2011

What's Wrong with Race-Based Medicine?: Genes, Drugs, and Health Disparities

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Recommended Citation
Available at: https://scholarship.law.umn.edu/mjlst/vol12/iss1/3

The Minnesota Journal of Law, Science & Technology is published by the University of Minnesota Libraries Publishing.
I. INTRODUCTION

In June 2005, the Food and Drug Administration (FDA) announced a historic decision: it approved the first pharmaceutical indicated for a specific race. BiDil, a combination drug that relaxes the blood vessels, was authorized to treat heart failure in self-identified black patients. BiDil had been tested in the African-American Heart Failure Trial (A-HeFT) launched in 2001. A-HeFT enrolled 1,050 subjects suffering from advanced heart failure, all self-identified African Americans. A-HeFT showed that BiDil worked; in fact, it worked so spectacularly that the trial was stopped ahead of schedule. BiDil increased survival by an astonishing 43 percent. Hospitalizations were reduced by 39 percent. It was a momentous
accomplishment for Jay Cohn, the University of Minnesota cardiologist who invented BiDil and had pioneered vasodilators as an important treatment for heart failure.

Given evidence of BiDil’s efficacy, but little evidence that race mattered to its efficacy, the FDA should have made one of two decisions: reject the request for race-specific approval or approve BiDil for all heart failure patients, regardless of race. Instead, the FDA put race at the center of its decision, sparking controversy and paving the way for a new generation of racial medicines.

No one is complaining that BiDil is available to people who will benefit from it. The problem is that BiDil was made available on the basis of race. Its racial label elicited three types of criticism: scientific, commercial, and political. I will discuss the first two controversies en route to what I consider the main problem with race-based medicine, its political implications. By claiming that race, a political grouping, is important to the marketing of drugs and that race-based drugs can reduce health disparities, which are caused primarily by social inequality, those who promote racialized medicine have made it a political issue. Yet, having made these political claims, these very advocates answer criticism by saying that we must put aside social justice concerns in order to improve minority health. This article explains why marketing pharmaceuticals on the basis of race is more likely to worsen racial inequities than cure them.

II. RACE-SPECIFIC MEDICINE IS SCIENTIFICALLY FLAWED

What does it mean for a pharmaceutical to be race-specific? A drug that is labeled for use by a particular race sounds like it has been developed based on scientific evidence that its ingredients work for one group and not for others because of some underlying biological difference. But there is no such drug or scientific evidence supporting it. BiDil is a case in point. It does not contain new ingredients. It was not designed only for black people. Nor was it developed to target any particular genotype that only black people supposedly have or are more likely to have. Instead, it combined into a single pill two generic drugs that had been prescribed to patients regardless of race for over

a decade. In fact, Dr. Cohn originally intended to market it to patients of any race who could benefit from it. There is not even scientific proof that BiDil works differently in black people because the clinical trial that tested BiDil enrolled only “self-identified” African Americans. There is no basis for a comparative statement if only one group has been tested.

Its maker, NitroMed, asked the FDA to authorize BiDil as a race-specific drug on grounds that its clinical trial involving only African American patients showed a dramatic reduction in their heart failure deaths. In other words, the company argued that, because BiDil was tested only on blacks, the FDA should label it as a drug for blacks only. As Jane Kramer, NitroMed’s vice president of corporate affairs, would later explain, “That doesn’t mean that it works in all African Americans and it doesn’t mean that it doesn’t work in other patients. It just means that we know it clearly works in African Americans.”

This kind of logic had never resulted in a racial indication before. In the past, the FDA has had no problem generalizing clinical trials involving white people to approve drugs for everyone. That is because it believes that white bodies function like human bodies. However with BiDil, a clinical trial involving all African Americans could only serve as proof of how the drug works in blacks. By approving BiDil only for use in black patients, the FDA emphasized the supposed distinctive, and substandard, quality of black bodies. It sent the message that black people cannot represent all of humanity as well as white people can.

Why did BiDil work especially well in black patients? NitroMed had no scientific evidence to answer this question, but it speculated that the mechanism had to do with biological difference. In a March 2001 press release, NitroMed explained

6. Taylor, supra note 2, at 2049.
8. Taylor, supra note 2, at 2050.
that BiDil works especially well for African Americans because “observed racial disparities in mortality and therapeutic response rates in black patients may be due in part to ethnic differences in the underlying pathophysiology of heart failure.”

NitroMed sought FDA approval based on pure speculation that race was a good enough proxy for some underlying genetic or pathophysiological difference without conducting any investigation whatsoever to test this claim. The researchers who reported BiDil’s effectiveness for African American heart patients recognized this flaw and simply promised to correct it at some future time.

The FDA expounded the race-as-proxy theory in a January 2007 article in *Annals of Internal Medicine* explaining its approval of BiDil. “We hope that further research elucidates the genetic or other factors that predict the usefulness of hydralazine hydrochloride-isosorbide dinitrate,” the author wrote, “Until then, we are pleased that one defined group has access to a dramatically life-prolonging therapy.” In other words, a racially-defined group could serve as a temporary substitute for the yet undiscovered genetic or other factor that identifies who will benefit from BiDil.

The issue crystallized during the FDA hearing on BiDil in a debate between two Advisory Committee members. Vivian Ota Wang from the National Institutes of Health’s National Human Genome Research Institute challenged the use of race in the A-HeFT trial as a proxy for an underlying biological trait that explained how BiDil worked. “There is a presumption here that somehow this self-identified social identifier is somewhat equivalent or representative of a biological process, and I am not sure it really is,” Ota Wang pointed out. “We need to real-

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14. Id.

15. Transcript of Department of Health and Human Services Food and Drug Administration Center for Drug Evaluation and Research, Cardiovasc-
ly carefully look at the issue of self-identified racial categories because if the assumption is that these population differences are biological, the self-identified population is a social and political construct.”\textsuperscript{16} The Committee Chair, Cleveland Clinic cardiologist Steven Nissen, dismissed Ota Wang’s concerns. “We are using self-identified race as a surrogate for genomic-based medicine and I don’t think that is unreasonable.” Nissen said.\textsuperscript{17} “I wish we had the genetic markers . . . to decide who is going to respond to what drug but, in the absence of that, we have to use the best available evidence, . . . and that evidence was used in this trial and it worked.”\textsuperscript{18} Later, Dr. Nissen more bluntly reiterated, “We’re using self-identified race as a surrogate for genetic markers.”\textsuperscript{19} Nissen dismissed the worry that this rationale for approving a race-specific drug would reinforce a genetic definition of race by asserting, “Drugs aren’t racist; people are.”\textsuperscript{20}

Not only was the racial indication scientifically flawed, but race became a reason for lowering the FDA’s scientific standards. In 1989, Dr. Cohn obtained a patent on a “method of reducing mortality associated with congestive heart failure.”\textsuperscript{21} The patent made no mention of race. In 1996, the company he licensed the rights to submitted a New Drug Application for BiDil to the FDA. Like Cohn’s patent application, this application for marketing approval did not mention race. Its evidence of the drug’s efficacy consisted of a retrospective analysis of clinical trials Cohn conducted in the 1980s. The FDA Advisory Committee that reviewed the application was not convinced.\textsuperscript{22} The issue was not BiDil’s effectiveness; it was the FDA’s statistical standards. The Advisory Committee found that the reanalysis of old data, which were not collected to test a new drug for FDA approval, failed to meet the narrow criteria for statistical

\textsuperscript{16}. Id. at 357.
\textsuperscript{17}. Id. at 355 (statement of Dr. Steven Nissen).
\textsuperscript{18}. Id. at 360.
\textsuperscript{20}. Committee Transcript, supra note 15 at 354 (statement of Dr. Steven Nissen).
\textsuperscript{21}. U.S. Patent No. 4,868,179.
\textsuperscript{22}. Temple & Stockbridge, supra note 13, at 59.
significance. Another problem was that BiDil would be prescribed as an adjunct to standard ACE inhibitor and beta blocker therapies, therapies that were not combined with the BiDil ingredients in the earlier trials. Consequently, the FDA denied approval in 1997.

During the 2004 FDA hearing on Cohn’s application to market BiDil as a race-specific drug, the question of statistical standards was raised again. Ota Wang and Nissen also differed on the standards the FDA should use to evaluate BiDil. Nissen took the position that the remarkable improvement in health experienced by the A-HeFT patients on BiDil should outweigh concerns about the statistical strength of trial data. “I have to approve a drug when I think there’s evidence you can reduce mortality by 43 percent,” he said, “As a clinician, I find the evidence more than adequate to vote for approval.” Comparing heart failure among African Americans to an “orphan disease,” Nissen argued that “you make some adjustments sometimes because you want to encourage trials in special populations and diseases which are of public health importance which we have few therapies for.” In other words, to Nissen, heart failure suffered by African Americans was a special type of illness that warranted exceptions to the rules, as in the case of rare medical conditions.

Ota Wang objected to the “notion that for some types of research, for some types of communities or populations we can actually lower the bar in terms of scientific integrity that we are using to evaluate the research.” In response, Nissen reiterated the orphan disease analogy. “So, if you are developing a drug for a disease and there are not many people that have it, you get some points for doing that.” he said. “I am arguing that it is not unreasonable public policy to make some adjustment

23. Id.
27. Committee Transcript, supra note 15, at 304 (statement of Dr. Steven Nissen).
for that.”28 Statistical weaknesses in the data that ordinarily posed problems for FDA approval were overlooked because BiDil was a drug for black people. Apparently, Dr. Nissen was not referring to heart failure in general, a common ailment that is far from an “orphan” disease, but to black people’s heart failure. The very issues about statistical data that led the FDA to deny approval for BiDil when it was for the general population were now overlooked because BiDil had become a race-specific drug.

Perhaps the overwhelming evidence from the A-HeFT trial that BiDil was beneficial for many patients was a compelling reason to discount the statistical concerns and make it widely available on the market. The stunning trial results gave the FDA grounds to approve the drug without insisting on its statistical rules or further investigation of why BiDil worked so well, but what the A-HeFT trial did not do was give the FDA grounds to base its decision on race. We should be concerned that the FDA’s acceptance of race-specific drugs will turn into a rationale for “lowering the bar” of the scientific standards the agency usually applies in evaluating new drugs.

III. RACE-SPECIFIC MEDICINE IS COMMERCIALY MOTIVATED

A second problem with racial indications for pharmaceuticals is that they are guided by the market and not by science. Race is becoming a niche market that gives pharmaceutical companies an opportunity to extract new profits from existing drugs. Again, BiDil provides an illustration.

Recall that the FDA denied Cohn’s original application to market BiDil without regard to race. With his original race-neutral patent due to expire in less than 10 years, Cohn needed a strategy for salvaging his pharmaceutical venture. Cohn’s second chance came from re-conceiving BiDil as a race-specific drug.29 It was only after the FDA rejection that Cohn turned BiDil—the exact same drug that he had patented without regard to race—into a therapy for African Americans. Cohn submitted a new patent for BiDil with a critical difference from the original one. The new patent added the key language: the “present invention provides methods for treating and preventing mortality associated with heart failure in an African Amer-

28. Id. at 305–06.
29. Kahn, supra note 11, at 3.
The new patent, issued in 2000, lasts until 2020, buying thirteen more years of intellectual property control over the drug.

NitroMed’s success at using race to gain FDA and patent approval, as well as support from influential political players, signaled the potential profitability of race-specific drugs. Legal scholar Jonathan Kahn argues that BiDil’s racial labeling gave the pharmaceutical industry “a new model of how to exploit race in the marketplace by literally capitalizing on the racial identity of minority populations,” providing “a cheaper, more efficient way to gain the US Food and Drug Administration’s approval for drugs.”

Supporting his view is evidence of the growing use of race as a genetic category to obtain patent protection. Using the U.S. Patent Office database, Kahn reviewed gene-related patent applications filed between 1976 and 2005 that employed racial or ethnic categories. Kahn discovered a five-fold increase in racial patents during that period. Race was not mentioned in any application filed in 1976–1997. From 1976–2005 twelve gene-related patents were issued using race or ethnicity, and from 2001–2005 sixty-five gene-related patents applications were filed using race or ethnicity.

Using racial categories to patent an invention and to carve out a racially-defined market for it is nothing new. There is a long history, dating back to the 1800s, of patenting all sorts of products that involve race: chemicals to straighten kinky hair, creams to lighten dark skin, and toys that celebrate or mock people of color. For example, a patent from 1940 for an arcade game featured a figure of a “negro stealing a chicken” as a target. “As soon as the target is initially moved, with the negro moving toward the hen house, a successful hit will cause him to reverse his direction of movement and leave the hen house,” the

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31. Kahn, supra note 26, at 737–38 (2008); see also Jonathan Kahn, The Politics of Patenting Race, 20 GENE WATCH 3, 3 (2007) (arguing that BiDil is serving as a model for the “strategic use of race as a genetic category to obtain patent protection and drug approval.”).
33. Id.
35. Id.
inventor explained. The Civil Rights Movement ushered in patents for dolls, games, and teaching materials that celebrated diversity, ethnic holidays, and civil rights leaders. A more recent patent filed in 2006 for a device that quickly removes natural or synthetic braids that have been attached to human hair claims to benefit African Americans because they “genetically have hair that resists the formation of longer lengths.”

Race and ethnic heritage are used by patent applicants in a variety of ways, some more harmful than others. The problem with recent gene-related patents is that they treat race as a biological category. Treating racial identity as a component of a genetic commodity further solidifies the view that race is biological. “The patent process takes race as a social category and recodes it as ‘natural,’” Kahn writes. The growing number of biomedical studies and patents that rely on race suggests that biotech companies are poised to launch a new generation of racial pharmaceuticals.

It would be naïve to believe that, given our market-driven system, biomedical research can proceed without private funding and without converting discoveries into marketable products. But it is equally naïve to ignore the influence of pharmaceutical money on the way biomedical research is conducted. The A-HeFT researchers surely were motivated by a desire to alleviate the suffering of black heart failure patients, but their ties to the pharmaceutical industry helped to steer their path to a cure.

My quarrel with the commercial aspects of BiDil’s development is not that the people involved made money. Congress has ensured that profit is the central incentive for the pharmaceutical industry to research and market medications. In his commentary on my lecture, Jay Cohn conceded that “there’s a commercial benefit to this—of course. How else do drugs get developed in this country except on the basis of commercial potential? So if you want to criticize that, you can criticize the entire economic strategy in America and maybe Dorothy would like to do that.” It is one thing for biomedical researchers and

36. Id. at 441.
37. Id. at 431.
pharmaceutical companies to profit from scientific innovation; it is quite another for the profit motive to steer the science that is being innovated. Commercial interests induce pharmaceutical companies to exaggerate or invent the therapeutic importance of race. NitroMed did not make money from a drug that was developed to treat heart failure in black patients. It made money by converting a drug for heart failure into a drug for African Americans based on unscientific claims about racial difference.

IV. RACE-BASED MEDICINE IS POLITICALLY DANGEROUS

Despite these criticisms, is it not a good thing that BiDil is on the market? Supporters of race-specific drugs counter the scientific and commercial challenges I discuss by arguing that these drugs are critical to advancing health in two very important ways: they are a step toward developing personalized medicine, and they are immediately addressing health disparities based on race.40

I argue that it is precisely these two claims that make race-based medicine not only scientifically flawed but politically dangerous. By reinforcing a biological definition of race and cure for health disparities that are false, race-based medicine supports a new biopolitics of race that threatens to make health and other social inequalities even worse.

For the last decade, genetic scientists have promised to soon develop personalized medicines that will enable doctors to predict, diagnose, and treat illnesses according to each patient’s own unique genome.41 Researchers in the field of pharmacogenomics, which studies the genetic origins of disease and differential responses to medications, are trying to develop “tailored” drugs that are safer and more effective than conventional medicines. But a decade after completion of the Human Genome Project, researchers have failed to discover the genes that cause common diseases, which were predicted to enable the development of gene-targeted medicines.42 “There is

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40. Kahn, supra note 26, at 748.
42. Nicholas Wade, A Decade Later, Genetic Map Yields Few Clues, N.Y.
absolutely no question that for the whole hope of personalized medicine, the news has been as bleak as could be,” summed up molecular biologist David Goldstein, director of Duke University’s Center for Human Genome Variation, in September 2008. Despite statistically linking hundreds of common variants to various diseases, scientists discovered that they account for only a tiny fraction of the genetic risk. Instead, most common diseases are caused by a host of rare genetic variants that evade detection by genome-wide association studies.

Despite the lack of genetic data—or perhaps because of it—race has become the magic fix to bridge the gap between the promise and disappointment of personalized medicine. Pharmacogenomic researchers treat race as a crucial first step to producing designer drugs because, they argue, race can serve as a proxy for individual genetic difference. Until science is able to match therapies to each individual’s unique genotype, race functions as a handy surrogate.

The FDA’s press release announcing its approval of BiDil stated that the decision “represent[ed] a step toward the promise of personalized medicine.” But what did BiDil have to do with personalized medicine? It was not a drug designed for black people at all, let alone one tailored to match some race-based genetic difference. It was developed to treat heart failure regardless of race and regardless of genetics. Yet despite having nothing to do with pharmacogenomics, “the step toward personalized medicine” claim became one of the leading rationales for race-based medicine. In FDA Week, Michael Warner, a former regulatory affairs specialist for the Biotechnology Industry Organization, falsely asserted that “BiDil is the first time, the highest profile time, the model of ‘let’s identify a target population and let’s develop a drug for that population’ has been pursued.”

A second basis for defending race-based pharmaceuticals is the claim that their health benefits outweigh their power to reinforce race as a biological category. Prominent African American scientists, doctors, and advocates endorsed BiDil to

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43. Nicholas Wade, A Dissenting Voice as the Genome is Sifted to Fight Disease, N.Y. TIMES, Sept. 16, 2008, at F3.
44. Wade, supra note 42.
45. Temple & Stockbridge, supra note 13, at 58.
47. Kahn, supra note 26, at 740.
redress past discrimination against African Americans in medical treatment and access to health care.48 Ever since their enslavement in the United States, African Americans have been victims of both medical abuse, such as the infamous syphilis study in Tuskegee, Alabama, and medical neglect.49 BiDil supporters argued that a race-specific drug fulfilled a longstanding demand that science attend to the particular needs of African Americans whom historically had been excluded from good medical care and clinical trials while suffering disproportionately from heart disease. Representative Donna Christenson implored the FDA to approve BiDil as a remedy for medical wrongs against African Americans “for whom treatment has been denied and deferred for 400 years.”50

Many used the health disparities as a reason to ignore the scientific flaws in race-based medicine. Gary Puckrein, executive director of the National Minority Health Month Foundation, has championed BiDil as an important response to high rates of heart disease among African Americans. Although he acknowledged “[c]oncern about the medical and scientific validity of the concept of race,” he dismissed such concern as “under present circumstances, impractical.”51 Similarly, Keith Ferdinand, chief science officer of the Association of Black Cardiologists, wrote that “race lacks any true biologic definition,”52 but BiDil is a “life-saving drug”53 that addresses “evidence of racial and ethnic differences in cardiac care in the United States which may significantly affect health outcomes.”54 In other words, these BiDil advocates argue that the urgency of addressing the African American health crisis with race-specific drugs overrides objections that race is a social and not a genetic grouping.

49. See, e.g., HARRIET WASHINGTON, MEDICAL APARTHEID 6 (2006).
53. Id.
54. Id. at 459.
When I stated at an April 2006 conference on race-based therapeutics at the Massachusetts Institute of Technology that there was no consensus among African Americans on the benefits of these pharmaceuticals, Juan Cofield, president of the National Association for the Advancement of Colored People (NAACP) New England branch, stood up in the audience and objected. “There is a consensus supporting BiDil,” he stated, “The NAACP supports it, the Association of Black Cardiologists, and the Black Congressional Caucus supports it.” “There isn’t even a consensus on BiDil among the black people in this room,” I replied. That may have elicited laughter from the audience, but it did not assuage Cofield. “Young lady,” he responded, “you are jeopardizing the lives of black people.” According to Cofield, I had no right to suggest that blacks are not united behind promoting racial therapeutics.

Upon further investigation, I discovered that in December 2005, the NAACP announced its partnership with NitroMed “to implement measures to narrow health disparities that exist between African Americans and Caucasians . . . .”55 As part of this alliance, NitroMed promised the NAACP a three-year $1.5 million grant.56 In return, the NAACP vigorously promoted BiDil in black communities as a life-saving drug for African Americans.57 NitroMed’s Chief Executive Office, Dr. Michael Loberg, described of the partnership’s chief aims as “together with the NAACP . . . doing our part to remove all barriers to access to BiDil.”58

This was not the first time I had heard this accusation. At a 2005 University of Minnesota race in biomedical research conference, Jay Cohn objected to my legal and moral concerns about labeling drugs by race. Another speaker, Georgetown bioethicist Gregg Bloche, although also opposed to racial labeling, asked how I could justify sacrificing the lives of black grandmothers merely to avoid classifying people by race.59 I
was shocked four years later to hear Jay Cohn on a Radio Lab broadcast say, “And there was a very well known law professor who said, ‘I would rather die from heart failure than take Bi-Dil!’” (referring to my talk at the conference). Fortunately, the host intervened to say, “Well, that’s not quite what she said,” and played my actual words: “I would be terrified about a doctor making a diagnosis like that based on his view of me as belonging to a particular racial category.”

Again, in his commentary on my 2009 lecture, Dr. Cohn charged that “the hostility that has grown in the community against this therapy has impaired its use, and, therefore, the vast majority of blacks with heart failure are not receiving life-prolonging therapy.”

These activists and researchers with ties to the pharmaceutical industry try to stifle criticism of racial medicine by portraying objections as roadblocks to African Americans’ access to lifesaving treatment. They imply that objecting to race-specific medicine is tantamount to denying black patients the medicine they need. Behind this argument is the false assumption that it is impossible to develop drugs that benefit blacks without classifying people by race.

It is unfair to accuse people who oppose racial labeling of trying to keep lifesaving drugs from dying patients. No critic of race-specific medicine seeks to deny lifesaving drugs to African American people. We never argued that BiDil should be withheld from the market. Just the opposite is true: we argued that if it were to be marketed, it should be made more widely available—without regard to race. We simply see no justification for marketing medicines according to race and worry about their potential to divert attention away from more significant social reasons for health disparities. Studying and eliminating the social determinants of health inequities is a far more promising course than searching for race-specific genetic differences.

Portraying BiDil as a solution to a racial gap in mortality implies the gap stems from racial differences in disease and drug response. Adding a genetic explanation for this difference

61. Id.
62. Cohn, supra note 39.
63. Puckrein, supra note 51, at w368.
64. See Jonathan Kahn & Pamela Sankar, Being Specific About Race-Specific Medicine, 25 HEALTH AFF. w375, w375 (2006).
attributes health disparities to flaws inside black people’s bodies rather than to flaws in the society they live in. It supports the increasingly popular but misguided view that the tiny percentage of genetic difference among human beings is distributed by race and that this difference creates inequities in health.

In his commentary on my lecture, Dr. Cohn objected that I raised “concerns that all of us have about inequality of healthcare that BiDil was never developed to address.” Although Cohn belatedly protests the insertion of health disparities into the debate about BiDil, it was BiDil advocates who first relied on the mortality gap between blacks and whites to gain support for the drug and who painted BiDil was a response to centuries of discrimination against African Americans. As Susan Reverby has observed, the “shadow of Tuskegee” hung over the FDA hearing and put pressure on the advisory committee to approve BiDil as a therapy for black people.

While listening to African American advocates for race-specific medicine, the FDA Advisory Committee ignored the evidence presented by Dr. Charles Rotimi, a researcher at Howard University’s Human Genome Center, that showed high rates of hypertension among blacks stemming from environmental rather than genetic causes. To Dr. Rotimi, it made no sense to conclude that blacks are “selectively acquiring bad genes” for the numerous conditions marked by racial gaps in mortality. It is implausible that one race of people evolved to have a genetic predisposition to heart failure, hypertension, infant mortality, diabetes, and asthma. There is no evolutionary theory that can explain why African ancestry would be genetically prone to practically every major common illness. “There must be something in our social environment that drives people toward poor health, and only by addressing that can we reduce health disparity,” Dr. Rotimi concluded.

Many scientific studies that show that racially unequal

65. Cohn, supra note 39.
68. Committee Transcript, supra note 15, at 212 (statement of Dr. Charles Rotimi).
69. Id. at 242.
70. Id. at 212.
health outcomes stem from unequal social conditions support Dr. Rotimi’s views.\textsuperscript{71} Some, like a study of racial gaps in breast cancer mortality in Chicago, show that the geographical concentration and historical changes in racial health disparities could not possibly stem from genetic difference.\textsuperscript{72} White women in Chicago are slightly more likely than black women to get breast cancer.\textsuperscript{73} Black women are twice as likely to die from it.\textsuperscript{74} That is a startling statistic by itself. But what is equally shocking is that Chicago’s black and white breast cancer mortality rates were identical in 1980. The astounding gap emerged over the course of the next twenty-five years.\textsuperscript{75} The most likely explanation is that black women did not have access to the technologies and therapies that lowered white women’s cancer mortality rate.\textsuperscript{76} Moreover, the disparity in breast cancer mortality in New York City is only fifteen percent, making the racial gap in Chicago ten times greater than in New York—a disparity unexplained by genetic difference.\textsuperscript{77}

Dr. Richard Cooper’s global comparison of hypertension similarly refutes a genetic explanation for race-based health inequities. His study revealed that blacks in Nigeria and Jamaica have rates of hypertension similar to that of whites in the United States and much lower than that of African Americans.\textsuperscript{78} Perhaps Nigerians and African Americans are genetically prone to high blood pressure, but there is something in the environment that is causing elevated rates in this country. But that is just the point: if our goal is eliminating the gap between white and black hypertension in the United States, our focus should be on the social causes of the gap. Continuing to hunt...


\textsuperscript{73} Id.

\textsuperscript{74} Id.

\textsuperscript{75} Id.

\textsuperscript{76} Id.

\textsuperscript{77} Id.

for a genetic component of racial differences only distracts us from the more relevant issue of identifying and tackling the preventable causes of hypertension, which have a similar impact regardless of race. This approach would help everyone who lives in conditions that cause high rates of hypertension.

Perhaps the most powerful evidence of the importance of social determinants is the relationship between an entire nation’s health and its level of inequality. Numerous studies tracking the health of people along the social ladder show that health gradually worsens as socioeconomic status, including race, declines.79 In their recent book, The Spirit Level, Richard Wilkinson and Kate Pinkett present remarkably consistent evidence of “a very strong tendency for ill-health and social problems [including life expectancy and infant mortality] to occur less frequently in the more equal countries.”80 To put it another way, “Health and social problems are indeed more common in countries with bigger income inequalities.”81 People in Japan, Sweden, and Norway live longer, are less obese, and have fewer teenage births than people in the United States, the United Kingdom, and Australia because their societies are more equal.82 The United States is unhealthier, despite spending far more money on genetic research and drugs. The reason is not that the United States has not done enough genetic research. It is because the United States has more social inequality and has not done enough to eliminate it.

Instead, attention has turned sharply toward the possible genetic reasons for drug effectiveness and health disparities. The NitroMed research team began searching in 2005 for the


81. Wilkinson & Pickett, supra note 80, at 20.

82. Id.
gene that explained how BiDil worked—after it was approved as a race-specific drug. The Genetic Risk Assessment in Heart Failure Trial (GRAHF) compared the frequency of aldosterone synthase (CYP11B2) alleles in 354 patients who participated in A-HeFT to their frequency in white participants in the Genetic Risk Assessment of Cardiac Events (GRACE) study conducted at the University of Pittsburgh.\textsuperscript{83} Activation of aldosterone appears to hasten the progression of heart failure. The GRAHF study found that the -344 T/C promoter polymorphism of CYP11B2 influenced clinical outcomes in the African American patients and that it was more common in African American patients than in the white patients who participated in GRACE. The authors observed that these findings “suggest that the genetic variation in aldosterone production may contribute” to differences in heart failure rates between blacks and whites. “In determining optimal heart failure treatment for an individual, race is likely a surrogate marker for differences in genetic background,” they concluded.\textsuperscript{84}

By comparing genotypes in black and white patients, the researchers seemed stuck on finding a biological mechanism based on race. NitroMed’s Vice President of Corporate Affairs stated in 2007 that the company might eventually use the genetic data to develop a diagnostic test for BiDil, though it is not clear what financial incentive it had to invest in genetic screening if it could use race instead. NitroMed researchers had a vested interest in finding racial differences that could justify FDA approval. By leaping to a genetic explanation, they foreclosed a potentially more fruitful investigation of the environmental factors that separate white and black health—and that could improve prevention of heart disease for everyone.

Researchers still do not know why blacks get and die from heart failure at a much earlier age than whites.\textsuperscript{85} But if I were a scientist, I would start looking at the effects of young black men’s seventy-five per cent chance of being incarcerated in some cities.\textsuperscript{86} Heart disease researchers should be more inter-

\textsuperscript{83} Dennis M. McNamara et al., Aldosterone Synthase Promoter Polymorphism Predicts Outcome in African Americans with Heart Failure: Results from the A-HeFT Trial, 48 J. AM. C. CARDIOLOGY 1277, 1277 (2006).
\textsuperscript{84} Id. at 1281.
\textsuperscript{85} Roni Caryn Rabin, Blacks Suffer Heart Failure More than Whites, Study Finds, N.Y. TIMES, Mar. 19, 2009, at A15.
\textsuperscript{86} Michelle Alexander, The New Jim Crow: Mass Incarceration In
ested in the fact that black men are seven times more likely than whites to be imprisoned in this country than the less significant genetic differences many are so fixated on. Attributing health inequities to genetic difference is part of a broader trend, what I call a new biopolitics of race, that is focusing on race at the molecular level while discounting its impact on society. At the other end of the political spectrum from the African American advocates I quoted earlier, are conservatives who claim that racial differences are real at the genetic level and also charge their critics with relying on political ideology rather than science. They argue that race is a natural category that became politicized only in the last few decades because of post-civil rights identity politics. This ignores the real origins of racial classifications that accommodated European, and later American, imperialism and slavery—the quintessential example of using science to achieve political ends. Conservatives point to racial medicine as scientific confirmation of racial differences that liberals have denied in order to be politically correct.

Sally Satel, a fellow at the American Enterprise Institute, has long defended the use of race in medical practice in response to biological differences between members of different racial groups. At a 2004 American Enterprise Institute Symposium entitled, Race, Medicine, and Public Policy, she concluded, “It is evident that disease is not colorblind, and therefore doctors should not be either.” Not surprisingly, Satel supports

87. Id. at 98.
89. See, e.g., Sally Satel, PC, M.D.: How Political Correctness Is Corrupting Medicine 6 (2001) (“Though activists appear to be waging “the good fight” for better health through social justice, their actions do not prevent disease . . . . At best they create distractions and waste money; at worst, they interfere with effective treatment.”); Sally Satel & Jonathan Klick, The Health Disparities Myth: Diagnosing the Treatment Gap 12 (2006) (“[M]inority health was transformed from a public health issue to a civil rights issue.”).
90. See Satel, supra note 89.
91. Race, Medicine, and Public Policy, American Enterprise Institute,
race-specific pharmaceuticals. “Social race is the phenomenon constructionists have in mind . . . . Biological race, however, is what BiDil’s developers are concerned with—that is, race as ancestry.”92

According to this view, racial differences are real at the molecular level and merely constructed in society; therefore, doctors and researchers cannot be colorblind, but social policy should be. Genomic science, these conservatives argue, now gives people license to act on biological differences between races to better understand their health and identities. In this ingenious twist of political logic, those who criticize racial medicine because of its social impact are seen as interfering with health on the basis of racial ideology.

A renewed trust in inherent racial differences provides a convenient but false explanation for persistent inequities despite the end of de jure discrimination. It is also the perfect complement to social policies that implement the claim that racism has ceased to be the cause of African Americans’ unequal status. Race consciousness in social programs like affirmative action is under assault at the very moment that race consciousness in medicine is ascending. As Chief Justice Roberts stated in one of several recent Supreme Court decisions chiseling away at government’s use of race to address institutionalized inequality, “The way to stop discrimination on the basis of race is to stop discriminating on the basis of race.”93

There is a long history of using a biological definition of race to make social inequities seem natural—the result of inherent difference instead of societal injustice. As Evelynn Hammonds has noted, “[T]he appeal of a story that links race to medical and scientific progress is in the way in which it naturalizes the social order in a racially stratified society such as ours.”94


94. Evelyn M. Hammonds, Straw Men and Their Followers: The Return of Biological Race, IS RACE REAL? (June 7, 2006),
While the racial gap in life expectancy widens,\textsuperscript{95} owing largely to the government’s failure to address structural inequities, the poor health of African Americans opens new markets for pharmaceutical companies. The claim that race-based biotechnologies will shrink the gap based on genetic difference is a powerful way to deflect concerns about their unjust social impact and the social inequality that actually drives poor minority health. We should be against an approach that promotes individual health through technological cures as a way of ignoring larger social inequities. This view sets up a false dichotomy between health and social justice: it treats health and justice as opposing values, weighs them against each other, and declares health the winner. It hides the social factors that determine health not only for individuals but for the entire nation. Letting health trump social justice does not really improve the welfare of most people; it supports the interests of big business and the most privileged members of society.

The promotion of race-based medicine misrepresents the relationship between genes, drugs, and health disparities. Of course, pharmaceuticals can help improve sick people’s health and effective pharmaceuticals should be available to people who would benefit from them. But health inequities are not caused by genes and cannot be eliminated with drugs. Promoting race-based medicine with the myth that poor minority health is caused by genetic difference will only widen the gap, diverting us from the real solution. It makes no sense to put aside social justice concerns in order to improve minority health. A more just society would be a healthier one.

\footnote{http://raceandgenomics.ssrc.org/Hammonds/}.