

Human, Study Thyself

Learning Series: Genes, Race, and Medicine [Part 1]

'Race has been a surrogate for biology. We don't have that luxury anymore'

By Jeff Wheelwright

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Georgia M. Dunston, a black specialist in human genetics, is speaking with a white reporter. We are in her office at Howard University's College of Medicine in Washington, D.C., at the end of the day. The question is whether race, in the scientific sense, is relevant to the health status of African Americans and other minorities. Even if this weren't the topic, race in its unscientific aspects—the thorn in the side of American history—would be with us too.

Dunston, 60, a spirited talker, has long held that race has little to contribute to medicine because the physical characteristics of race are superficial and misleading. For just as long she has contended that black people—those who participate in studies as well as black scientists—should be included in greater numbers in medical research. Her two positions don't fit together neatly—at least they don't to me, but that's probably because the social dimensions of race complicate my view.

After hours of questioning, Dunston finally becomes impatient, as she tries to make me comprehend the tenuous connections between genes, biology, health, and race. “There's no direct correlation between a racial group and anything in the human genome,” she says. “There's no diagnostic test for African Americans or Asian Americans or any other kind.”

She leans forward and presses her forefingers into her cheeks. “But we can see there are biological differences,” she said. “It's self-evident that races exist. No one is saying that features are not biologically captured [by race], but that's not all there is. There's much more biology that isn't being captured. What we see is only a small part of our biology.”

Photomosaic® by Robert Silvers; Original photography by Matthew Spiegelman

To capture the full array of human variation, Georgia Dunston, a geneticist at Howard University in Washington, D.C., leads studies of common diseases such as prostate cancer and hypertension among people of African descent. “There's knowledge inherent in these diseases, and we can unravel it if we can bring the right thinking to it,” she says. This portrait of Dunston is made up of faces spanning the continuum of human physical appearance.



She sits back. “Race is a social grouping based on phenotypic [visible] characteristics. There have been laws based on this social grouping, and I use the term because it’s relevant to our society. We can’t dismiss the factors that have led to tremendous disparities in health—which don’t have to do with genes, by the way. I’m not trying to make race go away but to redefine it using what we have learned about biology through the Human Genome Project. Race has been a surrogate for biology, and we don’t have that luxury anymore.”

Dunston has covered a lot of ground, compressing many issues. I put them into columns to explore. Column one: the dueling definitions of race, social versus biological. Column two: the health gap—blacks’ health in the United States is demonstrably poorer than whites’. Column three: race as the double-edged tool for uncovering medical and genetic differences between populations. (A recent article in *The Journal of the American Medical Association* warns: “Racial labeling, even if done in an effort to better diagnose and treat patients, can reinforce racial stereotyping.”) Column four: Georgia Mae Dunston. Besides being the first black female Ph.D. in her field and the founder of the National Human Genome Center at Howard, a historically black university, where was she coming from? Where was she going?

RACE AND RACISM

Biologists have clamored over race from the beginning of modern science. In the natural world the concept has seemed straightforward: Populations of the same species that didn’t overlap in breeding territory could safely be called races. The separation usually caused small differences in appearance between populations. Applying the same framework to humanity, early biologists decided that because black Africans and white Europeans lived on separate continents and didn’t normally interbreed, the two represented races.

Linnaeus, the 18th-century taxonomist, declared four human races (Asians and Native Americans were the other two). This was the so-called classical system of race, and its divisions were roughly geographic. Johann Blumenbach, one of the first anthropologists, endorsed the Linnaean categories but didn’t believe that human beings belonged to discrete groups. “One variety of mankind does so sensibly pass into the other,” Blumenbach wrote in 1795, “that you cannot mark out the limits between them.” Still, he put Caucasians at the top of the heap by claiming that other races had descended from them and migrated elsewhere. In the Blumenbach scheme Africans were Caucasians twice removed—the opposite of what modern genetic studies show.

For 200 years some scientists have made findings of human continuity and kinship while other scientists have measured and ranked people according to types. As a result, governments wishing to base their social policies on race could choose from different interpretations. It almost goes without saying that the social applications of race emphasized people’s distinctions rather than similarities, and that the discrimination has been cruel. The apartheid system in South Africa, the eugenics policies of Nazi Germany, and our own history of slavery and segregation come to mind.

Georgia Dunston, who was born in the South and attended segregated schools, grew up under an exclusionary social definition of race. As a scientist she joined the school of doubt about race in order to rectify the ramifications of race. Francis Collins, who directs the National Human Genome Research Institute in Bethesda, Maryland, an arm of the National Institutes of Health, praises Dunston as a “passionate-researcher-advocate. . . . Georgia has a vision for how the coming genomic revolution in medicine and public health can be used to help the African American people.” Vence Bonham, Collins’s colleague at the institute, says: “She brings her whole self to the table. She has a ‘lived experience’ as a

black woman in America who also has a knowledge of genetics.” Here were the two strands of her life, science and social identity, that would have to be untangled.

Medical genetics, the straighter thread, dates to the early 20th century. In 1901 laboratory studies revealed that human beings carry in their veins one of four types of blood, which are distinguished today as types A, B, AB, and O. Karl Landsteiner, who made the discovery, kept count of the immunologic reactions between random pairings of blood. “There were antibody reactions when blood from different individuals was combined,” said Dunston, whose specialty is immunogenetics. “There were four patterns of clumping.” When two samples were of the same type, no clumping occurred. Such immunologic matches make blood transfusions possible.

The so-called ABO blood group is inherited. It is a single trait that assumes multiple forms. Not until 1990 was the genetic site pinpointed, the place in the DNA where the ABO gene resides in its various spellings. Well before then geneticists had figured out that the variants didn’t match race. You cannot look at a person’s skin and know what type of blood he or she has. Still, there are patterns and probabilities that can guide your guess because the distribution of the ABO variants in the world’s population changes according to people’s continent of origin. The same holds true for variants of other genes.

The formal term for variant is allele. The major lesson in genetics over the last half of the 20th century is how the genes and large chunks of noncoding DNA between the genes vary from person to person. The interpersonal variation is much more extensive than group variation. Our bodies work generally the same way, but at the level of DNA we are wonderfully allelic. The Human Genome Project, when it unfurled the biochemical letters of “the” human DNA sequence in 2000, sent the wrong message. There is not one human sequence but billions, each very similar, each slightly different. As Dunston put it, “We are all the same, and we are all unique.”

UNETHICAL SCIENCE

Segregation wasn’t a bad thing for her education, Dunston said unexpectedly. Even after her home school district in Norfolk, Virginia, was opened up by court order, Dunston remained at the black school. She studied under dedicated black teachers, whose encouragement made up for the thinness of resources.

Her parents, a cook and a dishwasher, never finished high school. Georgia Mae blazed through at the top of her class and entered Norfolk State College on a scholarship when she was only 16. In 1965 she went to Tuskegee University in Alabama, enrolling in the master’s program in biology. She saw white teachers there for the first time.

Tuskegee’s fine record of educating African Americans may be overshadowed by a sorry biomedical episode, the Tuskegee Syphilis Study, which was still in progress during Dunston’s graduate education. Although she didn’t pay much attention at the time, the biological premise of the study was just the kind that she would battle later. In the 1930s researchers for the U.S. Public Health Service suspected that black people and white people might respond differently to syphilis infection.

Begun in 1932, the Tuskegee Syphilis Study documented how the disease progressed among infected black men in Macon County, Alabama. Treatment was withheld even after penicillin—the first effective medication for the infection—became available in 1943. The study ended in 1972 after a newspaper revealed the scandal. The NAACP sued the government and reached a settlement that paid the subjects more than \$9 million and included medical care for them and their families.

One way to find out was not to treat those who tested positive for the venereal disease and simply monitor their cases as they developed.

The project continued even after an effective treatment for syphilis—penicillin—became available. After the scandal of the Tuskegee study broke in 1972, U.S. health agencies overhauled the ground rules for human research. President Clinton apologized to black Americans for the study in 1997. To this day, Dunston and her Howard University colleagues find themselves addressing Tuskegee-inspired fears when they recruit Washington residents for genetic studies.

Dunston earned her Ph.D. in human genetics at the University of Michigan in 1972 and joined the Howard faculty as an assistant professor of microbiology. She also worked as a consultant in Washington for the Job Corps Sickle Cell Anemia Program. There she found a real-life controversy over purportedly black genes. Prompted by health officials with a dim sense of genetics, the testing of African Americans for sickle-cell disease was a social program that, like the Tuskegee study, backfired on the group it was meant to benefit.

Sickle-cell anemia, a dangerous deformity of hemoglobin in red blood cells, is not limited to blacks any more than cystic fibrosis is to whites. However, a skew in the prevalence of the sickle-cell gene puts African Americans at much higher risk of the disorder than other Americans—about one in 10 blacks are carriers. The numbers in those days were vague, but the issue was the same as the one debated today: Was a person's racial background a sufficient substitute for his or her genetic vulnerability to a disease? Policymakers decided it was. Between 1970 and 1972 a dozen states and the District of Columbia passed laws mandating sickle-cell screening for African Americans.

Today the laws would be condemned as racial profiling. The stigma was made worse by a misunderstanding of the inheritance of the condition. Contrary to reports of premature deaths, carriers of the sickle-cell gene were in almost all cases healthy; because genes come in pairs, carriers had one normal copy of the gene, making normal hemoglobin. The sick, who were far fewer, inherited two bad copies of the gene from their parents.

Patients having the disease didn't need to be screened for it, for most already knew. The carriers needed information about their status only if they were planning to have children, who would have a one-in-four risk of getting the disease. But the blood test that was used, the Sickledex test, could not tell people with the sickle-cell trait, called genetic heterozygotes, apart from those with sickle-cell disease, the homozygotes. Therefore black children who had positive results were misdiagnosed and their families unnecessarily frightened. Workers who tested positive lost their jobs in some instances because employers didn't want to be held responsible for stressing their health.

When Dunston became involved, the federal government had taken over the program, and the coercive laws had been repealed. Proper genetic counseling was supposed to accompany voluntary testing. At the Job Corps center she gave talks to young black adults about the transmission of sickle cell, trying to correct misinformation and mistrust in a community with a long memory.

“A 10 percent frequency [of the trait] doesn't define a group,” Dunston said. “People assumed that every black had it, as if homozygotes were out there in numbers. It did an injustice to the group because employers now had the power—the “science”—if they needed a reason not to hire someone.”

The great irony of the sickle-cell allele, said Robert Murray, head of Howard's human genetics department, is that in its singular form “it is a beneficial disease gene, the only one there is.” The allele evolved in Africa, southern Asia, and around the Mediterranean (where many Caucasians carry it)

because these zones are plagued with malaria. People who are sickle-cell carriers make hemoglobin that changes shape when the red blood cell is invaded by the malaria parasite, rendering the cell less hospitable to the pathogen. Murray, who advised the federal government on the issue in the early 1970s, recalls pointing out: “Forty percent of Nigeria has this trait. Why aren’t they dying?”

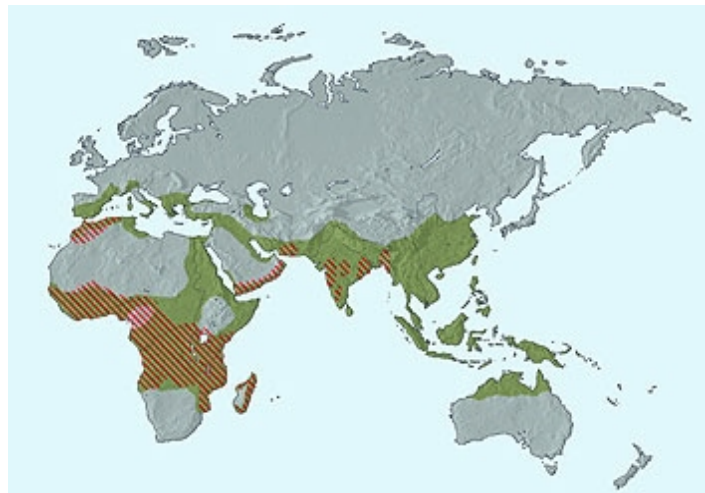
Dunston elaborated. “The gene conferred a biological advantage in an area where malaria was wiping out a lot of folk. We can look at the history of other variants in the same way. It’s all related to population history. The gene tells a history of a population much better than the phenotype [the gene’s outward expression] does.

“Sickle-cell testing was an example of tying genetics to disease in a way that was bad. But now we have an opportunity of getting a better rap for genetics, if you will. With the tools we have now, let’s define the gene not as the culprit or the pathological entity but as the unit interacting with the environment and as part of the science of variation. Variation is a light we can use to understand biology.”

ORIGINS OF THE SICKLE-CELL TRAIT

The sickle-cell trait has spread far and wide, outside the Americas. Regions where the trait is more commonly found (shown in orange stripes) overlap areas where malaria is prevalent (shown in green). Graphic by Matt Zang

One of the most clear-cut examples of environmental influence on the human genome is the sickle-cell trait. Although the blood disorder sickle-cell anemia was first described for medical science early in the 20th century, it was not until 1956 that researchers pinpointed its cause: a single change in a nucleotide in the gene that codes for the oxygen-carrying molecule hemoglobin.



In 1954 molecular biologist Anthony C. Allison had surmised that alterations in the hemoglobin molecule protected against death from malaria. The defense works like this: Having a single copy of the allele confers protection from severe malarial infection by somehow rendering the red blood cell less hospitable to the malaria parasite, *Plasmodium falciparum*. The downside is that someone with two copies of the allele suffers from sickle-cell anemia, an incurable blood disorder. When two copies of the altered hemoglobin gene are present, they cause the shape of the hemoglobin to change so much that the “sickled” blood cells don’t flow freely in the blood vessels, causing excruciating pain. A child of parents who each possess one copy of the sickle-cell trait has a one-in-four chance of inheriting both sickle-cell alleles—and thus getting sickle-cell anemia.

Of the five variants of the sickle-cell trait, four appear to have originated in Africa and spread through trade routes or the transport of enslaved Africans. The fifth, which is predominant in the Middle East and India, appears to have originated in Asia. The prevalence of the single copy of the gene varies, but in some malaria-prone pockets of India, Africa, and Saudi Arabia, up to 35 percent to 45 percent of the population have the trait. Malaria was confined to the Old World until about 500 years ago, when the malaria parasite gained a foothold in the New World with the arrival of Europeans and Africans. The disease was common in the United States prior to the introduction of DDT and the clearing of swamps.

Other variants of the hemoglobin gene also confer some protection against malaria. One of the most common causes thalassemia, a type of anemia that occurs more often in parts of Africa, the Mediterranean, the Middle East, and Southeast Asia than it does elsewhere.

ANCESTRY AND AFRICA

The National Institutes of Health became the major funder of Dunston's laboratories at Howard. In the 1980s she investigated why kidney transplants were being rejected at higher rates by black patients. The problem was that the tests for making immunologic matches between organ donors and recipients were based on "the majority population." That is, the tissue samples for the antigen panels had come from European Americans and only scratched the surface of human antigenic variation. Antigens are flags on the surface of cells, and they trigger the immune response when the flags are foreign.

"These are genetically inherited characteristics," Dunston said. "People thought that there were black tissue antigens and white tissue antigens—and I knew that wasn't so." Now that antigens are better understood, cross-racial transplants often make better matches than those from within a racial group because, as Dunston is quick to point out, it's the individual response that counts, not the group's.

In the 1990s, after two genes for familial breast cancer were identified, Dunston secured a NIH fellowship to study the alleles of breast cancer genes in the African American population. Making an ally of Francis Collins, she laid the groundwork for the National Human Genome Center at Howard University, which was launched in 2001. Its plan, she said, is to search out genes for disorders other than breast cancer, such as prostate cancer, high blood pressure, and diabetes, all of which affect blacks disproportionately.

"My role is not solving the puzzles and problems," she said to me in her office. "I'm more of a conceptualizer. My strength is recognizing the deficits in our capacity to answer questions."

I must have implied that she traded on her race with sponsors, because she said acidly: "We're supposed to pride ourselves when we get government support? We don't need to be included just because we've been excluded. It's better for the science design of the project. When I've said that blacks have to be included, it's not from the point of view of the social group. I've also said that whites as a group can't serve as the only reference for whites."

She shifted to the big picture. "Africa may be instructive to Europeans, but it may not be vice versa. Eleven hours of the total 12 hours on the evolutionary clock was spent in Africa. That's where most human variation occurred. As it migrated out, the base was still there, the base of the diverse human group. The breadth of variation in African people is a tool to look at variation in all of humanity.

"Yes, there is a set of diseases that occur more frequently in blacks, but they're complex diseases, involving multiple environmental factors and multiple genes too. The causes are tied to biology, not to race. There's knowledge inherent in these diseases, and we can unravel it if we can bring the right thinking to it."

In 2003 Howard announced it would establish a repository of genetic samples from 25,000 persons of African descent, an effort lately expanded to include people from West Africa and the Caribbean as well as people from the Washington, D.C., area. But Dunston and her colleagues were unhappy with the press coverage of the biobank plan, which she said focused on "collecting DNA on blacks. That's only a small part. Other factors will come out of the research."

She and her colleagues, it seemed, walked a fine line. They pursued the genetic mechanisms behind disease while maintaining that environmental and social factors—unhealthy diets, inactivity, poverty, stress, and other tolls of urban life—were just as important as genes in explaining the prevalence of disease. They denied race biologically but at the same time represented their race in the political

competition for research dollars, and they objected when blacks were left out of studies. They called attention to their own work at Howard, then bristled at how the work was characterized by others. “We are gun-shy about other people’s definitions of what we are,” Dunston said.

Yet she has negotiated this minefield for 20 years without an explosion. “A couple of my colleagues say, ‘You’re the Martin Luther King of the group, not the Malcolm X.’ ” She has repeatedly arranged collaborations between Howard and the richer institutions in the neighborhood, such as Georgetown University, Johns Hopkins University, and NIH.

Howard, which has trained black doctors proudly for more than a century, rivals big institutions in quality, but its budget doesn’t compare, and its staff is stretched thin. There’s no padding in the place, no room for error in dealing with the medical establishment. Nor was there eagerness to deal with a medical reporter nosing around the campus and asking about their activities. After a time doors at Howard that had been open to me were quietly closed—except for one. All the more power to Georgia Dunston, I thought.

The National Medical Association—the black counterpart to the American Medical Association—held its annual convention last August in San Diego. Over four days at the cavernous San Diego Convention Center, 6,000 physicians attended seminars and lectures, including a plenary session on Howard’s National Human Genome Center. Collins made a presentation, followed by Charles Rotimi, who had just taken over as the director of the center, and two others from Howard. Dunston was in the audience, sitting by herself. “I can be a cheerleader too,” she said.

The attendance was sparse and got worse because the session started late and ran into the lunch hour. The implied message, as doctors drifted out, seemed to be that genomics was great for what it might deliver down the road, but they had their hands full right now with patient needs. They wanted help right now, for instance, to reduce deaths from prostate cancer. Its mortality rate in African American men is the highest in the world—at least twice that for white males with prostate cancer. Blacks’ mortality from hypertension and strokes is 80 percent higher. Although these diseases surely have genetic components, it is difficult to believe that genes could cause such a split in outcomes between the two groups.

In a 2003 report titled “Unequal Treatment: Confronting Racial and Ethnic Barriers in Healthcare,” the National Academies’ Institute of Medicine detailed the shortcomings in the delivery of health services to minorities. The shortcomings were attributed to lack of education, racial discrimination, and poverty. Citing the report, health analysts wrote in *The Journal of the American Association*, “health disparities and genetics may have little to do with each other, short of capturing public attention simultaneously.” Even Collins conceded in his talk that “most health disparities don’t have anything to do with genetics, but sometimes genes have a role, we think.”

Drug companies were much in evidence at the convention, and if there is one area of genomics that has attracted a critical mass of believers, it’s pharmacogenomics. In recent years reports have mounted that many drugs work better in white patients than in black patients, and vice versa. The drug companies, noting that as many as half of all drugs don’t work as intended, think that genetic variation is responsible. Scientists have illuminated alleles that may affect drug reactions. Not only are the alleles skewed between black and white study samples but their frequencies also line up with the groups’ overall responses.

Black physicians as well as white recommend patients for drug trials, and they act on those results when writing prescriptions. Clearly the practitioners in San Diego accepted race as a useful clinical criterion, regardless of geneticists’ objection that pharmaceutical scientists leave it to their subjects to say if they

are black or white, which is hardly a scientific method. Studies of genetic markers that are distinctive to ancestral groups show that typical African Americans have 20 percent European blood, although that figure can go as high as 80 percent in some people who call themselves black.

A firm called Nitromed has a new drug on the horizon, a blood-pressure medication called BiDil. A 1986 study suggested that it helps blacks more than whites. At the convention Nitromed officials discussed the results of a new BiDil trial, entailing more than 1,000 African American patients with heart failure. In heart failure the diseased muscle can't pump blood effectively against the resistance of the blood vessels.

BiDil, which dilates the vessels and eases the pressure, was found by study reviewers to have been so beneficial that the study was cut off early. The people who took the drug in addition to their regular blood-pressure medicines had fewer deaths than the control group not using BiDil. Nitromed was pushing for Food and Drug Administration approval to market the drug. Next, the company's chief scientist said, Nitromed would search for biochemical and genetic markers that might predict the sort of patient who best responded to the drug.

Charles Curry, the recently retired head of cardiology at Howard University Hospital, was concerned about racial profiling, but he supplied patients for the BiDil trial and was listed as a coinvestigator. "I objected to the hypothesis that blacks have a 'sick' blood vessel—that their veins and vessels are different," Curry said. "I still don't believe it. Then I decided that it [the drug trial] can't hurt. These were very sick patients, and they improved their life expectancy. Sometimes I sound like I'm flip-flopping. But I bet the data will show that BiDil helped in lowering hypertension." The connection between hypertension and heart disease, he noted, is somewhat different among blacks and whites.

Dunston rose above such circular reasoning. Without knowing all the details, she had plenty to say about the Nitromed project. The only thing she liked was the company's promise to look for genetic markers, a step she called "a minimum."

"You have to characterize the individuals in the group," she repeated. "What about those who didn't respond? The group just tells us where to drop the net, but we can't stop there and target the group without the mechanism known. Genetics can pass through the population on the way to personalized medicine."

Not being a physician was an advantage to Dunston, she said. It helped her to see the research more clearly. Race in biology was to her a transitional structure, a mean and sorry thing that was destined to wither away, with a better day to come for all. "It's an exciting time for the genetics of African American people," she said fervently. "Even with this controversy—I'm hoping it will bring openness. I know we'll get beyond this point." That was it, I realized. Her faith.

Shiloh Baptist Church, the Reverend Wallace Charles Smith presiding, is a cultural landmark in Washington, D.C. On a Sunday in June, which happened to be Music Appreciation Day, Shiloh's 150-member choir rocked the house with song. Reverend Smith's sermon drew a warmly antiphonal response, and when he had finished speaking, he introduced the day's visitors. Shiloh's odd couple, a Baptist and an Episcopalian sharing a hymnal, took a small bow.

A scientist who is savvy won't mention God unless asked. Dunston knew the two topics don't mix well; I was the one who broached the church. But in the parish hall after the service, where she mingled and did some light recruiting, she cited scripture easily and often.

“My people perish for lack of knowledge,” she said at the table, quoting the Old Testament (Hosea 4:6). Her listeners, middle-aged men and women who were having a late breakfast, nodded. “Anyone who studies nature closely will have problems believing this is a random process,” Dunston continued. “Science is a tool for revealing the work of God.” More agreement.

When the subject turned to the genetic testing conducted at Howard and elsewhere, Stan Williams, a deacon of the church, said, “People will want to know: How will this benefit me personally? What are the dangers to me?” Dunston, her shoulders hunched, paid close attention to him. “If they feel it’s safe for them, they might do it,” Williams said. “Historically we have a built-in fear of being tested.”

“I sometimes say that genetics is the underpinning of all biology,” Dunston replied. “Let’s use it to refine the medical diagnosis.”

“I’m interested in the diseases I may be susceptible to,” offered Edwin Washington, “but I’m not a big DNA fan. I feel a lot of leeriness about it.”

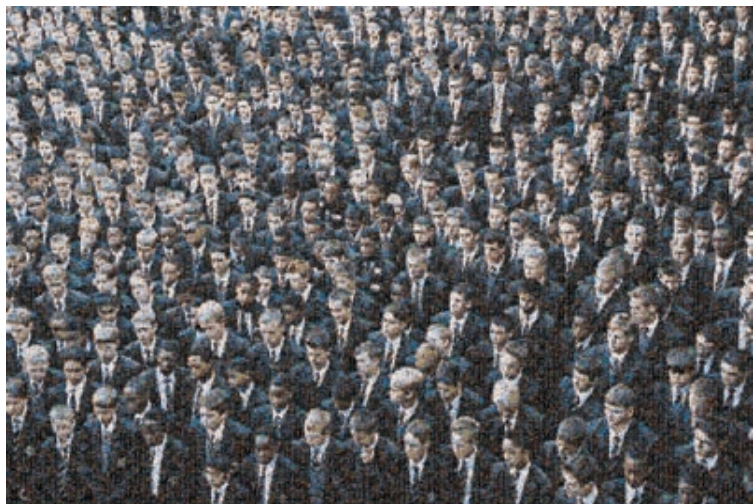
A woman named Constance Tate said sagely: “This project may not be at that stage yet. It may be four or five years yet before we have to worry about the risks and the benefits. But that’s why we need to understand the information now.” Dunston beamed and clutched Tate’s hand.

When we left Shiloh, Dunston said: “Do you want to know why I went into genetics? When I was a child, I had questions about God loving us all the same, the way we were taught. I saw people being treated differently. ‘Why are we different?’ I asked my parents. They couldn’t answer. They said I had to ask God. So my question for God was, ‘Why did you make us different when it leads to bad things for people?’

“Now, with genetics, I know. We are such exquisite beings. We have the gift of the genome. The genome is a gift.” She clamped both my arms with her big hands. “That’s why you and I are different. That’s why there is human variation. I get to know God through you!”

Photomosaic® by Robert Silvers; Original photograph from National Geographic/Getty Images.

The physical appearance of these South African schoolboys reflects the ancestry of peoples from three different continents: Africa, Europe, and Asia. But the variation that interests Dunston resides at a deeper genetic level. “Variation is a tool for seeing. But not the medical view, which sees variation as an aberration,” she says. “For me, science is the democratization of knowledge. We want to go from the patterns to the universal.”



ABOUT THE SERIES

This is the first of three articles exploring the relationship between race, genes, and medicine in three far-flung populations. Although race is a socially powerful concept, most geneticists think it has no foundation in biology. Modern DNA studies show that the world's population is too homogeneous to divide into races.

But while dismantling the barriers of race, scientists have uncovered patterns of genetic mutation and adaptation in human populations. As archaic bands of *Homo sapiens* left Africa and spread over the world's continents, their DNA evolved. Geography has left faint marks on everyone's DNA. Although the differences are small, they show up in the diseases that different groups get and how these groups respond to drugs.

To measure these differences is not to resurrect race by another name but to emphasize the role of history in shaping medical legacies. Researchers seeking genetic explanations for health have to explore the events written in the record of DNA. Thus in this first article, about African Americans, geneticist Georgia Dunston points out that Africa contains the richest DNA diversity because it is the site of humanity's oldest genes. Africans and their recent descendants in America may harbor clues to fighting diseases that other populations don't possess.

The second and third articles, to be published in our April and May issues, follow gene hunters into more isolated and homogeneous gatherings of people—the Finns at the top of the European continent and the Native Americans in Arizona and New Mexico.

In the future, doctors will examine the genetic portraits of individuals, not populations. The path to understanding how individuals fit into genetically similar populations would run straighter if not for the old stigmas of race. Two of the three groups in *Discover's* series, being minorities, are wary of genetic studies that may stereotype them further. In the past, science was not an innocent bystander when people were separated into races.