

In: *Race in 21<sup>st</sup> Century America*,  
Curtis Stokes, Theresa Melendez and Genice Rhodes-Reed (eds.),  
Michigan State University Press: East Lansing 2001 pgs 25-47.

2

## Six Wrongs of Racial Science

ALAN H. GOODMAN

### INTRODUCTION: AN UPSET STOMACH

A friend recently convinced me to buy a bottle of thirty-six TUMS® Ultras in assorted fruit flavors, a product of SmithKline Beecham, containing 800 mg of calcium per tablet. I also purchased less-expensive bottles of Drug Guild's "Calcium Antacid Tablets" and "Equate extra strength antacid tablets." The former contains 150 peppermint-flavor tablets, containing 400 mg of calcium per tablet. The latter, manufactured by Perrigo Company, contains 96 tropical-flavor tablets, each with 600 mg of calcium.

On all the bottles the first sentence under the heading IMPORTANT INFORMATION ON OSTEOPOROSIS is exactly the same: "Regular exercise and a healthy diet with enough calcium helps teen and young adult white and Asian women maintain good bone health and may reduce their risk of osteoporosis later in life." The producers of antacids and calcium supplements apparently have agreed that the scientific research is unambiguous on the fact that regular exercise, a healthy diet, and enough calcium may help young women of some race/ethnic groups. Conversely, the benefits are either not proven or not to be had for members of other race/ethnic groups.

Assuming for a moment that the osteoporosis information is based on solid research, problems quickly arise in moving from groups in research studies to real individuals. Who is white or Asian, and who, exactly, is not? Will the TUMS® benefit a teenager with an Asian mother and an African American father? Should a Latina regularly exercise to help prevent osteoporosis if she thinks of herself as white, but not if she thinks of herself as black? Whose opinion counts when she thinks of herself as black but her physician considers her to be white? Will the TUMS® benefit a dark-skinned white, but not a light-skinned black? Are Native Americans considered Asians, as they are in most racial typologies, or another race, as they are in the U.S. Census? If they are Asians, does that mean they can benefit from taking TUMS®? Conversely, if their race is Native American will they not benefit? Below the fluidities of group membership, deeper problems may exist. Is the generalization a valid one? Is racial science good science?

My research focuses on the everyday uses of race in science. The osteoporosis information is an example of medical advice and treatment that is based on an individual's purported race. Under the medical advice is "everyday racial science," in which individuals, their bones, bodies, and minds, are unproblematically divided into racial groups. Rather than focus on the more obvious racial science at work in studies of minds, behavior, and intelligence,<sup>1</sup> I wish to show that subtle uses of race, exemplified by the TUMS® label, may cause even more harm. The bottom line is that wherever race is used as shorthand for human biological variation, the results will be badly flawed. People who believe or act upon the results may cause harm to others and themselves.

This chapter is organized around six reasons why racial science is bad science, both wrong and harmful. The first has to do with the ideological and theoretical incompatibility of race with evolutionary theory. The second, third, and fourth reasons highlight the fundamental incompatibility of race with the structure or "facts" of human variation. The last two reasons focus on the incompatibility of a changing and ill-defined concept with the scientific necessities of replicability. No one reason shows certainly that race is a myth or a totally useless scientific concept. However, the combined consequences lead to the conclusion that biological race should be placed on the scrap heap of outdated scientific ideas.

## SIX REASONS WHY RACE IS WRONG AND HARMFUL

*Race is a Pre-Darwinian and Prescientific Concept  
that is Incompatible with Evolutionary Theory.*

The idea of biological race embodies two central beliefs (Hannaford 1996; Smedley 1999):

- Humans are divisible into a small and discrete number of types.
- Types are old, primordial, fixed, and unchanging.

In addition, biological race takes on power and meaning in the common implications that

- Types have defining characteristics and are hierarchically arranged.
- The type explains the individual: an individual's biology and behavior are in large part explicable by the type/race of which the individual is a member.

The idea of racial types emerged from the Platonic notion of ideal types and the Christian idea of a great chain of being. The Platonic notion of ideal types holds that the physical and material world is a reflection of a world of pure ideals. The real world of ideals is explicitly stable; change exists only in the potential for devolution or poor copy from the ideal. This method of science and philosophy is neither experimental nor empirical; rather, it is based on discovery through thinking about or imagining ideal types. An inanimate object, such as a chair, although a human construction, is evaluated as to how closely it conforms to the ideal type chair. But is there an ideal chair? What are its dimensions, materials, and characteristics? Who gets to imagine it?

In a similar way, ideal types of animals and plants were imagined. Existing fauna and flora may be evaluated as to how closely they resemble the ideal type of each animal and plant. Plato imagined ideal male and female types, and ideal soldiers, servants, and aristocrats. Individuals could be evaluated in relationship to how well they exemplified these ideal types.

Platonic idealism, of course, is flawed, because it rests on the assumption that there are ideal types "out there." It is thoroughly incapable of seeing how types are socially constructed. This lack of reflexivity is all the more obvious in the ranking of races embedded in the great chain of being. While Platonic idealism set up the

notion of ideal types, it is the idea of a "great chain of being" that provides an explicit ranking and worth.

The early Christian idea of a great chain of being arranges "all God's creatures" on rungs or stairs (Lovejoy 1936). The higher the rung one occupied, the closer one was to God, and the lower the rung, the further one was from God. As is made clear in drawing, Christian Europeans, the constructors of the great chain, occupied the top rungs, while other humans were further down the chain, typically placed between Europeans and the primate species.<sup>2</sup> Each type or species and each race had a fixed and unchanging place relative to God.

The preceding notwithstanding, there was little officially sanctioned and scientific concern over differences in humans before 1492. This changed when Europeans began to develop trade routes and expand markets (Hannaford 1996), along with the development of North American slavery (Smedley 1999). After 1492, the idea of race became useful as a means of justifying European capitalist expansion.

The goal of the now developing science of human variation was to name and describe types and to then demonstrate the unique behavioral and biological characteristics of each type. The agreed upon start of this natural science, which was later to become anthropology, includes Buffon's *Histoire Naturelle* (1749) and Linnaeus's racial classification in the tenth edition of *Systema Naturae* (1758). For Linnaeus, race explained, or at least correlated with, systems of government and psychological characteristics. In addition to classifying a "monstrous" species of homo with various subspecies types, and a wild man (*Ferus*) type of *Homo sapiens*, Linnaeus recognized four racial types: *Americanus*, *Europaeus*, *Asiaticus*, and *Afer*. According to Linnaeus (1758), *Americanus* is regulated by custom and paints himself with fine red lines; *Asiaticus* is haughty and covetous and governed by opinion; *Afer* is indolent, anoints himself with grease, and is governed by caprice; and *Europaeus* is gentle, acute, inventive, and governed by law. Unsurprisingly, Linnaeus was a European.

Following Buffon and Linnaeus, other French, German, British, and American natural historians continued to tinker with racial classifications and to consider why variation existed. After the middle of the eighteenth century, race became a worldview and a paradigm for both scientists and politicians. It became a popularly recognized concept, so much so that race was often taken to be natural and real. It filtered into languages and etched itself on the minds of eighteenth- to twentieth-century Europeans (Hannaford 1996; Smedley 1999; Stepan 1982; Todorov 1993). The process by which a folk idea such as race becomes a scientific

one and is then made to seem real is surely variable. However, it is clear that ideas that are useful to the ruling class (with control of legislation, access to and control over information, and so on) tend over time to be accepted as certain, natural, and real (Fields 1990; Mills 1997).

There is disagreement over whether race is any less typological today than it was centuries ago. In a 1985 survey, half of all physical anthropologists agreed with the statement, "there are biological races within the species *Homo sapiens*" (Lieberman, Stevenson, and Reynolds 1989). Unlike in Buffon's time, typology has now fallen out of vogue. In fact, "typologist" is one of the worst epithets one can hurl at a physical anthropologist. Physical anthropologists who believe race is biologically real, therefore, claim that they are not typologists. George Gill, a forensic anthropologist, defends this position by arguing:

Confusion and ambiguity surrounding the controversial four letter word "race" was alleviated greatly by the early 1950s following the classic work of Coon, Garn and Birdsell (1950). . . . The underlying basis for the race concept (and racial taxonomy) has shifted entirely in recent decades from a typological to a population one. (Gill 1994, 163)

To the contrary, Coon, Garn, and Birdsell (1950) did not and could not transform race. They tried to graft race into a population framework, but the graft failed to take. Populations are dynamic and respond to evolutionary pressures at local levels. Races, on the other hand, are poorly defined and unchanging groupings. If races become populations, they no longer are races. In a review of Coon's *The Living Races of Man* (1965), a book in which Coon expands upon the prior study (Coon, Garn, and Birdsell 1950), Buettner-Janusch summarizes: "Typology, typology, typology, nothing but typology" (1966, 187).

The concept of race remains a typological and nonevolutionary concept. As well as race fit an idealist and typological worldview, it misfit Darwinian theory, in which variations accumulate and humans change over time and place. No matter how race is recast, it cannot shake its typological core and accommodate the fluidity of human variation. To paraphrase the paleontologist G. G. Simpson, all ideas about humanity before 1859—not least, type and race—are invalid.

Surprisingly, then, many continue to use race despite the fact that the notion of fixed, ideal types should logically have been replaced well over a hundred years ago with the advent of Darwinism and the ascension of evolutionary theory in biology and anthropology. I suggest that the concept survives where it does not fit

either fact or theory, because: (1) it became reified by constant use, (2) it became conflated with human variation, and (3) it was and is politically useful.

*Most Traits Are Continuous, Varying, and Clinally Distributed.*

Human variation tends to be gradual, with only subtle differences between nearby groups. The more distant two groups are from each other, the greater the difference will be between them. In contrast, the idea of race implies that variation is discontinuous and sharp between racial groups, and inconsequential within a race. Human variation, however, is not like that.

A biological variable or trait is a measurable quality or quantity. It can be most anything one might think of, such as the color of one's eyes, the blood group one belongs to, or the rate at which one loses hair with age. In addition to traits possessed by individuals, traits can also define and characterize groups. On average, females are shorter than are males and African Americans have darker skin than do whites. The frequency of the Duffy  $Fy^a$  allele is 9.5 in Guyana and 7.8 among the Bantu of the Republic of South Africa (Molnar 1992). The rate of low birth weight in Illinois in the 1990s was 13.2 percent among infants born to African American women and 4.3 percent among infants born to white women (David and Collins 1997).

A potential problem with moving from individual-level traits to group-level traits may occur when the group's statistics (average, frequencies, etc.) are used to characterize individuals. In the preceding examples, knowing that the Duffy  $Fy^a$  allele is more common in Guyana than among the Bantu does not ascertain whether a specific individual actually has the allele. Each person needs to be tested individually. Similarly, females are shorter than males, but this does not mean that every female is short or that every male is tall.

Furthermore, if groups are defined on the basis of biological trait frequencies, then there are typically no clear borders where one group begins and another ends. Say, for example, we decide to use height to define groups. If we determined that there were to be two groups, where would we make the division between tall and short people? The "cutoff" point is arbitrary and often chosen for convenience. Those near the cutoff are more like each other than they are like others in their group.

Similarly, it is impossible to fix boundaries between races. There are no natural gaps. The division point is arbitrary and up to the whim of the classifier. The continent from which one originates, no less than one's race, is meaningless.

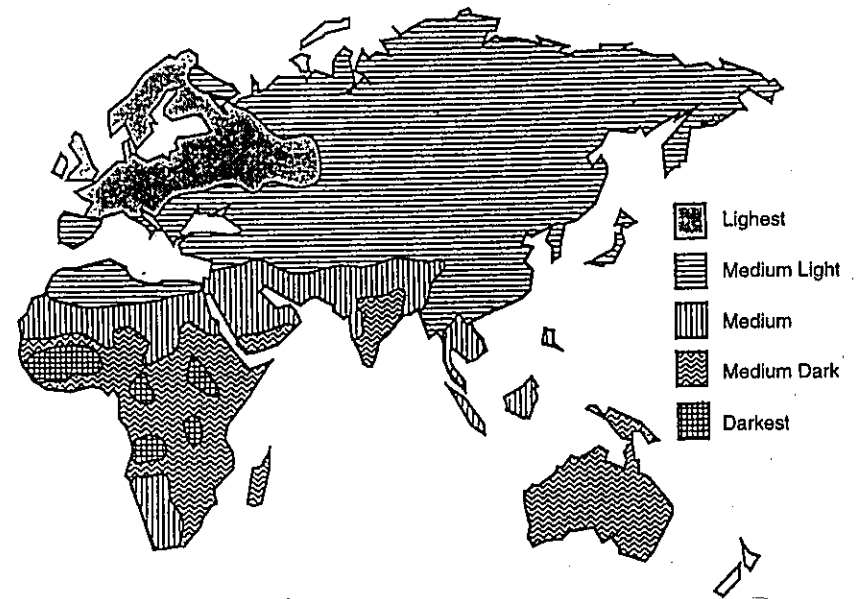


Figure 1. The distribution of skin color in the "old world" circa 1492. Note that variation is mostly on a north-to-south gradient.

The classification of a continuous trait such as skin color into discrete units diminishes the complexity of human variation, and, as a result, how well we can understand that variation. With continuous variation central tendencies become nearly meaningless; types are nonpredictive. Individuals near the race/type borders are more like individuals just over the border, though classified differently, than they are like most individuals in their race/type. This form of continuous variation is called clinal variation.

The geographic distribution of skin color varies clinally mainly on a north-south gradient (Figure 1). Africa and Asia are home to both light- and dark-skinned people. Taking into account the pattern of variation is the first step to understanding the cause of skin color variation. Might it be related to the protection dark skin affords against skin cancer or the greater ability of light-colored skin to facilitate the synthesis of vitamin D? These are the types of questions that can be asked when variation is not reduced to race.

The frequency of the sickle cell gene peaks in West Africa and gradually decreases as one moves away from West Africa (Angola) or another epicenter such as Northeast Arabia (figure 2).

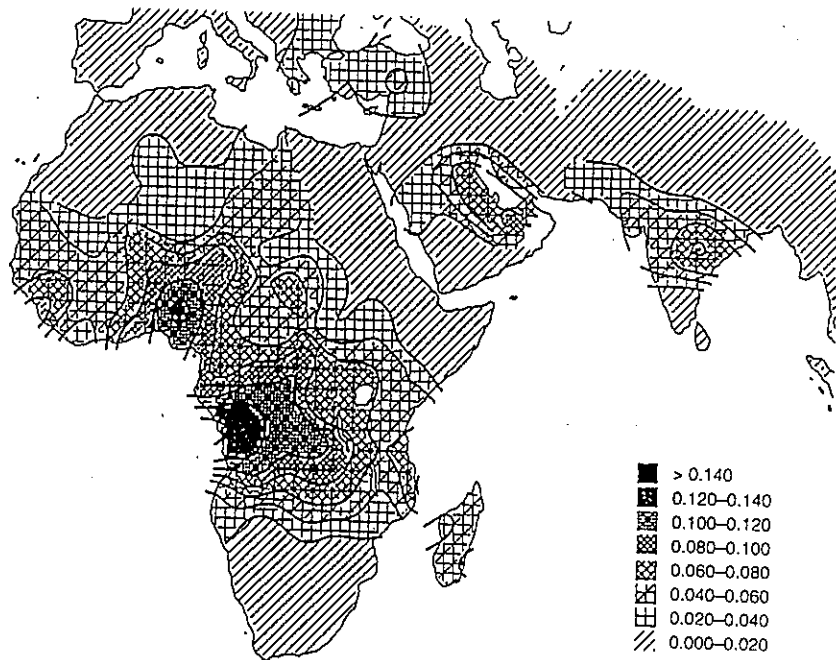


Figure 2. The distribution of sickle cell trait in the "old world" circa 1492. This is an example of clinal variation.

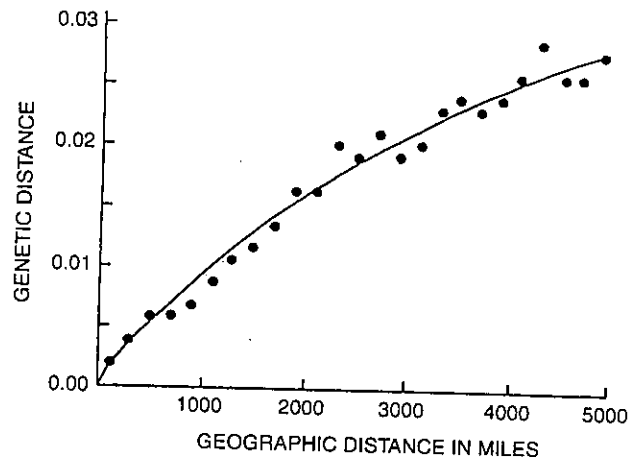


Figure 3. A plot of the relationship between genetic distance and geographic distance (from Templeton 1998).

Clinal variation, or variation that accumulates with distance, is the rule. Templeton (1998) has recently redrawn a graph of human genetic distance versus geographic distance (figure 3). Geographic distance almost totally explains genetic distance. In 1962 Frank Livingstone pointed out that "there are no races, only clines." Moreover, as will be reinforced in the following, thinking racially prevents one from understanding how variation evolved and why it might be significant.

#### *Most Trait Pairs Are Nonconcordant.*

The racial approach to human variation rests on the assumption that two or more traits tend to vary together in a consistent way. The importance of this, if it were true, is that one trait could explain or predict another. The truth, however, is exactly the opposite.

A few traits, the exceptions, are concordant. Height is concordant with leg length, and slightly less strongly with weight. In a similar way, skin color is concordant with hair and eye color, and less so with hair texture. However, knowing skin color provides no insight into height or any other anthropometric attribute. There is no reason it should. This is one reason why it might be said, "race is skin deep."

To get a feel for nonconcordance, imagine a layer cake in which each layer is a trait (figure 4). The top layer is skin color and the third layer is ear length. Also imagine that geographical groups from around the world occupy each place in the layer. Placing a pin through them would link up the values for a specific group for each trait as the pin passes through the layer. Alternatively, one could imagine that individuals are represented, and passing the pin through a specific spot would provide the individual's values for each trait. If the layer cake exemplifies human variation, then the color of the top layer will not give any insight into the color (or trait distribution) in the deeper layers. Knowing skin color tells us nothing of deeper traits.

#### *Within-Group Variation Is Much Greater than Among-Group Variation.*

There is no agreed upon test for whether humans are in any reasonable way groupable into subspecies or races. However, the clearest test is by statistical apportionment of variation within races versus among races. This test assesses the proportion of variation that is statistically explained by race. Humans fail the test.

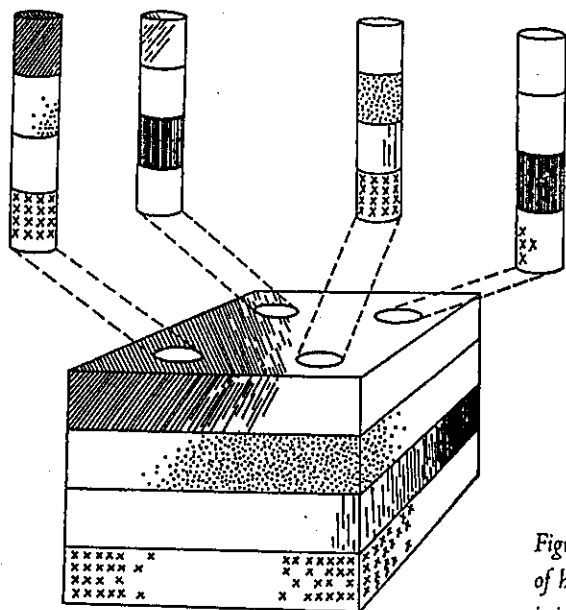


Figure 4. A "layer cake" model of human variation. Each layer is independent of the others.

The notion of subspecies has been under intellectual attack in zoology since the 1950s (Wilson and Brown 1953). At this time it was realized that many local varieties simply had *not* been reproductively isolated for long enough to have evolved and differentiated to a consequential degree. If they did not separately evolve, then most of the variation within the species would be distributed rather uniformly over the species, so that small groups of individuals would contain most of the species variation. If, on the other hand, they had been isolated for some time, then some of the variation would concentrate in the groups. The same small group of individuals would explain a smaller proportion of the species variation.

In thinking about humanity's past, we are faced with the realization that we are a relatively young species. In addition, we have a long generation time, and we seem to have encountered few major barriers that would cause reproductive isolation. It would therefore seem that there should not be very much variation among groups or so-called races. Although this model of small variation among races is not a common perception, it turns out to be true.

To think about the distribution of human variation, imagine a simple scenario: a trait that takes on two values—short or tall, type O blood or not type O blood. Green marbles represent one value and red marbles represent the other.

There are three races represented by three clear jars. On a species level there are 50 percent green and 50 percent red marbles. Thinking about human variation, should the green and red marbles go into each race jar in the 50 percent proportion, or in some very different proportion, perhaps as much as 100 percent green marbles in one race jar and 100 percent red marbles in another? The former might be called a state of extreme "racelessness" and the latter a state of extreme "raceness." Interestingly, most students think of human variation as more like the latter. Yet, in reality, human variation is more like the former.

In a classic article published in 1972, Richard Lewontin estimated the proportion of human variation that could be statistically explained by races. Lewontin took data on blood group polymorphisms (those who have two or more alleles in high frequency, such as blood types A, B, AB, and O) and tested how the blood groups were distributed and how much variation was explained at three levels:

- by local group
- within a race but among local groups
- among races

The first level of variation is equivalent to the small group of individuals discussed previously. The second level is the additional variation that is found within races, which is equivalent to several small chunks combining to form one large chunk. Finally, the last level is what is actually explained by race. Lewontin found that on average 85.4 percent of variation was explained at the first level, while only 6.3 percent of variation was explained by purported race (Lewontin 1972). In other words, the green and red marbles are nearly equally distributed. On average, two individuals of the same purported race are only marginally more genetically alike than any two individuals chosen at random.

Because of nonconcordance and within-group variation, race has little explanatory power. If we know a person's race, we know little more about that person. Race tells us little about the processes governing human variation, and its predictive value for individual traits is trivial. Finally, the roughly 6 percent of biological variation that is statistically explained by race is better explained by geographic proximity or clines (figure 3). Because genetic variation simply increases with geographic distance, race is not even necessary to explain this small fraction of variation. Race is totally replaceable.

Although there is no single test for subspecies or racial structure, the one most often agreed upon builds off of Lewontin's analysis of the apportionment of

variation. If the relative degree of variation among races is great compared to the variation within a single race, then one might suggest that subspecies or races are meaningful in at least an evolutionary perspective. On the other hand, if the relative degree of variation among races is small compared to the variation within a single race, then races are less meaningful. The  $F_{ST}$  of Wright (1969) is essentially the proportion of variation explained between races versus the proportion explained within race.

In the nonhuman literature the standard quantitative threshold used to determine if there is a potential subspecies structure is an  $F_{ST}$  of 0.25 to 0.30 or greater (Smith, Chiszar, and Montanucci 1997). From a larger set of data than Lewontin's, the human  $F_{ST}$  has recently been calculated to be 0.156 (Barbujani et al. 1997). Humans thus fail the test for biological races.

To summarize to this point, human variation tends to be continuous and nonconcordant. The fact of continuous variation makes problematical any efforts to think an individual is like the average of his or her group. It also makes the dividing lines between races rather arbitrary. The fact of nonconcordant variation calls into question any effort to do more than explain a single trait based on that trait. We end up with circularities: skin color explains race and race explains skin color, but neither explains much else. In some senses, these two problems parallel and forecast the two practical ways that race is used: as an identification method, and as a predictor.

The fact that almost all variation is statistically found within local groups seems counterintuitive. We are socialized into keying on physical features that do vary and have come to signify different groups. Yet, below the skin we are much the same. As previously noted, we have not had time to differentiate and we are not reproductively isolated.

#### *The Classification Is Not Stable across Space and Time.*

The practice of classification of objects into groups is something we do regularly. Classification helps us to order and make sense of our world. Even when classification systems are imperfect, even when they skip complexities, they may still be useful. Hat size does not account for the shape of the head, but it is a useful place to start looking for a good fit. Without much thought we divide the entire variation in human morphology into a few sizes: small, medium, large, and sometimes extra small and extra large. With this great reduction of human morphological variability, almost everyone can find a sweater that fits well enough. The

idea of social class is an abstraction; one might even say it is socially constructed. It is defined in different ways by different people, and it is certainly dynamic and unstable.

So why not think of races as a useful first effort at classification? Unfortunately, because of instabilities in racial classification, combined with the conflation of biology and culture, this analogy fails with terrible consequences when it is applied to human variation. With racial classification, division points are arbitrary and up to the whim of the classifier. Thus, an individual who is classified as "European" or "white" at one time and place may be classified as "mixed," "Hindu," "quadroon," "octoroon," "colored," "mulatto," "mestizo," or "black" at another time and place (Lee 1993). Jews were considered to be a separate race (or even many racial types) before World War II, but they came to be classified as white after World War II (Sacks 1998). Similar "whitening" happened for the Irish and Southern Europeans (Jacobson 1998). Fish (1995) describes how the race of his wife and daughter changes when they fly from the United States to Brazil. Their biologies do not change, but the cultural classification system does.

Changing racial classification is fine if one has a good sense of how it is changing. If one manufacturer's size small is too tight, you try a size medium. However, because sciences such as medicine are based on repeatability, changing classifications are often disastrous. Would you want your physician to base a critical decision about your treatment on his understanding of a racial group in Brazil or based on races from the late 1800s? As I will detail in the following, the TUMS<sup>®</sup> label is exactly this type of mistake.

Research into the identification of an individual's race based on his or her skull shape provides an example of the difficulties inherent in any system of racial classification. A common problem in forensic science is identification of homicide victims, so much research has gone into the effort to enable scientists to determine race based on measurements taken from skulls. Imagine a large group of skulls from individuals who have been socially defined as white, black, and Native American. Giles and Elliot (1962) showed that one could separate about 85 percent of the skulls into the appropriate racial groups by applying a statistical procedure called discriminant function to a series of skull measurements. In this case, all the skulls were from the previous century or older.

However, for this procedure of "racing skulls" to have some utility it would seem that a high percentage of correct classification would also be necessary when this procedure is applied to skulls today. When the method of Giles and Elliot was applied to contemporary skulls of known race—that is, when it was re-tested—

the percentage of skulls that were correctly classified was reduced to around one in three. The procedure is therefore no better than random. The reasons for this seem obvious enough: (1) there is great variation within race and little variation among races, and (2) the race groups that Giles and Elliot's skulls came from no longer exist (Goodman 1997).

If you do not believe that races change—not just by social definition, but in their biology, too—then consider the following. Steve Ousley reports in the introduction to a computer program (FORDISC-2) that a discriminant function formula can separate the skulls of U.S. white males born between 1840 and 1890 from those born between 1930 and 1980 with 88-96 percent accuracy.

The point is that racial classification and the perceptions of racial boundaries are thoroughly fluid. Because of this fluidity one can never be certain how well a racial generalization will apply.

#### *The Conflation of Biology and Lived Experience.*

When pronouncements are made about racial variation in health or some other trait, there is typically little discussion of the underlying source of the variation. Is a racial difference in hypertension due to genetic differences among races, or does such a difference have a more transient association with race, perhaps being due to differences in living conditions? If the former is true, then it will lead to research and practice in one direction. If the latter is true, it will lead in an entirely different direction. Leaving race as a proxy variable for either lived experience or genetics makes extremely problematical any effort to confidently act on the data.

The conflation of biology and lived experience is a double leap of scientific faith (Goodman 1997; 2000). The first leap of faith is in assuming that a difference between two groups is genetic without controlling for environmental factors and lived experience. The second leap is in assuming that a genetic difference falls along racial lines. The first leap might be called geneticization (Lippman 1991). We live in an age in which many things are thought to be genetic in their origins, but this is often an underexamined theory. The second leap might be called the geneticization of race. Even if there are race differences, and even if an outcome is genetically determined, there is still no reason to leap to the conclusion that race is a proxy for genetics or that the genes break along socially constructed and fluid racial lines.

If a disease turns out to be genetic, then we are still faced with the assumption that races are genetically distinct. However, the preceding discussion on the

apportionment of variation has made it clear that this is not so. If a disease is in large part nongenetic, then this implies that race is only a transitional covariate of a nonracial cause. As the conditions of life, such as access to medical care, diet, and exposure to lead in childhood change, the strength of the racial generalizations change.

A recent example of this type of double leap of scientific faith comes from a recent article on race and birth weight (David and Collins 1997). It is widely believed that a large portion of the variation in mean birth weight between black and white babies in the United States is genetic. Thus, the relationship of *in utero* conditions to low birth weight in blacks has been largely neglected in the research. In addition to presenting and comparing the birth weights of babies born to U.S.-born white and black women in Illinois between 1980 and 1995, David and Collins (1997) also presented the birth weights of babies born to African-born women during that timespan. If differences in birth weight between the races are due to genetics, then birth weights of babies born to African-born women should be near the birth weights of babies born to U.S.-born black women. If one invokes an additive model of human variation, then the birth weights of babies born to African American women would be expected to be between the African and U.S. white means, and approximately 25 percent of the way from the African mean and 75 percent from the white mean.<sup>3</sup>

The results of the study, however, are nothing like this. The mean birth weight of the babies born to U.S. white women was 3,446 grams, and that of the babies born to African women was 3,333 grams. Babies born to U.S.-born black women had an average birth weight of only 3,089 grams, much lower than either of the other groups. The explanation for this distribution of birth weights is, therefore, certainly not genetics. Seemingly, the place to look is in diet, health care, and other socioeconomic conditions that are known to affect *in utero* conditions. Such an example clearly shows how erroneous it can be to make scientific generalizations based on racial classifications.

To summarize, these six points show that human biological variation is real, but that racial classification is not a useful way to think about it. Race and racism are real, and so is human variation. However, taking race to be a determinant of human biological variation simply does not fit the facts. Such an assumption holds back scientific work, and may even cause harm.



RACE AND OSTEOPOROSIS:  
AN EVERYDAY MISUSE OF RACE

In the nineteenth and early twentieth centuries, the idea was pervasive that certain races got certain diseases. Whites, for example, got cancer, a disease of civilization.<sup>4</sup> This paradigm of racially distinct diseases has slowly been updated in epidemiological discourse by the idea that race is a disease risk factor. Work on osteoporosis and bone loss provides an example. Osteoporosis is an age-related disorder characterized by decreased bone mass and increased susceptibility to fractures. By 1980 it was estimated that osteoporosis affected fifteen to twenty million people in the United States and that it was the underlying cause of about 1.3 million fractures per year (Wasserman and Barzel 1987). Osteoporosis is a serious health problem, and better understanding of its etiology is obviously critical for improved screening, treatment, and prevention.

Since at least the nineteenth-century, white scientists have thought that blacks have thicker bones than whites. In his "Introduction to Anthropology" Dr. Theodor Waitz wrote: "The skeleton of the Negro is heavier, the bones thicker. . . . This is especially the case with regard to the skull, which is hard and unusually thick, so that in fighting, Negroes, men and women, butt each other like rams without exhibiting much sensibility" (1863, 93).

Over a century later, a review of the etiology of osteoporosis listed race as the third risk factor, after age and sex and before heredity, physical activity, and dietary factors (Wasserman and Barzel 1987). The section on race begins with the declarative sentence: "It is a well-known fact that blacks do not suffer from osteoporosis" (1987, 285). That "fact" is backed up by reference to one and only one study: a seminal paper by Mildred Trotter, G. E. Bowman, and R. R. Paterson (1960) on bone density changes by age, sex, and race. Given that physicians routinely vary osteoporosis treatment decisions based on their perceptions of the race of their patients, and now given that TUMS® excludes black women from its label, it is worth ensuring that the study of Trotter and colleagues (1960) proves the point that "blacks do not suffer from osteoporosis."

DECONSTRUCTING THE TROTTER STUDY

Trotter, Bowman, and Paterson (1960) measured the bones of eighty cadavers from Washington University, selected to provide twenty black males, twenty black

females, twenty white males, and twenty white females. Individuals' ages at death ranged from twenty-five to one hundred years, and the mean group ages at death varied from 59.6 in black males to 67.2 in white females. The authors do not provide a description of the method of selecting cadavers or matching their race-sex samples for causes of death, socioeconomic status, diet, or other known risk factors. The dried bones were weighed and their volumes estimated by displacement of millet seed. Ten different bones were studied on each cadaver.

The authors conclude that cervical, thoracic, and lumbar vertebra, sacra, humeri, and ulnae are heavier in blacks than in whites. They did not find a significant racial difference for radii, tibiae, ribs, and femora. The latter bone is particularly noteworthy because femoral neck fractures are one of the most serious effects of osteoporosis. Finally, Trotter and colleagues note that the decline in bone density with age, "occurred at approximately the same rate" in each sex-race group (57, emphasis added). Race or sex differences, where they did occur, were due to the fact that one group started with greater mean density. For example, bone density of black males at seventy years may be equal to or less than that of white males at sixty years of age. The clinical significance, in my opinion, is that different sex-race groups cross a threshold of bone density at different mean ages. On average, blacks start with more bone, but they suffer bone loss at much the same rate as do whites.

Remember for a moment that races do not get diseases; individuals do. It is of more than theoretical importance to know if a proclamation of group protection pertains to individuals. Related to this, in a follow-up study, Trotter and Hixon (1973) provide scatterplots of the original data of Trotter, Bowman, and Paterson (1960) on individual bone densities by age, sex, and race. The scatterplots are instructive because they provide a visual sense of the degree of variation within race-sex groups, and of how well individuals conform to the central tendency of the group. Figure 5 is a copy of the scatterplot of bone densities for the radius. White males are represented by open circles, white females by open squares, black males by black dots, and black females by black squares. This scatterplot clearly illustrates an overall trend of declining bone densities with age. Far less clear is a racial difference in the rate of decline in density with age. In fact, of the six lowest bone density points, one represents a black female and the remaining five represent black males.

What, then, is the basis for the "well-known fact that blacks do not suffer from osteoporosis" (Wasserman and Barzel 1987, 285)? What seems to have occurred was a double leap of faith. First, a condition (cysts, osteoporosis) was determined to be genetic (although environment was not adequately examined).

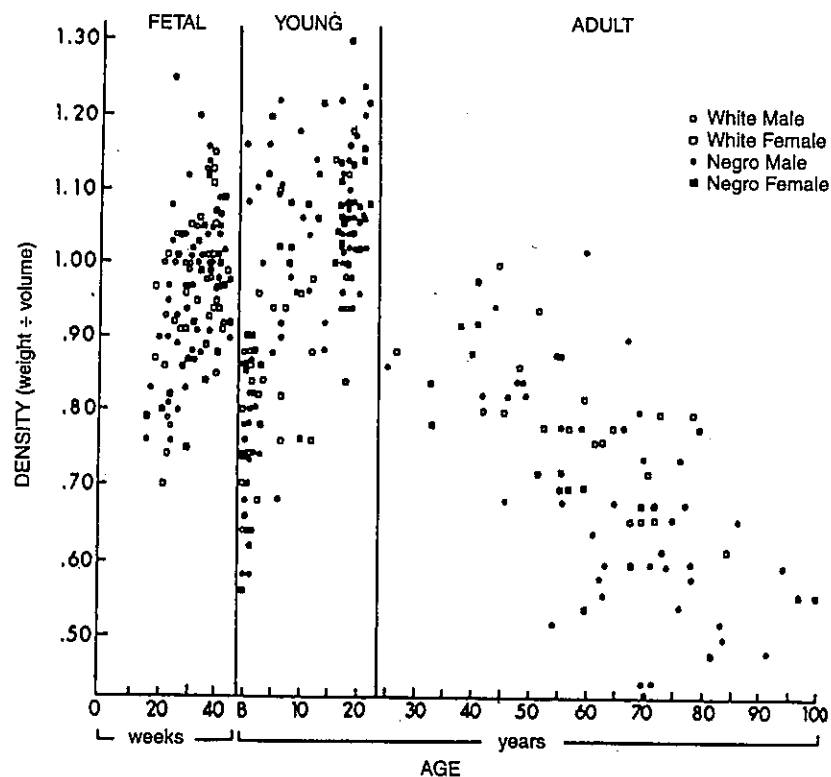


Figure 5. Scatter diagram of radii bone density values by race, sex, and age (modified from Trotter and Hixon 1973, 12, fig. 2).

Could the differences in bone density of the subjects have been due to diet or other known risk factors, such as exposure to sunlight and activity patterns? In the absence of such data there was an assumption of genetic etiology. Then, it was assumed that anything that was genetic was panracial, a characteristic of all members of the racial-type.

Although Wasserman and Barzel (1987) do not cite them, other studies have also found "racial differences" in bone density and fracture rates. However, even if there were a million studies showing such a difference, it would still not follow from this that blacks are immune to osteoporosis, that they should not be given advice and treatment to help prevent or slow bone loss, or that they should not be encouraged to take TUMS®. Blacks do suffer from age-related bone loss, they do

suffer from bone breaks due to bone loss, and they do suffer the consequences of medical neglect.

## CONCLUSIONS

In a *Newsweek* cover story, Sharon Begley recently declared that when it comes to a critique of race, "scientists got there first" (1995, 67). Ashley Montagu (1942) made clear over a half century ago that race is not a reality; it is not a thing. Rather, it is the phlogiston of our time. We tend to think of it as a reality because constant use and lack of questioning of its underlying reality have reified it. Race is a way of constructing and thinking about human variability, but it is a poor and outdated construct.

Definitions of race are varied and protean. For example, some classifications are based on geographic origin (with some assumed biological concordance), others are based on clusters of traits, and others still are based on bureaucratic and social definitions (again, with an assumption of a biological basis). There is no agreed upon definition (Brace 1982). Furthermore, all efforts at a scientific (widely accepted, reliable) definition have failed. Brace captures the assumptive and protean nature of race when he comments on racialist research:

The connection between the biology discussed and the races named at the end is never clearly spelled out, and in fact the attentive reader cannot discover, from the information presented, just how the racial classification was constructed—other than the fact that this just seems to be the way anthropologists have always done things. (Brace 1982, 21)

With so many differences in definition it is not surprising that there is also no agreement on the names and numbers of races. The inability to define race, to agree on how many there are, or to agree upon what biological criteria make a race, shows that this concept is slippery at best, making for problematic politics and biology.

Despite efforts to reinvent it, race remains a typological concept that has little use as an explanatory variable. Race and adaptation do not fit well together. Adaptations occur on the scale of individuals and local groups, responding to specific, local environmental conditions, and the responses are mostly nongenetic. Human variation is far more complex and intricately varietal than race allows for,

and certainly does not obey racial boundaries. Race in epidemiological studies should be considered scientific malpractice. It is a form of ideological iatrogenesis.

Unfortunately, the same flawed logic that is inherent to racial studies of intelligence and behavior also applies to racial studies of less politicized traits, such as susceptibility to osteoporosis. If one considers the countless, everyday places where race is used inappropriately, then one can get a sense of the hidden harms of thinking race and human variation are one and the same. Thus, I do not call for an end to race. Race is very real, but it is not biology. Realizing this makes clear some research challenges: studies of human variation free of a racial model, studies of social differences in racial thinking and racial formation, and studies of the consequences of racism on health and other outcomes.

#### ACKNOWLEDGMENTS

Thanks to Debra Martin (Hampshire College) for bringing to our attention the racial assumption in writing on osteoporosis, and to Michelle Murrain (Hampshire College), who alerted me to the "tums" label. Special thanks to Curtis Stokes for providing the best of forums in which to share ideas about the significance of race. Parts of this paper are a revision of Goodman (1997).

#### NOTES

1. My focus here is not on the clearly racist science that all too often continues. For example, J. Philippe Rushton, the contemporary devotee of esoteric racial measurements, claims that black males produce more testosterone and have larger genitalia than do whites (1994). Rushton uses this fact to explain a diversity of phenomena, from the AIDS epidemic to purported intelligence differences among races. In Rushton's pop evolution, races put their energies into either sexuality or intelligence—that is, big brains or big penises. Pseudoscientific works such as this need to be challenged, and many have done so. Fewer have focused on the more subtle uses of race.
2. Although the great chain of being is no longer a common motif, the idea of a ranking of progress or evolution remains. In nearly all trees of racial evolution published to this date, "Caucasians" are placed on the highest branches, most separated from apes, while non-Caucasians are placed closer to the apes and sometimes on lower branches (see discussion in Wolpoff and Caspari 1997)

3. That Europeans contributed to the gene pool of African Americans (perhaps contributing as much as 25 percent of the African American gene pool) is widely acknowledged in the genetics literature (Kittles and Royal 1999). The recent finding that Thomas Jefferson fathered a child of a slave, Sally Hemming, is one example of the permeability of racial borders (Foster et al. 1998).
4. This same essentialist thinking is what provided the ideological basis for the infamous Tuskegee syphilis study of 1932. The belief was that the course of syphilis would be different in blacks than in whites, that syphilis might transfer to whites via sexual relations, and that an epidemic of syphilis in blacks might further show their inability to cope with civilization (Brandt 1978).

#### REFERENCES

- Barbujani, G., A. Magagni, E. Minchi, and L. L. Cavalli-Sforza. 1997. An apportionment of human DNA diversity. *Proceedings of the National Academy of Science* 94:4516–19.
- Begley, Sharon. 1995. Three is not enough. *Newsweek*, 13 February, 67–69.
- Brace, C. L. 1982. The roots of the race concept in American physical anthropology. In *A history of American physical anthropology, 1930–1980*, edited by F. Spenser. New York: Academic Press, 11–29.
- Brandt, Allan M. 1978. Racism and research: The case of the Tuskegee syphilis study. *Hastings Center Reports* 8(6):21–29.
- Buettner-Janusch, J. 1966. Review of *The living races of man*, by Carleton Coon. *American Journal of Physical Anthropology* 25:182–88.
- Buffon, Counte de. 1749. Varieties of the human species. In *Natural history, general and particular*, translated by William Smellie. London: T. Cadell and W. Davies.
- Coon, Carleton S., Stanley Garn, and Joseph Birdsall. 1950. *Races: A study in the problem of race formation in man*. Springfield, Ill.: Charles C. Thomas.
- Coon, Carleton (with Edward E. Hunt). 1965. *The living races of man*. New York: Alfred A. Knopf.
- David, R. J., and J. W. Collins. 1997. Differing birth weight among infants of U.S.-born blacks, African-born blacks, and U.S.-born whites. *New England Journal of Medicine* 337:1209–14.
- Fields, Barbara J. 1990. Slavery, race, and ideology in the United States of America. *New Left Review*, 95–118.
- Fish, Jefferson. 1995. Mixed blood. *Psychology Today* (Nov./Dec.): 61, 76, 80.
- Foster, E. A., M. Jobling, P. Taylor, P. Donnelly, P. de Kniff, R. Mieremet, T. Zerjal, and C. Tyler-Smith. 1998. Jefferson fathered slave's last child. *Nature* 396:27–28.
- Giles, E., and O. Elliot. 1962. Race identification from cranial measurements. *Journal of Forensic Sciences* 7:247–57.

- Gill, George W. 1994. A forensic anthropologist's view of the race concept. Abstract, American Academy of Forensic Sciences, 46th Annual Meetings, pp. 163.
- Goodman, Alan. 1997. Bred in the bone: *The Sciences* (March/April): 20-25.
- . 2000. Why genes don't count (for racial differences in health). *American Journal of Public Health* 90(11):1699-1702.
- Hannaford, I. 1996. *Race: The history of an idea in the West*. Washington, D.C.: Woodrow Wilson Center Press.
- Jacobson, Mathew. 1998. *Whiteness of a different color*. Cambridge: Harvard University Press.
- Kirtles, R., and C. Royal. 1999. Genetic variation and affinities of African Americans: Implications for disease gene mapping. Paper presented at Wenner Gren International Symposium no. 124, 11-19 June, Teresopolis, Brazil.
- Lee, S. M. 1993. Racial classifications in the U.S. census: 1890-1990. *Ethnicity and Racial Studies* 16(1): 75-94.
- Lewontin, R. C. 1972. The apportionment of human diversity. *Evolutionary Biology* 6:381-98.
- Lieberman, Leonard, B. W. Stevenson, and Larry T. Reynolds. 1989. Race and anthropology: A core concept without consensus. *Anthropology and Education Quarterly* 20:67-73.
- Linnaeus, C. 1758. *Systema naturae*. 10th ed. Stockholm: Laurentii Salvii.
- Lippman, A. 1991. Prenatal genetic testing and screening: Constructing needs and reinforcing inequalities. *American Journal of Law and Medicine* 17(1-2):15-50.
- Lovejoy, A. O. 1936. *The great chain of being*. Cambridge: Harvard University Press.
- Mills, C. 1997. *The racial contract*. Ithaca, N.Y.: Cornell University Press.
- Molnar, S. 1992. *Human variation: Races, types, and ethnic groups*. 3d ed. Englewood Cliffs, N.J.: Prentice Hall.
- Montagu, Ashley. 1942. *Man's most dangerous myth: The fallacy of race*. New York: Columbia University Press.
- Rushton, J. Phillipe. 1994. *Race, evolution and behavior*. New Brunswick, N.J.: Transactions.
- Sacks, Karen. 1998. *How Jews became white folks*. New Brunswick, N.J.: Rutgers University Press.
- Smedley, Audrey. 1999. *Race in North America*. 2d ed. Boulder, Colo.: Westview Press.
- Smith, H. M., D. Chiszar, and R. R. Montanucci. 1997. Subspecies and classification. *Herpetological Review* 28:13-16.
- Stepan, N. 1982. *The idea of race in science: Great Britain 1800-1960*. London: Macmillan.
- Templeton, A. R. 1998. Human races: A genetic and evolutionary perspective. *American Anthropologist* 100(3):632-50.
- Todorov, T. 1993. *On human diversity*. Cambridge: Harvard University Press.
- Trotter, Mildred, G. E. Bowman, and R. R. Paterson. 1960. Densities of bones of white and Negro skeletons. *Journal of Bone and Joint Surgery* 42:50-58.

- Trotter, Mildred, and Barbara B. Hixon. 1973. Sequential changes in weight, density, and percent ash weight of human skeletons from an early fetal period through old age. *Anatomical Record* 179:1-18.
- Waitz, Theodor. 1863. *Introduction to anthropology*. London: Longman, Green, Longman and Roberts.
- Wasserman, S. H. S., and U. S. Barzel. 1987. Osteoporosis: The state of the art in 1987: A review. *Seminars in Nuclear Medicine* 4:283-92.
- Wilson, E. O., and W. L. Brown. 1953. The subspecies concept and its taxonomic applications. *Systematic Zoology* 2:97-111.
- Wolpoff, M., and R. Caspari. 1997. *Race and human evolution*. New York: Simon and Schuster.
- Wright, S. 1969. *Evolution and the genetics of populations, volume 2. The theory of gene frequencies*. Chicago: University of Chicago Press.