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The Epistemic Usefulness of Race in Approximating Diseases in Medical Genetics

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Abstract

This paper critically investigates whether there is a racial scheme that is epistemically useful in approximating disease in biomedicine. Quayshawn Spencer, Risch *et al.*, and Burchard *et al.* argue that racial schemes are classified based on the different continental populations that exist according to the US census data of 1997. These continental populations have diverse genetic differences that could account for various diseases that exist in the different populations. I argue contra this view from Quayshawn Spencer, Risch *et al.* and Burchard *et al.* It is my contention that race is not genetic and/or biological. I contend that race is socially constructed due to some social features; as a result, this social constructivism of race gives clear epistemic insights about diseases and how they can be approximated. I conclude that what these theorists conceive as genetic diseases is mistaken. Instead, these diseases occur due to the divide amongst the different populations based on social grounds such as skin colour, financial affluence, and environmental differences.

Keywords: Epistemic usefulness. Racial scheme. Medical genetic. Genetic-based race. Folk race.

Introduction

Whether any racial scheme is epistemically useful in medicine, especially medical genetics, is debatable in the biomedical race literature. Michael Yudell, Dorothy Roberts, Rob DeSalle, and Sarah Tishkoff (2016) argue that using the “biological concepts of race in human genetics is problematic at best and harmful at worst” (2016: 565). They argue that racial groups are heterogeneous and lack clear-cut genetic boundaries (2016: 565).

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On the contrary, Neil Risch, Esteban Burchard, Elad Ziv, and Hua Tang (2003) argue that racial categories are useful in medicine because genes are susceptible to diseases or variations in drug response are based on genetic diversity (2003:11). Furthermore, for Risch *et al.* (2002) and Burchard *et al.* (2003), racial classification is important in biomedicine to understand the causation of some diseases by using racial categories to generate hypotheses to account for essential risk factors. In contrast, Jonathan Kaplan (2014) argues that folk race should be used in medicine to approximate the socioeconomic and environmental causation of diseases.

This paper answers the question of whether there is a racial scheme that is epistemically useful in medicine and especially in medical genetics. I argue that the folk racial scheme, the social construction of race (Kaplan 2014), is epistemically useful in medicine, especially medical genetics for researching, approximating, and treating diseases caused by socioeconomic and environmental issues.

It is my view that race is important in the field of biomedicine and other medical research due to the insights we can get into some diseases due to the racial divides in society. From here, it is not immediately clear how we can correlate disease and race; however, I make this point apparent in section two of this paper. Furthermore, this paper contributes to the literature on biomedicine and the philosophy of medicine by making a case of a particular conception of race, folk race, which plays a significant role in researching, understanding, and approximating diseases in some populations that are not present in others.

I structure this paper as follows. The first section of this paper clarifies the important terms that I use, such as epistemic usefulness, medical genetics, and racial scheme, in order to contextualise these terms to the issue discussed here. In the second section of this paper, I defend my claim that the folk racial scheme is robust enough to warrant epistemic usefulness in medicine, especially medical genetics,² in order to explain the causation of diseases. To arrive at this conclusion, first, I show why a racial scheme is needed in medicine. Second, I argue by analogy to show that the widespread view that the genetic-based racial scheme is sufficient to explain epidemiology lacks robustness. I eliminate and replace the genetic-based

² In my argument, I first show why the genetic-based racial scheme cannot be used to approximate disease. I do so in order to eliminate and replace the view with a more robust conception of race- folk race.

racial scheme with the folk race to show that the latter best explains the cause of diseases more than the former. Finally, I look at a possible objection from a critic. For example, a critic might argue that if a medication is effective in a population to cure some diseases and the same medication is not effective in another, we can postulate a genetic-based racial scheme to explain the disparity. I respond to the above objection by arguing that the objection suffers from what is conceived as the reification and granularity problem.

Clarification of Terms

In this section, I begin by clarifying the key terms that I use throughout this paper. These terms include epistemic usefulness, medical genetics, racial scheme, and biomedical race debate. I do this to contextualise these terms and relate them to the central problem of this paper.

Epistemic Usefulness in Medicine

Epistemology, the study of knowledge, has been used differently in different fields but with a particular outcome: understanding how knowledge is produced and gained. In medicine, epistemology has been used to understand the causation of diseases and how to treat these diseases (Khushf 2013). In other words, epistemic usefulness in medicine means what biomedical scientists, clinicians, and care practitioners know as individuals or as a group concerning the medical sphere (Khushf 2013: 461). The activities associated with knowledge production are essential to biomedical and clinical literature. Thus, developing an epistemological framework to understand and examine the causal relationship of disease plays a vital role in diagnosing that disease.

For example, in Evidence-Based Medicine (EBM) and Medical-Decision Making (MDM), the use of normative epistemology is relevant to account for how knowledge ought to be arrived at and used (Khushf 2013: 463). The normative epistemology is vital in medicine for at least two reasons. First, normative epistemology involves claims about how clinicians ought to make judgments involving logic and the necessary justification in distinguishing knowledge from presumed knowledge (Khushf 2013: 463-4). Second, EBM gives guidelines on

reflecting substantive judgment about what ought to be considered good and valuable and how to rank the outcome, especially the ethical obligations they owe to patients (Khushf 2013: 464; Smart 2017; Broadbent 2019).

In applying races, genetic-based or folk racial schemes, to medicine is to know how best races play a part in understanding the causal relationship between diseases and how clinicians ought to make judgments about these diseases on the one hand. On the other hand, it is to know what evidence best supports the role that races play in making a disease without clinicians making unethical judgments. In the second section of this paper, I show that the best way to understand the causation of disease is to use the best available evidence that supports the causal link between a disease and a population. In this case, only the folk race provides such best available evidence for the diseases mentioned in this paper.

Medical Genetics

Medical genetics is a branch of biology that is precisely concerned with studying the Deoxyribonucleic Acid (DNA) of organisms and showing how DNA manifests itself as genes and how these genes are transferred from parents and inherited by their offspring (Greenwood 2021: 1; Richmond 2021: 2). Genes are passed down or inherited by parents to offspring through sexual or asexual reproduction. Over time, natural selection can accumulate variations amongst people of a group through evolution (Greenwood 2021: 1). This transfer of genes through reproduction is what some theorists use as a yardstick to account for the different races on some biological grounds. As it will become evident in this paper, I argue otherwise; I argue that there is no correlation between genetics and race, especially in the approximation of diseases.

Racial Scheme

Racial schemes are racial classifications of human beings according to ontological differences, be it biological or social. Racial schemes are divided mainly into folk races (social construction of race) and biological (genetic based). Folk races mean the ordinary conception of race or the self-reported race based on social categories (Kaplan 2010: 281). In contrast, a biological conception of race is the

division of the human population based on genetic differences and self-acclaimed races (Spencer 2014). In the later part of this paper, I show that theorists such as Risch *et al.* (2002), Burchard *et al.* (2003), and Spencer (2018) variously use the genetic-based racial scheme to argue for its relevance in medicine. However, I argue that they are mistaken. The genetic-based racial scheme cannot be used in medicine for the following important reasons: races are socially constructed, in my view and a view shared by Kaplan (2020). In addition, the socioeconomic differences amongst races are mainly responsible for the causation of diseases. Given these reasons, we need a racial scheme that considers the different races' social differences, such as the folk racial scheme in medicine, to understand how diseases are socially caused. I now briefly clarify the biomedical race debate.

Biomedical Race Debate

The biomedical race debate is the application of the metaphysical race debate to medicine, which includes clinical practices and medical research (Spencer 2018: 1014). There are three camps in the biomedical race debate: eliminativism, which argues that race should be eliminated in biomedicine (see Yudell *et al.*, 2016); conservationism, those who provide arguments that human races should be used in biomedicine as a first step toward understanding the causation of disease, the risk to individual intake of drugs and the outcome (see Burchard *et al.*, 2003; Spencer 2018); and social conservationism, those who argue that racial classification can and should be used to approximate social and environmental differences between the population of people (see Kaplan 2010). I argue in the next section that only the social conservationism camp, which gives room for the folk racial scheme, is useful in medicine. In what follows, I now defend the epistemic significance of race in biomedical research.

Defending the Epistemic Usefulness of Race Biomedicine and Medical Genetics

I argue that there is a racial scheme that is epistemically useful in medicine, especially medical genetics. It is the case that races exist, and race is widely used in medicine as a medical category (Root 2003: 1173). Various research has shown that

people of different races respond to treatment differently. For example, the US health statistics are primarily stratified by using race data on the mortality and morbidity of patients to deal with risk factors. Human races are used as good indicators to predict certain diseases and how patients might respond to these diseases. According to the Alzheimer Association Report of 2019, in the US, African Americans are twice as likely to get Alzheimer's Disease (AD) compared to their white peers (Alzheimer Association 2019: 333). It becomes evident that racial classification plays a part in the contraction and mortality of AD diseases. Another example is heart disease in the US.

According to the American Heart Association (2001), black people in the US are likely to have more heart failures than white people. This evidence suggests that there could be something in the black race that causes heart failure that is possibly not in the white race. The question now becomes, is the above problem genetic, or is heart disease caused by socioeconomic and/or environmental factors? I argue that these problems are caused by socioeconomic and/or environmental factors associated with races in the US; as a result, a genetic racial category is not sufficient and robust to answer the question. In the next subsection, I bring the genetic-based racial scheme as an explanation of diseases. I apply this explanation to diseases such as sickle-cell anaemia to see if it is robust enough to explain the causation of these diseases. I argue that it is not robust enough; thus, I eliminate this explanation. Second, I bring the second racial scheme, folk race, to see if it is robust enough to explain the causes of disease. I explain and apply it to sickle-cell anaemia and even low-birthweight to know if it works as an explanans for these diseases. I argue that it is more robust than the genetic-based racial scheme.

The Epistemic Insufficiency of the Genetic-Based Racial Scheme in Medicine, Especially Medical Genetics

There is a widespread conception that the racial scheme that could be epistemically useful in medicine, especially medical genetics, is the genetic/human continental population racial scheme. Risch *et al.* (2002), Burchard *et al.* (2003), and Spencer (2018) variously argue that the difference in health outcomes, especially individual differences in response to drugs, is somewhat caused by variation in medically relevant genetic, environmental, and social variation factors.

These variations, which are caused by relevant genetic factors, are correlated with self-identified races

The above argument rest on the assumption that there is “a racial classification with medically relevant genetic differentiation” (Spencer 2018: 1014). This racial classification with genetic relevance is supported by Spencer (2018) using the following premises. First, Spencer argues that the set of the 1997 The Office of Management and Budget (OMB)³ races is a racial classification (Spencer 2018: 1015). Second, the above racial classification is identical to human continental populations (Spencer 2018: 1031). Third, “there are medically relevant genetic differences among human continental populations” (Spencer 2018: 1015). Fourth, these “medically relevant genetic differences among the human continental population” (Spencer 2018: 1031) result in racial classification that is useful in medicine. Therefore, “there is a racial classification of people that is useful in medicine” (Spencer 2018: 1015).

The justification for Burchard *et al.* (2003) and Spencer’s (2018) conclusion rest in the understanding that there are five major racial classifications in the (2000) US census (Spencer 2018: 1016). This racial classification from the US census comes from the OMB. Given that these populations exist and are human continental populations, there is a justification that these populations are useful in medicine for the following reason. These continental populations stratify “samples of people to better represent human genetic diversity” (Spencer 2018: 1031) in medical genetics. If these population groups have unique medical samples that are useful in medicine to explain, let us say, sickle-cell anaemia, which is present in black people and “not” white people (Root 2003), it follows that sickle-cell anaemia is genetic in the black population. Thus, resulting in a racial classification based on some genetic justification. This racial classification stems from the claim that there is no mismatch in the OMB racial classification. The set of the human continental population is identical to the OMB races, just like Blacks are identical to Africans (Spencer 2018: 1031). In what follows, I argue that this conception of race is false; as a result, it cannot provide robust epistemic insights into medical genetics.

³ The Office of Management and Budget (O.M.B), is considered as the expert in defining race according to the US law (Spencer 2014: 1028). The OMB defines in document 97-28653 of the US federal register revised in 1997 does not define race as a ‘kind’ or ‘category’; instead, it defines race as a set category that has five members (Pacific Islander, white, black, Asian, and American Indian). This definition of race comes from the data collection of the five-member population groups. The US meaning of race is whatever the OMB decides.

The Epistemic Failures of the Genetic-Based Racial Scheme

I argue in this section that the genetic-based explanation suffers from problems such as the reification and granularity problems which I explain shortly. I use diseases such as sickle-cell anaemia to make a case for why the genetic-based explanation is epistemically insufficient due to the aforementioned problems. I begin by explaining what sickle-cell anaemia means in the next paragraph.

Sickle-cell anaemia, a recessive genetic disease⁴, is more prevalent in the black population than in the white or Asian population (see Root 2003: 1176). For many years, sickle-cell anaemia was conceived to be a black disease than it was conceived to be a disease that anyone from tropical regions with malaria could contract. Even if sickle-cell is passed down from one generation to the next, the inheritance does not suggest it results from some biological features that define race. In addition, it does not suggest anything about genetic diversity in the human population, as Spencer (2018) argues. There is no need to theorise the cause of sickle-cell to be based on genetic diversity since anyone can contract the disease. I now show why the genetic-based racial scheme should be eliminated in medicine as a plausible explanation for diseases.

According to Kalewold (2020), the genetic-based explanation of Epidemiological Race Disparities (ERDs)⁵ has two main explanatory challenges in researching the aetiology and solutions to ERDs (Kalewold 2020: 2). These problems are the granularity and reification problems (Kalewold 2020: 2). Furthermore, amongst other normative and epistemic problems that the genetic-based explanation faces, some social factors that best explain the aetiology of diseases could be overlooked. In what follows, I show how Kalewold problematises the use of the genetic-based explanation of race in biomedicine.

First, Kalewold contends that a genetic-based approach to ERDs shows only the statistical “association between genetic factors and disease” (Kalewold 2020: 2). The genetic-based racial scheme fails to explain causally productive difference-makers⁶ and how they form a disease. As a result, we cannot correlate disease with

⁴ I provide a clear definition of sickle-cell anaemia disease in the section where I apply the genetic-based explanation of race to the disease to see the robustness of this explanation.

⁵ According to Kalewold (2020:2), ERDs is the “statistically significant differences in the incidence of disease between racialised groups”.

⁶ A difference-maker is “a philosophical notion which captures both our ordinary intuitive sense of what matters in causation and accords with scientific practice (Kalewold 2020:12).

a population using a genetic-based factor. Correlating a disease with a population cannot provide sufficient epistemic insights to precisely know the cause of the disease that has been associated with a given population. Other factors of a disease in a population could be socioeconomic, comorbidities, and genetic variants (Kalewold 2020: 6). Thus, it is unclear if one of the explanations or all three explanations is robust enough to adequately explain the cause of a disease.

Risch *et al.* (2002) and Burchard *et al.* (2003) may critique Kalewold (2020) here by arguing that the genetic-based explanation does not insinuate that diseases are caused by genes found in various races. Instead, the point of the genetic-based racial scheme as an explanation of disease is to identify the causal relationship between diseases and race. For example, Burchard *et al.* (2003: 1171) argue that socio-cultural and geographic factors also contribute to the cause of some diseases and the making of a gene.

I respond to the above critique by arguing that race should be used as a proxy for diseases, but not the genetic-based racial scheme because of the subsequent two problems I discuss in the paragraphs below. I contend that it is paramount that we understand the causal link between diseases and race. This causal link should not be based on some imagined genetic diversity in the human population but should be grounded on clear facts. I now consider other problems the genetic-based faces.

The granularity problem is the second problem Kalewold identifies with the genetic-based explanation of ERDs (Kalewold 2020: 7). The granularity problem is that there is no relevant grain for population analysis in the genetic-based explanans of races (Kalewold 2020: 7). What do we mean when using a blanket term such as “blacks” to identify a particular population? For example, when we say black people, do we mean black people in general or African Americans, black Caribbean, black South Africans, or black West African since they all share common ancestry (Kalewold 2020: 7). This shows that the lumping of race according to common ancestry is problematic because race, especially the so-called black race, is heterogeneous in different medically relevant ways. In applying the genetic-based racial scheme to sickle-cell disease, I show why this problem persists.

Third, the genetic-based explanation of ERDs suffers from a reification problem. Kalewold (2020: 8) argues that the reification problem comes from the fact that the genetic explanation of ERDs is conceived to be the “best” explanation

for ERDs, while other robust explanations for ERDs are side-lined (Kalewold 2020: 8). The genetic-based explanation does not take racist exposome as a contribution to disease. Instead, the genetic-based racial scheme “reverses the temporal and causal relationship between race and disease risk” (Kalewold 2020: 8). It is not the case that race constitutes the “difference-making factor in many of the epidemiological cases discussed above” (Kalewold 2020: 8). Rather, the “social form that race takes with the well-known history of racism, exploitation, and discrimination results in significant biomedical, but not genetic, differences between racialised groups” (Kalewold 2020: 8). I further speak about this problem in this next subsection. In what follows, I apply the genetic-based explanation to diseases such as sickle-cell anaemia to see if it works.

Applying the Genetic Racial Scheme to Sickle-Cell Anaemia to Show how these Problems Persist

In this section, I test the genetic-based explanation of race to diseases such as sickle-cell anaemia to see if it passes the granularity and reification problem. Sickle-cell disease is “an inherited haemoglobin disorder and the result of a single recessive gene; individuals who are heterozygous for the gene have the trait, while only individuals who are homozygous have the diseases” (Root 2003: 1176). From this definition of sickle-cell anaemia, it is almost *prima facie* that the disease is causally explained by the genes that we inhabit. Therefore, it falls under the genetic-based racial scheme’s province to explain this disease’s causation. However, a more critical analysis proves this conception to be false.

The implication of explaining these diseases using a genetic-based explanation is that it lumps these diseases on a population who “predominantly” have this disease. However, even though this disease is biological and becomes heritable, it is so because it has been embodied due to socioeconomic and environmental factors like malaria. Suppose it is the case that malaria causes sickle-cell anaemia, and malaria is found in a particular environment; in that case, it follows that whoever lives in an environment with malaria will develop sickle-cell anaemia. Research has shown that white people in high-risk malaria regions like Southern Italy carry the sickle-cell gene more than black people in low-risk malaria regions like South Africa (See Root 2003: 1177). This follows that sickle-cell disease

is not genetic according to the conception of race, as explained by proponents of the genetic-based categorisation of race. If this is correct, then the genetic-based racial scheme is insufficient to explain the cause of sickle-cell anaemia. This means that the genetic-based explanation does not escape the reification problem.

Furthermore, another problem of the genetic-based racial scheme is that it over-predicts sickle-cell traits in black more than in the white population. Using a genetically based explanation, one can argue that black people suffer from sickle-cell anaemia because it is a black disease. This is because this disease has been diagnosed to predominantly “come” from black people; therefore, sickle-cell anaemia affects only blacks. However, the second premise is false. It is false because not all black people have sickle-cell anaemia. There is no malaria disease in some parts of South Africa; as a result, black South Africans in these areas do not suffer from sickle-cell anaemia. Therefore, sickle-cell anaemia is context-laden and can be contracted by everyone in a high-risk malaria environment. This implies that the above explanation does not pass the granularity problem. As a result, the genetic-based categorisation of race as an explanans of disease should be eliminated. In the section that follows, I consider the folk racial scheme to see how it works and if it passes the granularity and reification problem.

The Folk Racial Scheme as Epistemically Useful in Medicine and Medical Genetics

The folk racial scheme, as previously mentioned, is a social categorisation of human races based on some social features. For example, in the US, races such as black and white are not technically classified only with phenotypical differences but socioeconomic and environmental differences (Root 2003; Kaplan 2014). In relation to health outcomes, black people in America have worse health outcomes than their white peers. As a result, black Americans’ life expectancy is shorter than white Americans’ (see Kaplan 2014: 286). Black Americans are more likely to die often than white Americans due to poor health conditions caused by socioeconomic-related issues such as poor health care, stress, and discrimination (Kaplan 2014: 286).

The cause of the high rate of disease contraction and death rate of blacks in America than white goes concomitantly with some socioeconomic and

environmental factors and poor education faced by African Americans rather than genetic factors (see Kaplan 2014: 286). As a result, if black people have the same mortality profile as white people in America or even in countries such as South Africa, where there is a high racial divide, the death risk of black people to some diseases would be circumvented.

For example, Kaplan (2014: 287) and Kalewold (2020) variously argue that there is a profound difference between first-generation African Americans or native-born African Americans in the US and black people who recently migrated to the US. Recent immigrants do not suffer from diseases such as low birth rate, hypertension, and heart diseases like native-born black Americans (Kaplan 2014: 287). Native-born black Americans are conceived to have worse health than other recent black immigrants in the US. For one to argue that the cause of diseases such as low-birth-weight or hypertension is caused by “genes” that are predominant in a “population” is to run into the granularity and reification problems discussed above. Why so?

Suppose it is the case that recent immigrants of the black racial group in the US do not suffer from the same disease in similar ways as native-born black Americans; in that case, it follows that these diseases such as hypertension, sickle-cell anaemia, or heart disease are not racial. These diseases can only be racial if everyone within that racial population suffers from such diseases. This means another explanation is robust to explain the aetiology of diseases such as sickle-cell anaemia and low-birth-weight. The other explanation that is more robust is the race-based explanation.

The race-based explanation can be best understood using the folk racial scheme. The folk racial scheme explains the socioeconomic factors that are attributed to different socially constructed races. I now apply the folk racial scheme to sickle-cell anaemia and low birth weight⁷ to show how it properly explains the aetiology of this disease.

⁷ I am aware that I did not apply the genetic-based categorisation of race as explanans of disease to disease such as low-birth-weight. This is because, this paper focuses on one example, which is sickle-cell anaemia. I apply the folk race scheme to low-birth-weight as additional to make my argument robust.

Applying the Folk Racial Scheme to Sickle-Cell Anaemia

I bring back diseases such as sickle-cell anaemia to show that the folk racial scheme gives a more robust explanation than a genetic-based explanation. As previously mentioned, sickle-cell anaemia is a recessive genetic disease found in some black people (Root 2003). However, as I have shown earlier, sickle-cell anaemia is caused by the environment that a person inhabits rather than some genes in a continental population. If black people inhabit places with malaria, it becomes obvious that black people will have sickle-cell disease. However, it is not the case that only black people can contract the disease. If white people inhabit the same environment, they will contract the same disease. This shows that the contraction of sickle-cell depends on a population's social status. In the US, most white people are mostly affluent and can afford to live in low-risk malaria areas, while most black people are economically disadvantaged. The genetic-based explanation cannot explain the affluence of white people because socioeconomic factors are not biological categories like genes.

On the contrary, the folk-racial scheme is a social construction of race that recognises these socioeconomic differences between races. Since socioeconomic and environmental factors cause sickle-cell disease, it follows that folk race can explain these factors as they fall under social categories. Furthermore, more evidence supports socioeconomic and environmental factors to be the cause of sickle-cell than genetic diversity. Let me make an additional case to tighten my argument on why the folk racial scheme is more useful by applying it to low-birth-rate.

Applying Folk Racial Scheme to Low-Birth-Weight

According to Kalewold (2020:15), socioeconomic and environmental factors are better explanations for diseases such as low-birth-weight. Low-birth-weight is caused by excessive stress. In the US, for instance, African Americans experience high-stress levels due to their experiences of discrimination and their internalisation of the various discrimination they face (Kalewold 2020: 15). Evidence has shown a link between racial discrimination and low-birth-weight; racial discrimination gives rise to chronic stress (Kaplan 2014). African American

women suffer from chronic stress due to allostatic load, a “cumulative biological burden exacted on the body through attempts to adapt to life’s demands” (Kaplan 2014: 291). This condition then gives rise to low-birth-weight.

The folk racial scheme can show that low-birth-weight is caused by socioeconomic and environmental factors rather than genetic diversity. It provides evidence to show that low-birth-weight, common in African American populations, is not common in recent immigrants and other black populations. Social factors such as racism are the best explanations for low-birth-weight. Since race is a social category (Kaplan 2014: 293) and social issues cause low-birth-weight, we need a racial scheme that accounts for social differences to deal with social problems.

Returning to the central question of this paper, can any racial scheme be epistemically useful in understanding the cause of diseases? I answer in the affirmative. However, as shown in this paper, a genetic-based racial scheme cannot justify that genetic differences causes diseases such as low-birth-weight or sickle-cell. Rather, the only racial scheme that can explain the cause of low-birth-weight and sickle-cell anaemia is the folk racial scheme based on the reasons that I have provided throughout this paper. In the next section, I consider a possible objection from a critic, and I respond to the objection.

Objections

A critic might argue that my argument against the genetic-based racial scheme as plausible explanans to the causation of disease is flawed. The critic’s contention may be that research has shown that medication used in treating heart disease, such as beta-blockers, is more effective than the ACE inhibitor in the black population. Black people with heart disease do not respond to ACE inhibitors as they do to beta-blockers (Root 2003:1180). If this is the case, it follows that there is something genetic that causes black people not to respond to Ace-inhibitors the same way they respond to beta-blocker. Using the following conclusion, it is then possible for one to postulate that, rather than a social racial scheme, genetic diversity in the human population best explains the ineffectiveness of the Ace-inhibitor medication than the beta-blocker in treating heart diseases in the black population. If so, one can infer that the human population’s genetic diversity causes these diseases and others.

Response to the Objection

The above objection is a very plausible one. However, this objection still suffers from the granularity and reification problem I dealt with in the previous section. How so? The objection suffers from the granularity problem because it uses a particular black population (African Americans) to generalise to the whole black population. No evidence shows that all black people react to these medications similarly.

In addition, the second problem which the above objection suffers is the reification problem. The objection assumes that only genetic factors can be responsible for heart disease. However, as I have argued in this paper, other factors best explain the cause of heart disease. Such factors are socioeconomic and environmental factors. For example, what causes heart disease in African Americans could be different in the black population of West Africa. Thus, knowing the best medication that works better in treating heart diseases is a prerequisite to understanding the social causes of these diseases. The folk race offers a more robust explanation of the causes of heart diseases; thus, the folk race is the best racial scheme to account for the cause of diseases.

Conclusion

In this paper, I have argued that the folk racial scheme is epistemically useful in medicine, especially in medical genetics. I arrive at this conclusion by eliminating the genetic-based racial scheme as a plausible explanation for the aetiology of diseases due to its lack of robustness. I began this paper by clarifying the recurrent terms in the first section. In the second section, I first affirmed that there is a racial scheme that is epistemically useful in medicine, especially medical genetics. Second, by eliminating the genetic-based racial scheme as insufficient, I argued that the folk racial scheme is more robust in medicine, especially in medical genetics. Third, I provided a possible objection to my argument and responded to it afterwards. Finally, I contend that understanding the role race, especially the social construction of race and some social and economic factors, plays in the making of diseases is a step toward understanding how to deal with these diseases. Given this, this paper offers insights to those interested in biomedical research relating to the

causation of diseases and how to offer solutions to diseases associated with a particular continental population.

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