Epigenetic Legacy Inherited Shadows PDF - Enaya Wafa Majid.pdf

by Sanaul Haque

Submission date: 13-Oct-2025 09:41PM (UTC+0700)
Submission ID: 2779888411
File name: Epigenetic_Legacy_Inherited_Shadows_PDF_-_Enaya_Wafa_Majid.pdf (224.58K)
Word count: 1908
Character count: 11575

Epigenetic Legacy, Inherited Shadows: The Neuroscience of Trauma and PTSD

Scholar's Name:
Enaya Wafa Majid

Scholar's Affiliation:
Sunnydale School

Date of Submission
30/09/25

Research Aim: The purpose of this study is to examine how DNA methylation and other trauma-induced epigenetic processes control the generational transfer of susceptibility to PTSD. This study aims to clarify the biological mechanisms by which trauma has a unique impact on brain functions and emotional regulation across generations by combining data from neuroscience, psychology, and most notably genetics. The research's ultimate goals are to deepen our understanding of generational trauma, teach and educate preventative techniques, and offer therapeutic intervention.

Research Questions:

1) How do stress-regulation genes and the susceptibility to PTSD across generations get influenced by trauma-induced epigenetic changes, specifically DNA methylation?
2) What structural and functional alterations are brought about by intergenerational trauma in the amygdala, hippocampus, and prefrontal cortex, and how do these relate to epigenetic mechanisms?
3) To what degree can interventions that focus on psychological resilience and epigenetic regulation reverse or lessen intergenerational trauma?
4) In what ways do research on humans and animal models support the idea that trauma is inherited biologically?

Introduction:

Background and context: Although trauma is a universal human experience, its effects frequently go well beyond the individual. Recent findings in neuropsychology and neurobiology indicate that trauma can affect not only mental health but also the systems that control emotion, stress, and memory. Environmental stressors can change gene expression without changing the DNA sequence itself through a process commonly referred to as "epigenetics." DNA methylation is one of the most important processes, and it has frequently been connected to conflicts in brain areas like the prefrontal cortex, hippocampus, and amygdala.
Problem Statement: Although PTSD has been extensively researched as a psychological disorder, there are still important gaps in our understanding of how trauma affects people of all ages. Research on children of war survivors, descendants of Liberation War survivors from 1971, and experimental animal models consistently demonstrates patterns of intergenerational susceptibility to high levels of stress and anxiety. However, little is known about the precise biological processes that underlie these recurrent patterns. In the absence of coherence, trauma is still primarily seen as a personal ailment rather than a genetic cycle with significant social and health ramifications.
Research importance: This study is important because it reinterprets trauma as a genetic and psychological trait. This study will draw attention to epigenetics and demonstrate how unresolved suffering can subtly influence the mental health of subsequent generations. Although the liberation war of 1971 offers unique proof of inherited trauma, the problem is widespread,

impacting communities that have experienced collective violence, displacement, and war. Comprehending these processes can help direct early interventions, enhance mental health services today, and help end intergenerational cycles of suffering.
Research Objectives: This study aims to investigate the role that trauma-related epigenetic modifications, particularly DNA methylation, play in the generational transmission of post-traumatic stress disorder. It looks for ways to end generational trauma cycles and relate biological processes to psychological effects.

***Literature review*:**

Theoretical Foundations and Key Concepts: Epigenetics refers to modifications on DNA or chromatin that regulate gene expression without altering the underlying DNA sequence [6], [8]. Common mechanisms include DNA methylation, histone modifications, and non-coding RNAs [6]. Recent reviews emphasize how these modifications act as interfaces between environmental exposures (stress, trauma) and lasting biological change [1], [9]. For PTSD and trauma, epigenetic alterations can influence the regulation of stress-related systems such as the hypothalamic–pituitary–adrenal (HPA) axis via genes like NR3C1 and FKBP5 [5], [9]. Researchers distinguish intergenerational inheritance (effects passing from parent to child, potentially via gametes or in utero environment) from transgenerational inheritance (effects showing up beyond the first exposed generation) [1], [2]. While animal models often provide stronger evidence for transgenerational epigenetic inheritance, human studies are more constrained by confounds and ethical boundaries [2], [5].

Empirical Evidence: Animal and Human Studies: In rodent models, traumatic conditioning (e.g., olfactory fear conditioning) has been shown to lead to behavioral and epigenetic changes in offspring—even when offspring are never directly exposed to the original stressor [2], [3]. These findings suggest that parental experiences can leave lasting effects that are passed to the next generation.

In humans, epidemiological and clinical studies link parental traumatic exposure to increased risk of PTSD, anxiety, or altered stress reactivity in children [5], [9]. For example, PTSD–epigenetic association studies have found differential methylation at NR3C1, FKBP5, and other loci in trauma-exposed populations [5]. A recent review also explores how epigenetic resilience factors might interact with susceptibility to PTSD, showing that not all trauma-exposed individuals exhibit the same molecular signatures [8]. Other applied research has explored using epigenetic markers to predict treatment response or therapeutic outcome in anxiety, depression, or trauma-related therapies [7], [8]. This suggests a translational potential between molecular findings and clinical practice [7], [8]

Gaps in Prior Research

1. Causality and confounding factors: Since many human studies are retrospective or cross-sectional, it can be challenging to distinguish between shared environmental, social, or lifestyle factors and inherited epigenetic effects [2], [5].
2. Absence of extensive longitudinal cohorts: Few studies use epigenetic profiling to track several generations over time, particularly in situations involving collective trauma or war [1],

[8].
3. Limited studies conducted in non-Western settings: The majority of PTSD-epigenetic research is conducted in Western or wealthy nations; cultural and population relevance is limited because contexts like Bangladesh (such as the Liberation War of 1971) are rarely examined [10].
4. Inadequate integration of neurobiological and epigenetic data: A lot of research only looks at peripheral biomarkers (blood, saliva), not linking methylation changes to the structure or function of important brain regions (amygdala, hippocampus, PFC) [7], [5].

Implications for Research Design and Focus Future studies should focus on longitudinal or multigenerational designs in light of the gaps, adjusting for environmental covariates like lifestyle, diet, and socioeconomic status to support causal inference [2], [5]. Epigenetic assays can be used in conjunction with neurobiological or neuroimaging measures to help connect molecular changes with brain function [5]. In the Bangladeshi context, focusing on descendants of 1971 Liberation War survivors offers a culturally grounded case study that can fill the geographic as well as population gap [10]. The design should also consider resilience factors and potential reversibility of epigenetic marks, rather than only risk [8]. Moreover, mixed methods such as quantitative epigenetics + qualitative trauma histories and interdisciplinary collaborations (neuroscience, psychiatry, genetics) will strengthen the study's depth and relevance [7], [8].

Methodology: As a result, this study will use a comparative case-control design, looking at matched controls and the descendants of Liberation War survivors from 1971. Semi-structured interviews, cortisol profiling, psychological testing, and DNA methylation assays (NR3C1, FKBP5) will all be used to gather data. In order to investigate the mechanisms and reversibility of intergenerational trauma, statistical and thematic analyses will combine biological, neuropsychological, and experiential data.

***Quantitative Phase*:** To find quantifiable patterns of intergenerational trauma, the quantitative phase will collect biological and psychological data. DNA methylation markers (e.g., NR3C1, FKBP5) and cortisol reactivity as markers of HPA axis function will be evaluated using peripheral blood and saliva samples. The Connor-Davidson Resilience Scale and the PTSD Checklist (PCL-5) are two standardized psychological tests that will measure resilience levels and trauma-related symptoms in both exposed and control groups.

***Qualitative Phase*:** The qualitative phase will complement the molecular and psychological data by collecting individual accounts of trauma and coping across generations through semi-structured interviews. These interviews will focus on real-life resilience experiences, cultural background, and family histories.

This stage will enable the research to place biological results in the context of psychosocial realities and identify variables that could mitigate or increase inherited susceptibility to PTSD.

Project Reliability: For sensitive interviews and biological sampling, the project will need

ethical approval. Participants will also need to provide their full informed consent and have access to psychological support. In order to ensure cultural relevance, recruitment will take place in Bangladesh through survivor associations and community outreach. Large-scale neuroimaging may be limited by resources, but accessible biomarker assays and mixed-methods integration make the study possible. A reasonable schedule allots two months for synthesis and write-up, four months for laboratory and qualitative analysis, and six months for recruitment and data collection.

Conclusion: By redefining trauma as a biological imprint as well as a psychological inheritance, this research forces us to address how trauma is passed down through the generations. By combining the breadth of lived human experience with the precision of neuroscience, the project offers a novel viewpoint on how PTSD is embedded in the epigenome and endures into subsequent lives. In addition to academic research, its conclusions could influence novel therapeutic approaches, change public health practices, and lessen generational suffering in Bangladesh and other conflict-affected communities worldwide. This research seeks to transform inherited pain into a foundation for healing and the disruption of trauma cycles by illuminating the biological pathways of vulnerability and resilience, ultimately ensuring a healthier legacy for coming generations.

Bibliography: [1] A. Jawaid, M. Roszkowski, and I. M. Mansuy, "Transgenerational epigenetics of traumatic stress," *Progress in Molecular Biology and Translational Science*, vol. 273, pp. 273–298, 2018, doi: 10.1016/bs.pmbts.2018.03.003.

[2] B. Dias and K. Ressler, "Parental olfactory experience influences behavior and neural structure in subsequent generations," *Nature Neuroscience*, vol. 17, pp. 89–96, 2014, doi: 10.1038/nn.3594.

[3] I. S. Kiselev, O. G. Kulakova, A. N. Boyko, and O. O. Favorova, "DNA Methylation As an Epigenetic Mechanism in the Development of Multiple Sclerosis," *Acta Naturae*, vol. 13, no. 2, pp. 45–57, 2021, doi: 10.32607/actanaturae.11043.

[4] J. D. Bremner, "Traumatic stress: Effects on the brain," *Dialogues in Clinical Neuroscience*, vol. 8, no. 4, pp. 445–461, 2006, doi: 10.31887/DCNS.2006.8.4/jbremner.

[5] N. Youssef, L. Lockwood, S. Su, G. Hao, and B. Rutten, "The effects of trauma, with or without PTSD, on the transgenerational DNA methylation alterations in human offspring," *Brain Sciences*, vol. 8, no. 5, p. 83, 2018, doi: 10.3390/brainsci8050083.

[6] B. Weinhold, "Epigenetics: The science of change," *Environmental Health Perspectives*, vol. 114, no. 3, pp. A160–A167, 2006, doi: 10.1289/ehp.114-a160.

[7] F. Serpeloni *et al.*, "Does prenatal stress shape postnatal resilience? An epigenome-wide study on violence and mental health in humans," *Frontiers in Genetics*, vol. 10, p. 269, 2019, doi: 10.3389/fgene.2019.00269.

[8] E. J. Nestler, "Epigenetic mechanisms of depression," *JAMA Psychiatry*, vol. 71, no. 4, pp. 454–456, 2014, doi: 10.1001/jamapsychiatry.2013.4291.

[9] R. Yehuda and A. Lehrner, "Intergenerational transmission of trauma effects: Putative role of epigenetic mechanisms," *World Psychiatry*, vol. 17, no. 3, pp. 243–257, 2018, doi: 10.1002/wps.20568.

[10] A. Muhammad, "The spirit of 1971: Reflections on liberation, aspirations, and modern challenges," *The Daily Star*, 2024. Accessed: Dec 21, 2024. [Online]. Available: https://www.thedailystar.net/opinion/views/news/the-spirit-1971-reflections-liberation-aspirations-and-modern-challenges-3780941

[11] S. Dandapat and P. Tripathi, "Tracing Memory, Trauma, and Childhood: The Bangladesh Liberation War in Sorayya Khan's Noor," *R Discovery*, 2024. Accessed: Oct 18, 2024. [Online]. Available: https://discovery.researcher.life/article/tracing-memory-trauma-and-childhood-the-bangladesh-liberation-war-in-sorayya-khan-s-noor/f6469e3fb6bb332ab786508f245e7659

Epigenetic Legacy Inherited Shadows PDF - Enaya Wafa Majid.pdf

ORIGINALITY REPORT

14%	14%	9%	0%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	cris.maastrichtuniversity.nl Internet Source	1%
2	kjar.spu.edu.iq Internet Source	1%
3	www.preprints.org Internet Source	1%
4	escholarship.org Internet Source	1%
5	annsim.org Internet Source	1%
6	www.mdpi.com Internet Source	1%
7	www.hindawi.com Internet Source	1%
8	discovery.researcher.life Internet Source	1%
9	jlupub.ub.uni-giessen.de Internet Source	1%
10	tel.archives-ouvertes.fr Internet Source	1%
11	bora.uib.no Internet Source	1%
12	bobbyhajjaj.com Internet Source	1%

13	pub.uni-bielefeld.de Internet Source	1 %
14	terra-docs.s3.us-east-2.amazonaws.com Internet Source	<1 %
15	Bart P.F. Rutten. "Neuroepigenetics of Mental Illness: The Inside Outs of the Outside Within", Elsevier BV, 2018 Publication	<1 %
16	link.springer.com Internet Source	<1 %
17	mountainhyperbarics.com Internet Source	<1 %
18	www.frontiersin.org Internet Source	<1 %

Exclude quotes	On	Exclude matches	Off
Exclude bibliography	Off		