Abstract

Race, a concept with scientific origins, has come to permeate North American society on many levels, including one of the most respected North American institutions: medicine. Strangely, race continues to enjoy acceptance as a valid biological variable in medical literature despite the fact that in the past fifty years scientists have discredited the notion of race. References to race, and racial differences, abound in the medical literature, especially in studies from the past fifteen years. Most of these references concern observed differences in health status among racial/ethnic groups in North America. This article considers two of these noted health status differences: the higher prevalence of hypertension in African-Americans as compared to European-Americans (whites) and the higher prevalence of diabetes mellitus in Native-Americans as compared to European-Americans. A review of the proposed mechanisms for these observed differences is included in this article, followed by a conclusion that discusses the merits of using race as a biologic variable in medical literature.

Introduction

Few ideas in the history of humankind have generated more controversy than the concept of race, a notion that grew out of the scientific discipline of taxonomy several centuries ago. From early on, race has been directly linked to science, and therefore has enjoyed a great deal of legitimacy in the modern, “scientific” world in which we currently live. Over the centuries this concept of race has moved beyond the realm of science and has permeated nearly every aspect of human society in the twenty-first century. This is particularly true in North America, where race was the foundation for a system of human slavery. Although slavery ended in the United States nearly one hundred fifty years ago, the contentious issue of race continues to affect the daily lives of Americans. Ironically, the people who first developed the concept of race, scientists, are among the few who now reject it as a valid means of identifying human

subgroups.

This change of scientific opinion concerning race is of recent origin:

> Until World War II, race was usually considered to be a scientific concept, a biological category used to 'measure' geographical, religious or skin-colour-based groupings, primarily associated with physical anthropology. Subsequently, scientific opinion has rejected race as a useful classificatory tool.[1]

Unfortunately, neither the general public, nor many medical researchers seem to be aware that the concept of race has been completely discredited by scientists. This perpetuation of the validity of race has significant consequences for the field of medicine. We will examine some of these consequences in this essay.

References to race, and specifically racial differences, are ubiquitous in the medical literature. Some authors are beginning to point out this problem, yet medical research continues to employ race as a biological variable. Comments in an article by Witt et al. illustrate the perceived validity of race that many medical researchers maintain. These authors explain that race, culture and ethnicity do not have the same meaning:

> It is important to realize that culture, race and ethnicity are not interchangeable. Many different cultures exist among persons of the same race. Persons who share ethnicity may not be of the same race. People of different races may share cultural ideology.[2]

The explicit idea put forth by Witt and colleagues is that culture, ethnicity and race are separate concepts, with separate meanings; however, the authors' comments implicitly argue that all three concepts enjoy the same degree of scientific legitimacy. The clear difference between race and the concepts of culture and ethnicity is that the former is derived from a rejected biological paradigm, while the latter concepts belong to accepted sociological constructs.

So the question that medical authorities must ask themselves is why does “race” continue to appear in the medical literature, and what consequences result

from the erroneous use of this concept? The answer to the first question is that data from various studies have revealed alleged differences in health status among different subpopulations in North America. In attempting to explain these observed differences, many researchers look to the rapidly expanding field of genetics for answers. This focus on genetics, or biologic variation, naturally leads to racial rhetoric in the medical literature. Perhaps a better term to be applied to these subpopulations is ethnic groups.

The language of race

A brief discussion of the language of race used in the medical literature is necessary before we proceed to examine the issue of differential health status among North American sub-populations. As defined above, race is a fallacious idea that categorizes human beings based solely on supposed biological (genetic) differences. Ethnicity, on the other hand, is a means of classifying human subpopulations based on the aggregation of a broad spectrum of identifying information:

> [It] is defined as the real, or probable, or in some cases mythical, common origins of a people with visions of a shared destiny, which are manifested in terms of the ideal or actual language, religion, work, diet, or family patterns of that people.1

Ethnicity does not imply any biological or genetic uniqueness of a specific group. Nonetheless, as Witt et al. pointed out, many people confuse race and ethnicity. Some authors use these two terms interchangeably, while others refer to ethnicity as a euphemism for race. As an example of the euphemistic terminology of ethnicity, let us examine the terms used to refer to the descendants of Africans

who were brought to the Americas as slaves. Following the Civil War, the terms Negro and coloured were in general use as non-pejorative labels for these people. Negro eventually overtook coloured as the most widely used term for this ethnic subgroup of Americans (hence the eventual capitalization of the label), and the dominance of the label Negro stretched until mid-twentieth century.[3] Black, which originally had a derogatory connotation, gradually replaced Negro during the Civil Rights era of the 1960's and early 1970's as the “Black is Beautiful” movement gained strength.3 In the past ten to fifteen years, African-American has gained popularity as a replacement for these racial terms because it supposedly connotes one's ethnicity, not one's race. Yet this is misleading. One cannot equate the meaning of “African-American” to the ethnic term “Italian-American;” with regards to the latter, the “-American” can easily be dropped without losing the original meaning of the term, as in an “Italian” neighborhood.[4] Referring to a neighborhood of African-Americans as an “African” neighborhood would not make sense (this also points to the difficulty in linguistically distinguishing people of recent African immigration from those who are ancestors of forced immigration centuries ago). Thus, “African-American” is in fact a racial term masquerading as an ethnic one.4

The terms applied to the ethnic majority in North America are equally confusing. “White” and “Caucasian” are the two common words used to refer to this ethnic group. Like “black,” “white” is a racial term, yet it is considered by many people to be an equivalent to “African-American.” “Caucasian” is mistakenly considered by many to be a racial term, when in fact it originally

referred to a particular ethnicity.4

To further confuse the meaning of “ethnicity,” this term is commonly used to refer only to the minority subpopulations of North America. For instance, politicians often refer to the “ethnic” vote. But in fact, “[e]thnicity is something that everyone has, not just the minority ethnic groups....”1

Inconsistent use of racial and ethnic terms is not unique to the medical literature. In the United States one is continually asked to choose a self-identifying racial or ethnic label when completing forms such as census surveys and applications to educational institutions. Commonly, the following options are listed: white, African-American, Hispanic, Native-American, Asian, and other. As one can see, the meaning of each option is not equivalent to the meanings of the others. Hispanic people may find it difficult to choose only one option since some Hispanics may identify themselves as white, while other Hispanics would identify themselves as black (African-American). Other ethnic groups, such as Middle Eastern Arabs or East Indians, who do not fit neatly into one of the above specified ethnic labels, may experience conflict when having to choose among these labels.

So as not to perpetuate the perceived scientific validity of race, in this paper, subpopulations will be referred to in terms of ethnicity (though admittedly, most of the studies cited in this article employ racial terms, or otherwise euphemistic proxies). Thus, African-American will be preferentially used over black (despite the above cited euphemistic nature of this term). Likewise, “white” and “Caucasian” will not be used when “European-American” can suitably be

substituted. Aboriginal populations will be referred to as Native-Americans or Native-Canadians, and populations of immigrants from Asia will be referred to as Asian-Americans. Although these terms have their own limitations (e.g., those people of North African descendant would not be included in “African-American” populations), they are the most adequate terms available at this time to refer to common North American ethnic groups.

Why race/ethnicity is important in North American medicine

Although the nations of Europe (and to a lesser extent other Old World nations) have become more multi-ethnic over the past half century, the ethnic diversity of these Old World countries pales in comparison to the rich ethnic heterogeneity of the New World nations, particularly Canada and the United States. The US Census Bureau predicts that the percentage of the US population belonging to minority ethnic groups will reach 40% by the year 2035.2 According to the 2001 Canadian census, both Vancouver, British Columbia and Toronto, Ontario (two of the three largest Canadian cities) have “visible minority populations” that comprise 37% of their total populations.[5] The impact of the growing minority ethnic populations on the health care systems of North America is compounded by the fact that many of these minority ethnic groups suffer disproportionately more morbidity and mortality than the majority North American ethnic group[6] (hereafter referred to as European-Americans, or whites). Despite the proportionally higher burden of disease experienced by these minority ethnic

groups, “[f]or reasons of scientific and practical convenience, minority groups were commonly excluded from clinical trials until the mid-1990s.”[7] In recent years, however, a large body of literature has developed that addresses health status disparity in North America among the various ethnic groups. Unfortunately, race, rather than ethnicity, is often cited as a variable in these studies. The following sections of this essay will examine how race is presented as a determinant of health status in the medical literature.

Race as a determinant of health status

Since the concept of race was first introduced into the medical literature in 1684 by the French physician, François Bernier,[8] hundreds, if not thousands, of published medical studies have cited health-related differences among racial/ethnic groups. During the past decade, there has been an explosion of medical literature pertaining to health disparity among North American subpopulations. The subject has gained so much notoriety in the United States that in 2000 the American government created a division of its National Institutes of Health (the National Center on Minority Health and Health Disparities) to specifically address this issue. In North America these health status differences are usually reported as a comparison of a minority ethnic group to the reference (majority) ethnic group, who are invariably classified as whites (and on occasion, non-Hispanic whites). Unless otherwise specified, comparative incidence and prevalence rates cited in this paper for minority ethnic groups are always with

reference to European-Americans (i.e., white-Americans).

Increased prevalences in the African-American community of cancer (notably prostate cancer in African-American men),[9] hypertension,[10] and diabetes mellitus[11] have all been reported, while osteoporosis is reportedly less prevalent among African-Americans.[12] Native-Americans, like African-Americans, have been shown to have higher rates of diabetes mellitus than European-Americans.[13] The impact of alcohol abuse (mortality due to alcoholic cirrhosis) appears to disproportionately affect Hispanic-Americans in comparison to European-Americans.[14] Asian-Americans are more likely to be infected with tuberculosis than their European-American counterparts.[15] Similarly, Native-Canadians have a much higher incidence of tuberculosis than Canadian-born, non-aboriginal Canadians.[16] Proposals have been made to explain each of these observed differences, but it would be impossible to summarize all of these theories here. Therefore, we will have a limited discussion on the proposed mechanisms of the most important of these findings: increased prevalence (as compared to European-Americans) of hypertension in African-Americans and of diabetes mellitus in Native-Americans.

African-Americans and hypertension

A large body of evidence from the medical literature has observed that the percentage of African-Americans with hypertension is higher than the percentage of European-Americans. The importance of these data is evident: hypertension is

the most common reversible risk factor for cardiovascular disease, a major killer among all populations and age groups.[17] Hypertension is also a significant risk factor for end-stage renal disease, which is four times more common among African-Americans compared to European-Americans.[18] Three decades ago, studies indicated that hypertension was twice as common among African-Americans as European-Americans, although over the past 30 years, this ratio has decreased by approximately 50%.10 Investigators have sought evidence to support the various suggested mechanisms that lead to this disparity between African-Americans and European-Americans, while others have attempted to explain why this difference may be narrowing.

Because the concept of race (erroneously) implies that sub-populations of the human species can be categorized based on biological (i.e., genetic) differences, when race is used as a variable in scientific studies, readers commonly formulate an initial hypothesis that genetic differences may account for any observed health status discrepancies between the identified racial populations. Thus, when discussing the increased prevalence of hypertension in African-Americans, the first question to consider is whether or not genetic differences between African-Americans and European-Americans can explain the former group's propensity to hypertension. The body of research that attempts to answer this question is relatively small. The *American Journal of Hypertension* recently published (in the same issue) three different genome-linkage studies, each involving two sample groups, one African-American and one European-American, the results of which were not very conclusive. Kardia et al. found no evidence of “linkage to

hypertension susceptibility genes,"[19] while Thiel et al. found a possible linkage on chromosome 1 in European-Americans but not African-Americans,[20] and Rau et al. found a possible linkage on chromosome 2 in African-American sib-pairs but not European-Americans.[21] Likewise, a genomewide linkage scan based on the results of the HERITAGE Family Study provided only limited evidence of linkage in African-American families.[22] Although not enough research has been conducted to confidently state that the increased prevalence of hypertension among African-Americans is not due to genetic factors, many researchers agree that alternative explanations, both biological and social in nature, probably are more important to examine.

Researchers have advanced several biological hypotheses concerning the cause of African-Americans' increased prevalence of hypertension including: increased sensitivity to dietary sodium, enhanced vascular reactivity, impaired glucose tolerance, increased insulin resistance, and increased frequency of obesity.[23] According to Williams, "[o]besity is the strongest factor associated with blood pressures."[24] Data from the National Center for Health Statistics have revealed that fifty-three percent of obese people are hypertensive, while only twenty-two percent of non-obese people have hypertension.[25] These same statistics cite a forty-four percent prevalence rate of obesity among hypertensive people, as compared to a fifteen percent prevalence among normotensive people. These data suggest that obesity and hypertension are closely related. Interestingly, hypertensive African-Americans have a fifty-four percent prevalence rate of obesity, as compared to the forty-four percent prevalence rate among all

hypertensive people.25 Thus, the correlation between obesity and hypertension appears to be particularly strong among African-Americans. Although the prevalence of obesity among African-American males is roughly equal to that of European-American males, African-American females are twice as likely to be obese as European-American females.[26] It has been shown in one study that controlling for obesity decreases the difference in prevalence rates of hypertension between African-Americans and European-Americans.[27] In another study adjusting the data for obesity eliminated the difference in rates of hypertension between African-American and European-American women.[28] Although an increased prevalence of obesity in African-Americans may explain a portion of this population's high prevalence of hypertension, the data from the above cited studies do not suggest that obesity is the sole factor involved.

Recently, a new theory (that low birth weight leads to hypertension in adulthood) has received a great deal of attention in the medical literature. Low birth weight may be an all-encompassing biological explanation for the increased prevalence of hypertension among African-Americans since African-American mothers give birth to low birth weight children nearly twice as often as European-American mothers.[29] The suggestion that low birth weight tends to precipitate hypertension in adulthood has been eponymously termed the “Barker hypothesis” after one of the researchers who first cited a correlation between these two phenomena.[30] Several subsequent articles have corroborated the “Barker hypothesis,”[31] and attempts to explain this correlation have been made. Some researchers have proposed that low birth weight leads to impaired

maturation of the kidneys, which then induces progressive renal dysfunction and eventual hypertension.[29, 31] This decreased nephrogenesis would also explain why African-Americans are notably susceptible to the salt-sensitive, low-renin form of hypertension.29 A second possible link between low birth weight and hypertension is that low birth weight "programs" the body to overcompensate with growth in childhood, leading to obesity.[32] As noted above, obesity is associated with glucose intolerance and insulin resistance, both of which have been proposed as causes for the increased prevalence of hypertension in African-Americans, and is itself independently correlated with hypertension. Rather than structuring these pathological processes into a chain of events (i.e., low birth weight results in obesity which results in hypertension), it is reasonable to believe that obesity, glucose intolerance, insulin resistance and hypertension all result from the same pathological process initiated by low birth weight.* Current evidence for the causal relationship between low birth weight and obesity in African-Americans is scant. Only one trial[33] with a small sample number has shown a correlation between low birth weight and obesity, and no trials with large sample sizes have been conducted. If the scientific community were to agree that the increased incidence of low birth weight in African-Americans explains the increased prevalence of hypertension in adult African-Americans, this fact would lead to a second line of scientific inquiry, namely, why do African-Americans have a higher incidence of low birth weight than European-Americans? This question

* An emerging concept in medicine, known commonly as Syndrome X, describes individuals who have central (android) obesity, hypertension, insulin resistance/glucose intolerance, and atherogenic dyslipidemia. Many researchers purport that a common pathological process leads to the four features of Syndrome X.

must also be answered in terms of genetic, environmental and social factors. Lopes believes that there is more evidence that environmental and behavioural factors contribute to the increased incidence of LBW in African-Americans as opposed to genetic factors.29

Many researchers have argued that environmental factors can explain the tendency of African-Americans to be hypertensive. The most common of these contentions is that differences in socio-economic status (SES) between African-Americans and European-Americans account for the increased prevalence of hypertension in the former group. Many studies have entertained this hypothesis, but the aggregate results are not conclusive. In a study conducted by Diez Roux et al., there was an observed inverse relationship for both European-Americans and African-Americans between the risk of developing hypertension and SES.[34] Likewise, the Hypertension Detection and Follow-up Program, a prospective study of 4800 hypertensive blacks conducted in the late 1970's revealed an inverse relationship between SES and hypertension for both European-Americans and African-Americans. In this study African-Americans were twice as likely to be hypertensive as their European-American counterparts. Interestingly, European-Americans in the lowest socio-economic bracket had less hypertension than African-Americans in the highest socio-economic bracket. A recent prospective study by Matthews et al., which assessed the impact that change in SES had on the incidence of hypertension in both European-American and African-American participants, concluded that “cumulative economic difficulties are associated with incident hypertension.”[35]

Despite the evidence supporting the role of SES in hypertension development, the difference in prevalence of hypertension between European-Americans and African-Americans may not be entirely explained by differential socio-economic indicators. In a study examining elderly patients, African-Americans had higher prevalence of hypertension than European-Americans even when the data were adjusted to account for differences in SES between the two groups.[36] Similarly, a Brazilian study found that "black" women had a higher prevalence of hypertension than "white" women independent of SES.[37] Given the results of these studies, it appears that socio-economic status has a definite effect on the prevalence of hypertension in a given community, but the differences in SES between European-Americans and African-Americans cannot entirely explain the increased prevalence of hypertension in African-Americans.

Increased levels of stress in African-Americans has also been proposed as a mechanism leading to their increased prevalence of hypertension. Dresser, who has argued that dark skin color is an indicator of low SES and exposure to discrimination, contends that the stress experienced by dark-skinned individuals as they struggle to achieve economic and social rewards in a color-conscious society leads to increased hypertension among these individuals.[38] Other authors argue that the stress due to urban and industrial life, which disproportionately affects African-Americans, leads to the increased prevalence of hypertension in the African-American community. Williams has provided a concise explanation of this argument:

> [U]rbanization and industrialization tend to be predictive of increases in blood pressure levels. This suggests that environmental stress may make an important contribution to

> hypertension. Because of past and present economic exploitation, blacks are likely to experience more stress than whites. If stress is causally linked to hypertension, its role may be crucial in accounting for racial differences in high blood pressure.24

Further evidence to support the impact of urban stress on hypertension comes from a study in which black Africans experienced increases in blood pressure following relocation from smaller communities to larger, urban centers.[39]

A final potential explanation for the observed difference in hypertension prevalence between African-Americans and European-Americans is faulty methodology. No serious critiques have been made of the methods employed by the studies that observe this differential prevalence; however, a recent study by Gorey et al. hints at the risks of various biases that may obscure the results of studies that employ race as a variable.[40] Gorey and colleagues note that the observed disparity between prevalence rates of hypertension in African-Americans and European-Americans has narrowed over the past three decades by 50%. These authors discovered a significant relationship between the narrowing of this gap and the decrease in response rate cited in relevant studies (n=25) over the past 30 years. They propose that this relationship is due to the "healthy participator effect." As response rates declined over the past three decades, respondents tended to be younger and to have a higher SES, and thus healthier than earlier respondents. In fact, Gorey et al. state that European-American and African-American respondents in recent studies tend to resemble each other more than they resemble their respective population counterparts. Although one can conclude from this evaluation of epidemiological methods that the current estimate of the difference in prevalence between European-Americans and African-Americans is artificially low, the study begs the question

as to whether or not other methodological errors might explain the perceived difference in hypertension prevalence.

Native-Americans and diabetes mellitus

A second example of the inequality of health status based on race that is classically cited in the medical literature is the high prevalence of type 2 diabetes mellitus in North American aboriginal populations. One group of authors has boldly claimed that "[g]lobally, no other ethnic/racial group suffers more with type 2 diabetes than Native-Americans."13 In Canada 6% of Native-Canadian adults suffer from diabetes mellitus as compared to 2% of all-Canadians, although prevalence varies greatly among various aboriginal subgroups (e.g., the prevalence of type 2 diabetes in the Oji-Cree of Ontario is approximately 40%, among the highest rates in world, while the prevalence among the Inuit is less than 1%)[41] Given the wide range of prevalence rates among Native American tribes, Young has grouped them into three categories: tribes with high prevalence (more than 30%) such as the Pima, Havasupai, and Oji-Cree; tribes with low prevalence (less than 10%) such as the Inuit and Aleut; and all other tribes whose prevalence rates are intermediate.[42] The population believed to have the highest prevalence of type 2 diabetes mellitus globally are the Pima Indians of the southwestern United States. It has been noted that 50% of adult Pima Indians suffer from diabetes.[43]

The classical hypothesis for the cause of type 2 diabetes mellitus, the so-

called “thrifty genotype,” was put forth in 1962 by Dr. J. V. Neel.[44] The essential argument submitted by proponents of the “thrifty genotype” hypothesis is that diabetes arises when genetic susceptibility is triggered by environmental factors such as increased caloric intake and decreased physical activity.[45] Claims have been made that anthropological data concerning Native-Americans supports this theory. Advocates of this hypothesis reason that the alternating feast and famine conditions experienced by early migrants from Asia to North America, such as the Pima Indians, have selected over the centuries for individuals who are relatively insulin resistant, which allows for better conversion of serum glucose to fat when glucose is available.[46] The extra body fat that these individuals would store during times of abundant food sources would sustain them through the leaner periods. As Native-Americans' food sources became more constant and richer in carbohydrates and fat, and as the Native-Americans' previously strenuous lifestyle was supplanted by the sedentary lifestyle typical of reservation life, the supposed “thrifty gene” proved to no longer be adaptive. Thus, though diabetes was a virtually unknown disease among Native-Americans at the turn of the twentieth century,[47] prevalence rates have sky-rocketed over the past fifty to one hundred years among many Native-American tribes.

This theoretical paradigm may also explain why the Native-Americans of later migration waves, such as the Inuit and Aleut, have lower rates of diabetes than their predecessors since less time has been allotted to these populations for selection of thrifty genes in their gene pool.13 The fact that the Inuit still have active lifestyles and continue to eat healthier diets such as arctic fish also

explains why their prevalence of diabetes is much lower than that of the Cree or the Pima Indians.41 Since Neel asserted his hypothesis of the “thrifty genotype” over four decades ago, a great deal of research has been published in search of a type 2 diabetes gene. Thus, genetic differences between Native-Americans and other North American populations has been the leading theory to explain the difference in their respective prevalences of diabetes.

There are several clues that point to the genetic etiology of type 2 diabetes mellitus in Native-Americans: diabetes tends to aggregate in Native-American families; individuals from non-Native ethnic groups living in the same environment as Native-Americans have a lower prevalence of diabetes; and individuals of mixed ethnicity have an intermediate prevalence rate.[48] Further evidence of the genetic etiology of diabetes comes from twin-twin studies, which show higher concordance between monozygotic twins as opposed to dizygotic twins.[49,50] Hegele has pointed out that both the Cree and the Inuit have more “deleterious” alleles than whites living in the same environmental conditions (although, as explained above, the prevalence of type 2 diabetes among the Inuit is much lower than among the Cree, and is in fact lower than among European-Americans).41 For instance, among the Oji-Cree a population-specific mutation of the gene *HNF1A,* which “clearly confers susceptibility to...type 2 diabetes...”[51] has been found. Despite this and other evidence for the role of genetics in diabetes, the forty year search for identifiable diabetogenic genes has not been exceptionally fruitful.

Recently, some researchers have begun to reëxamine the evidence for the

“thrifty genotype” hypothesis. As Benyshek et al. point out, many Pacific Island populations have prevalence rates of diabetes nearly as high as the Pimans, yet there is little evidence that these islander populations suffered the same feast and famine conditions as the North American aboriginals.13 These authors also cite differences in prevalence rates between the Pima Indians and the Dogrib Indians as evidence against the “thrifty genotype” hypothesis. The ancestors of the Dogrib Indians, like the Pima Indians, crossed the land bridges from Asia to North America with the first migratory waves over 11,000 years ago, and they subsequently were exposed to feast and famine conditions. Therefore, the Dogrib Indians should have the same “thrifty genotype” as the Pima Indians. The lifestyle of the Dogrib Indians has gone through a transformation similar to that experienced by the Pima Indians in that they currently enjoy high calorie diets and lead relatively sedentary lives. However, the Dogrib Indians have a much lower prevalence rate of diabetes than the Pima Indians. Thus, the “thrifty genotype” hypothesis does not explain the varied prevalence of diabetes among Native American tribes, nor does it appear to explain the high rates of diabetes in non-Native American populations.

There is a great deal of emerging evidence that supports the importance of environment rather than genetics with regard to the development of diabetes. For instance, Pima Indians living in Mexico, who do not currently share the same sedentary lifestyle as the Piman Indians of the Southwestern United States, have a much lower prevalence of diabetes than their American counterparts.[52] Among the Pima Indians, individuals who were breast-fed for at least two months have

lower prevalence of diabetes than those who were exclusively bottle-fed.[53] Further evidence of the importance of environmental variable comes from another twin study.[54] In this study twins with abnormal glucose tolerance had lower birth weights than twins with normal glucose tolerance. Furthermore, "...twins with the lowest birth weights among the two co-twins had the highest plasma glucose concentrations..." following an oral glucose challenge. This final point leads us to an alternative hypothesis for why some Native-Americans have such high rates of diabetes: the so-called "thrifty *pheno*type" hypothesis.

The idea behind the "thrifty phenotype" hypothesis is that low birth weight promotes development of type 2 diabetes mellitus in adulthood. Evidence from both animal and human studies support this hypothesis. A rat model has been developed in which pregnant rats are given insufficient nutrition, leading to low birth weight offspring. The nutritional deprivation in fetal rats leads to changes in the liver and pancreas resulting in decreased insulin secretory capacity and glucose intolerance.[55] A human study has also shown that the children of pregnant women with poor nutritional status are more at risk for increased levels of serum glucose, insulin, and triglycerides.[56] It has been shown that in the Pima Indian population there is an excess of diabetes in low birth weight individuals.[57] However, Bennett does not find evidence that Pima Indian mothers are undernourished.[58] He has proposed two explanations for the correlation of low birth weight and diabetes. First, he has suggested that "...the excess of diabetes might be due to selective survival in low-birth-weight offspring predisposed to develop diabetes."58 The second hypothesis is that these low birth weight

individuals are insulin-resistant and therefore do not grow as large as non-insulin-resistant fetuses in a normal intrauterine environment.58 Given that insulin-resistance is a significant risk factor for diabetes, it is not surprising that these low birth weight individuals have higher prevalence of the disease.58

Benyshek and colleagues argue that although Native-American mothers are currently well-nourished, perhaps over-nourished with foods rich in fat and carbohydrates, the explosion of diabetes among certain Native-American populations began with previous generations who were in fact subjected to impoverished conditions (e.g., long forced marches to reservations, poor reservation conditions and punitive boarding schools).13 Perpetuation of the high prevalence of diabetes in subsequent Pima Indian generations may be explained by the fact that the offspring of diabetics have a high risk of developing diabetes themselves, thus creating a vicious cycle.57 The offspring of diabetic mothers tend to have low birth weight and relatively large pancreata. When these offspring are subsequently well-nourished (i.e., following weaning) they develop significant insulin resistance.13 Given the increased caloric intake and decreased physical activity characteristic of recent generations of Pima Indians coupled with this vicious cycle of diabetic mothers giving birth to diabetic children, it is easy to understand why the prevalence of diabetes among the Pima Indians has increased so dramatically over the past fifty years.

Discussion

As illustrated by the above examples, many differences in health status among North American racial/ethnic groups have been identified in the medical literature, yet these seemingly unbiased observations are in fact quite controversial. Lillie-Blanton and colleagues have shed light on the dubious nature of these observations. The authors note that references to differential health status among different ethnic/racial groups are routinely made "...without adjustments for social class differences."[59] These unadjusted descriptive statistics are "...particularly useful in answering the question: 'What is occurring?' Descriptive statistics, however, can be misleading if the primary question of interest is: 'Why are the observed patterns occurring?'"59 How do descriptive statistics mislead? When observations of differences between two racial/ethnic groups are made without controlling for environmental factors such as social class, racial (i.e., genetic) differences are assumed to be the etiological agents that lead to the differential health statuses. This presupposes that race, and therefore racial differences, are bona fide scientific facts. As noted several times above, the idea that discreet races of humans exist, and that they are thus endowed with identifiable genetic differences, has been proved false. Hence, these studies that present only descriptive statistics are perpetuating the myth that racial differences exist.

How then, might we explain the studies that link genetic factors to the observed health status differences? The evidence that genetic factors contribute significantly to determining an ethnic/racial group's disease prevalence is minimal

and inconclusive. As Cooper explains, “no 'discrete' package” of genes exists for each “race,” only “relative gene frequencies of one or another trait.”[60] Furthermore, diseases such as hypertension and diabetes, as well as cancer, are most likely influenced by multiple genes, not single ones that are present or absent in a given racial/ethnic population. Indeed, four decades of searching for the hypothesized “diabetes gene” have proved fruitless. Cooper compellingly argues for a multivariate analysis to explain the observed health status differences among ethnic groups.

Fortunately, there is a movement in the medical literature away from genetic theories toward environmental hypotheses that will explain the differential health status of North American subpopulations. Environmental theories specific to diabetes and hypertension have been discussed above; however, there are a few general hypotheses proposed to explain the excess morbidity and mortality suffered by minority ethnic groups in North America. As Williams et al. points out, increased levels of stress experienced by minority ethnic groups likely contributes to several pathologies,[61] not just hypertension. These authors further note that “[t]he extent to which minority populations are disproportionately exposed to environmental toxic exposures has been a neglected issue in studies of racial differences in health status.”61 Additionally, the negative impact of our modern, industrialized society on human health disproportionately affects minority ethnic groups. The so-called “diseases of modernization” such as diabetes, hypertension, hyperlipidemia and obesity (members of the previously mentioned Syndrome-X) have high correlations with high calorie, high fat diets

and sedentary lifestyles. Unfortunately, in the modern, Western world, only the wealthy can afford “healthy” diets and active lifestyles. To lead an active life in an urban setting requires money (to pay for gym memberships or to get involved with organized activities) as well as adequate leisure time. The low pay and long working hours of the employed poor make it difficult for them to escape a sedentary lifestyle. Likewise, the convenience and low-cost of high calorie, high fat foods (such as the fare at fast food restaurants) and comparative high cost for “healthy” alternative foods has likely fueled the rates of obesity in ethnic minority groups. In general, the privileges and wealth enjoyed by many European-Americans has probably shielded them from many of the environmental dangers to which minority ethnic groups are routinely exposed.

Role of race in medicine

Even if the controversy over the mechanism of health status differences were resolved, the scientific soundness of publishing studies that include race and/or ethnicity as a scientific variable is itself contestable. As noted above, scientists now view the notion of race as a fallacious biological construct, yet race continues to be used as a biological variable in the medical literature. Should a campaign be initiated to reject all use of race variables in future medical studies? Some would argue that despite the fact that race has been discredited as a valid biological concept, race is a suitable proxy for genetic similarity, which is why its use in medicine to study the relation of disease to genetics is appropriate.[62] For

example, the ranges of normal values for the Body Mass Index (BMI) are specific to one's “race” (the normal range for people of Asian descent is lower than the range for those of European heritage).

Many authors, however, have argued strenuously that race has no place in medical literature. Osborne and Feit claim that “[w]hen race is a study variable, the likelihood increases that the scientific merits of the investigation will suffer.”[63] Although race may be a convenient variable to use, its non-scientific basis calls into question the scientific value of any study that uses it. Osborne and Feit go on to say:

> When race is used as a variable in research, there is a tendency to assume that the results obtained are a manifestation of the biology of racial differences; race as a variable implies that a genetic reason may explain differences in incidence, severity, or outcome of medical conditions.63

Thus, one can argue that the use of race as a biological variable in medical studies is in fact a “subtle form of racism.”63 Williams and colleagues concur that using racial categories in scientific studies encourages thinking in “racial stereotypes.”61

This subtle racism that pervades medical research may overflow into medical practice, leading to important consequences for patients. For instance, an African-American woman with an ectopic pregnancy or an appendicitis may be misdiagnosed by the practitioner who recalls that there is a high incidence of pelvic inflammatory disease in “black” women.63 Another physician may miss the diagnosis of sickle cell anemia, which “... is more accurately conceptualized as a geographical rather than a race-based disease,”[64] for a patient who is Greek because there is a common misconception that sickle cell anemia only affects

"blacks." In psychiatry, it has been noted that "clinician prejudice" has led to over-diagnosis of schizophrenia in African-Americans.[65] Thus, the use of race in the medical literature may contribute substantially to racism, subtle though it may be, in medical practice.

It may be difficult to understand why race continues to appear in medical literature if race has been invalidated by science and if many authors have pointed out the danger of racial stereotyping. Williams and colleagues have made an astute attempt at explaining this inconsistency. They claim that:

> [c]onceptions of race that emphasize biology are least threatening to the status quo. If racial or ethnic differences in health result from innate biological differences, then societal structures and policies that may be involved in the production of disease are absolved from responsibility and can remain intact.66

If genetics did indeed explain all the observed health status differences among various ethnic groups, then physicians could do little more than shrug their shoulders and wait until effective gene therapies arrive. If, on the contrary, these observed differences were the direct result of social inequities, physicians would be forced to perform the difficult task of eliminating institutional racism in their profession. Furthermore, they would be impelled to lobby other societal institutions to identify and eradicate the conditions that lead to increased morbidity and mortality in minority ethnic groups. The medical community may be reluctant to take such actions so as not to admit their culpability. Physicians may also hesitate to play the role of lobbyist since this would extend beyond the traditional doctor-patient relationship. Nevertheless, the physician, as a professional and as a patient advocate, has the duty to perform both of these tasks because the best medicine is to prevent disease, not simply to observe and

treat it.

The consequences of not removing race from the medical literature are significant. It perpetuates the racialization of medical treatment. If you establish a “biologic” or “scientific” difference among races, then you can argue for different treatment of each group.[66] This is just as true in medicine as it is in politics. We have learned this lesson once already in medicine following the racist management of the Tuskegee Syphilis Study. Must we wait for a second lesson to come along before we change our ways?

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