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Epigenetics and Health Inequities in Indigenous, Black, and Hispanic Communities

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ABSTRACT

Health disparities persist along racial and ethnic lines, with Black infants being three times as likely to be born prematurely and with low birth weight as their White counterparts, underscoring broader patterns of racial and ethnic inequities in health outcomes. Drawing on interdisciplinary literature, this paper highlights how epigenetic processes, including DNA methylation and stress-related gene regulation, may biologically embed historical trauma, creating social, ethical, and policy implications. Case studies across Indigenous, Black, and Hispanic populations underscore disproportionately adverse outcomes across a range of conditions, including cardiometabolic disease, mental health disorders, and reduced life expectancy. While these disparities have been traditionally attributed to socioeconomic status, access to care, and lifestyle factors, the emerging field of social epigenetics offers a molecular perspective on how social and environmental conditions can influence the genome itself and contribute to generational health inequities. The objective of this research is to examine the role of epigenetic mechanisms in shaping health disparities, inherited disadvantage, and social immobility among three historically marginalized populations in North America, primarily the United States: Indigenous, Black, and Hispanic communities. It further seeks to identify gaps in the existing literature, suggest areas for future research, and explore possible policy interventions to address persistent disparities and inequities.

Keywords: *epigenetics, health disparities, health inequities, Black, Hispanic, Indigenous, public policy, minority populations*

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INTRODUCTION

The notion of a “generational curse”—the idea that the suffering of one generation is inherited by the next—has long existed in cultural and historical narratives. While often framed metaphorically, emerging research in epigenetics suggests that this concept may have a biological basis. This review argues that epigenetic mechanisms serve as a critical, if underexamined, biological link between structural inequities and persistent health disparities across generations. Specifically, it examines how lived experiences of historical trauma, discrimination, and chronic stress may become biologically embedded in Indigenous, Black, and Hispanic populations through molecular processes such as DNA methylation and stress-related gene regulation.

This review synthesizes current literature on epigenetic mechanisms and their role in shaping health outcomes among Indigenous, Black, and Hispanic populations in North America. Epigenetics has transformed the modern perspective of how environmental and social conditions influence human health and the inherited genome by offering an interdisciplinary framework through which trauma, social experiences, and chronic stress can shape health outcomes. By attempting to understand the cumulative impact of structural inequities at the biological level, these developments offer a critical yet fragile and incomplete basis for understanding health disparities. As such, their application must be accompanied by ethical safeguards, inclusive research practices, and structural policy reforms. Epigenetics functions not simply as a biological mechanism but also as a mediator of structural inequality, and without careful interpretation, it risks reinforcing the very disparities it seeks to explain.

Indigenous, Black, and Hispanic populations were selected for this analysis because each has been subject to distinct but overlapping forms of structural inequity in North America, such as colonization, forced assimilation, chattel slavery, systemic racism, and immigration-related marginalization. While specific historical contexts differ across communities, these three populations share disproportionate exposure to chronic stress, environmental hazards, and barriers to healthcare, making them particularly relevant case studies for examining epigenetic pathways underlying health disparities and for understanding inequity. Before examining these populations, it is necessary to establish the biological mechanisms through which social experiences become epigenetically embedded.

THE BIOLOGICAL BASIS OF EPIGENETIC REGULATION

Epigenetics is how environmental factors affect the gene expression of an organism without modifying the DNA sequence. As articulated by Rothstein et al. (2009), epigenetics regulates gene expression through “marks,” which are chemical alterations that govern the accessibility of the gene promoter. The most extensively researched mechanism, methylation (DNAm), adds a methyl group to DNA and acts as a silencer, while

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acetylation activates genes by adding an acetyl group to histone proteins. A set of epigenetic marks constitutes the epigenome, which can be transmitted through generations through mitosis. Epigenetics is essential to an organism's ability to adapt and regulate gene expression and cellular functions. All cells in an organism contain the same DNA sequence, but play different roles through cell differentiation. In normal cells, epigenetic mechanisms determine which genes are expressed without changing the underlying genetic code. Transposable elements, or "jumping genes," are sequences that move around in a genome, which can increase genetic diversity at the risk of being disruptive. Epigenetic mechanisms such as methylation allow the cell to silence harmful transposable elements (Rothstein et al., 2009).

The study also noted that genomic imprinting is the process by which only one copy of a gene—the maternal or paternal—is expressed. This substantiates the parental conflict hypothesis, which posits that certain paternal genes induce the extraction of resources from the mother to enhance the offspring's development and the father's reproductive success, whereas maternal genes prioritize conserving the mother's resources for her own survival and future offspring. Epigenetics also serves as a rapid feedback mechanism that allows a developing organism, in utero or postnatally, to adjust its genetic response to increase fitness. While essential to the organism, epigenetics can also give rise to adverse effects, such as cancer, adult-onset diseases, and transgenerational effects (Rothstein et al., 2009).

Social epigenetics is an emerging field that examines how social and environmental factors, such as stress, socioeconomic status, and exposure to harmful environments, can influence epigenetic expression and health inequalities. Mechanisms, including DNAm, histone modifications, and microRNAs, interact to regulate gene expression, yet are often studied independently (Martin et al., 2022). Epigenetics provides a molecular link between lived experiences and long-term, transgenerational health outcomes. In 2016, the National Institute for Minority Health and Health Disparities (NIMHD), the National Cancer Institute (NCI), and later the NIH, funded an initiative focused on the connection between social epigenomics and minority health disparities (Cerdena, 2022). Although social epigenetics has gained increasing recognition, significant limitations persist, particularly in research that simplifies the complexity of racial issues and fails to capture the interaction of the social construct with structural inequities.

INDIGENOUS POPULATION

Indigenous groups in North America disproportionately struggle with diet-related conditions such as obesity, alcohol abuse, and type II diabetes, as well as infectious diseases such as pertussis, chlamydia, hepatitis A, and tuberculosis. In 2021, American Indian and Alaska Native (AIAN) populations had the lowest life expectancy at birth (70.6 years) compared to Asian/Pacific Islanders (84.1 years), and non-Hispanic Whites (76.3 years), highlighting persistent racial and ethnic health disparities (Singh et al., 2024). Among the most

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significant health burdens is type 2 diabetes (T2D), which is three to five times more likely among Indigenous individuals, with Indigenous youth aged 10-19 exhibiting more than double the prevalence compared to non-Indigenous peers (Munns et al., 2025).

The epigenetic dimension of T2D risk in Indigenous populations is particularly important because disease progression of T2D often begins years before clinical diagnosis; robust biomarkers are critical for early identification and intervention. Current screening centers primarily focus on plasma glucose concentration and glycated hemoglobin (HbA1c), but these strategies do not fully capture early or intergenerational risk. Advances in epigenetics and the study of DNA methylation offer avenues for identifying biomarkers of T2D; genomic analysis across the global population has identified hundreds of loci linked to T2D susceptibility, with emerging evidence that Indigenous subsets exhibit distinct DNA methylation patterns compared to broader cohorts. Evidence also indicates a higher incidence of T2D among individuals born to First Nations mothers with T2D during pregnancy compared to those born to mothers with gestational diabetes or no diabetes, suggesting intergenerational biological transmission of risk. Thus, DNA methylation serves as a promising and biologically robust biomarker for T2D risk and progression (Munns et al., 2025).

Beyond cardiometabolic conditions, advancements in epigenetics research provide a possible connection between historic and ongoing abuses—such as residential schooling, physical and sexual abuse, cultural suppression, and forced assimilation—and the transmission of intergenerational trauma, resulting in increased long-term health disparities and allostatic load, a measure of physiological damage. A study by Kathrina Chief Moon-Riley (2019) examined the association between parental residential school attendance and the resulting adverse childhood experiences (ACEs) and allostatic load among Indigenous students at the University of Lethbridge. Residential schools were church-run institutions designed to assimilate Indigenous children aggressively, which led to physical abuses as well as a disruption of family structures, parenting practices, and cultural suppression. Results indicated that participants whose mothers attended residential schools had significantly higher allostatic load scores than those whose mothers did not; these individuals were five times more likely to have an allostatic load score in the mid-range than the low range. After controlling for age, gender, the mother's education level, and perceived socioeconomic status, the study yielded that participants whose mothers attended residential schools were 12.6 times more likely to be in the mid-range. Critically, this association was not significantly related to parenting style, suggesting biological intergenerational transmission rather than direct psychological and social influence—a finding consistent with epigenetic mechanisms of stress embedding (Chief Moon-Riley, 2019).

Inequities persist in the representation of Indigenous populations within the reference genome. The most widely used human reference genome, GRCh38, is derived primarily from African and European ancestry; millions of single-nucleotide variants identified in Indigenous populations are absent from major global

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genomic databases. The failure to account for population-specific differences risks introducing bias into biomarkers, invalidating their accuracy for Indigenous populations that experience the highest burden of the disease. Addressing these gaps is critical to ensure that advances in medicine address, rather than exacerbate, existing health inequities (Munns et al., 2025).

BLACK POPULATION

According to the National Library of Medicine, Black populations in North America are disproportionately affected by cardiometabolic diseases, including hypertension, cardiovascular diseases, and diabetes, contributing to higher mortality rates and shorter life expectancies compared to White populations. Cardiovascular disease, in particular, is significantly more prevalent, occurring in 47.3% of African Americans versus 34.1% of White Americans. As such, life expectancy is approximately four years shorter for individuals of African descent (75.2 vs. 78.8 years) (Vick & Burris, 2017). These disparities often appear earlier in life, with higher rates of hypertension, diabetes, and stroke observed in younger Black adults. In the 18-34 age range, stroke is present in 0.7% of Black individuals compared to 0.4% of their White counterparts, while high blood pressure is common in 12% of Black versus 10% of White young adults (Centers for Disease Control and Prevention, 2017). Additionally, Black infants have a 50% increased mortality risk compared to White infants, and African American women are at higher risk of early-onset breast cancer diagnoses and higher associated mortality.

Epigenetic research provides direct biological evidence linking these disparities to chronic environmental and psychosocial exposures. DNA methylation is known to be associated with risk factors linked to cardiovascular disease, breast cancer, and preterm birth. Environmental exposures such as air pollution, smoking, diet, and endocrine-disrupting chemicals—alongside chronic psychosocial stressors such as racism and discrimination—alter this epigenetic process (Vick & Burris, 2017). A stress study in mice showed how early-life adversity and the defeat stress paradigm induce long-term epigenetic changes that resemble depression, associated with reduced BDNF in the hippocampus and increased BDNF in the nucleus accumbens. Interestingly, animals that are exposed to another animal undergoing stress display the same depressive behavior—a pattern consistent with human PTSD data, suggesting that witnessing trauma can itself trigger epigenetic changes. It has also been observed that epigenetic effects can be transferred to offspring without direct behavioral contact, which supports the idea that epigenetic marks and repeated social adversity can be preserved genetically (Post, 2021).

As such, these epigenetic findings carry significant social and policy implications for African Americans, who experience disproportionate levels of discrimination, bullying, and violence, which may contribute to why this population experiences disproportionate health issues in the United States. Post (2021) discusses how

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advances in epigenetics offer new perspectives on HR 40, a bill that would begin discussions on reparations for Black Americans. The argument is not simply that historical injustice warrants remedy, but that epigenetic research provides a molecular framework demonstrating how past and ongoing discrimination may be biologically embedded across generations. Post notes, even if reparations were enacted, epigenetic marks and associated health disadvantages remain without targeted clinical intervention. For instance, shortened telomeres resulting from stress are indicators of accelerated aging and increased vulnerability to mental and physical illnesses. Studies have shown that shorter telomeres are associated with racial discrimination and that telomere length increases vulnerability to multiple relevant illnesses. Animal and human data reinforce the idea that violence, direct or indirectly witnessed, can create generational scars (Post, 2021). Separately, structural disparities in clinical care compound these biological disadvantages: emergency department data reveal that Black patients have comparatively longer wait times and lengths of stay compared to White patients and a 10% lower chance of admission (Karaca & Wong, 2013). Epigenetic evidence and structural evidence underscore the need for continued molecular research, which may spark valuable discussion and justification for reparative efforts and wider medical reforms.

HISPANIC POPULATION

Hispanic populations in the United States, irrespective of immigration status, exhibit elevated levels of chronic stress and a disproportionately higher risk of cardiometabolic conditions, including obesity and hypertension. Data indicate a 1.65-fold higher prevalence of diabetes and a 1.2-fold higher prevalence of obesity in this population. Among children aged 2-19 years, obesity rates are approximately 1.6 times higher, with particularly pronounced disparities from ages 2-5, where rates are four times higher (Clausing & Non, 2021).

Hispanic populations are more likely to be exposed to multiple chronic stressors, including legal status, uncertain immigrant policies, and acculturative stress. Notably, adverse health outcomes among Hispanic children tend to worsen with the duration of their United States residence, suggesting adverse effects of structural factors such as racism, rather than race itself, and substandard living conditions. Empirical studies suggest associations between discrimination and elevated body mass index (BMI), waist circumference, and blood pressure (Clausing & Non, 2021).

Biologically, stress-related epigenetic mechanisms provide key pathways through which chronic stress is biologically embedded. The FK506 binding protein 5 (FKBP5) gene, a central regulator of glucocorticoid receptor sensitivity within the hypothalamic-pituitary-adrenal (HPA) axis, has been widely implicated in stress-related epigenetic modification. Other common environmental stressors upon this community--including adverse childhood experiences, socioeconomic disadvantage, and discrimination--are associated with altered DNA methylation patterns in FKBP5, particularly reduced methylation at regulatory regions. This association

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has been observed in pregnant Latina women: in a sample of 147 participants, greater exposure to discrimination over time was associated with decreased methylation of the FKBP5 gene in blood (Clausing & Non, 2021).

Interestingly, a study that explored the connection between immigrant status and DNA methylation, using a sample of 2,336 nationally representative United States adults, found that immigrants experienced significantly lower GrimAge2 scores, which indicate epigenetic age. Nwanaji-Enwerem et al. (2025) found that both immigrant status and citizenship were associated with differential levels of GrimAge2-related DNAm protein markers, including adrenomedullin and C-reactive protein. This supports the idea of the immigrant mortality advantage, including the Hispanic paradox, which is an epidemiological finding that Hispanic Americans often live longer than White Americans despite a disadvantaged risk profile (Ruiz et al., 2013). Despite this phenomenon, these patterns still underscore the importance of addressing structural inequities and chronic stressors, as well as the investigation of epigenetic pathways overall, to better understand and reduce health disparities in Hispanic populations.

POLICY IMPLICATIONS

The rising significance of epigenetics in public health research signals greater recognition of the importance of trauma mitigation and the echoing implications of the historical abuses of minorities in America. However, without proper policies in place to protect patient data, the study of social epigenetics could be used to discriminate in the workplace. Originally enacted in response to state-mandated sickle cell anemia screenings, which disproportionately focused on Black Americans, the Genetic Information Nondiscrimination Act (GINA) aimed to prevent genetic discrimination in health insurance and employment (Grant, 2023). The law allows United States citizens to pursue genetic testing and learn about potential health risks without the fear that these results could influence higher insurance costs or limit job prospects. However, epigenetic data is not clearly protected under this law and can be used to decipher specific DNA sequences. The ambiguity surrounding its protections for epigenetic discrimination may open up opportunities for employers and insurance companies to bypass the original intent of GINA (Song & Li, 2025). Historical policies such as Johnson Controls' fetal-protection rule demonstrate how employers may discriminate against workers under the guise of safety, particularly fertile women, due to perceived vulnerability. Moreover, epigenetic predispositions are unlikely to qualify as disabilities under current law (Rothstein et al., 2009). Thus, regulatory frameworks such as GINA must be clearly defined to protect epigenetic information as new tests, such as the detection of abnormal methylation, become increasingly normalized.

Furthermore, addressing disparities in biomedical research is essential to ensure equitable application of epigenetic advances. As demonstrated in the Indigenous case study, the underrepresentation of minority groups in genomic databases risks producing biased diagnostic tools and ineffective treatments. GRCh38, the most

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commonly used human reference genome, is primarily based on one individual of African and European ancestry (Schneider et al., 2017). Given that many minority groups—Indigenous populations in particular—experience disproportionately worse health outcomes and exhibit population-specific genomic variants absent from standard databases, federal funding agencies and research institutions should prioritize inclusive data collection to make sure research addresses all populations equitably. Similar concerns apply to the Black and Hispanic populations discussed in this paper, where biomarker research must be validated across diverse cohorts to avoid producing tools that perform well for the majority but fail those with the greatest need. These policy considerations highlight the importance of aligning scientific advancements in epigenetics with structural reforms that address existing health disparities.

ETHICAL IMPLICATIONS

While epigenetic research could bolster genetically defined improvements in healthcare based on racial disparities, it could also open the door to the perpetuation of harmful racial bias if not approached critically and transparently. Although the term “race” is frequently used in biological and genomic research, it is more accurately understood as a socio-political construct than a biological classification, because the concept of biological race implies clear biological distinctions between races (Graves, 2023).

This distinction has direct relevance to each of the case studies in this paper. In Indigenous communities, conflating ancestry with race risks overgeneralizing findings across heterogeneous nations and populations. In the context of the Black population, the historical misuse of biological race concepts—including the false belief that Black patients have higher pain tolerance, which resulted in under-prescription of pain medication (Hoffman et al., 2016)—illustrates how epigenetic research framed around race rather than social experience can amplify rather than remedy disparities. In the Hispanic context, race-based categorization erases diversity within the population and obscures structural stressors such as immigration status and discrimination.

As such, research using ancestry data remains inconsistent and vaguely defined, reflecting a lack of standardized definitions and interpretation across studies. Approaches such as genomic sequencing and principal component analysis (PCA) offer improvements over self-reported race; however, PCA-based clustering overlooks shared social experiences, potentially complicating downstream analyses (King et al., 2024). Thus, epigenetic research must emphasize this distinction by focusing on environmental and social influences on the epigenome, which supports the concept of race as a social construct rather than a biological one.

Moreover, Santaló and Berdasco (2022) articulated that communicating epigenetic information to patients requires a balance of technical validity with ethical and practical considerations. Effective disclosure of disease risk can improve healthcare outcomes, especially in an era of personalized medicine, but must be tailored to

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individual characteristics, conveyed clearly in plain language, and delivered with sensitivity. Thus, professional training in ethics is critical to support epigenetic research. Before communicating epigenetic findings, biomarkers must meet key criteria, including replicable data accuracy, plausible causality, and clinical utility. Ethical challenges arise when risk information lacks clear clinical actionability or when the individual has limited capacity to act due to structural or environmental constraints. Disclosure must thoughtfully consider patient autonomy, potential psychological burden, and the risk of stigmatization (Santaló & Berdasco, 2022).

CONCLUSION

The study of epigenetics has provided an alternative interpretation for systemic health disparities: building on the widely accepted role of lifestyle factors in shaping health outcomes, epigenetics offers a biological framework through which lived experiences become embedded across the life course and, in some cases, across generations. As demonstrated through case studies involving Indigenous, Black, and Hispanic populations, epigenetic mechanisms and markers provide transformative insight into how measurable structural inequities are reflected not only in the present but also in the future.

However, while epigenetics supplies a powerful exploratory model, it remains incomplete and potentially dangerous when applied in isolation. The risk of misinterpretation--particularly the reification of race as a biological construct or the overemphasis of inaccessible personalized medicine--can inadvertently reinforce the very disparities this research seeks to address. These concerns are compounded by the underrepresentation of minority populations in genomic databases and the lack of regulatory protections for epigenetic data.

Moving forward, the integration of epigenetics into public health must be accompanied by careful ethical consideration, inclusive research practices, and structural policy reform. Expanding legal protections and emphasizing the social determinants of health are essential to ensure that epigenetic insights remedy, not exacerbate, existing inequities. Ultimately, epigenetics should not be viewed as a deterministic account of inherited disadvantage, but as a tool for understanding and mitigating the biological consequences of inequality.

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