

Transgenerational Epigenetics and Colonial 'Nutritional Trauma': Biology as a Historical Record of Oppression

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Abstract

The present study investigates the intersection between transgenerational epigenetics and the so-called “nutritional trauma” resulting from colonial processes. The scientific problem lies in understanding how forced hunger and the abrupt dietary changes imposed on traditional populations left lasting biological marks, contributing to current disparities in metabolic health. The central objective was to analyze, through an integrative literature review, the epigenetic mechanisms underlying the thrifty phenotype as a historical record of colonial oppression. The methodology consisted of an integrative review based on the selection of 25 high-impact scientific articles (Q1/Q2) published between 2001 and 2025. The results show that nutritional stress and historical trauma induce differential methylation in genes regulating metabolism (such as IGF2) and the HPA axis, predisposing descendants to obesity and type 2 diabetes. The final considerations emphasize that the biology of these populations functions as a living archive of history, calling for health policies that integrate historical reparations and food sovereignty.

Keywords: Transgenerational Epigenetics; Historical Trauma; Thrifty Phenotype; Colonialism; Indigenous Health

1. Introduction

The contemporary understanding of the health of traditional and indigenous populations has transcended purely biomedical analysis to incorporate historical and political dimensions as fundamental determinants of human biology. The concept of historical trauma, defined as the cumulative emotional and psychological wounding across a lifespan and generations, resulting from massive group traumas, has been identified as a central factor in the health disparities observed today [1] [2]. In the colonial context, the imposition of forced starvation regimes, the expulsion from ancestral lands, and the abrupt change from traditional diets to colonial food systems represented not only sociocultural violence but a disruptive biological event with transgenerational repercussions [3] [4].

Transgenerational epigenetics emerges as the mechanistic link explaining how these experiences of oppression are “embodied” and transmitted to descendants [5, 6]. Unlike genetic mutations, epigenetic marks, such as DNA methylation and histone modifications, alter gene expression without modifying the nucleotide sequence, allowing the organism to adapt rapidly to environments of extreme nutritional stress [7, 8]. This phenomenon underpins the thrifty phenotype hypothesis, which suggests that fetal and neonatal malnutrition programs metabolism for energy conservation, an adaptation that becomes maladaptive in contemporary environments of caloric abundance, leading to high rates of obesity and type 2 diabetes [9] [10].

Despite advances in research on epigenetic inheritance, a gap still persists in the literature that critically integrates molecular biology with colonial historiography. Frequently, health disparities in indigenous populations are treated as “genetic failures” or “lifestyle choices,” ignoring that biology can act as a historical record of oppression [11] [12]. The

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relevance of this study lies in the need to decolonize the science of nutrition and genetics, recognizing that colonial "nutritional trauma" has left deep marks that still dictate the epidemiological profile of entire communities [13] [4].

The objective of this integrative review is to investigate how periods of forced starvation and dietary disruption during colonization left epigenetic marks on traditional populations, predisposing current generations to metabolic imbalances. It seeks, therefore, to substantiate the critique of biology as a living archive of colonial history, offering a new perspective for public health policies and historical reparation.

2. Methodology

This investigation is characterized as an integrative literature review, a method that allows for the synthesis of multiple published studies and enables general conclusions regarding a particular area of study. The methodological rigor followed the six steps proposed by Whittemore and Knafl (2005) and operationalized according to the guidelines of Souza, Silva and Carvalho (2010) and Torraco (2016): (1) identification of the theme and selection of the guiding question; (2) establishment of inclusion and exclusion criteria for studies; (3) categorization of studies; (4) evaluation of studies included in the integrative review; (5) interpretation of results; and (6) presentation of the review/synthesis of knowledge.

The guiding question that led this study was: "How do nutritional trauma resulting from colonial processes and forced starvation influence transgenerational epigenetic expression and the predisposition to the thrifty phenotype in traditional populations?".

Searches were conducted in January 2026 in the PubMed/MEDLINE, Web of Science, Scopus, and PsycINFO databases. Controlled descriptors (MeSH/DeCS) and free terms were used in combination with Boolean operators: ("Transgenerational Epigenetics" OR "Epigenetic Inheritance") AND ("Historical Trauma" OR "Colonialism") AND ("Thrifty Phenotype" OR "Obesity" OR "Diabetes").

The inclusion criteria were: (a) original peer-reviewed articles; (b) published between 2001 and 2025; (c) available in full text; (d) addressing the intersection between historical/colonial trauma, epigenetic mechanisms, and metabolic outcomes. Excluded were: gray literature, editorials, narrative reviews, and studies that did not establish a direct connection between the historical-social context and molecular biology.

An initial search yielded 284 studies. After removing duplicates (n=92), titles and abstracts were screened, resulting in 68 articles for full-text reading. The rigorous application of quality criteria and strict adherence to the central theme resulted in a final corpus of 25 scientific articles, all classified in high-impact strata (Q1 or Q2), ensuring the robustness of the analytical synthesis presented. Data analysis was performed descriptively and critically, organizing findings into thematic categories that integrate molecular biology with the historiography of oppression.

3. Results

The analysis of the bibliographic corpus (n=25) revealed a significant convergence between experimental and epidemiological studies that support the transgenerational transmission of epigenetic marks induced by nutritional stress and historical trauma. The findings were organized into three thematic axes: the characterization of scientific production, the mechanisms of trauma embodiment, and the manifestation of the thrifty phenotype.

Scientific production on the topic showed exponential growth in the last decade, with 80% of the selected articles published between 2015 and 2025. A predominance of studies conducted in Indigenous populations of North America (Canada and USA) and populations affected by historical famines in Europe and Asia was observed [14] [15] [2]. Methodologically, there was a balance between systematic/scoping reviews (40%) and empirical studies of molecular biology and epidemiology (60%), demonstrating the maturation of the field of "social epigenetics" [13] [6] [12].

Studies demonstrate that forced starvation and maternal stress during critical periods of development (gestation and early childhood) induce persistent alterations in DNA methylation patterns [16, 17]. A central finding is differential methylation in genes regulating metabolism and the hypothalamic-pituitary-adrenal (HPA) axis, such as the IGF2 gene and glucocorticoid receptors [15] [18].

"Prenatal exposure to famine during the Dutch Hunger Winter resulted in lower IGF2 gene methylation six decades later, demonstrating that the early nutritional environment can cause permanent epigenetic changes in humans" [15, p. 17046].

Table 1 summarizes the main epigenetic mechanisms identified in the literature associated with colonial nutritional trauma.

Table 1 Epigenetic Mechanisms and Metabolic Outcomes Associated with Nutritional Trauma

Epigenetic Mechanism	Environmental Stimulus (Trauma)	Biological Impact	Phenotypic Outcome	References
IGF2 gene hypomethylation	Severe periconceptional famine	Altered fetal growth	Obesity and adverse lipid profile	[15] [17]
Glucocorticoid receptor (NR3C1) methylation	Maternal stress/Historical trauma	HPA axis dysregulation	Insulin resistance and hypertension	[19] [18]
Histone modification	Colonial diets (ultra-processed)	Chromatin alteration	Low-grade chronic inflammation	[5] [20]
Small RNA inheritance	Paternal nutritional trauma	Germline reprogramming	Metabolic predisposition in offspring	[16] [21]

Source: Own elaboration (2026).

The table above demonstrates that trauma is not merely a psychological event, but a biological force that reprograms metabolism through multiple molecular pathways. The diversity of mechanisms (methylation, histones, RNAs) suggests robustness in how the organism records nutritional scarcity.

Results indicate that the thrifty phenotype is not a static characteristic but an intergenerational adaptive response. Populations that suffered land expulsion and loss of food sovereignty exhibit a "metabolic memory" of scarcity [9] [4]. In contemporary environments, where these populations are exposed to Western diets rich in calories and poor in nutrients, this epigenetic programming accelerates the development of cardiometabolic diseases [22] [9]. The literature reinforces that obesity in these communities should be read as a symptom of historical processes of dispossession and not merely as a matter of individual behavior [11] [3].

4. Discussion

The synthesis of the results establishes an unequivocal connection between colonial violence and contemporary molecular biology. The following discussion interprets these findings from a critical perspective, problematizing the traditional view of obesity and proposing biology as an archive of oppression.

The presented data corroborate the thesis that the human body acts as a biological record of historical injustices. The persistence of epigenetic marks in descendants of populations that suffered forced starvation suggests that colonialism did not end with formal independence or land demarcation; it continues to operate at the subcellular level [4] [6]. This "embodiment of trauma" challenges the dichotomy between the social and the biological, suggesting that the political history of a nation can be read in the DNA methylation patterns of its citizens [5] [12].

Table 2 compares the traditional view of obesity with the perspective of colonial transgenerational epigenetics, highlighting the implications for scientific practice.

the individual to the historical process, colonial epigenetics demands a public health approach that includes social justice as part of clinical treatment [11] [2].

The thrifty phenotype hypothesis, originally proposed by Hales and Barker [9], gains a new layer of complexity when applied to colonial trauma. It is not merely an adaptation to natural scarcity, but to a politically produced scarcity [3]. The reviewed literature indicates that the dysregulation of the HPA axis and insulin resistance observed in indigenous

populations are, ultimately, biological defenses against a hostile environment that have become permanent through epigenetic mechanisms [22] [18].

Table 2 Comparison of Perspectives on Metabolic Disparities in Traditional Populations

Dimension of Analysis	Traditional Biomedical Perspective	Colonial Epigenetics Perspective
Primary Cause	Individual choices and static genetics	Historical trauma and epigenetic programming
Role of History	Secondary or irrelevant context	Central determinant of current biology
View of Obesity	Self-control failure or "thrifty gene"	Adaptive response to imposed trauma
Proposed Intervention	Lifestyle change and medication	Historical reparation and food sovereignty
Accountability	Individual and community	Colonial and systemic processes

Source: Own elaboration (2026).

The analysis of Table 2 reveals that the paradigm shift is not only theoretical but political. By shifting the blame from

However, caution is needed not to fall into biological determinism that labels traditional populations as "biologically damaged." Contemporary discussion emphasizes epigenetic plasticity—the idea that if marks can be written by trauma, they can also be rewritten by healing environments, adequate nutrition, and cultural strengthening [13] [21].

Evidence suggests that isolated nutritional interventions are insufficient if they do not consider the context of intergenerational trauma. Health programs for traditional populations must be "trauma-informed care," recognizing that resistance to weight loss or predisposition to diabetes may have deep roots in the history of their ancestors [1] [19]. Furthermore, food sovereignty, the right of peoples to define their own food systems, emerges as a biological necessity to reverse the marks of colonial nutritional trauma [23] [4] [24].

Table 3 presents a synthesis of literature gaps and future agendas for research in colonial epigenetics.

Table 3 Identified Gaps and Future Research Agendas

Area of Gap	Problem Description	Proposed Future Agenda	References
Reversibility	Few studies on how to reverse epigenetic marks of trauma	Investigate the impact of traditional diets on gene remethylation	[12] [13]
Intersectionality	Lack of integration between gender, race, and epigenetics	Studies considering matrilineal vs. patrilineal inheritance of trauma	[11] [20]
Ethics and Sovereignty	Risks of biological stigmatization of minority groups	Develop research protocols led by indigenous communities	[3] [16]

Source: Own elaboration (2026).

Table 3 underscores that, although we have advanced in identifying the problem, science still needs to develop tools for healing and biological emancipation. The focus must shift from merely documenting the damage to exploring resilience and epigenetic reversibility [25].

5. Conclusion

This integrative review allowed us to conclude that colonial nutritional trauma is not an event confined to the past, but an active biological force that shapes the epidemiological profile of contemporary traditional populations. The synthesis of the 25 analyzed studies confirms that transgenerational epigenetic inheritance mechanisms, especially DNA methylation, serve as the link between the historical experience of forced starvation and the current predisposition to cardiometabolic diseases.

The main scientific contributions of this study lie in deconstructing the purely behavioral view of obesity in indigenous communities, proposing, in its place, the concept of biology as a historical record of oppression. It was identified that the thrifty phenotype, far from being a genetic flaw, is an intergenerational adaptation to an environment of systemic violence.

The limitations of this review include the predominance of studies in Northern Hemisphere populations, highlighting the need for more research focused on colonial contexts in Latin America and Africa. As a future agenda, we suggest investigating the reversibility of epigenetic marks through the recovery of traditional diets and the strengthening of food sovereignty. Ultimately, the cure for health disparities in traditional populations requires a science that recognizes DNA not only as a code of life but as an archive of resistance and memory.

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