PANDION: The Osprey Journal of Research & Ideas

Volume 2 | Number 1 Article 3

2021

Embodied Injustices: COVID-19, Race, and Epigenetics

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Abstract

The co-occurrence of the COVID-19 pandemic with the long-lasting effects of systemic racism has been devastating, and results in vast inequities in infection and mortality rates within communities of color. In this article, I analyze the potential for epigenetic research to operationalize the social science theory of embodiment, which describes how the social and material worlds manifest in our physical bodies. Epigenetic modifications can be triggered by environmental stressors, to which minority populations are more likely to be exposed. In turn, these stressors are linked to disorders that increase COVID-19 susceptibility. Thus, epigenetic modifications provide an avenue by which racialized social experiences may become embodied as comorbidities that enhance vulnerability to COVID-19. I contextualize the epigenome's permeability in larger discussions about the social construction of race, inheritance, and calls for racial equity.

Introduction

Racial identity defines boundaries of community and impacts the experiences of individuals, including how people live and die during a pandemic. At the time of this publication, the COVID-19 pandemic has killed 2.5 million and infected 114 million people worldwide (Johns Hopkins University & Medicine 2021). In the racialized society of the United States, infections expose vast racial inequity. When adjusted for age, Pacific Islanders, Latinos, Indigenous, and Blacks are respectively 2.7, 2.4, 2.2, and 2.1 times more likely to die from COVID-19 than Whites (APM Research Lab 2021). Simultaneously, residents of the United States grapple with the aftermath of the killing of George Floyd at the hands of Minneapolis police officers. The resulting resurgence of the Black Lives Matter movement, which counters white supremacy and violence against Black communities, sparked national conversations about systemic racism (Black Lives Matter n.d.).

The intersection of these events throws into sharp relief how biology and culture are intertwined in relation to human health. Although race is a culturally determined social construction, it also constitutes a health risk. This year's jarring experiences are a single snapshot of this country's long history of racial health inequity. Marginalized racial groups routinely experience worse outcomes across multiple dimensions of health and across multiple generations. In this paper, I discuss how COVID-19 outcomes reflect the blurred line between biology and culture. I propose that racialized social experience may affect the human body through intergenerational biological mechanisms.

Defining Inheritance

Humans inherit a variety of items ranging from property and generational wealth to genetic material in the form of "genes." I will focus on the biological inheritance of both genetic and environmentally induced traits, which roughly correspond to the concepts of "hard" and "soft" inheritance. Hard heredity generally refers to how genetic traits are inherited at conception without significant environmental influence (Bonduriansky 2012). The field of genetics has largely operated off this understanding of heredity to analyze the role of the genome, which refers to the totality of an organism's genetic information packaged into genes. An organism's genetic makeup is generally referred to as its genotype. Genes are specific DNA nucleotide sequences that lie on chromosomes and are characterized by their unique combination of nitrogenous bases: Adenosine, Guanine, Cytosine, and Thymine. Each sequence of As, Gs, Cs, and Ts codes for a specific protein. Given that proteins are important building blocks and regulators of the body, inheriting a set of proteins means that the organism also inherits the physical traits, or phenotype, to which these proteins correspond.

Although most of us are not geneticists, most people's understanding of how traits are acquired is based upon this limited concept of hard heredity. This contributes to overly deterministic understandings of heredity – that every trait in our body can be directly linked to our genotype. This fascination with finding a singular pivotal gene for specific conditions is so widespread that headlines like "Scientists Discover So-Called 'Skinny Gene'" are at once spectacular and unexceptional (Anderer 2020). The "geneticization" of human biology in which individual differences, behaviors, and disorders are primarily attributed to one's genetic code was first described by feminist and epidemiologist Abby Lippman

(1991). Her warnings joined other critiques that using genes as proxies for human identities and traits while dismissing the impact of sociocultural and historical environments masks and facilitates oppression (Arribas-Ayllon 2016).

In large part, our understanding of how genes become expressed traits is based on a two-step pathway. The DNA of genes is transcribed into RNA, which is then translated into proteins. Hard heredity has enshrined this strictly unidirectional pathway as the "Central Dogma" of molecular biology with no opportunity for environmental factors to add significant modifications (Crick 1958). However, there is evidence that this process is not as impervious to environmental influence as previously believed.

In contrast to hard heredity, soft heredity describes how the environment influences the transmission of traits. It explains how traits, including those acquired within the parents' lifetimes, are passed to offspring (Bonduriansky 2012). How our environments shape our bodies has been a topic of much theorizing. Specifically, the theory of embodiment suggests we incorporate aspects of our material and social worlds into our biology. Embodiment is a continuous process spanning from conception to death (Krieger 2001). Up until recently, the discourse around embodiment has mostly focused on bodily manifestations of our environments within our own lifetime. However, the transmittance of these embodied traits might not be so neatly confined by the bookends of conception and death. The emerging field of epigenetics suggests biological traits determined by the outside environment can be passed on *intergenerationally*.

Epigenetics is the study of changes beyond the nucleotide sequence of DNA that affect gene expression (Moore 2012). There are several epigenetic mechanisms, such as histone modification and DNA methylation. Both can be altered by environmental factors (Handy et al. 2011). Histones, the proteins around which DNA strands are wound, can be modified by changing the chemical tags (like acetyl, methyl, or phosphoryl groups) attached to them (Handy et al. 2011). This results in a shape change of the histone that either exposes more or less of its associated DNA. This affects the ability of transcription proteins to access the DNA, thus impacting translation, and ultimately leading to elevated or lowered protein expression (Handy et al. 2011). DNA methylation describes the addition of a methyl group directly to the DNA sequence which disrupts the attachment of transcription proteins or recruits gene repression proteins (Moore 2012). While both mechanisms alter gene expression, DNA methylation is more stable under DNA replication and cell

division; methylation patterns are found to be conserved across multiple generations (Handy et al. 2011). For epigenetic markers to be heritable, they must be retained during gametogenesis: the creation of egg and sperm cells. DNA methylation is particularly capable of surviving the "cleansing and resetting" that occurs during gametogenesis and subsequent fertilization, explaining the longevity of these markers (Krippner and Barrett 2019).

The regulation of protein expression is not new to the field of genetics. The body is constantly elevating or lowering the expression of proteins to adapt to an ever-changing environment. However, the idea that these epigenetic markers are heritable is both revolutionary and familiar. It contradicts strict hard heredity and bolsters soft heredity by positing that the environmental influences underestimated in previous models of inheritance play an important role. The epigenetic view recognizes the interplay between both genes *and* environment in determining offspring phenotype. Furthermore, the study of epigenetics expands the scope of embodiment. Traits displayed as phenotypes may be the result of an embodiment of our own environment and also the environments of our ancestors. Accordingly, identifiable epigenetic markers offer an opportunity to operationalize the influences of our physical and social worlds, like racial status.

Epigenetics and Race

Science is always political. Despite the posturing that the natural sciences are a strictly empirical discipline, they have been used to entrench existing positions of power and status or to define new ones. The entanglement of science and politics is evident in the development of bioweapons, human stem cell research restrictions, and the debate around climate change where human belief systems dictate what science gets prioritized. Science exists in the social world; thus, society will always determine who scientists are, what they focus on, and how findings are communicated and framed. Interconnection is unavoidable. Still, we should attempt to disentangle narratives that have actively led to the subjugation of vulnerable populations. Few constructs have so saliently ossified power differentials than the concept of "race" by creating a biological "other."

In this paper, I capitalize all racial categories to signal that they are social constructions (Appiah 2020). I elect to use the same categories as the authors I reference in each section in lieu of standardizing terms referring to racial categories

throughout the article. For example, I may use "Black" in one section and "African American" in the next, following the lead of the literature I cite. The oscillation underscores the impreciseness and inconsistency of how researchers define race. Thus, my conclusions are inevitably constrained by the same limitations of all studies that rely on racial categories. Racial labels often mask dramatic disparities between populations that fall under the same group. For example, 72% of Indians and 9% of Bhutanese Americans hold bachelor's degrees, reflecting the vast differences in socioeconomic status among those labeled as "Asian" (Kochhar and Cilluffo 2018). Furthermore, Native American populations are routinely excluded from research findings due to small population sizes, chronic misidentification of their race, and the lack of funding and institutions that serve Native communities (Nagle 2020). These blind spots and the lack of standard definitions for racial categories reflect the inadequacy of race in characterizing human populations.

Over the past few decades, biologists have presented evidence that racial categories have little biological meaning. Criticisms of race as a biologically significant category gain strength occasionally throughout history, most recently following the Human Genome Project, which successfully sequenced the entire human genome in 2003 (National Human Genome Research Institute 2020). The advances in DNA sequencing technology that made the project possible continue to develop. Accordingly, the genomes of multitudes of individuals from different racial backgrounds have been sequenced and compared.

As with any species, humans display genetic variation. However, the majority of this variation is not between socially defined racial categories. There are significant genetic differences between populations separated by time and geography but these differences do not line up neatly with the subjective characteristics associated with race such as skin color or hair texture (Kolbert 2018). In fact, there is more variation *within* racial categories than *between* them. Humans are remarkably genetically similar due to the relative brevity of our evolutionary existence, with 99.5–99.9% of our genome being identical (Perez-Rodriguez and de la Fuente 2017). Of the remaining "0.1–0.5% variation between any two unrelated individuals, the largest amount of variation, 85%, is between individuals within the same local population" (Perez-Rodriguez and de la Fuente 2017).

So, if race is not primarily founded on biological difference, from where did the concept of race arise? French physician Bernier was the first to use the term "race" in

his 1684 publication (Meloni 2016). Shortly thereafter, Carl Linneaus, the famous taxonomist responsible for binomial genus/species nomenclature, introduced familiar categories as part of his delineation of Homo sapiens. Sparing no organism in his zeal for meticulous classification, he identified four main groups: *H. americanus, H. africanus, H. europaeus, and H. asiaticus* (Smedley et al. 2020). He characterized geographic subspecies like Europaeus albus as "gentle and governed by laws" and *Afer niger* as "black, crafty and governed by caprice" (Meloni 2016). Given the political dominance of Europe during this period, the scientific findings of the time attributed positive characteristics to cultures most similar to those of Europe. In contrast, unfamiliar peoples were assigned "savage" caricatures, and this was used to justify the "civilizing" colonialist wave of this era.

While multiple fields have contributed to scientific racism beyond this early taxonomy, genetics has a particularly influential role. In 1883, in addition to coining the term "gene," Sir Francis Galton also coined the term "eugenics" (Meloni 2016). In pinpointing the unit of inheritance, he saw the potential to selectively breed humans for traits like intelligence to better the human race (Meloni 2016). As a member of the English elite, Galton attributed superior traits to the upper classes. Yet, his ideas never permeated Britain to the extent they did in the United States. The American Eugenics movement of the late 19th and 20th centuries sanctioned forced sterilizations of those deemed "unfit:" the poor, minorities, and people with mental and physical disabilities (History 2019). These efforts to wipe out the perceived negative influence of marginalized social groups became fixtures of American society. Civilian-led institutions like the Race Betterment Foundation founded by cereal titan John Harvey Kellogg, and state and federal governments, advocated and executed eugenics policies (History 2019). Even after the eugenics experiments and genocides committed by Nazi Germany during World War II garnered international condemnation, the United States continued similar atrocities on its own soil. According to reports that surfaced during a 1976 Government Accountability Office investigation, between 1970 and 1976 an estimated 25-50% of Native Americans were sterilized (Rutecki 2010).

One of the dangers of the eugenics movement is that it framed "inferior" traits as inescapable, an idea that bleeds into modern conceptual frameworks. Poorer health outcomes continue to be routinely conflated with inherent genetic inferiority rather than being seen as products of systemic racism and depressed socioeconomic

conditions. The acceptance of substantial genetic differences between "races," even among health experts, persists. For example, a study investigating the disparity between premature birth among Black and White women claimed "important genetic contributors to the timing of birth" despite an absence of genetic data to back the claim (Kistka et al. 2007). In his commentary on this case, medical anthropologist Clarence Gravlee (2009) points out the absurdity of providing a genetic hypothesis without genetic data, underscoring how easily weak conclusions bleed into public discourse. Shortly after the study's initial publication, the *New York Times* featured it in a piece titled "Study points to genetics in disparities in preterm births" (Bakalar 2007).

Epigenetics and soft inheritance might offer an alternative avenue to explain racial differences. Epigenetics offers a way to view phenotypes as the embodiment of racialized social experiences, not as hardwired inevitabilities. Although race is a social construct, the consequences of this categorization are real. Racial identity defines the boundaries of community and the experiences of individuals. Epigenetics may hold the key to understanding how populations experiencing widespread extraordinary experiences, like trauma and chronic stress, impact future generations of offspring.

Epigenetics confirms what many communities of color already know. Many indigenous scholars have embraced epigenetics as an acknowledgment of the lasting health effects stemming from colonial violences. For example, LeManuel Bitsoi, who belongs to the Navajo Nation and whose background is in genomics and bioinformatics, said of the link between adverse experiences and illness that "'Native healers, medicine people and elders have always known this and it is common knowledge in Native oral traditions'" (Pember 2017).

Epigenetics illuminates the ways humans embody social experiences like inequity, stress, and trauma. Although a person's biology is not equivalent to their race, race may *become* biology (Gravlee 2009) through the epigenome. Our genome might not vary significantly by race, but our *epi*genome might. There is now a multitude of research into the epigenetic markers behind alcoholism, cardiovascular disease, and metabolic diseases that show racial minorities suffer poorer health outcomes (Handy et al. 2011, Knight and Smith 2016). Without proper contextualization of the oppressions behind many of these differential outcomes, minority groups and individuals are often blamed as individual choices – not societal imbalance – come under scrutiny.

COVID-19 and Minorities

The coronavirus disease 2019 (COVID-19) is the disease caused by a specific virus: severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). There is long-standing research on coronaviruses, as some of the most notorious viruses, such as SARS and MERS, are of the same family origin (Cascella et al. 2021). But viruses are rapidly and constantly evolving; SARS-CoV-2 is unique compared to any other previously identified coronavirus. Accordingly, although the eyes of the global research community are fixed on COVID-19 and the SARS-CoV-2 virus that causes it, there are still many unknowns surrounding what makes certain groups particularly susceptible to infection and higher mortality.

In the initial months of the pandemic reaching the United States, SARS-CoV-2 was portrayed as a virus to which everyone is equally susceptible. Responding to his brother's infection, New York Governor Andrew Cuomo noted that "this virus is the great equalizer" (Cuomo 2020). This statement was intended to encourage caution. If the virus is indiscriminate in its selection of targets, we all have an equal stake in heeding orders to social distance, wear masks, and limit the extent to which we circulate in an effort to protect ourselves and others. Yet, as the disease spread within the U.S. and beyond, it became increasingly clear that rather than being an equalizer, COVID-19 is an amplifier of existing class and racial inequities. Governor Cuomo would come to see this play out in his own state. In New York alone, Black and Hispanic individuals accounted for 23% and 24% of all deaths despite only making up only 14% and 19% of the population respectively (Kaiser Family Foundation 2021).

Several explanations have come to the forefront of the discourse regarding the racial divide in health, with varying degrees of empirical soundness. Since COVID-19 has emerged as an issue of national security and policy, government officials have a particularly strong reach irrespective of their epidemiological backgrounds, leading to false claims about minority health permeating public discussion. When addressing a question about the disproportionate toll COVID-19 was taking on Black communities in his state, Louisiana Senator Bill Cassidy, himself a physician, pointed to African Americans´ suffering from underlying conditions as the "fundamental reason" for this disparity (NPR Morning Edition 2020). He chalked up the disparity to innate "genetic reasons" citing that "the virus likes to hit what is called an ACE2 receptor... African Americans are going to have more of those receptors inherent in their having the diabetes, the hypertension, the obesity" (NPR

Morning Edition 2020). This was a likely reference to research proposing that the ACE2 receptor facilitates the virus' entry into the body. According to recent research, SARS-CoV-2 has proteins that form "spikes" on its surface. These spikes have special regions that bind to receptor proteins on the body's cells. Following attachment, SARS-CoV-2 may then cross into the new host, resulting in infection. The ACE2 receptor may have a structure that favors this binding (Cascella et al. 2021). However, a link between different levels of ACE2 expression and race is far less certain. Results have shown higher ACE2 expression among Asians, for example, only for later studies to show no significant difference across race at all (Zhao et al. 2020, Li et al. 2020, Chen et al. 2020). Senator Cassidy proposes a link between higher ACE2 expression and disorders like hypertension (high blood pressure) in African Americans. However, evidence suggests expression is actually lower in hypertensive Black populations including Afro-Caribbeans (Cohall 2020).

By critiquing Senator Cassidy's remarks, I am not rejecting the existence of any genetic link behind SARS-CoV-2 infection or mortality rates. If evidence were to emerge tomorrow that African Americans do have higher ACE2 expression levels, that would not detract from my critiques. I argue that genetics alone cannot explain the monumental racial disparity in COVID-19 outcomes nor health disparities more generally. I criticize the senator's fixation on a genetic explanation at the cost of ignoring the social factors that impact our biologies. When asked about how the underlying conditions he mentioned are "rooted in years of systemic racism," he responded, "that's rhetoric, and it may be. But as a physician, I'm looking at science" (NPR Morning Edition 2020).

Genetics and sociocultural influences are not mutually exclusive. Our bodies experience and adapt to our environments and leave tangible epigenetic markers as evidence of the interplay between biology and culture. These markers may impact health outcomes by contributing to the comorbidities that make individuals more vulnerable to SARS-CoV-2. Since these conditions typically are attributed to behavioral components such as diet and exercise, patients are often blamed for their own poor health. However, the higher prevalence of these diseases in minorities also suggests that oppressive external factors play a significant role. I argue that epigenetic mechanisms may further embody racialized experiences through the metabolic, stress, and respiratory disorders that put one at increased risk for severe illness from SARS-CoV-2.

Metabolic Disorders

Metabolic disorders encompass a wide category. They generally refer to any disorder that disrupts our ability to properly break down and utilize the food we ingest (MedlinePlus 2019). In my discussion, I reference type 2 diabetes and hypertension. Both are listed as leading risk factors for COVID-19 and often coincide in relationships that are cyclical and reinforcing (Centers for Disease Control and Prevention 2021).

Diabetes interferes with the production or function of insulin, which regulates the amount of glucose in the blood, affecting the body's metabolism and growth (Wilcox 2005). Type 2 diabetes (T2D) interferes with the body's ability to properly respond to insulin and typically emerges later in life. Like other metabolic disorders, minorities are more likely to have T2D. Given that 90-95% of diabetes cases are T2D, national estimates as compiled by the CDC do not differentiate by diabetes type. In ascending order, diabetes is diagnosed in approximately 7.5% of non-Hispanic Whites, 9.2% of non-Hispanic Asians, 12.5% of Hispanics, 14.2% of non-Hispanic Blacks, and 14.7% of American Indians and Alaska Natives (Centers for Disease Control and Prevention 2020). It is important to note that these racial categories blur significant variance, particularly between different Asian, Hispanic, and Native communities.

I discuss how malnutrition and stress contribute to T2D and how they may be passed on through epigenetic mechanisms. However, T2D is a complex disease; epigenetics joins a wide host of other factors that determine susceptibility. Yet despite this large field, lifestyle and diet dominate views towards T2D. This focus can be problematic as it places the blame squarely on the patient. A 2017 study found the majority of T2D patients felt stigmatized, most commonly citing perceptions that T2D is a character flaw or personal failure due to "overeating, poor diet, inactivity, laziness, or being overweight or obese" (Liu et al. 2017). When these negative stereotypes about T2D collide with existing stereotypes of minority communities, what results is a pathologization of minority culture that distracts from structural barriers.

Caricatured versions of "ethnic" foods have contributed to a false impression that many minority diets are inherently unhealthy in comparison to the average American diet. Yet, it is the Americanized versions of minority diets that are overly carbohydrate and fat dense. The Latino food familiar to mainstream culture is a far cry from the original dishes. In fact, when accompanied by the fresh produce that first or second-

generation immigrants are more likely to incorporate into their diet, Latino traditional diets are relatively well balanced (Heuman et al. 2013). Latinx communities may be experiencing increased rates of diabetes and metabolic disorders because they are increasingly incorporating elements of the mainstream diet, not because they are retaining traditional foods (Heuman et al. 2013). Similarly, a long-term study concluded that Pima Indians who ate diets typical of the U.S. developed diabetes at 2.5 times the rate of those who followed a traditional diet (Williams 2001).

Black communities, especially those in southern regions of the U.S., have not escaped scrutiny. Ibram X. Kendi notes how the senior editor of *The American Conservative*, Rod Dreher, considered "to what extent black folks all over the country still eat the traditional soul food diet with lots of grease, salt, pork, sugar, and carbs" in defense of Senator Cassidy's previously mentioned remarks. Also included is a reader's response that "I am especially amused by the implication that a racist conspiracy is keeping brussels sprouts and kale from black neighborhoods. If people wanted fresh vegetables and salads and tofu, stores would provide them" (Kendi 2020).

The problem is that the stores where minorities live *are* far less likely to carry fresh and healthy foods, or at least they are less likely to carry them at affordable prices. Black, Hispanic, and Native people are more likely to live in urban and rural "food deserts": areas that lack affordable healthy food within reasonable distances (Dutko et al. 2012). For example, instead of being able to frequent larger grocery stores equipped to carry perishable foods, individuals living in an area characterized as a food desert may only have access to corner stores that do not regularly stock fresh vegetables and fruits. The food available in these environments is typically high calorie but nutrient deficient. The resulting food insecurity makes people in food deserts more susceptible to T2D, hypertension, and obesity (Pan et al. 2012).

Before the pandemic hit, food security was a major concern for communities of color. According to the U.S. Department of Agriculture (USDA), 21% and 16% of Black and Hispanic households respectively suffered from food insecurity in 2018 in comparison to 8% of White households. This means that at some point over the course of that year, a member of those households had insufficient access to food for a healthy and active lifestyle. In some cases, this progressed to the point that some members disrupted their normal eating patterns (USDA 2020). Not getting sufficient nutrients, whether due to a simple lack of food or an excess of unhealthy foods, contributes to health effects that linger for generations.

Epigenetic research offers explanations as to how malnutrition can be translated into metabolic disorders appearing in individuals and their offspring. Both maternal and paternal malnutrition, especially during times close to early gestation of the fetus, can result in epigenetic modifications that lead to metabolic disorders in addition to endocrinological and general organ dysfunction (Patel et al. 2017). One of the most famous cases in the field of epigenetics studied the descendants of those who lived through a famine known as the Dutch Hunger Winter. The investigators found that the DNA methylation of the IGF2 (insulin growth factor II) gene of children carried by mothers who were pregnant during the famine was reduced in comparison to their same-sex siblings six decades later (Heijmans et al. 2008). Although IGF2 activity is complex and not completely understood, it appears that this difference in methylation is linked to physiological changes. A later study connected the famine to increased fat tissue in newborn grandchildren, suggesting that epigenetic modifications may remain after two generations (Veenendaal et al. 2013). Additionally, higher expression of IGF2, which coincides with reduced methylation, has been proposed as a possible trigger for the onset of T2D (Casellas et al. 2015).

Contemporary minority populations are more likely to be malnourished, and malnutrition is often experienced across generations. The diets of slaves were largely monotonous and not well balanced, leading to malnutrition, especially in children and infants (Steckel 1986, Guzmán 2012). Even if times of such severe malnutrition are long past, the marks may still linger. Today, IGF2 methylation patterns appear to vary by race. In studying newborn DNA extracted from their umbilical cords, investigators found racial differences in the methylation of IGF2 even after results were adjusted for socioeconomic status (King et al. 2015).

The occurrence of these epigenetic modifications following times of malnutrition suggests these changes may be adaptations to promote survival under nutrient shortages in the short term (Vaiserman and Lushchak 2019). For example, if your parents are not getting enough nutrients, it is likely that you will be born into a similar environment. Thus, insulin resistance and extra fat stores in newborns would be advantageous during events like famines or slavery. This "Predictive Adaptive Response" in which parental cues trigger the development of a phenotype optimally adapted to the parental environment can result in poorer health if these adverse conditions dissipate during the offspring's life (Bateson 2014). Most Americans do not face famine-level calorie shortages, even if their ancestors did. These adaptations

that promote fat accumulation become harmful if that fat is not used, leading to conditions like obesity and T2D.

The fetal epigenome is incredibly malleable, especially when compared to other life stages. Thus, like much epigenetic research, many of the aforementioned studies focus on pregnant mothers, fetuses, and newborns. This focus extends to premature birth, another health condition with massive race disparities. Black women are the most likely to deliver premature infants, closely followed by American Indians, Native Alaskans, and Pacific Islanders (Martin et al. 2018). These disparities in pregnancy and birth complications remain, regardless of the socioeconomic status of the mother. Even when adjusted for variables like income and education, Black women are five times more likely to die during childbirth than White women who similarly hold at least a college degree (Petersen et al. 2019). This suggests that the heritability of premature birth (in other words, women born prematurely are more likely to give birth to premature babies) cannot be wholly explained by depressed socioeconomic conditions in the mother's lifetime alone (Knight and Smith 2016). Nor can it be wholly explained by inherent biological differences between races. Non-Hispanic Black women born in the U.S. continue to have significantly higher rates of premature birth in comparison to Caribbean and Sub-Saharan born non-Hispanic Black immigrants (Elo et al. 2014). When analyzed together, these data suggest that the unique cultural environment experienced by minorities in the United States creates specific health outcomes.

Epigenetics may provide us with a way of better understanding this embodied phenomena. T2D increases the risk for premature births and premature infants have higher rates of T2D (Knight and Smith 2016). Therefore the ability for T2D to be possibly passed on through multiple generations via epigenetic modification may make premature birth more heritable by association. Furthermore, methylation patterns for thousands of genes in newborns change with each additional week of gestation (Merid et al. 2020). In other words, premature babies have significantly different epigenetic profiles in comparison to babies born at the end of the gestational term. IGF2 is one of the affected genes. Specific regions of IGF2 are differentially methylated in individuals born prematurely, with these patterns still persisting for at least 20 years (Wehkalampi 2013). Since the field of epigenetics is still relatively new, longitudinal studies with longer time frames are still needed to determine exactly how long these modifications persist.

Hypertension, another leading risk factor for severe COVID-19, also increases the likelihood of delivering prematurely and is likewise linked to epigenetic modifications (Catov 2008, Liang 2019). As stated previously, hypertension and T2D often occur together and individuals with one are at an increased risk of developing the other. However, there are more factors contributing to the development of hypertension that involve epigenetic changes like stress, as described in the next section.

Stress Disorders

Experiences of severe stress can lead to a variety of mental health issues, including Post Traumatic Stress Disorder (PTSD). Strong evidence taken from different populations indicates that PTSD is heritable. The children of Holocaust survivors, Cambodian refugees, and the Rwandan genocide are more likely to develop PTSD despite having never directly experienced these conflicts (Yehuda et al. 2008, Sack et al. 1995, Shrira et al. 2019). For Holocaust survivors, sustained psychological distress was both in their children and grandchildren (Scharf 2007). While many studies of intergenerational trauma argue that behavioral causes (such as abuse) lead to the reproduction of PTSD in children, new studies pinpoint DNA methylation as another possible link between parent and offspring.

Parental stress is associated with differential methylation of genes with functions ranging from protein transport to immune response (Wankerl et al. 2014, Cao-Lei et al. 2014). Most epigenetic studies of PTSD focus on NR3C1, which codes for a glucocorticoid receptor (Bowers and Yehuda 2016). Although different studies have found both increased and decreased methylation of NR3C1, it is clear that atypical levels of methylation are linked to PTSD and parental stress (Radtke et al. 2011, Mulligan et al. 2012). Special attention is accorded to NR3C1 given its important role in the stress response.

Glucocorticoids include a wide range of hormones that regulate how we perceive and respond to threats, quickly mobilizing our body to defend itself. This is commonly referred to as the "flight or fight" response (Cannon 1915). Glucocorticoid stress hormones function in a negative feedback loop that regulates the secretion of hormones from the hypothalamus, anterior pituitary, and adrenal cortex (Gjerstad et al. 2018). This means that in a properly functioning system, the release of these secondary hormones eventually triggers the end of the stress response. This allows the body to relax. If parts of this cascade are altered, such as the

glucocorticoid receptors NR3C1 codes for, this can impair the proper functioning of this negative feedback loop. This may extend the stress response, prolonging the amount of time our body is awash with stress hormones, resulting in an over-taxation of the body, particularly the cardiovascular system (McEwen 2006).

If traumatic events spanning years, like those experienced by the Holocaust or Rwandan Genocide victims, are able to leave such tangible genetic markers, what modifications might be present in the epigenomes of minority populations that have endured traumas lasting centuries? It is undeniable that populations from all races have suffered traumatic events at various points throughout history. Still, traumatic experiences are more recent and numerable for minority communities in the United States. Our nation's foundation coincided with the decimation of Native communities via disease and targeted genocide. Black people were enslaved for two and a half centuries, enduring back-breaking work, torture, and psychological traumas (History 2021). Spanish speakers (who along with European blood, share indigenous and African roots) were regularly subjected to mob violence in the 19th and 20th centuries, joining the thousands of Blacks who were lynched during this time period (Blakemore 2017). While massacres of minorities are not as common now, the 21st century is far from being an egalitarian utopia.

To list all the forms of stress-inducing violence and discrimination that continue to be perpetrated against Native, Hispanic, and Black populations would be impossible. Although I mention other races in the subsequent section, I mainly focus on Black populations here. My narrow focus is not intended to dismiss the severity of the traumas experienced by other minority populations. I acknowledge that 2020 is historic not only because of the COVID-19 pandemic but because of the added attention the Black Lives Matter movement has brought to the public health crisis of systemic racism and police brutality.

With the exception of Asians, all minorities are more likely to die during encounters with law enforcement (Hansen 2017). Native Americans are killed at about three times the rate as Whites, a death rate that is followed closely by African Americans (Hansen 2017). This disparity is more pronounced for Black men; in fact, police use of force is one of the leading causes of death among Black men (Edwards et al. 2019). Still, the full extent of the effects of police brutality are unknown. As stated previously, science is always political. It has often been weaponized to uphold existing power structures, and police organizations are core enforcers of state power.

This complicates the investigation of police violence, a state-sanctioned use of force against civilians, regardless of the integrity of individual researchers. Accordingly, intergenerational transmission of police brutality trauma has not been well researched (Bryant-Davis et al. 2017), particularly within an epigenetic framework. Existing studies on the adverse health effects of police interactions rely on data collected from victims who may be disincentivized to report incidences for fear of the repercussions. This is justified fear given the history of retaliation and extensive cover-up attempts perpetrated by police officers and the government officials that protect them (Ralph 2020). Protection measures against brutalization often fall to potential victims themselves. Proposed remedies in the healthcare field even task children to educate themselves on how to diffuse police interactions and assert their rights (Maroney and Zuckerman 2018). This advice from the mouths of pediatricians is a testament to how large the barriers to systemic reform are in the eyes of citizens.

Although epigenetic studies into police brutality are lacking, it is reasonable to conclude that similar modifications to the epigenome occur in light of the wide range of traumas committed by police officers against minority citizens. At their worst, police officers have committed torture. As defined by the United Nations Office of the High Commissioner for Human Rights (1987), torture is "any act by which severe pain or suffering, whether physical or mental, is intentionally inflicted on a person". Although instances of police torture have occurred in cities ranging from New York to Los Angeles, the most extensive records come from Chicago (Kane and White 2013, Amnesty International 1992). The Illinois Torture Inquiry and Relief Commission was founded in response to hundreds of verified cases in which police beat, electrocuted, raped, suffocated, and burned Chicago citizens (Ralph 2020). While the majority of documented cases occurred between 1972 and 1991, the commission continues to receive three to five new cases a week (Ralph 2020).

These traumas may stretch into intense decades-long experiences under an era of mass incarceration. According to the NAACP, African Americans and Hispanics account for over half of the prison population despite only constituting 32% of the U.S. population. Although race relations are perceived as having improved over the past few decades, the prison population has ballooned since the 1970s, constituting a 700% increase. Prisons have been the sites of physical assault, sexual violence, and inadequate healthcare or neglect (Ford 2019, Pinto 2019, Wilper et al. 2009). A United Nations special rapporteur called for the ban of solitary confinement,

which is routinely used to discipline inmates, "considering the severe mental pain or suffering solitary confinement may cause, it can amount to torture" (United Nations 2011). A history of solitary confinement is strongly associated with PTSD symptoms (Hagan et al. 2018).

Beyond police torture and murder, Black individuals are subject to racial slurs, threats, and assaults by officers (Bryant-Davis et al. 2017). Staggers-Hakim (2016) links the stress elicited by media portrayals of police violence to negative impacts on mental health and hypervigilance in African American boys. These may seem like minor offenses in light of the previously mentioned brutalities. However, when they become everyday experiences, the cumulative stress can be extremely harmful because our bodies respond to different types of stressors using the same pathways. Ultimately, the perception of any threat, physical or psychological, results in the release of the same stress hormones that trigger the physiological stress response. Therefore chronic stress related to social factors, like one's placement in social hierarchies, is linked to a host of detrimental health effects including increases in morbidity and cardiovascular health risk factors (Ferrie et al. 2002, Spruill 2013). Minorities are more likely to be subject to these "everyday" stressors including police harassment, discrimination, and poverty. This has contributed to the development of the "weathering hypothesis" which describes detrimental health effects as results of repeated exposure to depressed socioeconomic conditions and political marginalization (Geronimus 1992). The ability for these racialized experiences of stress to manifest not only as visible mental health and metabolic conditions but as accompanying epigenetic markers is a testament to the longevity of the harmful impacts of racism.

Respiratory Disorders

The lungs are the primary site of exchange between the environment and our body. With each breath, we potentially absorb everything in our immediate environment including pathogens, carcinogens, and the oxygen needed for survival. Respiratory disorders are thus highly dependent on the environment, extending past the behavioral causes (such as smoking) that often occupy the stage when explaining poor minority health. SARS-CoV-2 primarily attacks the cardiovascular system, disrupting the body's ability to exchange and circulate oxygen throughout the body. Therefore respiratory disorders highly impact COVID-19 patient prognoses. The

severity of COVID-19 cases is largely characterized by the patient's respiratory function, in particular the inflammation and accumulation of fluid in the lungs that impairs breathing along with possible scarring (Cascella et al. 2021).

Accordingly, any pre-existing conditions that place additional strain on the patient's respiratory system heavily compromise the patient's ability to combat the virus. The most common respiratory conditions are asthma and chronic obstructive pulmonary disease (COPD), both of which are identified by the CDC as putting one at increased risk for severe illness (Centers for Disease Control and Prevention 2021). Asthma disproportionately affects Native Americans and Blacks (American Lung Association 2020). The data for COPD (which is an umbrella term for a variety of chronic lung diseases) incidence is more unclear. There is a notable gap in comprehensive population studies investigating racial/ethnic disparities, especially in Native populations, but existing studies indicate minorities may have slightly lower rates of COPD (Gilkes et al. 2016). Regardless, both disorders are subject to environmental factors. In specific, COPD is linked to exposure to irritants like ambient air pollution and fumes (KC et al. 2018). Asthma is linked to similar exposures and is also described as having a genetic component that is not well understood (National Institutes of Health 2020).

The genetic aspect of these disorders might be tied to epigenetic modifications associated with environmental factors that minorities are more likely to experience. As illuminated by the pandemic, minorities are disproportionately represented in essential jobs, especially in the sectors like agriculture (McNicholas and Poydock 2020). In addition to putting them at greater risk of direct exposure to SARS-CoV-2, employees are often exposed to workplace pollutants. Sustained exposure to these pollutants is lasting. The sperm of the grandsons of men exposed to vinclozolin, pesticides, plastics, dioxin, and jet fuel retain differentially methylated regions in their DNA (Manikkam et al. 2012).

Prominent biostatistician Dr. Melody Goodman says, "your zip code is a better predictor of your health than your genetic code" (Roeder 2014). Although workplace pollutants play a role in respiratory health, residential pollution has a higher impact on minorities. Communities of color are more likely to be disproportionately impacted by contaminated air, water, and soil (Mohai et al. 2010). Low income and minority communities live closer to Superfund sites, which are areas that have been contaminated by hazardous chemicals through mismanagement or dumping

(Environmental Protection Agency 2018), and industrial plants (Mohai et al. 2010). Blatantly discriminatory housing practices, like redlining, contribute to this co-occurrence of marginalized racial and economic status. Yet, race persists in being a significant predictor of exposure even when socioeconomic status is accounted for (Ash and Boyce 2018). Saha and Mohai argue that this relates to discrimination in siting decisions. In contrast to Black communities, White communities may be perceived by government and industry officials as more capable of fending off the placement of hazardous waste facilities. White communities are more likely to have the political connections and affluence to utilize legal resources and delaying strategies, leading siting attempts to fail and for businesses to seek the path of less resistance by targeting minority communities (Saha and Mohai 2005).

The environment may become internalized as predispositions to asthma and COPD through mechanisms ranging from changes to the body's microbial community to white blood cell expression (Brand et al. 2011, Malhotra and Vaarala 2017). A large and well-studied mechanism centers around inflammation, a staple of both asthma and COPD. Specifically, differential demethylation along with histone acetylation of pro-inflammatory genes is linked to upregulated expression of these genes and increased asthma and COPD severity (Kabesch and Adcock 2012). Chronic inflammation is linked to oxidative stress, which results from an imbalance of reactive oxygen molecules and the antioxidants that combat their damaging effects (Pizzino et al. 2017). When exposure to oxidizing pollutants becomes too high, epigenetic modifications to pro-inflammatory genes further exacerbate the inflammation associated with asthma and COPD (Kabesch and Adcock 2012). These epigenetic modifications can be triggered by residential and workplace pollutants such as diesel fuel and traffic-related air pollution (Cao et al. 2007, Ji et al. 2016). Thus, a possible cyclical relationship crystallizes in which greater severity of COPD-related or asthmatic inflammation triggers the formation of heritable epigenetic markers which beget even greater inflammation.

As with other respiratory disorders that obstruct airways, asthma and COPD are differentiated and diagnosed by spirometry: the measure of lung capacity and function (Rogliani et al. 2016). Since modern spirometers are nearly entirely mechanized, many are unaware they automatically adjust for race by using population-specific norms or scaling factors for non-White patients. These scaling factors are the most dramatic for Blacks, adjusting measurements 10-15% (Braun

2015). Studies proposing Black populations have decreased lung function due to their biology have deep historical roots. Thomas Jefferson noted "a difference in the structure of the pulmonary apparatus" to justify Blacks suitability for enslavement in the warm climate of the South (Braun 2015). Since then, studies spanning from the Civil War era to the modern day have found that African American populations have decreased lung capacity (Gould 1869, Kumar et al. 2010). Yet, when adjusted for socioeconomic status, racial differences in lung function and capacity shrink dramatically, showing that the environmental influences excluded from spirometry algorithms play a strong role (Hegewald and Crapo 2007).

Race is an inadequate proxy for biological difference. Explanations for decreased lung function among minorities that focus mainly on anthropometric measures, such as smaller trunk to leg ratios, fail to recognize the lasting influence of a racialized environment that can live on through epigenetic modifications (Hankinson et al. 1997). The continued automation of race correction in spirometry assumes innate racial difference while masking the social mechanisms behind these differences. For respiratory disorders like COPD or asthma, minorities have to be significantly sicker than their White counterparts to receive the same diagnoses and the associated benefits, like qualifying for worker's compensation. This exacerbates the existing problem of undiagnosed obstructive respiratory disorders. Approximately 60% of all cases go undiagnosed and minority populations are disproportionately represented in these undiagnosed cases (Martinez et al. 2015). Even beyond pulmonology, race-correction factors are ubiquitous, appearing in calculations ranging from kidney function to fetal growth rates (Braun et al. 2021). This widespread normalization of minority sickness that requires minorities to reach different thresholds for equal treatment is a barrier to equitable healthcare.

Conclusion

Minorities are disproportionately dying during this pandemic due to racialized social experiences tied to systemic racism such as housing discrimination, poverty, and chronic stress. Minorities are not dying because of an immovable and natural biological predisposition to disease. Yet, the generational nature of minority health disparities suggests that the human body embodies and passes down memories of this marginalization through epigenetic mechanisms. Ultimately, epigenetic research increases the urgency of social activists' calls to transform the revolutionary moments

of this year into lasting movements. The detrimental effects of both dramatic and subversive acts of racial marginalization assert that it is not enough that mass lynchings and genocides of minority populations are becoming less common. To truly achieve equitable health conditions for ourselves and the generations to come, we also need to eliminate the everyday environmental stressors that unrelentingly tax minorities.

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