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From Social Environment to Molecular Biology: A Review of How Social Changes Drive Biological Adaptations

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ABSTRACT

A growing body of interdisciplinary research demonstrates that social environments exert significant influence on biological systems without altering the underlying genetic code. This review synthesizes empirical findings from studies published between 2000 and 2024 to examine how social determinants—including socioeconomic status, early-life adversity, and social relationships shape molecular and physiological processes. Central mechanisms include epigenetic modifications, particularly DNA methylation, as well as neuroendocrine and immune regulation. Evidence consistently indicates that adverse social conditions are associated with dysregulated stress responses, altered gene expression, and increased disease susceptibility, whereas supportive environments exert protective, buffering effects. However, challenges in establishing causal relationships persist due to methodological variability and inconsistencies in measuring social exposures. This review highlights key empirical trends, theoretical debates, and research gaps, emphasizing the need for integrative and longitudinal approaches to better understand how social inequalities become biologically embedded and to inform future public health interventions.

Keywords: Social Environment, Biological Embedding, Epigenetics, DNA Methylation, Gene–Environment Interaction, Social Determinants of Health, Socioeconomic Status, Social Stress, Early-Life Adversity, Social Support, Health Inequalities, Neuroendocrine Regulation

INTRODUCTION

The relationship between social environments and human biology has become a central focus of contemporary research across disciplines, including sociology, genetics, and public health. Traditionally regarded as distinct domains, social conditions and biological processes are now recognized as deeply interconnected, with accumulating evidence demonstrating that factors such as socioeconomic disadvantage, psychosocial stress, and interpersonal relationships exert measurable effects on physiological functioning (Hertzman and Boyce, 2010; Tung and Gilad, 2013). A key conceptual framework underpinning this shift is *biological embedding*, which posits that lived experiences particularly during critical developmental periods become incorporated into biological systems, thereby shaping long-term health trajectories (Hertzman and Boyce, 2010).

Advances in epigenetics have further strengthened this perspective by revealing how environmental exposures regulate gene expression through reversible biochemical modifications that do not alter DNA sequences (Bollati and Baccarelli, 2010). These findings challenge deterministic views of genetics by highlighting the dynamic interplay between genes and environment. Despite significant progress, research remains fragmented across disciplines, limiting a comprehensive understanding of the mechanisms linking social environments to biological outcomes. This review aims to synthesize empirical evidence, critically evaluate current findings, and identify key gaps in the literature.

Objective

The primary objective of this review is to synthesize and critically evaluate existing research on the influence

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of social environments on biological adaptations and health outcomes. Specifically, the review examines the biological mechanisms through which social experiences affect human health, with particular emphasis on epigenetic modifications as mediators of environmental influences on gene expression. It explores the impact of key social determinants, including socioeconomic status, chronic social stress, social relationships, and early-life experiences, on molecular, physiological, and health-related outcomes.

METHODOLOGY

Study Selection Process

The study selection process was conducted in multiple stages. Initially, titles and abstracts of the identified articles were screened to determine their relevance to the review objectives. Studies that appeared relevant were subjected to full-text assessment. Articles were selected based on their focus on the relationship between social factors and biological outcomes. Duplicate records, studies unrelated to the research topic, and articles with insufficient methodological information were removed during the screening process.

Inclusion and Exclusion Criteria

Studies were included if they were peer-reviewed empirical research articles, systematic reviews, or meta-analyses that examined the effects of social determinants on biological, physiological, genetic, or epigenetic outcomes. Human studies were prioritized; however, animal studies were included when they provided mechanistic insights into biological pathways linking social experiences and health. Eligible studies investigated factors such as socioeconomic status, chronic social stress, social relationships, discrimination, and early-life adversity and reported measurable biological outcomes, including epigenetic modifications, neuroendocrine responses, immune function, or gene expression patterns.

Studies were excluded if they were non-peer-reviewed publications, editorials, conference abstracts, opinion papers, theoretical articles lacking empirical evidence, duplicate publications, or studies that did not assess biological outcomes.

Data Extraction and Synthesis

Relevant information from the selected studies was extracted and organized according to study objectives, population characteristics, social determinants examined, biological mechanisms assessed, and key findings. The findings were synthesized using a thematic review approach. Major themes identified included epigenetic mechanisms, social stress and neuroendocrine regulation, socioeconomic inequalities and biological health, early-life social environments, gene–environment interactions, and the protective role of social support. This thematic synthesis facilitated the identification of common patterns, emerging evidence, methodological challenges, and research gaps across the literature.

Limitations

Although a systematic search strategy was employed, certain limitations should be acknowledged. Variability in study designs, measurement of social exposures, sample characteristics, and analytical techniques limited direct comparison across studies. Furthermore, the predominance of observational studies restricted the ability to establish causal relationships between social environments and biological adaptations. Despite these limitations, the review provides a comprehensive overview of current evidence regarding the biological embedding of social experiences and their implications for health outcomes.

This review is based on a systematic examination of peer-reviewed literature published between 2000 and 2024. Studies were identified through database searches using keywords such as “social epigenetics”, “DNA methylation”, “gene - environment interaction” and “social determinants of health”. Inclusion criteria encompassed empirical studies and systematic reviews that examined associations between social factors and biological outcomes in human populations, with animal studies included where necessary to clarify underlying mechanisms. Exclusion criteria included non-peer-reviewed sources, purely theoretical papers lacking empirical data, and studies without measurable biological outcomes.

Although this approach allows for a comprehensive thematic synthesis, several limitations must be acknowledged. Variability in study design, measurement of social variables, and analytical techniques introduces challenges in comparing findings across studies. Furthermore, the predominance of observational designs limits the ability to establish causal relationships.

Thematic Review

Epigenetic Mechanisms as Central Mediators

Epigenetic processes represent a primary

mechanism through which social environments influence biological systems. Modifications such as DNA methylation, histone modification, and non-coding RNA regulation enable environmental factors to alter gene expression without changing the underlying DNA sequence (Bollati and Baccarelli, 2010). Empirical evidence indicates that social experiences—including socioeconomic disadvantage, psychosocial stress, and early-life adversity—are associated with distinct patterns of DNA methylation, which in turn influence physiological functioning (Evans *et al.*, 2021). These findings suggest that environmental exposures are biologically embedded at the molecular level, linking social conditions directly to cellular processes.

However, despite robust associations, establishing causality remains challenging due to potential confounding factors such as lifestyle behaviors, genetic predispositions, and environmental heterogeneity. Consequently, while epigenetic evidence provides strong support for gene–environment interactions, further longitudinal and experimental research is required to clarify causal pathways.

Social Stress and Neuroendocrine Responses

Chronic exposure to social stress activates the hypothalamic–pituitary–adrenal (HPA) axis, resulting in sustained cortisol release and subsequent dysregulation of stress-response systems (Tung and Gilad, 2013). While acute stress responses may be adaptive, prolonged activation of the HPA axis is associated with impaired immune function, increased inflammation, and heightened risk of chronic disease. Empirical studies consistently demonstrate that individuals exposed to persistent social adversity exhibit altered cortisol rhythms and reduced physiological resilience.

Nevertheless, most existing studies rely on observational data, limiting the ability to distinguish causal effects from correlational associations. Additionally, variability in stress measurement further complicates comparisons across studies.

Socioeconomic Status and Biological Inequality

Socioeconomic status (SES) is a critical determinant of health, influencing biological processes through multiple pathways. Lower SES has been consistently associated with increased inflammation, altered gene expression, and elevated risk of chronic disease (Cerutti *et al.,* 2021). These effects are often cumulative, reflecting prolonged exposure to adverse environmental conditions. In contrast, higher SES is generally associated with more favourable biological profiles and improved health outcomes.

However, inconsistencies in the operationalization of SES ranging from income and education to composite indices limit comparability across studies. This lack of standardization represents a significant methodological challenge in the field.

Early-Life Social Environment

Early-life experiences play a crucial role in shaping long-term biological outcomes. Exposure to childhood adversity has been linked to persistent alterations in stress-related gene expression and increased vulnerability to disease in adulthood (Weeland *et al.*, 2015). These findings align with the concept of sensitive developmental periods during which environmental influences exert particularly strong effects on biological systems.

While animal studies provide compelling evidence of causal relationships, human research remains largely correlational. Longitudinal studies are therefore essential to better understand the temporal dynamics of these effects.

Gene–Environment Interaction

The framework of gene–environment interaction (G×E) emphasizes that genetic predispositions and environmental exposures jointly influence biological outcomes. Rather than acting independently, genes and environments interact dynamically, resulting in context-dependent patterns of gene expression (Halldorsdottir and Binder, 2017). This perspective challenges deterministic models of health and underscores the importance of considering both biological and social factors in understanding disease risk.

Protective Role of Social Support

Emerging evidence highlights the protective effects of supportive social environments. Social relationships have been shown to buffer stress responses, regulate cortisol levels, and enhance immune functioning (Hostinar *et al.,* 2014). Despite these findings, research in this area remains relatively underdeveloped compared to studies focusing on adverse social conditions, indicating a potential bias in the literature and an important avenue for future investigation.

Summary of Key Findings

The review identified strong evidence supporting the influence of social environments on biological functioning

Table 1 : Biological Pathways Linking Social Determinants to Health Outcomes

Social Determinant	Biological Mechanism	Health Outcome	Limitation
Socioeconomic status	DNA methylation, inflammation	Increased chronic disease risk	Inconsistent measurement of SES
Chronic social stress	HPA axis dysregulation	Impaired immunity, elevated cortisol	Predominantly correlational evidence
Early-life adversity	Epigenetic modification	Long-term health vulnerability	Limited longitudinal data
Social support	Hormonal and immune regulation	Reduced stress, improved resilience	Understudied compared to adversity
Gene–environment interaction	Context-dependent gene expression	Individual variability in health outcomes	Complexity in isolating causal pathways

through multiple interconnected pathways.

DISCUSSION

The reviewed evidence strongly supports the concept of biological embedding, demonstrating that social experiences influence biological systems through epigenetic, neuroendocrine, and immunological pathways. Epigenetic mechanisms appear to play a central role in translating social experiences into long-term biological outcomes.

Despite these advances, several challenges remain. The predominance of observational studies limits causal inference, while variability in social and biological measurements reduces comparability across studies. Additionally, debates concerning transgenerational epigenetic inheritance remain unresolved.

Another concern is the risk of biological reductionism; whereby complex social phenomena are oversimplified into biological mechanisms. Future research must balance molecular explanations with broader social, economic, and structural determinants of health.

Greater use of longitudinal designs, standardized measurements, and diverse population samples is essential for advancing the field

Conclusion

Human biology is dynamically shaped by social environments through complex and interconnected mechanisms involving epigenetic regulation, stress physiology, immune functioning, and gene –environment interactions. Evidence from the reviewed literature demonstrates that socioeconomic disadvantage, chronic stress, and early-life adversity contribute to biological changes that increase disease susceptibility, whereas supportive social relationships promote resilience and better health outcomes.

The findings highlight how social inequalities become biologically embedded across the lifespan, contributing to persistent health disparities. Although substantial progress has been made, important gaps remain regarding causality, standardization of social measures, and long-term biological effects. Future research should prioritize interdisciplinary, longitudinal, and cross-cultural investigations to deepen understanding of social-biological interactions and inform evidence-based public health interventions aimed at reducing health inequalities and improving population well-being.

REFERENCES

Bollati, V. and Baccarelli, A. (2010). Environmental epigenetics. *Heredity*, **105**(1) : 105–112.

Cerutti, J., Lussier, A.A., Zhu, Y., Liu, J. and Dunn, E.C. (2021). Associations between indicators of socioeconomic position and DNA methylation: a scoping review. *Clin. Epigenetics,* **13**(1):221

Evans, L., Engelman, M., Mikulas, A. and Malecki, K. (2021). How are social determinants of health integrated into epigenetic research? A systematic review. *Soc. Sci. Med.,* **273**:113738

Halldorsdottir, T. and Binder, E. B. (2017). Gene–environment interactions: From molecular mechanisms to behavior. *Annual Review of Psychology,* **68** : 215–241.

Hertzman, C. and Boyce, T. (2010). How experience gets under the skin to create gradients in developmental health. *Annual Review of Public Health,* **31** : 329–347.

Hostinar, C.E., Sullivan, R.M. and Gunnar, M.R. (2014). Psychobiological mechanisms underlying the social buffering of the hypothalamic-pituitary-adrenocortical axis: a review of animal models and human studies across development. *Psychol. Bull.,* **140**(1):256-82

Tung, J. and Gilad, Y. (2013). Social environmental effects on gene regulation. *Cellular & Molecular Life Sciences*, **70**(22) : 4323–4339.

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