

# Racial Disparities in Obesity Epigenetics: An Overview for Nurses

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## Article

### Abstract

Obesity represents a major public health challenge among adults in the United States and globally, significantly contributing to the development of chronic diseases such as cardiovascular disease, type 2 diabetes, certain cancers, osteoarthritis, and psychological disorders. Notably, the Centers for Disease Control and Prevention has reported a disproportionately high prevalence of obesity among Hispanic and African American adults compared to other racial groups. This review focuses on the role of epigenetic mechanisms, specifically DNA methylation patterns, in mediating the observed racial disparities in obesity. Epigenetic regulation, influenced by diet and activity, impacts gene expression and disease risk. Unlike genetic variation, which remains stable throughout life, epigenetic modifications are plastic and tissue-specific, making them potential biomarkers for obesity and related diseases. Nurses play an important role in understanding and addressing health disparities. By studying racial disparities in obesity epigenetics, nurses can gain insights into how genetic and environmental factors contribute to obesity among different racial groups. Understanding epigenetic factors that contribute to these disparities can help nurses develop more effective, culturally sensitive interventions to address obesity and the associated health risks.

**Key Words:** epigenetic, health disparity, obesity, diet behaviors, physical activity, DNA methylation, biomarkers

Obesity remains one of the major public health issues in the U.S. and globally. It affects more than 90 million Americans and over 600 million people worldwide (Serverin et al., 2019). Obesity contributes to chronic diseases like diabetes, heart disease, and cancer, increasing morbidity and mortality (Bulló et al., 2011; Kanwal et al., 2015; Pantalone et al., 2017; Samblas et al., 2019; Serverin et al., 2019). Evidence suggests that the average medical cost for individuals with obesity is approximately \$132 annually, which is higher than that for individuals with normal weight; this cost is projected to rise to \$48 to \$66 billion per year in the U.S. by 2030 for treating obesity and obesity-related diseases (Byrd et al., 2018; Cawley & Meyerhoefer, 2012; Centers for Disease Control and Prevention [CDC], 2020; Wang et al., 2011).

According to recent National Center for Health Statistics data, the prevalence of obesity is highest among non-Hispanic Black adults (49.6%) and Hispanic adults (44.8%), compared to non-Hispanic White adults (42.2%) and non-Hispanic Asian adults (17.4%) (Hales et al., 2020). The frequency of obesity is highest among middle-aged adults (ages 40-59) at 44.8% and older adults aged 60 and over at 42.8%, compared to younger adults aged 20-39 at 40% (Hales et al., 2017; Petersen et al., 2019). Studies suggest that common explanations for disparities in obesity include genetic and epigenetic factors, physical inactivity, and unhealthy eating behaviors (Karamangla et al., 2010; Krueger et al., 2011, 2015; Samblas et al., 2019). Recent findings indicate that epigenetic regulation, particularly DNA methylation patterns, can be modified in response to these exposures and lifestyle choices, such as eating habits, sedentary behavior, and sleep patterns, which are linked to racial disparities in obesity (Biddle et al., 2017; Kanwal et al., 2015; Mauvais-Jarvis, 2015; St-Onge, 2017; Taveras et al., 2006, 2010; Vick & Burris, 2017).

Studies have also revealed that not all individuals exposed to the same environmental factors develop obesity. The interaction between genes and the environment results in the phenotypic outcome of obesity; it has therefore been suggested that epigenetic mechanisms play a role in the development of obesity and obesity-related diseases (Akinyemiju

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et al., 2018; Sambals et al., 2019; Singh et al., 2010; Światowy et al., 2021). This review is focused on the role of epigenetic regulation, particularly DNA methylation, in response to exposure to diet behaviors and physical activity among minority groups.

Epigenetics and DNA Methylation

Epigenetics regulates gene expression without altering the DNA sequence, with DNA methylation serving as a key mechanism (Rozek et al., 2015; Williams, 2013). Methyl groups added to cytosine in CpG dinucleotides, particularly in gene promoters, typically repress transcription (Antequera & Bird, 1993; Van Dijk et al., 2015). Unmethylated CpG islands allow for gene expression (Grazioli et al., 2017; How Kit et al., 2012; Jones, 2012; McBride & Koehly, 2017; Sepulveda et al., 2009). In human cells, 5-methylcytosine accounts for 70-80% of CpG dinucleotides (Antequera & Bird, 1993; Klose & Bird, 2006; McBride & Koehly, 2017; Rozek et al., 2015; Ziller et al., 2013). While methylation suppresses gene expression, demethylation activates it (Antequera & Bird, 1993; Aslibekyan et al., 2015; How Kit et al., 2012; Klose & Bird, 2006; McBride & Koehly, 2017; Samblas et al., 2019; Ziller et al., 2013). The key aspects are summarized in Table 1.

Table 1. Key Aspects of Epigenetics and DNA Methylation

Feature/Concept	Description	Significance/Impact	Relevance to Obesity Disparities
Epigenetics Definition	Heritable changes in gene expression that occur without alterations to the underlying DNA sequence.	Crucial for regulating gene activity in response to environmental stimuli and cellular differentiation. Plays a role in development, disease, and adaptation.	Potential mediators of environmental and lifestyle influences obesity risk, potentially contributing to observed racial disparities.
Mechanisms	Includes DNA methylation, histone modifications, and non-coding RNA.	Each mechanism contributes to gene regulation at different levels and through distinct processes.	
DNA Methylation	Addition of a methyl group (CH3) to the 5' carbon of cytosine, primarily within CpG dinucleotides.	Typically represses gene transcription when located in promoter regions. Influences gene expression and cellular function.	Responsive to diet, activity, and stress; may explain racial differences in obesity-related gene expression and contribute to disparities in obesity prevalence.
CpG Islands	Regions of DNA with a high frequency of CpG sites, often found in gene promoters.	Important for regulating gene expression. Methylation of CpG islands often leads to gene silencing.	Changes in CpG methylation in these regions may link environmental exposures (common in disparate communities) to obesity-related gene regulation and contribute to health disparities.
Methylation and Transcription	Methylation of CpG sites in promoter regions often inhibits gene transcription; demethylation promotes gene expression.	Controls which genes are active or inactive in different cell types or under different environmental conditions.	Studies exploring stress, diet, and physical activity effects on epigenetic markers in minority groups, to determine what changes in DNA methylation occur, and how those changes affect obesity.
Tissue Specificity	Epigenetic marks, including DNA methylation, can vary significantly between different tissues and cell types.	Allows for tissue-specific gene expression patterns, contributing to cellular specialization.	
Plasticity	Epigenetic changes are plastic and can be influenced by environmental factors such as diet, physical activity, and stress.	Enables organisms to adapt to changing environments and may contribute to disease susceptibility or resilience.	Provides opportunities to target epigenetic modifications to mitigate obesity disparities through lifestyle interventions or targeted therapies.
Reversibility	Some epigenetic changes can be reversed through interventions, although not all.	Offers potential targets for therapeutic interventions aimed at modifying gene expression in disease states.	

<b>Biomarkers</b>	DNA methylation patterns can serve as biomarkers for various diseases, including obesity, cancer, and cardiovascular disease.	Provides a means for early detection, risk assessment, and monitoring of disease progression.	May serve as indicators of obesity risk and response to interventions in different racial groups, allowing for more targeted and personalized prevention and treatment strategies.
<b>Environmental Influence</b>	Environmental exposures can induce epigenetic changes, contributing to disease susceptibility.	Highlights the importance of lifestyle factors and environmental exposures in shaping individual health outcomes.	Explains why not all individuals with similar lifestyles develop obesity; highlights environmental contributors to disparities by showing how they can change gene expression.
<b>Transgenerational Epigenetics (Potential)</b>	Studies suggest that some epigenetic modifications may be transmitted across generations (needs more study).	Provides evidence for potential long-term impacts of environmental exposures on offspring health.	

Dynamic Nature of Epigenetics and Environmental Influences

DNA methylation patterns are dynamic, influenced by lifestyle and the environment ([Kanherkar et al., 2014](#); [Klose & Bird, 2006](#); [Rohde et al., 2019](#)). Epigenetic changes are reversible and tissue-specific, serving as potential biomarkers and targets for interventions ([James-Todd et al., 2016](#)). Environmental exposures, such as diet and stress, can alter DNA methylation and impact health ([Grazioli et al., 2017](#); [Thayer & Kuzawa, 2011](#)). Early experiences and transgenerational inheritance may also play a role ([Notterman & Mitchell, 2015](#)). Epigenetic markers link environmental factors, lifestyle, and disease risk, especially among minority groups ([Akinyemiju et al., 2018](#); [Aslibekyan et al., 2015](#); [Grazioli et al., 2017](#); [James-Todd et al., 2016](#); [Min et al., 2021](#); [Notterman & Mitchell, 2015](#); [Światowy et al., 2021](#); [Vick & Burris, 2017](#)). These dynamic epigenetic changes, influenced by environmental factors, play a critical role in the observed racial disparities in obesity. The dynamic aspects of these processes are also highlighted in [Table 1](#).

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Racial Disparities

Despite advancements in healthcare access, racial disparities in the United States continue to significantly impact minority and disadvantaged groups, leading to substantial differences in mortality and morbidity ([Marmot, 2018](#); [Obama, 2016](#); [Vick & Burris, 2017](#)). These disparities, particularly concerning obesity, stem from a complex interplay of factors, including individual behaviors, social determinants of health, and environmental influences ([Min et al., 2021](#); [Van Vliet-Ostaptchouk, 2012](#)).

Racial disparities create conditions of daily life marked by inequalities in resources, education, environmental exposures, and employment opportunities, potentially predisposing minority groups to obesity and related diseases ([Akinyemiju et al., 2018](#); [Singh et al., 2010](#); [Taveras et al., 2006, 2010](#)). Addressing these disparities is crucial, given the profound impact of obesity on health outcomes.

The fundamental cause of obesity is an energy imbalance: calories consumed exceeding calories expended. This imbalance is often attributed to societal and behavioral changes observed over recent decades ([Milliken-Smith & Potter, 2021](#)). Studies consistently demonstrate higher rates of obesity among African American and Hispanic populations, linked to environmental factors like physical inactivity, diet, and socioeconomic status ([Byrd et al., 2018](#); [Petersen et al., 2019](#)). For example, according to the CDC, the prevalence of obesity among non-Hispanic Black adults is 49.9%, and among Hispanic adults is 45.6%, compared to 41.4% among non-Hispanic White adults ([CDC, 2021](#)). Research also highlights the protective effects of healthy lifestyle habits, such as moderate alcohol consumption, balanced diets, and regular physical activity, against abdominal obesity, particularly in elderly individuals at high risk for heart disease ([Bullo et al., 2011](#); [Ntanasis-Stathopoulos et al., 2013](#)).

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Minority groups exposed to chronic stress face an increased risk for obesity and other chronic conditions. Studies have suggested that individuals raised in adverse living conditions, characterized by low-paying jobs, lack of health insurance, inadequate childcare, and polluted environments, may transmit these behaviors, lifestyles, and eating habits to subsequent generations. Limited access to healthy foods, impacting maternal and paternal nutrition, further contributes to childhood obesity risk. This social gradient of health inequality is reflected in the distribution of diet quality across minority

populations ([Ling & Ronne, 2019](#); [Ronne et al., 2013](#); [Sharp et al., 2015](#); [Taveras et al., 2006, 2010](#); [Vick & Burris, 2017](#); [Yeshurun & Hannan, 2019](#)). Further research is needed to elucidate the interactions between genes and environmental exposures, specifically how epigenetic mechanisms may differentially affect racial groups exposed to similar environmental stressors.

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Physical Activity and Epigenetic

Given the established link between environmental factors and epigenetic changes, it is essential to examine how specific lifestyle factors, such as physical activity, influence these disparities. Physical activity significantly influences gene expression related to metabolism, impacting muscle growth, diabetes, and other metabolic disorders ([Heinonen et al., 2013](#); [Kanzleiter et al., 2015](#)). Even moderate physical activity can induce global hypomethylation within muscle cells, potentially activating regulatory genes involved in repair and growth pathways ([Kanzleiter et al., 2015](#)). Notably, the intensity of physical activity correlates directly with the extent of hypomethylation and the activation of a greater number of genes ([Grazioli et al., 2017](#); [Heinonen et al., 2013](#); [Ntanasis-Stathopoulos et al., 2013](#); [Plaza-Diaz et al., 2022](#)). DNA methylation, a well-established epigenetic mechanism, plays a crucial role in regulating gene expression. While the underlying DNA sequence remains constant, environmental factors and exercise can alter the epigenetic regulation of these genes ([Grazioli et al., 2017](#)). This

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plasticity suggests a potential mechanism for how parental or grandparental exposure to physical inactivity negatively impact chronic disease susceptibility in subsequent generations ([Biddle et al., 2017](#); [Denham, 2018](#); [Kanzleiter et al., 2015](#); [Marmot, 2018](#); [Penner-Goeke & Binder, 2024](#); [Plaza-Diaz et al., 2022](#)).

The prevalence of sedentary behavior, particularly leisure-time sitting, has increased substantially due to the widespread adoption of technologies. Concurrently, both occupational and leisure-time physical activity have declined ([McCullough et al., 2015](#)). Studies have consistently demonstrated an association between prolonged television viewing and increased obesity risk, attributed to reduced energy expenditure and increased consumption of energy-dense foods ([Biddle et al., 2017](#); [Denham, 2018](#); [Pantalone et al., 2017](#); [Zhang et al., 2016](#)). Research in rats by [Kanzleiter et al. \(2015\)](#) demonstrated that regular physical activity induced hypomethylation and increased expression of genes related to muscle growth and metabolic function. These findings suggested that exercise can regulate muscle gene expression, particularly myostatin, and contribute to energy homeostasis. Furthermore, studies have shown that exercise benefits both parental health and offspring health. ([Axsom & Libonati, 2019](#); [Carter et al., 2013](#); [Gapp et al., 2014](#); [Hernández-Saavedra et al., 2022](#); [Hittel et al., 2010](#); [Leite et al., 2017](#); [McPherson et al., 2015](#); [Plaza-Diaz et al., 2022](#); [Stanford et al., 2018](#); [Yeshurun & Hannan, 2019](#)). For example, paternal physical activity during the preconception period and maternal physical activity during pregnancy have been shown to improve offspring glucose tolerance ([Axsom & Libonati, 2019](#); [Grazioli et al., 2017](#); [Hall et al., 2019](#); [Stanford et al., 2018](#)).

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Additionally, preconception paternal exercise has shown promising effects on offspring body weight and C-reactive protein levels ([Hernández-Saavedra et al., 2022](#); [Sharp et al., 2015](#); [Stanford et al., 2018](#); [Taveras et al., 2006](#); [Yeshurun & Hannan, 2019](#)). A study by [Petersen et al. \(2019\)](#) indicated a significant association between physical activity (walking or biking) and lower body mass index (BMI) and waist circumference (WC) in both White and African American populations ( $P < 0.001$ ). However, while the benefits of physical activity on gene expression through epigenetic mechanisms are well-documented, the optimal duration and intensity of physical activity required to modulate DNA methylation patterns and prevent obesity disparities remain to be fully elucidated ([Aslibekyan et al., 2015](#); [Hernández-Saavedra et al., 2022](#); [Leite et al., 2017](#); [Światowy et al., 2021](#); [Voisin et al., 2015](#)). There is a notable gap in research concerning epigenetics and obesity within minority

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populations. Therefore, further investigations are needed to identify DNA methylation biomarkers that can be used to assess the epigenetic effects of physical activity interventions in disadvantaged and minority groups. Additionally, more research is needed to determine the epigenetic effects of various types of exercise. obesity disparities among

Diet and Epigenetic

Dietary behaviors have significantly contributed to health challenges within minority groups. Several factors influence obesity development, including infant breastfeeding practices, the timing of solid food introduction, the composition of family meals, fast food consumption, and the intake of sweetened beverages ([CDC, 2020](#); [Hall et al., 2019](#); [Taveras et al., 2010](#)). Studies indicate that African American and Hispanic children are more likely to consume lower-quality foods, such as sweetened beverages and fast foods, by the age of two compared to Asian or White children ([Burdge & Lillycrop, 2010](#)). Socioeconomic status within racial and minority groups significantly influences dietary choices. Individuals with limited



financial resources often consume calorie-dense, nutritionally poor foods, such as those high in hydrogenated oils and saturated fats, while those with greater financial resources tend to consume more vegetables, lean proteins, and fresh fruits (Gapp et al., 2014; Taveras et al., 2010).

Exposure to excessive nutrition and obesogenic products can induce epigenetic modifications, predisposing individuals to obesity (Burdge & Lillycrop, 2010; Dick et al., 2014; Lorenzo et al., 2022; Severin et al., 2019). If these epigenetic changes are transmissible across generations, they may explain how parental or grandparental exposure to unhealthy eating habits, sedentary lifestyles, and social inequalities increases obesity susceptibility in subsequent generations (Heinonen et al., 2013; Izquierdo & Crujeiras, 2019; Van Vliet-Ostaptchouk et al., 2012). Research has identified several epigenome-wide methylation sites in obesity-related genes, including CPT1A, ABCG1, PGC1A, and HIF3A, demonstrating a strong association between DNA methylation patterns and body mass index or obesity (Aslibekyan et al., 2015; He et al., 2019; Lindholm et al., 2014; Loi et al., 2013; Penner-Goeke & Binder, 2024; Schröder et al., 2007; Voisin et al., 2015). Epidemiological and observational studies have linked unhealthy eating habits, such as diets low in fruits and vegetables, high in fat, and high alcohol consumption, with increased obesity risk.

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Dietary epigenetic research in humans remains limited by small sample sizes, but has suggested that epigenetic mechanisms may reinforce social health inequalities across generations (Rönn et al., 2013). For example, it is well-known that maternal nutrition during gestation significantly influences offspring obesity risk, highlighting the parental role in intergenerational obesity transmission (Antequera & Bird, 1993; Rönn et al., 2013). Further research is needed to investigate differential DNA methylation patterns and the potential paternal role in intergenerational obesity transmission. Additionally, research into the effects of specific diets, such as the western diet, and their epigenetic effects is needed. (Gapp et al., 2014; Lorenzo et al., 2022)

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### Conclusion and Future Directions

This review highlights the profound impact of epigenetic modifications, particularly DNA methylation, on the development of obesity disparities among minority groups. It is imperative that we move beyond simply documenting these disparities and translate this knowledge into actionable strategies. Future research must focus on validating epigenetic biomarkers, developing targeted interventions, and exploring the long-term effects of environmental exposures. This work necessitates a robust, interdisciplinary approach, bringing together experts from epigenetics, public health, nursing, social sciences, and community advocacy. Only through collaborative efforts can we develop and implement culturally sensitive interventions that effectively address the complex interplay of factors contributing to obesity disparities. The health of future generations depends on our commitment to close the gap between scientific discovery and real-world impact.

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### Application to Nursing Research and Practice

Nursing research can significantly leverage epigenetic studies to understand and address health disparities. By investigating how environmental and lifestyle factors influence DNA methylation patterns in diverse populations, nurses can identify biomarkers for early obesity risk and develop targeted interventions (Kronfol et al., 2017; Min et al., 2021; Notterman & Mitchell, 2015; Voisin et al., 2015). For example, studies exploring the impact of stress, diet, and physical activity on epigenetic markers in minority communities have the potential to inform culturally tailored nursing interventions that can promote healthy behaviors (Akinyemiju et al., 2018; Grazioli et al., 2017). Furthermore, longitudinal research to examine intergenerational epigenetic effects can guide family-centered nursing practices aimed at preventing childhood obesity and fostering long-term health (Rönn et al., 2013). These applications are summarized in Table 2.

Table 2. Applications of Epigenetics in Nursing Research and Practice

Area	Application/Action	Benefit/Outcome	Example
Nursing Research	Identify DNA methylation biomarkers linked to obesity risk in diverse populations.	Early risk detection and targeted interventions	Studies exploring stress, diet, and physical activity effects on epigenetic markers in minority groups.
Nursing Research	Investigate intergenerational epigenetic effects on obesity.	Family-centered prevention strategies.	Longitudinal studies on parental lifestyle impact on offspring obesity.

Nursing Education	Incorporate epigenetics and health disparities into nursing curriculum.	Prepared future nurses to address these issues effectively.	Developing coursework and training programs on the impact of epigenetics on health.
Interdisciplinary Collaboration	Collaboration with genetics, public health, social sciences, nurse practitioner, pharmacologist, physician	Comprehensive understanding of obesity disparities and multifaceted interventions, that will lead to a reduction in those disparities.	Working together to develop a comprehensive understanding of obesity disparities.
Practice/clinical	Personalize risk assessments using epigenetic biomarkers.	Tailored interventions for vulnerable populations.	Creating a personalized diet and exercise plans based on genetic predisposition and environmental context.
Practice/clinical	Implement evidence-based lifestyle interventions (nutrition, exercise) informed by epigenetic research.	Empowered patient choices and mitigated obesity risks.	Nutritional counseling and exercise programs informed by DNA methylation studies.
Practice/clinical	Educate patients and communities about lifestyle factors influencing gene expression.	Increase awareness and healthier choices.	Workshops on nutrition and physical activity emphasizing epigenetics.
Practice/clinical	Advocate for policies addressing social determinants of health.	Equitable access to resources and reduced health disparities.	Supporting initiatives for healthy food access and safe environments for physical activity.

In nursing practice, epigenetic knowledge allows for personalized patient care. By utilizing biomarkers, nurses can tailor risk assessments and interventions, especially for vulnerable populations ([Kanwal et al., 2015](#); [Kronfol et al., 2017](#); [Panuganti et al., 2023](#); [van Dijk et al., 2015](#)). Evidence-based lifestyle interventions, such as nutritional counseling and exercise programs, informed by epigenetic research can empower patients to make healthier choices ([Heinonen et al., 2013](#); [Ielapi et al., 2020](#); [Panuganti et al., 2023](#)). Furthermore, nurses can advocate for policies to address social determinants of health, recognizing their impact on epigenetic regulation and health disparities ([Marmot, 2018](#)) and thereby advancing health equity. These applications are also summarized in [Table 2](#).

**In nursing practice, epigenetic knowledge allows for personalized patient care.**

Finally, nurses play an important role in understanding and addressing health disparities. By studying racial disparities in obesity epigenetics, nurses can gain insights into how genetic and environmental factors contribute to obesity among different racial groups. This knowledge can help nurses develop targeted interventions to address these disparities and improve the health outcomes related to the prevalence of obesity populations described above ([CDC, 2021](#)). Understanding epigenetic factors that contribute to these disparities can help nurses develop more effective, culturally sensitive interventions to address obesity and the associated health risks.

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