

# **PLAYING THE GENE CARD?**

A REPORT ON RACE AND  
HUMAN BIOTECHNOLOGY

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## Chapter 1

# Race-Based Medicine: One Step Forward, Two Steps Back?

It is well known that people often have different reactions to medications. In most cases, the causes of these differences are unknown, but they may be connected to subtle variations in individuals' DNA. Efforts to prescribe the right medication for each patient's genome, to custom-tailor therapies for patients with a particular genetic makeup, are known as "personalized medicine" and considered by many one of the great promises of modern biology.

This promise of personalized medicine, however, has barely begun to be realized. While there are limited examples where drugs can be tailored to individual genotypes, genetic knowledge is not yet robust enough to do this on a large scale. Nevertheless, pharmaceutical companies are beginning to develop drugs that claim to be tailored for a specific racial group, otherwise known as race-based medicines. Such medicines are based upon the idea that specific genetic variations that are most common within particular racial populations explain certain health outcomes and disparities.

The first race-specific drug was BiDil, approved in 2005 by the FDA to treat African Americans suffering from heart failure. Marketed by the biotechnology company NitroMed as a way to address what were perceived as racial disparities in heart failure, BiDil quickly became the poster child for revamped efforts to approach

race not merely as a social category, but as a genetically relevant mechanism for understanding human difference and medical outcomes.

This interest in race-based medicines is part of a broader trend, most notably articulated by doctors such as Sally Satel who believe racial profiling in medicine is good, or even necessary.<sup>44</sup> For Satel and others, social categories of race are useful proxies for understanding underlying genetic variations that may be unique to certain racial populations—even when such variation is known to be relatively small.<sup>45</sup> From this perspective, race-specific therapies "illuminate the future of medicine."<sup>46</sup>

Despite this enthusiasm and the supposed benefits for minority health care, the story of BiDil is a cautionary tale that raises a number of important questions:

- Is it reasonable to assume without specific evidence that genetic variations, which can play a substantial role in individuals' drug response, can be meaningfully grouped by social categories of race?
- How might lingering biological theories of race influence well-intentioned research agendas?
- Is race-specific medicine the best way to use limited resources to address racial disparities in health?

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## Major Projects on Human Genetic Variation

- **The NIH Pharmacogenetics Research Network** examines the less than 1% of human genetic difference to explore how tiny variations might underpin group differences in disease susceptibility and drug response.<sup>47</sup>
- **The Human Genome Diversity Project** (now defunct) tried to use genetic data from indigenous groups around the world in order to examine human genetic diversity.<sup>48</sup>
- **The International HapMap Project** compares the genetic sequences of individuals with African, Asian, and European ancestry to catalogue genetic differences and similarities that may help find genes linked to certain diseases or that affect drug response.<sup>49</sup>
- **The NIH Center on Genomics and Health Disparities**, launched in March 2008, promises to devote substantial resources to using genomics to understand health disparities across different populations. The Center’s director, Dr. Charles N. Rotimi, notes that “the priority of our center will be to understand how we can use the tools of genomics to address some of the issues we see with health disparities.”<sup>50</sup>

These efforts do have scientific merit. Many researchers hypothesize that the less than 1% of variation in DNA might be relevant to racial disparities in health outcomes. The tiny difference among individuals’ shared three billion base pairs corresponds to up to 15 million genetic dissimilarities; these may correspond to genetic variations that are linked to ancestral evolutionary dynamics in a manner that can be proxied by individuals’ outward appearance, or phenotype.

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Before delving into these questions, it is necessary to have a brief understanding of the underlying scientific concepts used to support not only claims about the propriety of race-based medicines, but also other claims linking race and racial outcomes to genetic difference.

### Pharmacogenomics: The Concept Behind Race-Based Medicines

The Human Genome Project (HGP) revealed that humans have between 20,000 and 25,000 genes, many fewer than was once thought. The completed sequence can now identify their locations; further research is likely to shed greater light on how these genes work.

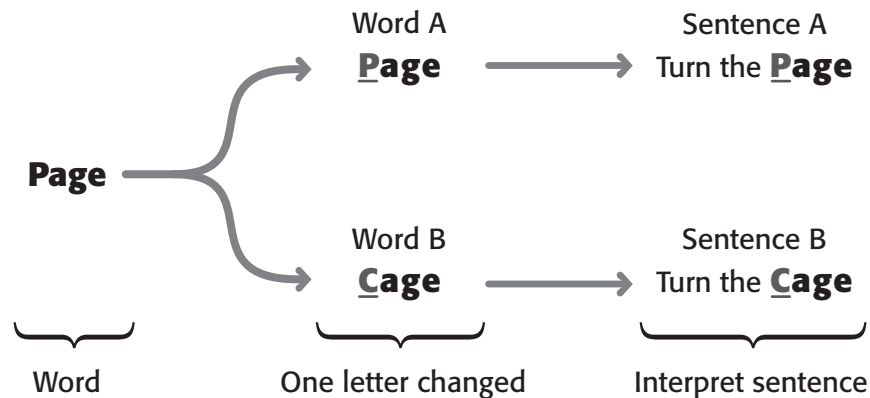
Individuals’ genetic sequences are remarkably similar. When two people’s chromosomes are compared, their DNA sequences can be identical for several hundred bases.<sup>51</sup> But the sequences will differ at about one in every 1,200 “letters”; one person might have an “C” (cytosine) at a given location while another person has a “T” (thymine), or a person might miss part of a DNA segment at any given point or have extra bases.

Each unique “spelling” in a chromosomal region is called an *allele*, while the collection of

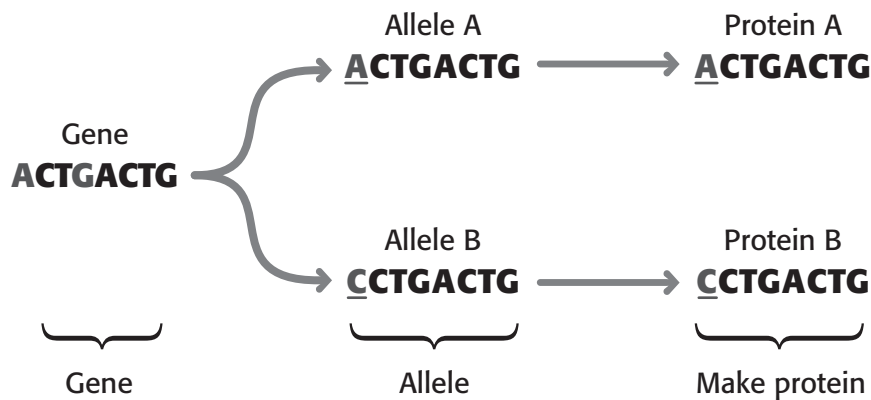
alleles in a person’s chromosomes is called a *genotype*. This is often contrasted with *phenotype*, which is a person’s outward characteristics resulting from their genes’ interaction with the environment during development. For example, identical twins have the same genotype but their phenotypes differ, though sometimes only slightly.

Pharmacogenomics is a biomedical field that studies how these different spellings, or genetic variations, might affect which drugs are most effective for particular genotypes. (See Figure 1, on page 9, and “Why Genetic Variations Matter,” on page 10.) Knowing that, researchers hope to be able to predict which patients will respond best to certain medications.

Pharmacogenomic research into which genetic variants correlate with drug response or disease susceptibility coupled with population geneticists’ research into which haplotypes correlate with particular ancestries—what many scientists and laypersons closely associate with “race”—are slowly but surely moving biomedicine in the direction of developing treatments that use race and ancestry as *proxies* for groups’ genetic predispositions.<sup>52</sup> In other words, race-based medicine works from the premise that social categories of race defined largely by pheno-



**A** Changing one letter in a word.



**B** Changing one base in a gene sequence.

**Figure 1 Grammatical analogy.** (A) A one-letter variation can change the meaning of a word or affect its meaning in a sentence. (B) Similarly, a one-letter variation in a gene sequence can affect its meaning and the proteins that are produced. The different protein may also have further consequences, such as affecting a person's susceptibility to certain diseases or their response to certain drugs. (Image based upon work by Esteban González Burchard.)

type or self-identification can “stand in” for specific genetic differences between races that have yet to be found—and may never be.

### First on the Scene: BiDil

The FDA's approval of NitroMed's BiDil in June 2005 as a treatment for African Americans with heart failure was the first time that regulatory approval had ever been given to a drug specified only for one racial group.

Five million Americans currently suffer from heart failure.<sup>53</sup> Medical literature and popular media frequently repeat the claim that Blacks die from heart failure twice as often as their White counterparts. This two-to-one disparity has been shown to be misleading,<sup>54</sup> but it has nevertheless provided the moral, scientific, and commercial justifications for a race-specific approach to treating Black heart failure. The National Association for the Advancement of Colored People, the

## Why Genetic Variations Matter

The “letters” or base pairs in a genetic sequence make up “words” (in this analogy, genes) that instruct cells to make proteins that allow them to perform their assigned functions. These genetic sequences contain information that might influence physical traits, predisposition to disease, and responses to environmental influences.

The misspelling of one letter can change a word’s connotation and thus how it functions in a sentence to convey meaning, as shown in Figure 1A. This is no less true for genes, as shown in Figure 1B.

The most common types of genetic variation are these alternate spellings in individual base pairs, which affect whether and how certain proteins are made. These genetic differences are called *single nucleotide polymorphisms*, or SNPs (pronounced “snips”). Several million have been identified, but the total number is not known. Some of these differences seem to be immaterial or compensated for elsewhere; others can be critically important.

In addition, SNPs can be used as markers to identify and find particular genes in sequences of DNA. For example, a “spelling change” in a gene might increase the likelihood that a person suffers from asthma, but researchers might not know its location on a chromosome. They might be able to compare the SNPs in people who suffer from asthma with those of people who do not. If they find a particular SNP that is more frequent among asthma sufferers, that SNP could be used as a marker to locate and identify genes that may influence this outcome. As the International HapMap Consortium notes, “systematic studies of common genetic variants are facilitated by the fact that individuals who carry a particular SNP allele at one site often predictably carry specific alleles at other nearby variant sites. This correlation is known as linkage disequilibrium; a particular combination of alleles along a chromosome is termed a haplotype.”<sup>55</sup>

Association of Black Cardiologists, and other organizations have supported BiDil as an effective way to curb the perceived disparity in heart failure between Blacks and Whites.<sup>56</sup>

The story of BiDil’s clinical development goes back many years. The original patent, which did not mention race, was submitted in 1987.<sup>57</sup> Even then, BiDil was not entirely new; rather, it combined two generic drugs (hydralazine and isosorbide dinitrate) into one pill.

This is not to underestimate BiDil’s potential contribution to treating heart failure; simplifying administration can increase the likelihood that patients will use prescription drugs correctly and thus optimize benefits. But it does draw attention to the curious fact that these particular drugs have been used to treat heart failure in all races for decades.

BiDil was put through the required clinical trials, but initially failed to receive FDA approval in 1997.<sup>58</sup> Only then, through a retrospective analysis of data from older clinical trials, did researchers begin to argue that the outcomes of Blacks taking BiDil were better than those of other racial groups. In 2002, after researchers published a paper highlighting these race-specific findings, the United States Patent and Trademark Office issued a patent for BiDil to treat heart failure in African Americans. This patent was subsequently assigned to the biotech firm NitroMed.

With this new patent in hand—and an extended thirteen years of market exclusivity—NitroMed amended BiDil’s failed application for FDA approval with a new clinical trial, called the African-American Heart Failure Trial, or A-HeFT. This study included only “self-identified” Blacks, and yielded astonishing results: adding BiDil to conventional heart failure therapy reduced one-year mortality by 43%. This finding, along with the oft-cited 2:1 racial disparity in heart failure mortality, fast-tracked BiDil for the FDA’s 2005 approval as the first race-specific medicine.

BiDil’s approval represented at least three different claims about the relevance of race to health care and health disparities. It was:

- the first drug to be patented as race specific (a *legal* claim about race and biology)
- the first to receive FDA approval as race specific (a *regulatory* claim about race and biology)
- the first to be marketed as race specific (an *economic* claim about race and biology)

BiDil represents an important step in framing racial difference as an indicator of significant genetic differences in human populations. Steven

Nissen (chair of the FDA Cardiovascular and Renal Drugs Advisory Committee that endorsed BiDil's approval) could not have been clearer in affirming this, noting that his committee took self-identified race in the A-HeFT studies "as a surrogate for genomic-based medicine."<sup>59</sup> In the absence of knowing the specific genetic markers that presumably correspond with BiDil's efficacy in some patients, the advisory committee concluded that self-identified race is a suitable stand-in for this genetic difference.

### Concerns about BiDil

Many ask, why not support BiDil, if it really helps African Americans who suffer from heart failure? The issue is that much of the evidence supporting this claim is not as convincing as it initially seems.

*African Americans are not twice as likely to die from heart failure as anyone else.* The statistic behind the moral impetus for a race-specific approach to treating Black heart failure—the 2:1 ratio—is not accurate. Legal scholar Jonathan Kahn, who followed the BiDil story very closely, traces this claim to a series of misquotes concerning what is now quarter-century-old data.<sup>60</sup> More recent data from the Centers for Disease Control puts the ratio at 1.1:1. Essentially, there is no difference in population-wide mortality between Blacks and Whites.

After this inaccuracy was brought to NitroMed's attention, the company amended its claim to say that "African Americans *between the ages of 45 and 64* are 2.5 times more likely to die from heart failure than Caucasians in the same age range." This is technically correct. Yet it fails to highlight a key point: the population aged 45 to 64 accounts for only 6% of heart failure mortality; after age 65—when most heart failure mortality occurs—the statistical difference evaporates.<sup>69</sup>

### Top-Down Marketing to the Black Community

*"NitroMed did what other pharmaceutical companies have always done. It gave money to people who later gave its medication the thumbs up."*<sup>61</sup>

NitroMed invested heavily in mainstream Black organizations to promote BiDil. It gave the National Association for the Advancement of Colored People \$1.5 million "to develop health advocacy initiatives towards equal access to quality healthcare."<sup>62</sup> The Association of Black Cardiologists was a co-sponsor of its clinical trials, and was paid \$200,000.<sup>63</sup> The company also gained the support of the Congressional Black Caucus.<sup>64</sup>

Analysts predicted sales of \$200 million in 2007 and potentially as much as \$825 million a year.<sup>65</sup> In practice, however, physicians and insurance companies were reluctant to spend the extra \$3000 a year that BiDil cost compared with the existing generic counterparts.<sup>66</sup> Sales for the first nine months of 2007 were only \$11 million, and in January 2008 the company announced that it was laying off most of its staff and suspending marketing of BiDil while still making it available.<sup>67</sup> In October 2008, NitroMed announced that it planned to sell all of its BiDil-related assets to JHP Pharmaceutical.<sup>68</sup>

These data undermine the claims about racial disparity upon which BiDil's supporters have based their moral argument. And given the robust research demonstrating that environmental and socio-economic factors such as poverty and lack of preventive health care worsen cardiovascular health outcomes, it is difficult to assert *a priori* that genes play a significant role in any population-wide disparities in heart failure that might exist.

*The clinical trial showing that BiDil is a race-specific drug had significant flaws.* The A-HeFT trial that propelled BiDil's FDA approval does not clearly support the claims of race specificity made by the drug's proponents. Those affiliated with the

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## Historical Theories of Race

Concepts of difference have been part of the human experience for millennia, as have prejudicial attitudes towards groups perceived to be physically different. During the taxonomic phase of biology, capped by Linnaeus in 1758, there were several attempts to categorize humanity into races; Linnaeus identified four.<sup>70</sup>

The 19th century ushered in more systematic attempts to give subjective prejudices an air of objective truth by using biological theories of race. Among those who tried were such notables as Georges Cuvier, who effectively established the discipline of paleontology, and Louis Agassiz, perhaps the leading biologist of his day, who identified twelve human races. Agassiz and others advocated for “polygenism,” the theory that human races had separate origins.

It is noteworthy that Charles Darwin was a “monogenist” who rejected race as a biological construct, having lived with South American natives and been struck by “how similar their minds were to ours.”<sup>71</sup> Nevertheless, he did suggest that stronger tribes would always eliminate the weaker, and what became known as “Social Darwinism” provided a foundation for racist investigation.

The development of eugenics by Francis Galton (1822–1911), who helped pioneer skull measurements and the statistical technique of correlation, was closely related to theories of race.<sup>72</sup> Among his many and varied efforts, Galton once advocated introducing “the Chinaman” to Africa, in order to “out-breed and finally displace the negro,” since “the Chinaman [has] a remarkable aptitude for a high material civilization.”<sup>73</sup>

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FDA have justified this trial design by noting that “the decision to conduct the trial in [only] black patients reflected careful analyses of 2 previous trials in racially mixed populations [V-HeFT I and V-HeFT II]. . . . Both trials showed little or no overall effect . . . in the mostly white patient population but hinted at substantial effect in subsets of black patients.”<sup>74</sup> They also note that conducting a full study within a mixed race population would have been an “unreasonable delay” in approving a drug for a group for which there is evidence of its benefits.

Any clinical trial that yields a 43% reduction in mortality is a stunning feat. Yet by only enroll-

ing self-identified Blacks, the trial strongly implies (and is indeed used to show) that it is *only* effective in African American populations. But this is not the case. Dr. Jay Cohn, the person who developed BiDil, has repeatedly noted that non-Blacks can receive a substantial benefit from the medication.<sup>75</sup>

Since patients other than African Americans were not included in the clinical trial, the results cannot speak to whether the drug works differently in Blacks. As Kahn notes, “The only responsible scientific claim that can be made on the basis of these trials is that BiDil works in some people who have heart failure, period.”<sup>76</sup>

***There is little robust evidence that race is a suitable proxy for genetic differences in drug response.*** No genetic component to BiDil’s efficacy has been demonstrated, despite assumptions by Dr. Nissen and other BiDil supporters who believe that self-identified race can be used as a proxy for genetic differences until specific genetic variations are located. Racial pharmacogenomics, as discussed above, is based upon the idea that specific genetic variations that are most common within particular populations explain certain health disparities, and that these disparities can be remedied with therapies that take such knowledge into consideration. BiDil’s clinical trials arguably put the cart before the horse, replacing a scientific approach with the theory that racial difference equals genetic difference connected to heart failure.

BiDil’s presumed race specificity is based upon the idea that self-identified race can be a reliable placeholder for inherited genetic variations that ostensibly explain disparate health outcomes. However, Francis Collins, former director of the National Human Genome Research Institute, writes: “A true understanding of disease risk requires a thorough examination of root causes. ‘Race’ and ‘ethnicity’ are poorly defined terms that serve as flawed surrogates for multiple environmental and genetic factors in disease causation, including ancestral geographic origins, socioeconomic status, education and access to health

## Are More Race-Based Medicines Around the Corner?

University College London biologists Sarah Tate and David Goldstein note in a 2004 *Nature Genetics* article that while controversial, “at least 29 medicines (or combination of medicines) have been claimed, in peer reviewed scientific or medical journals, to have differences in either safety or, more commonly, efficacy among racial or ethnic groups.”<sup>78</sup> Examples include:

- AstraZeneca is currently trying to salvage Iressa—a drug that blocks carcinogenic cell growth—after a clinical trial showed its efficacy to be statistically insignificant.<sup>79</sup> The company claims to have found data suggesting that Asians responded particularly well to it and has begun developing marketing strategies for Asian countries.<sup>80</sup>
- While the cholesterol-lowering drug Crestor is currently available to all qualifying patients, AstraZeneca has conducted a racially exclusive clinical trial (similar to A-HeFT) called STARSHIP to demonstrate its particular effectiveness in Hispanics.<sup>81</sup> The FDA has also issued a Public Health Advisory because some Asian Americans had an unusually strong reaction to Crestor at some dosages.<sup>82</sup>
- In 2003, the pharmaceutical company VaxGen took another look at data showing that its HIV vaccine,

AIDSVAX, was not effective in the general population. It hoped to find that the vaccine significantly reduced HIV infections in Blacks and Asians, but abandoned the effort after a subsequent clinical trial in Thailand also failed to demonstrate efficacy.<sup>83</sup>

The Pharmaceutical Research and Manufacturers of America (PhRMA), the pharmaceutical industry’s trade group, released a report in December 2007 noting that its member companies “are developing 691 medicines for diseases that disproportionately affect African Americans or diseases that are among the top 10 causes of death for African Americans . . . [to] help close the health disparity.”<sup>84</sup> While this report does *not* specifically pertain to medicines claiming to be genetically tailored for Blacks, the report’s framing highlights a perspective that drug companies are promoting and that is becoming increasingly popular within the biomedical sciences: health disparities are linked to group predispositions that are best addressed through targeted medications. The idea that some racial groups are inherently different from others is at the heart of the moral impetus for race-based medications.

care. Research must move beyond these weak and imperfect proxy relationships to define the more proximate factors that influence health.”<sup>77</sup>

Arguments based on loose correlations and unreliable proxies can play dangerously into lay notions that racial difference equals fixed genetic difference and may thus erroneously give the impression that racial disparities are caused by genes.

## Addressing Disparities in Health Through Race-Specific Pharmaceuticals

The assumptions and missteps embedded in efforts to develop and market race-specific medicines raise some concerns. They contribute to three possible outcomes that may work against sensible approaches to addressing health disparities.

***Social determinants of health may take a back seat.*** Studies have repeatedly demonstrated the relevance of poverty, environmental contaminants, lack of education, and other social determinants to overall health and health disparities.<sup>85</sup> Even the most enthusiastic supporter of BiDil’s race-specific indication acknowledges that many factors—such as diet and stress—contribute to hypertension, diabetes, and other conditions that lead to heart failure.

Scientific studies that root health disparities in genetic differences might obscure the social and environmental factors that affect groups’ disparate health outcomes. Thinking about race in genetic terms attracts public attention and deemphasizes the ways in which poor social treatment leads to poor health outcomes.<sup>86</sup>

*Claims about a genetic basis for racial disparities in health outcomes can quickly influence how we understand other social disparities.* A key concern is the temptation to use the notion that “racial disparities in health are genetically linked” to explain racial disparities in other areas such as employment, education, and criminal justice. These disparate outcomes might then be attributed to people’s genes rather than to the treatment groups are afforded and their access to resources. Discussion of Blacks’ unemployment rate, educational underachievement, and grossly disproportionate representation in the criminal justice system becomes detached from society’s long history of discriminatory practices, and can become intertwined with assumptions about groups’ inherent (and inheritable) tendencies. This may allow old theories of racial minorities’ biological inferiority to be legitimated in new and different terms, shaping how we understand inequalities in other fields.

*Race-specific medicines can shift the responsibility for resolving racial disparities in health from public health initiatives to private biomedical ventures.* This is not to say that profit interests can never converge with genuine opportunities to reduce health disparities. Indeed, profit-driven research and development might lead to treatments that can greatly benefit minority communities. But there is significant evidence that commercial motives might also lead companies to make claims about race, genes, and medicine that the available scientific evidence simply does not support. And ceding the problem of racial disparities in health to biomedical companies might devalue public health mechanisms that tackle these disparities’ core social and environmental causes.

Examples abound of how commercial dynamics can distort the public interest in drug development. With regards to race-based medicine, BiDil’s original patent as a race-neutral drug expired in 2007; the new patent based on the claim of racial specificity extended exclusive rights over what is essentially two generic drugs packaged as one. It is

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## The Slavery Hypothesis

Exaggerated ideas about what genes can explain have shaped popular culture to the point of creating urban legends.<sup>87</sup> And genetic reductionism affects medical professionals as well as pop culture. One example is the so-called “slavery hypothesis,” which has received high-profile coverage on *The Oprah Winfrey Show* and the CNN mini-series *Black in America*.

According to this theory, African Americans tend to have high blood pressure because the slaves who survived the grueling journey across the Atlantic to North America had a genetic predisposition to retain salt—in short supply on the slave ships—which gave them a survival advantage. Given the supposed genetic roots of this advantage, the heritable characteristic was supposedly passed on to subsequent generations, who then developed hypertension in epidemic proportions once their daily salt intake increased.

No evidence supports this theory despite its prevalence and persistence. Even if there were a “salt sensitivity” gene, slave ships’ overall mortality rate, while high, was insufficient to create a lasting genetic bottleneck effect that would shape the entire African American gene pool in perpetuity.<sup>88</sup> And there is no evidence for this hypothesized gene among native Africans. Indeed, Nigerians have lower hypertension rates than White Americans, while Finns have higher rates than Black Americans.<sup>89</sup>

Such genetic reductionism can distract from the documented social determinants that affect hypertension such as poverty, diet and stress. Saying something is “in the genes” is tantamount to saying we can do nothing about it—except perhaps sell expensive custom-made medications. And that is a prescription not for health equity, but for continuing disparities.

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not unlikely that this influenced Nitromed’s re-packaging of BiDil as a race-specific drug.

Such intellectual property rights have the potential to increase some African Americans’ cost for heart failure treatment. Some have been encouraged to pay BiDil’s premium rather than continue a medical practice that has been going on for years prior to BiDil’s FDA approval: taking its generic counterpart. Though there is some contention as to whether BiDil and its generic components are bioequivalent,<sup>90</sup> the broader point is that leaving the resolution of health disparities to the market can increase costs in ways that, in the

end, make health care less accessible to minority populations.

In a similar vein, using less-than-robust scientific evidence to racialize drug indications might prevent broader populations from potentially benefiting from a therapy. Some doctors may avoid prescribing what the federal government deems to be a Black drug to non-Black patients. And some non-Black heart failure sufferers might not want to take a so-called “Black” drug.

## Conclusion: Evaluating Race-Based Medicine

Taken together, BiDil presents at least four inter-related concerns that should give pause when considering continued efforts to produce and market race-based medicines:

1. The claim that BiDil’s effects are race-specific is based on less than convincing science.
2. Its marketing suggests that health disparities are best addressed through technology rather than by addressing social determinants.
3. It might give unwarranted credence to biological notions of racial difference.
4. It may obscure the real potential of personalized medicines based upon individuals’ genotypes rather than self-identified race or group phenotype.

What unites these initial forays into personalized medicine with our broader concerns about race and biotechnology is their tendency to work from the outside in: to assume that race (self-identified or otherwise) reflects genetic variation that explains groups’ disparate health outcomes. This is fundamentally different than pharmacogenomics’ scientific promise: that specific genotypes, regardless of an individual’s racial categorization, can be identified and correlated with particular therapies to improve drug response. Loose correlations between the phenotypes and genotypes of racial groups belie the promising science behind pharmacogenomics.

## Recommendations

- The Food and Drug Administration should require that clinical trials used to support race-specific indications *not* be racially exclusive. Rather, these clinical trials should occur across racial populations and empirically demonstrate not only that the proposed drug is more effective than standard therapy in the targeted population, but also no better than standard therapy in the non-targeted group.<sup>91</sup>
- When race-specific drug labels are sought, the FDA should seek authority to convene separate advisory committees that look at implications beyond safety and efficacy. In particular, these committees should examine the broader social impact that might occur. A key concern should be the avoidance of any government action that might give undue legitimacy to biological understandings of racial difference or unnecessarily restrict medications that might benefit more than one racial population.<sup>92</sup>