Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 299

CHAPTER 12

GENETIC PERDITION

The Rise of Molecular Bias

In the age of the technological fix, this country is heading for genetic and behavioral control of society. Who will exercise the control? Who will make the decisions about which genes are defective and which behavior abnormal? Who will make the decisions about the genetic worth of prospective human beings?

—JONATHAN BECKWITH, 1974

"When I went to prison, the concern and worry literally broke my mother's heart. She suffered a series of heart attacks and strokes and died in 1997. She knew I was innocent, because I had been at home with my parents when the crime occurred. And over the years, things just wore her down. When you are in prison, if you are close to your family, your whole family is in prison."

The burden of guilt is common coin in prison, but Calvin Johnson knows the crushing agony of innocence. The twenty-five-year-old Atlanta resident had a bright future, a close-knit family, many friends, and a wedding date when he was convicted of raping a white woman in 1983. He had never seen his "victim" before, but he was convicted, although pubic hairs recovered from her body did not match his. They did come from an African American man, and that, apparently, was enough. "I still had faith in the justice system. I believed it would be just a matter of time before officials realized that they had made a mistake. I was really kind of naïve: I didn't believe that I would be sentenced or convicted of the crimes."

Although the woman identified photographs of someone else as her assailant and although he did not match key elements of her description (the actual rapist had only a mustache, and Johnson wore a full beard), Johnson was convicted by an all-white jury. For seventeen years, Johnson fought to survive in "the hardest work camp in the state of Georgia. I

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 300

worked in snake-infested swamp waters up to my knees." He also had to stave off assailants. "When you're in prison for a sex offense, if you're not physically strong, the guys around you, they'll try to pick at you. So I lifted weights and became a pretty good size. People left me alone." Johnson lost his youth, his fiancée, and his naïveté, but, he says, "I always believed that God would save me." Faith in God sustained his spirit, and in 1986, Johnson finally found physical deliverance in DNA, which proved him innocent. He was forty-two years old.

Nearly all human cells contain genes, which, in turn, contain deoxyribonucleic acid, or DNA, the molecule that encodes life itself. DNA's genetic code is composed of building blocks called nucleotides, and this code dictates and directs the development of a fertilized egg through processes of protein manufacture so complex that they remain incompletely understood. DNA is passed from parents to children, and it determines or influences many traits, from your eye color to many disease propensities. There is DNA in nearly all your cells, but there are several types of DNA, and less than 1 percent codes for differing traits such as eye color, height, or disease susceptibility. Unless you are an identical twin or the product of another such multiple birth, your DNA is unique. No one else on the planet has your exact genetic code, although humans share a great many genetic similarities.

Today, "DNA fingerprinting" technology enables scientists to identify distinctive genetic patterns.[1] In Johnson's case, the DNA samples from his body ultimately proved that the pubic hairs and other biological evidence left behind by the rapist were not his. At least three types of DNA fingerprinting are in use, but despite the terminology, none is as accurate an identification method as matching a fingerprint. The most popular method at the time of Johnson's conviction, restriction fragment length polymorphism, or RFLP, analysis, compares the DNA of two or more individuals, which varies by only 0.1 percent. That's one difference in a thousand, useful for establishing paternity—or guilt. A newer form of DNA comparison utilizing single nucleotide polymorphisms (SNP) has rapidly outstripped RFLP.

Anyone who doubts that genetic technology can be an important blessing for African Americans should consider its pivotal role in freeing black men such as Calvin Johnson. Johnson was freed by the Innocence Project, the brainchild of O.J. Dream Team members Barry Scheck and

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 301

Peter Neufeld, lawyers at the Cardozo School of Law in New York. So far, DNA evidence has helped them and the fifteen to twenty similar projects they have inspired to exonerate more than 328 inmates,[2] including Kirk Bloodsworth and Earl Washington, Jr., who were sentenced to die in Maryland and Virginia, respectively.[3] "These are mostly African American men convicted of raping white women," says Neufeld. "Only 10 percent of reported sex assaults are allegations of white women attacked by black men. Yet most—54 percent—of all convictions proven to be unjust involve African American men wrongfully convicted of assaulting white women. This is a crime that seems associated with many wrong convictions."

So many men have been freed by DNA testing that laws ensuring prisoners' rights to DNA appeals have been passed in some states, including California, New York, and Illinois. Illinois declared a moratorium on capital punishment after an embarrassing string of investigations uncovered many innocent prisoners in its penal institutions.

However, deployment of DNA technology is no panacea. Relatively few inmates can afford the requisite five thousand dollars, and the backlash triggered by the Illinois embarrassment was swift. Some cities, such as Lansing, Michigan, passed laws restricting the use of DNA evidence in inmate appeals. Then again, some criminals leave no testable materials behind, and according to Barry Scheck, even when biological evidence exists, 70 percent of the time it is allowed to deteriorate, is lost, or is discarded during the decades an innocent person languishes in jail.

Human error sometimes sabotages genetic wisdom, as when courts ignore compelling DNA evidence.[4] Scientists and technicians in genetic laboratories have made errors and have even falsified DNA test results. For example, Chicago Laboratory worker Pamela Fish lied or made errors that bolstered at least one erroneous conviction, according to forensic experts who reviewed her testimony before the release of inmate John Willis.[5]

A study by University of Michigan law professor Samuel R. Gross determined that tens of thousands of innocent people are trapped in jail: "If we reviewed [all] prison sentences with the same level of care that we devote to death sentences, there would have been more than 28,500 non-death-row exonerations in the past 15 years rather than the 255 that have in fact occurred."[6]

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 302

Even for freed men such as Johnson, justice remains elusive: How do you compensate a man for consigning him to spend his youth in hell? For the loss of his family, friends, income, and good name? States such as California offer a nonnegotiable settlement of one hundred dollars for each day of unjust imprisonment. But two-thirds of those freed by DNA evidence get nothing.[7] And money means nothing to some, such as Frank Lee Smith, a Fort Lauderdale man exonerated by DNA evidence nearly fifteen years after he was sent to death row and eleven months after he died there of cancer.

Clearly, DNA testing is no substitute for justice. In fact, according to experts such as Neufeld, "the real significance is not that DNA got them out, but that DNA provides a window into the criminal justice system to see what went wrong with the system to let so many innocent people be convicted."

But DNA evidence has powerful uses beyond liberating the innocent.

Shades of Gattaca

The film *Gattaca* held a not-too-distant mirror up to a genetic dystopia in which human decisions—and discretion—are removed from all-encompassing judgments about men's worth. In this film, only one's DNA, recognized and assessed by machines, determines one's fate, leaving character, personality, drive, and intent all sublimated to the tyranny of the gene. The biometric dystopia of *Gattaca* doesn't exist yet, and perhaps it never will. But developments over the past few years evoke an unmistakable glimmer of recognition. The FBI, Secret Service, IRS, Social Security Administration, Census Bureau, and Department of Veterans' Affairs all maintain extensive collections of genetic data. Since May 1998, sex offenders have been required to surrender DNA samples to federal databases, and today every state maintains its own DNA database that contains the DNA profiles of felons—and of others, including people merely suspected of crimes or even of innocent people rounded up in DNA sweeps. The samples of 450,000 convicts are stored with identifiers, such as the person's name, description, criminal record, Social Security number, and image. The government has also sponsored the creation of national databases, such as the FBI's Combined DNA Index System

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 303

(CODIS), which stores DNA samples, most without identifying information. CODIS went online in 1998 with samples from 8,000 convicted child molesters, and by 2001, it contained the profiles of 1.5 million felons. In 2002, the U.S. Attorney General ordered the FBI to expand CODIS to 50 million profiles, and by 2004, CODIS stored 2.6 million samples containing the DNA of people convicted of almost any crime. In October 2005, the Senate Judiciary Committee approved a law forcing anyone who is merely *detained* by federal authorities to provide DNA, and in 2006 the database contained more than 3 million samples. The FBI predicts that CODIS will accommodate 50 million samples "in the near future".[8]

Some scientists warn that the very DNA evidence and technology that has freed hundreds of African American men like Johnson may soon be wielded by police to criminalize and convict black and Hispanic men. From California to London, DNA data banking has allowed the collection of genetic evidence for convicted felons on the premise that those who have been convicted have sacrificed some of their rights to privacy. But Troy Duster, a professor of sociology at Berkeley and author of *Backdoor to Eugenics,* warned in 2001, "The same technology that will exculpate people today is also being used to put people who have merely been *stopped* by the police into genetic databases." He is correct. In 2000, Miami police seeking a violent criminal described vaguely as "black or Hispanic" stopped 2,300 black and Hispanic men on the street and quickly took a buccal swab from each, swabbing the interior of each man's cheek. The police now had samples of their DNA, accompanied by identifying information—suspect profiles—and each man was free to go, for the time being. The samples were tested against DNA left by the rapist at the scene, but none of these men's DNA matched that of the putative assailant. Therefore, all these men have demonstrated their innocence, but police have stored their genetic data in a database to be tapped when they next seek a perp.

This database of innocent black and Hispanic men constitutes a collective presumption of guilt. When weighing the ethical and scientific unacceptability of this tactic, it is important to realize that (1) the term *DNA fingerprinting* is a misnomer: the genetic profile is not as specific as a fingerprint and cannot provide a unique identifier; (2) the description of a "black" or a "Hispanic" suspect is so vague that it yields a racial drag-

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 304

net, not a description of a suspect; and (3) some "rare" differences that allow one to differentiate individuals based upon a genetic profile become less rare when one looks only within ethnic or kinship groups.

DNA profiling has been questionably imposed upon white men, too, but with important differences. For example, the ACLU of Massachusetts denounced DNA testing as "a serious intrusion on personal privacy" when police in Truro, Massachusetts, used it in investigating the 2002 killing of white fashion writer Christa Worthington. The ACLU also cited the technology's failures in sites such as Baton Rouge and Virginia when DNA samples were coerced from up to eight hundred area men, most of whom were white (in contrast to the thousands taken from black and Hispanic men). The ACLU also argued that the seven thousand forensic DNA samples tested in sweeps have resulted in only one arrest, making DNA sweeps a very expensive and inefficient way of targeting suspects.[9] This is partly because guilty suspects typically refuse to give a sample, even under considerable pressure; it is the innocent who allow themselves to be cajoled or bullied into a buccal swab.

A DNA sweep targeting all Caucasian men, in which police coerce men into supplying DNA to eliminate themselves as suspects, then store it for use the next time they seek a criminal, would be as ethically repugnant as a similar sweep of black men. However, in Truro the donors were not exclusively white and were not targeted on the basis of skin color, so racial bias was not a factor: Truro police asked "all local men" over eighteen years old to provide samples and recorded their various races. What's more, the police agreed to destroy the Truro samples after collection, unlike sites in Miami and Washington, D.C., where the police sought DNA only from men of color.[10] The Truro sweep was still a privacy violation: Many white men felt pressured to give samples and complained that the demand for a DNA sample violated Fourth Amendment protections against unreasonable search and seizure.

Moreover, a black man was arrested for Worthington's murder in April 2005, under troubling circumstances. According to the *Boston Herald,* this suspect, who had an extensive criminal history of violent crimes against women, had given the police permission to take his DNA in April 2002, but police declined to do so until March 2004. During the three years it took them to take, analyze, and act on his DNA analysis, the DNA dragnet of Truro's eight hundred adult men was completed. Some now

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 305

complain that their privacy was invaded for no reason by DNA testing because police failed to investigate an obviously promising suspect or even to analyze his DNA sample.

California, too, is forcibly taking DNA samples from people presumed innocent—people who have been arrested but not tried and convicted. Defenders of the practice often say that taking and storing such samples is no more intrusive than the common practice of taking a suspect's fingerprints. It is true that fingerprints are taken of arrested persons without too much protest that the innocent are being stigmatized, but again, DNA markers are not fingerprints: They are less specific and far more invasive. In practice, a fingerprint is not a forensically infallible means of identification, but it verifies a person's identity with enough accuracy to satisfy the legal system. However, one's DNA contains intimate information not only about one's identity but also about one's health, including one's future risks of becoming prematurely senile, or developing Huntington's disease or a hard-to-cure cancer. Besides harboring the markers for four thousand disease risks, DNA also contains information about the health and identity of one's forebears and descendants. With a sample of your DNA, a person can predict certain disease and disorder probabilities for you *and* for your children. George Annas, a law professor and bioethicist at Boston University, has referred to one's DNA profile as a "future coded diary," and with the completion of the Human Genome Project, the code has essentially been broken. Therefore, taking the fingerprints of an arrestee and taking a sample of his DNA are not comparable acts; the latter is far more intrusive and revealing—but far less likely to yield a uniquely definitive identification.

In the United States, laws prevent the federal government from retaining DNA samples of the innocent, but the states are doing just this. In 1994, police took samples from 160 black men in Ann Arbor, Michigan, many of whom complained that they had been coerced by police officers who ignored their alibis and threatened to prosecute them if they refused to submit. San Diego police similarly pressured eight hundred black men in order to catch a serial killer described only as "dark-skinned." Black Ann Arbor residents complained that the police tactics "bordered on harassment and abuse," but the men who were approached in Truro often cited subtler peer pressure and vague fears that police would scrutinize them more heavily if they refused to give a sample.

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 306

However, Ann Arbor law-enforcement officials denied that their investigation was discriminatory; they insisted that police were simply targeting individuals who met the description of the perpetrator. The Ann Arbor killer—along with several other men—refused to provide police with a DNA sample and was identified only after he was arrested for an unrelated crime.[11]

In mid-April 2001, Syracuse University's Lubin Center hosted a program on forensic genetic technologies, moderated by television journalist Catherine Crier and with a panel of experts that included NYU sociology professor Troy Duster and Howard Safir, the police commissioner of New York City under Mayor Rudolph Giuliani. Safir's new career as a proponent of high-technology security includes the promulgation of his view that police should soon be allowed to use brave new genetic technologies to stop people on the street, take a buccal swab with a portable device, run the database off a satellite, and use their portable computers to see whether they have a "hit."

Such on-the-spot DNA testing is not yet reality, but several biotechnology firms are endeavoring to perfect portable solutions that can allow cops to stop a person, obtain a quick DNA sample, and check it against a database in minutes. One such firm, located in San Diego, is called Nanogen. It utilizes single nucleotide polymerases (SNPs), small DNA fragments that are sites of genetic difference distinctive enough to identify a suspect. Nanogen can put SNPs on a microchip the size of a stamp, technology that scientists have taken to calling "SNPs on chips."[12] Or by analyzing and comparing small areas of DNA called short tandem repeats, or STRs, a police officer armed with DNA from spittle, a buccal swab, or a semen sample can very speedily check thirteen STRs within minutes and match them to a suspect's DNA profile. Police outfitted with portable computers will be able to access the DNA databanks to screen the profiles of thousands of men. The FBI felons' database has samples from eight thousand unsolved crime scenes and state law enforcement has accrued approximately 620,000 samples from lawbreakers, including those suspected or convicted of minor crimes.

Every state now maintains genetic databases that are matched to genetic samples taken from crime scenes, such as blood traces, in order to facilitate finding the person who has committed the crime.[13] Crier echoed the sentiments of many present when she asked why being in the

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 307

genetic database would be a problem for an innocent black man. "If he is not guilty, what is the problem for a man in the database? He has nothing to worry about."

But he does. Multiple levels of bias feed the all-black and Hispanic databases, and lawsuits such as the Pamela Fish case cited earlier already have verified that DNA evidence is no more immune to fraudulent or incompetent manipulation than is other evidence. Then, too, there is the issue of collective stigmatization: If only men of color are in the database, only men of color become suspects and only they can be convicted. Databases that exclude white men, the numerical majority group, will miss most criminals. As the *American Criminal Law Review* points out, "Optimal effectiveness, however, would require a universal DNA database that contains DNA fingerprint of *every* citizen, otherwise potential matches would be missed."[14] Although a universal DNA database would be more efficient than one based upon skin color, it is also ethically unacceptable because it would necessitate coercion. The DNA sweeps, from Miami to London to Truro, have met with varying levels of resistance and resentment and so cannot be described as voluntary.

Will the novel DNA fingerprinting technology lead to the imprisonment of more African American men than have been freed because of it? This technology's benevolent face has been seen most often, but it has another, sinister, visage. This dual nature holds true for almost every application of genetic science to African American health and welfare. Historically, every boon appears to have been accompanied by a stigmatizing threat to health or freedom. For American blacks, genetics has always been wielded as a two-edged sword.

Sickle-Cell Misstep

African Americans are no strangers to genetic innovation, but unfortunately, genetic therapy has long been sabotaged by racial myths and bad science. The agenda-driven nature of much genetic research with African Americans has rendered many blacks wary of all genetic science. One of the most infamous examples within recent memory has been the family of troubled genetic initiatives surrounding sickle-cell disease.

Chapter 6 described how in 1910, cardiologists James B. Herrick, M.D., and Ernest E. Irons first identified the "thin, elongated, sickle-

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 308

shaped" red blood cells of a desperately ill twenty-year-old dental student from Grenada. A year later, a Virginia medical journal published a description of a twenty-five-year-old black woman with similar symptoms. Soon, reports of African Americans with sickle-cell anemia, a constellation of dire conditions ascribed to misshapen "sickled" red blood cells, began to flood medical journals. When people with the disorder are exposed to environmental insults such as low-oxygen environments, their red blood cells deform into a sickled shape and become adhesive, sabotaging the cells' ability to carry sufficient oxygen and causing them to block small blood vessels, including capillaries. These events trigger excruciatingly painful episodes, known as sickle-cell crises. A sickle-cell crisis can generate not merely anemia but also bleeding ulcers, strokes, a heart attack, or the loss of limbs and tissues, depending upon the location of the compromised blood vessels. Thus physicians often prefer the term *sickle-cell disease,* pointing out that most of the sufferers' worst medical crises have little to do with anemia. By 1920, an erroneous belief had become firmly entrenched that sickle-cell disease was a racial condition that struck only African Americans.[15] However, it also affects people from Mediterranean, Middle Eastern, and West African regions, but not those from South African and East Asian regions.[16]

After the supposed postwar conquest of infectious disease via antibiotics and after the discovery of DNA's double-helical structure in 1951, genetics gained primacy as the preeminent mode of understanding and attacking disease. In 1949, sickle-cell anemia became the very first molecular disease to be identified. Scientists learned that sickle-cell anemia was the worst of several sickling-cell disorders and that it struck one in every four hundred African American newborns. They also knew that sickle-cell disease and a slew of closely related blood disorders called hemoglobinopathies struck not only blacks but also persons of other races. For example, one such blood disease, thalassemia, affects people of Mediterranean, Middle Eastern, and African extraction. But sickle-cell anemia's identity as a black disease was so firmly entrenched that blacks with thalassemia are still often misdiagnosed with sickle-cell disease.[17]

Sickle-cell disease is recessive: A person must carry two of the recessive genes for sickle-cell disease to develop the illness. People with only one sickle-cell gene are said to be heterozygotes, or carriers, who are essentially well. But if two heterozygotes for sickle-cell disease marry, their

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 309

offspring run a one-in-four chance of developing the disease. If a carrier marries a person without the gene, none of their children will develop sickle-cell disease, but their children run a one-in-two chance of becoming carriers themselves. Carriers of sickle-cell disease are sometimes referred to as having the sickle-cell trait, but despite the connotation of illness that the word *trait* carries, they are well. (Because of the potential for confusion, this chapter avoids the term *sickle-cell trait* whenever possible.)[18]

By the late 1960s, workplaces instituted genetic screening, ostensibly to protect vulnerable employees by avoiding their placement in work environments that could trigger illness such as a sickle-cell crisis. The federal government supported initiatives that encouraged widespread genetic screening of sickle-cell disease, and African Americans themselves pushed for many of these initiatives to test for and counsel people at risk for sickle-cell disease, so there is no doubt that many of the projects were well intentioned. However, some were not. And in many cases, good intentions paved the medical road to perdition.

"Sickle-cell screening created huge problems," recalls Vernellia Randall, professor of law at Dayton University. "Airlines, for example, said pilots with the trait couldn't fly."

Why not, if they were healthy? In 1968 and 1969, doctors at Fort Bliss in El Paso, Texas, grew concerned that army basic training was suddenly proving more than usually hazardous—even deadly. Within eleven months, four recruits had collapsed and died suddenly, all of them black. Even more alarming were the autopsy results, which showed the men's red blood cells were now sickle-shaped. The soldiers were black and the high altitude of the boot camp—4,060 feet—suggested that the deaths might have been due to sickle-cell disease crises triggered by the low-oxygen environment characteristic of high altitudes. But *The New England Journal of Medicine* report on the men's deaths noted that the sickled cells didn't necessarily mean that the men had sickle-cell disease, because the misshapen cells could have been a consequence, not the cause, of their deaths. When the National Academy of Sciences studied the deaths, it could neither rule out sickle-cell anemia nor prove that it had killed the men.[19]

But the U.S. Air Force Academy rushed to judgment, promptly issuing a directive barring the admission of all black sickle-cell carriers—

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 310

healthy people. Carriers were permanently grounded, were banned from copiloting, and were reduced to ground jobs. It is worth noting that by banning black carriers from admission, the academy was effecting a large-scale restoration of its long-standing, nakedly race-based ban on blacks entering the academy, but now it could offer the rationale of protecting them.[20]

Strangely, scientists as well as laypersons confused well sickle-cell carriers with the homozygotes who had both genes for sickle-cell disease and therefore had the disease. However, this confusion was no accident: It resulted in profits for Ortho Pharmaceutical Company of McNeil Laboratories, the company that sold the so-called sickle-cell screening test, which did not differentiate between the sickle-cell trait and sickle-cell disease. Ortho was promoting and distributing a test it called Sickledex that could not discriminate between sickle-cell carriers and people with sickle-cell disease. That is, Sickledex detected the presence of the gene, but not whether one or two genes existed. In order to market the test, employers, military hospitals, and the government extended to carriers the same advice and restrictions that applied to people genuinely ill with sickle-cell anemia. Otherwise, these agencies would have had to admit that the test was of extremely limited therapeutic value because it could not tell a sick person from a well one.[21]

The National Institutes of Health, hospitals, and private organizations disseminated brochures and booklets equating carrier status with the disease, and millions of well black people were informed that they were ill and genetically tainted. Some were told that they had a life expectancy of twenty years. The very first sentence of the preamble of the National Sickle Cell Anemia Control Act, enacted in 1972 to foster sickle-cell research, screening, counseling, and education, is untrue: "Two million Americans suffer from sickle cell disease." Actually, 2 million people were healthy carriers[22] and fewer than 100,000 Americans suffered from sickle-cell anemia. The erroneous claim coupled with its constantly reinforced perception of sickle-cell disease as a black disorder left Americans with the mistaken impression that a good portion—one in twelve—of African Americans suffered from sickle-cell anemia.[23]

The perception of sickle-cell heterozygosity as a disease state is an eloquent illustration of ethnocentrism, because far from being unhealthy, this carrier status confers the distinct biological advantage of immunity to the deadliest strain of malaria. This helps sickle-cell carri-

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 311

ers in malarious areas to survive. At the Eighth International Congress of Genetics in 1949, evolutionary biologist J. B. S. Haldane first proposed that people with one gene for sickle-cell disease were "more resistant to attacks by the sporozoa that cause malaria." In parts of Africa and other countries where malaria-carrying mosquitoes thrive, people who have one gene for sickle-cell anemia and one gene for normal hemoglobin are not only healthier than people with sickle-cell anemia but also healthier than people without the trait—those with normal hemoglobin. Being a heterozygote for sickle-cell anemia protects one from invasion by the deadly *P. falciparum* strain of malaria in several ways. A form of the malaria parasite—the plasmodium—infects the person's red blood cells, but in heterozygotes, the plasmodium causes only the infected red blood cells to sickle by making the cell environment more acidic: this increased acidity, in turn, makes the hemoglobin lose oxygen, which further escalates the sickling of the infected cells. However, the resulting lack of oxygen also depletes the infected cells of potassium, which kills the malaria parasites. Any surviving parasites are picked off by the person's immune system, and the sickled cells are taken out of circulation, destroyed, and eliminated from the body along with the parasites. The uninfected red blood cells do not sickle and the person suffers neither from sickle-cell disease nor from malaria.

In malarious environments, sickle-cell heterozygotes are 15 percent more likely to survive and to reproduce than their neighbors with normal hemoglobin.[24] This is called the "heterozygote advantage" and it helps to explain why the common denominator for groups carrying the sickle-cell gene is not being black, but living in proximity to the malaria-bearing anopheles mosquito. Other genetic diseases that also are thought to confer a heterozygote advantage include cystic fibrosis, the most common genetic disease among people of European descent, which protects against the fatal dehydration of cholera and typhoid, and scientists have suggested that heterozygotes for Tay-Sachs disease, which preferentially strikes Ashkenazi Jews, may enjoy increased protection against tuberculosis.

Today, the United States sees only about one thousand cases of malaria annually, so that the heterozygote advantage is not terribly useful to a North American, except for travelers to malarious areas and as an object lesson in the interplay among genetics, disease, and culture.

African Americans were among those confused by the erroneous

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 312

medical advice the government was dispensing. Many states mounted compulsory genetic-screening programs, which many blacks welcomed, but which caused others, including genetic experts, to feel stigmatized. For example, James Bowman, M.D., an African American professor of genetics at the University of Chicago, was the lone voice crying out in the genetic wilderness when he was invited to address a 1971 Black Panthers event. There, sickle-cell screening was being conducted by community leaders, who warned that anyone who tested positive could expect to live only twenty years longer. Bowman forcefully objected that the testing was unable to identify the genuinely ill, and that in any case, the clinical picture was far less dire. Despite Bowman's credentials and protests, the black and white organizers persisted in the erroneous testing and counseling.

Seventeen states enacted sickle-cell screening laws, often in response to requests from African Americans. But black Americans did not clamor for workplace screenings, which threatened privacy and raised questions that could create a genetic underclass of workers. In 1971, almost nine hundred diseases were known to be genetic, yet screening tests could identify the carriers of only fifty genetic diseases.[25] However, screening for sickle-cell disease was the genetic test performed most often by employers. By 1975, tens of thousands had been screened for Tay-Sachs and thalassemia, but half a million blacks had been screened for sickle-cell disease. *In the Name of Eugenics,* a social history of genetics by Daniel Kevles, notes, "No one argued seriously for the screening of every possible parent, but some did urge the screening of people from groups at comparatively high risk for particular genetic diseases, notably blacks. . . ."[26]

The National Institutes of Health's policies and publications focused exclusively on African Americans, solidifying sickle-cell anemia in the American psyche as a black disease. Unfortunately, the government policies still confused the disease state with being a carrier. Screenings were performed en masse at a variety of sites in an assembly-line fashion with agenda-driven, inaccurate counseling. When screening revealed that a person carried the trait for sickle-cell disease, that information was dumped upon her; she was informed she was sick, given a brochure that erroneously equated the disease with the trait, then often dismissed without further support or answers except for the one piece of advice

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 313

that was always dispensed—the inadvisability of marriage between two people with the trait, because they could produce children with sickle-cell anemia. This was often the main informational point of the screening, to identify affected people so that they would know not to have children. Such advice led many African Americans to accuse genetic counselors and counseling programs of genocide, especially after 1973. That was the year amniocentesis allowed prenatal testing of the amniotic fluid, first for life-threatening disorders, then for genetic defects, and later for sickle-cell anemia. This was also the year that *Roe v. Wade* gave American women access to legal abortion on demand.

Genetic counselors, who had dispensed pointed advice along with diagnoses since the 1950s, were supposed merely to provide diagnosis and disease information, but they still practiced virtually unregulated and many recommended abortion on the basis of testing that could not discern the trait from the disease.[27] "For at-risk couples who conceived at that time," recalls Vernellia Randall, "the advice was pregnancy termination. Some viewed these as attempts to limit the fertility of blacks."

Discrimination against sickle-cell carriers has been slow to dissipate, lagging well behind scientific knowledge. The U.S. Air Force Academy's admission bar and grounding of heterozygous pilots, for example, was ended only in 1981, by a lawsuit.[28]

Testing, Testing

Today, unscrupulous employers continue to wield genetic screening, but they now do so surreptitiously, without employees' informed consent. In 2001, the Equal Employment Opportunity Commission charged Burlington Northern Santa Fe Railroad with running genetic tests on workers who filed claims for carpal tunnel syndrome. If tests had shown them to have any genetic predisposition to the condition, the railroad could have argued that it should not be held liable.[29] Some lawsuits spawned by such abuses allege racial bias. Perhaps the most egregious was the case of *Norman-Bloodsaw v. Lawrence Berkeley Laboratory,* a research center that the federal government ran in cooperation with the University of California. In 1998, 172 employees, all but one of them black, sued LBL when they learned that they had secretly been tested for syphilis, pregnancy, and sickle-cell trait without their knowledge that the

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 314

blood and urine they had supplied during required physical examinations would be tested in this manner. These tests were insulting as well as intrusive, and were illegal under the Americans with Disabilities Act. But what is particularly disquieting is the lack of scientific sophistication the laboratory demonstrated in testing only its black employees for the sickle-cell trait: Scientists should have known that not only blacks were at risk and they should also have known that carrier status imparted no reasonable disability risk. The blatantly racial nature of the screening was suggested when plaintiffs learned that the only white employee to have been tested for venereal disease was a white man married to a black woman. In August 2000, the University of California settled the $2.2 million suit brought by these black employees.

The privacy of these workers was illegally assailed and they could have been unfairly stigmatized. But there is another reason that being tested for genetic issues without one's consent is damaging: The price of genetic knowledge can be intolerably high. The health information contained in one's genes can give clues to prevention and self-care, but such information can also generate futile anxiety and lay one open to layers of medical and financial discrimination. "If you know of a genetic condition and lie about it to your insurance company, they can refuse to cover you," observed Marian G. Secundy, Ph.D., the late director of the National Center for Bioethics in Research and Health Care. "If you learn you are at risk for a disease that cannot be treated, the information can be worse than useless: The knowledge will not enable you to protect yourself, and you will suffer mental anguish over an illness that you may never acquire."

Employers who refuse to hire people when they learn of genetic indicators for a disease may relegate them to an "unemployable" biological underclass. And that's not just a concern for those with known genetic disorders, because everyone's genome harbors a few bad apples—genes that could, but do not necessarily, indicate a health problem. The more people are forced to reveal about their genome, the greater their risk of suffering genetic discrimination. Currently, black people are most likely to be subjected to such testing, in large part because testing for sickle-cell disease is the most common genetic screen used by employers and insurers. A 2000 congressional report predicts that such discrimination may become widespread as employers are pressured to contain health-care costs.

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 315

Already, black women, who have a higher-than-normal risk of the BRCA1 gene, fear their insurers and employers may discover their status should they seek genetic testing. "Some women seek gene testing on their own and pay for it out of their own pockets because they don't want their insurance company to know," noted Tene Hamilton, an Alabama genetic counselor.

Might other genetic tests preclude African Americans from desirable jobs in the near future? Consider, for example, that a genetic mutation affecting resistance to chemotherapy occurs more frequently in African and African American populations than in Caucasian or Asian populations.[30] A 1998 research study of African Americans and Hispanics living in Manhattan revealed that they harbor a genetic variant (APOE-epsilon4) that places them at a higher relative risk of developing Alzheimer's disease than whites.[31] African Americans are more likely than whites to be healthy carriers of glucose-6-phosphate dehydrogenase (G6PHD) syndrome,[32] which can cause the loss of red blood cells and affects many medical risks and medication reactions. If this carrier status is detected by tests and is miscategorized as a disease state, will blacks be barred from desirable jobs? Of course, each of these genetic complements appears in other ethnic groups as well, but the rates—and thus the risks—are higher among black Americans.

There is also the widespread misconception that simply having a disease gene means you have the disease. This is not so. Most common adult-onset genetically influenced diseases, such as Type II diabetes, hypertension, and cancer, typically result from several genetic factors, not from a single gene. It often also takes environmental triggers (obesity, nutrient deficiency, exposure to noxious chemicals, for example) to cause the disease to manifest. What's more, genes interact to temper one another's effects. All these factors complicate determining who is at risk, and they also hamper scientists' attempts at gene therapy.

Less Than Global: The Human Genome Project

Used therapeutically, genetics hold out promises of enormous improvements in African American health, but the promises have as yet gone unrealized. For example, research into sickle-cell disorder, the first identified molecular disease, remains underfunded and the disease still awaits an effective treatment, but effective genetic therapies were

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 316

mounted within just a few years after the gene for cystic fibrosis was discovered in 1989. Whites are at much higher risk than blacks for cystic fibrosis.[33] Therapeutic research sometimes bypasses blacks because finding a gene for an illness and curing an illness are two very different things and decades may separate one from the other. Also, the interests of African Americans too often fall below the radar screen of mainstream genetic research, and much more quality research should be undertaken into blacks' genetic risks. This may seem an ironic concern for a book that has focused upon the experimental abuse of blacks, but it is merely the obverse of the research-abuse coin: As research has become an important avenue of therapy the proportionate inclusion of African American in ethical, therapeutic research has become imperative.

Take the Human Genome Project (HGP), which has been touted as a unifying global enterprise to map all of humanity's genes and has been sold to the public on the strength of its role in finding cures for many illnesses. The U.S. National Institutes of Health and London's Wellcome Trust have completed the vital arms of the project, which began in 1990. The 30,000 genes constituting the genetic makeup of a human being have all been identified and mapped.

However, Dr. Georgia Dunston, a geneticist at Howard University, claimed in the mid-1990s that of the more than sixty families whose genes were analyzed by the project, there were no people of African descent.[34] She lamented that severing the African branch of the family tree is a critical error because African gene pools are the oldest and consequently the most diverse on the planet, due to human life's having evolved in Africa. Dunston asked, "What picture of humankind can emerge without Africa?"

Also, of the 100,000 professional HGP scientists from sixteen separate research universities in six countries, only a few, aside from laboratory assistants, were black.[35] Dr. Bettie J. Graham, program manager for the National Human Genome Research Institute at the National Institutes of Health, told the *Journal of Blacks in Higher Education,* "Unfortunately, African Americans have not been involved in the first phase of the Human Genome Project." However, the relatively small numbers of blacks conducting biomedical research for the project also proved a factor.[36]

Howard University was, however, belatedly invited to contribute

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 317

data and has since received considerable support, which enabled it to open the National Human Genome Center, with Dunston as its director. Today, the center is pursuing several projects of importance to African American health. Among them is a search for candidate genes of complex diseases that are common in African American populations. These include prostate cancer, breast cancer, asthma, Type II diabetes, hypertension, and HIV/AIDS.

The near homogeneity of the HGP is ironic, because the stirring message of the Human Genome Project data is a ringing denunciation of race. Analyses found so little variation among the genomes of what have been thought of as separate racial groups and so many genetic characteristics in common that race was found to have no basis in biology.

This book uses the term *race* because it is accepted argot, a convenient, commonly used way of designating ethnic groups that are perceived as distinct. We all know what we mean (or think we do) when we denote someone's race as "black" or "white." In our nation, race is inarguably important in discussions of health and disease. However, the Human Genome Project has erased any lingering doubts: Biological race does not exist, because all humans share the same genes. Although the proportions of genes differ, meaning that genetic differences exist, these variations map very poorly onto what we think of as races. This seems to introduce a logical contradiction: If race is not real, how can we speak of race-based therapeutics? The answer is that race *is* real, but it is not biological: It is social. What correlates very closely to most "racial" differences in life expectancy, mortality, disease susceptibility, and survival is the race to which one is perceived as belonging.

This is contrary to conventional wisdom and at first blush seems easily refuted: The racial differences between an Icelander and a Nigerian seem obvious. But so do the differences between a dark-skinned Asian from southern India and a pale North African, yet the former person is classified as Caucasian and the latter as "black." Historically, confusion has been sown by the fact that in the early days of the republic and of African enslavement, the Africans who were imported represented only the polar opposite of pale-skinned Europeans in skin color and hair types. Africa is home to people of every skin color, hair type, stature, or other physical measure, but the rich diversity of Africa and, for that matter, of Europe was not represented in seventeenth-century America. Only

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 318

the dark-skinned denizens of West Africa and principally pale-skinned Anglo-Saxons populated the colonies. If our forebears had included dark-skinned Finns and Mediterraneans on the one hand and North Africans, East Africans, Egyptians, and Somalians on the other, they would have had a better appreciation for the presence of similar phenotypic traits in all ethnic groups. When one looks at the diverse bounty of all peoples, it is easier to appreciate that most of the various criteria we have for sorting people into races—skin color, eye color, hair texture, body type, blood types, disease susceptibility—map very poorly onto genetic frequencies, albeit with a few dramatic exceptions.

For there are exceptions, and although they are rare, it is important from a medical point of view to recognize them when we see them if we want to devise the best-possible medical treatments. However, many genetic diseases are no respecters of race: As we have seen, sickle-cell disease affects Mediterranean peoples, Africans, and South Asians, among others; the autoimmune disorder sarcoidosis afflicts principally African Americans *and* Scandinavians. Some genetic risk factors for diseases such as heart disease, prostate cancer, and low birth weight are present in African Americans but not in Nigerians and West Indians, suggesting that factors other than African heredity are at work.

Today, the commercial marketing of genetic theories is being undertaken with data from the HGP with African American markets very much in mind. A vital part of this marketing plan involves African American pharmacogenomics, the custom-tailoring of medications to exploit genetic variations. But statistically, only a small percentage of genetic variations—about 0.1 percent, one in a thousand—can be laid to race.

Exploiting that real one genetic difference in a thousand to develop more effective medications for African Americans or for any other group is an exciting, very positive tool, especially if it can focus upon major killers such as cancer, heart disease, stroke, and HIV. However, most genetically distinct diseases and differences between ethnic groups account for only a small fraction of the illness and death in any community.

Heart of Darkness

In the late 1990s, the Pharmaceutical Research and Manufacturers of America (PhRMA) boasted that its members had 99 medications in de-

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 319

velopment that addressed the particular medical needs of African American patients. By 2004, that number had grown to 249 medicines. But these were not drugs tailored specifically to black patients' medical needs; nearly all of these medications treated illnesses that African Americans suffer at higher rates than whites, which encompasses nearly every serious ailment.[37] It is certainly laudable that drug companies are producing medications that address black health needs. However, the implication that these were *tailored* to racial needs is easily recognizable as a marketing ploy.

The case of BiDil, a heart drug approved by the FDA in July 2005, is different. BiDil is an oral combination of two drugs, hydralazine and isosorbide dinitrate, that act as antioxidants, widen blood vessels, and produce nitric oxide, which, BiDil makers say, provides beneficial effects for African American heart failure patients. It was developed for its potential to reduce deaths and serious illness among African Americans diagnosed with congestive heart failure.[38] CHF is a condition in which the heart muscle, which has been weakened or otherwise compromised by injury or disease, fails to maintain circulation properly. The overwhelmed heart triggers a cascade of functional deterioration that culminates in a slow death: It is commonly fatal within a decade of diagnosis. People with congestive heart failure may suffer from constant fatigue, swollen legs, and respiratory problems. Or heart failure may be insidiously asymptomatic. BiDil's patent holders say their medication's mechanism of action addresses a genetic anomaly that makes African Americans particulary susceptible to CHF. This medication is in the vanguard of new commercial marketing of genetic therapies for blacks.

NitroMed, the Cambridge, Massachusetts, biotechnology firm that developed BiDil, claims that it is the first specifically tailored medication to treat congestive heart failure in an estimated 750,000 African Americans patients. Clearly, BiDil should be embraced and supported if it works to decrease death and disability due to CHF. But its marketing as an exclusively African American genetic medication is just as clearly troubling for both scientific and social reasons.

First, is the medication driven by a true biological dimorphism in black heart patients or is it the product of a fertile market? In an illuminating analysis in the *Yale Journal of Health Policy, Law, and Ethics,* Jonathon Kahn has weighed the medical evidence and found it wanting. His investigation reveals that BiDil began life not as a specialized med-

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 320

ication tailored for African American heart patients, but as a heart drug aimed at the general public. Neither its first clinical trials in 1987 nor its patent application in 1988 mentioned racial applications, and only after the FDA Advisory Committee refused to approve BiDil's use for a general population in 1997 did NitroMed reanalyze twenty-year-old data from its first trials, looking for possible special applications that might allow it to approach the FDA with a revised application.[39] The Food and Drug Administration's Modernization Act had recently required inclusion of racial minorities and women in clinical trials, and in 1997 Surgeon General David Satcher drafted the resolution that made resolving racial health disparities a national priority. In 1998, BiDil was reborn as a black medication, rescuing the drug from pharmaceutical oblivion.

But how did NitroMed make a case for BiDil's transformation from a medication for everyone to a genetic drug that addresses specific weaknesses in African Americans, even before clinical trials were conducted? Was it based upon a proven special utility for black patients?

In part, NitroMed achieved this by creating a perception of CHF in blacks as a racially distinctive disease, then supplying the medication that was "necessary" to address this biological dimorphism. First, as Kahn has pointed out, BiDil's makers made a case for CHF as a racial disease claiming that there is a huge difference in the mortality rate between black and white patients with CHF. NitroMed scientists claimed that CHF kills blacks at twice the rate it does whites, and publications from *Science* to *Today in Cardiology,* as well as press releases from the Association of Black Cardiologists, affirmed this disparity.[40]

But the data contradict this claim. It is true that proportionately twice as many blacks as whites died of CHF in 1988, but reducing the rate of heart failure in African Americans has been a medical success story, and by 2003 the gap had nearly closed. Most recent CDC figures indicate that the racial ratio of heart-failure deaths is 1.1 blacks for every 1 white—they are almost identical.[41] Kahn traced the provenance of NitroMed's widely disseminated figures and found that they were based upon very old studies, including National Heart, Lung, and Blood Institute (NHLBI) data collected in 1995. At the time NitroMed was using this data, it was already woefully outdated and no longer accurate. NitroMed's researchers used numbers that were not only old but also inappropriate, because they cited National Health and Nutrition Examination

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 321

Survey (NHANES) data from 1988 that described prevalence, the number of people *suffering from* CHF, which is very different from mortality, the number of *deaths from* CHF.[42] One 1987 study does seem at first blush to support the NitroMed figures because it indicated that 1.8 black men died of CHF for every affected white man and that 2.4 black women with CHF died for every afflicted white woman. But in addition to being old superseded figures, these figures describe deaths within a specific age range, from thirty-five to seventy-four. Thus they reveal a serious disparity in the age at death, not in absolute deaths. The same percentage of blacks and whites die of CHF, but 50 percent of blacks who die of CHF are between the ages of thirty-five and seventy-four, while only 30 percent of whites who die of CHF are seventy-four or younger: Most whites who die of CHF do so quite late in life. In short, bad data helped BiDil boosters to portray CHF as a racial disease by exaggerating its death rates in blacks and raising the specious question of why so many more blacks than whites die of the disease.

NitroMed explained that only physiology could explain such a dramatic disparity in the death rate. In doing so, BiDil's promoters discount the well-substantiated research into myriad nongenetic factors that drive CHF death rates. Nongenetic interventions in the form of better access to medical care, more preventive lifestyle changes, and high-tech interventions have already cut the African American CHF death rates from twice that of whites in 1988 to essentially the same as whites in 2003. This fairly quick reduction didn't emanate from genetic techniques or changes and thus strongly suggests that nongenetic factors are most important. So does recent research that suggests heart failure is fed by hypertension and kidney disease. Hypertension in blacks, in turn, has been shown to be driven by stress (including the stress of racism), by diets that are high in fat, possibly by salt sensitivity, by overweight, and by obesity. There is even evidence that hantavirus infection spread by rodents in urban settings can cause kidney disease and hypertension.[43] So can exposures to some poisons in such urban settings. A slew of reports, beginning with those published by *The New England Journal of Medicine* in February 1998, have shown that limited access to high-tech care has also fed blacks' higher mortality from heart disease. But researchers and news articles that discuss the merits of BiDil tend to give the nongenetic factors short shrift. As Kahn points out, Clyde Yancy, a black cardiologist

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 322

on the steering committee of BiDil's trial, says that the data "do not support socioeconomic factors as important contributors to the excess mortality rate seen in African Americans affected with heart failure."

BiDil patent holder Jay Cohn, M.D., and his colleagues wrote papers positing a genetic mechanism for CHF in blacks: "a pathophysiology found primarily in black patients that may involve nitric oxide insufficiency," which makes the cause of their heart failure different from that of whites. Clyde Yancy agreed, saying, "Heart failure in blacks is likely to be a different disease" and adds "the emerging field of genomic medicine has provided insight into potential mechanisms to explain racial variability in disease expression."

But even if the putative difference in nitric-oxide metabolism were found primarily in African American patients, this would not mean that *all* African American patients in heart failure harbor it, or even most African American patients. Nor would it mean that such an anomaly is restricted to blacks.

Since the publication of Kahn's analysis, NitroMed has quietly revised the numbers in its promotional materials. It no longer claims that African American CHF deaths are double those of whites. But the alarm sounded by its earlier claims already served its purpose: The FDA gave the drug another opportunity in clinical trials, this time to prove that the drug is efficacious against CHF in African Americans

In 2003, NitroMed, with the Association of Black Cardiologists as a highly visible participant and supporter, mounted a clinical trial. NitroMed enrolled 1,050 African Americans for the trial of BiDil as a treatment for heart failure in African American subjects. The trial was called A-HeFT, an acronym for the African American Heart Failure Trials,[44] and it tested BiDil not on its own but in conjunction with fully approved heart medications. In August 2004, the clinical trials to demonstrate BiDil's safety and efficacy were halted because, its makers say, the results were clearly beneficial to blacks suffering from heart failure. The results showed that 6.2 percent of patients given BiDil died; 10.2 of patients who did not receive BiDil died, constituting a 43 percent survival advantage for those taking the medications.

The FDA has approved BiDil's race-based labeling. This means that although a doctor may choose to prescribe it for non–African Americans in an "off-label" use, insurers will not have to cover its cost for them. The

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 323

study should have included whites in order to provide evidence that the drugs works differently in blacks, but because the patents for use in all races will expire in 2007, there is no economic incentive to test the drug in whites. (NitroMed will hold the patent for the use of BiDil in blacks until 2020.) In an ironic twist, whites are being subjected to racial exclusion by being denied access to testing or use of a heart drug that could benefit them or even save their lives.

NitroMed stock rode the good news from the A-HeFT trials to a 73 percent leap in share price. Because it was tested only with other drugs, BiDil typically will be prescribed for use in concert with other drugs, not instead of them, so that BiDil will not compete in the marketplace with established heart medications. This will help BiDil's sales and this could even explain why BiDil was tested only against a placebo: Had BiDil been tested alone, researchers would have run the risk that the study results could have been different, finding that BiDil provided less protection to black patients than standard medications.

Because heart disease is the number-two killer of blacks—and whites—BiDil should be embraced if it indeed conveys a racial benefit to blacks with CHF. So should any other therapy that accurately targets clinically meaningful disease vulnerabilities in African Americans. But the development of a genetic drug for what has been newly dubbed "a racial disease" also raises long-term issues that temper its immediate benefits.

We soon will see other medications marketed for "genetically distinct" populations of African Americans. The glaucoma medication Travatan is being promoted to African Americans as "the first glaucoma drug to demonstrate greater effectiveness in black patients," although the FDA-required informational insert indicates in fine print that eye color may be a better indicator of its effectiveness than race. Prostate-cancer therapies genetically tailored for African American men are in the pipeline. Recently, 89 percent of breast-cancer tumors from African American women tested positive for a newly found gene, BP1, compared with 57 percent of those from Caucasian women. Can a special medication tailored to the black breast be far behind? It will also be important for African Americans to study and, where applicable, to support such research efforts by joining ethical therapeutic trials that offer the best-possible safety protections. To find these trials, African Americans

PDF

Wash_0385509936_2p_all_r1.qxp 8/24/06 12:51 PM Page 324

should discuss them with their personal physicians and consult resources available on-line that offer "how-to" primers on joining clinical trials.

But unsurprisingly, given the subject of this book, I also advise African Americans to look before they leap. Although many black cardiologists and many in the African American news media applaud the BiDil innovation,[45] the specter of neoracial disease based upon questionable genetics should give one pause for many reasons. African Americans must actively support the search for disease risks and therapies, but they must also be conscious of the long-term import of funneling scarce resources into race-based medications unless they provide the best therapeutic approaches.

A genetic fix for a nongenetic disease is unlikely to be the most efficient approach. What's more, racializing CHF allows scientists and policy makers to ignore the environmental factors that are the chief causes of the racial heart-disease disparity. Racial genomics also raises profound social questions. If physicians fall back into the antebellum habit of treating blacks' ailments according to race, will not this condemn many to poorer, stereotyped, less appropriate care? Because race is not a biological reality, medications based upon group biological differences will work only for some African Americans. This will lead to a false sense of security, and will stymie the search for more inclusive, more efficacious, and, in a word, better treatments. We must recognize the powerful stigmatizing potential of genetic approaches to disease, especially when they are touted as the only approach.

From tools that could release or convict to the troubled history of genetic disease fixes that may provide cures or mere stigmatization, genetics offers a cornucopia of medical answers and pitfalls to blacks. The next chapter gives the history of another mixed blessing: research into infectious diseases.

PDF