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ARTICLE

From Colonial Trauma to Biological Memory: A Critical Review of Intergenerational Epigenetic Mechanisms

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Abstract

Colonialism has historically been examined through economic, political, and cultural frameworks; however, emerging evidence from epigenetics suggests that trauma may leave enduring biological imprints across generations. This review integrates interdisciplinary literature from molecular biology, neuroscience, and postcolonial theory to examine how prolonged exposure to famine, systemic violence, and racialized oppression under British colonial rule in the Indian subcontinent may have contributed to epigenetic modifications with intergenerational consequences. A systematic review guided by PRISMA methodology was conducted using major scientific databases. Evidence from trauma-exposed populations and famine cohorts suggests that environmental stress can induce heritable changes in DNA methylation, histone modification, and gene regulation, particularly in pathways associated with the hypothalamic-pituitary-adrenal (HPA) axis. Although direct empirical evidence from colonial India remains limited, comparative findings support a conceptual framework in which imperialism may have biologically embedded long-term health disparities. The review emphasizes methodological limitations and ethical considerations, advocating for cautious interpretation within an interdisciplinary framework.

Keywords: intergenerational epigenetic effects, psychological trauma, stress-regulation pathways, neuroplasticity.

1. INTRODUCTION

British colonial rule in the Indian subcontinent from the eighteenth to mid-twentieth century constituted a prolonged period of structural violence marked by economic extraction, racial hierarchies, and repeated famines that resulted in mass mortality (Sen, 1981) [1]; (Davis, 2001) [2]. By the end of this period, the colonized population was psychologically left uncertain of its identity and collective survival (Rao, 2021) [3]. These events were not merely economic disruptions but deeply embodied experiences of suffering that reshaped both social identity and psychological stability (Dirks, 2001) [4]; (Kleinman et al., 1997) [5]. The imposition of racial ideologies that framed colonized populations as inferior further intensified this trauma, legitimizing exploitation and systemic violence (Kolsky, 2010) [6].

Frantz Fanon's assertion that "Imperialism leaves behind germs of rot which we must clinically detect and remove from our land but from our minds as well" (Fanon, 1963) [7] is particularly relevant in this context, as it highlights the enduring psychological and structural residues of colonial domination. The acts that the British conducted through nearly 200 years of British Rule can now be labelled as war crimes under the Geneva Convention (Mukerjee, 2010) [8]. While Fanon articulated this in sociopolitical terms, contemporary biological sciences suggest that such "germs" may also manifest at

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the molecular level.

Advances in epigenetics have provided a framework through which environmental exposures, including trauma, may influence gene expression without altering DNA sequences (Meaney, 2010) [9]. These mechanisms—primarily DNA methylation, histone modification, and non-coding RNA regulation—play a central role in stress response and neurodevelopment. Increasing evidence suggests that such modifications may be transmitted across generations, thereby linking historical trauma to present-day biological outcomes (Bale, 2014) [10]; (Yehuda & Lehrner, 2018) [11]. These epigenetic markers have been suggested to be heritable across at least three generations. According to some studies the heritability may even stretch across 14 generations (Klosin et al., 2017) [12].

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It is important to acknowledge that much of the empirical evidence linking trauma to epigenetic modifications derives from non-Indian populations, including studies on Holocaust survivors and the Dutch Hunger Winter. While these studies provide valuable mechanistic insights, direct epigenetic investigations within populations affected by British colonial rule in the Indian subcontinent remain limited. Therefore, the present review adopts a hypothesis-driven approach, drawing on cross-contextual evidence to explore potential biological consequences of colonial trauma. Further, this review synthesizes evidence from epigenetics, neuroscience, and historical scholarship to explore whether the trauma experienced under British colonial rule in the Indian subcontinent may have contributed to intergenerational biological effects.

Distinction Between Intergenerational and Transgenerational Epigenetic Inheritance

In the context of epigenetic research, it is important to distinguish between intergenerational and transgenerational inheritance, as these terms are often used interchangeably despite representing biologically distinct processes. Intergenerational inheritance refers to effects observed in directly exposed generations. For example, in humans, environmental exposures affecting a pregnant individual (F0) may influence both the fetus (F1) and the developing germ cells within that fetus (F2), making these generations directly exposed. In contrast, transgenerational inheritance refers to epigenetic effects that persist beyond directly exposed generations (i.e., F3 and subsequent generations in humans), in the absence of continued environmental exposure.

Current human evidence, including studies on populations exposed to severe trauma and famine, largely supports intergenerational effects. While transgenerational epigenetic inheritance has been demonstrated in model organisms, its existence and extent in humans remain under investigation. Therefore, in the context of colonial trauma, the available evidence is more appropriately interpreted within an intergenerational framework, with transgenerational effects remaining hypothetical.

2. MATERIALS AND METHODS

This review was conducted using a systematic approach guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework to ensure transparency and reproducibility. A comprehensive literature search was performed across PubMed, Scopus, and Google Scholar databases to identify relevant studies examining epigenetics, trauma, intergenerational inheritance, famine, and colonial health impacts (Figure 1). Search terms included combinations such

as “epigenetics and trauma,” “intergenerational epigenetic inheritance,” “DNA methylation and stress,” “famine epigenetics,” and “colonialism and health,” with Boolean operators applied to refine results.

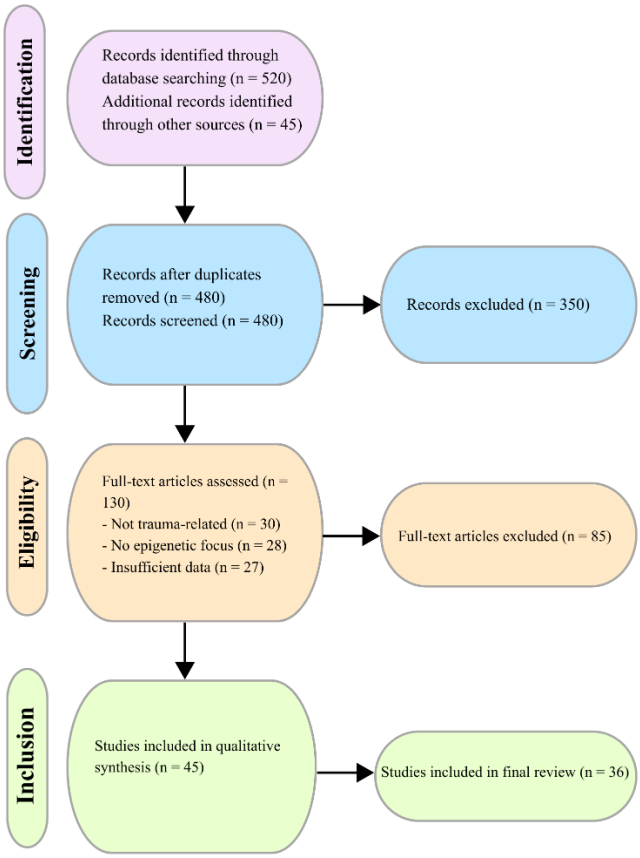

Figure 1. PRISMA Framework Chart

The initial search identified 520 records, with an additional 45 records obtained through other sources. After removal of duplicates, 480 records remained and were screened based on titles and abstracts. During the screening phase, 350 records were excluded due to lack of relevance.

Subsequently, 130 full-text articles were assessed for eligibility. Of these, 85 articles were excluded for specific reasons, including not being trauma-related (n = 30), lacking epigenetic focus (n = 28), or providing insufficient data (n = 27).

A total of 45 studies were included in the qualitative synthesis. From these, 36 studies met the criteria for inclusion in the final review. Included studies were categorized into thematic domains such as epigenetic mechanisms, neurobiology of trauma, intergenerational inheritance, famine studies, and colonial trauma frameworks. Data synthesis was qualitative, focusing on identifying patterns and constructing a conceptual model linking colonial trauma to biological outcomes.

3. RESULTS AND DISCUSSION

3.1 Epigenetic Mechanisms of Trauma

Epigenetic processes provide a mechanistic basis for understanding how environmental stress may become biologically embedded. DNA methylation, mediated by enzymes such as DNMT1, DNMT3a,

and DNMT3b, plays a critical role in regulating gene expression and neural development (Liester & Sullivan, 2019) [13]. These processes are particularly significant in the brain, where they influence neuronal differentiation and plasticity (Hsieh et al., 2017) [14] (Figure 2).

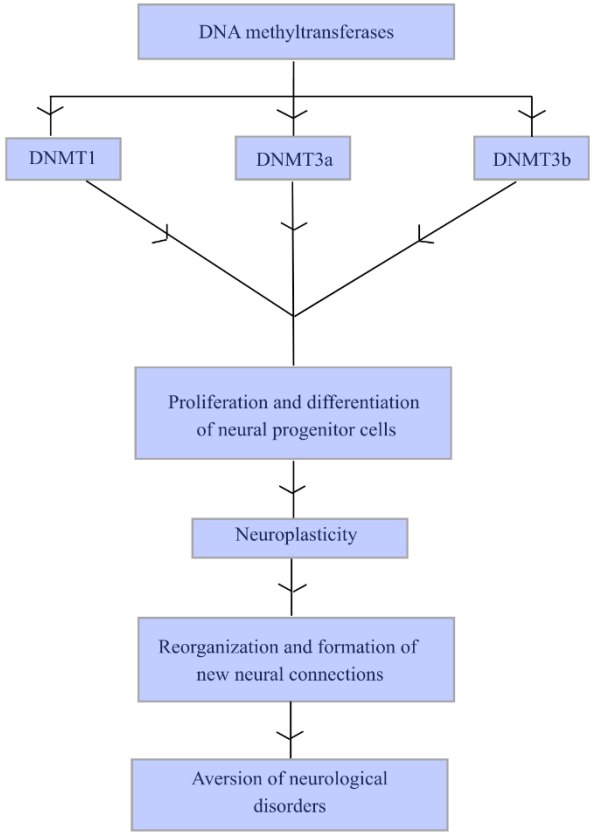

Figure 2. A version of Neurological Disorders Through the Neuroplastic Mechanism

Empirical studies have suggested that early-life adversity can lead to persistent changes in DNA methylation patterns, particularly in genes regulating the stress response. For example, altered methylation of glucocorticoid receptor genes has been associated with exposure to childhood trauma (McGowan et al., 2009) [15]. Similarly, FKBP5 gene regulation has been implicated in stress-related epigenetic modifications (Klengel & Binder, 2015) [16]. Histone modifications and non-coding RNAs further contribute to gene regulation and neural adaptation to stress (Nestler, 2014; Bale, 2014) [17,10]. But not all consequences of these modifications are negative—some studies suggest that many epigenetic adaptations also lead to resilience within organisms (Lehrner & Yehuda, 2018) [11].

These mechanisms collectively support the concept of biological embedding; wherein environmental experiences are encoded within gene regulatory systems.

3.2 Neurobiological Impact of Trauma and Racism

Heterogeneous colonial stress exposures may also result in dysregulation of the HPA axis, leading to prolonged cortisol secretion and structural changes in key brain regions such as the amygdala, hippocampus, and prefrontal cortex (Daskalakis et al., 2018) [18]. These changes are associated with impaired emotional regulation and increased vulnerability to psychiatric disorders (Figure 3).

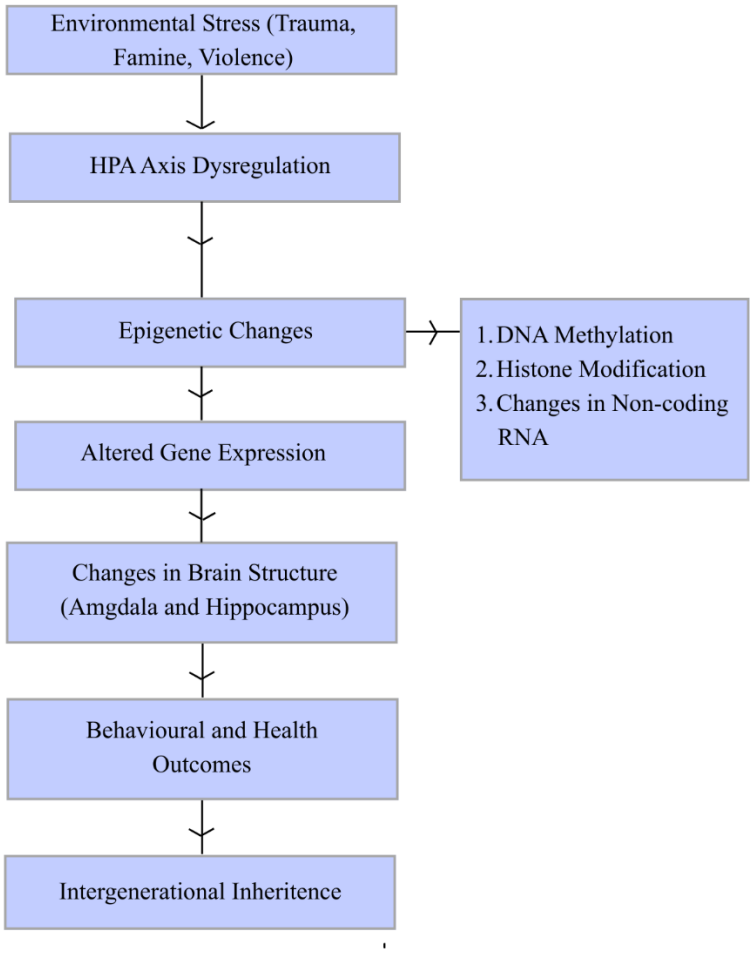

Figure 3. Epigenetic Mechanism of Trauma

Recent research has demonstrated that exposure to racism can alter neural connectivity patterns. Maternal experiences of discrimination have been linked to changes in neonatal brain connectivity, particularly in the amygdala and hippocampus (Kral et al., 2024) [19]. Such alterations may induce hypervigilance and heightened stress sensitivity (van Marle et al., 2009) [20]. Heterogeneous colonial stress exposures have also been shown to reduce dendritic branching, thereby impairing neural connectivity and cognitive function (Cook & Wellman, 2004) [21].

Additionally, studies suggest that racial discrimination accelerates epigenetic aging, increasing susceptibility to disease (Elbasheir et al., 2024) [22]. These findings highlight the profound neurobiological consequences of sustained psychosocial stress.

3.3 Intergenerational and Transgenerational Transmission

The intergenerational transmission of trauma represents one of the most significant developments in epigenetics. Evidence from Holocaust survivor studies suggests that trauma-related epigenetic changes can be observed in both survivors and their offspring (Yehuda et al., 2016) [23]. Animal models further support this phenomenon, demonstrating that stress exposure may lead to heritable changes in behavior and gene expression (Dias & Ressler, 2014; Franklin et al., 2010) [24,25]. However, such findings from model organisms may not be directly translatable to humans.

Some studies suggest that these effects may persist across multiple generations, indicating

transgenerational inheritance (Klosin et al., 2017) [12]. However, this field remains contested due to the difficulty of disentangling epigenetic inheritance from cultural and environmental influences (Horsthemke, 2018; Daxinger & Whitelaw, 2012) [26,27]. Despite these challenges, the evidence strongly supports the plausibility of trauma-induced biological inheritance.

3.4 Famine, Developmental Programming, and Metabolic Outcomes

The Great Famine of 1876-1878 affected people across the subcontinent under the crown rule. This famine was especially devastating because it could have easily been prevented if it weren't for the exportation of millions of tons of grain from India to Britain during a time in which 30-35 million people were dying of starvation (Tharoor, 2017) [28]. For comparison, 50-55 million people died worldwide in WWII. While this famine claimed millions of lives, Lord Lytton was busy preparing for an assemblage in Delhi which would declare Queen Victoria as the Empress of India. The ceremonial included a week-long feast where 68,000 officials were fed grand meals every day (Davis, 2001) [2].

Famine studies provide compelling evidence for long-term biological effects of environmental stress. Research on the Dutch Hunger Winter suggests that prenatal exposure to famine results in persistent DNA methylation changes and increased risk of metabolic and cardiovascular diseases (Heijmans et al., 2008; Tobi et al., 2014) [29,30]. These findings are consistent with the concept of developmental programming, which posits that early-life exposures have lasting health consequences (Gluckman & Hanson, 2004) [31].

Similar patterns have been observed in descendants of famine survivors, who exhibit increased risks of kidney dysfunction and metabolic disorders (Tolkunova et al., 2023) [32]. Studies also indicate that famine exposure increases susceptibility to mental disorders such as depression and schizophrenia (Eichenauer & Ehlert, 2023) [33].

We can also see cultural projections of our history in terms of a subconscious food insecurity within previously oppressed populations wherein habits such as the 'overfeeding' of children within the family are highly prevalent.

Given the scale and severity of colonial famines in India, these findings provide a strong comparative framework for understanding potential long-term biological impacts.

3.5 Colonial Trauma as a Biological Exposure

The conditions experienced under British colonial rule constitute a form of prolonged environmental stress. The conditions suffered by the Indian population align closely with known triggers for epigenetic modification.

Colonialism disrupted social structures and cultural identities, contributing to collective psychological trauma (Rao, 2021) [3]. The persistence of social divisions and communal tensions in postcolonial India may reflect these historical disruptions (Sahgal, 2021) [34]. A lot of these communal tensions can be attributed to the highly prevalent British policy of 'Divide and Rule'—they intentionally integrated sociopolitical structures that would widen the gap between communities and hence breed contempt where there once was unity. Additionally, colonial-induced lifestyle changes likely altered the microbiome composition within the gut, with implications for immune and metabolic health. Dysbiosis within the microbiome composition has proven to result in an increased risk of diseases such as rheumatoid arthritis (Skelly et al., 2018) [35].

These factors collectively support the conceptualization of colonialism as a biologically relevant exposure.

3.6 Biological Embedding of Imperialism

The concept of biological embedding provides a framework for linking historical experiences with present-day health outcomes (Marmot, 2005) [36]. Epigenetics offers a mechanism through which social and environmental conditions may influence gene expression and disease susceptibility (Thayer & Kuzawa, 2011) [37] (Figure 4).

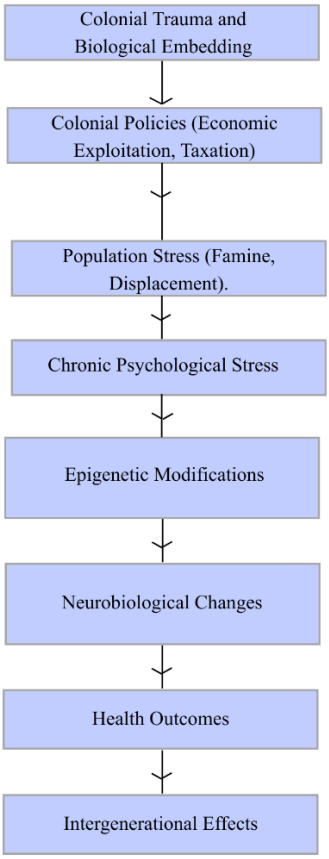

Figure 4. Colonial Trauma and Biological Embedding

However, the application of epigenetics to colonial history must be approached with caution. There is a risk of oversimplification or deterministic interpretations that fail to account for the complexity of social and cultural factors (Keaney et al., 2023) [38]. Other factors such as differential susceptibility also inhibit us from drawing simplifications. Importantly, epigenetic changes can confer resilience, enabling populations to adapt to adverse environments (Lehrner & Yehuda, 2018) [11].

3.7 Heterogeneity of Colonial Stress: Exposure Windows and Stressor Types

Colonial trauma cannot be considered a uniform or singular exposure, as its biological impact likely varied depending on both the timing of exposure and the nature of the stressor. Epigenetic responses are known to be highly sensitive to developmental windows, making it important to distinguish between different periods of exposure.

Exposure timing plays a critical role in shaping biological outcomes. Prenatal exposure, such as maternal famine or stress during pregnancy, may influence fetal development and result in long-term metabolic and neuroendocrine changes. Early childhood represents another sensitive period, during which environmental stressors can affect developmental programming and stress-response systems. In contrast, exposures during adulthood are more likely to contribute to cumulative physiological burden rather than developmental reprogramming.

In addition to timing, the type of stressor is also significant. Colonial experiences encompassed a wide range of stressors, including nutritional deprivation (e.g., famine), acute physical and psychological trauma (e.g., violence), and chronic psychosocial stress (e.g., systemic racial

discrimination). Each of these stressors may engage distinct biological pathways and result in different epigenetic signatures.

Therefore, grouping these diverse exposures under a single category of "chronic stress" may oversimplify the complexity of colonial experiences. A more nuanced framework that distinguishes between exposure windows and stressor types is necessary for accurately interpreting potential epigenetic outcomes.

3.8 Potential Confounding Factors and Alternative Explanations

While epigenetic mechanisms provide a compelling framework for understanding the biological embedding of historical trauma, several confounding factors must be considered. Observed intergenerational health patterns may not solely reflect heritable epigenetic modifications but could also arise from shared environmental and socioeconomic conditions across generations.

One major factor is nutritional transition following colonial and post-independence periods. Variations in early-life nutrition, including famine exposure followed by periods of relative abundance, are known to influence metabolic outcomes and may mimic epigenetic inheritance.

Additionally, persistent socioeconomic inequalities and structural disparities may contribute to health outcomes across generations through non-biological pathways. Limited access to healthcare, education, and resources can create patterns that resemble inherited biological effects.

Cultural continuity and behavioral transmission also play an important role. Practices related to diet, stress coping, family structure, and social norms are often transmitted across generations and may confound attempts to isolate purely biological inheritance mechanisms.

Environmental exposures, including urbanization, pollution, and lifestyle changes, further complicate the interpretation of intergenerational effects.

Taken together, these factors highlight the complexity of disentangling epigenetic inheritance from broader social and environmental influences. Therefore, the proposed links between colonial trauma and epigenetic outcomes should be interpreted with caution and viewed as part of a multifactorial framework.

3.9 Limitations

The primary limitation of this review is the lack of direct empirical studies examining epigenetic effects of colonial trauma in Indian populations. Additionally, methodological challenges make it difficult to distinguish between biological inheritance and ongoing environmental influences. Ethical concerns regarding the interpretation of epigenetic data further complicate the analysis. Further, the interpretation of colonial trauma within an epigenetic framework must be approached with caution, as current evidence is largely extrapolated from non-Indian cohorts. Differences in genetic background, environmental exposures, and sociocultural conditions may influence the extent and nature of epigenetic modifications. As such, the conclusions drawn here should be considered exploratory and indicative of potential mechanisms rather than definitive causal relationships.

4. CONCLUSION

This review has examined the potential intersection of epigenetics, trauma biology, and colonial history, proposing that heterogeneous colonial stress exposures associated with British colonial rule in the Indian subcontinent may have contributed to biologically embedded effects with possible intergenerational consequences. By integrating insights from molecular biology, neuroscience, famine research, and postcolonial theory, the analysis positions colonialism not only as a socio-political system but also as a sustained environmental exposure capable of influencing long-term health outcomes.

A key strength of this work lies in its interdisciplinary approach, which allows for the development of a conceptual framework linking historical trauma to biological processes such as stress-axis

dysregulation and epigenetic modification. The evidence reviewed supports the broader idea that severe and chronic environmental stress can shape gene expression and potentially influence subsequent generations. However, this framework remains largely inferential when applied to colonial India, as direct empirical epigenetic data from historically exposed populations is currently lacking.

This limitation highlights a central challenge: the reliance on analogies from other trauma-affected populations and experimental models. While these comparisons provide valuable insights, they cannot fully capture the unique historical, cultural, and environmental context of colonial India. Moreover, it remains difficult to distinguish between true biological inheritance and the persistence of adverse social conditions across generations. This raises important questions about causality and underscores the need for caution in interpreting epigenetic evidence within historical frameworks.

Another critical concern is the risk of reductionism. Explaining colonial trauma primarily through biological mechanisms may inadvertently obscure its structural, political, and cultural dimensions. At the same time, epigenetic responses should not be viewed solely as markers of damage; they may also reflect adaptive processes that enabled survival under extreme conditions. Recognizing this dual role is essential for developing a balanced understanding of trauma.

Ultimately, while definitive conclusions cannot yet be drawn, the convergence of interdisciplinary evidence supports the plausibility that colonial-era experiences may have had lasting biological implications. Future research integrating epigenetic data with historical and social analysis will be essential to advance this field. Such work has the potential not only to deepen scientific understanding but also to illuminate the enduring legacies of colonialism in shaping present-day health and inequality.

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