetics

Though the role of genetics in the context of chronic pain susceptibility is in its infancy, recent pain-related epigenetics studies have been beneficial in adding to the understanding of nociceptive mechanisms [63]. “Epigenetics” is a biological mechanism by which environmental stressors can directly affect the expression of genes without modifying the genetic sequence [64]. At the molecular level, three types of epigenetic modifications have been identified: non-coding RNAs (NcRNAs), acetylation, and DNA methylation [6]. Previous studies of epigenetic changes in chronic pain have primarily employed the use of DNA methylation [65].

The process of gene regulation through DNA methylation is sensitive to environmental input and life experiences [66]. As we previously discussed, the unequal burden of pain and pain-related disability experienced by BIPOC may stem, in part, from differential exposure to a host of psychosocial and environmental stressors. The frequency and duration of exposure to stressors, such as racial discrimination, poverty, adverse childhood events, and depression, have the potential to confer biological consequences [6, 67]. For example, exposure to stressful events in the early life of an individual has been linked to DNA methylation augmentations in genes that regulate stress response in the brain (e.g., glucocorticoid receptor genes) [6, 68]. These DNA methylation changes that take place during childhood have the potential to influence stress and cortisol responses well into adulthood [6]. Further, it is a well-documented chronic stress and these associated maladaptive biological processes confer increased risk for the development of chronic pain [67]. Though the primary focus of this review was to highlight biopsychosocial determinants that underlie racial disparities in chronic pain, it should be noted that other factors such as exposure to pollution can also contribute to epigenetic modifications and are factors that BIPOC communities are disproportionately exposed to [69, 70].

Taken together, this review highlights the complex interplay between biological processes, psychological factors, and social determinants of health that underlie the disproportionate

burden of chronic pain experienced by BIPOC; however, it must be noted that the impact of these mechanisms is further compounded by the racial disparities in chronic pain treatment.

Racial Disparities in Pain Treatment

Inequities in the assessment and treatment of pain among BIPOC individuals are well-documented. BIPOC individuals are consistently rated by providers as having less pain than White individuals [71, 72]. These disparities are wide-spread and can be found among children as well as adults and have even been shown among athletes. For example, Druckman and colleagues found that medical staff perceive Black collegiate athletes as having a higher pain tolerance for an ACL injury compared to White athletes [73]. The underassessment of pain among BIPOC individuals results in inadequate pain care, which extends beyond access to care (though this in itself is problematic). Indeed, BIPOC are less likely to be prescribed analgesic treatments such as opioids, are prescribed lower doses of opioids, and are less likely to be referred for specialty care [74•, 1, 75]. This pattern is also observed in the context of emergency medical services. Among traumatically injured individuals requiring emergent transport to hospitals, significantly fewer Black patients received opioids prior to arriving at the hospital compared to other ethnic groups [76, 77]. In addition, providers are more likely to discontinue opioid therapy for Black patients who test positive for illicit substances, compared to White patients [78].

There has been a recent call to not only identify racial and ethnic treatment disparities but also address these disparities [79-82]. To accomplish this, the mechanisms driving these treatment disparities must be identified. One of the primary focus areas has been on identifying bias among pain treatment providers. For example, Hirsh and colleagues found that half of providers demonstrate race and/or socioeconomic status bias in the treatment of virtual human patients with chronic pain [83]. In a follow up study, results indicated that both implicit and explicit attitudes about race influenced providers' pain treatment decisions [84•].

There is evidence that provider bias is perpetuated by multiple mechanisms. For instance, perceptual process may contribute to racial bias in pain recognition. As demonstrated by Mende-Siedlecki, White individuals have more stringent thresholds for perceiving pain on NHB faces due to a disruption in configural processing associated with other-race faces [85]. Similarly, the same group has found that there is a general bias in recognizing negative emotions on Black male faces, compared to White male faces, an effect particularly robust for pain faces [86].

Empathy plays a critical role in social behavior, including discrimination, and may further influence racial bias in pain care. Studies have demonstrated racial ingroup bias in empathy —or greater empathic response to individuals who belong to the same group as a given subject (e.g., a White person has more empathy for other White folks compared to Black folks) [87]. These racial differences in empathy may be associated with specific neural responses. Indeed, several studies demonstrated increased neural responses to perceived pain in same-race compared to other-raced individuals across brain regions including the anterior cingulate cortex (ACC), supplementary motor area (SMA), anterior insula (AI), and temporoparietal junction (TPJ) [88-90].

Despite continued efforts to identify mechanisms of racial and ethnic disparities in pain care, future research must move toward actionable steps to reduce these disparities. Researchers have begun doing so by modulating underlying neural activity in three ways: (1) shifting attention, (2) fostering intergroup relationships with other-race individuals, and (3) increasing interaction with other-race individuals. In a study asking individuals to pay attention to the painful feelings of other-race individuals, researchers found increased activity in the ACC, SMA, AI, and TPJ [89], regions related to empathy [91]. These fundings suggest that cognitive interventions aimed at understanding others' perspectives may reduce racial ingroup bias in empathy and thus alter bias related to pain treatment. Another study showed that bringing other-race individuals into a team can weaken racial ingroup bias in empathy as evidenced by comparable empathic neural responses to both same- and other-race faces on one's own team but not for an opponent team [92]. To determine whether experiences interacting with other-race individuals reduced racial ingroup bias in empathy, researchers conducted an fMRI study of participants with who engaged in considerable interracial communication and interaction. Results showed comparable ACC, SMA, AI, and somatosensory activity and empathic neural response in response to same- and other-race models of pain [93].

It is also important for providers to address their own implicit bias. Edgoose and colleagues recommend providers use the mnemonic *IMPLICIT* to help address their own biases [94•]. They suggest that providers (1) engage in *Introspection* to explore and identify their own implicit biases; (2) practice *Mindfulness* to reduce stress and increase mindful awareness which will reduce the use of problematic cognitive shortcuts that are more prevalent under pressure; (3) engage in *Perspective taking* to consider experiences from other points of view; (4) *Learn to slow down,* pausing to reflect on potential bias thereby reducing reflexive actions; (5) engage in *Individuation*, evaluating others on personal characteristics rather than characteristics of the groups to which they belong; (6) *Check messaging* such that they use evidence-based statements that embrace multiculturalism; (7) *Institutionalize fairness* by promoting change at the organizational level and moving the health system toward health equity; and (8) *Taking two*, continuing the practice of cultural humility, a life-long commitment to self-evaluation and critique, redressing power dynamics in physician–patient dynamics, and developing mutually beneficial and non-paternalistic partnerships with communities.

As described by Mathur et al., in their recent review, much work to reduce BIPOC inequities in health has focused on individual-level variables without contextualization within the broader cultural, structural, and interpersonal systems of injustice [95••]. This focus on the individual places the responsibility for change on the people experiencing systemic oppression and greater pain burden. Indeed, interventions focused on the individual have not ameliorated pain disparities. Future interventions should aim to target multiple levels of injustice in order to eliminate pain inequities.

Consistent with this framework, there needs to be a change in the way we are educating providers. For example, medical school curricula frequently misuse race which may be propagating physician bias. Specifically, there is use of imprecise and nonbiologic labels that inaccurately conflate race and ancestry, present racial ethnic differences in disease

burden without context, present links between racial groups and disease (e.g., sickle cell disease), link minorities with increased disease burden therefore pathologizing race, and teach guidelines that endorse use of racial categories in diagnosis and treatment of disease [96]. Not only does the curricula need amended to prevent the perpetuation of these issues, but additions should also be made to the curricula. In fact, there have been numerous calls made for the inclusion of anti-racism training in medical school curricula [97-99]. Such training may capitalize on the use of perspective-taking to reduce disparities.

Conclusions

It is crucial to recognize that the experience of pain is multifaceted and involves complex interactions between a range of factors. This topical review provides a summary of recent findings related to biological, psychological, and social determinants of health that underlie racial differences in pain and provides an overview of studies that address the disparities in pain treatment. Though the studies highlighted in this review provide clinicians and pain researchers with a great deal of insight regarding racial disparities in chronic pain, it must be noted that there are limitations in the research to date as well, including limited participant diversity and inconsistent operational definitions of social constructs and pain outcomes. It must also be noted that much of the research on pain and health disparities in BIPOC focus on Black and White comparisons. However, recent findings suggest that pain differences between these two groups are modest, and other groups, such as Native Americans and multiracial adults, may be at higher risk of pain. In the future, not only should researchers investigating racial disparities in chronic pain consider utilizing the biopsychosocial model but should also aim to be more racially inclusive.

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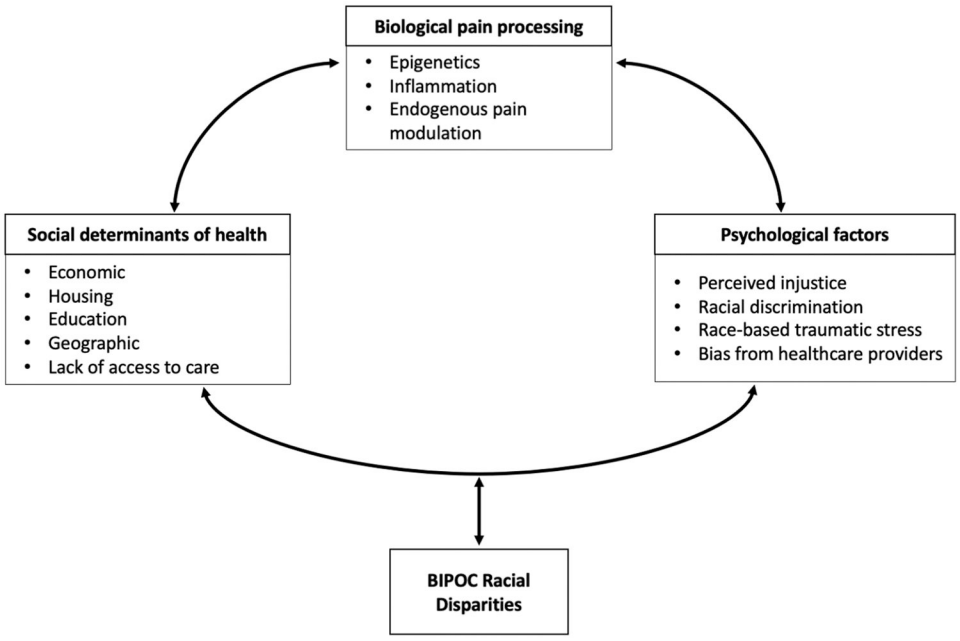

Fig. 1.
Biopsychosocial determinants of BIPOC disparities