

2014

Repackaging Racism: The Role of Sickle Cell Anemia in the Construction of Race as Biological

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Repackaging Racism: The Role of Sickle Cell Anemia in the Construction of Race as Biological

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Thesis submitted in partial fulfillment of the requirements

for a major in the program in

Science, Technology, and Society (STS)

at Vassar College

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April 2014

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Prologue

In the spring of 2013 I attended a lecture by Dorothy Roberts. She was discussing her new book, *Fatal Invention: How Science, Politics, and Big Business Re-Create Race in the Twenty First Century*. She described how we have been bamboozled into believing the races are more separate than they actually are. Specifically, she argues that though it has been proven again and again to be unrelated, popular belief has it that there is a genetic component to race. Since race plays such a potent role in my everyday life it seemed reasonable to think that there must be something substantial to it. She argues however, that the foundation of this term rests in its political origin and not in the imagined biological one. I found her arguments mesmerizing, especially her ability to illustrate how the archaic categories of race have become engrained to the point that we are inevitably recreating them through the realm of genetics by the assumption that they already exist. She pointed to articles and studies that used these politically generated racial categories to produce documentation of genetic difference.

While her study was logical, it almost seemed unbelievable due to my preconceived notions about the existence of race. This is not to say that I did not question its definition. I am a biracial individual who is half West African and half white American, so I have been confronted with the contradictions of race since birth. Whether it was filling out the demographic information on paperwork, or walking into a store with my mother versus my father, it is clear that I walk a charged yet elusive line. As a result, the navigation of racial borderlands plays a significant part in how I conceptualize myself and how I relate to others. Nonetheless, while I am an example of the incongruity of racial categories it was challenging and confusing to learn that there is nothing behind them. The most difficult area to grapple with was the concept of race

specific disease: specifically sickle cell. How could Roberts argue that this disease did not affect mainly individuals of the black race? How was this clear correlation not proof of the genetic connection between people of one race?

Working through Roberts' work I realized that the answers to these questions were in her original point: it's because race itself was socially and politically constructed. There cannot be a race specific disease because there are no races. The way sickle cell disease was presented to the public created an image that this specific disease is tied to race (blackness) because it aligns in some capacity with the predetermined categories. The fact that sickle cell happens to affect a majority of people considered black is simply coincidence, not inherent, as illustrated through the fact that it affects a people of a variety of races and is strongly suggested to have a geographic origin based on positive selection for malaria resistance. Of course the overarching factor is that races themselves are arbitrary and as a result there is not one genetic factor that is consistent for every member within these categories, sickle cell allele included.

Nonetheless, as stated earlier, it aligns in part with a racial category that has real social and political consequence, therefore policies affecting sickle cell disease sufferers and trait carriers inevitably affect those who are considered black. This is not to say that there should be more policy surrounding sickle cell, but is instead a call to recognize the types of policies surrounding this disease and the social context they are operating in. The way sickle cell was often sidelined in comparison to other genetic diseases is likely a product of its association with blackness, but the later obsession with counteracting the disease as a remedy for the black condition housed its own problems, as it distracted from the actual societal ills affecting the black community at large.

Due to sickle cell anemia's position as a genetic disease and its association with a racial category, it has remained an active player in the repackaging of race through modern genetics. In recent years, genetic sequencing has become more prominent as the ultimate definer of human life, yet the ways in which we continue to incorporate and enforce past racial categories is where a new form of the problem lies. Throughout this thesis I hope to deconstruct these current notions of race as biological by using the disease I originally found to be the most convincing example to the contrary, sickle cell anemia.

Introduction

Currently in the United States, outward characteristics and behaviors provide the basis for divisions we call “races”. Race is a readily accessible marker of difference between people and is a concept that is integral to most Americans' understandings of the world (Roberts 11). While there has always been a recognition of difference among peoples, whether it physical, religious, or cultural, the use of the broad overarching divisions of race to define and box this heterogeneity only happened recently (Roberts 2011, Smedley A., Smedley D. 2012).

The generation of this worldview, while recent, has been pervasive (Smedley A., Smedley D. 2012, Hammonds and Herzig 2008). Through its manifestation in major institutions race has impacted the way we relate to one another and who has opportunities in life. I will argue that science and medicine have played significant roles in how we define race. Specifically, I will examine how the positioning of sickle cell anemia as a race-specific disease has reinforced the concept of race as biological and contributed to the framework through which current genetic research constructs race as an innate structure.

In chapter one I will outline how the concept of race developed in the United States. I will examine its relationship with power dynamics and its role in the founding principles of the nation. This section will end with a brief discussion of the transition to current theories on race. The second chapter will provide an overview of sickle cell anemia, such as its background, origins, symptoms and treatment. The third chapter will illustrate how sickle cell anemia developed into a racialized disease in light of the worldview solidified only two centuries earlier. Finally, the fourth chapter analyzes at the consequences of the intertwining of race with sickle cell in addition to expanding the discussion on how race interacts with medicine more broadly. It

looks at the ways that race influences disease, and disease, race, and explores the the possibility of a genomic future. The conclusion examines the role of race in public health and policy now and the possibility of resistance.

In order to complete this task I must define the key terms and concepts central to my thesis: race, science, the categories of black and white, and the difference between family history and race. In Evelyn Hammonds and Rebecca Herzig's anthology entitled "The Nature of Difference" (2008), they provide a chronology of how the definition of race has changed since 1886. They begin with J. Thomas' definition in "A Complete Pronouncing Medical Dictionary" (1886) where he defines races as "permanent varieties of the human species, characterized by certain prominent distinctive traits". According to Hammonds and Herzing, the next development in the accepted definition of race is documented by C. Winick (1956), because he provides a link between race, inheritance, and genetics. He defines race as "a major division of mankind, with distinctive hereditarily transmissible physical characteristics, e.g, the Negroid, Mongoloid, and Caucasoid races. It may also be defined as a breeding group with gene organization different from other intra-species groups". This concept of race portrays it as natural unquestionable divisions that are linked to our genes, a belief that profoundly affected the conception of race and continues to color how many view it today. This line of thinking is not directly challenged in Landeu's 1986 definition of race in the "International Dictionary of Medicine and Biology", but complications are beginning to be recognized. As he states,

"human races are generally defined in terms of original geographic range and common hereditary traits which may be morphological, serological, hematological, immunological, or biochemical. The traditional division of mankind into several well-recognized racial types such as Caucasoid (white), Negroid (black), and Mongoloid (yellow) leaves a residue of populations that are of problematical

classification and its focus on a limited range of visible characteristics tends to oversimplify and distort the picture of human variation.”

This is a marked change from the concept of fixed, clear cut races. As we continue to move through history, it may seem like a backwards step is taken in the “Dictionary of Genetics” (1997) when it defines race as a “phenotypically and/or geographically distinctive subgroup”. To its credit, it also includes this statement: “the number of racial groups that one wishes to recognize within a species is usually arbitrary...”. Unique to this definition over the earlier ones is the argument that there is no set number of races and that distinction between them is “usually arbitrary”. One of the first times that biological race is explicitly challenged and presented as a social construction in a major dictionary is in 2005 when Mai, Young Owl, and Kerstig state that “the biological concept of race is very difficult to apply to observed patterns of human variation; rather human varieties can be considered cultural constructs that are contingent facts of history”.

As these definitions imply, race is intricate, convoluted, and continuously evolving, particularly its categorization as “natural or social, fixed or mutable, inherited or acquired” (Hammonds and Herzig 2008). This instability of the notion of race yet the widespread confidence in its existence provides the basis for this thesis. Race is something that everyone intrinsically experiences (to varying degrees), and it shapes how most Americans navigate their lives in the U.S. (Delgado and Stefancic 2012). As the basic philosophies of critical race theory lay out, race impacts how we interact with each other on even the most minute levels, and it affects our conception of self (Delgado and Stefancic 2012). Nonetheless, while race has real social and political consequences, the differences are not based in our biology (Tattersall and Desalle 2011, Smedley A., Smedley D. 2012, Roberts 2011, Pollack 2006). As Dorothy Roberts cleverly states in *Fatal Invention* (2011), “Race is not an illusion. Belief in intrinsic racial

difference is a delusion”. While that statement sums up the large volume of research supporting the belief in the social construction of race (Detailed documentation of this support is presented in Smedley A., Smedley D. 2005), it reduces a very challenging concept into a deceptively simple statement. As Delgado and Stefancic (2012) argue in *Critical Race Theory*, a racialized worldview has infiltrated the way that almost all major institutions are run, let alone the way we think about people on an interpersonal level. Where this worldview has been particularly insidious is in the way it has seeped into the institution that we often deem as the ultimate generator of “objective knowledge”, *science* (Hammonds and Herzig 2008, Bloor 1974). This infiltration has resulted in the upholding of race as a biological concept on varying levels through today (Roberts 2011).

Science, while perhaps not intentionally, has been consistently used to uphold racial divisions since their inception (Savitt 2007, Roberts 2011). For the purpose of this thesis, science will have a shifting definition. In the first chapter it will be defined as commonly held views, practices, and understandings within multiple disciplines of the sciences and social sciences - including but not limited to: anthropology, genetics, and biology – that support outdated views of the differences between the races. Views, for example, that Agassiz and others championed in the mid-1800s, such as the undeniable inferiority and mental feebleness of blacks. The beliefs have now been debunked overtime and their aftermath analyzed by authors such as Audrey and Brian Smedley, Eveleen Hammonds, and Keith Wailoo. This is not to say that the science carried out between the mid-18th and mid-19th century was pseudoscience or poor quality necessarily. It should be seen in context, paying attention to the shifting perspectives on race at the time. I do not want to excuse their biases, especially as there were active anti-racist voices during that time,

such as Benjamin Bannaker, but it is necessary to keep in mind the dominant viewpoints of the period. I will later focus on science in terms of recently conducted studies in those areas. This science is also not to be presented as neutral, but perhaps as more subtly influenced by and influential on the current construction of race. As a result of the continued challenges faced by science and its perpetuation of biological races, I do not wish to create a dichotomy between studies that clearly played a role in “scientific racism” (science used to directly support racist ideologies) and more current studies which are presumably absolved of this prejudice (Roberts 2011). Medicine, referring to the specific branch of science relating to human health is used interchangeably with science as it too is relevant in the generation of views around race.

Another term integral to understanding this thesis is “black”. If race is defined as socially constructed, it seems that “black” and “blackness” should lose their meaning. On the contrary, current society recognizes race as extremely significant, and who is defined as black has implications for how they will be treated. As a result, black will be defined as a member of a population that has been systematically oppressed based on perceived phenotypic (outward expression of a gene) and cultural difference. The characteristics I am referring to as racial markers for black individuals include visual cues such as dark skin and eyes and tightly curled hair. For the most part, I will use black over African American because I am referring specifically to the group of people viewed as black regardless of if they have recent African History. Further, in some instances, such as when I discuss the role of racism in health risk factors and disparities, I am excluding those who *do* have recent African family history but *do not* possess the stereotypical phenotypes associated with the black race because they do not necessarily experience the same type of immediate interpersonal discrimination. With that I am focusing

more on peer-identified race, because it is the perception of others that affects how one is treated though I recognize the importance self-identification as well. I am focusing on blacks, though statistics often also emphasize the high rates of sickle cell among Latino/a populations, because as currently defined by the Census, Hispanic is not a race, but an ethnicity, so someone who identifies as Hispanic must also choose a race, which includes them in this discussion if they identify and can be identified by others as black.

White I will also define within the current social sphere as someone who is peer and self identified as white, possesses characteristics attributed to the white population – most notably light skin, and experiences the benefits of white privilege (a list of which can be found in Peggy McIntosh's *Unpacking the Invisible Knapsack*). This definition may seem vague, but the boundaries of who is defined as white have changed dramatically over time (Painter 2010, Roberts 2011). The one constant is the power that whiteness provides, whether psychological or physical over people of color (Delgado and Stefancic 2012).

The final distinction I want to make is between race and family history. Race is sometimes viewed as a stand-in for family history because there is an assumption of a common ancestry. What a viewpoint like this ignores is the fact that “there is more genetic diversity within races than between them” (Roberts 2011), and that there is no genetic factor that links all members of one race together (Strain et al 2003, Roberts 2011). As a result, race is not a reliable indicator of genetic commonality and attempts made to treat it as such have failed horribly, an example of which, BiDiI will be discussed later on. Unlike race, family history is directly related to some risk factors for illness, appearance, and personal propensities because genes are passed down from parents directly to children (Snustad 2001). This is not to present family medical

history as deterministic, because to be expressed genes must interact with the environment, but it plays a much more significant role than race in constructing our genetics. The qualities and phenotypes that we use to define race (hair color and texture, skin tone, nose shape), are not intrinsically linked to genes for other more complex characteristics like musical talent (Strain et al 2003). As a result, it is much more significant to examine the genes of someone with a known relationship than with someone you may arbitrarily share physical characteristics with.

Chapter 1: The History of Racial Divisions

Race in the United States was only constructed a few hundred years ago (A. Smedley and B. Smedley 2012, Roberts 2011, Tattersall and Desalle 2011). The racialization of people over this time period was a multiphase process that employed (and continues to employ) science, the judicial system, and manipulation of the common man to carry out its end goal of maintaining a system of power (A. Smedley and B. Smedley 2005, 2012, Gross 2008, Painter 2010). This effort produced a lasting system that continues to govern our actions today (Delgado and Stefancic 2012). What is unique to the racial system carried out in the U.S. is the harsh dichotomy that was created between white and black. The development of this cavernous, unbridgeable distinction between whites and blacks provided justification for the inherent contradictions of slavery (Roberts 11, Hammonds and Herzig 2008). With the abolishment of this institution and increased immigration to the United States from all areas of the world, race has been under the microscope in the last century and its construction is frequently reevaluated to accommodate new people.

While the first mention of “race” as a word and concept has been highly contested, one of the first descriptions of race as it relates to the modern day definition was by François Bernier in 1684 (Bernesconi, Loft 2000). Bernier was a french physician that traveled to a number of places in Asia and Africa. In each location he noted the physical differences among the people he met distinguishing the broad groups as races (Bernesconi, Loft 2000). Bernier excitement in the discovery of difference among people shines through in his writing (1684), “I have remarked that there are four or five species of men in particular whose difference is so remarkable that it may be properly made use of as the foundation for a new division of the earth”. In any case, prior to

Bernier's more official classification of races, people in the United States had already begun grappling with the acknowledgment of human difference.

Africans first arrived in the U.S. in 1619, but they did not occupy the space of chattel slaves as we might think of today (Painter 2010). Prior to the explosion of the African slave trade, Africans (not yet defined as blacks so I will refer to this population as African) were entering in smaller numbers and occupied a minority of the labor market in the colonies (Painter 2010). In fact, the majority of indentured servants' origins stemmed from Britain (especially orphans and criminals) until the 18th century (Painter 2010). During this period, miscegenation laws were not yet in place and intercourse between Africans, Europeans, and Native Americans was not specifically outlawed (Gross 2008)

Dissension between the African and European (not yet classified as white so I will continue to refer to this population as European) laborers did not begin to surface until post-1676 as a result of Bacon's rebellion (Roberts 2011). This rebellion occurred when African and European servants bound together under the influence of the prosperous planter Nathaniel Bacon to insist that Virginia governor William Berkeley work harder not only to protect the frontier from attacks by Native Americans, but to further expand the colony into Native territory (Roberts 2011). Despite the speculated spiteful intentions of Bacon's involvement in the uprising and the nonrecognition by slaves of the shared dehumanization happening to Native Americans, this rebellion marked the first major collective action among European and African slaves as a result of a recognition of common subjugation along class lines (Gross 2008, Roberts 2011). The solidarity generated between these groups in an effort to fight back against the common thread of

economic injustice and wealth distribution between the affluent members of the colony and the indentured/formerly indentured servants was enough to worry those in power.

As a preventative measure, the wealthy began to vary their treatment of the groups in order to curtail their unity and stifle the people power that was being harnessed to address economic inequality (Gross 2008). Over the next few decades favoritism emerged, European slaves received freedom dues upon the end of their indentured servitude whereas African slaves did not (Roberts 2011). The separation escalated when Africans became bound to a lifetime of slavery that would eventually extend to their children, in direct contrast with Europeans, who continued to be guaranteed freedom at the end of a set term (Roberts 2011). This resulted in the biggest push toward the assumed humanity of one group and dispensable subhuman nature of the other. Building camaraderie and solidarity between these two groups became less feasible when they no longer held the same social position (Roberts 2011). Solidarity between the groups was also undercut by the privileges afforded to those in the emerging category of “whites” that were distinctly not afforded others, such as those classified as “black” (Roberts 2011). The benefits provided this group generated feelings of superiority that hindered unity (Painter 2010, the physiological superiority attached with whiteness discussed in *Critical Race Theory*). Nonetheless, while the divisions provided the foundation for the argument of fundamental differences between these groups, the concept of race as we think of it today was only in its initial stages.

It was in the 18th century that the scientific classification of people into races became a serious, widely valued idea . This transition was in large part due to the centering of the labor market around the African slave trade. The increased use of African slaves over European ones

had benefits for U.S. landowners in addition to preventing a united struggle against the wealthy. First of all, many Africans already possessed the skills needed to farm sugar and other major products of southern plantations because many of these crops were being grown in West Africa (Roberts 2011). Africa also offered what many viewed as an “endless supply” of labor, which was becoming increasingly important because many laborers from Britain and Ireland were refusing to make the treacherous trip to America (A. Smedley and B. Smedley 2012). Finally, and perhaps most significantly in light of racial formation, the vast majority of Africans brought to the U.S. had distinctly darker skin, hair, and other phenotypic differences that prevented them from blending in with the current settlers (A. Smedley and B. Smedley 2012). Though there were a few free blacks, the vast majority were not, and this visible difference among the enslaved and the free vastly decreased the possibility of servants running away as they would not have blended in with the surrounding free populations. Thus, the the emphasis on one's physical characteristics as a marker of inferior status in society began (A. Smedley and B. Smedley 2012).

This division gave rise to chattel slavery in the 1700's, a type of slavery where humans as solely viewed as property to be bought and sold, with no more rights than an animal. Though chattel slavery generated vast wealth for plantation owners, this inhumane treatment of people was in direct conflict with the goals of liberty and justice that United States intended to uphold as a nation (Strain et al 2003). While one might have speculated the end of the institution of slavery in light of this fundamental clash in ideals, the majority of the founding fathers were slave owners themselves and slavery clearly brought a significant benefit to the economy (Britannica). In addition, the ideals themselves, when viewed through a racialized lens, could provide justification for the continuing of slavery. The founding principles advocated for self-

determination, self discipline, and individualism, all necessary to the development of any capitalist society. This belief system emphasized the importance of property rights, and as slaves were viewed as property at the time, it seemed “un-American” to strip men of their possessions (Smedley 2012). This argument was aided greatly by the then accepted concept of unequal races (Strain et al 2003). If blacks were viewed as feeble minded and subhuman, then slavery gained justification. Nonetheless, at the time there were no dominant scientific theories on the superiority of one race over another. On the contrary, there was still a lot of work around classification of races and findings were indicating that there is one species of humans, but multiple varieties (Bernasconi and Lott 2000). Nonetheless, the lucrative institution of slavery still needed more substantial backing and a shift in view to the equality of the races would have far-reaching political consequence (Hammonds and Herzig 2008). As a result, Thomas Jefferson “called on science” to investigate the inherent differences between the races (Smedley 2012, Strain et al 2003). There was already suspicion that racial differences extended well beyond skin color but he was looking for confirmation:

“Whether the black of the negro resides in the reticular membrane between the skin and the scarf-skin, or in the scarf-skin itself; whether it proceeds from the colour of the blood, the colour of the bile, or from that of some other secretion, the difference is fixed in nature, and is as real as if its seat and cause were better known to us” (Jefferson 1781, Hammonds and Herzig 2008).

This set the stage for the transition to the genetic basis of race only a century later (Strain et al 2003).

Science responded to Jefferson's call to action, and by the mid-1800's articles about the scientific differences between these already assumed races began to surface en masse. One of the most well-know works to contribute to the discourse on race during the 1850's, specifically the hierarchy of the races, was Louis Agassiz et al's *The Types of Mankind*. Of course works

appeared before this one boldly claiming the inescapable inferiority of other races, such as John Campbells “Negro-mania”, but Agassiz was a well respected Harvard professor and naturalist, and his work had far reaching implications. Other influential works included that of Galton in 1869 suggesting the heredity of race and Gobineau in 1853 (Bernasconi and Loft 2000). These two theorists focused on carefully planned reproduction and are considered founders of the eugenics movement (Bernasconi and Loft 2000), With the support of many acclaimed scientists the entrenchment of the racial divide reached its most convincing peak. This is not to say that views of white supremacy and racial superiority were not without their critics. To the contrary, the late 1850's was during the height of the abolitionist movement and many argued against unbridgeable racial differences including famous academics such as Frederick Douglas. Counter-arguments became even more important when the suggestion of eugenics and the dying off of the black race started to encroach on the fight for black equality in the late 19th and early 20th century, which lead to the involvement of prominent theorists such a W.E.B. Dubious. Nonetheless, the addition of an inherent and inherited inferiority added a new element to the struggle for equality, one that would capture peoples' subconscious for a long time following its “official” end.

The 20th century sought to deal with the conflict between a large, free population of black Americans and a belief system now structured around a biological basis to race. Since blacks were no longer legally property creative methods of containing the black population had to be developed (Smedley 2012). One step merely required linking the unpreparedness of black Americans for entrance into mainstream society with an innate deficiency. Many blacks failed to effectively incorporate into mainstream society and was attributed to their incompetence without

recognition of their little education and lack of experience with the economic systems due to their prior subjugation (Roberts 11). The struggles that blacks had with this process (particularly the high rates of mental illness) supported arguments for the paternalism of slavery, stating that blacks were simply too feeble minded to handle life on their own (Smedly 2012).

Another way that effectively served to regulate blacks to a position of second class citizenship was the judicial system. For example, laws preventing intermarriage between blacks and whites date back as far as the seventeenth century, but they rose to a new importance when the possibility of free blacks interacting with whites became more likely (Gross 2008). These laws in conjunction with laws about full citizenship, the ability to own land, and the ability to travel freely fostered the structural subjugation of blacks, and fed into the prevailing ideology of blacks as not fully human in the way that whites were. These regulations were most pervasive in the south, where the Jim Crow era made the obstacles to obtaining basic rights seem insurmountable for those who could be identified as black (Gross 2008).

Nonetheless, the process of identifying someone as “black” was not always as simple as studies about the vast caverns between the races made it seem. Significant “mixing” (often through sexual assault) occurred among those of European, African and Native American ancestry to create individuals of all shades, sizes, and facial make-up (Smedley 2012). Still, despite this intermixed heritage, all ambiguous individuals were often recognized as a part of the sliding scale of blackness. The whiteness of these individuals was generally ignored if possible as to many it seemed irrelevant; one either carried the privileges of being white or did not and there was a continued belief in a “pure” white race (Goodman 2003).

This lack of official recognition of mixed individuals in the first half of the 20th century as their own category or as one that at least as indefinite was in large part due to the harshness of the dichotomy created between blacks and whites, and the pedestal on which whiteness was placed. Unlike in other slave holding countries, such as Brazil, where white masters were required to recognize children they produced with their female slaves as their own, whites in the U.S. were never legally bound to acknowledge their relationship to their offspring born to slaves (Smedley 2012). Smaller steps such as this one, contributed to the belief in the tainting ability of blackness (Goodman 2003). The level to which the infiltration of “black blood” constituted blackness varied from state to state (in some it was 1/16th, in others 1/8th and in a few, as small as one drop) (Roberts 2011). This association of “blackness” with blood developed as medicine became more involved in race, perhaps intensifying with the discovery of a blood related disease that seemed to be unique to African Americans, sickle cell anemia.

As we move through history into current times, the worldview around races has changed significantly. There are continued attempts to categorize people into set races, as illustrated by the Census and standardize testing, but the recognition of the role of social construction in race has become more evident. The most recent works, such as that of Roberts (2011), Krimsky and Sloan(2011), Nelson, Lee, and Wailoo (2012), and Tattersall and Desalle (2011), were in some ways born out of the aftermath of the Human Genome Project (2001). Many of the authors listed above had the hope that this project would debunk the concept of biological race once and for all, but while the evidence describing the vast similarities between humans did provide sufficient proof for an elimination of biological race, the way the study has been manipulated within the scientific sphere has done the opposite (Roberts 11). The true separation of social and political

race from a made-up biological race is a difficult conclusion to draw and in my experience has not yet entered mainstream sensibilities. What scientists such as Evelyn Hammonds and Dorothy Roberts find most frightening is the way in which the gene map has been speculated as the final breakthrough for determining the differences between races. The worry among these social scientists is that a new worldview will develop that bypasses the difficult but important concept of racial social construction and aims for genetic validation (Wailoo et al 2012). The consequences of a worldview in which race is seen as intrinsic will be discussed in a later chapter, but it is important to note that the the concept of race is still evolving and changing, and at present it is passing through a major threshold that could shape how a generation sees and thinks about race.

Chapter 2: Overview of sickle cell anemia

General Overview :

Sickle cell anemia is a genetic blood disorder that is characterized by relatively frequent bouts of severe pain, organ damage, and eventual early death (Patterson 2009). The disease affects hemoglobin proteins, which are integral to the transportation of oxygen by red blood cells (Peterson 2009). As oxygen is used in numerous biological processes that maintain our systems, such as in the digestion of food and the production of energy, it is necessary that all tissues receive this vital molecule. In the case of sickle cell anemia, red blood cells are less capable of transporting oxygen because they have a damaged form of hemoglobin, known as hemoglobin S.

90-100,000 Americans currently have the disease, which is approximately 3% of the U.S. population (CDC 2009). In addition, though the last chapter exposed the social construction of race, it is necessary to recognize that as currently defined, sickle cell anemia disproportionately impacts black Americans. Nonetheless, the disease still only occurs in 0.2% of the black population. The trait, which consists of solely the genetic marker and not the actual disease, is a lot more common consisting of approximately 8% of the black population (CDC). The life expectancy for sickle cell anemia is approximately 40-50 years which is significantly higher than it was only a few decades ago (CDC 2009).

Symptoms:

The most prominent experienced symptom of sickle cell is pain in various forms, and one of the earliest indicators of the disease is pain in the hands and feet as well as fever and swelling at the joints (Bloom 1995). This type of joint and bone pain generally lasts a week or so, but it

can occur at any time (Wailoo 2001). The cause of the pain is blockages of veins and arteries that cut off circulation to changing areas of the body. When large blockages occur, sickle cell anemia sufferers experience “pain episodes” (Peterson 2009). These large blockages result in the cutting off of oxygen from certain locations and the affected tissue starts to die (Nelson and Cox 2008). The process through which a tissue dies is extremely painful, from the moment that it becomes oxygen deprived to when the dead tissue is digested by hydrolytic cells (Bloom 1995). As Tiffany, a featured individual in a video by the NIH describe, “[it is] like repeatedly being stabbed, with a butcher knife, in the same spot, nonstop.” There is no way to predict when these episodes will occur or the duration they will last. When the episodes are not very severe then over-the-counter medication such as acetaminophen can be used, but when they are long lasting and severely painful, the sufferer can go to the emergency room where he or she can receive stronger medication, such as morphine, and intravenous fluids (NIH).

One of the most painful locations to have blockages is in the bones (Bloom 2009). Bones, like other living tissues in the body, need the oxygen carried by red blood cells in order to stay alive. They are important structural units in the body in addition to providing the home bone marrow which is responsible for producing red blood cells (Saylor). Due to the frequent destruction of defective red blood cells in a person with sickle cell, the bone marrow is distinctly important as it is constantly producing replacement cells (Bloom 1995). Oxygen rich blood enters bones through *sinusoids*, which are miniscule holes around the ends of bones (Bloom 1995). Unfortunately the unusual, inflexible cells unique to sickle cell anemia cannot always pass through these holes and they get stuck causing swelling and joint pain.

Other parts of the body that can be seriously affected by sickle cell are the lungs, liver, and spleen and heart (Stuart et al 2004). In sickle cell patients, the chest is often smaller than average, and the heart larger than normal, which lessens the space where the lungs can expand (Bloom 1995, Stuart et al 2004). As a result, the amount of oxygen that these individuals inhale with each breath is less than normal, and it gets thinly dispersed (Stuart et al 2004). If a blood vessel becomes blocked in the lungs fever, chest pain, and breathing challenges may occur and immediate care must be sought out to prevent permanent damage to the lungs (Paterson 2009). Another organ that can become damaged in part due to blockages, but more often as a result of the build up of red blood cells in the organ, is the liver (Bloom 1995). As the blood gets filtered through the liver in people that have sickle cell, deviant cells can build up, causing damage to the organ which inevitably can lead to jaundice, a disease in which the whites of an individual's eyes become yellow because of excess chemical waste in the blood (Bloom 1995). This high concentration of waste products can also create gallstones, which are hard crystallizations of bile (used in digestion) with other minerals. Finally, the spleen has the possibility of becoming enlarged and damaged due to cells building up in the small vessels that run throughout it. It plays a significant role in housing white blood cells, so when damaged, it can negatively impact the immune system and increase one's susceptibility to disease (Bloom 1995). The list of organs negatively impacted by sickle cell continues as all organs are fed oxygen by red blood cells that must travel through small veins and arteries (Patterson 2009).

Biology:

In the course of sickle cell anemia, normal hemoglobin, *hemoglobin A (Hb A)*, is replaced by a version that does not function as well, known as *hemoglobin S (Hb S)* (Peterson 2009). The less effective version of hemoglobin occurs because of a tiny mutation that results in the swapping amino acids glutamic acid and valine, in turn affecting the solubility of hemoglobin (Alexy et al 2010).

Sickle cell anemia is one of the three major categories of sickle cell disease. The other two are sickle-hemoglobin C (*Hb C*) and beta-thalassemia (HbS) (Peterson 2009). Both sickle cell anemia and sickle-cell C affect the shape and activity of red blood cells, whereas beta-thalassemia creates issues by improperly regulating the amount of hemoglobin produced (Bloom 1995).

It is important that red blood cells remain flexible as they are required to fit through progressively smaller locations on their way back to the heart, and the liquid center provides the cell with the necessary flexibility to fit through narrow spaces such as veins (Bloom 1995).

When red blood cells travel throughout the circulatory system, the oxygen carrying hemoglobin molecules can be found solubized in the cytoplasm (liquid) inside of the cells (Peterson 2009).

Humans have approximately 270 million hemoglobin per cell (Saylor). Ideally, those millions of proteins float unattached from one another inside of the cell because when they clump together, the cell becomes rigid and inflexible (Bloom 1995).

In the case of sickle cell disease, the abnormal hemoglobin does not consistently remain soluble in the liquid inside of red blood cells and therefore clumps. The initial reason has to do with the the construction of the hemoglobin protein itself. Hemoglobin S is susceptible to

clumping because a mutation causes a water soluble amino acid (glutamic acid) to be replaced with an insoluble amino acid (valine) (Nelson and Cox 2008). As a result, when the abnormal hemoglobin proteins are not fully saturated with oxygen, they seek to protect the location where the insoluble amino acid is from the liquid center of the cell (Nelson and Cox 2008). To do this, they clump together with other hemoglobin S proteins in a way that is mutually beneficial for both of them. In this position, the insoluble portions of the proteins stick together leaving only the soluble portions exposed to the liquid, which is ideal. The clumps that these groups of hemoglobin form end up forming long strands that alter the shape of red blood cells from soft, circular, and inner tube-like to a hard crescent moon shape (“a sickle”) (Stuart et al 2004). This change in shape prevents the cells from passing through narrow spaces which can lead to blockages in veins and arteries (Bloom 1995). In people that only have one copy of the allele (“sickle cell trait”), this process of clumping still occurs, but much more infrequently than in sickle cell disease patients, because people with only the trait make normal hemoglobin as well (Patterson 2009).

Treatment:

A few treatments exist to prevent further complications with the disease, but no consistently effective treatment exists. Currently, the medication hydroxyurea is the best way to prevent pain episodes (Peterson 2009, Wailoo 2009). This medication increases the amount of fetal hemoglobin produced which a type of hemoglobin that does not sickle, but which we unfortunately stop producing on our own soon after birth (Peterson 2009). One type of care that is always recommended for sickle cell patients is a healthy diet that includes a large volume of

fluids (Petersoon 2009). Despite some progress in treatment, the most significant measure taken in the last ten years regarding sickle cell care is that newborns are now tested for sickle cell disease at birth. This early diagnosis allows doctors to immediately provide antibiotics to babies with sickle cell disease which helps to prevent threatening infant diseases as they are susceptible to infection (U.S. Preventative Task Force 2007).

Inheritance:

Sickle cell anemia is the result of a heritable genetic mutation in the hemoglobin gene, which is a small section of DNA (Bloom 1995). It is one of the few well-studied inherited diseases that follow Mendelian genetics. Mendelian genetics refers to traits whose inheritance patterns are controlled by a single gene (monogenetic) (Pierce 2010). Often heritable traits are controlled by multiple genes that all work in tandem to provide a final phenotype – the observed outward representation of gene expression (Pierce 2010). Phenotypes such as skin color and height are very difficult to predict or form probabilities for because they are controlled by a number of genes simultaneously, in addition to necessitating the incorporation of environmental factors (Pierce 2010). For the purposes of this thesis, sickle cell trait will be defined as someone with one copy of the mutant allele and sickle cell anemia as someone with two copies of the allele (Patterson 2009).

Monogenetic traits such as sickle cell anemia only depend on one gene inherited from both parents. Of course there are a number of permutations for the final genotype depending on the alleles of one's parents, but the probabilities are much easier to determine. Each parent contributes one of the two alleles they received from their parents to their child and the

combination of the two denotes what the eventual phenotype will be (Pierce 2010). As Mendel discovered, there are both autosomal dominant alleles and autosomal recessive ones. If a dominant gene is paired with the same dominant gene or with a recessive one the dominant gene will be expressed (Pierce 2010). The gene for sickle cell is considered to be autosomal recessive, so a person must carry two copies of the gene to have sickle cell anemia, one from each parent (Patterson 2009).

Despite how easy it seems to do a simple genetic test to determine if someone has two copies of the sickle cell trait or just one, the multiple manifestations and severity of symptoms across the populations who possess it indicate that other factors contribute to the expression of the gene.

History:

It is possible to trace a disease back to its origins through the use of genetic markers. Within genotypes, scientists can isolate the surrounding bases pairs (the letters through which the instruction manual of DNA is written) to determine the origins of the disease. The combination of the gene with its neighboring alleles is called a haplotype (Pierce 2010). Often long genes are inherited, but only a small portion of the DNA is actually expressed. The the part that gets expressed may seem most important, but the surrounding DNA can tell us a lot about the origin of a disease when compared with the DNA of large sample populations (Bloom 1995). *Hb S*, one of the major mutations found in the majority of sickle cell cases, has five origins according to the study of haplotypes. Four of the five origins are in Africa and include the Bantu haplotype (equatorial Eastern and Southern Africa), Sengal haplotype (Atlantic West Africa around the

Congo), Benin haplotype (central West Africa), and Cameroon haplotype (the center of Cameroon) (Bloom 1995). The fifth is unclear and thought to originate in either India or Saudi Arabia (Kulozik 1986). The fifth haplotype also contains other differences from the *Hb S* mutations found in Africa and is thought to have an independent origin (Kulozik 1986).

Over the centuries, these haplotypes have dispersed among other populations, particularly in North Africa, the Americas, and the Mediterranean for multiple reasons, including migration, trade, war, and slavery (Gonçalves 2003)[See Appendix 1]. The only haplotype that has failed to spread is the Cameroon one, which with a few exceptions has remained relatively concentrated in central Cameroon (Bloom 1995). The main haplotype found in North Africa (regions such as Morocco Tunisia, and Egypt) is the Benin haplotype from central West Africa. It is believed that thousands of years ago, people from the central West African region migrated up north through a fertile land that over those centuries has become what is now considered the Sahara desert (Bloom 1995). This same haplotype is located in the Middle East within populations of Syrians, Israeli Jews, and Saudi Arabians, specifically in the parts of Saudi Arabia that participated in the slave trade (sections of the western portion of the country) (Bloom 1995). The eastern portions of Saudi Arabia often carry a haplotype that is non-specific, having origins in either India or Saudi Arabia. The Benin haplotype appears to be the most pervasive North of Africa and is also found among all of the affected countries in the Mediterranean excluding Portugal. Portugal carries the Bantu and Senegal haplotypes which is reflective of their colonial past, occupying areas in Angola and Mozambique as well Senegal (at that time known as Portuguese Guinea).

In the U. S., constant migration has led to the presence of all of the haplotypes, however the majority of cases stem from the Benin, Senegal, and Bantu haplotypes (Bloom 1995). The

main cause for the concentration of cases in these three haplotypes is the slave trade. Depending on the location of the trading port and the countries participating in the slave trade in at a specific location, slaves were abducted and brought over from a number of locations in Africa (Gonçalves 2003). For example, the majority of slaves brought to Jamaica by the British came from the Central West Africa, which is the location of origin for the Benin haplotype (Bloom 1995). The second largest population was from Equatorial East and South Africa (Bantu haplotype), and the third the Atlantic Coast of West Africa (Senegal haplotype) (Bloom 1995). The percentages of people living in Jamaica with these haplotypes coincides with the records reflecting the number of slaves brought from those regions in Africa (Bloom 1995). Depending on the location, in the United States, the breakdown of haplotypes of sickle cell differs. For example, the breakdown of haplotypes in Baltimore (over sixty percent Benin haplotype) is very different from that of South Carolina, where there is a large number of Senegal haplotypes (Bloom 1995). Even further, one of the largest groups of patients with sickle cell in and around the New York area are Italian Americans (specifically Sicilian) carrying the Benin haplotype as a result of an invasion in Italy by people from the Middle East over 1200 years ago (Bloom 1995). While sickle cell has origins in Africa, as a disease it has spread around the world into all different populations over centuries.

The reason that sickle cell as an allele has persisted over the years, despite its contributions to spreading a debilitating disease, is due to a benefit it must provide. After the realization that the highest rates of sickle cell anemia were found in the highest incidence malaria regions it was speculated that there might be a connection between malaria prevention and sickle cell trait (Alison 1954) [See Appendix 2]. Since that initial realization, data has been found in

support of a link, particularly in the form of correlations between the distribution of sickle cell around the world and that of malaria (Aidoo et al 2009). Nonetheless, it was not until recently that it was understood how the process of resistance was taking place. In 2011, Ana Ferreira et al discovered that sickle cell trait does not stop infection of red blood cells from happening, but prevents serious infection from occurring by making the body “tolerant” of the parasite. She describes how the trait does this by causing the cell to release specific hormones when it becomes infected. These hormones break down down heme, a component of hemoglobin, in turn releasing a byproduct (carbonmonoxide) that prevents the parasite from moving the infection forward to cerebral malaria, which is the stage at which it becomes very dangerous (Ferreira 2011). As a result of this process, the sickle cell trait hampers the malaria parasite before it can become deadly, a substantial benefit in locations and during time periods when medications were unavailable.

Due to this unforeseen benefit of the sickle cell trait, natural selection likely played a role in its continued dispersion among populations. When introduced into locations where there were high rates of malaria, such as Sicily, those carrying the gene probably fared better than those without it. As a result, it was more likely one would reach reproductive age if they had the trait inevitably making it more likely that future children in the population would also carry the trait (Bloom 1995). There were a number of locations with very high rates of malaria a few thousand years ago that the gene was probably received a welcome introduction that quickly took hold because it prevented a major cause of early death. The individual trait must have provided an effective resistance as it seems unusual that a trait under which two copies of the gene creates an incapacitating and deadly disease persists for so long. Now that people exist in locations where

malaria is no longer as rampant the trait does not serve the same protective purpose. Nonetheless, considering the number of people that have it, it would take a while to completely select it out of the population especially since one copy of the allele does not have any negative effects on many carriers.

Chapter 3: How Sickle Cell Became a Race Specific Disease

This complex, devastating illness became attributed to the black body as a result of its emergence in science during a period of high racial transformation and the political campaigns waged in relation to it.

With the involvement of natural science in the creation of race in the mid-nineteenth century, medicine, like all aspects of life carried racialized undertones, specifically when it came to black individuals. By the beginning of the twentieth century, black people were viewed as disease carriers and associated with diseases of prominence such as syphilis and tuberculosis (Wailoo 2001). Their susceptibility to illness was thought to be a product of their ignorance, superstition, and inescapable physical and mental inferiority (Tapper 1999). Nonetheless, while blacks were associated with sickness in general, few diseases have maintained as strong a connection with race as sickle cell anemia has throughout the decades. This disease's association with blackness was a combination of its presentation in medicine and the way attention was captured around the disease: first as a means to prove black inferiority and later in the black power movement as a way to create solidarity awareness of the state of blacks in the U.S.

The first documented case of the disease that would soon be entitled "sickle cell anemia" was in 1910 by James B. Herrick (Herrick 1910/2001). In his article, Herrick describes the case of Walter Clement Noel, an immigrant from Grenada attending dental school in Chicago who possesses a strange set of symptoms: shortness of breath, yellowness of the sclera (whites of the eyes), and frequent leg ulcers since childhood (Herrick 1910/2001). Along with his description of the symptoms, analysis of bodily fluids, and proposed treatment course, Herrick includes sections on patient history and physical examination. In these two segments, Herrick describes

Noel as an “intelligent negro” and “a young man of typical negro facies” (Herrick 1910/2001). He also writes in his discussion section of the article of his surprise at the lack of history Noel had of syphilis, a disease for which increased susceptibility was already attributed to the black population (Hammonds 2003). The next documented case was by Benjamin Earl Washburn in 1911. This article describes Ellen Anthony, as a “fairly well developed negro woman, with facies characteristic of her race”, again clearly taking note of the race of the individual as it was seen as significant to health (Washburn 1911). Though these two articles alone only note that their patients were black, the fact that both clearly feature the race of the subjects sparked connection in the coming years.

The third case appeared in scientific literature in 1915. In this account, Jerome E. Cook and Jerome Meyer describe their female patient as a “very light mulatto” which illustrates a recognition of a problem with black and white binary. One would expect this realization to contribute to the complication of a connection of the disease with race, but the opposite held true; when Cook and Meyer compare their case to the previously documented ones of Herrick and Washburn they confidently begin the list of similarities between the cases with “all three of the patients were of negro blood”(Cook and Meyer 1915). This description plays to the idea that any “blackness” distorts whiteness to the point of non-recognition. In addition to beginning the link between sickle cell anemia and blackness, Cook and Meyer also realized the possible link to inheritance, inevitably originating a connection between blackness, sickle cell, and genetics.

Articles clarifying and categorizing the disease began to surface less than ten years after Cook and Meyer's article. For example, Verne R. Mason wrote an article in 1922 that designated

the disease the title “sickle cell anemia”. This article also supported the notion that it may be deeper than a typical bacteria or parasite saying:

“Nevertheless, they do point to an hereditary or congenial anomaly as the factor of importance in the development of the disease, and this assumption is supported by the history of disability in early life, by the occurrence of the disease only in negroes, and by the reports of Dresbach and Bishop of otherwise healthy and presumably normal persons whose red blood cells were elliptical” (Mason 1922).

Despite more prominently featuring the mixed nature of early patients' ancestry, Mason still saw their “blackness” as a significant link between the patients. This speculation about the heredity of the disease was confirmed a year later, though exact understanding about the patterns of inheritance would not become clear for a while (Sydenstricker et al 1923, Taliaferro & Huck 1923).

During the next twenty years the connection between sickle cell anemia and blackness was forged in the scientific community. If cases appeared in individuals who appeared to be another race, it was assumed that there must have been racial mixing in that person's history (Tapper 1999). In cases where the link to a black ancestry was not apparent there was active denial that the person had sickle cell because sickle cell only occurred in blacks (Tapper 1999). Due to the multiple origins of sickle cell and the unsubstantiated method of classifying people based on highly variable outward “race characteristics”, it is very possible that people who could be classified as white carried the allele or had the disease. Nonetheless, to admit the fact that racial categories were only skin deep and not indicative of important internal markers one would have to come to terms with the idea that there is no pure white race (Tapper 1999). That conclusion would have been difficult to imagine for someone who had grown up with immense racial prejudice, not to mention that the conclusion would also have been undesirable because it

would have chipped away at some of the power and privilege that whiteness provides. As a result, instead of loosening the dichotomy between whiteness and blackness, science looked for new ways of locating pollution of the white race by blacks (Tapper 1999). This push in turn generated more studies of the origins of the disease.

While the appearance of sickle cell anemia as a significant race-based disease took place in the scientific world during the 1920's-1940's, there was little input on the subject from black academics. This silence was in part due to worry about the fatalistic implications of an inherited disease on black people, particularly in light of the limited understanding of the disease over those decades, such as within the inheritance patterns and treatment options (Wailoo 2001). Alerting the black community to a genetic disease that did not have any treatment possibilities when there were so many black Americans dying from preventable and treatable disease did not seem like the most strategic option for the improvement of black health (Savitt 2007). Particularly when the diseases having the greatest impact on the black community at the time did not carry the same suggestions of inherent flaws in blacks.

Since so little was said about sickle cell in the public sphere, in part due to the assumption that the disease was for blacks only and the limited interest in black health in a white society, only a few articles surfaced between the 1950's and early 1960's. The concept of sickle cell was not in many American's sensibilities during this time, especially African American's, who by happenstance of disease origin and the way racial lines were developed along skin color divides, were the most affected. There were however a few articles that addressed sickle cell, such as the 1959 article "I'm Living on Borrowed Time" in the black publication *Ebony*, which detailed an African-American girl's struggle living with sickle cell and trying to negotiate college

aspirations. In addition there was one in *Negro Digest* in 1953 that was later reprinted in 1963, and one 1959 article in the *American Mercury* that emphasized the importance of getting tested (Savitt 2007). These few articles might have piqued the occasional interest in the disease, but they did not create nationwide awareness. The more widely circulated articles appeared in publications such as *Time* magazine and *Life*, where sickle cell was portrayed as a “discriminating disease” in which African Americans were illustrated as the only ones at risk. The knowledge that the disease was inherited and perpetuation of tie between African Americans served to reinforce the preconceived notions of the separation between black and white blood. The emergence of the disease was in many cases used by white supremacists as proof for the maintenance of miscegenation legislation in addition to other methods of segregation during the mid 1900’s (Nelson 2011).

One notable article of dissent appeared in a 1963 issue of *Phylon*, a quarterly journal for which W.E. B. Du Bois was a founder. This article, “Race, Disease, and Desegregation”, acknowledged the dangers of looking at race as biological, particularly by connecting it with this new disease. The article brings up anthropological research on the impossibility of variation between humans to the point of separate scientifically defined races (Pettigrew and Pettigrew 1963). Humans simply have not been around long enough nor has there been enough separation between populations for distinct subspecies or “races” to exist (Strain et al 2003). It also discusses the social ills that plague those classified as black Americans and the effect of poverty on health outcomes. Nonetheless, this article did not reflect popular understanding of sickle cell anemia (particularly in relation to its racial construction), both within the world of science and outside of it.

A breakthrough mirroring arguments made in the Pylon article seemed possible when the connection between sickle cell and malaria resistance was discovered. Articles such as A. C. Allison's (1954) emphasized the role that environmental circumstance had on the distribution of the sickle cell allele over the “racial specificity” of the disease. In one of the early paragraphs of Allison's article he references the high rates of sickle cell found within Greece and India and describes the possible selective advantage of one copy of the allele in regions with high rates of malaria. Statistics and explanations such as these make sense of the variety of individuals with the disease and reduce the automatic connection to continent based races. Nonetheless, after the introduction, the rest of the article focuses on African (specifically East African) inheritance patterns. Despite knowledge of the diversity of the disease bearers, articles with the opportunity to change the discourse on the disease from race specific to environmentally beneficial in the past, failed to fully do so by focusing more attention and energy to the African roots of the disease. With the increased literature on African inheritance, more fodder was provided to those who argued that the disease stems from “blackness”, and that it only occurs in non-blacks due to pollution of the family line that occurred at some point, even if in the distant past.

Due to sickle cell's association with the black population, there was a distinct lack of serious investment in its research despite affecting larger numbers of people than its better funded counterparts, such as cystic fibrosis (Wailoo and Pemberton 2006). This situation, in conjunction with the fact that many of the victims of the disease fall under the classification of African American contributed to the viewpoint that it was up to black Americans to bring light to the situation. As a result, activist groups, such as the Black Panthers, took it upon themselves to educate and increase awareness about the disease. Extensive campaigns about sickle cell

targeted at the black community inevitably served to solidify sickle cell as a “black disease” in the public sphere during the 1970's. Some effort was made to try and structure the conversations around framing the severity of the sickle cell situation as a symptom of a larger social problems, but as the fight became more mainstream only the basic connections between blackness, sickle cell, and genetics withstood.

The visibility of sickle-cell took off from 1969-1970 when the Black Panthers decided to launch a nationwide campaign. Sickle cell, though not inherent to all black people, did affect many classified in the category of African American, and it was largely being ignored, like so many other aspects of black health. This current and concrete injustice seemed like a logical entry point for healthcare advocacy.

The Black Panthers tackled the issue of limited awareness through mobilizing grassroots campaigns, recruiting celebrity speakers to disseminate knowledge through the media, and opening clinics all over the United States (Nelson 2011). As a result of their efforts, basic information about the disease became common knowledge among the black population who had previously been largely uninformed about the disease. In addition, wide scale testing became available to black Americans at the clinics they set up allowing individuals the benefit of knowing their status. Nonetheless, some worried that the emphasis on knowing if one was a carrier of the defective allele could have implications for eugenics if the only solution was simply that those with a copy of the allele do not mate (Nelson 2011).

In any case, while the disease was intended to be a gateway into discussing black health on a broader scale, there were some pitfalls in the way the Black Panthers and other activist organizations presented the disease. They framed the limited knowledge and funding for disease

research as part of a “black genocide”(1971) in which the white population had been systematically harming and killing off black people beginning with their initial removal from their homeland during slavery to their current neglect for black livelihoods. Though the Black Panthers were not in direct support of the “back to Africa” movement that had become popular in the 1920's by Marcus Garvey, they emphasized the benefit of the allele in Africa and how the change in environment was the originator of the illness (Nelson 2011, Black Genocide 1971). Though the latter half of that statement wasn't true, the benefit that the sickle cell trait provided was genuine and offered a positive side to the portrayal of the disease. In some respects, blacks could have been seen as superior for carrying the trait. In particular, during the time of chattel slavery, blacks suffered horrific conditions constantly, yet when malaria outbreaks hit the south blacks survived at a higher rates than their white slave owners in numbers that almost made the situation biblical (Kiple 1981). This focus on the African origins of the disease and the previous benefit in black bodies (never mentioning the other locations where the allele exists in high frequency), solidified any previous notions that the disease was a “black disease”.

Since so many multifaceted problems exist that hinder black health in America, the Black Panthers used sickle cell as a way to bring attention to black illness in a focused and easily digestible way. The disease lent itself to a number of platforms for summarizing the black experience: it was originally better suited for Africa, it went unrecognized and was undermined, and it consisted of constant, debilitating pain and suffering (Nelson 2011). Black people had been socially and politically oppressed for so long, as the racial categories were created with the goal of stripping the humanity from those of classified as blacks, that sickle cell provided a concrete example of blacks being ignored and relegated to the status of second class citizens.

Unfortunately, this attachment to blackness, despite efforts by the Black Panther Party to portray the neglect of sickle cell as a symptom of larger societal ills, shaped treatment, testing, and funding for disease research in dubious ways.

Chapter 4: Sickle Cell, Race, and Biology: the Aftermath

When a disease is aligned, even in part, with one of the politically and socially constructed categories of race, it both defines and is defined by that category and inevitably reinforces its existence. One example of this interaction is in the way that sickle cell disease's association with blackness has affected the perceptions of the black population. The first connection this relationship enforces is the concept that blackness has a genetic component. Sickle cell is a well known genetic disease that follows a simple inheritance pattern. Though its severity can depend on the expression of the gene in relation to environmental factors, someone is determined to have the disease based on a genetic allele. As a result, sickle cell's link with black people speaks to more than other stigmatized diseases. Unlike tuberculosis or syphilis, which whites could attribute to blacks' hygiene or susceptibility, sickle cell runs deeper, to the immutable core of a being (Roberts 2011). The association of sickle cell with blackness increased the pervasiveness of the ideology that blacks are tainted right down to their genes (Roberts 2011).

The interlinking of blacks and “bad blood” was one of the tenants that allowed race to blossom into an inseparable feature of man (Roberts 2011, Gross 2008). Specifically this concept was used as fodder during the mid-20th century to uphold miscegenation and segregation laws (Nelson 2011). Proponents of these laws argued that the subhuman blood of blacks should not be mixed with that of whites because its inferiority would dilute the purity of white race (Tapper 1999). The concept of bad blood and the terror at its possible infiltration into white bodies gained support and affirmation with the discovery of sickle cell, due to the nature of the disease as a blood disorder and its localization in people considered black.

Examining sickle cell's history as a racialized disease highlights its symbiotic relationship with perceptions of the black population. On one hand, the disease contributed to the reinforcement of popular perceptions of the black people, yet on the other, those very same stigmas of the black individuals affected the way the disease was positioned and treated by everyday citizens and medical professionals alike. For example, sickle cell is under-diagnosed due to the way the disease presents itself in combination with stereotypes surrounding the black population. As described in an earlier chapter, sickle cell disease expresses itself through debilitating pain episodes that can be triggered as a result of the constant degeneration of organs. When the symptom of extreme pain is seen in black people in isolation of other complaints, such as pain in the hands and feet or a fever, it is often not taken seriously, which can lead to a misdiagnosis or non-diagnosis of the disease (Wailoo and Pemberton 2006). This dismissal of black pain, layered with the constant fight black Americans face to obtain recognition as equal humans in the U.S., wreaks havoc on the black psyche. Black bodies are once again undermined when it comes to care in emergency rooms. Though it is well known that the main treatment option during a pain episode is a high strength pain medication, people who come into the ER with regular frequency seeking treatment for pain are often looked down upon because the ailment is judged unrealistic. Medical professionals treating sickle cell anemia patients often confront the question of whether it is more or less ethical to continue to prescribe high doses of what can be viewed as addictive narcotics. Once again, people question the authenticity of the pain and assume that because many of the patients in inner cities are poor black Americans, they will inevitably misuse the medication (Wailoo and Pemberton 2006). This stigma surrounding drug addiction and the general mistrust of the black community, gives medical professionals the

opportunity to decide who is worthy of treatment, and in turn define who is a morally responsible being. The questioning of integrity, whether or not it is racially motivated, contributes to the long history of dis-empowerment within the black community. Stigma also contributes to structural social norms surrounding black Americans, such as the criminalization and devaluation of their bodies.

The dichotomy between a chronic illness in need of expensive constant care and the low socioeconomic status of the population most affected by sickle cell disease has led to further implications about U.S. spending on healthcare. Comprehensive treatment for sickle cell is costly, especially the high strength prescription pain medications, hospital stays, antibiotic cycles, and other treatment options. As so many of the patients seeking this treatment happen to be poor and black due to historical race constructions and circumstance, many patients' expenses are covered by medicaid, which was particularly true during the 1980's and early 1990's (Wailoo and Pemberton 2006). However, this level of government assistance was often chided by the fiscally conservative community as excessive spending and some of those in office looked to cut back during this same period (Wailoo and Pemberton 2006). The desire to reduce funding for public healthcare programs that were thought to benefit black Americans was exacerbated by the increasing prominence of poor-shaming and “welfare queen” rhetoric, the concept that black women were living in luxury off of the government's dime while contributing nothing to society (for further reading on origin of the Welfare Queen see Ange-Marie Hancock's *The Politics of Disgust: The Public Identity of the Welfare Queen*). The idea of the welfare queen is not true for multiple reasons, including the fact that the majority of people who receive welfare are white (BLS 2012), yet the association of sickle cell with both long term health care and blackness

provided more ground to argue that once again blacks were siphoning off money from the state. Sickle cell has been used as a tool throughout history to shape and enforce societal norms around race.

In addition to access to treatment, the actual treatment options themselves were marred by the association with the historical category of blackness. As noted in an earlier section the only real treatment options generated surrounding for sickle cell were urea, which was an unsuccessful desickling agent, hydroxurea, which met some success by producing hemoglobin that does not sickle, blood transfusions, which only provide short term relief, and bone marrow transplants, which are unreasonably risky (Linde 1995). The most research surrounding sickle cell anemia centers around its role as a pivotal genetic disease. Due to its simple mutation and the well understood inheritance pattern it seems researchers did not fully engage in finding treatments to relieve the symptoms and looked more definitively toward eradication. This may not be true, as there were a few drugs that came out of sickle cell research such as urea and hydroxurea, nonetheless, neither one has proved to be a long term effective treatment. It is possible that because of the complicated history of black Americans with healthcare, few treatments came to the market for fear they would not be well received unless they were proven to be effective. Many black Americans were distraught in the 1970's after the exposing of the Tuskegee debacle in which black men in the rural south were used as guinea pigs to examine the effects of syphilis (Washington 2007). Men in this study were infected with the disease but not given treatment as a way to examine the course of the disease in black bodies, extending as far as death. As a result, many black Americans were not highly receptive to the newest fad treatments and wanted to use medications that were well tested and supported (Wailoo and Pemberton

2006). This sentiment was felt particularly after urea was marketed as a premier de-sickling agent only to disappointment by providing only short term relief, if even that. Once again when one thinks about treatment options, the population most affected by the disease, lower class black Americans-as currently defined, must be taken into account. As a result, even if effective, expensive treatment options like gene therapy and bone marrow transplants are brought to the table, they might be unrealistic. The amount of funding going toward individualized high tech initiatives seems like a mismanagement of resources when due to an ineffective healthcare system much of this population does not have a basic level of healthcare.

This brings about the issue of funding withing sickle-cell research in general. Overall, sickle cell research has historically been severely underfunded when examined next to its comparable genetic disease counterparts (Wailoo and Pemberton 2006, Nelson 2011). In her book, Nelson (2011) describes an article exposing the differences in funding provided by the National Institute of Health for research of genetic disease. The disparities in funding one again exemplify the social and political ramifications when a disease is associated with a subordinate race. This observation is not to say that millions more in grants should be poured into researching sickle cell disease, because if one truly considers the state of black public health, funding an uncommon genetic disease is not going to be the fulcrum on which society begins to balance out.

Nonetheless, in part due to the commitment of the Black Panthers to bring sickle cell to the forefront of American consciousness in the early 1970's, changes in funding and policies ensued around this disease. The most notable passage of legislation occurred in 1972 when Nixon issued the "National Sickle Cell Control Act"(Nelson 2011). This act increased funding

significantly to the research of sickle cell and to the implementation of health programs aimed at testing, counseling, and treating those with sickle cell and carriers of one copy of the allele. Though the creation of this legislation seemed like a significant win for black Americans, particularly the Black Panthers who had been fighting for recognition of the disease over the preceding three years, it missed the point of true racial justice that the Black Panthers were trying to keep central to their campaign.

First of all, it changed the lens through which the projects were carried out, overlooking the overarching social problems generating black sickness. In particular, Nixon's passage of this bill was portrayed as an effort to tackle black health inequity, but sickle cell was not the origin of this issue. In his speech on the subject he discusses the longstanding neglect of the black community, but continues on to make it seem as though increasing funding to sickle cell (a disease misconstrued as a main factor in everyday black health), would right past wrongs (Nelson 2011). His speech was particularly ironic in light of the fact that to increase funding to this project, he significantly slashed the budgets of social programs that affected the livelihoods of many more black Americans than those affected by sickle cell disease (Nelson 2011). This misguided effort to help "black Americans" was a direct consequence of attachment of sickle cell disease to the black race, and illustrates the reciprocal relationship between perception of the disease and perception of the population affected by it. It is also argued that this effort by Nixon had the end goal of improving his black vote for the next election (Nelson 2011, Washington 2007). The manifestation of blackness in this single disease led to a reductionist viewpoint of the race, ignoring the social and political creation of the races, and therefore the more complicated consequences in that arena. It is much easier to tackle a difficult multifaceted problem when it is

made to seem as the result of a single factor. It would have been much more challenging to work on social causes of inequity, but it would have been more significant. The epitomizing of “black illness” in a singular, specifically genetic, disease obscured the social relationship between disease and race and completely blanketed the possibility of understanding race as socially constructed.

Nixon offered blacks the possibility of bio-citizenship at the expense of true citizenship. Explained further, through testing for the trait, black Americans gained biological knowledge that provided them with control over an aspect of their health, in addition to provide them with the ability form communities around the trait, but this line of testing did not actually improve their position in society. In his speech announcing the act he states, “In February 1971, I pledged that this Administration would reverse the record of neglect on this dread disease”(Nixon 1972). Though he is directly referring to the disease in this statement, by increasing funding to the sickle cell process, it seems as though the intention was that blacks would feel cared for by the medical community and included in the most up-to-date medicine. This focus on genetic medicine as the cure-all for health disparities persists today, and in many portrayals of an imagined future, where we are categorized by genotypes and not by race. For example, there is a film entitled “GATTACA” that portrays a future in which everyone's genome is sequenced at birth and as a result all information about the person, right down to how and when he or she will die, is recorded in a database. Of course this period is not in the near future as there is so much to learn about the human genome, not to mention the critical role that the environment plays on the expression of genes. Even if it were possible to have everyone's genome sequenced at this time and subsequently have treatment catered to the individual, a problem with this concept still

arises in how current divisions (such as gender and race) are to play out in that future. While the film *GATTACA* claims that the only discrimination that will exist in this imagined future is genetically based, how we would get to that point other than simply ignoring race is not explained. I question whether a post-racial society is a possibility, because even in the imagine version that *GATTACA* provides, nearly all of the main characters are white and in general have greater representation in the movie. The level of bio-citizenship that *GATTACA* suggests, a world where we strictly define ourselves and relate to each based on our genes, could lead to a level of colorblindness where any racial inequity is not considered, on top of the erasure of the history and national memory of race itself. Prior to moving ahead to a world where race is no longer relevant, we must combat racism on the institutional level and must reconcile with its current manifestations. We must work toward a just society in which every member has the same life expectancy and exposure to opportunities at birth and the human rights of all people in the U.S. are recognized. True citizenship can only exist in a U.S. where everyone's human rights are met, including the rights to housing, job security, and education. Genotyping the population does not redistribute wealth or change the language we use to create the "other" in a way that would make that world possible, only through building communities and coalitions can steps truly be taken to citizenship.

Race in medicine cannot be simplified to race and genetics because there are many more factors that influence differences in health outcomes between the races, and these areas cannot easily be controlled for. On the opposite end of the spectrum from sickle cell anemia, which is a fixed genetic disease with social repercussions, there are diseases that are not just slightly influenced by social and environmental conditions, but actively developed as a result of their

influence. Three major factors that can lead to an increased risk of illness are poverty, sexism, and racism (Marmot & Wilkinson 2007). These three factors consistently intersect to produce high stress environments that often result from limited resources and little support. So many social determinants of health (conditions in which people live and develop) are impacted by poverty that simply knowing one's socioeconomic status one can predict that they will have a higher risk to develop more serious health problems as compared with their wealthier counterparts (Marmot & Wilkinson 2007). The correlation between poverty and health is so distinct that socioeconomic status serves as a channel through which one can predict health outcomes all the way down the chain (Adelman et al 2008, Marmot & Wilkinson 2007). Poverty results in decreased access to healthy food, adequate healthcare, and well-funded schools, and with the limitation of these options arises a feeling of a lack of control over one's life and decisions. Whereas those who are the managers in organizations have the ability to choose what projects to work on or the order of their schedules, unskilled workers often do not have this freedom and generally have to follow the rules and whims of their supervisors. In addition those who do not have the capital to move where they chose are generally subjected to the schools and resources that their current neighborhood has to offer.

In cities, many neighborhoods are still segregate and internally homogenous both racially and socioeconomically (census). Instead of a concept of integrated housing, there are neighborhoods that consist of the wealthiest members of society just blocks from the poorest members of that same area. As a result of biased housing practices and home construction there is systematic discrimination in housing which makes it very difficult to exercise freedom of movement. In addition, high concentrations of poverty can lead to higher crime rates and a more

unstable neighborhood climate as everyone is competing for the same resources (Sampson et al 1997). The resources themselves are very limited which only increases the competition. In addition, due to neglectful policies around zoning, poor areas often contain endless fast food restaurants, pawn shops, and beauty supply stores, but very few, if any, well stocked grocery stores (Adelman et al 2008). Instead of passing laws that limit the number of lottery stores (and comparable entities) allowed in the area, or increase the number of supermarkets, space is not delineated, stores repeat, and options narrow. The frustrations of limited choice arise again as people are forced into a corner.

The physical environmental factors, such as dilapidated housing, often coalesce with the emotional pressures of limited options and resources, to lead to a perpetual state of stress. This constant activation of psychological systems to stress over time increases creates “wear and tear” on the body, increasing the likelihood of future negative health outcomes (Evans and Schamberg 2009). Systems that are particularly affected by the steady high stress are the cardiovascular and regulatory systems. For example, high levels of cortisol (stress hormone) can affect blood pressure, mood, and one's ability to regulate glucose. While the level of cortisol in one's system may not be significant enough to notice on a daily basis, the consistent exposure can have an additive effect, leading to long term illness such as hypertension or diabetes which are experienced at higher rates in those of lower socioeconomic status (Adelman et al 2008).

Due to disproportionate levels of poverty and the added factor of racism the experience of constant stress is even more salient among the black population. Over 25% of black people in the U.S. live below the poverty line, as compared with approximately 11.5% of white people (Macartney et al 2013). All people that live in poverty experience severe stress, but the added

factor of institutionalized and internalized racism serves to amplify it significantly. The increased psychosocial stress that blacks experience is in part due to microaggressions, small acts of racism and discrimination that people of color experience on a daily basis throughout their lives. These occurrences were not always recognized, and it was a pivotal moment in health disparities research when it was realized that health outcomes are directly impacted by these subtle forms of racism. One prominent study in providing the base for this understanding was one conducted by Richard J. David and James W. Collins in 1997. In the study they were searching for an answer to the question of persistently high rates of low birth weight babies among African-American women. To do this they studied the birth weights of white women in comparison with African-American women and recent immigrant women from Sub-Saharan Africa. The idea of studying African women in conjunction with black ones was to see if low birth weight was the result of environmental factors of living in the U.S. or genetic ones (it was assumed that the African and black women had related genetics). What the study found was that white women and recent African immigrant women had babies of relatively similar healthy birth weight whereas the black U.S. born women had babies of low birth-weight. In addition, as socioeconomic status improved among the the black American participants, the disparity persisted. These results indicate that there is a difference in experience not just between black and white women, but also between black and African women that causes low birth weight babies. The findings also negate their theory that there is a genetic link between all black and African women, a point which though proven in science is not always heeded to. As David and Collins recognized that the disparities in birth-weight were not genetic, they looked to environmental factors, and concluded that it must be the particularly insidious form of racism in the U.S. While this study made the

problematic assumption that African women and black women in the U.S. share a genetic history, what it found disproved that assumption and provided concrete support for the way race in the U.S. has become an inescapable component of our everyday lives with serious health consequences.

As a result of the intangible compounding factor of racism, studies that try and control for race are ineffective. Occasionally studies will control for socioeconomic status or income as a proxy for race, assuming that the only manifestations of it are in class. While class has a significant role in the black experience, it clearly does not comprise of all of the disparities that blacks and whites experience and therefore provides an insufficient place holder for race (Lu et Halfon 2003). In addition, studies that control for socioeconomic status as a proxy for race are often trying to create a platform on which to prove an objective difference between the races, such as predisposition to a disease or a propensity to have certain abilities. This process is ineffective because the basic social markers of racial disparity do not explain the language and culture of racism that manifests itself in intangible but influential ways throughout society, both interpersonally and through institutions.

There is a deep contradiction in the way races are used in science and the way they are defined. Race is often assumed to be a set of standard and accepted categories at the beginning of a study, yet the only way to separate subjects into them is through self-identification. The fact that there is no definite way to demarcate between the races other than self identification should exemplify the ambiguous and incongruent nature of race. Depending on context one's race can change, especially for those who are multiracial or have physical features that are otherwise

racially ambiguous. As a result, the self-identification process is unscientific in its very existence, as it necessitates a high level of subjectivity.

A number of authors, such as Roberts (2011) and Jonathan Kahn (2012), have previously illustrated how race continues to live on despite the move into a genomic age. They argue that race has morphed into a new entity that is not challenged by the commonality affirmed through genetics, but is instead further supported by it. This is not through direct endorsement necessarily, but through the use of racial categories as objective groupings that are simply the base for other studies and need little explanation. For example, certain experiments will begin with research questions and hypotheses that seek to find the genetic explanation for disparities between the races. This line of questioning is problematic because it legitimizes the assumption that there is already a genetic component to race, a jump many make due to perceived scientific support from examples such as sickle cell disease. Once this initial assumption is made and people are divided into these categories, one is bound to find both differences between them, but they will find nothing that links every person within one race nor should they look for a factor. In one of her speeches, Dorothy Roberts describes how the discovery of minor statistical difference would be the case in any system of organization, whether we were divided by height or by shoe size. At least if we were divided by certain shoe sizes there would be an explicit factor that connects that each subgroup group together, a feature that is not found in the description of race. Regardless of how humans could be divided, a number of scientific studies currently use race, and as a result, any findings, whether similarities within each grouping or differences between them, serves to further support the existence of separation along racial lines.

In some respects, scientists appear to be still searching for the answer to Thomas Jefferson's original "call to science", in which he challenged researchers to find scientific support for the obvious hierarchy of the races. It is possible that this call came from a place of guilt at a recognition of the inhumanity of slavery at the time, or a place of greed that necessitated a justification of slavery while in the midst of demanding of a free nation. While perhaps to a lesser degree, studies today that try and attribute disparities between the races to genetic differences may be similarly searching for a way to explain inequity in a way that does not self-implicate. During the 1700's many people were operating within a framework that upheld beliefs of the clear inferiority of the black race; while many white Americans today make serious efforts not to appear overtly racist, subtler manifestations of this same racist ideology persist (Bonilla-Silva and Forman 2000). For example, we commonly accept studies that search for racial "predispositions" to disease as helpful to the populations they study because the ultimate goal is understood to be a path toward prevention or new treatment and not simply the degradation and stigmatization of the race as would likely have been the outcome of a study like that in the 1700's.

Nonetheless, though likely unintentional, the quest for, and discovery of, these genetic predispositions continues to make certain races seem inferior, whether it is through attaching them with an increased likelihood of heart disease or lower survival rates from cancer. Though these disparities may exist between the races, the root of the differences cannot be attributed to a genetics because as argued throughout this thesis, race has been continuously constructed and deconstructed over time and is arbitrary at its core regardless of coincidental similarities along

these lines. To study genetic links is to search for something that cannot be found, specifically on a wide enough scale within races for it to be a defining feature.

Despite a the lack of a genetic link, it is easy be deceived into thinking race and has a genetic component; it does not require that someone be intentionally racist, nor does it require that that he or she be white. In fact, there are black Americans that make the same assumptions about the races, though because of their inherently disadvantaged position in this belief system it is possible that viewpoint is a result of internalized racism, the set up science education, or the presentation of illnesses like sickle cell anemia as race-specific. One example of black people supporting this type of research occurred shortly after the human genome project first illustrated our similarities. The case was the release of BiDil, a African-American specific heart medication. This medication was targeted at black Americans under the guise of individualized medicine (Roberts 2011, Kahn 2012). It was portrayed as specifically effective on blacks with high blood pressure though who was defined as black or why it worked this way was never explained. In fact it was later reveled that the clinical trials involving BiDil only tested the medication on black Americans; therefore it was never fully understood whether or not it would affect other races in a similar fashion (Kahn 2012). It was also realized that generics of BiDil already existed on the market as heart medications that were applicable to everyone, so the ploy to make it a “race-specific” drug was unfounded and simply a play to exploit a target population (Roberts 2011).

Nonetheless, the NAACP along with a number of other prominent black American individuals and organizations, publicly supported the release of the medication because it showed an appreciation for the black condition (Roberts 2011). Just as was the case when the Black Panthers fought for recognition of sickle cell anemia only to find out that their support in some

ways detracted from their overall mission a similar situation happened regarding BiDil. Many black Americans were excited about the release of BiDil, because heart disease is a constant threat to the black community due to the factors discussed earlier, and a drug made specifically for this population seemed like acknowledgment of the disproportionate statistics of heart disease in blacks (Roberts 2011). What they failed to keep central to their vision of addressing heart disease in blacks was the fact that race is not based in the human genome and therefore the disparities cannot be addressed by a medication. The knowledge that heart disease affects blacks more than whites was important however, just as it is significant that sickle cell anemia affects significantly more black Americans than white ones. However, it is the framing of that knowledge and the further research that comes out of the it that's important. When research about health disparities centers on genetics, it depoliticizes the inequality, and makes the roots of issues seem written in our genes instead of our legislation, a problem that the black panthers encountered after organizing around sickle cell anemia for a number of years.

Conclusion

It is a continuous process to determine the contexts in which race can and should play a role in science and the pathways to racial justice. Sickle cell provides a case study to explore the various ways race intersects with science and the lasting effects and repercussions. For this disease, the connection with race has been more of a barrier for steps toward racial justice than a benefit. This is the case in large part due to sickle cell anemia's existence as a genetic disease. It

takes very careful framing to present a genetic disease as a racial concern and struggle without inevitably drawing a link between the genetic make up race and the disease. Of course, throughout recent history, there have not been many genetic diseases that align so noticeably with an already existing race, especially a subordinate one. Further, this alignment was discovered during a time when racial discrimination was overt and consistent, so it makes sense that mobilization was required to get recognition of the disease by the black and white communities alike. As power was built around the disease, and its portrayal as a beacon of the black condition grew in popularity, the fight for recognition lent itself to mainstream repackaging. The presentation of the disease as the tip of the iceberg on the path toward racial equality faded, and the focus shifted to how to best address the disease alone. As a result, the understanding of sickle cell anemia as a “black disease” became common knowledge and formed one of the most prominent arguments for proof of the separation of the races. This entrenched definition of race as a naturally occurring system of organization has laid the groundwork for research that continues to study the differences between the races.

Controversy over the handling of sickle cell in the public sphere continues transpire today. One recent source of controversy is the newly passed National Collegiate Athletic Association (NCAA) mandate of sickle cell screening for Division I, II, and III athletes. This mandate was passed in direct response to a lawsuit filled against the NCAA by the family of Dale Lloyd II after his sudden death following an intense football workout at Rice University. His death was one of 10 since 2000 that have been from heat exhaustion in connection with sickle cell anemia (Pasquerella 2012). Though considered a mandate, this testing requirement does not actually necessitate that everyone get tested. It provides three options: get tested at the

beginning of your incoming season, submit the results of a previous test, or finally, opt out of the test. A number of critics understand the last option to be an illustration of the college's lack of real concern for their athletes and sole interest in protecting themselves from lawsuits (Pasquerella 2012, Quick 2011). Since there is an opt-out option, in addition to limited privacy of one's results, people might actually be deterred from taking the test who otherwise would.

In fact, the right to privacy is one of the strongest complaints about the new policy because these test results carry implications that can affect one's livelihood if found out (Quick 2011). The NCAA does not have a solid set of guidelines in place to protect athletes' genetic information, especially in light of the fact that a number of schools require their athletes sign waivers to allow medical information to be released to the media (Quick 2011). As a result, if athletes get tested, their results may become widely available, which could result in discrimination and affect future employment opportunities (Pasquerella 2012 Quick 2011). For example, professional football player Cam Johnson was not drafted until the 7th round, not due to a lack of superior abilities, but because he possessed the sickle cell allele and therefore a possible liability (Kahn 2012). The vast majority of sickle cell trait carriers do not experience any symptoms from having the trait, and unless tested, many will be unaware they are carriers throughout their lifetime. As a result, there is only a very small possibility that serious complications will arise, which means that the carrying of the trait should not negatively impact one's career as severely as it did to Cam Johnson, since it does not pose a serious threat to his ability to perform. Other health factors, such as congenital heart defects, pose a greater threat statistically, yet there is no standardized testing for this risk factor (Muller & Colgate 2013). In

2012, heart related deaths accounted for over half of indirect football related fatalities (7 out of the total 13), which when compared with heat stroke, at less than 8% of that number (often linked with sickle cell), seems to be a much greater treat (Muller & Colgate 2013). In fact, currently, heart-related emergencies pose a great enough risk that an Automated External Defibrillator (AED) is expected to be on site at all school sporting events (Muller & Colgate 2013).

Not only does sickle cell trait only physically affect a small number of its carriers, with simple modifications to training methods used in practices, the risk for an sickle cell crisis among that small population can be drastically reduced. Beyond that small group, these changes in routine would create a safer training environment for all athletes, especially those who have other risk factors for illnesses. Some modifications include, consistent and frequent hydration and building up to high intensity activity. One entity that implemented a training regime to account for hydration and difficult but reasonable conditioning, is the army (Thomas 2010). The army no longer tests for sickle cell due to these changes and have not suffered dire consequences. In fact, they have had an overall positive experience because fewer people have to be singled out from participation, which provides a more inclusive environment (Thomas 2010).

Race is noticeably left out of the discussion around testing policies, changes to exercise regimes, and sickle cell stigma within sports, but it is important to recognize that policy on this disease has disproportionate impact on black Americans. Though the common academic definition of sickle cell separates the disease from race, the current perception of the disease still supports a strong tie between race and illness (NIH). As a result, stigma around the disease and testing will likely target black athletes more than their white counterparts. In addition, the public

nature of the testing and the possible impacts on future employment will likely be more acutely felt by black Americans, who are often at a disadvantage in the job market before they hand in their first application. Finally, as stated multiple times throughout this thesis, the actual number of black sickle cell allele carriers is in fact greater than most other races, though this concentration is a result of coincidence. With the increased proportion of carriers, all of the downsides of the previous policies are disproportionately felt within the black population.

Past experience tell us that if race and illness are presented as influential on one another it is easy for ordinary citizens to misconstrue the relationship between the two, especially when it is a genetic disease. Nonetheless, when appropriate, there are ways to incorporate race into the response to a disease in a way that combats racism. Public health has the space to do this type of work, since it is often social justice oriented in nature (Ford & Airhihenbuwa 2010). Public health is addressed through communities and therefore requires the use of divisions among people to be effective. Initiatives along these divides might be as basic as looking at smoking in a specific neighborhood, or as broad as the effects of obesity in the black population. The question of whether there is a benefit to the use of racial categories is grappled over by Keith Wailoo, a frequently cited expert on the history of stigmatized disease in the U.S. He takes a positive approach to the use of these groupings in the effective control of disease and describes the communities that form out of the struggle for recognition as beneficial in some circumstances because they allow groups to take ownership of an illness. Marginalized groups can gain a sense of agency when they feel as though they are united for a cause and the campaign may extend to larger ones addressing better health for all. It is unclear, particularly in relation to sickle cell anemia as a genetic disease, whether targeting racial groups enforces the notion of biological

race, or if it helps to address one manifestation of racism. Nonetheless, for diseases that take place at high rates in certain races as a result of social and environmental factors, the link with race is important to recognize and document, as it is a clear marker of the unequal state we live it. In addition, recognized racial disparities allow space for coalitions to form among those affected which provide a network that allows for active resistance.

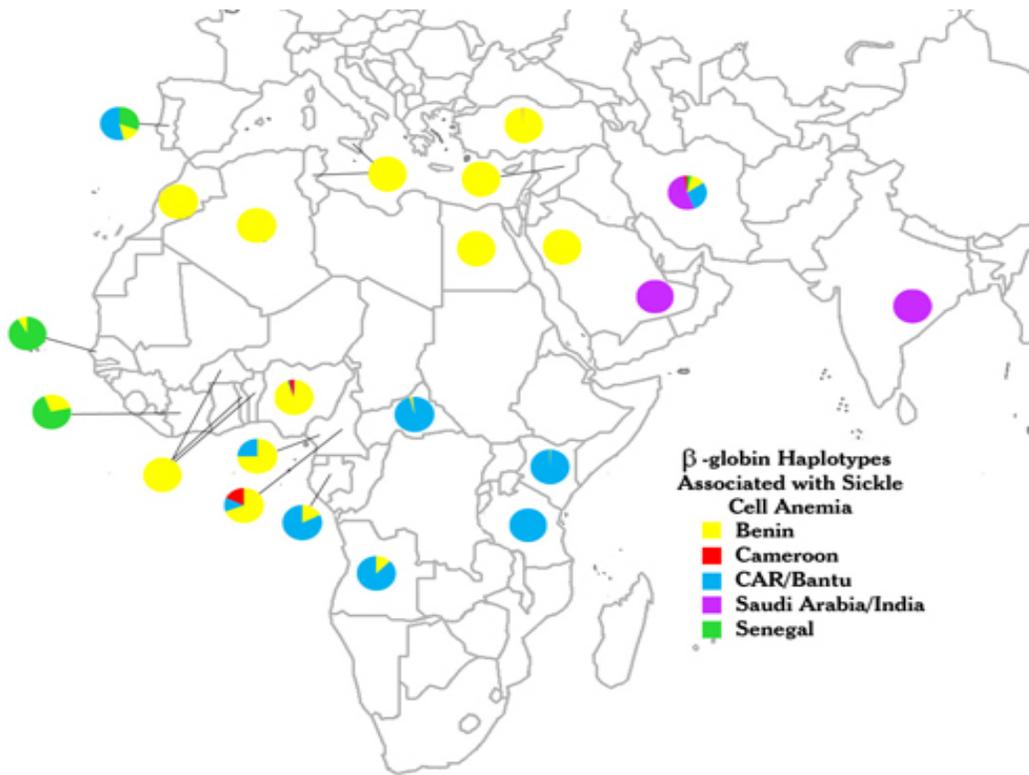
The transformation from a demoralized group that suffers more than others to an empowered people fighting back can occur as a result public health research, particularly public health research that applies a Critical Race Theory (CRT) framework. CRT centered public health focuses on the approach of research and the recognition of race and power dynamics in how studies are carried out. When studying race, one of the first steps is understanding that one's subjects come from their own racialized contexts, and that neither you nor them has an objective interaction with the subject being studied (Ford & Airhihenbuwa 2010). As a result, what part of a topic is addressed and how the study does this is important. For example, the framing of a study as “why this population is resistant to sickle cell testing” assumes that the initial population is intentionally avoiding the act of testing, which based on the socioeconomic status of the area, in addition to other factors such as time constraints or a lack of information, may not be the case. Some groups may know about the disease and not act because they disagree with the principles behind the test, whereas others may simply not have the time or energy to think about taking care of themselves. Without understanding the population one is studying, it may be unclear what question is most pertinent to ask. Asking relevant questions increases the chances that the community will eventually mobilize for justice, a goal that extends beyond documentation of disparity into future equality.

Another important aspect of a CRT approach to public health is understanding one's structural position when entering particular spaces to carry out studies. One paper describes a researcher's initial assumption that because she could be easily identified as black, she possessed the ability to navigate a poor black community. She later realized that her agent identity, as a college educated researcher affiliated with a private university, prevented a basic level of trust from forming (Ford & Airhihenbuwa 2010). Due to the stress this mistrust may cause to those around her, the researcher's presence in the community could be considered more harmful than helpful, and as a result, the bodies best suited to carry out the study were reevaluated (Ford & Airhihenbuwa 2010). Considering the population's wishes, in addition to intentionally incorporating them in the carrying out study, exemplifies health disparities research that does not reinforce racial hierarchy and by providing the subjects with a degree agency. Public health research that is actively oppositional to genetic assumptions highlights our similarities and provides a space for collective resistance to the current structures that maintain social inequity.

The understanding that race cannot exist genetically as currently defined provides us with the basis to see the common humanity of our neighbors, regardless of our initial perceptions of one another, a feature that Dorothy Roberts (2011) describes as integral to ending racism. Once a sense of connection is established, then role of society in the maintenance of inequity becomes more clear and the sense of social responsibility can develop. Social inequality cannot be attributed to faults in character and therefore must be seen in relation to one's privileges. With this sense of self-implication, true anti-racist work can begin, specifically research that goes further than simply documenting the issue to the level of disparity to actively organizing against it. Nonetheless, it is important that colorblindness, the belief that race is no longer relevant, not

develop from knowledge that race is superficial. As said in the beginning of this thesis, race, though not biologically based, has visible and tangible affects socially and politically and therefore must still be addressed in those realms. Active anti-racist work and organizing provide outlets of resistance especially as science continues to recreate a biological origin of these categories. If targeted at the researchers carrying out these studies, anti-racist work could lead to a paradigm shift in the way that racial difference is addressed in science, from an assumed genetic basis to an understood social and political consequence. Instead of a focus on race and difference, as Dorothy Roberts argues, genetics could focus on finding out more about our similarities, such as how the genes themselves function. This shift is necessary for current inequality to be seen as necessary to address on a large scale.

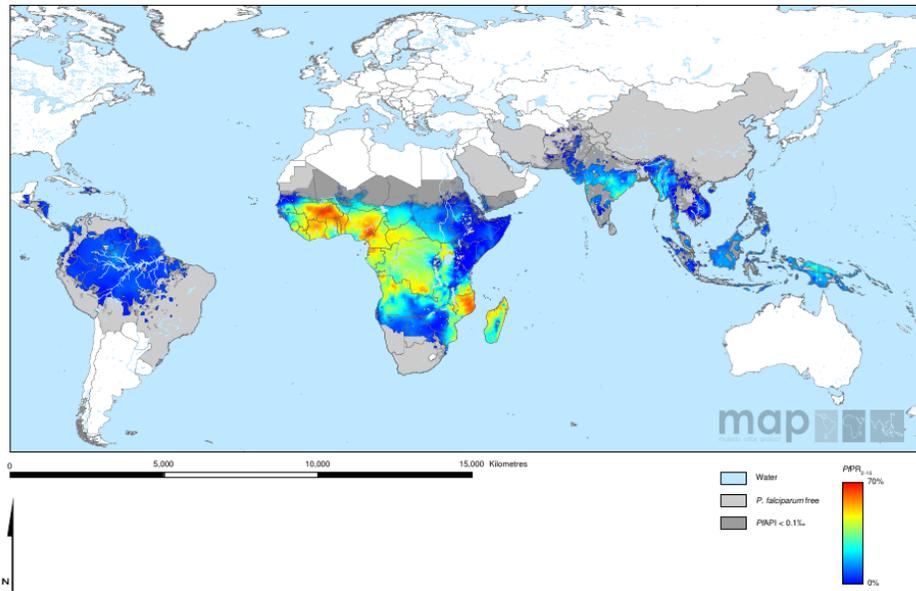
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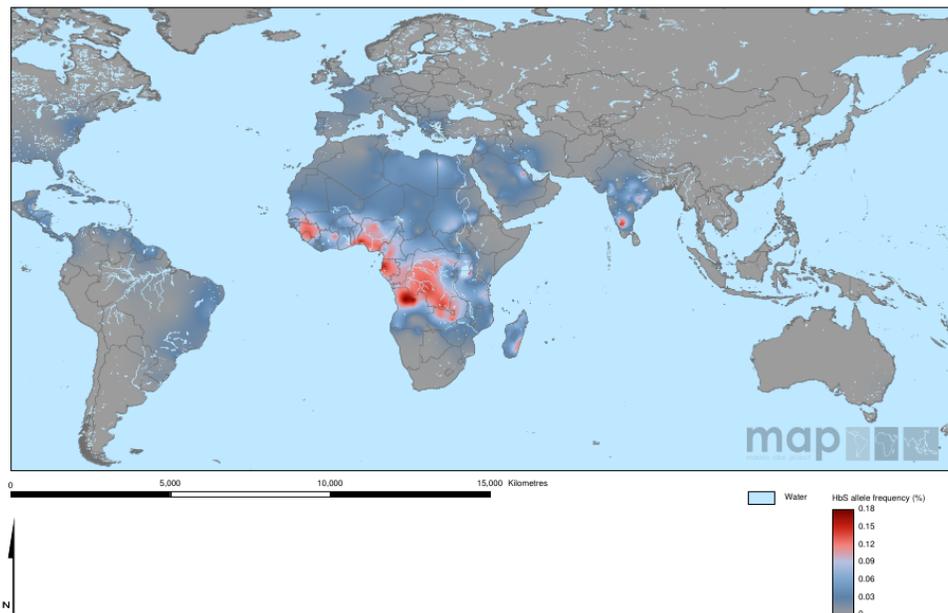
Gabriel, A., & Przybylski, J. (2010). Sickle-cell anemia: A look at global haplotype distribution.

Appendix 2:

The spatial distribution of Plasmodium falciparum malaria endemicity map in 2010 globally



Mean estimates of sickle haemoglobin allele frequency map in 2010 globally



<http://www.map.ox.ac.uk/explore/about-malaria/malaria-maps/>

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