

Epigenetic Inheritance and Its Impact on Health and Development

Elena R. Vasquez*

Department of Epigenetics and Molecular Biology, Biomark Research Institute, Madrid, Spain

DESCRIPTION

Epigenetic inheritance, the transmission of heritable changes in gene function without altering the underlying DNA sequence, has emerged as one of the most intriguing areas of modern biology. This realization has profound implications for our understanding of development, evolution and disease. At the core of epigenetic inheritance are mechanisms such as DNA methylation, histone modifications and non coding RNAs. These mechanisms regulate gene expression by altering chromatin structure or recruiting protein complexes that activate or silence specific genes. Unlike mutations, these modifications do not change the DNA sequence itself but can still influence cellular function in meaningful ways. When these epigenetic marks are transmitted through the germline, they can affect the phenotype of the next generation, leading to heritable traits that do not follow classical Mendelian patterns. One of the most well documented examples of epigenetic inheritance comes from studies on DNA methylation. In mammals, methylation of cytosine residues in DNA can silence gene expression and certain methylation patterns established during gametogenesis can escape the usual epigenetic reprogramming that occurs during early embryonic development. These persistent marks can influence offspring development and metabolism. For instance, animal studies have demonstrated that parental exposure to environmental stressors, such as nutrient deprivation or toxins, can result in altered methylation patterns in offspring, affecting growth, stress responses and susceptibility to metabolic disorders. Such findings highlight that the environment experienced by one generation may leave molecular imprints on the next.

Histone modifications also play a crucial role in epigenetic inheritance. Histones are proteins around which DNA is wrapped and chemical modifications to histones such as acetylation, methylation or phosphorylation can influence chromatin accessibility and transcriptional activity. Certain histone modifications established in germ cells have been shown to persist after fertilization, guiding gene expression patterns in embryos. These marks can contribute to long-term phenotypic outcomes and may even influence behavior and cognition, as

suggested by recent studies in model organisms. Non coding RNAs, including microRNAs and long non coding RNAs, represent another layer of heritable epigenetic regulation. Evidence suggests that small RNAs present in sperm can modulate gene expression in offspring and affect metabolic and stress related traits. Although the mechanisms are still being explored, it is becoming increasingly clear that RNA mediated epigenetic inheritance complements DNA methylation and histone modification pathways in transmitting information across generations. The concept of epigenetic inheritance has far reaching implications for human health and disease. Traditional genetic studies have sometimes failed to fully explain the heritability of complex diseases, such as diabetes, cardiovascular disorders and psychiatric conditions. Epigenetic inheritance offers a potential explanation for these missing heritability gaps, as inherited epigenetic marks could predispose individuals to disease independently of DNA sequence variants. Moreover, understanding epigenetic inheritance may allow for the development of interventions that target reversible epigenetic changes, offering new strategies for disease prevention and therapy.

Despite its promise, epigenetic inheritance remains a complex and sometimes controversial field. In mammals, for instance, epigenetic reprogramming occurs extensively during early development, potentially erasing most epigenetic marks. Yet, accumulating evidence suggests that a subset of marks can escape this reprogramming, allowing heritable effects to persist. Further research is required to clarify which mechanisms are robustly heritable and how they influence physiology and behavior across generations. Ensuring proper nutrition, reducing exposure to toxins and addressing stress in populations may have consequences not just for the current generation but for the generations that follow. While the field is still developing, the evidence to date suggests that epigenetic inheritance is a critical factor in development, evolution and disease susceptibility. Continued research promises to reshape our understanding of heredity and open new avenues for therapeutic interventions, emphasizing that inheritance is not simply a matter of genes, but also of the epigenetic marks that modulate them.

Correspondence to: Elena R. Vasquez, Department of Epigenetics and Molecular Biology, Biomark Research Institute, Madrid, Spain, E-mail: elena.vasquez@gmail.com

Received: 02-Jun-2025, Manuscript No. EROA-25-39553; **Editor assigned:** 04-Jun-2025, PreQC No. EROA-25-39553 (PQ); **Reviewed:** 17-Jun-2025, QC No. EROA-25-39553; **Revised:** 23-Jun-2025, Manuscript No. EROA-25-39553 (R); **Published:** 01-Jul-2025, DOI: 10.35248/EROA.25.7.214

Citation: Vasquez ER (2025). Epigenetic Inheritance and Its Impact on Health and Development. J Epigenetics Res. 7:214.

Copyright: © 2025 Vasquez ER. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

REFERENCES

1. Braakman I, Bulleid N.J. Protein Folding and Modification in the Mammalian Endoplasmic Reticulum. *Annu. Rev. Biochem.* 2011;80:71-99.
2. Shibata Y, Hu J, Kozlov M.M, Rapoport T.A. Mechanisms Shaping the Membranes of Cellular Organelles. *Annu. Rev. Cell. Dev. Biol.* 2009;25:329-354.
3. Obara C.J, Moore A.S, Lippincott-Schwartz J. Structural Diversity within the Endoplasmic Reticulum From the Microscale to the Nanoscale. *Biol.* 2023;15.
4. Westrate L.M, Lee J.E, Prinz W.A, Voeltz G.K. Form Follows Function: The Importance of Endoplasmic Reticulum Shape. *Annu. Rev. Biochem.* 2015;84:791-811.
5. Schuck S, Prinz W.A, Thorn K.S, Voss C, Walter P. Membrane expansion alleviates endoplasmic reticulum stress independently of the unfolded protein response. *J. Cell Biol.* 2009;187:525-536.
6. Karagoz G.E, Acosta-Alvear D, Walter P. The unfolded protein response: Detecting and responding to fluctuations in the protein-folding capacity of the endoplasmic reticulum. *Biol.* 2019.
7. Volmer R, Van Der Ploeg K, Ron D. Membrane lipid saturation activates endoplasmic reticulum unfolded protein response transducers through their transmembrane domains. *Proc. Natl. Acad. Sci.* 2013;110:4628-4633.
8. Walter P, Ron D. The unfolded protein response: From stress pathway to homeostatic regulation. *Science.* 2011;334.
9. Korenykh A, Walter P. Structural Basis of the Unfolded Protein Response. *Annu. Rev. Cell Dev. Biol.* 2012;28:251-277.
10. Tam A.B, Roberts L.S, Chandra V, Rivera I.G, Nomura D.K, Forbes D.J, et al. The UPR Activator ATF6 Responds to Proteotoxic and Lipotoxic Stress by Distinct Mechanisms. *Dev. Cell.* 2018;46:327-343.
11. Bommiasamy H, Back S.H, Fagone P, Lee K, Meshinch S, Vink E, et al. ATF6 α induces XBP1-independent expansion of the endoplasmic reticulum. *J. Cell Sci.* 2009;122:1626-1636.
12. Sriburi R, Jackowski S, Mori K, Brewer J.W. XBP1: A link between the unfolded protein response, lipid biosynthesis, and biogenesis of the endoplasmic reticulum. *J. Cell Biol.* 2004;167:35-41.