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How epigenetic inheritance fails to explain the Black-White health gap

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ABSTRACT

Slavery, legal segregation, and ongoing discrimination have exacted an unfathomable toll on the black population in the United States, particularly with respect to the impact on health outcomes. In recent years, various researchers and activists have suggested that racial disparities in the modern era can be attributed directly to the trauma of slavery, postulating that these unspeakable traumas led to epigenetic changes in slaves—changes that have since been passed down to subsequent generations. Investigating those claims in this paper, we comprise a review of previous literature that considers the potential for transgenerational epigenetic transmission of trauma in humans. However, we find that there is little evidence to indicate the presence of transgenerational epigenetic transmission of trauma in humans. We find no prior evidence that supports (or is relevant to) the notion that the black-white health gap stems from the inherited trauma of slavery. We conclude that, given the ongoing traumas black Americans are exposed to in modern America, it is much more likely that present-day racial health disparities are due to more direct and current mechanisms than transgenerational transmission of slavery-era trauma.

> Possibly the leading PTSS theorist is the well-meaning and well-respected healing ambassador, Joy DeGruy. Her critically acclaimed 2005 book, Post Traumatic Slave Syndrome: America's Legacy of Enduring Injury and Healing, remains the Bible for PTSS theorists. Black "infighting," materialism, poor parenting, jealousy, colorism, defeatism, frustration, rage—these "dysfunctional," these "negative" behaviors "as well as many others are in large part related to trans-generational adaptations associated with the past traumas of slavery and on-going oppression," DeGruy argued.
>
> DeGruy added PTSS to the library of theories imagining that slavery and on-going oppression had largely fashioned Black behavioral (and genetic) deficiencies. PTSS joined other recent additions to the library, theories with names like the "slavery-hypertension hypothesis" and "hood disease" and the "legacy of defeat."
>
> – Kendi (2016), "Post-Traumatic Slave Syndrome is a Racist Idea"

1. Introduction

Black Americans are nearly three times more likely to be hospitalized and nearly twice as likely to die from COVID-19 than white Americans (Feldman and Bassett, 2021). These figures serve as a reminder of the persistence of black-white health disparities in the United States. Compared with whites, black Americans face a disproportionate disease burden, including a higher incidence of and mortality from hypertension, stroke, and diabetes (Aggarwal et al., 2021).

Black life expectancy for many years has averaged four years less than white life expectancy; during the first year of the pandemic alone, the black-white gap widened almost an additional two years (Bosman et al., 2021). Black infant mortality is twice as high as white infants; perversely, black mothers with the highest levels of education experience the highest infant mortality rates among any racial or ethnic group in the United States (Smith et al., 2018). Black mothers are three times as likely as white mothers to die of pregnancy-related causes (Petersen et al., 2019).

These racial disparities in health outcomes are a function of racial disparities in environmental and experiential differences, including disparities in power, wealth, income, and what philosopher John Rawls aptly termed the social bases of self-respect. These modern gaps are products of both historical and ongoing racial disparities in federal land distribution policies, subsidization of homeownership, access to well-paid employment, access to quality education, and access to adequate sanitation, nutrition, and medical care.

The housing market provides a strong illustration. Structural racism

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and interpersonal discrimination in housing markets via historical redlining, racial covenants, and other sanctioned tactics have created, in many locations, a form of modern-day segregation (Rothstein, 2018; Massey and Denton, 1993). Such a constraint imposes barriers to healthy lifestyles, limits access to quality medical care, decent housing, healthy food, clean water, and chronically strains physiological systems—increasing susceptibility to illness (Smith et al., 2019; Williams and Collins, 2001). Such a relationship between individual and contextual factors, expansively construed, and health is often referred to as the social determinants of health (SDOH). Krieger (1994) emphasizes, in outlining the beginnings of what would be called "Ecosocial Theory," the web of these of various factors in explaining the spread of disease. Subsequently, numerous studies have confirmed the central role of SDOH in explaining health outcomes and, in particular, health disparities (Taylor et al., 2016; Parolin and Lee, 2022; Javed et al., 2022a; Javed et al., 2022b). This direct connection between racism and health disparities is explicated further by Fundamental Cause Theory as detailed by Phelan and Link (2015). As the authors explain, racism is a "fundamental cause" of racial disparities in socioeconomic status (SES); these SES disparities, in turn, cause racial health disparities (Phelan and Link, 2015).

All the manifold and interwoven environments that contribute to health disparities, including the prenatal environment, are *experienced*. Moreover, the acute nature of modern-day, adverse social exposures—accrued through one's neighborhood social environment, racialized stressors, lower socioeconomic status, and exposure to ongoing racism and discrimination—can lead to negative health outcomes through epigenetic mechanisms (Martin et al., 2022). One needs not consult the past to find epigenetic evidence for current health disparities.

Still, an idea has gained traction in recent years that the environmental conditions faced by one's ancestors, not directly experienced by subsequent generations, is critical for understanding the current black-white health gap. One position that has gained increasing currency is the idea of Post-Traumatic Slave Syndrome (DeGruy Leary, 2005) or Post Traumatic Slavery Disorder (Reid et al., 1994).

Both versions posit that the extreme injuries inflicted upon the enslaved in the United States have been transmitted across multiple generations, resulting in poorer mental and physical health for their living descendants. Certainly, if there is trauma passed through generations, it could easily be transmitted via learned cultural practices. Adaptations of behavior like those DeGruy describes do not necessarily constitute genetic change. But this brand of the argument intersects with behavior genetics, since the multi-generational effects of slavery also are manifest in what she views as dysfunctional practices, particularly in parenting, self-discipline, and attitudes toward education. In some popular and academic iterations of this argument, epigenetics are invoked as shaping the potential transmission path of the trauma of slavery to subsequent generations (Karen Hunter Show, 2022; Evans, 2021; Kaufman et al., 2023). A similar argument from Shannon Sullivan (2013) invokes epigenetics as the mechanism through which white racism, dating to the days of slavery, delivers transgenerational health effects.

We shall refer to this as the transgenerational slavery trauma (TST) hypothesis, where "trauma" refers to any adverse health outcome. In short, the horrors of being subjected to slavery ostensibly induced epigenetic changes in enslaved blacks. These changes were inherited via a process of transgenerational epigenetic inheritance (or "trans-epigenetic inheritance," for short). These inherited epigenetic changes are presumed to have an adverse impact on the health of contemporary African Americans, 158 years after the end of slavery.

The idea's power can be seen in the broad audience it has reached: The seminal TST tract, *Post-Traumatic Slave Syndrome: America's Legacy of Enduring Injury and Healing* (2005), has been cited more than 2100 times on Google Scholar (Post-traumatic slave syndrome, 2024). DeGruy's work has found particular purchase in revamping age-old arguments that ascribe the plight of black Americans to individual shortcomings—however, in DeGruy's framing, the root cause for familial and social instability is not the individual but the trauma the individual carries in their genes (Halloran, 2019; St. Vil et al., 2019). DeGruy and other TST proponents continue to be cited widely in spaces related to psychology, therapy, social work, environmental studies, and other areas (Sule et al., 2017; Hicks, 2015; Campbell, 2022; Rodgers, 2021; Dilworth-Bart et al., 2024). Cross (2024) states how, in her work, DeGruy presents the epigenetics grounding of TST as "*settled science*" (p.399, emphasis in original). Moreover, Cross (2024) notes, DeGruy's work has been presented at conferences and spawned subsequent books about the trauma of black Americans (Bowser and Charles-Nicolas, 2021; Skinner, 2008). Notable is not just the spread of these ideas but also their invocation in discussions of policy and treatment. Of particular importance is the application of TST to clinician guidelines surrounding understanding collective trauma and the influence of TST on public and professional narratives involving mental health and healing (Williams et al., 2018; Mohatt et al., 2014; Hankerson et al., 2022; Treleaven, 2018). Even a recent report from a leading organization advocating for black reparations deploys TST as a core argument in its case (Kaufman et al., 2021).

Advocates of TST, when they marshal evidence for their claims, typically make an argument by analogy by referencing studies that purport to demonstrate the transgenerational epigenetic inheritance of ancestral trauma in humans. We argue in subsequent sections that these studies are seriously flawed and provide little support for either TST or the more general notion of the transgenerational transmission of trauma via a genetic-level mechanism. We consider, critically, the hypothesis advanced by DeGruy Leary (2005)—the intergenerational, epigenetic transmission of the trauma of slavery—as falling within the confines the general yet similarly flawed concept of the intergenerational, epigenetic transmission of trauma. However, we limit our scope of discussion in this article to TST given the argument's high-profile nature, as well as its insertion into the growing discourse surrounding the topic of black reparations.

Notably, we believe the TST argument is actively harmful in pursuing remedies to these modern-day disparities for at least five reasons. First, any argument's reliance on dubious mechanisms like transgenerational epigenetic inheritance in humans makes it unnecessarily vulnerable to counterarguments. Second, TST frames health inequities arising from structural factors as predestined curses manifesting themselves on the individual level—or rather, the level of one's genetic code. This framing, which maintains that the cause of present-day disparities is the product of transmitted trauma, risks perpetuating a discourse in which the victimized (traumatized) families are indirectly responsible for passing on this trauma (Maxwell, 2014). Of course, there has been a long history of pathologizing black Americans and suggestions that black family structures explain present-day disparities across social spheres, perhaps most notably in Daniel Patrick Moynihan's 1965 report "The Negro Family: The Case for National Action." Such "cultural" explanations fall apart upon close inspection (Smith et al., 2018; Darity et al., 2018).

Third, the TST argument is harmful for its seductive nature. While opening the door for this damaging discourse, TST does (appropriately) absolve the individual from ultimate blame for current-day disparities. Moreover, it is poetic, and it retains appeal as a potential coping mechanism for African Americans facing constant discrimination and racism. Fourth, as we have mentioned, TST is gaining purchase in more reputable spaces and is beginning to influence discussions around policy. Fifth, by identifying the root of the black-white health gap to be inescapable traumas of the past rather than quantifiable conditions that exist in the present, TST risks treating these disparities as far more intractable than they are.

Instead, we argue that there is no justification for the assumption (or proposal or hypothesis) that the black-white health gap is influenced by transgenerational epigenetic inheritance and, particularly, that there is no justification needed beyond the key role of structural racism experienced directly by African Americans today. There have been no

systematic studies of TST, nor, for reasons we shall explain, could there have been experimental investigations. One notable complication is the reality, as Joseph L. Graves Jr. (2011, 2015) has pointed out, multiple generations of blacks in America continuously have been exposed directly to (different) factors that affect epigenetic change.

Section two provides an overview of the process of transepigenetic inheritance. Section three considers the intersection of the TST argument and evidence of transepigenetic inheritance—particularly with regards to the possibility of trauma causing transgenerational epigenetic change in humans. By combing the references of key TST texts, here we compile and review the major studies that purport to detail the biological mechanisms of TST. Last, in the concluding section, we expound upon the impossibility—and irresponsibility—of the TST argument and consider more appropriate alternatives.

2. Review: epigenetics and inheritance

Epigenetics is defined as the molecular factors and processes that regulate genome activity (i.e., gene expression) independent of DNA sequence (Gibney and Nolan, 2010). Epigenetic modifications include DNA methylation (the addition of a methyl compound to specific DNA sequences), histone modifications (chemical modifications to the histone proteins around which the DNA molecule is wrapped), and non-coding RNAs. Epigenetic modifications are the reason why different cells only give rise to cells of the same type when they divide mitotically, despite having the same genome. Inasmuch as there are epigenetic differences between different cells and tissues of the same individual, the term "epigenome" (without qualification) will be used to refer to the sum total of epigenetic "configurations" in all the cells of a given individual at a given time.

Epigenetic changes can be induced by environmental factors such as nutrition, toxicant exposure, and stress and are an important mechanism by which organisms change their gene expression in response to their environment (Mazzio and Soliman, 2012). They likely play a role in translating a variety of adverse environments into adverse health outcomes. To what extent epigenetic modifications are involved in, or can serve as markers for, the black-white health gap is an ongoing area of investigation (Vick and Burris, 2017; Pepin et al., 2021; Liu et al., 2019).

2.1. Epigenetic inheritance

All the epigenetic changes that are part of the developmental process and all the environmentally induced epigenetic "marks" (i.e., changes in DNA methylation and histone modifications) are erased and "reprogrammed" during gametogenesis in primordial germ cells and post-fertilization in the zygote (Morgan et al., 2005). This erasure and reprogramming are necessary for sexual reproduction and to generate totipotency (i.e., the ability of a cell to form all the cell types in the human body). The concept of epigenetic inheritance rests on the assumption that, somehow, at least some environmentally induced epigenetic marks escape this reprogramming. It is still unclear whether in humans (and mammals more generally) they do escape and how this is accomplished (Perez and Lehner, 2019). Hence, whether epigenetic inheritance is possible in humans still is unresolved.

We draw a distinction between intergenerational and transgenerational epigenetic inheritance (or transepigenetic inheritance) (Perez and Lehner, 2019; Skinner, 2008).

When discussing inheritance, F0 refers to the parental generation or any member of that generation, F1 the children, F2 the grandchildren, and so forth.

In intergenerational epigenetic inheritance, a male or non-pregnant female (F0) is exposed to an environmental input that alters F0's epigenome and most importantly, the epigenome of F0's germ cells (egg and sperm) which will generate F0's children (the F1 generation). F1 manifests a certain phenotype due to the epigenetic alteration of F0's germ cells. However, the epigenetic change/phenotype occurs only in F1 and not F2 (F0's grandchildren) or in any other generations. An example of this would be paternal alcohol consumption prior to conception, leading to epigenetic differences in his children that increase their risk of birth defects (Zhou et al., 2021). These epigenetic alterations/phenotypes are not transmitted beyond F1.

In transgenerational epigenetic inheritance, epigenetic alterations acquired by a male or non-pregnant female (F0) would need to be observed, *in the absence of the original environmental input*, through at least the F2 generation (F0's grandchildren). This is because F2 would be the first generation *with no direct exposure to the environmental input.*

For pregnant females exposed to an epigenome-altering environmental input, the mother (F0), fetus (F1), and the fetus's germ cells, which will generate the F2 generation, all are exposed directly. In this case, intergenerational effects encompass inheritance from F0 to F2. In the absence of the inducing environmental input, only persistence to the F3 generation (F0's great-grandchildren) would be considered a transgenerational effect.

Given slavery's termination in the United States in 1865 and the average human lifespan, attributing present-day health disparities to epigenetic responses stemming from slavery-era traumas requires transmission across multiple generations. While there are many exceptions (Darity Jr and Mullen, 2022, p.241-243), assuming a generational turnover of 30 years typically the youngest generation would be the fifth generation born since the end of American slavery (p.242). As such, when we evaluate in the subsequent section the epigenetic mechanisms that potentially lend credence to TST, we are focused on evidence of transgenerational epigenetic inheritance, not merely epigenetic inheritance from one generation to the generation immediately following, nor even the next.

3. Review: the evidence for transgenerational slavery trauma

As noted, there have been no studies providing evidence for TST. This absence is due not simply to the paucity of relevant data for African Americans dating back five generations and more (enslaved African descendants were not recorded in the pre-Civil War Censuses) but to the obvious lack of any valid control group. While being an enslaved black had to be worse than being a free black, free blacks' lives were not devoid of racialized trauma, so they would not be an appropriate control group (Northup, 2014; Maris-Wolf, 2015; Wilson, 1994). Moreover, to the extent their descendants intermarried with blacks whose ancestors had been enslaved there literally is no potential control group at present.

Instead, the case for TST is typically made by referring to the results of other studies. It is worth reiterating that the bulk of evidence of intergenerational epigenetic transmission is found in animal studies and has not been demonstrated in human studies (Perez and Lehner, 2019; Yehuda and Lehrner, 2018). As a result, studies claiming evidence for epigenetic inheritance in humans are generally of two kinds. First, researchers might report evidence of inter- or transgenerational epigenetic inheritance and, from this, infer the involvement of unknown epigenetic mechanisms. Evidence is generally based on a process of elimination involving cases and controls. For example, researchers may report that the grandchildren of grandparents exposed to famine are more likely to exhibit a certain phenotype compared to controls whose grandparents were not exposed to famine. Based on this data, they might conclude that the only possible explanation for this difference is transepigenetic inheritance. Second, researchers might report evidence of inter- or transepigenetic inheritance by a similar process but also report epigenetic differences between cases and controls.

3.1. Studies that have invoked epigenetic mechanisms to explain the effect of Holocaust induced parental trauma on offspring

It is often assumed that parental trauma is associated with higher rates of posttraumatic stress disorder (PTSD) in children. However, while Holocaust survivors, in general, have higher rates of PTSD and

other psychological problems, it is not clear whether this is true of their children. Studies have yielded conflicting results and there is no consensus (van IJzendoorn, Bakermans-Kranenburg and Sagi-Schwartz, 2003; Sagi-Schwartz, van IJzendoorn, & Bakermans-Kranenburg, 2008).

That said, in 2016 Yehuda and colleagues conducted a study of 32 Holocaust survivors and 22 of their adult children conceived after parental "Holocaust exposure," defined as being interned in a Nazi concentration camp, having witnessed or experienced torture, or having had to flee or hide during World War II (Yehuda et al., 2016). Based on blood samples, they reported differences in methylation levels of a region of the gene FKBP5 which plays a role in the regulation of the neuroendocrine stress response. Mothers whose exposure took place at age ten or earlier showed 10% higher methylation levels than controls. As the authors noted, higher FKBP5 methylation has been associated with increased PTSD risk (Binder et al., 2008). By contrast, Holocaust children showed 7.7% *lower* methylation levels at the same site, which has been associated with *decreased* risk of PTSD.

In a 2020 study, researchers reported the partial replication of these findings. While they failed to replicate the finding of higher FKBP5 methylation among mothers exposed at age ten or earlier, they reported the replication of the finding of lower FKBP5 methylation levels among adult children of Holocaust mothers (n = 147) (Bierer et al., 2020). Of this surprising finding, the authors commented:

> In the present study … the effect of maternal Holocaust exposure on [FKBP5] methylation … was not associated with trauma-related psychopathology. Rather, lower … methylation was associated with diminished self-reported anxiety symptoms, suggesting the possibility of a protective effect. Offspring of mothers exposed in childhood showed the greatest reductions in methylation but also the least psychopathology (Bierer et al., 2020, p.747).

Regarding the supposed relationship between FKBP5 methylation levels and PTSD: The authors assume that higher FKBP5 methylation is associated with higher PTSD risk and lower methylation with lower risk. However, there have been conflicting results concerning the direction of this supposed relationship, as well as studies finding no relationship at all (Flasbeck and Brüne, 2021; Lee et al., 2010; Di Sante et al., 2018). The authors speculated that FKBP5 methylation influences levels of cortisol which is the main stress hormone in the body.

However, researchers recently reported that FKBP5 methylation is unrelated to FKBP5 expression in whole blood, and that there is no association between FKBP5 gene methylation in whole blood and acute and long-term cortisol output (Klinger-König et al., 2019; Alexander et al., 2020). This is a reminder of the following problem with most studies that attempt to identify epigenetic differences: Because different cells have different epigenomes, the methylation status of a gene based on a blood sample may tell us nothing about its methylation status in other cells or tissues that are relevant for the phenotype under consideration (Husby, 2020).

This finding does not provide support for TST (beyond the fact that the study concerns inter-, and not transgenerational epigenetic inheritance). A central assumption of TST is that a negative ancestral environment results in negative health outcomes that are transmitted across multiple generations. Here, parental trauma and associated epigenetic alterations are reported to result in *positive outcomes* in children (reduced anxiety and psychopathology).

3.2. Studies concerning the health effects of changes in grandparental food consumption on grandchildren's health

3.2.1. The Överkalix studies

The Överkalix studies involved data on 303 probands born in 1890, 1905, or 1920, in the Överkalix locality in northern Sweden and their 1818 parents and grandparents (Pembrey et al., 2006; Kaati et al., 2002; Kaati et al., 2007; Bygren et al., 2001; Pembrey et al., 2014). Throughout the nineteenth century, Överkalix harvest records were kept every year, along with records of births, deaths, and family trees. Using historical harvest data researchers classified the harvests (primarily of barley and rye) for each year from 1799 to 1889 as poor, moderate, or good (Bygren et al., 2001). They used this as an indicator of food availability for the year. Food availability in turn, was treated as a proxy for food consumption. The health of probands was then compared with the quality of harvest experienced each year by parents and grandparents.

The only associations between grandparental food availability and the health of grandchildren occurred when the grandfathers were ages 9–12 and the grandmothers were ages 8–10. These time frames have been referred to as the "slow growth periods" (SGPs) since they are periods prior to the growth associated with adolescence.[1]

The main findings of the Överkalix studies were:

1. If there was at least one good harvest (and no year of poor harvest, but possibly years of moderate harvest) during the paternal grandfather's SGP, his grandsons had an increased all-cause mortality rate[2] (compared to the mortality risk of males whose grandfathers had different experiences during their SGP).
2. If there was at least one good harvest during the paternal grandfather's SGP, his grandchildren had an increased risk of death from diabetes and cardiovascular disease.
3. If there was at least one good harvest during the paternal grandmother's SGP, her granddaughters had a higher mortality rate.
4. If there was at least one good harvest during the paternal grandmother's SGP, her grandchildren had an increased risk of developing diabetes.

It is hard to know how to interpret these inscrutable patterns of inheritance. Some skepticism is warranted. From a methodological standpoint, the finding of statistically significant associations between grandparental consumption and the health of grandchildren appears to rely on the multiplication of covariates by drawing ever finer distinctions (e.g., paternal grandfather v. paternal grandmother, maternal grandfather v. maternal grandmother, sons' sons v. sons' daughters, daughters' sons v. daughters' daughters, ages 9–12 [v. any other age]). Regardless, the results of the Överkalix studies are the converse of the results of the Holocaust studies. Whereas in the latter, a "trauma-inducing" environment was associated with good outcomes in offspring, here, a good environment (abundant food supply) was associated with bad outcomes (greater all-cause mortality). Both are contrary to the narrative of TST.

In 2018, utilizing the Uppsala Multigeneration Study, which has 40 times the number of subjects as the Överkalix study, (F0, n = 9039; F1, n = 7280; F2, n = 115610) researchers set out to replicate the main Överkalix findings (Vågerö et al., 2018). They reported replication of the first finding but failed to replicate the last three. One new finding they reported was that the elevated mortality rates among grandsons were, apparently, due entirely to a higher risk of dying from cancer.

One noteworthy feature of the single finding that appears to have survived replication thus far—one good harvest during the paternal grandfather's SGP is associated with an increased all-cause mortality rate in grandsons—is that the supposed transgenerational epigenetic inheritance appears to have skipped a generation. As the authors of the original study note:

[1] This generates the impression that the SGP is an important developmental period and that this could explain why only exposures during this period affected grandchildren. However, the only references to the SGP in this sense occur either in the Överkalix studies themselves or in studies attempting to replicate or expand the Överkalix findings.

[2] All cause morality refers to death from all causes in a given time interval for a given population. This is sometimes carelessly and inaccurately referred to as longevity. All-cause mortality could include, for example, deaths from auto accidents.

> Parents' longevity [by which the authors mean all-cause mortality; see footnote b.] was … not significantly affected by grandparents' access to food during the SGP … The parental generation appeared to have been skipped (Bygren et al., 2001, p. 57).

Generations are not "skipped" in transgenerational inheritance. Since we are dealing with only three generations, this fact alone is sufficient to draw the conclusions into doubt.

The Överkalix studies highlight some of the difficulties involved in studies of transgenerational inheritance, particularly when dealing with the behavior of persons who lived a century or more ago. Researchers often make broad assumptions based on sparse data, opening the door to omitted variable bias. Consider a few examples:

- Periods of good harvest were treated not only as periods of good food availability, but as periods of *overeating.* Is this a reasonable assumption? Was storing grain an impossibility? Was there no trade or exchange of farmed goods?
- Exposures to all poor harvests up through the end of the nineteenth century were treated as exposures to equivalent environments, despite significant variations in food availability (Bygren et al., 2001).
- All grandparental exposures occurred between the ages of 8–12 years old. Parents often shield children from experiencing food insecurity, particularly very low food security, even when the parents themselves are food insecure (Nord, 2013). While this may not have been possible during a time of extreme famine, it may have affected children's exposure during less severe food harvests.

3.2.2. Dutch famine studies

The Dutch famine studies concern the effects of the 1944–1945 Dutch famine on the offspring of pregnant mothers (Bleker et al., 2021). The health outcomes of offspring in utero during this time were found to be considerably worse than controls in many ways (Roseboom et al., 2000a; Roseboom et al., 2000b; Ravelli et al., 1999; Roseboom et al., 2001). These studies, however, are not studies of epigenetic inheritance, either inter- or transgenerational. They do not involve the inheritance of environmentally induced epigenetic changes (and corresponding phenotype) via the germline. Instead, they concern the effects of the maternal prenatal environment on offspring, and this is the case even if epigenetic mechanisms play a role in the negative outcomes of offspring.

Researchers have looked for evidence of intergenerational inheritance by considering the health outcomes of the children of parents exposed to famine prenatally. These would be examples of intergenerational and not transgenerational inheritance because the parents' germ cells would have been exposed to the famine in utero. The evidence for intergenerational inheritance is meager. According to one study, children of mothers prenatally exposed to famine were slightly shorter on average at birth, and according to another study, offspring of fathers prenatally exposed to famine had a higher BMI at age 37 years (Painter et al., 2008; Veenendaal et al., 2013). However, no differences in the offspring of men and women who were exposed to famine in utero have been identified for prematurity rates, cardiovascular and pulmonary disease, elevated cholesterol, diabetes, hypertension, obesity, psychiatric conditions, mortality rates, and poor health in general (Veenendaal et al., 2013).

4. Conclusion: the problem with the argument for the transgenerational inheritance of the trauma of slavery

In this paper, we have argued that studies typically invoked in support of inherited slavery trauma are seriously flawed and provide little support. Specifically, we have reviewed the literature to find that there does not appear to be an epigenetic pathway for the transgenerational transmission of trauma. The mechanisms of transmission outlined in the reviewed evidence provide no evidence of a pathway extending across five or more generations, the minimum to attribute modern-day outcomes to slavery-era trauma. Moreover, there is no evidence of transmission, let alone effects of transmission leading to any disparate outcomes in later generations on the scale of modern-day black-white disparities in health or other aspects of society.

At the risk of belaboring our point, however, it warrants mention that the analogy between TST and the phenomena investigated in these studies of potential epigenetic transmission breaks down in a critical way.

As noted above, documenting transepigenetic inheritance requires the absence for several generations of the environmental input assumed to have caused the initial epigenetic change in the F0 generation. For this reason, studies of the transepigenetic inheritance of, for example, famine-induced epigenetic alterations have concerned famines with an identifiable onset and end. TST, on the other hand, clearly does not qualify for these experimental conditions: While chattel slavery as a legal institution was abolished in the United States with the passage of the Thirteenth Amendment in 1865, central features of American society have perpetuated racialized disadvantage and adversity among African Americans over the last 160 years—and continue to do so.

With a few minor exceptions (i.e., so-called imprinting disorders), the role of specific epigenetic modifications on phenotypes is not known. Epigenetic information derived from whole blood tells us little about the epigenetic state of relevant cells (e.g., in the brain or pancreas) and tells us little about transmission (which would require, at the very least epigenetic information from germ cells). Hence, the role of epigenetic information in demonstrating transepigenetic inheritance is, for the most part, ancillary, and many studies do not examine DNA methylation status. Instead, they rely upon phenotypic differences between cases and controls.

As noted, this phenotypic information is taken to provide evidence of transepigenetic inheritance by a process of elimination or "proof by elimination" (e.g., given the phenotype observed in great-grandchildren, the only reasonable explanation is the fact that great-grandparents were exposed to a trauma-inducing environment) (Costa et al., 2018). In short, researchers' *justification* for invoking inter- or transgenerational epigenetic inheritance is that it is *the only possible explanation for the phenomenon under consideration.* Researchers believe they are justified in making this claim because they have taken into account (i.e., "eliminated") any other possible environmental (or genetic) explanations, which is why these studies are so vulnerable to omitted variable bias.

Imagine a study that claimed to have found evidence for transgenerational epigenetic inheritance, extending from parents to great-grandchildren, of adverse health effects associated with the parents' exposure to a traumatic environment (of any kind). However, while the parents experienced this traumatic environment, so did the members of each subsequent generation (to a greater or lesser extent), that is, their children, grandchildren, and great-grandchildren. If the members of every generation exhibited the same phenotypic differences, then the explanation would be each generation's *direct exposure* to a trauma-inducing environment (Green and Darity, 2010).

If every generation is exposed to a trauma-inducing environment and exhibits signs of trauma, then there is no justification for proposing transepigenetic inheritance of trauma as an explanation. What is it explaining that is not already explained by the experience of a trauma-inducing environment? To propose transepigenetic inheritance in such a situation is to posit a hidden, indemonstrable, and otiose causative agent (the very existence of which as a biological phenomenon in humans is uncertain).

Such a proposal is analogous to supposing that TST explains (either wholly or in part) the current black-white health gap. Consider a recent report by the National Coalition of Blacks for Reparations in America titled, "The Harm is to Our Genes" (Kaufman et al., 2021). The authors note, correctly, that "Addressing the key structures in American society that perpetuate cycles of disadvantage and ongoing experiences of

adversity and trauma for African descendants is critical" (Kaufman et al., 2021, p.25). Yet, simultaneously, the bulk of their report is devoted to a defense of TST.

If every generation of blacks in the US from the time of slavery to the present has been exposed to "structures that perpetuate cycles of disadvantage," and has ongoing experiences of trauma and adversity, then what is the justification for proposing the transepigenetic inheritance of trauma? What does TST explain that is not already explained by ongoing exposure to a day-to-day trauma-inducing environment (and by SDOH)?

The notion that these historical harms still exist in "our genes" is potent and poetic. It is reasonable that hearing the causes of modern-day disparities stem from centuries prior is comforting to members of these populations. It is meaningful to feel solidarity in such a struggle, to know that the headwinds one faces have been felt for quite some time. Moreover, the connections that the TST hypothesis makes across different knowledge domains may have allowed it to gain more traction and spread more widely (Deichmann et al., 2020). However, as this theory and similar discussions of trauma's transmission move into popular spheres of discussion, the language around these discussions has slipped into imprecision, making it "increasingly important to be clear about what is being transmitted, and how such transmission might occur" (Yehuda and Lehrner, 2018, p.14).

The TST hypothesis makes a key misstep: these harms are not biologically inherited but are socially produced. A more precise explanation for modern-day health disparities can be found at a systemic level, via Fundamental Cause Theory, Ecosocial Theory, or any theory that connects historical and ongoing discrimination to social determinants of health. Indeed, the best explanations are those that span generations. Black Americans do not suffer from DNA damaged from slavery but starkly reduced opportunities for social mobility and far greater economic insecurity (Sharkey, 2008; Chetty and Hendren, 2018). What black Americans have inherited is not a psycho-genetic disadvantage but a *resource disadvantage*: a wealth gap in a society that refuses to enact policies to close it, such that the gap has persisted since 1860 and is, in fact, widening in recent years (Derenoncourt et al., 2024).

Building the argument for black reparations (or any policy to promote racial equity) on a foundation of TST is shoddy craftsmanship: It is much cleaner and safer to make these arguments using the tangible evidence—seen in wealth, health, housing, education, and other areas of society—of the outcomes of the systemic discrimination that has persisted since the country's founding (Darity Jr and Mullen, 2022; Darity et al., 2023). One need not travel to the genetic level to see these effects.

There is no basis for assuming that the trauma of slavery is inherited. Rather, what has been "inherited," and, barring drastic change, will continue to be inherited, is a social environment still shaped by the pernicious legacy of slavery.

CRediT authorship contribution statement

Evan Charney: Writing – original draft. **William Darity:** Writing – review & editing. **Lucas Hubbard:** Writing – review & editing.

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Data availability

No data was used for the research described in the article.

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