LOYOLA UNIVERSITY CHICAGO

WHY IS RACE A RISK FACTOR FOR INFANTS BORN WITH BIRTH DEFECTS?:
DECONSTRUCTING THE BIOLOGICAL BASIS OF RACE IN MATERNAL-FETAL
MEDICINE THROUGH THE LENS OF REPRODUCTIVE JUSTICE

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—Rosalind Franklin
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ABSTRACT

Several studies have shown that marginalized populations, especially those of non-white race/ethnicity, have an increased risk of having infants born with severe birth defects. Existing hypotheses from the scientific literature on the topic of birth defects have primarily suggested that these trends may be the result of differential genetic susceptibilities within certain racial groups, a theory that reifies the (currently disproven) biological basis of race. Through this thesis, I argue that the myth of the biological basis of race continues to exist within maternal-fetal medicine today, where it is used to further the narrative that the bodies of women of color are hosts of degeneracy, as exemplified through the case study of increased rates of birth defects. I suggest that the assumption that disparities in birth defects are based on race-specific genetics turns our attention away from other explanations that are more likely to be the problem – explanations that are likely tied to systemic racism. This process perpetuates a cycle defined by white supremacy and reproductive injustice that works to deny women of color the right to have healthy offspring who are not affected by adverse environments and stressors that stem from systemic oppression. Ultimately, through this project, I hope to investigate how we can meaningfully acknowledge differences, including those differences that contribute to racial health disparities in the field of maternal-fetal health, in ways that do not reify the biological basis of race but instead work to affirm reproductive justice for our most marginalized populations.
CHAPTER ONE
INTRODUCTION

Over the past few decades, racial healthcare disparities have started to gain more attention in the sphere of medicine, with scientists and healthcare providers realizing that people of color face higher rates of mortality and morbidity of several diseases and disorders. As one example of this, several studies have shown that women of non-white race/ethnicity have an increased risk of having infants born with severe birth defects.\(^1\) In seeking to explain this disparity, hypotheses from the scientific literature on the topic of birth defects have suggested that these trends may be the result of differential genetic susceptibilities within certain racial groups, a theory that reifies the (currently disproven) biological basis of race. However, if we know that race is not a biological reality, why does current literature still indicate that race should be considered a risk factor for birth defects? In pursuit of this question, this thesis will investigate how the myth of the biological basis of race continues to exist within maternal-fetal medicine as a harmful concept that perpetuates inequity for women of color and their children.

Since its construction in modernity, the category of race has been used to justify sociopolitical hierarchies of oppression that name Black and Brown populations as lesser than their white counterparts. In many ways, this history of racism, at both systemic and individual levels, has relied upon the biological basis of race, a concept that has been disproven in the

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\(^1\) The CDC (2020b) defines birth defects as “structural changes present at birth that can affect almost any part or parts of the body (e.g., heart, brain, foot)” (para. 3). They vary widely in severity, in outcome, and in effect upon the neonate. While some prefer the language of congenital abnormalities, congenital disabilities, or other terminology, I have chosen to use the term ‘birth defect’ throughout this project because of its standardized use within the scientific literature.
modern day, to make a case for the inferiority of people of color. The concept of biological race (falsely) argues that “there are naturally occurring hierarchies of humans determined by brain size, genes, morphology, phenotype, or pigmentation” that can be visualized by grouping humans into definitive racial categories (Isoke, 2016, p. 748) These racial categories have manifested in a variety of ways, with people being grouped according to skull size, skin color, facial features, voice, and even genetic variants. Noticing the differences or variations that exist from individual to individual or even population to population – such as patterns of skin color – is not what makes the biological basis of race so harmful, though. The problem is that biological race essentializes an entire community of people – who are not homogenous in any way – into one unified group who are assumed to share similar traits due to inherent, natural, and unchangeable aspects of their biology. These traits (as well as their associated populations) are then hierarchicalized to differentially oppress and privilege certain groups, all the while using the institutionalized logic of biological race to justify this injustice.

In recent decades, the scientific logic behind the biological basis of race has been particularly dependent on studies of genetics. Proponents of the scientific nature of race have argued that there exist specific genetic variants or alleles that can only be found in certain populations, populations that coincide with the racial categories (Sussman, 2014). They have claimed that these variants drive phenotypes that are definitively linked to one’s racial classification (such as skin color, hair texture, and/or facial features) and are so immutable that each race can be categorized as biologically distinct from another – to the point where the different races can be conceptualized as different subspecies or species altogether (Angier, 2000).
Today, these arguments have been rightfully discredited, with the literature coalescing around the fact that all humans today are one biological species (Angier, 2000). Much of this has been the result of studies demonstrating that there exists greater overall genetic diversity within constructed racial populations rather than between them (Fairbanks, 2015). A 2004 meta-analysis conducted by Lynn Jorde and Stephen Wooding confirmed this, finding that out of the 0.1% of human DNA that varies from individual to individual, 85-90% of genetic variants are found within all groups, and only 10-15% are unique to certain continental populations (Fairbanks, 2015). From this data, Jorde and Wooding concluded that “these estimates…tell us that humans vary only slightly at the DNA level and that only a small proportion of this variation separates continental populations” (Fairbanks, 2015, p. 23).

The small percentage of population-based variation that does exist is the result of human evolutionary patterns from hundreds of thousands of years ago. Race has been used as a proxy for these population-level patterns of ancestral genetics, meaning that the racial categories we use today are failed attempts to classify individuals according to the true source of some of their genetic diversity as per migration patterns out of Africa more than 100,000 years ago (Angier, 2000). All of this evidence indicates that race cannot be used as a category to group people together on the basis of inherent, defining biological characteristics, despite what outward appearances may suggest to a lay onlooker.

While the creation of categories that are based on a false understanding of biology is problematic in and of itself, it is the hierarchicalization of these categories – the assignment of ‘desirable’ and ‘undesirable’ social traits to racialized human phenotypes – that has been used to serve the logic of white supremacy. Western society has rationalized this ‘logic’ as value-neutral, objective, and legitimate by constructing race as a biological concept through the field of science
(Grigg & Kirkland, 2016). Robert Sussman, an American anthropologist, has summarized the current beliefs about racial categories while eloquently disputing the biological basis of race at the same time. Essentially, according to Sussman (2014):

> There are no major complex behaviors that directly correlate with what might be considered human “racial” characteristics. There is no inherent relationship between intelligence, law-abidingness, or economic practices and race, just as there is no relationship between nose size, height, blood group, or skin color and any set of complex human behaviors. However, over the past 500 years, we have been taught by an informal, mutually reinforcing consortium of intellectuals, politicians, statesmen, business and economic leaders, and their books that human racial biology is real and that certain races are biologically better than others. These teachings have led to major injustices to Jews and non-Christians during the Spanish Inquisition; to blacks, Native Americans, and others during colonial times; to African Americans during slavery and reconstruction; to Jews and other Europeans during the reign of the Nazis in Germany; and to groups from Latin America and the Middle East, among others, during modern political times. (p. 2)

As Sussman has explained, while biological race is not real (in the sense that it is not factually accurate), its consequences have been and continue to be extremely harmful for those who have been labeled as ‘other’ by the dominant (and often white) society.

From an intersectional lens, we can recognize that the adverse effects of the institutionalization of the biological basis of race throughout history have been most profound for women of color, who face the ‘double bind’ of race and gender.² When it comes to the biological basis of race, the interaction of gender-based oppression and race-based oppression has painted women of color as “both biological and social reproducers” of race and its assigned ‘degeneracy’ (Roberts, 2014, p. 21). In her book *Killing the Black Body*, Dorothy Roberts (2014) has focused on how this assigned ‘degeneracy,’ specifically with regards to Black women, has resulted in a white supremacist agenda to control Black women’s reproduction. In fact, Roberts has argued that reproductive control has been a key tenet of racial oppression, especially for the

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² The idea of the ‘double bind’ – or double discrimination – of race and gender comes from Kimberlé Crenshaw, who coined the term ‘intersectionality’ in 1989 (Crenshaw, 1989).
Black woman, from the early origins of the United States. She has explained this further, writing that:

The systematic, institutionalized denial of reproductive freedom has uniquely marked Black women’s history in America. Considering this history—from slave masters’ economic stake in bonded women’s fertility to the racist strains of early birth control policy to sterilization abuse of Black women during the 1960s and 1970s to the current campaign to inject Norplant and Depo-Provera in the arms of Black teenagers and welfare mothers—paints a powerful picture of the link between race and reproductive freedom in America. (Roberts, 2014, p. 14)

On an individual level, the intertwined nature of racism and reproductive control has undermined the right to choice that Black women should have over their bodies and their lives when it comes to whether to not to reproduce, whether or not to be a mother, how they choose to mother, etc. On a systemic level, this has contributed to the dehumanization of the Black woman and her offspring, to the point where we see both the Black mother and child as the source of all social problems. This stereotype subsequently ignores the marginalization that Black bodies have faced in the United States and instead blames them for their own oppression. Thus, “not only do these policies injure individual Black women, but they also are a principal means of justifying the perpetuation of a racist social structure” (Roberts, 2014, p. 16). Ultimately, this means that, in accordance with Roberts’ analysis, when problematizing the biological basis of race, it is necessary to emphasize the damaging effects that this ideology has had on the reproductive freedom of Black women and, more broadly, all women of color.

Unfortunately, the biological basis of race is not a relic of the past, nor is the connection between racial oppression and reproductive control. Today, the biological basis of race continues to be reified in many spheres of existence, but especially in the practice of race-based medicine, which relies on using socially constructed racial categories to better ‘understand’ an individual’s biological makeup, especially their genetic variation (Roberts, 2011). Under race-based
medicine, patients of color are asked to either self-declare their race according to predetermined categorical boxes or have their race assumed by their provider based on phenotypic racial stereotypes (such as skin color, voice, etc.). The patient’s assigned race contributes to how a provider will diagnosis them, what tests they will use and how they will analyze those tests, and what course of treatment will be used. This differential care is not only the result of implicit bias (though that can contribute to the problem) but actually written into the rules of medicine. In all of these ways, race-based medicine relies on the rationale that people of different races need different healthcare because they are profoundly biologically different from one another.

While ancestral genetic variation is something that does have clinical implications, as previously mentioned, race is not a valid proxy for these population-level patterns. For this reason, race-based medicine ultimately becomes a reification of the biological basis of race because the genetic, molecular, and even phenotypic variants that providers are using to

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3 As one example of how diagnosis is influenced by assigned race, many healthcare providers still believe the myth that African-Americans can’t get psoriasis. In actuality, a number of African-Americans do have psoriasis, though diagnosis is often impaired because of how the disease manifests on darker skin tones than it does on lighter ones (HealthDay News, 2018).

4 Assigned race also influences how medical tests are analyzed, which affects both diagnosis and treatment. One example of this is the estimated glomerular filtration rate (eGFR) which includes a correction factor to be used in Black patients. The correction factor is based on the idea that “black individuals release more creatine into the blood, perhaps because of more muscle mass” though there is debate as to the accuracy of this statement (Eneanya et al., 2019, p. 113). Even if this statement is accurate in some Black individuals, the use of such a correction factor encourages healthcare providers to believe race is a homogenous category with essentialized biological components.

5 Perhaps the most famous example of race-specific treatment is BiDil, a heart drug that was FDA approved exclusively for use in African-Americans. While “the clinical trial data [for BiDil] failed to show sufficient statistical power of the drug’s efficacy for a multiracial population with heart disease,” the scientists behind the study claimed it did meet these standards in a trial exclusively made up of self-identified African-Americans (Krimsky, 2012, para. 2). However, the creators of BiDil failed to demonstrate exactly why this was, thus relying on the inaccurate biological basis of race to justify its use (or misuse) in Black populations.

6 To clarify, the idea of differential care is not necessarily the problem here. It is the fact that differential care is being provided based on an inaccurate rationale. Differential care, including differential care on the basis of race, has the potential to be useful and helpful, such as when alleviating health disparities, but it must be done for the right reasons and in the right way to actually provide better care for marginalized populations.
determine a patient’s course of care are not necessarily true to that individual’s actual biology but rather assumed to be part of their biology based on the racial category they have been assigned. Therefore, race is acting as a scientific variable that homogenizes people’s genes, phenotypes, and potentially even their social characteristics under the guise of providing ‘race aware’ care. Rather than supporting people of color, though, this phenomenon only validates the disproven idea that race is a natural, inherent, and ‘scientific’ category that carries biological meaning – meaning that has been wrongfully used to rank people of color as fundamentally inferior to their white counterparts.

Race-based medicine has infiltrated the healthcare field widely, but it has some of the most visible impacts in the field of maternal-fetal medicine, where the biological basis of race is being used as an explanation for reproductive health disparities in inaccurate, harmful ways. Health disparities that affect people of color are being traced back to the ‘bad genetics’ of people of color, particularly women of color, who are faulted for passing on ‘defective’ genes to their offspring. This is no different than the history that Roberts (2014) has described, in which Black mothers were thought to “pass down to their offspring the [physical and moral] traits that marked them as inferior to any white person” (p. 20). The field of medicine has perhaps moved past the explicit idea that Black bodies pass on moral inferiority to their offspring, but it has not stopped suggesting that they are responsible for passing on and reproducing physical inferiority. Women of color are still considered the bearers of degeneracy, but, in medicine, this has shifted from an emphasis on them being the “bearers of ‘incurable immorality’” to being the bearers of incurable disease (Roberts, 2014, p. 20).

These ideas are exemplified prominently in the case of birth defects among women of color. Several studies have shown that marginalized populations, especially those of non-white
race/ethnicity, have an increased risk of having infants born with birth defects. For example, an analysis by the Centers for Disease Control and Prevention [CDC] (2018) of a study by Canfield et al. has stated that, as compared to non-Hispanic white populations, non-Hispanic Native American populations had much higher occurrences of encephalocele,\(^7\) anotia/microtia,\(^8\) cleft lip,\(^9\) limb deficiency,\(^{10}\) and trisomy 18;\(^{11}\) non-Hispanic Black populations had higher occurrences of encephalocele and trisomy 18; and Hispanic populations had higher occurrences of anencephaly,\(^{12}\) encephalocele, and anotia/microtia.

This disparity is especially concerning because birth defects account for 20% of all infant deaths, making it the leading cause of infant mortality in the U.S. (CDC, 2020a). Although the evidence demonstrates the existence of specific trends regarding birth defects, very little research has been done to thoroughly investigate why these trends might be occurring. When analyzing the data, scientific researchers have predominantly left it upon future research to determine why these trends exist. However, current hypotheses that do exist within the scientific literature have suggested that these trends may be the result of differential “genetic susceptibilities” within certain racial groups (Egbe, 2015, p. 183; Ibrahim et al., 2014, col. 2). However, there is very little to no evidence demonstrating that non-white populations have inherent genetic differences that would result in an increased risk for birth defects at the scale at which we see the current

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\(^7\) Encephalocele is a neural tube birth defect that results in sac-like protrusions on the head of a neonate.

\(^8\) Anotia and microtia are two birth defects of the external ear.

\(^9\) Cleft lip is a birth defect that is characterized by an opening in the upper lip of the mouth.

\(^10\) Limb deficiency is a syndrome that describes any sort of maldevelopment of the limbs, ranging from a complete absence of the upper and lower limbs to the absence of a digit on one of the limbs.

\(^11\) Also known as Edward’s syndrome, Trisomy 18 is a condition that results from the presence of an extra chromosome 18. Neonates with Trisomy 18 are often born small and/or with heart defects.

\(^12\) Anencephaly is a neural tube birth defect that results in the absence of a major portion of the brain or skull.
trend emerging. Because healthcare providers are not using a scientifically and socially sound rationale in analyzing these trends, they characterize reproductive health disparities as problems that are ‘natural’ to the bodies of people of color, subsequently naming race-based disparities as problems that are unchangeable and biologically predetermined. This undermines the necessity of intervention on behalf of the medical field and allows the disparity to be continuously ignored, subsequently perpetuating systemic inequity within the medical field and in society more broadly.

By perpetuating the ignorance of health disparities within the field of maternal-fetal health, the concept of biological race is responsible for allowing not only inaccurate, negligent healthcare but reproductively unjust healthcare. According to Ross et al. (2017), reproductive justice is defined as:

a theoretical paradigm shift and a model for activist organizing centering three interconnected human rights values: the right not to have children using safe birth control, abortion, or abstinence; the right to have children under the conditions we choose; and the right to parent the children we have in safe and healthy environments. (p. 14)

In the case of increased rates of infants with birth defects among women of color, simply labeling reproductive disparities as a problem resulting from racially-specific defective genes prevents an investigation into factors such as environmental injustice, lack of access to nutritious food, lack of access to quality healthcare, etc. – all of which stem from the intertwined effects of systematic race-based, class-based, and gender-based oppression. Thus, rather than looking at changeable social and environmental factors that can and should be addressed by healthcare providers, the biological basis of race serves as an excuse that prevents women of color from
being able to have healthy\textsuperscript{13} children (i.e., children who are not affected by factors that we know cause infant mortality and morbidity).

While the existing body of literature has focused predominantly on the types of birth defects that certain populations are facing, I believe there is a need to investigate whether the increased rates in birth defects among marginalized populations could actually be the result of social and environmental causes in order to identify the real driving factors behind this disparity, rather than blaming it on an outdated, false understanding of race. However, before this can be done, it is necessary to first establish the theoretical context behind this problem so as to understand how the reification of the biological basis of race is taking place within maternal-fetal health and why it is a problem for women of color’s healthcare today. As such, through this thesis, I argue that the idea of the biological basis of race continues to exist within maternal-fetal medicine today, where it is used to further the narrative that the bodies of women of color are hosts of degeneracy. This problem is manifested within the existing scientific analysis of trends of birth defects among women of color, where the ‘bad genes’ of women of color are being blamed for (re)producing ‘defectiveness’\textsuperscript{14} among their infants in order to account for this health disparity. This analysis falsely ignores the real root of the problem, which is likely directly tied to systemic racism, and instead places the blame upon women of color. This process perpetuates a cycle defined by white supremacy and reproductive injustice that works to deny women of

\textsuperscript{13} It is important to investigate the idea of ‘health,’ not only from a race, gender, and class perspective but also through the lens of disability studies. Conceptions of health have often excluded those who are disabled, which is an injustice that needs to be problematized, deconstructed, and further addressed, including in medicine. However, at this time, a full investigation of this idea is beyond the scope of this thesis.

\textsuperscript{14} Defectiveness as well as degeneracy have often been code words for disability. Further research should deconstruct this connection through the lens of disability studies as part of problematizing of the biological basis of race and its impacts upon reproductive autonomy.
color the right to have healthy offspring who are not affected by adverse environments and stressors that stem from the realities of systemic racism.

In pursuit of these ideas, chapter two will investigate the historical construction of the concept of biological race, particularly within the United States. The chapter begins by tracing the origins of the biological basis of race among European scientists in the 1600s. The chapter then discusses the formation of the American School of Ethnology, which propagated ideas of biodeterminism within U.S. scientific and social spheres, especially in the 19th and 20th centuries. From these early conceptions of biological race came the full-fledged ideological movements of Darwinism, social Darwinism, and eugenics, all of which have had lasting implications for people of color in the U.S. The chapter ends by discussing how women of color became targets of race-based biology, where they were conceptualized as carriers of genetic degeneracy, especially through the interactions of scientific movements and women’s rights movements in the late 19th and early 20th century.

Building on this history, chapter three focuses on how the biological basis of race continues to be reified in maternal-fetal medicine, as demonstrated through a review of the existing scientific literature on the prevalence of birth defects among racial/ethnic minorities. The chapter focuses on detailing how these explanations of racial health disparities are reliant on the biological basis of race and how they ultimately cause inaccurate and reproductively unjust healthcare for women of color and their children.

Noting the problems of using race as a risk factor for the health disparities like the increased rates of birth defects seen among infants born to women of color, chapter four focuses on how a reproductive justice framework can be used to reconceptualize racial health disparities, especially those related to adverse pregnancy outcomes. By centering women of color,
embracing intersectionality, and meaningfully addressing difference, a reproductive justice framework mandates that we deconstruct and dismantle the remnants of biological race within healthcare in order to promote more equitable care to our most marginalized populations.

To conclude, chapter five summarizes the key arguments of the thesis and points towards how medicine exists as an institution of bio-power that has normalized race as a legitimate biological category. Specifically, the chapter focuses on how deconstructing the harmful effects of the biological basis of race demands that we not only utilize a reproductive justice lens to see and solve existing problems within maternal-fetal medicine but that we rethink the very foundations of how we care for the health of our most marginalized populations.
CHAPTER TWO

A BRIEF HISTORY OF THE BIOLOGICAL BASIS OF RACE

In order to understand how and why the biological basis of race is pervasive within maternal-fetal medicine today, it is important to first situate the theory of biological race within a historical context. As such, this chapter will investigate the following question: how has the biological basis of race emerged in history as a ‘valid’ scientific theory that has influenced women of color’s reproduction? In pursuit of this question, this chapter is divided into three sections: origins, mutations, and evolutions. The first section discusses the origins of the biological basis of race within the scientific community, starting with European scientists in the Renaissance and Enlightenment eras and then moving into the work of American scientists in the 19th century. The second section, which begins in the mid-1800s, demonstrates how the belief in the biological basis of race mutated into the social Darwinism and eugenics movements, both of which applied evolutionary theory to human populations in order to ‘understand’ sociopolitical inequity. The third section depicts how U.S. scientific racism movements evolved as the result of interactions with white feminists from the 1870s onwards to enact reproductive control, particularly among women of color. Ultimately, through this historical examination, I hope to uncover how scientific racial constructs have fueled reproductive injustice in the United States, particularly when thinking about who is allowed to birth healthy babies.

Origins: The Scientific Beginnings of Biological Race

Human variation, including variation along the lines of skin color, facial morphology,
and other phenotypic characteristics, has likely been philosophized about since the birth of humankind. However, the first recorded use of the term ‘race’ to refer to distinctive human categories in western scholarly writings is attributed to François Bernier (1620-1688), a French physician and traveler (Bindon, 2012b). Bernier notably embarked on a 12-year journey throughout Africa, South Asia, and the Middle East, during which he recorded the presence of four species, or races, for the first time.

The first race, ‘the Europeans,’ consisted of populations from Europe, Northern Africa, Persia, India, and/or the Americas and were predominantly defined by their light skin (Bindon, 2012b). Interestingly, Bernier still considered those with slightly coppery or tan skin tones, such as those from Egypt or India, as European because their “colour [was] only an accident in them…because they [were] constantly exposed to the sun” (Bindon, 2012b, slide 16). Bernier’s conception of race, which was much broader than what would be seen in later years, implied that there was something inherently ‘purer’ about light skin tones, including ones that had been slightly ‘corrupted’ by accidental exposure to environmental conditions. Skin color, therefore, was more than just an external, visual trait but something indicative of the nature of the inner being.

This is seen in Bernier’s second racial category as well, which he defined as those populations living in the rest of Africa (outside of Egypt). The ‘Africans,’ as he named the second category, were defined by their “thick lips,” “squab noses, “wool-like” hair, and “blackness of skin which is not caused by the sun” (Bindon, 2012b, slide 17). Here, the “blackness of skin” that Bernier describes is not influenced by the outside environment; rather, this “blackness” is something intrinsic to the being, which later scholars would associate with being biological in nature (Bindon, 2012b, slide 17).
Bernier’s third race, which he named as ‘some Asians,’ was made up of those living in Japan, China, Georgia, and Muscovy who shared the following characteristics: “broad shoulders, a flat face, a small squab nose, [and] little pig’s-eyes long and deep-set” (Bindon, 2012b, slide 18). The fourth race, ‘the Lapps,’ were described by Bernier as “little stunted creatures with thick legs, large shoulders, short neck, and a face elongated immensely” (Bindon, 2012b, slide 19). Obviously, all of these descriptions are crude, incorrect, and racist stereotypes. That being said, they demonstrate how the conceptualization of the ‘other’ has been rooted in biological notions of difference in the western world since at least the 17th century. More specifically, they reveal how differences in the outward appearances of individuals were homogenized and then applied to entire populations in order to create categories based on phenotypic appearance. These constructed categories also showcase the beginning of phenotypic appearance being misinterpreted as a representation of the inner being, which later would manifest as a complete conflation of skin color, biological traits, and character traits in scientific thought.

Bernier’s four races were based on the notion of differential worth, a concept that argued that the races could be hierarchicalized based on their scientific classification. In the 18th century, George-Louis Clérel, Comte de Buffon (1707-1788) expanded on the idea of differential worth in the 44 volumes\(^1\) of his encyclopedia collection *Natural History*\(^2\) that were published between 1749 and 1804 (Bindon, 2012b). In his work, Buffon divided the human species into six racial categories: Laplanders or Polar People, Tartars or Mongolians, Southern Asiatics, Europeans, Ethiopians, and Malays. This categorization was based on a “devolutionary theory”

\(^{1}\) 36 of these volumes were written and published within Buffon’s lifetime, while 8 additional volumes were finished and published posthumously by his colleagues (Hoquet, 2010).

\(^{2}\) The original title of Buffon’s work is *Histoire Naturelle, générale et particulière, avec la description du Cabinet du Roi*, which, in English, translates to *Natural History, General and Particular, with a Description of the King’s Cabinet*. 
that argued that humans “over time fell off by degrees from their originally perfect state” (Curran, n.d., para. 5). Of course, according to Buffon, the original perfect state was the European (white) Adam and Eve, from which all other races ‘degenerated’ due to environmental conditions, such as temperature, sunlight, food access, etc. (Harris, 2001, pp. 83-85). While Bernier did not believe that ‘accidental exposure’ or environmental conditions led to the inferiority of a particular group, Buffon redefined the role of genetic-environment interactions. Under Buffalo’s devolutionary theory, any being or group that experienced changes in skin color due to environmental conditions was labeled as less evolved or ‘degenerate’ By naming environment as a contributor to devolutionary theory, Buffon was able to rank the devolutions of humankind based on how much the environment had changed them: the white man devolved into the Brown man who then devolved into the Black man. The darkness of their skin indicated the level by which they had fallen from the ‘original perfect state,’ ultimately causing those with Black skin color to be ranked as the most inferior ‘type’ of human.

While Bernier and Buffon used skin color to construct biological categories along the lines of race, their successors began to study other anatomical parts as well, a process that resulted in the racialization of the entire physical body. For example, Johann Friedrich Blumenbach (1752-1840), a professor of medicine and a natural historian, published his dissertation “On the Varieties of Mankind” in 1775, in which he argued that there were five ‘varieties’ of human beings based on skull shape: Mongolian, American, Caucasian, Malay, and Ethiopian (Bindon, 2012a). He devised these categories based on his ‘extensive’ measurements of the human skull, for which he was named the father of craniology (Bindon, 2012a). Blumenbach’s research expanded the biological categories of race to not only include external phenotypes but internal phenotypes as well, namely the size and shape of the skull. The skull is
one of the most intimate parts of the body, connected to the entity that gives us our intelligence, our personality, and potentially even our humanness: the brain. Thus, in the following years, Blumenbach’s work connecting the skull and the brain to the concept of biological race would quickly allow for more expansive characteristics of ‘degeneracy’ to be associated with Black and Brown bodies.

Bernier, Buffon, and Blumenbach were all European scientists. However, by the 19th century, American scientists had also become involved in conversations on the biological basis of race, conversations that would form the foundation of the American School of Ethnology (Smith, 2014). Samuel George Morton (1799-1851), a Pennsylvanian physician and disciple of Blumenbach, became one of the primary founders of the American School and conducted his own craniological studies to investigate biological race (Smith, 2014). Fascinated with Blumenbach’s theory of the five races, Morton collected (or more accurately, stole) hundreds upon hundreds of skulls from various geographic origins of the world, upon which he performed numerous ‘experiments’ (Renschler & Monge, 2008). Morton’s most famous publication was perhaps the *Crania Americana, or a Comparative View of the Skulls of Various Aboriginal Nations of North and South America* (1839), in which he characterized the skulls and the phenotypic characteristics of Blumenbach’s five races (Renschler & Monge, 2008). Like Blumenbach’s work, Morton’s cranial measurements claimed to demonstrate significant differences in size and capacity by race; however, Morton extrapolated this data to be indicative of differing levels of intelligence amongst the races, which he saw as validation for the differential value of the races through the ‘science’ of craniology (Renschler & Monge, 2008).

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3 In later years, Morton’s measurements of the skulls themselves were actually found to be completely unreliable, and even potentially falsified, though in either case his analysis was fully reliant on racist understandings of the body (Menand, 2001).
While Morton’s predecessors had characterized the non-white races as inferior, Morton was one of the first scientists to make an explicit connection between non-whiteness and inferior intelligence.

The repercussions of Morton’s ‘discovery’ were devastating for people of color, as decreased intelligence (an idea that was false in the first place) begin to be (mis)applied as indicative of decreased civility or even decreased humanity. Morton began to apply these ‘scientific’ theories to the sociopolitical sphere in his 1844 publication, *Crania Aegyptiaca, or Observations on Egyptian Ethnography*. After obtaining and analyzing an extensive collection of skulls from Egypt, which were his most ancient samples yet, Morton claimed that the “distinct racial differences shown in modern ‘Caucasoid’ and ‘Negroid’ crania were equally discrete in the past” as they were in the present (Renschler & Monge, 2008, p. 34). Essentially, he argued that this was evidence for the devolutionary theory being applicable not only to the recent past but actually from the beginning of human history itself. By demonstrating that racial differences in craniology, and subsequently in intelligence, existed even in ancient history, Morton used historical precedence to naturalize the ‘degeneracy’ of the non-white races.

Alongside differential skull measurements and their corresponding intelligence levels among the races, Morton claimed that his ‘evidence’ also showed that those belonging to the ruling class of ancient Egypt had been Caucasians, while those of the slave class were ‘Negroids’ (Renschler & Monge, 2008). Thus, he argued that the hierarchies of antiquity provided evidence for a natural order based on race (and race-based intelligence) that justified enslavement of Black bodies by white owners, including the enslavement of Black bodies in the United States (Renschler & Monge, 2008). This was one of the first times that the American School had directly used biological race to answer the ‘question’ of slavery, an ethical dilemma that had
been plaguing the American public at the time. By using craniology to justify slavery, Morton’s work began to more clearly connect scientific ideology and social oppression in the United States, specifically by characterizing people of color as fundamentally unintelligent and potentially even as non-human beings.

Now that people of color had been ‘proven’ to be biologically inferior, could this inferiority be passed on? This was the question that Josiah Clark Nott (1804-1873) sought to address through his discussions on intermarriage between Black and white individuals (Smith, 2014). Nott, a physician who first became noteworthy for his research on Yellow Fever in the American South, published an article in 1843, entitled “The Mulatto a Hybrid – Probable Extermination of the Two Races if Whites and Blacks Are Allowed to Intermarry,” in the New England Journal of Medicine that established him as part of the American School of Ethnology (Smith, 2014). In this piece, Nott utilized the biological species concept, which defines a species as a “group of organisms that can successfully interbreed and produce fertile offspring,” to argue against inter-race relations (Ereshefsky, 2007, p. 412). Nott’s main argument was that “mulatto children were inter-species hybrids and would ultimately prove to be infertile because their parents were not of the same species” (Smith, 2014, p. 43). Even if these offspring were not infertile in the first generation of ‘inbreeding,’ he claimed that they would be “less capable of endurance and shorter lived” and “that mulatto women were more delicate and less able to bear children” (Smith, 2014, p. 43) Nott’s claims were based solely on his own observations and his own “veracity” (Smith, 2014, p. 43). He had no distinctive evidence when making these claims, and later scientists would come to recognize his work as inherently false and racist.

Nott’s article was considered revolutionary because he had ‘proved’ that the different races were not only devolutions one of another, but entirely different species – a concept that
only further validated the nonhumanity of people of color. He also had addressed the question of reproduction between the (white) human and now (non-white) ‘non-human,’ arguing that any relations resulting in these racial ‘hybrids’ were wrong, not only from a moral lens but from a scientific perspective as well. It was ‘unnatural’ and perhaps even threatening, from Nott’s evolutionary perspective, for the offspring to be born as infertile, something that Nott assured would happen either sooner or later as the result of ‘interbreeding’ between Black and white individuals in the U.S. Ultimately, by combining race-based biology with questions of reproduction, Nott laid the foundation for later eugenicists who would argue that the biological basis of race – and its associated ‘degeneracy’ – demanded an imperative to control who had the right to reproduce.

It is also important to note that for Nott, the offspring was never a point of concern. Though Nott’s idea that the offspring between a Black and a white individual would result in an infertile offspring was not factually correct, he operated under the assumption it was. Despite this, Nott never theorized about if or how the offspring needed to be cared for if they were affected by infertility. It was as if the offspring inherited the supposed ‘inhuman-ness’ of its Black parent and subsequently didn’t deserve further care or consideration. Reproductive limitation was the solution for Nott, but if by chance a ‘degenerate’ offspring was born, under Nott’s view, they were invisible or ignored – even if they needed medical help or care. Nott’s work was the culmination of years of scientific racism, of years of the biological basis of race being reified within the scientific scholarship. What began as an examination of differences in skin tones transformed into a value-based hierarchicalization of populations based on differential skin color; into a conceptualization of race as a wholly biological concept, representative of internal and external elements; into the idea that non-whiteness was associated with inferior
intelligence and even nonhumanness; *into* an imperative to control reproduction to limit the birth of ‘unnatural’ offspring.

While the biological basis of race was being popularized within the scientific sphere, public opinion in American society was more divided on these race-based theories, at least until Louis Agassiz (1807-1873), a Swiss-born scientist, immigrated to the United States in 1846. Agassiz’s reputation as an expert natural scientist who valued factual “observation over speculation” preceded him, and he even became known as “Harvard's most prominent professor” by 1850 (Strain, 2003). In 1850, at the Third Annual Meeting of the American Association of Science, Agassiz made a public and authoritative statement in which he asserted that “zoologically, the several races of men were well marked and distinct” (Strain, 2003). When Agassiz explicitly recognized the Black and white races as different species in this statement, the public took his word for it, causing public opinion as a whole to shift towards a firm acceptance of the biological basis of race. In fact, Agassiz’s speech was so influential that, following the meeting, Nott wrote a letter to Morton in which he famously stated that, “with Agassiz in the war the battle is ours” (Strain, 2003).

It is thought that Agassiz’s need to differentiate the races stemmed from a fear of ‘race-mixing,’ a concept that Nott also focused on in his scholarly work. Building on Nott’s idea of the infertile offspring, Agassiz thought that the offspring of an interracial couple would result in a ‘degenerate’ half-breed. This pattern of thought is reflected in a quotation from Louis Agassiz in an annotated version of his wife’s diary, where he wrote that:

> The natural result of an uninterrupted contact of half-breeds with one another is a class of men in which pure type fades away as completely as do all the good qualities, physical and moral, of the primitive races, engendering a mongrel crowd as repulsive as the mongrel dogs, which are apt to be their companions, and among it which is impossible to pick out a single specimen retaining the intelligence, the nobility, or the affectionateness
of nature which makes the dog of pure type the favorite companion of civilized man.
(Agassiz & Agassiz, 2020, pp. 298-299)

This statement moved beyond Nott’s dismissal of the infertile offspring and replaced it with a
dehumanizing picture of the offspring as a dangerous threat. Through this statement,
reproduction became further racialized so that the offspring of people of color were
conceptualized as posing a direct threat to the good, pure, and ‘civilized’ (read: white) society.
Moreover, because of Agassiz’s reputation, these ideas were being popularized at unprecedented
levels. Non-whiteness was constructed as a biological threat, and in fear of this threat, Agassiz
succeeded in convincing the American public that people of color needed to be viewed as
sources of degeneracy.

Nott, Agassiz, and peer Egyptologist, George Glidden (1809-1857) built on the growing
public attention around the biological basis of race by leading the charge on the 1855 publication
of *Types of Mankind: Or, Ethnological Researches* – a comprehensive, 700-page work that
outlined the central beliefs of the American School on the subject of race up until that point
(Smith, 2014). The book, which was an amalgamation of the theories of the time, was considered
revolutionary, as it was “the first time that scientists pulled together all of the research that
justified the argument that African Americans, Native Americans, Asians, et cetera were
different species” (Strain, 2003). Moreover, it explicitly laid out why the different races had
differential values in society. Through its ‘scientific’ evidence, *Types of Mankind* claimed that:

> Nations and races, like individuals have each an especial destiny: some are born to rule,
and others to be ruled. And such has ever been the history of mankind. No two distinctly
marked races can dwell together on equal terms. (Nott et al., 1855, p. 79)

Ultimately, *Types of Mankind* naturalized inequity, specifically inequity on the basis of race, in
ways that hadn’t been seen before. It argued that racial injustice was ‘inevitable,’ and as such
unchangeable, because of the biological inferiority of certain races. The book became a best-seller, popularized by all sides of the American public as well as government leaders such as the Secretaries for the Treasury, Navy Department, and State Departments (Smith, 2014). In fact, scholars have suggested that “Types of Mankind, for all its scientific and factual failings, was also the book that fixed the idea of ‘race’ in the consciousness of the American reading public” (Smith, 2014, p. 12).

Following the publication of the Types of Mankind, the public was on the side of science, allowing the biological basis of race to be institutionalized within the U.S. as an indisputable truth. People of color were the most negatively affected by this ideology, as non-whiteness had become associated with undesirable physical and character traits that could be passed on directly from one generation to the next, subsequently tainting a ‘civilized’ society wherever it existed. Thus, with the biological threat of non-whiteness fully established in society by the 1850s, the next question would be how to control it.

**Mutations: From Darwinism to Social Darwinism to Eugenics**

While the American School of Ethnology had established the basic principles of biological race both within the spheres of science and public opinion, the scientific racism movement was popularized at unprecedented levels in American society following the publication of Charles Darwin’s *The Origin of Species* in 1859. In this groundbreaking text, which is still considered foundational in the field of Evolutionary Biology, Darwin (1809-1882)

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4 Alongside the institution of science, white supremacist Christianity also played a role in shifting public opinion about biological race. Essentially, Christian theologians/preachers were also complicit in establishing the (biological) degeneracy of people of color, particularly through natural law arguments. Delving into this history further, Terence Keel (2012) has comprehensively traced the way in which Christianity has “shaped modern scientific perceptions of race” (iii).
concluded that species could evolve through natural selection,\(^5\) which he defined as the “preservation of favourable variations and the rejection of injurious variations” (Darwin, 2011, p. 35). Darwin explained that natural selection could be seen in all aspects of the natural world, which caused organisms to be in a constant state of competition, or what he called the “consequences of one general law, leading to the advancement of all organic beings, namely, multiply, vary, let the strongest live and the weakest die” (Darwin, 2011, p. 105). While Darwin was specifically describing natural selection in the non-human world, his thoughts on evolution, survival, and speciation would soon be appropriated and misapplied to human populations. In fact, Darwin’s quotation about letting “the strongest live and the weakest die” would be removed from its context and evolve into the motto of the social Darwinism movement: ‘survival of the fittest’ (Darwin, 2011, p. 105).

Though the phrase ‘survival of the fittest’ was extrapolated out of Darwin’s words, it was actually coined by the British biologist and social philosopher Herbert Spencer (1820-1903) in his 1864 book *Principles of Biology* (Falk, 2020). Drawing connections between conservative economics, social structures, and theories of evolution, Spencer argued that human societies and their organizing principles were subject to the same laws of nature – namely natural selection – as the flora and fauna Darwin described in *The Origin of Species* (Falk, 2020). More specifically, according to Gregory Claeys (2000), the theory of social Darwinism applied “the idea of evolution to a higher social type on the basis of social competition between ‘fit’ and ‘unfit’ groups and individuals, whose ‘fitness’ or ‘value’ to society [could] be defined in a number of

\(^5\) Today, we recognize natural selection as an evolutionary process through which “non-random difference in reproductive output among replicating entities, often due indirectly to differences in survival in a particular environment, lead[s] to an increase in the proportion of beneficial, heritable characteristics within a population from one generation to the next” (Gregory, 2009, p. 156).
ways” (229). The implications of Spencer’s theory of social Darwinism were profound, causing new ideas about natural hierarchies to be accepted in the public sphere. In fact, because of Spencer’s work, class stratification was seen as natural – the result of the natural order of the “unimpeded growth of society” (Hofstadter, 1944, p. 27). In advocating for laissez-faire capitalism, Spencer linked the control of economic property with success, including moral and character success (Hofstadter, 1944). While the rich were classified as the holders of both wealth and morally desirable traits under this system, the poor were seen as unfit and potentially worthy of elimination from the human race (Hofstadter, 1944). While much of Spencer’s early work discussed class-based hierarchies and capitalism specifically, social Darwinism was used to justify ideologies of political conservatism, imperialism, and racism as well.

In the United States, the social Darwinist movement was responsible for laying the foundation for the eugenics movement, an ideological force that would add the notion of reproductive control on the basis of heredity to Spencer’s arguments (Kurbegovic, 2014). The term ‘eugenics’ itself was coined in 1883 by Sir Francis Galton (1822-1911), a British anthropologist and the second cousin of Darwin (Kurbegovic, 2014). Following the publication of Darwin’s The Origin of Species, Galton (1908) became obsessed with investigating questions “clustered round the central topics of Heredity and the possible improvement of the Human Race” (p. 288). Galton’s 1883 book, Inquiries into Human Faculty and its Development, pulled together his life’s work – “the results of his twin studies, his thoughts on anthropometrics and statistics, as well as psychometrics (another term we owe to Galton), psychology, race, and population – to argue that the human civilization could be improved through the racialized process of selective breeding” (Gillham, 2001, p. 98).
Galton (1883) named this field of inquiry “eugenics,” which was to be focused on those questions that have “bearing on what is termed in Greek, *eugenēs*, namely, good in stock, hereditarily endowed with noble qualities” (p. 24). Essentially, this field of ‘science’ was based on the idea that the human race could be improved or advanced by promoting the reproduction of individuals/populations with ‘desirable’ traits (a process known as positive eugenics) and by preventing, even forcibly, the reproduction of individuals/populations with traits deemed to be ‘undesirable’ (a process known as negative eugenics) (Wilson, 2013). This movement was a full-fledged version of the concerns that Nott and Agassiz, among others, had brought up about racialized reproduction. Through the eugenics movement, scientists had come up with potential ‘solutions’ to their predecessors’ concerns, solutions that would use the biological basis of race to enact harm upon people of color and their offspring.

As Michael Yudell (2014) has written in his book *Race Unmasked*, “If Francis Galton was the theoretician of eugenics, then Charles Davenport was its engineer and American torchbearer” (p. 31). American eugenicist Charles Davenport (1866-1944) was a Harvard-trained biologist who, in 1904, established the Station for Experimental Evolution at the Cold Spring Harbor Laboratory, one of the first major sites for eugenics research in the U.S. (Largent, 2002). In 1910, Davenport added another layer to his research station and established the Eugenics Records Office (ERO), which would continue to be the American epicenter for eugenics research until it closed in 1939 (Cold Spring Harbor Laboratory, 2008b). According to the Canadian Eugenics Archive:

The ERO sent out investigators and questionnaires to families to get information on their talents, medical problems, personalities, social standing, and so on. The ERO’s public presence was used to promote restrictions on immigration, segregation of the so-called unfit, and sexual sterilization legislation. (Ball, 2013, para. 3)
Through his work with the ERO, Davenport was able to definitively add the concept of modern-day genetics to the biological basis of race. While it had long been hypothesized that internal ‘traits’ could be passed on from parent to offspring, including the ‘trait’ of race-based degeneracy, Davenport was able to advocate for the inclusion of Mendelian genetics into the eugenics literature. Mendelian genetics, named after Gregor Mendel and his famous pea-plant experiments, demonstrated that there were “regular statistical patterns [of inheritance] for features like height and colour” (DNA Worldwide, 2014, sec. 4). Mendel conducted his experiments between 1856 and 1863, and his results were rediscovered in the U.S. in the early 1900s by Davenport and his fellow eugenicists (DNA Worldwide, 2014).

Though the biological basis of race had plenty of ‘evidence’ by this point (especially from the ERO), Davenport used Mendel’s theories to add in the language of genetics to solidify the scientific basis of race-based biology. This was evidenced in his 1917 article “The Effects of Race Intermingling,” in which Davenport:

> offered what he called a modern geneticists’ definition of race—that is, “a more or less pure bred ‘group’ of individuals that differs from other groups by at least one character, or, strictly, a genetically connected group whose germ plasm is characterized by a difference, in one or more genes, from the other groups.” (Yudell, 2014, p. 40)

Thus, through Davenport’s work, the biological basis of race had evolved to be focused on heredity – particularly genetics – and this relationship was key to the success of the eugenics movement. Davenport’s predecessors had set up questions of reproduction in such a way that, with Davenport’s final addition, people of color could finally be named as having ‘bad genes.’ Environmental or systemic factors were nothing in comparison to the inherently defective genetics that these people carried. Such genetic deficiency could not be cured or solved, which made them a threat to the entire American (white) population, a threat that could not be solved
by rehabilitation. It was with this vision of eradicating degeneracy (which was now genetically associated with non-whiteness) from American society that the eugenics movement fully established science as an institution of power that all Americans would have to reckon with in the following centuries.

Despite the horror with which we view these ideologies today, eugenicists in the 19th and 20th centuries saw themselves as working toward the public good. In fact, eugenicists of the time were “optimistic that scientific changes in human breeding habits would solve many complex problems facing modern American society” (Cold Spring Harbor Laboratory, 2008a, para. 1). As the Cold Spring Harbor Laboratory (2008a) has further explained, “by sterilizing the mentally ill and restricting foreign immigration, eugenicists sought to isolate the American genetic stock from the taint of allegedly bad genes” (para. 1). Essentially, advocates saw eugenics as a way of affirming the health of many, though in reality, this was a racialized goal that extended exclusively to white populations and denied the right to health, particularly reproductive health, to people of color and their offspring on the basis that their genetics were inherently defective.

Evolutions: Women of Color as Targets of Scientific Racism

While the ERO wouldn’t be formed until 1910 and the term eugenics itself wouldn’t be coined until 1883, even in the 1870s, the idea of heredity was already starting to take hold within the American public at large. Even at this point in American history, the scientific racism movement was becoming pervasive, with eugenics ideologies commonplace and readily accepted among most of the (white) American families. White women, including those who were in the pursuit of women’s rights or liberation endeavors (which we may name as early feminists today), were not excluded from the effects of these ideologies. In fact, in many ways, the
intertwined interactions of women’s rights movements and scientific racism solidified the connection between reproduction and biological race and made genetic heredity an issue that demanded sociopolitical action. This not only brought race-based ‘science’ into the legal, social, economic, and political spheres but made women of color (as opposed to all people of color) the specific target of eugenics action. Essentially, because of the emphasis on reproduction that was brought up through the interactions between some early feminists and scientific racist movements, U.S. society began to directly focus on the woman of color as the source of the race-based degeneracy, a source that needed to be controlled through reproductive policies.

The relationship between feminist movements and scientific racist movements first became prominent during the 1870s, when some early feminists used their work to paint white women as harbingers of the solutions propagated in the eugenics movements (and, thus, by extension, non-white women as part of the problem). This characterization of white women primarily started to come about as the result of political maneuverings within the women’s suffrage movement, where racist ideologies were used to gain political prowess – specifically the right to vote – for white women. Elizabeth Cady Stanton (1815-1902), one of the dominant forces behind the Seneca Falls Convention and a leader within the women’s rights movement of the time, played a crucial role in this endeavor.

Suffragists like Stanton faced many barriers to securing the right to vote, though some of the most common were arguments that cited “biological differences” that characterized women

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6 Of course, the women’s rights movements, even those led predominantly by white women or white feminists, were not universal in their goals, practices, or effects. The instances mentioned here are just a subject of the experiences and realities of early feminist movements.

7 Even within the suffragist movement, Stanton’s views were not necessarily universally shared. In fact, several suffragists were aligned with the abolitionist movement, especially earlier. A more nuanced and detailed history of the relationship between racism and women’s liberation movements can be found in Daughters of Jefferson, Daughters of Bootblacks by Barbara Andolsen (1986).
as inferior to men (particularly with regards to their intelligence) and thus, unworthy of the right to vote (Rensing, 2006, p. 33). Fed up by her opponents’ ‘scientific’ arguments, Stanton decided to look to the latest science for an answer to fight back. In an unexpected twist of Galton’s early eugenics theory, Stanton actually used the concept of heredity to combat the sexist science that was preventing women’s suffrage. Much of Galton’s work focused specifically on non-white populations as holders of ‘undesirable’ traits, but he also clearly recognized that, as the bearers of future progeny, women had a pivotal role to play in deciding the fate of the next generations of humanity. Using this rationale, in her 1879 article, “The Other Side of the Woman Question,” Stanton used Galton’s argument, which stated that:

No matter how different men and women outwardly appear… inside them both flows hereditary material that is constantly mixed and shared in subsequent generations through procreation.’ Taking this argument from heredity one step further, Stanton cited the research of “Galton and Ribot” who illustrated that not only are hereditary characters inherited from both parents, but that there seemed to be a law of “cross-heredity” whereby “daughters more frequently inherit the characteristics of the father, and sons the characteristics of the mother.” Thus, if women inherited their weakness and biological inferiority, then they inherited it from their fathers. . . The corollary of this claim was that if society wanted to improve the hereditary quality of men, then they would have to go through their mothers. (Rensing, 2006, p. 33)

By using Galton’s concept of heredity to undermine the biological basis of sex and gender inferiority, Stanton – intentionally or unintentionally – validated its other applications as well, namely the biological basis of heredity, including racial heredity. The irony here is that the same arguments that deconstructed scientific sexism essentially affirmed scientific racism. In using the concept of heredity to fight for the right to sociopolitical access for white women, Stanton also caused white American women to be fundamentally tied to the procreation of the desirable traits identified through the eugenics movement, work that would continue to promote the biological basis of race. White women became the bearers of desirable traits to improve the quality of
‘white stock,’ but as a corollary, women of color specifically became the bearers of undesirable traits for generations to come.

With the ‘threat’ of women of color’s reproduction entering accepted social thought, other feminists used this opportunity to continue to advocate for white women’s rights. One example of this was seen in the late 19th century in the free love movement, which “emphasized the right for women and men to choose their sexual partners regardless of institutional sanction” and, as such, rejected marriage, seeing it as “an institution that fostered the degradation and inequality of women” (Hayden, 2013, pp. 2-3). In many ways, the free love movement was based on the idea of female ‘sovereignty,’ or a women’s right to bodily self-determination. Feminist scholar Stephanie Athey (2000) has argued, however, that during the late 19th century and early 20th century, women’s sovereignty emerged as a “racially charged concept,” particularly through the writings of Victoria Woodhull, that caused a differentiation between white women and women of color’s natural right to be seen as sovereign beings (para. 1).

Woodhull (1838-1927) was a leader in both the suffrage and free love movements, most well-known for her scathing critiques of marriage. Woodhull, like many of her fellow free love feminists,8 saw marriage as a manifestation of the “awful crime of sexual slavery,” a crime that denied women their right to sexuality and even bodily sovereignty (Woodhull, 1874, p. 33). However, for Woodhull, much like with Stanton, this right belonged only to white women, and in order to achieve this liberty for white women, Woodhull was willing to let it be at the cost of women of color. As such, Woodhull framed free love in the context of biological improvement, writing that sexual freedom (for white women) was a “place to begin the work of improving the race,” especially considering “the rapid multiplication of the Negroes in America” (Woodhull,

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8 Similar to the women’s suffrage movement, the free love movement was not universal nor were its beliefs. Several free love activists had connections to abolitionism, which should be further explored in future work.
1874, p. 29). Through Woodhull’s work, we see another example of white women advancing their sociopolitical agenda by reifying the biological basis of race in order to frame women of color as a threat to the American public, a threat that white women could help solve if they received increased liberation.

While Woodhull embraced the theory of the biological basis of race, a comprehensive analysis by Athey (2000) has demonstrated some key ways in which her thinking wasn’t a full manifestation of the eugenics movement, at least not yet. According to Athey (2000):

Though [Woodhull] decries the increasing numbers of “unfit” among the white populace, and though she warns against “the rapid multiplication of the Negroes of America,” she does not argue for reproductive restrictions on “unfit” whites or “lower” races; she does not advocate military control of the “vast hordes” multiplying in the non-white world; she does not, to my knowledge, openly or obliquely advocate lynching. Yet clearly this program for the economic, political, reproductive and sexual “freedom” of (white) women is simultaneously an argument for white supremacy; the independence of white women will stave off the racial decline of Western civilization. (para. 27)

So, while it can be inferred that, in Woodhull’s eyes, Black women posed a threat to the individual white body and perhaps even to the collective white body (read: society) of America for (re)producing ‘Blackness’ through their offspring, she didn’t advocate for negative eugenics policies to be used against them. Instead, she hinted at the use of positive eugenics (via increased sexually liberated procreation by white women) but didn’t go as far as to explicitly dictate a need for explicit reproductive control over women of color – though her successors most certainly would. Nonetheless, for Woodhull, this threat of Blackness, which was tied to a false understanding of Blackness as inferior, was clearly based in biology, as we see from her heavy emphasis on racialized reproduction. Therefore, through Woodhull’s statements, the Black woman became unworthy of bodily sovereignty because of the inherited ‘degeneracy’ she carried in her genetics. In later years, this lack of sovereignty would drive the idea that women of color’s
autonomy could be violated without consequence, which we would see manifested through targeted, racialized forms of violence such as sexual abuse and forced sterilization.

While feminist advocacy continued regarding both suffrage and free love, by the beginning of the 20th century, the biggest threat for all of U.S. society, feminists included, was race suicide, or the idea that the higher net rates of births among ‘unfit’ populations were going to lead to the extermination of ‘fit’ populations in the United States (Zimmerman, 2019). In fact, in 1905, President Theodore Roosevelt (2002) gave a speech to the National Congress of Mothers in which he addressed the pressing issue of race suicide and called for (white) women to be “able and willing to bear, and to bring up as they should be brought up, healthy children, sound in body, mind, and character, and numerous enough so that the race shall increase and not decrease” (para. 4). Roosevelt’s call to action sparked a new commitment, on behalf of himself, the American government, and social advocates of the time, to the growth of ‘fit’ (read: white) families to address the threat of race suicide.

The Better Babies Contests, which were established in 1908 by Mary de Garmo (1891-1986), a women and children’s health advocate, were just one of the feminist initiatives that arose out of Roosevelt’s call to action (Gerais, 2017). These contests, the first of which was held at the Louisiana State Fair, sought to improve infant health, especially in light of the high rates of infant mortality that the U.S. was facing at the time (Gerais, 2017). The nurses and doctors running the contests would measure babies according to behavioral and physical guidelines for normal child development as well as according to their “lineage and cumulative flawlessness” (Uenuma, 2019, para. 2). Implicit to the depiction of the ‘best’ baby was that the baby was white and came from a lineage of ‘pure’ whiteness (Cold Spring Harbor Laboratory, 2008a; Uenuma, 2019). The Better Babies Contests quickly spread across the United States from 1908 to 1935,
being held across states such as Indiana, Iowa, and Minnesota (Stern, 2002). As the contests grew in number and size, the implicit connection between ‘better’ babies and race-based eugenics became more and more apparent. In many states, African American children were explicitly excluded from these contests, which “promoted the idea that only White babies could achieve perfection” and further emphasized the biological ‘degeneracy’ of babies of color (Stern, 2002, p. 748).

While this segregation meant that the bodies of Black children couldn’t be criticized or exploited at the Better Babies Contests, there were other harmful consequences of being excluded from these contests. In many areas of the United States, the main push for improved fetal/neonatal health at this time came from the Better Babies Contests (Uenuma, 2019). So, if a child was ill, missing development milestones, or had a significant health problem after birth, it was often caught at these contests, where it could then be promptly addressed by doctors and nurses (Uenuma, 2019). By excluding non-white individuals, the Better Babies Contests were one way in which healthcare access was denied to women of color and their infants. This can be seen as a manifestation of negative eugenics, where infants of color were more likely to die out simply because of unequal access to neonatal healthcare. This scenario contributed to higher rates of infant mortality among populations of color and potentially even acted as a deterrent for women of color to have babies, as they knew that their ability to have healthy children would be impeded by a lack of access to the same care white women were getting for their infants (Uenuma, 2019). Overall, the race-based exclusion from the Better Babies contests emphasized that babies of color were undesirable as well as unworthy of being saved. It sent the message that their ‘degeneracy’ was so ingrained into their biology that they: a) would die regardless of care received or b) perhaps even deserved to die. This is the horror that can arise, and historically has
arisen, from the application of the biological basis of race onto women of color and their offspring.

While certain feminists were worried that not enough (white) babies were being born (and surviving), others like Margaret Sanger (1879-1966) were promoting the use of birth control so that women could more easily control the number of babies they wanted to have. In fact, in 1916, Sanger opened the first birth control clinic in the United States in a Brooklyn, New York neighborhood with the goal of providing women the reproductive freedom to choose whether or not to have children (Planned Parenthood, 2016). While birth control was being condemned by many who were concerned about the threat of race suicide, Sanger actually saw her work as a critical part of improving the quality of the human race as per eugenics ideologies. In fact, while other eugenicists were “focused only on differential fertility and placing eugenic demands upon women, Sanger argued that birth control developed ‘a higher standard of motherhood’ that enabled ‘the child to be better born, better cared for in infancy and better educated’” (Rensing, 2006, p. 162).

That being said, Planned Parenthood (2004) has asserted that Sanger firmly “repudiated any racial application of eugenics principles” (p. 3). Despite her supposed disdain for the racial applications of eugenics, Sanger still supported incentives for sterilization of those with “untreatable, disabling, hereditary conditions,” policies that prevented “diseased and ‘feebleminded’” immigrants from entering the country, and the isolation/exile of “illiterates, paupers, employable, criminals, prostitutes, and dope-fiends” – all of which were common propositions in the eugenics movement of the time (Planned Parenthood, 2004, p. 3). So, while her eugenics beliefs may not have been explicitly race-based, Sanger still believed in the concept of inherited degeneracy. From an intersectional standpoint, we can recognize that any kind of
policies based on assigned ‘degeneracy,’ including those that targeted the poor and those deemed to be less intelligent, could still indirectly target people of color (because of systemic inequities that resulted in poverty as well as racist ideas that non-white individuals genetically had a decreased capability for intelligence). Thus, Sanger’s eugenics beliefs still conformed to the biological basis of race, even if she did not necessarily explicitly support race-based eugenics. This made the choice of whether or not to have children as one only belonging to white women, leaving so-called ‘degenerates’ like women of color to be forcibly sterilized, ultimately left without the sovereignty to make decisions about their own reproduction.

Sanger’s views on sterilization were further validated in the 1927 Supreme Court case *Buck v. Bell*. The case surrounded Carrie Buck, an 18-year-old woman who had been raped and subsequently became pregnant in 1924 (Gross, 2016). Upon the discovery of her pregnancy, her guardians had Buck diagnosed with ‘feeble-mindedness,’ a “capaciously defined condition that was diagnosed often using flawed intelligence tests and by identifying symptoms such as moral degeneracy, an overactive sex drive, and other traits liberally ascribed to poor people (especially poor women) [and poor women of color] who were seen as having stepped out of line” (DenHoed, 2016, para. 2). With this diagnosis, in 1924, Buck was institutionalized at the Virginia Colony for Epileptics and Feeble-Minded, where she was the first person forcibly sterilized under a new Virginia law that targeted those whose biology posed a ‘threat’ to the reproductive success of the race (Antonios & Raup, 2012). Three years later, *Buck v. Bell* arrived at the Supreme Court, where the justices upheld Carrie Buck’s sterilization as well as forced sterilization for anyone who met the category of ‘feeble-mindedness’ in the United States in an 8-1 decision (Antonios & Raup, 2012). Ultimately, from 1927 to the 1970s, *Buck v. Bell* allowed for the legal compulsory sterilization of 70,000+ women across the United States, many of whom
were women of color who had been falsely labeled as ‘feeble-minded’ under a eugenics mindset (Gross, 2016).

Similar laws enacted by the U.S. government after the *Buck v. Bell* precedent allowed for the forced sterilization of 33% of all women in Puerto Rico during the 1930s-1970s and 25% of all Native American women of childbearing age during the 1970s (Andrews, 2017; Theobald, 2019). In 1974, a similar court case, *Relf vs. Weinberger*, revealed widespread sterilization abuse by the U.S. federal government (Southern Poverty Law Center, n.d.). The case demonstrated that 100,000-150,000 poor individuals per year had been coerced into sterilization procedures, being forced to sign documents they did not understand and/or threatened to have their welfare revoked if they did not undergo sterilization, making the overall number of women who underwent compulsory sterilization into the hundreds of thousands, if not millions, throughout the 20th century (Southern Poverty Law Center, n.d.). These forced sterilizations, which deprived individual women of color of the right to their own bodies, their autonomy, and their reproductive choices and enacted a racial genocide on their communities, were based on the biological basis of race – the notion that their racial identity was associated with an inherent, homogenous, and unchangeable ‘degeneracy’ that could be passed on via reproduction.

Throughout this documented history, the biological basis of race was used to label women of color in the United States as debased beings who did not deserve any bodily sovereignty, including when it came to reproduction. Forced sterilization was perhaps the most prominent example of this, where women of color were coerced or deceived into giving up a lifetime’s option of having children – a clear violation of reproductive justice. But what about those babies who were born to women of color? Because of the biological basis of race, the
offspring of women of color have been seen as unnatural beings that carried defectiveness within 
their genes and, as such, did not deserve the resources necessary to live a thriving life.

Essentially, in line with eugenics ideals, those programs, resources, and support that 
could (and should) have been offered to women of color and their offspring to counteract the 
systemic discrimination they faced from class-based, race-based, and gender-based oppression 
simply weren’t provided to them– because a biological understanding of non-whiteness was used 
to justify their oppression and their non-survival as natural. Historically, this denial of care 
manifested across institutions of power, though it had some of the most visible effects on the 
health of women of color and their offspring when it was enacted within the institution of 
medicine. The Better Babies contests were just one example of this, but there are many others⁹ 
that indicate that the children of women of color were (at best) ignored by and (at worst) actively 
harmed by institutions of medicine, healthcare, and science because said offspring were deemed 
biologically unworthy of saving. Dorothy Roberts (2014) has described this phenomenon in 
detail, writing that, during the eugenics boom in the early 1900s:

[ Eugenicists ] argued that adequate medical care, better working conditions, and minimum 
wages all harmed society because those measures enabled people with inferior heredity to 
live longer and produce more children. The Harvard geneticist Edward East, for example, 
complained that the provision of prenatal care and obstetric services to the poor through 
clinics and public hospitals was “unsound biologically” because it “nullifie[d] natural

⁹ Other examples in more recent history of the denial women of color to have healthy children raised in healthy 
environments can be found in Dorothy Roberts’ Killing the Black Body. For example, Roberts (2014) has discussed 
how medical institutions have been a part of criminalizing women of color who used drugs such as crack in their 
pregnancy. The Medical University of South Carolina (MUSC) was one hospital in which physicians became 
mandated reporters of drug use in pregnant women, despite the right to physician-patient privilege and the fourth 
amendment’s protections against warrantless searches (Roberts, 2014). As Roberts has further explained, in the 
1990s, MUSC “instituted the Interagency Policy on Cocaine Abuse in Pregnancy (Interagency Policy), a series of 
internal memos that provided for nonconsensual drug testing of pregnant patients, reporting results to the police, and 
the use of arrest for drug and child abuse charges as punishment or intimidation” (p. 260). Ultimately, these policies 
did not help women of color receive treatment for their addiction nor did they seek to help their offspring, many of 
whom would end up in the tumultuous foster care system. In these cases, the health of mother and child were not 
prioritized, as they were seen within the paradigm of non-white degeneracy from the moment they entered the 
healthcare system.
elimination of the unfit.” (pp. 106-107).

While this example specifically focused on the poor, this eugenics-based rationale applied to anyone who was assumed to have “inferior heredity,” including people of color (Roberts, 2014, p. 106). As we see in this quotation, the biological, and specifically genetic basis of race, has been used historically as a rationale to deny using medical or healthcare services to women of color and their children in an effort to limit their reproduction. Thus, it is crucial that we recognize that women of color have not only been denied the choice of whether or not to have children (through forced sterilization) but also denied the right to raise the children in healthy environments that allowed them to thrive, which must also be considered as a blatant violation of reproductive justice.

Today, this historical trend is still ongoing. The biological basis of race is still being used to deny necessary reproductive care for women of color and their offspring through the institutions of medicine, healthcare, and science. Within the field of maternal-fetal medicine, one way that this has manifested is in the discussion around increased rates of birth defects among women of color. In seeking explanations for this trend, physicians and scientists have attributed the increased rates of birth defects among infants of color as the result of inherited ‘defective’ traits from their mothers – traits that they assume are the result of a high prevalence of ‘defective’ alleles within racial/ethnic minority populations. This is a hypothesis that is based on outdated, inaccurate science but also one that is incredibly dangerous, as it can be used to further the idea that these birth defect trends are ‘natural’ – or as earlier eugenicists might have phrased it, a “natural elimination of the unfit” (Roberts, 2014, p. 107).
CHAPTER THREE

THE BIOLOGICAL BASIS OF RACE AS A FALSE EXPLANATION OF HEALTH DISPARITIES AMONG WOMEN OF COLOR TODAY

Within the 21st century, it has become well established that the biological basis of race is an idea that is not factually supported by any evidence – scientific or otherwise. Instead, we have shifted to recognizing race as socially constructed, meaning that “no coherent, fixed definition of race actually exists” (Coates, 2013, para. 3). There is no objective factual conception of race; it is an identity that has been ascribed malleable meanings by the collective society and sub-societies we live in (Zach, 2017). However, while much of humanity now accepts that race is not a biological category, this does not erase the fact that the biological basis of race was heavily reified through much of early scientific thought – particularly through the social Darwinism and eugenics movements – as well as through early U.S. (white) feminist liberation movements. Because of this history, the biological basis of race has not simply disappeared now that we have entered the 21st century. In many ways, the institution of science, especially as manifested in medicine/healthcare, has sought to distance itself from and even deny the ‘dark’ history of scientific racism. By failing to reckon with this history – failing to acknowledge it, address it, and subsequently dismantle its remnants – the field of medicine has complacently allowed biological constructs of race to exist today (though perhaps in more subtle ways than they have historically), where they continue to harm people of color, and especially women of color.
Within maternal-fetal medicine, the pervasiveness of the myth of the biological basis of race has allowed race to become a ‘risk factor’ for health disparities, which relies on the idea that race is a homogenous, unchangeable category indicative of internal biological phenomena. This kind of race-based medicine is especially prominent in the case of increased rates of birth defects seen among infants born to non-white mothers,¹ in which women of color are blamed for having racially-specific “genetic susceptibilities” that result in the disparate rates of certain birth defects seen among Hispanic, Black, Native/Indigenous, and Asian populations (Egbe, 2015, p. 183; Ibrahim et al., 2014, col. 2). Not only is this hypothesis contrary to evidence we have about the biological basis of race today (as race is not a genetic category²), but it is a hypothesis reliant on a history that has constructed non-whiteness to be a defective trait that is passed down through degenerate women of color.

In order to deconstruct how the biological basis of race is prevalent within the field of maternal-fetal medicine today, this chapter will investigate the following two questions: 1) what scientific evidence has been used to justify the claim that race is a risk factor for certain birth defects? 2) what are the effects of using the biological basis of race to justify a health disparity like the increased rates of birth defects among infants of color? To answer these questions, the first section of the chapter explains why race has been considered a risk factor for birth defects; it includes 10 studies from a non-systematic literature review on scientific studies published on

¹ It is important to note that not all women who give birth may identify as mothers and that not all individuals who give birth are women. However, I have chosen to use women-focused language in this project because that is the language used within the current scientific literature. Further research should seek investigate how these trends manifest among all birthing people and encourage more inclusive language within the scientific literature.

² Race is not a biological construct, meaning that there is no genetic component to race. However, race has been often used as a proxy for genetic ancestry, though this is not extremely accurate and can actually do more harm than good. Further research should investigate how concepts of genetic ancestry can be used for individualized medicine without “resorting to notions of race and ethnicity” (Fujimura & Rajagopalan, 2011, p. 5).
PubMed\textsuperscript{3} since 2000 that specifically looked at the relationship between race/ethnicity and prevalence of birth defects in the United States. The second section of the chapter discusses why using race as a risk factor actually perpetuates this racial disparity for women of color and their offspring.

**Why is race considered a risk factor for birth defects?**

Many previous studies have shown a link between increased risk of certain birth defects among non-white populations as compared to their white counterparts. The most comprehensive and arguably well-known study on this topic is an article by Canfield\textsuperscript{4} et al. (2014) that examined the prevalence of 27 specific birth defects among 13 racial/ethnic groupings by analyzing records from birth defect surveillance programs of 13.5 million live births in the U.S. from 1999 to 2007. In their study, Canfield et al. (2014) demonstrated an overall stratification of certain birth defects by racial/ethnic identity, noting increased rates of birth defects among infants born to Hispanic women, non-Hispanic Black women, non-Hispanic Asian women, and non-Hispanic American Indian/Alaska Native women in comparison to infants born to non-Hispanic white women. Specifically, Canfield et al. (2014) found that infants born to Hispanic women were significantly more likely to have the following 7 birth defects (p<0.05\textsuperscript{5}): anencephaly\textsuperscript{6} (aPR\textsuperscript{7}=1.6), spina

\textsuperscript{3} Articles were primarily collected through the following search on PubMed: ("birth defects"[Title]) AND ((race[Title]) OR (ethnic*[Title]) OR (racial[Title]) OR (black*[Title]) OR (African*[Title]) OR (Asian*[Title]) OR (Native*[Title]) OR (Indian[Title]) OR (Hispanic[Title]) OR (Latin*[Title])) AND ((U.S.) OR (US) OR (United States))

\textsuperscript{4} Mark A. Canfield, PhD is the Co-Principal Investigator of the Texas Center for Birth Defects Research and Prevention and is a part of the National Birth Defects Prevention Study. His expertise is in clinical genetics and epidemiological research.

\textsuperscript{5} The p-value, or probability value, is a statistical tool that is used to determine statistical significance. A p-value of less than 0.05 indicates statistical significance.

\textsuperscript{6} Anencephaly is a neural tube birth defect that results in the absence of a major portion of the brain or skull.
bifida\(^8\) without anencephaly (aPR=1.2), encephalocele\(^9\) (aPR=1.5), anotia/microtia\(^{10}\) (aPR=2.4), rectal and large intestinal atresia\(^{11}\) (aPR=1.1), Down syndrome\(^{12}\) (aPR=1.4), and trisomy 18\(^{13}\) (aPR=1.2). Infants born to non-Hispanic Black women were significantly more likely to have the following 8 birth defects (p<0.05): encephalocele (aPR=1.8), Tetralogy of Fallot\(^{14}\) (aPR=1.2), atrioventricular septal defect\(^{15}\) (aPR=1.3), atrioventricular septal defect without Down syndrome (aPR=1.4), lower limb deficiency\(^{16}\) (aPR=1.2), omphalocele\(^{17}\) (aPR=1.4), trisomy 13\(^{18}\) (aPR=1.4), and trisomy 18 (aPR=1.7). Canfield et al. (2014) also found that non-Hispanic American Indian/Alaska Native women were significantly more likely to have the following 8 birth defects (p<0.05): encephalocele (aPR=2.1), anotia/microtia (aPR=4.0), cleft lip with or

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\(^7\) aPR is the abbreviation for adjusted prevalence ratio. In this case, an aPR of 1.6 indicates that infants born to Hispanic women are 1.6 times as likely to have anencephaly as compared to infants born to non-Hispanic white women.

\(^8\) Spina bifida is a neural tube birth defect that results from maldevelopment of the spinal cord.

\(^9\) Encephalocele is a neural tube birth defect that results in sac-like protrusions on the head of a neonate.

\(^10\) Anotia and microtia are two birth defects of the external ear.

\(^11\) Atresia is a birth defect that results in the abnormal narrowing of an opening or passage of the body, specifically of the rectum and large intestine in this case.

\(^12\) Down syndrome is a condition that is cause by the presence of an extra chromosome 21.

\(^13\) Also known as Edward’s syndrome, trisomy 18 is a condition that results from the presence of an extra chromosome 18. Children born with trisomy 18 often have low birth weight as well as life-threatening organ defects, typically in their heart and lungs.

\(^14\) Tetralogy of Fallot is a congenital heart defect that is characterized by four specific problems: ventricular septal defect, valve stenosis, an aorta that is in the wrong position, and a thickened right ventricle.

\(^15\) An atrioventricular septal defect is a congenital heart defect that is characterized by one or more holes in between the left and right chambers of the heart. Atrioventricular septal defect is often associated with Down syndrome.

\(^16\) Lower limb deficiency is a syndrome that describes any sort of maldevelopment of the lower limbs, ranging from complete absence of a lower limb to the absence of a digit on the lower limbs.

\(^17\) Omphalocele is a birth defect in which the abdominal cavity fails to fully cover the internal organs, causing the intestines, liver, or other internal organs of the neonate to be outside of the abdomen.

\(^18\) Also known as Patau syndrome, trisomy 13 is a condition that results from the presence of an extra chromosome 13. Trisomy 13 is associated with severe intellectual disability and physical defects.
without cleft palate\(^{19}\) (aPR=1.9), upper limb deficiency\(^{20}\) (aPR=1.5), lower limb deficiency (aPR=1.9), any limb deficiency (aPR=1.5), gastroschisis\(^{21}\) (aPR=1.4), and trisomy 18 (aPR=1.9). Alternatively, infants born to non-Hispanic Asian women were at a significantly higher risk for only 1 birth defect (p<0.05): anotia/microtia (aPR=1.4). It is interesting to note that, of all the racial/ethnic populations included in the study, infants born to non-Hispanic Asian women had the least number of individual birth defects associated with increased risk compared to their white counterparts. In contrast, infants born to Hispanic, Black, and American Indian/Alaska Native women all had a much higher number of birth defects associated with increased risk compared to their white counterparts.

To date, the study by Canfield et al. (2014) remains one of the largest population-based projects “to systematically examine the prevalence of a range of major birth defects across many racial/ethnic groups” (Canfield et al., 2014, p. e14). That being said, the study investigators did not propose any legitimate rationale for why these trends might exist within the U.S., merely noting that it warranted further investigation. While some of the birth defects highlighted in the study by Canfield et al. (2014) are somewhat explicitly related to genetic problems within egg/sperm cells, such as the chromosomal conditions trisomy 13 and trisomy 18, the analysis conducted by this study failed to account for other variables or factors that could help explain this trend at the large scale at which it seems to be occurring. Canfield et al. (2014) adjusted for maternal state of residence at delivery and maternal age but failed to incorporate other relevant

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\(^{19}\) Cleft lip and cleft palate are birth defects that are characterized by an opening in the upper lip or roof of the mouth. They can occur together or separately.

\(^{20}\) Similar to lower limb deficiency, upper limb deficiency is a syndrome that describes any sort of maldevelopment of the upper limbs, ranging from compete absence of an upper limb to absence of a digit on the upper limbs.

\(^{21}\) Gastroschisis is a birth defect in which the entire bowel of the fetus develops outside of the abdomen.
explanatory variables such as healthcare access or socioeconomic status into their model. Thus, while these data demonstrate evidence of the racial stratification of birth defects, the study as a whole failed to discern the real driving factors behind this trend.

Because of this limitation, for many readers, this article presents the trend of increased rates of individual birth defects among racial/ethnic minorities as a direct result of minority racial/ethnic identity itself. This conveys the idea that the risk factor itself is being a person of color – not because of the systemic inequity that people of color face – but because of race-based defective biological traits carried within the genes of people of color. This frames the entire conversation around the increased rates of birth defects as a problem related to the mother’s ‘inherent’ racial identity, an interpretation that fails to account for how race, as a social construct, intertwines with other factors, such as socioeconomic status or environmental injustice, to cause inequity in health outcomes. With this being one of the most prominent studies in the field, it sets the stage for the conversation around the disparate rates of birth defects faced by racial/ethnic minorities to be framed as one related to biological race.

While the study by Canfield et al. (2014) is one of the largest and most frequently cited scientific analyses of these trends, there is a substantive body of literature looking at the relationship between birth defects and race/ethnicity from as early as the 1980s (if not earlier) (Polednak, 1986). One of the earlier studies looking at this trend, specifically in relation to Hispanic populations, was by Kirby\(^ {22} \) et al. (2000) and analyzed data from birth defect surveillance programs within states with at least 10,000 resident Hispanic births per year. On average, the study found that the rates of neural tube defects were significantly higher among

\(^ {22} \text{Russel Kirby, PhD, MS is a “doctorally-trained human geographer with extensive training and experience in public health practice, academic medicine, and academic public health” (University of South Florida, n.d.). He has devoted his career to the study of maternal and child health.} \)
infants born to Hispanic women (6.1 cases/10,000 live births) than infants born to non-Hispanic white women (4.5 cases/10,000 live births) (p<0.001\textsuperscript{23}). More specifically, the data showed that infants born to Hispanic women were more likely to have the following neural tube defects in comparison to infants born to non-Hispanic white women: anencephaly (with Hispanic women having 1.3 cases/10,000 live births and white women having 0.7 cases/10,000 live births, p<0.001) and spina bifida without anencephaly (with Hispanic women having 4.2 cases/10,000 live births and white women having 3.0 cases/10,000 live births, p<0.001). These are in line with the results found in the study by Canfield et al. (2014), particularly regarding the high rates of neural tube defects among Hispanic populations.

Kirby et al. (2000) presented these data within the framework of existing healthcare inequity, noting that Hispanic women were 80% more likely to be teen mothers and 20% less likely to receive prenatal care than their white counterparts. That being said, however, their analysis failed to determine if the noted trends in teen pregnancy and prenatal care actually explained the racially disparate rates of birth defects. In fact, they did not even suggest a need to further investigate the rationale behind birth defect trends. Instead, Kirby et al. (2001) focused their analysis on how surveillance data could be improved to better identify the prevalence of birth defects within various racial/ethnic populations. While birth defect surveillance programs are essential, by failing to state the importance of investigating the factors behind these trends, Kirby et al. (2000) validated race/ethnicity as an unchangeable variable that is in and of itself explanatory.

This is further problematic because some of their ideas for birth defect surveillance improvement seem to indicate a connection between genetic abnormalities and racial

\textsuperscript{23} This p-value is substantially lower than 0.05, which indicates a high level of statistical significance.
populations. For example, Kirby et al. (2000) suggested that “if birth defects data are linked to clinical genetics databases… it may prove useful to collect race/ethnicity variables” (p. 27).

While this statement is not particularly clear, Kirby et al. (2000) seemed to indicate that this kind of record-keeping could help identify if certain racial populations have higher rates of clinical genetic abnormalities, which could then explain the increased rates of neural tube defects among infants born to Hispanic women. While certain geographical populations can have higher rates of certain genetic alleles, it is extremely difficult to push this historical population-level data onto the constructed racial categories. Because they failed to problematize this notion, Kirby et al. ended up conflating race/ethnicity and genetic ancestry, a false equivalency that ends up stereotyping Hispanic women as genetically ‘defective’ in order to explain existing rates of birth defects.

A similar study by Agopian et al. (2012) also found high rates of neural tube defects, specifically spina bifida, among infants born to Hispanic women. Agopian et al. (2012) analyzed spina bifida cases from 1997 to 2005 from the National Birth Defects Prevention Study to determine the relationship between different spina bifida subtypes/sub-phenotypes and maternal race/ethnicity. The results showed that “compared to non-Hispanic (N.H.) White mothers, offspring of Hispanic mothers had higher prevalences of each subtype and most sub-phenotypes, while offspring of NH Black mothers generally had lower prevalences” (Agopian et al., 2012, p. 109). Though they were able to corroborate the trends seen in Kirby et al. (2000), Agopian et al. (2012) still failed to provide further insight into why these disparities were occurring among racial/ethnic minorities. As they explained, “it is unclear if these apparent differences in maternal

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race/ethnicity might have been due to differences in socioeconomic/demographic characteristics, diet, or other environmental and/or genetic factors” (Agopian et al., 2012, p. 115). While rationales related to systemic inequity were suggested – namely socioeconomic status (which could be indicative of differential healthcare access, increased stress, lack of access to prenatal nutrition, etc.) and/or environmental exposure (which is related to race/ethnicity as the result of environmental racism), Agopian et al. (2012) still pointed to genetic factors specific to each racial/ethnic group as a possible source of birth defects.

While Kirby et al. (2000) and Agopian et al. (2012) specifically examined Hispanic populations, Forrester and Mertz (2004) sought to analyze birth defect trends among Native Hawaiian populations in comparison to white populations in Hawaii. Using data from the Hawaii Birth Defects Program, Forrester and Mertz (2004) analyzed records of infants and fetuses from 1986 to 2000 who had been diagnosed with 1 or more major birth defects. They found that infants born to Native Hawaiians had significantly increased risks of the following birth defects (p<0.05): microcephaly (RR=1.48), anotia/microtia (RR=3.66), Tetralogy of Fallot (RR=2.42), pulmonary valve atresia (RR=2.02), tricuspid valve atresia (RR=2.42), cleft

25 Mathias Forrester is an epidemiologist with 14 years expertise working with the Hawaii Birth Defects Program and 5 years with the Texas Birth Defects Monitoring Program.

26 Microcephaly is a condition that results in a child being born with a smaller brain than would be typically expected. According to Mayo Clinic (2021a), “microcephaly usually is the result of the brain developing abnormally in the womb or not growing as it should after birth” (para. 1).

27 RR stands for relative risk or risk ratio. It used in statistical analysis to estimate the strength of the association between risk factors and outcomes. A risk factor greater than 1 suggested an increased risk of a particular outcome in a particular group, while a risk factor less than 1 suggests a decreased risk of a particular outcome in a particular group – as compared to the reference group. Here, an RR of 1.48 suggests a 1.48 times increased risk of microcephaly among Native Hawaiians as compared to whites.

28 Pulmonary valve atresia is a birth defect in which the pulmonary valve (which controls blood flow from the heart to the lungs) does not form.

29 Tricuspid valve atresia is a birth defect in which the tricuspid valve (which controls blood flow from the right atrium to the right ventricle of the heart) does not form.
palate (RR=1.76), and polydactyly (RR=1.40). At the same time, Native Hawaiians had a significantly decreased risk for having infants with the following birth defects (p<0.05): coarctation of the aorta30 (RR=0.46), pyloric stenosis31 (RR=0.33), epispadias32 and hypospadias33 (RR=0.75), and congenital hip dislocation34 (RR=0.54). Overall, the crude relative risk was lower among Native Hawaiians for 25 (44.4%) of the birth defects analyzed within the study and significantly lower for 7 (13%) of the birth defects. Meanwhile, the relative risk was higher among Native Hawaiians for 30 (55.6%) of the birth defects included within the study and significantly higher for 7 (13%) of the birth defects. Ultimately, Forrester and Mertz (2004) could not identify “a clear pattern to those birth defects demonstrating the higher or lower crude rates among Native Hawaiians” (p. 239). They did, however, postulate that “such populations [referring to Native Hawaiian populations vs. white populations] were likely to differ with respect to genetics, behaviors, and exposures” and that an investigation of these three factors would be “useful in eliciting the causes of birth defects” (Forrester & Mertz, 2004, p. 239).

Although this analysis pointed to external factors as possible sources of birth defect etiology, Forrester and Mertz explicitly conflated population-level genetics35 with race-based

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30 Coarctation of the aorta is a birth defect in which the aorta, an artery that transports oxygen rich blood from the heart to the rest of the body, is narrower than typical.

31 Pyloric stenosis is a thickening of the muscle between the stomach and the intestines, which is known as the pylorus. This condition prevents food from entering the small intestine and results in severe vomiting.

32 Epispadias are a birth defect in which the opening of the urethra, the tube that allows for urine to exit the bladder, does not develop in the correct location.

33 Hypospadias are a type of epispadias in which the opening of the urethra is not located at the tip of the penis but rather anywhere from the end of the penis to the scrotum.

34 Congenital hip dislocation is when a child is born with an unstable hip as a result of maldevelopment of the ball and socket joint of the hip.

35 The field of population genetics is centered on the “origin, amount, frequency, distribution in space and time, and phenotypic significance” of genetic variation within groups of the same species that live in the same area (i.e., a population) (Templeton, 2018, p. 1). Human population genetics specifically centers on “genetic variation in human
genetics, just as Kirby et al. (2000) and Agopian et al. (2012) did in their work as well. Neither Forrester and Mertz (2004), Kirby et al. (2000), nor Agopian et al. (2012) presented any evidence that could support such a hypothesis; they did not cite any prior evidence that would actually suggest that there have been evolutionary migration patterns that have resulted in specific genetic anomalies being present at higher rates in certain population groups. Such evidence is likely nonexistent because groups such as Hispanics or Native Hawaiians (or any racial group really) are extremely heterogeneous, even genetically (Gannon, 2016). There is no one gene or group of genes that would be found in all members of this population because race is not a biologically determined category; it is socially constructed. Instead, Kirby et al. (2000), Agopian et al. (2012), and Forrester and Mertz (2004) all naively suggested a hypothesis that is entirely based on the idea of biological race, has no scientific backing, has had dangerous repercussions for women of color in the past, and has the potential to harm women of color and their offspring in the present.

While the analyses presented by these three groups did not heavily investigate other lifestyle factors as root causes of the increased rates of individual birth defects among infants born to women of color, Kucik et al. (2012) did take into account several external variables, including or maternal age, child sex, gravidity, and socioeconomic status when creating their model for birth defect trends. In their study, Kucik et al. (2012) analyzed the prevalence of 46 populations and its evolutionary and phenotypic significance based on the original human migration patterns out of Africa (Templeton, 2018, p. 1).

36 James Kucik, MPH, PhD has an academic background in Epidemiology and in Health and Public Policy. He served as a health scientist in the Division of Birth Defects and Developmental Disabilities for 13 years at the Centers for Disease Control and Prevention.

37 Gravidity is a term that refers to how many times a woman has been pregnant.

38 Kucik et al. (2012) specifically used the percent of the population below the federal poverty level as a measure of socioeconomic status.
specific birth defects among infants born to non-Hispanic white women, non-Hispanic Black women, and Hispanic women in metropolitan Atlanta between 1994 and 2005 as recorded in a population-based birth defects registry. After adjusting for other variables,\(^{39}\) the results of the analysis showed that infants born to Hispanic women were still significantly more likely to have the following birth defects as compared to infants born to white women (p<0.05): any atrial septal defect\(^{40}\) (aPR=1.31), muscular ventral septal defect\(^{41}\) (aPR=1.20), chromosomal defects\(^{42}\) (aPR=1.30), and Turner syndrome\(^{43}\) (aPR=2.48). Additionally, after adjusting for other variables, infants born to non-Hispanic Black women were significantly more likely to have the following birth defects as compared to infants born to white women (p<0.05): any atrial septal defect (aPR=1.34), secundum atrial septal defect\(^{44}\) (aPR=1.35), Hirschsprung disease\(^{45}\) (aPR=1.73),

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\(^{39}\) This indicates that after accounting for the impact of other factors, including socioeconomic factors, the statistical model still showed that infants born to women of color had higher rates of birth defects. This tells us that even after accounting for these variables, there was still some factor that race was acting as a proxy for or some factor that was not accounted for that was being masked by the variable of race within the model.

\(^{40}\) An atrial septal defect is a congenital heart defect characterized by the presence of a hole in the wall that divides the right and left atria (upper chambers) of the heart. There are many types of atrial septal defects; they are characterized by the location in which the hole occurs.

\(^{41}\) A ventricular septal defect is a congenital heart defect characterized by the presence of a hole in the wall that divides the right and left ventricle (lower chambers) of the heart. The most common type of ventricular septal defect is the muscular ventral septal defect, in which the hole occurs in the lower, muscular part of the wall between the two ventricles.

\(^{42}\) Chromosomal defects are a category of birth defects that include any chromosomal anomalies. This would include trisomy 13, trisomy 18, Down syndrome, etc.

\(^{43}\) Turner syndrome is a chromosomal birth defect that only affects girls. It is characterized by having only one complete X chromosome instead of two. According to Mayo Clinic (2021b), “Turner syndrome can cause a variety of medical and developmental problems, including short height, failure of the ovaries to develop and heart defects” (para. 1).

\(^{44}\) Secundum atrial septal defect is a type of atrial septal defect that occurs in the middle part of the wall that divides the right and left atria (upper chambers) of the heart.

\(^{45}\) Hirschsprung disease is a birth defect characterized by the absence of certain nerve cells in the large intestine of the neonate, which results in the neonate having difficulty passing stool.
cystic kidney\textsuperscript{46} (aPR=1.41), posterior urethral valves\textsuperscript{47} (aPR=1.84), polydactyly\textsuperscript{48} (aPR=1.66), and trisomy 13 or 18 (aPR=2.06). Based on these data, the authors concluded that racial/ethnic disparities were suggested in 57\% of the individual birth defects that were examined in the study (Kucik et al., 2012). Additionally, by comparing their adjusted prevalence ratios and their crude prevalence ratios,\textsuperscript{49} the authors found evidence of a potential interaction between race/ethnicity and the percentage of people in a mother’s census tract\textsuperscript{50} living below the federal poverty level for the ‘any defect’ category as well as for 43\% of the individual defects that were analyzed (Kucik et al., 2012).

To explain these trends, Kucik et al. (2012) suggested many possible avenues for further investigation. In their conclusion, they wrote that:

These data provide evidence to suggest that socioeconomic factors explain some of the variation in birth defect prevalence, with a hypothesis that inequity in access to quality medical and diagnostic services may explain a lower observed prevalence among poor racial/ethnic minority groups. Further examination of this interaction using both individual- and community level measures of SES could shed more light on the impact of the availability and access to healthcare services on the confirmed diagnosis of a birth defect. Disparities might be further explained by the differential use of elective pregnancy terminations, varying exposure to environmental teratogens, and differing genotypic profiles. (Kucik et al., 2012, p. 60)

Unlike some of the previous studies, Kucik et al. (2012) heavily emphasized alternative explanations for disparate rates of birth defects (other than biological race), such as unequal

\textsuperscript{46} Cystic kidney, also known as congenital renal cystic dysplasia of the kidneys, is a category of birth defects in which there are cysts in the kidneys that prevent the neonate’s flow of urine.

\textsuperscript{47} Posterior urethral valves are a birth defect in which obstructive valves develop in the urethra, which end up blocking the flow of urine in the neonate.

\textsuperscript{48} Polydactyly is a birth defect in which there extra fingers and/or toes that develop \textit{in utero}.

\textsuperscript{49} Crude prevalence ratios are the calculated prevalence ratio statistics in a model that does not adjust for other factors.

\textsuperscript{50} Census tract is a term that is used by the U.S. Census Bureau for analyzing populations. A census tract is an area that is approximately equivalent to one neighborhood.
healthcare access/medical care, nutritional risk factors, and/or other environmental influences.

They have also indicated that this disparity could be the result of differences in rates of birth defect diagnoses, potentially because of a lack of access to healthcare providers (who are required to make the diagnosis) or because of differential rates of abortion. However, at the end of this statement, they still indicated that differential “genotypical profiles” – specifically among the different racial/ethnic groups – could be a valid source of these birth defects (Kucik et al., 2012, p. 60). The abstract for the article also stated that disparities in the prevalence of birth defects could be the result of different “genetic susceptibilities” within certain populations (Kucik et al., 2012, p. 52). Despite going further than their predecessors, Kucik et al. (2012) still considered race-specific genetics as a potential rationale for the increased rates of birth defects among certain racial/ethnic minorities, which ultimately perpetuates the biological basis of race as a ‘valid’ scientific theory that can be used to write off health disparities.

While all of the studies highlighted thus far have demonstrated a link between increased rates of individual birth defects and minority racial/ethnic identity, some studies have found a lower rate of overall birth defects in non-white populations as compared to white populations. For example, Kucik et al. (2012) found that infants born to non-Hispanic Black women had a 0.85 fold lower prevalence of being born with any birth defect in comparison to infants born to non-Hispanic white women (p<0.05). Similarly, infants born to Hispanic women had a 0.86 fold lower prevalence of being born with any birth defect in comparison to infants born to non-Hispanic white women (p<0.05).

Similar results were also found in a study by Ibrahim et al. (2014) that examined the relationship between race/ethnicity and birth defects among infants born between 2006-2008 in
Louisiana. Specifically, Ibrahim et al. (2014) found that, in the total study population, the “distribution of [birth defects in] non-Hispanic white and non-Hispanic black children was 57.1% and 42.9%” respectively (col. 1). This trend was further demonstrated in a study by Egbe (2015) that analyzed the relationship between birth defects and race/ethnicity through records of live births from the 2008 Nationwide Inpatient Sample database. The study found that the relative risk of birth defects was slightly lower in African-American (RR=0.9, p<0.001) and Hispanic (RR=0.9, p<0.001) populations as compared to Caucasian populations. In addition, Asian populations were found to have the same relative risk for birth defects as populations. Despite these variable relative risk ratios, however, Egbe (2015) showed that the birth defect prevalence rates within each racial/ethnic group were not significantly different from one another. Specifically, the study indicated the birth defect prevalence per 1000 live births among African-Americans, Hispanics, Asians, and Caucasians was 27.8, 28.3, 30.1, and 29.8, respectively (p>0.05).

Thus, while women of color may have a higher risk for individual birth defects, such as neural tube defects in Hispanic populations, these studies have found the overall risk for birth defects to be about 10-15% lower among racial/ethnic minorities in comparison to white populations. There could be many reasons for this pattern. One possibility is that these data could indicate that racial/ethnic stratification is not the real driving factor behind birth defects, which

51 Ayn Ibrahim, MS, is a public health specialist with a focus on improving health for women and their families through education and advocacy.

52 Alexander Egbe, MBBS, MPH is a congenital cardiologist with an interest in the clinical outcomes for those born with congenital heard disease. He is an Associate Professor of Medicine at Mayo Clinic.

53 This p-value indicates a lack of statistical significance.
warrants further investigation into rates of overall birth defects and rates of specific types of birth defects among people of color and other marginalized populations.

Interestingly, Kucik et al. (2012), Ibrahim et al. (2014), and Egbe (2015) all also hypothesized that genetic abnormalities within certain racial groups could contribute to the racial/ethnic disparities that they found. As mentioned previously, Kucik et al. (2012) suggested “genetic susceptibilities” within certain populations as an explanation for racial disparities in their abstract (p. 52). Similarly, in their article, Ibrahim et al. (2014) emphasized that “genetic susceptibilities” should be explored alongside environmental factors as a rationale for any birth defects, including those specific defects that are higher in racial/ethnic minorities (col. 2). Egbe (2015) similarly mentioned that race/ethnicity could “serve as a surrogate for a variety of potential exposures (e.g., socioeconomic level, nutrition, stress, access to medical care, and migration decisions)” but that “genetic susceptibilities” should be one of the key factors to consider in order to better understand differences in health among various racial/ethnic population (p. 188).

Some of the more recent studies published in the scientific literature on the relationship between birth defects and race/ethnicity in the U.S have had a particular focus on the rates of birth defects among Native and Indigenous populations. For example, a study by Aggarwal et al. (2015) used the California Birth Defects monitoring program to analyze all live births and fetal deaths from 1983 to 2010 among American-Indian women and non-Hispanic women. The study investigators found that there were several birth defects significantly more prevalent among the offspring of American Indian women in comparison to non-Hispanic white women in

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54 Deepa Aggarwal, PhD received her academic training in statistics and works as a research scientist with the California Birth Defects Monitoring Program at the California Department of Public Health.
California. These included (p<0.00555): reduction deformities of the brain56 (PR57=1.85), anomalies of the anterior segments58 (PR=1.71), specified anomalies of the ear59 (PR=1.26), ostium secundum type atrial septal defect60 (PR=1.37), specified anomalies of the heart61 (PR=1.48), anomalies of the aorta62 (PR=1.63), anomalies of the great veins63 (PR=1.9), and cleft lip with cleft palate (PR=1.58) Meanwhile, there was only one birth defect group for which the offspring of American-Indian women had a significantly lower prevalence of: hypospadias (PR=0.78, p<0.05). Overall, these data demonstrated that “American-Indian women giving birth in California are at lower risk for hypospadias but at a higher risk for eight groups of birth defects of which half of them are related to heart defects” (Aggarwal et al., 2015, p. 109).

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55 This p-value is substantially lower than 0.05, which indicates a high level of statistical significance.

56 Reduction deformities of the brain is a category of birth defects that is defined by the absence of parts of the brain. An example of a specific birth defects in this category is agenesis corpus callosum, which is the complete or partial absence of the corpus callosum, a bundle of nerve fibers that connects the two cerebral hemispheres.

57 PR stands for prevalence ratio. In this case a PR of 1.85 indicates that infants born to American Indian women in California are 1.85 times as likely to have reduction deformities of the brain as compared to infants born to non-Hispanic white women in California.

58 Anomalies of the anterior segments is a category that refers to birth defects in the cornea, iris, lens, and the aqueous humor of the eye. One example of an anomaly of the anterior segments is Peter’s anomaly, which is characterizing by the thinning of the cornea, attachment of the iris to the cornea, and other lens abnormalities.

59 Specified anomalies of the ear include birth defects such as microtia or anotia.

60 An ostium secundum type atrial septal defect is a type of atrial septal defect in which there is a hole in the middle of the wall between the two upper chambers of the heart.

61 Specified anomalies of the heart are birth defects that involve structural abnormalities within the heart itself. An example of this includes dextrocardia, in which the heart faces towards the right part of the body instead of towards the left.

62 Congenital anomalies of the aorta involving the main artery of the body (the aorta) that supplies oxygen rich blood to the body. An example includes an interrupted aortic arch, a birth defect in which the aorta lies over a ventricular septal defect as opposed to its normal position over the left ventricle.

63 Anomalies of the great veins refer to birth defects involving the aorta, the pulmonary artery, and major intrathoracic vessels. An example is total anomalous pulmonary venous return, a birth defect in which the pulmonary veins do not connect to the left atrium, preventing oxygen-rich blood from returning to the left side of the heart as it should.
Unlike every other study examined thus far, Aggarwal et al. (2015) did not attribute this trend in birth defects, even in part, to being the result of genetic or biological abnormalities within certain racial/ethnic groups. Instead, they noted some of the major limitations of using race/ethnic categories in the first place, namely that it is self-reported and not reliably related to genetic ancestry whatsoever. Specifically, they wrote that many previous studies had “suggested questionable reportability and validity specifically for American-Indian race classification” (Aggarwal et al., 2015, p. 109). While this statement is perhaps not as explicit as it could have been, this is the first study that has indicated that race/ethnicity is not a valid explanatory variable in and of itself because it does not correlate to actual biological classification. Aggarwal et al. (2015) further noted that the trends they found were not necessarily generalizable to American-Indian populations outside of California because of the heterogeneity of American-Indian populations, hinting at the scientific reality of vast (genetic) heterogeneity within racial categories in general. While not outrightly said, all of this can be taken as a rejection of the biological basis of race, which is novel in comparison to many of the previous studies looking at the relationship between rates of birth defects and race/ethnicity.

In a more expansive retrospective study, Marengo et al. (2018) also looked at the rates of selected birth defects within American Indian/Alaska Native newborns by using data from 1999 to 2007 from 12 population-based birth defects surveillance programs across the United States. Unlike Aggarwal et al., these data expanded beyond California to look at nationwide populations. In looking at the relationship between race/ethnicity and birth defects, Marengo et al. (2018) also accounted for the confounding variables of maternal age, maternal education level, maternal diabetes, maternal smoking, and the type of case-finding surveillance program in

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64 Lisa Marengo, MS received her academic training in Microbiology and has focused on public health research throughout her career. She is an epidemiologist at the Texas Department of State Health Services.
their analysis. The study found that, after adjusting for these factors, only the following 2 birth defects were significantly more prevalent in non-Hispanic American-Indian populations as compared to non-Hispanic white populations (p<0.005): anotia/microtia (aPR=2.72) and cleft lip with or without cleft palate (aPR=1.69). This is far fewer than the number of defects associated with increased risk in Native/Indigenous populations in the study by Aggarwal et al. (2015). While there could be many reasons for this (such as an increased sample size and/or greater geographical variation), these data could indicate that external factors such as teratogenic influence (as measured in the variables for maternal diabetes and maternal smoking) may be behind many of the birth defects that are seen at higher rates in racial/ethnic minorities. However, the study by Marengo et al. (2018) still identified 2 birth defects that were significantly more prevalent in American-Indian populations, indicating that there is still some other root factor that is not being accounted for – that race is acting as a proxy for – in these studies. Like Aggarwal et al. (2015), this study also did not resort to explanations on the biological basis of race when deconstructing this trend. Instead of relying on false understandings of homogenous racial categories, Marengo et al. (2018) actually emphasized the heterogeneity of Native populations, writing that “the AI/AN population in the United States is diverse culturally, genetically, and socioeconomically” (p. 6).

Similar to Aggarwal et al. (2015) and Marengo et al. (2018), a recent study by Le et al. (2019) also looked at birth defect trends in Native populations, though they did also add in an

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65 Teratogens are agents or factors, typically of environmental origin, that result in the maldevelopment of an embryo or fetus in utero. There are many types of teratogens, though they typically fall into the following categories: infectious pathogens, maternal illnesses, maternal nutritional deficiencies, drug use, and/or exposure to environmental pollutants.

66 Mimi T. Le is a research coordinator with the Birth Defects Epidemiology and Surveillance Branch at the Texas Department of State Health Services.
analysis of Asian/Pacific Islander populations in their work. Le et al. (2019) used records from the Texas Birth Defect Registry from 1999 to 2015 to analyze birth defect trends among infants born to non-Hispanic white women as compared to American Indian/Alaska Native women and non-Hispanic Asian/Pacific Islander women. The results showed that, after adjusting for maternal age, infants born to American Indian/Alaska Native women had a significantly higher likelihood of the following birth defects in comparison to non-Hispanic white women (p<0.05): holoprosencephaly\(^{67}\) (aPR=3.05), anotia/microtia (aPR=3.27), cleft lip with cleft palate (aPR=2.68), esophageal atresia/tracheoesophageal fistula\(^{68}\) (aPR=1.36), biliary atresia\(^{69}\) (aPR=4.36), and talipes equinovarus\(^{70}\) (aPR=1.48). Two of these (anotia/microtia and cleft lip with cleft palate) were also found to be significantly higher in American Indian/Alaska Native populations in the comprehensive study by Marengo et al. (2018) that looked at data from across the U.S. and adjusted for several socioeconomic and environmental variables.

In alignment with the results from Aggarwal et al. (2015), Le et al. (2019) also found that infants born to American Indian/Alaska Native women had a significantly lower rate of only one individual birth defect in relation to their white counterparts (p<0.05): hypospadias (aPR=0.64). Looking at non-Hispanic Asian/Pacific Islanders, infants born to women of this population had significantly higher rates of only three individual birth defects in comparison to non-Hispanic white women (p<0.05): anotia/microtia (aPR=1.19), total anomalous pulmonary venous

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\(^{67}\)Holoprosencephaly occurs when the forebrain – a part of the brain that includes the cerebral cortex – fails to develop normally.

\(^{68}\)Esophageal atresia/tracheoesophageal fistula occurs when the esophagus (the tube that connects the mouth to the stomach) and the trachea (the windpipe) fail to separate from one another during development.

\(^{69}\)Biliary atresia is a defect in which the bile ducts inside or outside the liver fail to develop typically, blocking bile flow from the liver to the gallbladder and causing liver damage.

\(^{70}\)Talipes equinovarus, also known as clubfoot, is a birth defect in which the offspring’s foot appears to be rotated inward.
connection (aPR=1.36), and biliary atresia (aPR=2.50). On the other hand, infants born to non-Hispanic Asian/Pacific Islander women had significantly lower rates of several birth defects (p<0.05): spina bifida without anencephaly (aPR=0.38), anophthalmia/microphthalmia\(^71\) (aPR=0.69), congenital cataracts (aPR=0.63), truncus arteriosus\(^73\) (aPR=0.58), ventricular septal defect\(^74\) (aPR=0.84), pulmonary valve atresia (aPR=0.73), aortic valve stenosis\(^75\) (aPR=0.54), and hypoplastic left heart syndrome\(^76\) (aPR=0.46).

Out of the studies examined thus far, Le et al. (2019) was the first one to have expanded the category of Asians to also include Pacific Islanders (though the previously mentioned study by Forrester and Mertz (2004) did analyze Native Hawaiian populations individually). Interestingly, Le et al. (2019) noted that they chose to focus on these particular populations (American Indian/Alaska Native and Asian/Pacific Islander women) because these groups have historically been left out of the conversation. Specifically, they wrote that:

Asian/Pacific Islanders (PIs) and American Indians/Alaska Natives (A.I.s/ANs) generally make up a small proportion of the population in comparison to other racial/ethnic groups, making it difficult to conduct meaningful subgroup analyses. Researchers have frequently ignored these groups or lumped them into an “Other” category to increase sample size and power. When this is done, however, important differences with respect to culture, environment, and genetic makeup are missed. (Le et al., 2019, p. 1381)

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\(^71\) Total anomalous pulmonary venous return is a birth defect in which the pulmonary veins do not connect to the left atrium, preventing oxygen-rich blood from returning to the left side of the heart as it should.

\(^72\) Anophthalmia is a birth defect in which an infant is born without one or both eyes. Microphthalmia is when one or both eyes did not fully develop.

\(^73\) Truncus arteriosus is a birth defect of the heart in which, instead of the aorta and the pulmonary artery, there is only one blood vessel that comes out of the right and left cardiac ventricles.

\(^74\) A ventricular septal defect is a birth defect in which a hole forms between the two ventricles of the heart.

\(^75\) As a birth defect, aortic valve stenosis is a congenitally present obstruction within the aorta that blocks blood flow from the heart to the rest of the body.

\(^76\) Hypoplastic left heart syndrome is a birth defect in which the left ventricle of the heart is underdeveloped.
While it may appear that Le et al. (2019) aimed to correct for the historical marginalization of these groups in the conversation around birth defects, ultimately, they ended up adding to this racial oppression by validating the biological basis of race. This is demonstrated in the last sentence of the previous quotation, where they indicated that there are unique elements of “genetic makeup” within these racial/ethnic minority groups must be examined to ‘justly’ understanding this health disparity (Le et al., 2019, p. 1381). Once again, this is a clear conflation of genetic ancestry and racial/ethnic identity. Ironically, Le et al. (2019) acknowledged this conflation themselves, though they did not problematize it. Regarding the role of genetics in this conversation, they stated that:

In addition to cultural and environmental factors, genetics also play a role in birth defect outcomes. Research on birth defects and genetics, however, is incomplete due to its complex nature. Various studies have shown that individuals with similar biogeographical ancestry, which can be categorized broadly by race/ethnicity [emphasis added], have a similar genetic makeup. Therefore, it is important to examine specific groupings rather than the more heterogeneous “Other” category, to fully elucidate these differences. (Le et al., 2019, p. 1381)

What Le et al. failed to recognize, however, is that just like the supposed “heterogeneous ‘Other’ category” that they mention, those that they included within the racial groupings of American Indian/Alaska Native and Asian/Pacific Islander are themselves extremely heterogeneous (Le et al., 2019, p. 1381). For example, within the category of Asian/Pacific Islander alone, Le et al. have included people who have ancestral origins in four large subregions of the eastern hemisphere: the Far East, Southeast Asia, South Asia, and the Pacific Islands. These many geographical regions correlate to large numbers of ancestral genetic groups. Because these many diverse groups have been simplified into an assumed-homogenous category of Asians/Pacific Islanders, there is little to no actual biological meaning that can accurately be extrapolated out of this racial category.
Overall, out of the 10 studies that were examined in this literature review, 2 did not suggest any rationale for the increased rates of individual birth defects among women of color, which, as previously mentioned, is problematic because it could indicate to the reader that race should be interpreted as an explanatory variable for this health disparity. Out of the other 8 studies, 6, at least in part, relied on the notion of biological race when presenting hypotheses as to why this trend was occurring. While these 6 studies did indicate the possibility of other factors contributing to the increased rates of birth defects among populations of color, including socioeconomic and environmental factors, all 6 still continued to emphasize genetic susceptibilities within racial/ethnic categories as a key source for this disparity. Only 2 of 10 studies did not reify on the biological basis of race through their analysis of birth defects, namely Aggarwal et al. (2015) and Marengo et al. (2018). Instead, these 2 studies problematized race as an inconsistent variable that does not have meaningful biological implications. It

**What are the effects of using race as a risk factor for birth defects?**

There are many reasons why the hypothesis that biological differences are behind racial health disparities in birth defects is a manifestation of bad science. As discussed in chapter one, the implication that race – a category that relies on self-identification and does not carry a singular or stagnant definition – can tell us anything definitive about our ancestral inheritance patterns or genetic lineages is simply inaccurate. Many of the problems with using race as a proxy for population-level genetics based on migration patterns from early human evolution have been highlighted in an article by Hammerschmidt (1999). Specifically, Hammerschmidt (1999) has explained that racial categories have fluid meanings that can correlate to extreme variations in individual geographical ancestry and origin, writing that:
racial assignment in papers in the medical literature is made into one of several very broad categories, each including people of extraordinary genetic diversity. “Black” subjects, for example, may include slave descendents [sic] whose ancestors were from West Africa, but it also may include recent immigrants from South Asia, the horn of Africa, South Africa, certain Pacific islands, and even Australia. The genetic disparity between some of these groups (as estimated from a number of DNA homology studies) is as great as the disparity between some groups of “blacks” and “whites.” To draw an analogy to statistical analysis, the intragroup variance may often be sufficient to explain (or even be as great as) the intergroup variance. And of course, the ancestry of “black” people may be quite different in different places, depending on the immigration (and unfortunately, enslavement) history of each locale. A black/white comparison in one urban center or neighborhood might be largely a comparison of slave descendants against people of British heritage; in another location, it might be largely a comparison of recent Somali and Ethiopian immigrants against people of Slavic descent. (pp. 10-11)

Essentially, today’s socially constructed racial categories are extremely heterogeneous and made up of individuals who draw their geographical ancestry from many parts of the world. Trying to correlate this varied geographical ancestry with genetic ancestry may work on an individual level, but not by homogenizing an entire population through the lens of race. Because of this heterogeneity, race cannot be used as a variable\textsuperscript{77} that in and of itself explains biological outcomes, which is precisely what the existing literature on birth defects, at least in a majority of cases, has tried to do.

Beyond the pure scientific inaccuracy of using biological race to explain current birth defect trends, these hypotheses are harmful because they perpetuate the very disparities they are trying to explain – a major concern that has often been left out of the discussion around race-based medicine thus far. In this case, by attributing the cause of birth defects to being related to race-specific genetic anomalies, the institution of science perpetuates these disparities by distancing itself from having to take action on this inequity in two ways. First, because genetic

\textsuperscript{77} This does not necessarily mean that race is an entirely useless category or that it should never be used as a variable in scientific research. Ignoring the variable of race results in a colorblind approach, which can also further perpetuate systemic racial inequity. However, researchers must be aware of how using an identity as a variable is a implication of many social phenomena and should consider this more heavily in their work.
abnormalities are extremely difficult to address with the scientific options we currently have, labeling racially disparate rates of birth defects as ‘genetic issues’ excuses science from having to provide a solution. Currently, we don’t have great treatment or prevention options for genetic mutations. So, if these trends in birth defects are labeled as having their origins purely in genetics, scientific action to ‘fix’ this racial disparity is not necessitated, at least until scientific breakthroughs in gene therapy are made.

This ideology has already started to manifest within the scientific literature. For example, when analyzing racial/ethnic disparities in relation to birth defects, an article by Bryant et al. (2010) stated that “some disparities may have their origins in biology and therefore may not be modifiable in the short term” (p. 340). Ultimately, through the reification of biological race, the institution of science is able to dismiss this health disparity as a non-issue – because even if it exists, it cannot be solved. It excuses action on behalf of the scientific community, which further marginalizes people of color from receiving equitable health care.

Second, if this birth defect trend is characterized as being the result of genetic anomalies, it excuses scientists from having to look at other external causes, including causes related to systemic racism. For example, we know that people of color face increased levels of poverty, decreased quality and access to healthcare, and decreased quality and access to food, all of which could (and likely does) contribute to increased rates of adverse pregnancy outcomes like birth defects. Additionally, we know that environmental racism exists, which makes “poor communities and communities of color particularly vulnerable to environmental degradation,

78 While the article by Bryant et al. (2010) reviewed many types of disparities in obstetrical outcomes and care, they specifically indicated that congenital abnormalities likely have a biological component by referencing differences “in carrier frequencies of genetic polymorphisms associated with folate metabolism” (p. 337). However, it is important to note that these race-specific polymorphisms have been heavily inconsistent in the literature and have been associated with many (white and nonwhite) racial/ethnic groups.
exposure to toxicity, and the resulting health risks” – one such risk being fetal maldevelopment (Berila, 2006, p. 93). These should be the primary sources of investigation when thinking about the increased prevalence of birth defects within racial/ethnic minorities. However, because race-based genetics have been emphasized within the scientific literature, it has falsely propagated the idea that race (specifically non-whiteness) is the problem rather than racism.

As an example of this, an article by Thompson and Suter (2020) has fallen into this exact trap, where they have made the assumption that race in and of itself is the reason that high rates of adverse birth outcomes, including increased prevalence of birth defects, exist among racial/ethnic minorities. In fact, Thompson and Suter (2020) have gone as far as to suggest that future research on racial disparities only consider factors that “cause race” as opposed to socioeconomic (or other) co-mediators of race (p. 5). They have written that:

Much of the existing literature that describes racial disparities adjusts the disparity risk estimate for covariates like socioeconomic status (SES).…The rationale for conditioning on covariates has not always been clear. Following a unifying and serviceable definition for confounding, *there are no backdoor paths between race and any of the outcomes in the current study because none of the factors can “cause” race* [emphasis added]. Backdoor paths for the more abstract construct “racism” can be envisioned and confounding control defended in those models but controlling for confounding by racism was not an objective of the study and should not be performed for descriptions of potential racial disparities. For the *unchangeable factor of race* [emphasis added], any covariates used for conditioning should be considered potential mediators and it is the effect of the mediator in changing the risk estimate for each race that is of interest, not just the resultant conditional risk. (Thompson & Suter, 2020, p. 5)

By placing the onus of health disparities on aspects of biology that are considered ‘unchangeable,’ it is easier for science to forgo its role in trying to identify and address those structural inequities – both within science and outside of it – that are causing adverse effects on the health of people of color. Additionally, it breeds compliance with systemic racism, causing

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79 Because nothing can actually cause race (as per an understanding of race as socially constructed), it is implicit in this context that Thompson and Suter (2020) are operating under a definition of biological race, where they believe that there are biological factors (such as genetic variations or certain alleles) that *cause* race.
people of color continue to face existing health disparities as well as the other effects of structural inequity. Root causes of inequity are allowed to continue because biological race masquerades their impact upon people of color. Thus, the institution of science is able to use biological race (a theory that is itself racist) to ignore the realities of systemic racism, which only furthers the oppression of people of color, and especially in this case, women of color.

Ultimately, in these two ways, the institution of science perpetuates the increased rates of birth defects among racial/ethnic minorities. The overall effect of using biological race as an explanation for the increased rates of birth defects is that this disparity continues to exist: women of color continue to have higher rates of infants born with birth defects. While this is not a form of forced sterilization upon women of color or a direct denial of medical care to children of color as was seen in the eugenics era, using the biological basis of race as a justification for birth defects deprives both mother and child of the right to accurate, socially-cognizant reproductive healthcare today – and this is also reproductive injustice.

Previously, in chapter one, three key tenets of reproductive justice were defined: 1) the right to not have children, 2) the right to have children, and 3) the right to parent children one has in “safe and healthy environments” (Ross et al., 2017, p. 14). I believe that, within this definition, the second and third tenets also indicate that, all people should have the right to birth and raise healthy children, or children who have the opportunity to develop prenatally and postnatally in a healthy environment. We must consider how factors that specifically affect women of color – namely nonbiologic factors stemming from as systemic inequity – negatively influence both the health of the woman and the health of her future offspring. As Loretta Ross (2017b) has explained, in order to “explore the potential of reproductive justice today,” we must consider factors such as:
freedom of movement, immigration restrictions, the prison-industrial complex, racial and gender binaries, racial profiling and police brutality, racist and sexist media portrayals, resource allocations through tax policies, welfare and public assistance, health care systems, housing availability, food insecurity, lack of educational opportunities, zoning regulations, internal displacement through natural disasters or eminent domain, voting rights, religious bigotry, credit and finance regulations, restrictions on civil liberties, and environmental racism. (para. 7)

In considering these other factors, we can gain a larger picture as to how reproductive injustice actually manifests within the lives of all people, especially people of color, today.

Within the context of the biological basis of race and using this framework of reproductive justice, we can recognize how in perpetuating the disparate rates of birth defects faced by women of color, the institution of science is simultaneously perpetuating a reproductive injustice. This reproductive injustice specifically manifests in two ways. First, because ascribing this as a problem of genetics excuses science from having to find a solution, there is a lack of investigation into adequate treatment options that could alleviate the severity of birth defects within racial/ethnic minorities. If this disparity is considered the result of inherent, unchangeable race-based genetic mutation (as it currently is), then solutions may be hard to find. However, if non-whiteness is no longer seen as a source of natural degeneracy and other causes are investigated, it increases the necessity and the possibility of identifying treatment options that can mitigate the biological outcomes stemming from whatever is causing this disparity. Further treatment options can also be investigated to improve the quality of life for infants of color diagnosed with birth defects.

Second, because science is using biological race to distract from investigating other factors, namely factors that have roots in systemic racism, there is a lack of investigation into improved preventative care for women of color facing the effects of structural inequity. We know that despite race being socially constructed, systemic racism affects biological processes,
whether it be through increased stress, lack of adequate nutrition, increased exposure to toxins, etc., all of which can also affect the intrauterine environment. By failing to consider the profound effects of systemic racism on health, including reproductive health, women of color are denied the right to have a child who is able to develop – prenatally in this case – in a healthy environment. If we wish to prevent the effects of systemic racism, we must recognize racism as a threat to a healthy environment and work to address this problem. Overall, women of color deserve to have effective treatment options that allow their offspring to thrive as well as preventative options that affirm a healthy environment in which their offspring can develop. These are key elements of reproductive justice that must be emphasized when working to alleviate this health disparity in the future.

Adequate healthcare to both treat and prevent birth defects, especially in racial/ethnic minorities, is of particular importance considering that birth defects are the leading cause of infant mortality in the United States. On average, in the United States, birth defects affect 3.03% of babies but account for 20.6% of all infant deaths yearly (Almli et al., 2020; CDC, 2020a). These effects are often more severe in racial/ethnic minority groups as well. According to a study by Wang et al. (2015), after analyzing records from 12 different birth defect tracking programs of infants born between 1999 to 2007, it was found that the offspring of women belonging to racial/ethnic minorities had an increased risk of mortality for most of the major birth defects studied in all age groups. For younger children, the study found that “post-neonatal infant (28 days to <1 year) mortality risk was significantly (p<0.05) greater among children born to non-Hispanic black mothers for 13 of 21 defects analyzed (hazard ratios [H.R.s] 1.3-2.8) and among

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80 This must be framed as a call to action to address systemic racism and not as a way to further blame women of color for ‘subjecting’ their offspring to systemic racism. The ongoing effects of systemic racism should not limit the right of women of color to have children, but it does demand action to address the sources of these inequity.
children born to Hispanic mothers for 10 of 21 defects (HRs 1.3-1.7)” as compared to children born to non-Hispanic white mothers (Wang et al., 2015, p. 819). For older children, the study also showed that there was a significantly (p<0.05) increased risk for childhood (≤8 years) mortality among infants born to “Asian/Pacific Islander mothers for encephalocele (HR 2.6), tetralogy of Fallot, and atrioventricular septal defect (HRs 1.6-1.8) and among children born to American Indian/Alaska Native mothers for encephalocele (HR 2.8)” (Wang et al., 2015, p. 819). Not only are infants born to women of color facing increased rates of birth defects, but they are also facing increased rates of severe consequences from these birth defects, including those that result in death.

This means that infants born to women of color are surviving at lower rates because of this health disparity – a disparity that we do not know the exact roots of because it hasn’t been investigated due to the heavy emphasis in the scientific literature on the hypothesis of race-based genetics. From a reproductive justice lens, women of color deserve to have both treatment and prevention options for alleviating this burden in order to affirm their right to have a healthy child who does not face undue mortality risk as the result of birth defects. This work is only possible once we reject the biological basis of race as a legitimate theory and no longer use it as an explanation for racial health disparities today.
CHAPTER FOUR

MANIFESTING REPRODUCTIVE JUSTICE IN MATERNAL-FETAL MEDICINE

As chapter three discussed, the existing scientific literature around birth defects has utilized a hypothesis reliant on the biological basis of race in order to explain why certain birth defects are seen at higher rates in racial/ethnic minority groups. Many of the problems that stem from using biological race as an explanation for health disparities have been highlighted in the previous chapters, including: how race is an inaccurate proxy for ancestral genetics, how naming race-based genetics distances science from having to find a solution, and how race-based medicine masquerades the effects of systemic racism upon the health of people of color. In all of these ways, using the biological basis as a false explanation of health disparities ultimately allows for the disparity to progress, resulting in women of color (and their offspring) continuing to have poorer health than their white counterparts. Because birth defects are associated with adverse pregnancy outcomes, including high rates of infant mortality, this health disparity is a manifestation of reproductive injustice that prevents women of color (as well as other marginalized populations at higher risk for birth defects) from birthing healthy babies.

While it is important to recognize the perpetuation of this health disparity as a manifestation of reproductive injustice, what does it mean to use the reproductive justice framework to actually address this health disparity in a way that does not reify the biological basis of race? This is the question that this chapter will seek to investigate, specifically by focusing on how reproductive justice allows us to center women of color, embrace an
intersectional approach, and meaningfully address difference within the context of birth defects among racial/ethnic minority groups.

**The Reproductive Justice Framework**

Earlier chapters have mentioned the three vital human rights values of the reproductive justice framework: “the right not to have children using safe birth control, abortion, or abstinence; the right to have children under the conditions we choose; and the right to parent the children we have in safe and healthy environments” (Ross et al., 2017, p. 14). As previously discussed, fundamental to this definition is the right to have healthy children – or children who are able to develop prenatally and postnatally in environments that are free from harmful effects, including the harmful effects of systemic racism. This connection between reproductive justice and anti-racism is not incidental but rather quite intentional. As Loretta Ross (2017a) has stated in her article “Conceptualizing Reproductive Theory: A Manifesto for Activism,” “analyzing white supremacy is a cornerstone of reproductive justice. Failures to criticize it produce sterile theories and practices that are, in fact, complicit with white supremacy by airbrushing it to soften its lethality” (p. 173).

The “sterile theories” and practices that Ross (2017a) has spoken of include those that were found in earlier waves of the feminist movement in the United States, especially those that were used to propagate the biological basis of race and the racial inferiority of women of color in order to achieve increased liberation for white women. What makes the reproductive justice framework different from these earlier versions of “feminism-lite,”¹ white feminism, or even a

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¹ Feminism-lite is a phrase utilized by Chimamanda Ngozi Adichie in her book *Dear Ijeawele*. Adichie (2017) has written that this kind of feminism is the “idea of conditional female equality” which manifests as a “hollow, appeasing, and bankrupt” understanding of gender roles, identity, and behavior (para. 2).
reproductive rights\textsuperscript{2} approach is that it is firmly rooted in anti-racist work. It has been created \textit{by} women of color and \textit{for} women of color (as well as for all those who have been oppressed by white supremacy) as a way “to reimagine what reproductive freedom could look like for all people” (Ross, 2017a, p. 178). Applying these ideas to the case of increased rates of birth defects among racial/ethnic minorities, I find that there are three key ways in which a reproductive justice framework helps us intentionally be anti-racist in our work to better understand and eventually alleviate this health disparity so as to affirm the right for all populations to have healthy children. I believe that these three pillars – centering women of color, embracing intersectionality, and meaningfully addressing difference – provide a starting point for reproductive justice work in maternal-fetal medicine, especially when working to deconstruct health disparities such as the increased rates of birth defects among infants born to women of color.

\textbf{Pillar One: Centering Women of Color in Reproductive Justice}

To bring a social-justice lens to maternal-fetal health, especially when deconstructing racial health disparities, it is necessary that our work intentionally centers on women of color. While this focus is particularly applicable to birth defects and other pregnancy-related inequities, it also broadly applies to any sort of human rights work that we wish to do in the field of maternal-fetal medicine. As Beverly Yuen Thompson (2017) has written in her article “Centering Reproductive Justice: Transitioning from Abortion Rights to Social Justice,” a reproductive justice framework emphasizes focusing on women of color because, “by

\textsuperscript{2} The reproductive rights movement has been predominantly focused on “achieving women’s individualistic reproductive freedom through the legal system and has historically focused on the pro-choice and pro-life debate, sex education, and family planning” (Paciu, 2020, para. 5). However, the reproductive rights approach has been critiqued for ignoring and/or failing to address the issues of women of color, for whom a systemic deconstruction of social, political, and economic inequity must accompany any discussion on reproduction in order to affirm reproductive choice in an intersectional way.
defending the human rights of those in the most vulnerable position, the movement will ensure
the widest possible social benefit, rather than emphasizing the needs of those with the most
resources” (p. 269). Women of color, who face the double bind of racism and sexism and are
more likely to face class oppression as well, are not only the ones who are most affected by this
health disparity, but they are also the ones who have been historically left out of the medical
field, in terms of both clinical care and consensual, justice-oriented research. To center women
of color in maternal-fetal medicine goes beyond including them in research studies or clinical
trials as participants. Instead, this work mandates community-oriented processes that are focused
on building equitable collaborations and work towards the flourishing of women of color.
Essentially, women of color must be allowed the opportunity to drive the research we are doing
around birth defects; they must be involved in asking research questions, identifying elements
that could contribute to increased prenatal risk, and creating and implementing interventions to
reduce prenatal risk.

Behind this model is not only centering women of color but truly learning to care for
them as valued human beings. This model of care exists in direct opposition to the
institutionalized racism within medicine that has marginalized women of color for so long, which
is what Dána-Ain Davis (2019) has discussed in her book Reproductive Injustice: Racism,
Pregnancy, and Premature Birth. Citing a quotation from a radical birth worker and then
elaborating on the issue, Davis (2019) has written that:

“When we think about medical care and race, we have to think about the relationship to
the profit-driven medical industry. We have to think about the construct of race within a
medical industry that is rooted in the preservation of cis, white, male, straight, able-
bodied Christian concepts of wellness and existence.” Black women and people lose
because the medical and caring economies have little investment in their well-being,
safety, and value. That is the reality against which reproductive justice advocates fight.
(pp. 188-189)
The lack of care for women of color (and their offspring) is truly a life and death issue that affects generations of people of color. To fight the institutionalization of racism within medicine, including the persistence of the biological basis of race, it is necessary to rehumanize the woman of color – in body, mind, and soul – so that we are fully able to care for her. Within maternal-fetal medicine, this kind of radical care can manifest by starting to see the woman of color beyond a vessel for reproduction and as a whole human being. For example, a women of color-focused approach can start by “looking at the whole life cycle of women [of color] in reproductive age—to make sure they are as healthy as possible” (Davis, 2019, p. 154). If we work to affirm the health of the mother throughout her lifespan, it also improves the health of any of her future offspring.  

3 To think comprehensively about women of color’s health, especially within the context of reproductive justice, means thinking about comorbid conditions, reproductive planning, prenatal care, and more. As Davis (2019) has further explained:

We need to make sure [women of color] are as healthy as possible, and what we’re seeing in our population is that is not happening. There is more obesity, hypertension, diabetes, and higher smoking rates. All of those are conditions that affect women’s health in pregnancy…We need to look at the intentionality of pregnancy. Half the pregnancies are unintended, and it is up 70 percent or more when you get to poor women. So, we really need to factor in reproductive planning for women so that they can become intentional in their life about when they become pregnant—so it doesn’t drive them into poverty. Access is what we need to work on. Prenatal care is an access issue. But there is also the issue of . . . many women not coming for prenatal care. We can unpack all that too, about trust of the health care system. But we also need to make sure that they’re getting all the interventions that we know work. And that’s not happening. We’re not seeing drug and alcohol screens happening. With a history of preeclampsia, we’re not seeing the use

3 This idea is rooted in a reversal of the maternal-fetal conflict, an idea discussed by Dorothy Roberts (2014) in her book *Killing the Black Body*. Roberts (2014) has written that “feminists use the term “maternal-fetal conflict” to describe the way in which law, social policies, and medical practice sometimes treat a pregnant woman’s interests in opposition to those of the fetus she is carrying” (p. 68). By realigning the interests of the mother and the developing offspring, we can promote the health of both in ways that fundamentally affirm the autonomy of the mother.
of aspirin. So, often, the interventions that we know work, women are not getting. (p. 154)

While Davis’ (2019) work has focused on the disparity of premature birth among women of color, this care ethic is a model to preventatively address all health disparities related to reproduction, including the increased rates of birth defects among infants born to women of color. Through this kind of radical care approach, we can shift (at least in part) the burden of racism and its harmful effects from women of color onto the medical industry so that healthcare providers must do more to actively pay attention to the unique struggles by women of color faced as the result of systemic inequity. When we center the care of the most marginalized populations, namely women of color in this case, healthcare providers are forced to be more intentional about and attentive to the quality of care they are providing, the frequency of this care, and the kind of medical advancements they are pursuing in order to counteract the effects of systemic racism on both mother and child.

This kind of intervention-based approach is not where radical (medical) care ends. A radical care approach that aims to address health disparities must also directly target racism. If we know that racism is behind adverse health outcomes (which we do) and behind adverse pregnancy outcomes like birth defects (as we suspect it is), then healthcare providers who are committed to alleviating health disparities in maternal-fetal medicine must also target the existence of structural racism itself. As Davis (2019) has further explained, addressing racism through interventions that mediate or account for its effects is important, but it is a temporary fix to the larger structural issues at play. It doesn’t address the root harm that is systemic racism. As such, Davis (2019) has written that:

Intervening…is not enough to address racism…NICUs do not address racism; charts showing racial disparities do not address racism; health promotion campaigns focused on
patients’ behaviors and habits do not address racism. It is thus fundamentally important that medical and health care professionals who have been trained and work in that system be willing to take responsibility for their own behaviors and biases. They must look racism in the face and question the ways that the system within which they work might contribute to racist outcomes, draw from racist discourse, or perpetuate racist ideas. To move closer to reproductive justice, we must address medical racism. (p. 206)

To center – and care – for women of color is a significant undertaking and one that must take place on both individual and systemic levels in order to make change. We need a cultural shift at both levels in order to improve the health of women of color, including during their reproductive years, so as to affirm their health and the health of their offspring. We need medical interventions that are focused on this aim, but we also need doctors to adopt a care ethic for women of color that prioritizes their well-being from birth to death.

**Pillar Two: Embracing Intersectionality in Reproductive Justice**

Centering women of color in our social justice approach doesn’t transform one element of medicine; it changes everything. Within health disparities research, it changes the goals, the mission, the research questions, the data collection methods, the analysis, the intervention methods, and more. It is a transformational shift that allows for an intersectional understanding of the world. Ross (2017a) has noted this shift, writing that the reproductive justice movement does not just seek to “colorize an existing pro-choice framework by merely adding women of color and stirring, but to shake it up and offer our own radical paradigm that could account for the differential impacts of white supremacy and incorporate intersectionality [emphasis added]” (p. 178). Ross (2017a) has further elaborated to say that, within reproductive justice, “human rights is the goal,” but “intersectionality is the process” used to achieve this goal (p. 174). Intersectionality, therefore, is a crucial part of a reproductive justice approach that must be considered when seeking to bring this framework into the field of maternal-fetal medicine.
Simply defined, intersectionality is a “conceptual tool for analyzing differences” (Bromley, 2012, p. 48). Based on the work of Black feminist scholars and activists like bell hooks, Patricia Hill Collins, and Audre Lorde and officially coined by Kimberlé Crenshaw in 1989, intersectionality is a theoretical approach that emphasizes how multiple, intersecting identities – such as race, gender, sex, class, sexuality, nationality, ability, etc. – differentially oppress and privilege certain individuals and communities as the result of existing systems of power, historically and presently (Bromley, 2012). To clarify, intersectionality is not at odds with centering women of color; rather, it is a method that has arisen out of Black women’s experiences as a way to focus on differential aspects of collective and individual identity.

Using the pillar of intersectionality alongside the pillar of centering women of color prevents us from homogenizing ALL women of color. It allows us to notice the differences in individual experiences as well as the various layers of individual identity that may intersect with one another even as we focus on marginalized communities. Applying this to the case of birth defects among racial/ethnic minorities, an intersectional approach can help us distinguish between different groups of women of color based on their differential experiences of oppression. For example, a Native American woman living on a reservation in rural South Dakota may have different struggles, lifestyles, and challenges than a Black woman living in urban St. Louis, Missouri. Taking into account the specifics of each woman’s identity prevents the same kind of homogenization and essentialization that the concept of biological race has been responsible for. As we focus on the specifics, an intersectional approach can also let us see how other elements of identity and positionality beyond race – such as class, native language, skin color, nationality, and geographical location – all intersect with the social construct of race to
differentially increase or decrease the risk for adverse pregnancy outcomes even within one racial/ethnic category.

According to Patricia Hill Collins and Sirma Bilge (2016), there are two parts of an intersectional approach: intersectionality as critical inquiry and intersectionality as critical praxis. As Hill Collins and Bilge (2016) have written in their foundational feminist text Intersectionality:

Intersectionality as a form of critical inquiry invokes a broad sense of using intersectional frameworks to study a range of social phenomena... across different social contexts, e.g., local, regional, national, and global. Intersectionality as critical praxis does the same, but in ways that explicitly challenge the status quo and aim to transform power relations. (pp. 32-33)

Thus, not only is intersectionality a way to transform how we think/write about these issues in the scientific literature, but it also is a method that necessitates that we use our transformed scholarship as a way to ignite conversations about reproductively just care across the field and implement tools, methods, and resources that work towards this goal. Therefore, intersectionality is both a method for analysis and a demand for action.

In the case of birth defects among racial/ethnic minorities, this means that when we call for the end of false hypotheses that perpetuate the biological basis of race in the scientific literature, we are simultaneously calling for sweeping change in how women of color are cared for within the field of maternal-fetal medicine. This change may start in the scholarship but must go beyond it to change how providers think about birth defects among racial/ethnic minorities as well as how providers act with women of color and their offspring within clinical and research settings. Intersectionality as critical praxis shows us that we are not only calling for an end of the use of outdated ideas but for a complete transformation for how women of color and their infants are treated within medicine. We are calling for prevention, treatment, and a commitment to anti-
racist work to dismantle this manifestation of reproductive injustice as well as to dismantle the structures of systemic racism that have fueled it.

**Pillar 3: Meaningfully Addressing Difference**

Intersectionality helps us broaden our perspective so we can see *difference*, which is a crucial step to challenge how we see social inequity and subsequently how we enact change. To elaborate on this second component of making change, under the reproductive justice framework, we must not only acknowledge difference but meaningfully address it in order to alleviate racial health disparities. To more concretely focus on what it means to address difference, I suggest a framework detailed by Sharlene Nagy Hesse-Biber and Michelle L. Yaiser (2004) in their article “Difference Matters: Studying Across Race, Class, Gender, and Sexuality.” Hesse-Biber and Yaiser (2004) have suggested six themes that should be used in feminist research in order to conceptualize and address factors of identity⁴ to meaningfully acknowledge difference while still promoting equity across these differences. These six themes are: 1) Race, class, gender, and sexuality are contextual and constantly changing; 2) Race, class, gender, and sexuality are socially constructed and must be interpreted as such; 3) Race, class, gender, and sexuality are not only inequalities that are socially constructed and historically specific but are also related to systems of power; 4) Race, class, gender, and sexuality “must be examined in both the social structure (macro) and the social psychological (micro) contexts” (Hesse-Biber & Yaiser, 2004, p. 109); 5) Race, class, gender, and sexuality operate in intersectional, simultaneously occurring ways; and 6) Race, class, gender, and sexuality scholarship emphasizes the interrelatedness of theoretical and practical approaches.

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⁴ While Hesse-Biber and Yaiser (2004) specifically focused on race, class, gender, and sexuality these six themes can and should be extrapolated to understanding any element of socially constructed identity.
By ensuring that all future research questions, analyses, and hypotheses lie in accordance with these principles, we can prevent the recurrence of harmful theories that rely on bio-essentialism from appearing within maternal-fetal medicine, especially as we analyze racial health disparities. Using these six themes as guiding principles for future research can ensure that the work done in maternal-fetal medicine meaningfully investigates how issues of difference are related to the unequal impacts of birth defects that are faced by infants born to women of color. Therefore, if marginalized populations face increased rates of birth defects, a justice-based healthcare system must aim to alleviate these disparities in ways that are meaningful and useful to the populations at hand. This kind of healthcare must acknowledge the way differences have been used to construct social hierarchies and look at the driving factors behind an inequity rather than merely naming it as an issue of inherent ‘difference,’ which is precisely what future work must do in order to correct for the existing hypothesis in the scientific literature around racially disparate rates of birth defects. Ultimately, by using these six themes to guide our work in maternal-fetal research, we can affirm reproductive justice by ensuring that we “avoid a biological essentialism that centers sexual, gender, and racial subordination,” while at the same time working to “actively engage theories of embodiment that account for racial, class, ability, gender identity, and citizenship (among an infinite array of differences)” (Ross, 2017a, p. 193).

**Reproductive Justice in Action**

The three pillars of reproductive justice discussed above (centering women of color, embracing intersectionality, and meaningfully addressing difference) are heavily interconnected with one another and have several places of overlap. They are not the only way to enact reproductive justice nor are they all-encompassing. Despite being broad in nature, these pillars provide a starting place to reflect on how a reproductive justice framework can be used within
maternal-fetal medicine to affirm the right of women of color (and all people really) to have healthy children who are not affected by systemic inequity. While these frameworks may be more prevalent in the humanities field, the reality is that science researchers are already starting to utilize them, even if they don’t use the same terminology.

As one example of this, a study conducted at the University of Oxford by Barbeito-Andrés et al. (2020) sought to analyze why the prevalence of congenital Zika syndrome (CZS), a syndrome found among neonates characterized by developmental and physical birth defects such as microencephaly, was higher amongst women of color living in an economically disadvantaged region of Brazil. The study researchers noted that in Brazil, which has had about 95% of all cases of CZS, 75% of these cases were “found in the disadvantaged socioeconomic region of the Northeast” in Brazil (Barbeito-Andrés et al., 2020, p. 1). However, instead of attributing this disparity to an issue of genetic anomalies on the basis of race, geographical origin, or other another element of identity, Barbeito-Andrés et al. (2020) hypothesized that external “cofactors” such as malnutrition likely played “a key role in modulating ZIKV infection’s severity and the level of developmental impairment” (p. 1).

To test this hypothesis, the study utilized mouse models with pregnant mice who were subjected to protein malnutrition and subsequently infected with Zika virus. Ultimately, the study found that in undernourished pregnant mice, Zika virus presented with higher viral load, where it then resulted in severely altered placental structures as well as restricted embryonic body growth and neurogenesis of the developing offspring. In these mice models, the offspring (who were also born malnourished) were also found to have altered neural gene expression and reduced

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5 Microencephaly is a birth defect characterized by a small head, typically as the result of maldevelopment of the brain. It is commonly associated with Zika virus.
brain size. All of this evidence suggests that, in human beings, “maternal protein malnutrition increases susceptibility to CZS” (Barbeito-Andrés et al., 2020, p. 1).

What makes this approach different from other studies on birth defects among racial/ethnic minorities is not only that the hypothesis did not rely on the biological basis of race (or a bio-essentialist understanding of other elements of identity), though this is an important step forward. However, in addition to a more accurate hypothesis, the whole research process was driven by an approach that was rooted in an understanding of social constructionism and the realities of systemic inequity. From the beginning of the study, the investigators made it clear that their goal was to better understand an inequity that predominantly affected women of color in an economically disadvantaged region. Furthermore, the study itself was conducted after a call for help from members of the Brazilian community, namely after the “Brazilian Ministry of Health declared a Public Health Emergency of National Concern in response to an increased number of microcephaly cases, possibly related to previous Zika virus outbreaks” in 2015 (de Oliveira et al., 2017, p. 861). By seeking to help an affected population that was desperately searching for answers, Barbeito-Andrés et al. (2020) centered their study on the most marginalized, or in this case, poor Brazilian women who were facing increased rates of CZS, which is exactly what pillar one promotes. Concerning pillar two, Barbeito-Andrés et al. (2020) also used an intersectional approach when constructing their hypothesis. They utilized critical analysis to discern that this inequity was differentially affecting specific populations in Brazil and to identify the connections between poverty, class, geographical location, and maternal malnutrition. Building on this, they utilized the third pillar, meaningfully acknowledging difference, to pose a question focused on dissecting the rationale behind this health disparity.
Rather than simply naming it as an issue of ‘difference,’ the study investigators looked for plausible, tangible, and fixable sources of this disparity.

Additionally, their explanation for this disparity—which ended up being linked to increased maternal malnutrition among low-income populations—was not framed in a way that reified the biological basis of race or enforced maternal degeneracy. In fact, Barbeito-Andrés et al. (2020) went so far as to reject the biological basis of race in their discussion, noting that it was not genetic background but environmental factors that played the primary role in this disparity. Specifically, they wrote that:

Recently, it was suggested that fetal genetic background could predispose to malformation development after ZIKV vertical transmission. Beyond genetic predisposition, it was shown that environmental factors, such as the previous exposure to dengue virus, increase susceptibility to ZIKV congenital infection and lead to more exacerbated phenotypic alterations in the brain. (Barbeito-Andrés et al., 2020, p. 6)

In concluding their research, Barbeito-Andrés et al. (2020) stated that future work should continue to investigate external and environmental factors in order to fully understand how birth defects affect all members of the population, especially the most marginalized. They noted that, in order “to deepen current knowledge on the emergence of CZS, it is essential to understand the cofactors modulating previous outbreaks, thus improving preventive measurements facing future epidemics” (Barbeito-Andrés et al., 2020, p. 8). While this is perhaps not the most active form of critical praxis, as per an intersectional approach, this work still challenged the status quo within the institution of science to encourage more research to preventatively address adverse pregnancy outcomes now and for the future.

In all of these ways, we can see how Barbeito-Andrés et al. (2020) utilized key pillars of a reproductive justice framework, even if they didn’t use that exact language. Ultimately, their research worked toward affirming the right for everyone, especially marginalized populations, to
have a healthy child who is not affected by systemic patterns of inequity – and this is what reproductive justice is about.

**Key Lessons for Reproductive Justice in Maternal-Fetal Medicine**

The three pillars of centering women of color, embracing intersectionality, and meaningfully addressing difference are guiding principles through which clinical practitioners and scientists can manifest reproductive justice in maternal-fetal medicine. While these three pillars can be enacted in many ways, I have also provided some more tangible guidelines that can be a starting point for social justice transformation within the field. Healthcare providers in maternal-fetal medicine should:

1. Acknowledge the historical and present-day harms that have been done by the institutions of science in manifesting reproductive injustice. Only by recognizing and deconstructing this history can we dismantle the remnants of oppression, particularly racial oppression, within maternal-fetal medicine today.

2. Recognize race as a socially constructed category that has been used to fuel hierarchies of oppression and reject the biological basis of race.

3. Recognize systemic racism as a threat to reproductive health for both the pregnant individual and the child.

4. Utilize community-based research practices to empower those who are the most marginalized when constructing research questions, conducting analysis, and implementing clinical and public health interventions.

5. Pay attention to the particular. Note that differences are significant and should be acknowledged equitably and accurately, but that ‘difference’ is not an explanation for inequity in and of itself.
6. Affirm patient autonomy as well as the free flow of accurate information in all clinical care and research.

7. Remember that reproductive justice exists alongside and in harmony with other forms of anti-oppression work. Reproductive justice is environmental justice, is disability justice, is immigrant justice, and so on.

Overall, reproductive justice is a fluid framework that includes many aspects, only a few of which have been highlighted in this chapter. The broad pillars mentioned in this chapter, centering women of color, embracing intersectionality, and meaningfully addressing difference, provide a foundation for opposing inaccurate, harmful methods of race-based medicine within the field of maternal-fetal health. By sticking to these guiding pillars, we can not only work to eliminate the biological basis of race within the field but replace it with a framework that allows us to begin to correct for the systemic marginalized of women of color within medicine. This framework illuminates the path through which healthcare providers within maternal-fetal medicine can start to holistically, comprehensively, and justly address many of the racial health disparities related to reproduction that women of color in the U.S. face, such as the high rates of maternal mortality, infant mortality, birth defects, premature babies, and other adverse birth outcomes. By manifesting reproductive justice, I believe we can hope to provide equitable care for women of color in both clinical practice and research to affirm the right for all people to have healthy offspring.
CHAPTER FIVE

CONCLUSION

The previous four chapters have investigated how the biological basis of race has been constructed throughout the history of western science and into the present day, where it now exists in medicine as a problematic explanation for racial health disparities. More specifically, by analyzing the scientific literature around birth defects among various racial/ethnic groups, I have sought to demonstrate how the use of the concept of biological race within maternal-fetal medicine further marginalizes women of color and their offspring by perpetuating reproductive health disparities. These remnants of the biological basis may seem subtle to a lay onlooker, but they have the potential to drastically limit the type and quality of care women of color may receive within the field of maternal-fetal medicine.

As previously discussed, perhaps the greatest problem stemming from hypotheses that use biological conceptions of race as a risk factor for birth defects is that the perpetuation of the disparity itself, which results in infants born to women of color continuing to have higher rates of birth defects. Ultimately, this ends up preventing women of color from having healthy offspring who are able to develop, prenatally and postnatally, in a healthy environment. The realities of this health inequity are masked behind a simplistic, inaccurate explanation of differences in biological race, which only further prevents future research from actually solving this disparity. This leaves women of color and their offspring with the burden of the morbidity and mortality that can stem from severe birth defects, potentially preventing them from thriving at the same
rates as their white counterparts. We must recognize this as a manifestation of reproductive injustice, where women of color are not able to assert their right to have healthy children and to have healthy environments in which their children can grow and thrive. Once we recognize this healthcare disparity as a form of reproductive injustice, one that is being perpetuated by explanation reliant on the biological basis of race, we can use the framework of reproductive justice as a solution. By centering women of color, embracing intersectionality, and meaningfully addressing difference, reproductive justice provides a way forward for the field of maternal-fetal medicine to work to improve the health of women of color and their offspring in accurate, equitable ways.

Furthermore, in naming the persistence of the biological basis of race within the case of increased rates of birth defects among racial/ethnic minorities, we can begin to recognize medicine as an institution of bio-power that has been used throughout modernity to create and recreate the category of race. “Bio-power, according to philosopher Michel Foucault, refers to the practices of modern states to regulate their subjects through technologies of power” (Kang et al., 2017, p. 73). As Kang et al. (2017) have further explained, “Foucault argued that medical knowledge [emphasis added], combined with modern states’ collection of data on their populations, created new norms of health which populations internalize. Thus, the intended effect of bio-power is that people regulate themselves according to norms proliferated by medical knowledge and the state” (pp. 73-74). The “norm” of race has been constructed through the bio-power of the institution of medicine, allowing race to be seen as a valid, factual category that has a biologic, and specifically genetic, underpinning.
In the modern era, medicine has constructed the racialized body of the person of color to be the one that faces health disparities, health disparities that are deemed to be ‘natural’ to the non-white being based on their ‘degenerate’ genetics. This process reifies the category of race while simultaneously perpetuating health disparities that people of color actually do face. Racialization theory has argued that “races are produced through political mechanisms that create forms of inequality written on the body” (Isoke, 2016, p. 748). This is the process occurring in medicine, where the body of color is inscribed with biological degeneracy and then blamed for causing the very inequity it faced, subsequently causing a cycle of blame that fails to address the oppression faced by people of color. Within the case of birth defects, because of the way potential for disability and death surround a diagnosis for birth defects, medicine, as an institution of bio-power, has normalized an infant with birth defects to be seen as a ‘defective’ or ‘degenerate’ infant. Within this framing, the mother of said infant is labeled to be the host of this degeneracy, where her degenerate nature has been passed down biologically from mother to offspring. This is the mentality that is emphasized through hypotheses that ‘blame’ or falsely attribute the cause of increased rates of birth defects to race-specific genetic susceptibilities without actually investigating how class-based, gender-based, and race-based oppression can cause negative repercussions upon the health of marginalized populations.

In order to end the cycle of degeneracy that has been created by the bio-power of the institution of medicine, we not only need to utilize a reproductive justice lens to see and solve existing problems within maternal-fetal medicine but to rethink the very foundations of how we treat women of color in medicine. As one example of this, in hopes of spurring growth among
both institutions and individuals, Zenzele Isole (2016) has explained that contemporary studies of race and racialization should carefully consider the following questions:

1) Who gets to live and who gets to die? 2) Who gets to live well, and who gets to suffer? 3) Who has the right to hate and thereby the right to kill? 4) Which bodies are counted as persons and which are counted among the (living) dead? 5) How do states, governments, civil society, media, popular culture, the technologies of everyday life, and theories of political and social life either contribute to or disrupt the practices of violence that continue to make race real? (p. 758)

To deconstruct and dismantle the remnants of biological basis within the field, I believe that these questions must be investigated by healthcare providers under the framework of reproductive justice as part of providing better medical care. Tailored for this audience, I have adapted to Isole’s questions as follows: 1) How does the reification of biological race within medicine determine who lives and who dies? 2) How does this reification determine who can live well (thrive) and who can suffer? 3) Who has the right to power within the field – the power to make decisions, to choices, to empower or disempower? Who is deciding what marginalized patients deserve and don’t deserve? How does this contribute to who lives, who dies, who lives well, and who suffers? 4) Which bodies are prioritized and which are marginalized within scientific research, clinical practice, and medical education occurring in medicine? 5) How have the realities of bio-power influenced the development of the field of medicine, and what are the impacts of this upon women of color?

Some of these questions have been hinted at through the previous four chapters, while others have not. However, all of these questions provide a starting point for further work to more fully understand the persistence of biological race within the field of medicine and to affirm reproductive justice (which includes comprehensive, accurate, culturally-aware care) for all patients, especially women of color. While these questions may be daunting, they “also force
new generations of scholars to think carefully about the significance of small-scale movements, including common and recurring interpersonal and collective modes of relation that allow innovative forms of resistance for practices of freedom to flourish” (Isoke, 2016, p. 758). In this way, reproductive justice is not only a solution to deconstructing racial health disparities and the biological basis of race but a starting point for transformation for the entire institution of medicine in order to bring about ‘practices of freedom to flourish’ for our most marginalized populations.
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VITA

Amrita Bhagia was born and raised in Rochester, Minnesota. Before attending Loyola University Chicago, she attended St. Olaf College in Northfield, MN, where she earned a Bachelor of Arts in Biology and Women’s & Gender Studies, graduating Magna Cum Laude with Distinction in both her majors in 2019.

While at Loyola, Bhagia was a Graduate Assistant in the Department of Women’s Studies and Gender Studies (WSGS) and served as the WSGS Representative on the Graduate Student Advisory Council for the 2020-2021 academic year. She also worked as a Master’s Mentor for Achieving College Excellence (ACE), where she mentored underrepresented students from first-generation and low-income backgrounds.

Beginning in summer 2021, Bhagia will be attending medical school to pursue a dual MD/PhD degree, where she hopes to focus on integrating her feminist training into the field of healthcare.