

The Law and Genetics of Racial Profiling in Medicine

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Modern medicine has embraced the use of race. Race is routinely employed by medical researchers, clinicians, and community health officials. Moreover, medicine's use of race is not done in the shadows, but right before our eyes. Physicians note our race when treating us and medical researchers routinely publish results that classify subjects based on race. Researchers debate the relative merits of using race in prominent journals and doctors have freely claimed in major newspapers that they use race.¹ Recently, the *New York Times* featured Dr. Sally Satel on the cover of its Magazine Section proudly proclaiming, "I am a racially profiling doctor."² A year earlier, the same paper reported on FDA approval of clinical trials for a heart drug designed exclusively for African Americans.³

Curiously, the question of whether biological differences in the races should be taken into account by our health care institutions has gone largely unconsidered in the law journals. Given the pervasive role law plays in medicine and research, this is surprising. This omission is especially striking because race otherwise dominates law review articles.⁴ Whether the topic is affirmative action, employment discrimination, environmental justice or any of a myriad of areas where race encounters the law, the reviews have thoroughly canvassed the problem, often with a strong interdisciplinary focus. Accordingly, law journals have devoted significant space to race where it intersects health care in one area: the cause of racial disparities

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¹ See, e.g., Ariana Eunjung Cha, *Race Plays Role in New Drug Trials: Treatment by Genetic Origin, Ethnicity Divides Medical Profession*, WASH. POST, July 28, 2003, at A1.

² Sally Satel, *I Am a Racially Profiling Doctor*, N.Y. TIMES, May 5, 2002, § 6 (Magazine), at 56.

³ Sheryl Gay Stolberg, *Shouldn't a Pill Be Colorblind?*, N.Y. TIMES, May 13, 2001, § 4, at 1.

⁴ A Lexis search for "race" or "racial" in the title of law review articles yielded 1904 hits. U.S. & Canadian Law Reviews database, July 25, 2003. Obviously, a great many other articles address racial issues without using either word in the title.

in the health status of African Americans and other minorities.⁵ But the conscious use of race to diagnose and treat individuals continues with almost no discussion, despite all of the attention paid to the topic in both the popular media and medical literature. This Article fills that void.

Taking race into account in medical treatment seems at least as objectionable as other, explicitly prohibited uses, especially given the egregious acts perpetrated against racial minorities in this country in the name of medicine. For example, in the notorious Tuskegee Syphilis Experiment, the United States Public Health Service deliberately failed to treat nearly 400 African American males suffering from late-stage syphilis.⁶ Further, the notion of genetic racial differences triggers associations with the eugenics movement⁷ and repeated “scientific” efforts to prove the intellectual inferiority of African Americans.⁸

Even more dramatic is the increasing acceptance among researchers and clinicians of race as an appropriate focus of medical study and treatment. Indeed, this may be an unintended byproduct of the medical and legal literature on racial disparities in health. For example, to explain why African Americans have higher mortality rates from heart disease, researchers have studied whether the disparity may be partially accounted for by genetic differences between African Americans and whites—differences that enlightened modern medicine can identify and then address.⁹ To that

⁵ The same Lexis database yielded 112 results for “racial disparity w/p health.” U.S. & Canadian Law Reviews, July 25, 2003. *E.g.*, Joel Teitelbaum & Sara Rosenbaum, *Medical Care as a Public Accommodation: Moving the Discussion to Race*, 29 AM. J.L. & MED. 381 (2003); Sidney D. Watson, *Race, Ethnicity and Quality of Care: Inequalities and Incentives*, 27 AM. J.L. & MED. 203 (2001).

⁶ See JAMES H. JONES, *BAD BLOOD: THE TUSKEGEE SYPHILIS EXPERIMENT 1–7* (rev. ed. 1993).

⁷ See generally Paul A. Lombardo, *Taking Eugenics Seriously: Three Generations of ??? Are Enough?*, 30 FLA. ST. U. L. REV. 191 (2003).

⁸ The most recent effort in this direction was RICHARD J. HERRNSTEIN & CHARLES MURRAY, *THE BELL CURVE: INTELLIGENCE AND CLASS STRUCTURE IN AMERICAN LIFE* (1994). See also Paul A. Lombardo, “*The American Breed*”: *Nazi Eugenics and the Origins of the Pioneer Fund*, 65 ALB. L. REV. 743 (2002) (explaining aspects of the history of eugenics in further detail).

In light of this sad history, it is not surprising that Dr. Satel disclaimed any racist impulses, wrapping herself in the mantle of science. She and many of her colleagues note their patients’ race “because certain diseases and treatment responses cluster by ethnicity. Recognizing these patterns can help us diagnose disease more efficiently and prescribe medications more effectively. When it comes to practicing medicine, stereotyping often works.” Satel, *supra* note 2, at 56; see also Cha, *supra* note 1, at A10 (noting that “some doctors have for a long time adjusted dosages or favored certain medications over others because of a patient’s race”). Similarly, the makers of BiDil, a heart medicine being tested for African Americans, clearly sought validation of the purity of their motives when they obtained the imprimatur of the Association of Black Cardiologists and the support of members of the U.S. Congressional Black Caucus. See NitroMed, *BiDil® and the African American Heart Failure Trial (A-HeFT)*, at <http://www.nitromed.com/bildil/DOCS/background.html> (last visited Aug. 14, 2003).

⁹ *But cf.* Jonathan Kahn, *Getting the Numbers Right: Statistical Mischief and Racial Profiling in Heart Failure Research*, 46 PERSP. BIOLOGY & MED. 473–74 (2003) (challenging the accuracy of supposed fact that blacks die from heart disease at a rate twice that

end, medical journals increasingly explore possible racial connections with diseases and treatments. In 2001, a pair of studies in the *New England Journal of Medicine* focused on possible differences in drug responses among black and white heart patients. One study found racial differences for one drug;¹⁰ the other found no such differences for another.¹¹ Other examples abound.¹²

The notion that medicine should reject a colorblind model in favor of taking race into account marks a significant shift in perspective. Proponents argue that, unlike many of their predecessors in the medical and scientific community, they will take race into account only when it is appropriate to do so.¹³ But that claim was also made by predecessors whose views are now widely condemned.¹⁴ Furthermore, it occurs at a time when researchers are documenting the role that unconscious or semi-conscious racism plays in the delivery of medical treatment. For example, recent research has suggested that physicians prescribe different treatment for patients solely as a result of the patient's race and/or gender.¹⁵ One study showed that physicians recommended cardiac catheterization at a lower rate for African American female patients than for African American males, white males, or white females, even though the symptoms presented were exactly the same.¹⁶ Another study showed that physicians prescribed analgesics to patients at different dosages depending upon the race of the patient and the gender of the physician.¹⁷ All of these differences are in-

of whites).

¹⁰ Derek V. Exner et al., *Lesser Response to Angiotensin-Converting-Enzyme Inhibitor Therapy in Black as Compared with White Patients with Left Ventricular Dysfunction*, 344 *NEW ENG. J. MED.* 1351, 1351 (2001).

¹¹ Clyde W. Yancy et al., *Race and the Response to Adrenergic Blockade with Carvedilol in Patients with Chronic Heart Failure*, 344 *NEW ENG. J. MED.* 1358, 1358 (2001). See also *Shades of Doubt and Fears of Bias in the Doctor's Office*, *CHRON. HIGHER EDUC.*, May 25, 2001, at A16.

¹² See, e.g., sources cited *infra* notes 15–17.

¹³ See, e.g., Satel, *supra* note 2.

¹⁴ See, e.g., Lombardo, *supra* note 7.

¹⁵ INSTITUTE OF MEDICINE, *UNEQUAL TREATMENT: CONFRONTING RACIAL AND ETHNIC DISPARITIES IN HEALTH CARE* 177 (Brian D. Smedley et al. eds., 2003); Jay S. Kaufman & Richard S. Cooper, *Commentary: Considerations for Use of Racial/Ethnic Classification in Etiologic Research*, 154 *AM. J. EPIDEMIOLOGY* 291, 293–94 (2001).

¹⁶ Kevin A. Schulman et al., *The Effect of Race and Sex on Physicians' Recommendations for Cardiac Catheterization*, 340 *NEW ENG. J. MED.* 618 (1999). The findings of Schulman and his colleagues have sparked some debate. Compare Lisa M. Schwartz et al., *Misunderstandings about the Effects of Race and Sex on Physicians' Referrals for Cardiac Catheterization*, 341 *NEW ENG. J. MED.* 279 (1999) (challenging some of the analysis), with Kevin A. Schulman et al., *Letter to the Editor*, 341 *NEW ENG. J. MED.* 286 (1999) (responding). Even the critics, though, acknowledged that African American women were referred at a much lower rate. See, e.g., Schwartz et al., *supra*, at 280–81.

¹⁷ Carol S. Weisse et al., *Do Gender and Race Affect Decisions about Pain Management?*, 16 *J. GEN. INTERNAL MED.* 211 (2001) (finding that male physicians prescribed higher doses to white patients, but that female physicians prescribed higher doses to black patients). See also Marti Loring & Brian Powell, *Gender, Race and DSM-III: A Study of the Objectivity of Psychiatric Diagnostic Behavior*, 29 *J. HEALTH & SOC. BEHAV.* 1 (1988) (finding differences in psychiatric diagnoses based on race and gender of both patient and doctor).

appropriate in terms of the current state of medical knowledge. Even if physicians can be cured of conscious bias, they no doubt will be influenced by the unconscious biases that plague American society.¹⁸

The problems of using race in health care have not gone unnoticed in the medical community. In 2001, the *New England Journal of Medicine* ran two editorials—one praising the research¹⁹ and the other claiming that attributing medical differences to race “is not only imprecise but also of no proven value in treating an individual patient.”²⁰ The *New England Journal of Medicine* reprised the 2001 dispute with a pair of articles in March 2003. Esteban González Burchard of the University of California at San Francisco and Neil Risch of Stanford University argued that ignoring race will “retard progress in biomedical research,”²¹ while an opposing article by Dr. Richard S. Cooper warned that scientists have been too quick to view genetics as the reason for greater susceptibility of African Americans to certain diseases when the real reason may be social factors.²² The reality is that more and more articles in scientific journals are reporting results by racial groups, a result federal regulations encourage.²³

Almost completely ignored to date have been the legal implications of medicine’s use of race. Existing law, primarily the Equal Protection Clause, 42 U.S.C. § 1981 and Titles II and VI of the Civil Rights Act of 1964, renders many of these actions legally suspect. For instance, the use of race in selecting participants for clinical trials and in deciding the treatment of patients, we believe, may violate federal constitutional and statutory law. While nonclinical research that utilizes race is probably legal, it faces threats from proposals such as the California Racial Privacy Initiative,²⁴ rejected this past year.

Given the disconnect between what medicine does and what the law appears to allow, what should be done? We believe that, in quite limited circumstances, the law should permit the use of race in medicine. Race, although socially constructed, is a useful proxy for both a person’s ancestry and for environment.²⁵ As we explain in more detail in this Article, both ancestry and environment can play an important role in determining a person’s health. Of course, race is never more than a proxy, and other

¹⁸ See Rachel D. Godsil, *Expressivism, Empathy & Equality*, 36 U. MICH. J.L. REFORM 247, 269–70 (2003) (discussing scholars’ approaches to unconscious racism).

¹⁹ Alastair J. J. Wood, *Racial Differences in the Response to Drugs—Pointers to Genetic Differences*, 344 NEW ENG. J. MED. 1393 (2001).

²⁰ Robert S. Schwartz, *Racial Profiling in Medical Research*, 344 NEW ENG. J. MED. 1392, 1392 (2001).

²¹ Esteban González Burchard et al., *The Importance of Race and Ethnic Background in Biomedical Research and Clinical Practice*, 348 NEW ENG. J. MED. 1170, 1171 (2003).

²² Richard S. Cooper et al., *Race and Genomics*, 348 NEW ENG. J. MED. 1166, 1167 (2003).

²³ See *infra* note 396 (noting FDA requirement that such data be reported in clinical trials).

²⁴ See *infra* note 360.

²⁵ See discussion *infra* Part II.

and better methods can usually be used to obtain the same information about ancestry and environment. But, in a few cases, race may be the best, and perhaps only, means of obtaining this information. When and if this is true, the use of race can be justified.

We acknowledge that the use of race in medicine, as anywhere else, is fraught with peril. Researchers and clinicians in the past have visited grave injustices on individuals in the pursuit of race-driven medicine. The continued use of race by physicians and other health care professionals may only reinforce the unconscious biases that infect medicine, and it may tend to validate the racism of others in society more generally. These costs have to be weighed before the use of race should be permitted. But even after considering them, we still believe that there are some very limited circumstances where the use of race ought to be permitted. This Article is, in large part, designed to define carefully the rare circumstances in which the use of race will be appropriate.

The Article proceeds as follows. Part I sets the stage by sketching the underlying debate about racial disparities in health status and health care and the ways in which the question of race in health is likely to arise. It also addresses the special problems of using race. Part II then turns to the threshold question for any such discussion, "What is Race?," concluding that "race" as it is currently used in America is socially constructed. While race, as a biological construct, has no meaning, modern human evolutionary theory tells us that, in quite limited circumstances, differences in the frequency of some genes may arise between different races as they have been socially constructed. This is (generally) *not* because of natural selection, but rather the result of an evolutionary force known as genetic drift, which causes population groups that are separated from one another to diverge in the frequency of genes.

Part III then canvasses the scientific literature to assess the limited situations when "race" may be suitable for medical use because of genetic factors that cannot otherwise be efficiently taken into account. Race, when used as a proxy for ancestry, may tell us something about both disease susceptibility and drug sensitivity. In addition, when seen as a proxy for environment, race can also tell us something about disease susceptibility. Part IV moves from science to law, reviewing the various legal regimes that bear on the use of race in the medical context. We conclude that, in general, the use of race in medicine raises serious legal issues. The main exception is that race-based studies, with no clinical component, would appear to be legal. Finally, Part V brings together the themes of social construction of race, genetically related populations, and the existing legal framework in order to draw normative recommendations for the law's approach to "racial profiling" in medicine. In particular, we propose the creation of a defense for the limited use of race in treatment, which we describe as a *bona fide* treatment rationale defense. In addition, we suggest that efforts to include (but not to exclude) racial groups in

clinical trials ought to be permitted, and that efforts to exclude groups ought to be resisted. Finally, we accept—for now—the continued use of race in non-clinical studies.

I. DISPARITIES IN HEALTH STATUS AND HEALTH CARE

A. *The Existence of Racial Disparities*

Racial disparities in health status are scarcely news. As a recent article summarized, “[m]inority Americans have significantly higher rates of cancer, stroke, heart disease, AIDS, diabetes, and other severe health problems than white Americans.”²⁶ While there are differences among minorities in health status, racial and ethnic minorities generally have higher rates of a wide variety of diseases and have higher disability and death rates than whites in this country.²⁷

The causes for these differences are many, and it is certain that socio-economic status, rather than race or ethnicity per se, is an important contributing factor.²⁸ Obviously, poverty influences such health basics as nutrition and environment, and minorities are more likely to be poor than whites.²⁹ Further, access to health care is obviously directly related to income,³⁰ or at least insured status (which, in the United States, is strongly related to income since insurance is linked generally to employment).³¹ However, studies that have tried to hold such factors constant, and also

²⁶ Sidney D. Watson, *Race, Ethnicity and Quality of Care: Inequalities and Incentives*, 27 AM. J.L. & MED. 203, 205 (2001). See also Louise G. Trubek & Elizabeth A. Hoffmann, *Searching for a Balance in Universal Health Care Reform: Protection for the Disfranchised Consumer*, 43 DEPAUL L. REV. 1081, 1091–92 (1994) (“People of color have a shorter life expectancy due to higher rates of cancer, hypertension, infant mortality, alcohol and drug abuse, cardiovascular disease, and diabetes.”).

²⁷ See Barbara A. Noah, *The Participation of Underrepresented Minorities in Clinical Research*, 29 AM. J.L. & MED. 221, 223 (2003) (“The infant mortality rate among African-Americans is double that of whites, and African-Americans can expect to live six fewer years on average than whites. African-Americans die from complications of diabetes at three times the rate of whites, and experience higher incidences of several cancers, including breast, colorectal, and lung cancers, as well as the highest death rates from all cancers combined.”).

²⁸ See Trubek & Hoffman, *supra* note 26, at 1092–93 (“For the urban poor, the problem is that health care facilities are not often located in their communities For rural poor, the problem is that poor and widely dispersed rural communities rarely attract permanent medical staffs to their area.”).

²⁹ See Watson, *supra* note 26, at 218 (“[M]inority Americans are more likely than white Americans to be poor, uninsured and on Medicaid.”).

³⁰ See Linda C. Fentiman, *AIDS as a Chronic Illness: A Cautionary Tale for the End of the Twentieth Century*, 61 ALB. L. REV. 989, 1008 (1998) (“[F]or people under 65 health care coverage is tied closely to employment status.” (quoting PAUL FRONSTIN, SOURCES OF HEALTH INSURANCE AND CHARACTERISTICS OF THE UNINSURED 2 (Employee Benefit Research Inst. Issue Brief No. 192, 1997))).

³¹ This is not to suggest that the more health care, the better the health status; indeed it might be the least important factor. See generally Mary Crossley, *Infected Judgment: Legal Responses to Physician Bias*, 48 VILL. L. REV. 195 (2003) (discussing physician bias).

account for age, gender, clinical condition and severity of disease, have continued to find significant remaining differences in treatment strongly associated with race. One commentator reported that at least some of the difference could be attributed to disparities in health care provided to minorities:

Moreover, health care professionals provide different—and generally less—care to their minority patients. When hospitalized, African-Americans receive fewer surgical interventions, diagnostic tests, medical services, and less optimal interventions than whites—even when their diagnosis, symptoms, and source of payment are the same. The findings for African-Americans are consistent for every service studied: cardiology and cardiac surgery, obstetrics, general medicine, kidney transplants, hip replacements, mammograms, oncology and leg sparing surgery for peripheral vascular disease³²

The obvious question is: why do such disparities exist? There have been at least two major explanations offered for different treatment of individuals with similar medical conditions and equal access to medical care. The first focuses on the supply side and explores discrimination by health care providers, either because of blatant prejudice or, more likely, because of stereotyping.³³ The second looks at the demand side—resistance of minority populations to more advanced medical treatment because of cultural factors, including a widely documented suspicion of medical professionals.³⁴ These possibilities are, of course, not mutually exclusive, and a vicious cycle may be at work with minorities, particularly African Americans. Minorities may be resistant to medical recommendations because of a long and treacherous history “of several hundred years of pseudoscientific racism and pervasive discrimination in medicine, unethical and often brutal experimentation, and abuse of black people by both private institutions and government programs in health.”³⁵ Physicians may interpret such resistance as noncompliance that, in turn, justifies lesser care.

Our goal is not to address these questions, which have been dealt with effectively by a range of scholars.³⁶ Rather, in the midst of the de-

³² Watson, *supra* note 26, at 206–07. Watson also found that African Americans were provided with less outpatient care and that the outcomes for Hispanics and Native Americans were similar. *But see* S. S. Rothmore et al., *Race, Quality of Care, and Outcomes of Elderly Patients Hospitalized with Heart Failure*, 289 JAMA 2517 (2003) (finding no difference in treatment in the population of Medicare beneficiaries hospitalized for heart failure).

³³ *E.g.*, Crossley, *supra* note 31.

³⁴ René Bowser, *Racial Profiling in Health Care: An Institutional Analysis of Medical Treatment Disparities*, 7 MICH. J. RACE & L. 79, 94–95 (2001) [hereinafter Bowser, *Racial Profiling*].

³⁵ *Id.* at 94.

³⁶ *E.g.*, Bowser, *Racial Profiling*, *supra* note 34; Crossley, *supra* note 31; Watson, *su-*

bate about the causes of the problem and how to address it, one confounding possibility has arisen—that racial groups are biologically different in ways that impact the delivery of health care to patients. Theoretically, genetic and behavioral racial differences might not only explain some differences in health status but also justify racial differences in treatment. While, for reasons we will explore at some length, any such differences are unlikely to provide much explanatory power for racial disparities in health status or health care, the possibility has developed to the point where the medical, scientific, and legal communities must take it into account. This Article begins that task.

As a prelude to that effort, however, it is important to consider the areas in which medicine uses race. The first is race-focused statistical studies, by which we mean researchers' collection and dissemination of statistical information and analysis about the health of subjects apart from the diagnosis or treatment of subjects.³⁷ The next is race-related outreach, which refers to attempts to educate particular races on health issues, for example, attempts to inform African Americans that they are at higher risk of developing sickle cell anemia. The third is the use of diagnostic tests on whole populations, which we refer to as race-based screening.³⁸ The best example of such race-based screening was the requirements in some states that African Americans, but not others, be tested for sickle cell anemia.³⁹ The fourth way race is used is in race-based clinical trials, such as the BiDil trial described in the Introduction. In that case, presumably only African Americans are eligible to participate in the study.⁴⁰ Finally, there is race-based treatment, such as the practices Dr. Satel describes.⁴¹

B. The Special Harms of Race

The consideration of a person's race in the United States has rarely benefited members of minority groups. Instead, race has generally been used to subordinate non-whites: to limit their political power, to deprive them of economic opportunities, and more broadly, to deny them liberty. Given this background of race in the service of racism, extreme caution is warranted in the use of race in medicine.

While the use of race has resulted in concrete harms inflicted by governmental actors (most notably de jure segregation in the South), scholars over the last decade have also been concerned with the law's expressive

pra note 26.

³⁷ Since some studies seek to ascertain the influence of a wide range of factors on health status, the information sought can be quite sweeping. While these studies raise their own privacy and ethical concerns, that is not the concern of the present Article.

³⁸ See *infra* text accompanying note 372.

³⁹ See *infra* text accompanying note 377.

⁴⁰ See NitroMed, *supra* note 8.

⁴¹ See Satel, *supra* note 2.

power,⁴² arguing that laws and legal actors can send stigmatizing messages that result in concrete harms separate and apart from any denial of government benefits.⁴³ Under this view, the law can harm individuals by “express[ing] contempt, hostility, or inappropriate paternalism toward racial, ethnic, gender, and certain other groups, or that constitute them as social inferiors or as a stigmatized or pariah class.”⁴⁴ Similarly, expressive harms can arise from law “giving too much weight to suspect classifications, express[ing] a divisive conception of citizens—a conception that represents their racial, ethnic, religious, or other parochial identities as more important than their common identity as citizens of the United States.”⁴⁵

Legitimizing the use of race in health care runs both of these risks. Any use of race has the potential to perpetuate racism. Because race is socially constructed,⁴⁶ actions by health care providers that rely on race serve to reify that social construction, to the general detriment of racial minorities.⁴⁷ Of course, in a society that has already socially constructed race, the concrete benefits that flow to racial minorities from specific actions recognizing race may outweigh any harms that flow from further legitimizing race. Indeed, some scholars have suggested that the cure to racism is not to deny the existence of race, but rather to “consciously and vociferously contest[] the meaning of race and of the content of” the character of individuals.⁴⁸ Despite potential benefits, however, there may be something distinctive about using race in health care that counsels against its use here.

Other concerns focus on using genes to demonstrate racial distinctions. Although racial differences in health outcomes can be as easily ascribed to either genetics or environment, much of the research to date has focused on genetic causes. This focus creates the distinct possibility of increasing racism across the board by giving credence to claims that races

⁴² See, e.g., Elizabeth S. Anderson & Richard H. Pildes, *Expressive Theories of Law: A General Restatement*, 148 U. PA. L. REV. 1503 (2000); Cass R. Sunstein, *Incommensurability and Valuation in Law*, 92 MICH. L. REV. 779, 820–24 (1994). But see Matthew D. Adler, *Expressive Theories of Law: A Skeptical Overview*, 148 U. PA. L. REV. 1363, 1495 (2000) (arguing that these are not true “expressive” theories of law in the sense of linguistic impact, although they may be expressive theories in other senses of the term); Matthew D. Adler, *Linguistic Meaning, Nonlinguistic “Expression,” and the Multiple Variants of Expressivism: A Reply to Professors Anderson and Pildes*, 148 U. PA. L. REV. (2000) (same).

⁴³ See Godsil, *supra* note 18, at 274.

⁴⁴ Anderson & Pildes, *supra* note 42, at 1533.

⁴⁵ *Id.* at 1533–34.

⁴⁶ See discussion *infra* Part II.

⁴⁷ See LARS NOAH & BARBARA A. NOAH, *LAW, MEDICINE, AND MEDICAL TECHNOLOGY* 245 (2002); René Bowser, *Racial Bias in Medical Treatment*, 105 DICK. L. REV. 365, 376 (2001) (noting that “racialized research” “consolidates Whites as the group with which all ‘others’ should be compared,” thereby leading to increased white consciousness and an obscuring of heterogeneity among whites) [hereinafter Bowser, *Racial Bias*]; Newton G. Osborne & Marvin D. Feit, *The Use of Race in Medical Research*, 267 JAMA 275, 278 (1992) (suggesting that racial comparisons in health care may reinforce societal racism).

⁴⁸ Ian F. Haney López, *The Social Construction of Race: Some Observations on Illusion, Fabrication, and Choice*, 29 HARV. C.R.-C.L. L. REV. 1, 20 (1994).

are biologically distinct from one another. This notion lends itself to the mistaken belief that members of particular races have far more in common with one another than with other human beings. That is false. Nonetheless, the message that some racial differences in genes imply significant genetic differences between races may undermine our cohesiveness both as a nation and as a species.

A related worry is that the differences in frequencies for health-related traits will be seen as an indication that there are differences in frequencies for other traits. In particular, as Stephen Jay Gould notes in *The Mismeasure of Man*, there has long been a belief in differences in intelligence among the races and, at least in the twentieth century (and beyond), there have been attempts to demonstrate that such differences are the result of genetic factors.⁴⁹ Such conjectures are almost assuredly wrong.⁵⁰

Even though there is no reason to believe that real differences in intelligence, or other general behavioral traits, exist between races, some will seize on any differences in traits related to health as support for contrary views. The false validation of such stereotypes based on the use of race in health care may result in persons being denied educational opportunities, on the assumption that racial minorities are less intelligent, or employment, because a racial minority group is “too acquiescent.” It is worthwhile to recall the description of Africans by Carolus Linnaeus, the initial taxonomist of races, as being ruled in their behavior “by caprice,” as opposed to Europeans, who were ruled “by custom.”⁵¹ Such beliefs that racial groups differ in their intelligence and behavior persist in the minds of some, if not many, Americans today, and validating the use of race in health care may strengthen such views.⁵²

Furthermore, as Professor René Bowser has noted, the use of race in medical practice may itself lead to worse health outcomes for members of minority groups.⁵³ Focusing on genetic causes or environmental factors that can lead to poorer health in racial minorities can distract investiga-

⁴⁹ See *supra* note 8 and accompanying text; *infra* notes 92–93 and accompanying text.

⁵⁰ See STEPHEN JAY GOULD, *THE MISMEASURE OF MAN* 404, 367–78 (rev. ed. 1996).

⁵¹ *Id.* at 404–05 (translating from Linnaeus’ Latin). See also STEVE OLSON, *MAPPING HUMAN HISTORY: GENES, RACE AND OUR COMMON ORIGINS* 60 (2002) (setting forth Linnaeus’ other descriptions). Indeed, Thomas Jefferson, often viewed as a racial moderate at the time, remarked: “Comparing [African Americans] by their faculties of memory, reason, and imagination, it appears to me, that in memory they are equal to the whites; in reason much inferior, as I think one could scarcely be found capable of tracing and comprehending the investigations of Euclid; and that in imagination they are dull, tasteless, and anomalous.” THOMAS JEFFERSON, *NOTES ON THE STATE OF VIRGINIA* 139 (William Peden ed., U.N.C. Press 1982) (1787).

⁵² Lawrence D. Bobo, *Racial Attitudes and Relations at the Close of the Twentieth Century*, in 1 *AMERICA BECOMING: RACIAL TRENDS AND THEIR CONSEQUENCES* 264, 273–80 (Neil J. Smelser et al. eds., 2001).

⁵³ See Bowser, *Racial Bias*, *supra* note 47, at 378; cf. Cooper et al., *supra* note 22, at 1166 (“Promotion of a drug for a race-specific ‘niche market’ could distract physicians from therapies for which unequivocal evidence of benefit already exists.”).

tors from another important cause: racism.⁵⁴ The significant racial disparities that exist in health care⁵⁵ arise, at least in part, as the result of past or present racism,⁵⁶ although most current discriminatory practices probably result from either unconscious or semi-conscious stereotyping on the part of physicians and other health care professionals, rather than from the conscious racism that has characterized so much of our history.⁵⁷

Finally, the use of race in research may lead to what Professor Bowser describes as “bedside bias.”⁵⁸ Having been trained to believe that race is useful information for medical practice, medical practitioners may make unjustified assumptions about a patient’s likely disease or response to treatment based on the person’s apparent race without further investigation.⁵⁹ Assumptions about racial groups may be wrong, lead to great harm, and cause individual patients to suffer. But even if race is properly ascertained and the physician’s assumption is statistically “true” for that race, harm can result when the assumption turns out not to apply to individual cases. In a recent issue of *The Chronicle of Higher Education*, Doctor Richard Garcia told of a childhood friend who

wasn’t diagnosed with cystic fibrosis until she was 8 years old. Over the years, her doctors had described her as a “2-year-old black female with fever and cough . . . 4-year-old black girl with another pneumonia. Lela is back.” [sic] Had she been a white child, or had no visible “race” at all, she would probably have gotten the correct diagnosis and treatment much earlier. Only when she was 8 did a radiologist, who had never seen her face to face, notice her chest X-ray and ask, “Who’s the kid with CF?”⁶⁰

The fact that cystic fibrosis is far less common in African Americans than in whites⁶¹ no doubt led the treating doctors to overlook its possibility in Lela. But understanding why the doctors failed does not excuse that failure, nor the additional pain and suffering Lela endured. While race can be used in a beneficial manner, it can also be harmful when used improperly.

⁵⁴ See Bowser, *Racial Bias*, *supra* note 47, at 377; Cooper et al., *supra* note 22, at 1168.

⁵⁵ INSTITUTE OF MEDICINE, *supra* note 15, at 5–11.

⁵⁶ *Id.* at 8–11.

⁵⁷ *Id.* at 166–67.

⁵⁸ See Bowser, *Racial Bias*, *supra* note 47, at 378.

⁵⁹ See *id.* at 378–79; Cooper et al., *supra* note 22, at 1169 (noting that use of race can “leav[e] us blind to the meaning of the more relevant local and individual context”).

⁶⁰ Richard S. Garcia, *The Misuse of Race in Medical Diagnosis*, *CHRON. HIGHER EDUC.*, May 9, 2003, at B15.

⁶¹ See sources cited *infra* note 236.

II. WHAT IS RACE?⁶²

Before addressing the legality of using racial categories in health care, it is important to understand what is (and is not) meant by “race” and why race is understood by many medical researchers and practitioners to have a legitimate place in their work. Accordingly, we consider these questions in this and the next Part.

While the term “race” seems self-explanatory to most contemporary Americans, its precise meaning is far from clear, and race as a legal or scientific concept is even more contested. Present-day American law is inconsistent as to which groups are denominated “races.” Historical sources pro-

⁶² Terminology regarding race is particularly problematic for this Article, in part because it draws on a number of sources over a great span of time. As we develop at some length, there is neither agreement nor precision as to the meaning of “race” or the number or composition of races, or even the appropriate terminology for particular population groups. For example, the descendants of the peoples of Africa (largely West Africa) brought to the United States as slaves have been described over time as, among other terms, Africans, Ethiopians, Negroes, colored, black or Black, Afro-Americans and African Americans. Since this Article concludes that “races” are socially constructed, not biologically based, we opt for the socially preferred term for our writing; thus, we typically use “African Americans” to mean the descendants of African slaves in this country. We do so despite our acceptance of the “Out of Africa” theory, which suggests that all human beings are, in a very real sense, Africans. *See infra* text accompanying notes 145–147. Despite this preference, the other labels will be found in our sources, and the term is obviously ill-adapted for groups who are, say, the subjects of human research in the United Kingdom (where “black” seems to be the preferred descriptor in medical journals). *E.g.*, E. H. Baker et al., *Association of Hypertension with T594M Mutation in β Subunit of Epithelial Sodium Channels in Black People Resident in London*, 351 LANCET 1388 (1988).

However, since this Article’s focus is the relationship between social construction and genetics, it is sometimes important to draw distinctions that are otherwise unnecessary in social interactions. For example, we will see that African Americans have a substantial admixture of genes from other “racial” groups, *infra* text accompanying note 176, which requires us to sometimes stress the differences between African Americans, as society presently so calls them, and individuals whom we describe as “Africans” because of their lesser admixtures of white genetic material. We sometimes use the term “black” to describe both groups.

Similar problems arise with respect to Asians, who have been variously described collectively as Mongolians, members of the “yellow” race, and Orientals, although there is little sense in placing the various populations on or near the Asian continent in one group. Indeed, that question confounded the Supreme Court a century ago when it had to decide whether a high caste Indian was a Caucasian. *See* discussion *infra* note 74. The problem is, oddly enough, lessened for Hispanics or Latinos because it is widely recognized that those terms embrace more a cultural or national origin or language grouping than a “racial” one. *See infra* text accompanying note 175. Native Americans also can have heavy admixtures of other gene lines (both in terms of intermarriage with whites and, especially in some tribes, heavily intermixtures with African Americans), which can raise difficulties in trying to determine biological differences. Josephine Johnston, *Resisting a Genetic Identity: The Black Seminoles and Genetic Tests of Ancestry*, 31 J.L. MED. & ETHICS 262 (2003).

We use “white” to indicate the population group with its origins in Europe. While that group is sometimes called “Caucasians,” this pseudo-scientific name is problematic, in no small part precisely because it suggests a biological construction, rather than a social construction, of race. We have chosen “white” precisely because it lacks this “scientific” connotation, although we recognize that it is scarcely well-chosen as a descriptor of any, much less the range of, pigmentation in peoples originating most recently in Europe.

vide no more of a consistent answer, and biology and genetics also provide little help. In this Part, we begin by tracing the historical meaning of “race” before discussing the evolving social construction of that term. We then turn to what light biology and genetics can shed on the definition of race.

A. Definitions: Historical and Present

The definition of “race” remains one of the most important yet contested ideas in American life. As the Oxford English Dictionary notes, “[t]he term is often used imprecisely; even among anthropologists there is no generally accepted classification or terminology.”⁶³ This confusion over the meaning of “race” spans time and academic fields.

Much of the confusion stems from an inability to agree upon the criteria used to distinguish races. As others have noted, there are no “natural” races. Races, instead, are social constructions, the criteria for which must be agreed upon by society.⁶⁴ Historically, much of the debate has ranged over whether race should be defined simply by physical characteristics and/or through common ancestry.⁶⁵

In addition, confusion exists as to how narrowly or broadly one should divide races. Arbitrarily selecting a criterion or criteria will not suffice. So, for instance, racial divisions based solely on hair color could result in any number of races. If one wanted to create two races, humans could be divided into “blonds” and “non-blonds.”⁶⁶ From that point, the number of races could be almost infinitely expanded by sub-dividing these two categories further by shade: “brunettes,” “red heads,” “strawberry blonds,” etc. Indeed, the same is true about any physical or ancestral criterion. Thus, an indeterminate number of races is always possible. In modern America, of course, we have become used to race, distinguishing only among a small number of groups, but this has not always been the case.

The Supreme Court’s opinion in *Saint Francis College v. Al-Khazraji*⁶⁷ illustrates how different means of defining race have led to confusion. The case arose as a suit by a United States citizen who had been born in Iraq and claimed that he was denied tenure at St. Francis College based on his Arab ancestry.⁶⁸ He sued under 42 U.S.C. § 1981, which prohibits racial discrimination in making contracts.⁶⁹ The district court denied Al-

⁶³ OXFORD ENGLISH DICTIONARY 69 (2d ed. 1989).

⁶⁴ See generally Daniel J. Sharfstein, Essay, *The Secret History of Race in the United States*, 112 YALE L.J. 1473, 1477–84 (2003) (summarizing literature).

⁶⁵ See Adrienne D. Davis, *Identity Notes Part One: Playing in the Light*, 45 AM. U. L. REV. 695, 706 (1996) (noting these two approaches).

⁶⁶ Cf. OLSON, *supra* note 51, at 133 (noting the illogic of labeling “blonds” as a race).

⁶⁷ 481 U.S. 604 (1987).

⁶⁸ *Id.* at 606.

⁶⁹ The statute does not, in so many words, bar race discrimination, but instead requires that all persons within the United States shall have the same rights to make contracts (among other things) as white citizens. See 42 U.S.C. § 1981(a) (2000). However, the statute has

Khazraji's claim because Arabs are generally considered Caucasians.⁷⁰ The Supreme Court rejected this view as inconsistent with the understanding of "race" when § 1981 was enacted:

In the middle years of the 19th century, dictionaries commonly referred to race as a "continued series of descendants from a parent who is called the *stock*," "[t]he lineage of a family," or "descendants of a common ancestor." The 1887 edition of Webster's expanded the definition somewhat: "The descendants of a common ancestor; a family, tribe, people or nation, believed or presumed to belong to the same stock." It was not until the 20th century that dictionaries began referring to the Caucasian, Mongolian and Negro races, or to race as involving divisions of mankind based upon different physical characteristics. Even so, modern dictionaries still include among the definitions of race "a family, tribe, people, or nation belonging to the same stock."⁷¹

The *Al-Khazraji* Court thus chose ancestry as the proper criterion for "race," at least in interpreting § 1981 as its nineteenth-century drafters would have understood the term, but then hedged somewhat by referring to physical traits:

Congress intended to protect from discrimination identifiable classes of persons who are subjected to intentional discrimination solely because of their ancestry or ethnic characteristics The Court of Appeals was thus quite right in holding that § 1981, "at a minimum," reaches discrimination against an individual "because he or she is genetically part of an ethnically and physiognomically distinctive sub-grouping of *homo sapiens*." It is clear from our holding, however, that a distinctive physiognomy is not essential to qualify for § 1981 protection.⁷²

In sum, the Court concluded that in the middle of the nineteenth century, "race" was not necessarily connected to distinctive physical traits, sug-

been held to bar racial discrimination. See *McDonald v. Santa Fe Trail Transp. Co.*, 427 U.S. 273, 286–87 (1976).

⁷⁰ *Al-Khazraji*, 481 U.S. at 606.

⁷¹ *Id.* at 610–11 (citations omitted).

⁷² *Id.* at 613.

gesting that race is defined by ancestry.⁷³ But the Court's use of the words "not essential" may signal that race can *also* be defined by physical traits.⁷⁴

The Court's decision in *Al-Khazraji* also required a decision as to how finely to divide the races. After concluding that race was defined (at least primarily) by ancestry, the Court could have lumped people of North African and Arab descent together with Europeans as "White" or "Caucasian." The Court, however, instead took a much more fine-grained approach to race. While the nineteenth-century sources were "somewhat diverse," "Arabs, Englishmen, Germans, and certain other ethnic groups" were not considered "a single race."⁷⁵ The opinion also noted references to "race" for Scandinavians, Chinese, Spanish, Anglo-Saxons, Blacks, Mongolians, and Gypsies.⁷⁶ The result was a definition to create a large number of races.

Today, *Al-Khazraji*'s definition of race is jarring. Most Americans are accustomed to a conception of race based both on physical traits and in broader terms, that is, fewer races—black, white, Asians, etc.—than *Al-Khazraji* recognized. The disconnect between the modern social view and

⁷³ An expansive view of race has also been taken under the Equal Protection Clause, although the Court has not precisely defined "race" in that context. For example, *Korematsu v. United States*, 323 U.S. 214 (1944), which upheld wartime internment of Japanese-descended Americans, could have been framed as a "national origin" case but was instead viewed as a racial classification.

⁷⁴ Earlier decisions had adopted such an approach. In construing a statute that limited immigration into the United States "to aliens, being free white persons, and to aliens of African nativity and to persons of African descent," the Court held "a high caste Hindu of full Indian blood" not to be "white." *United States v. Bhagat Singh Thind*, 261 U.S. 204, 206–07, 215 (1923). The issue in *Bhagat Singh Thind* was difficult because Indians had been classified as Caucasian or Aryan by some ethnologists:

In the endeavor to ascertain the meaning of the statute we must not fail to keep in mind that it does not employ the word "Caucasian" but the words "white persons," and these are words of common speech and not of scientific origin. The word "Caucasian" not only was not employed in the law but was probably wholly unfamiliar to the original framers of the statute in 1790The words of the statute are to be interpreted in accordance with the understanding of the common man from whose vocabulary they were taken

They imply, as we have said, a racial test; but the term "race" is one which, for the practical purposes of the statute, must be applied to *a group of living persons now possessing in common the requisite characteristics*, not to groups of persons who are supposed to be or really are descended from some remote, common ancestor, but who, whether they both resemble him to a greater or less extent, have, at any rate, ceased altogether to resemble one another. It may be true that the blond Scandinavian and the brown Hindu have a common ancestor in the dim reaches of antiquity, but the average man knows perfectly well that there are unmistakable and profound differences between them today

Id. at 208–09 (emphasis added). So approached, the answer was easy: Indians were not "White." *Id.* at 213–14.

⁷⁵ *Al-Khazraji*, 481 U.S. at 612.

⁷⁶ *Id.*

the conception articulated in *Al-Khazraji* raises the question: how could we have moved so far in only a century?

As the *Al-Khazraji* opinion suggests, nineteenth-century scientists were themselves unable to use the term in a consistent fashion. From a scientific perspective, the notion of distinctive races emerged from Carolus Linnaeus, the great taxonomer, who attempted in the eighteenth century to classify the human species as comprised of four races which we might roughly translate as Native American, European, Asian and African.⁷⁷ Linnaeus's divisions were "defined primarily by geography"; that is, the most important piece of information in determining a person's race was where she came from (and presumably who her ancestors were).⁷⁸ This division, however, was "refined" by J. F. Blumenbach in his influential *On the Natural Variety of Mankind*, published in 1795, which recognized five races: Caucasian, Mongolian, Ethiopian, American, and Malay.⁷⁹ As Stephen Jay Gould notes, Blumenbach's approach was revolutionary because it relied not just on geography but also on physical appearance as an indicator of race.⁸⁰ Blumenbach, however, recognized that when race was viewed this way, "all [the races] do so run into one another, and that one variety of mankind does so sensibly pass into the other, that you cannot mark out the differences between them."⁸¹

This early classification system provided a useful rough-and-ready sorting that remains embedded in conventional wisdom today, continuing to have a major impact on how race is viewed. First, as Gould notes, the work of Blumenbach and others transformed the criterion for determining race from ancestral to physical, although the importance of ancestry has never completely faded. Second, and perhaps just as importantly, the result was a much less fine-grained approach to race. As the *Al-Khazraji* Court indicated, over time race has been popularly transmuted from meaning almost any distinctive group into three "major" races, which, by the middle of the twentieth century, were usually called Caucasian, Mongo-

⁷⁷ See GOULD, *supra* note 50, at 404; see also Donald Braman, *Of Race and Immutability*, 46 UCLA L. REV. 1375, 1386–92 (1999) (sketching scientific and political objections to taxonomic efforts to define race).

⁷⁸ See GOULD, *supra* note 50, at 404. Of course, geography is not precisely the same as ancestry because people sharing a similar ancestry may find themselves in quite distinct geographic locations.

⁷⁹ According to Gould, Blumenbach not only recognized a fifth division—the Malay variety, which included Polynesians, Melanesians, and Australian aborigines—but also was the originator of the term "Caucasian" for people from Europe and adjacent areas. *Id.* at 402–03.

⁸⁰ See *id.* at 403.

⁸¹ *Id.* at 407. The Court in *Bhagat Singh Thind*, therefore, was correct when it noted that "[t]he word 'Caucasian' not only was not employed in the law but was probably wholly unfamiliar to the original framers of the statute in 1790." 261 U.S. at 208. The word did not even exist as a signifier for race at the time.

lian, and Negro, at least when the terms were used descriptively rather than pejoratively.⁸²

This general paradigm—race defined by a combination of physical traits and ancestry (but more heavily relying on the former) and limited to a small number of races—has persisted into the twenty-first century. The only significant change has been increased recognition of Asians and Native Americans as separate races.

Commentators agree that American society continues to define race at least partly (or perhaps mostly) through reference to physical characteristics. For instance, Michael Omi and Howard Winant define race as “a concept which signifies and symbolizes social conflicts and interests by referring to different types of human bodies.”⁸³ Ian Haney López argues that the construction of racial identity is a complex matter but notes the importance of physical traits in facilitating that process: not only do physical traits determine the race into which others will categorize us but it also constrains our individual ability to construct our own racial identity.⁸⁴ Adrienne Davis also recognizes the importance of physical traits, along with ancestry, in racial classifications.⁸⁵

Despite the tendency for *society* to rely on physical traits, contemporary American *law* remains ambivalent about the “proper” criterion for race. While *Al-Khazraji* ensures that § 1981 uses an ancestral criterion for determining race, the term, as used in the Civil Rights Act of 1964, might not be interpreted the same way. The *Al-Khazraji* Court relied upon the understanding of the term at the time the statute was enacted during Reconstruction. By the time the 1964 Act was passed, however, the contemporary understanding of the term had changed, both in respect to the criteria and the number of races. It is possible, therefore, that *Al-Khazraji* had a claim for “race” discrimination under § 1981 but not under the 1964 Civil Rights Act.⁸⁶

Furthermore, the federal government clearly attaches a different meaning to “race” in other contexts. For instance, the guidelines for the 2000 Census (which apply to all federal data collection on race and ethnicity), much like the Court’s view of § 1981, appear premised on an ancestral definition of race: all of the racial categorizations make reference to the “origins” of the person or the “original peoples” of a geographic loca-

⁸² *Al-Khazraji*, 481 U.S. at 610–11.

⁸³ MICHAEL OMI & HOWARD WINANT, *RACIAL FORMATION IN THE UNITED STATES: FROM THE 1960S TO THE 1990S*, at 55 (2d ed. 1994) (emphasis omitted).

⁸⁴ See Haney López, *supra* note 48, at 39–53.

⁸⁵ See Davis, *supra* note 65, at 705.

⁸⁶ *Al-Khazraji* presumably could have asserted a claim for national origin discrimination under Title VII. The distinction between race and national origin can be legally significant. A race discrimination claim is subject to no statutory defense, while national origin discrimination may be justified as a bona fide occupational qualification. See discussion *infra* note 323.

tion.⁸⁷ But unlike § 1981, the Census guidelines employ a more “modern” limitation on the number of races: they refer to only five separate racial groups, plus one ethnic group—American Indian/Alaskan Native, Asian, Black/African American, Native Hawaiian/other Pacific Islander, White, and Hispanic/Latino.⁸⁸

Another federal statute, 18 U.S.C. § 1093, dealing with crimes against certain groups, goes a step further and defines race in a way less consistent with *Al-Khazraji* but more consistent with societal conventions by using a combination of ancestry and physical traits: “[T]he term ‘racial group’ means a set of individuals whose identity as such is distinctive in terms of physical characteristics or biological descent.”⁸⁹ Left unstated, however, is how finely to divide the races under § 1093.

The bottom line is that contemporary American law, even more than contemporary American society, is ambiguous about the “true” definition of race. In practice, race appears to be socially constructed through a complex process but most often relies on physical traits. In this Article, however, we will use the term “race” as defined in the Census guidelines. Thus, we use a definition of race that turns on ancestry, rather than physical traits, and we also accept the relatively small number of races used in the guidelines. This definition of race, we believe, is the most relevant to medical research for two reasons. First, defining race as a proxy for ancestry is the method most likely to make race salient to scientific and medical research and treatment. Second, although the number of groupings in the Census guidelines is certainly an arbitrary social construction, the groupings are currently the divisions most familiar to Americans and therefore again most likely to be of use to researchers and clinicians. It is only by analyzing such a best-case scenario, we believe, that we can truly judge the legal implications of such use.

In so defining race, we do not mean to validate the social choices that have led to the construction of these races in the first place. Instead, we attempt to evaluate whether “races,” as particular social constructions, might have any legitimate value to medical research and treatment and to what extent the law presently permits such uses.

B. The Genetics of Race

Almost since the conception of modern evolutionary theory by Charles Darwin and Alfred Russel Wallace in the middle of the nineteenth century, people have attempted to use it to validate racial distinctions.⁹⁰ In

⁸⁷ See Standards for the Classification of Federal Data on Race and Ethnicity, 62 Fed. Reg. 58,782, 58,788 (Oct. 30, 1997) (setting forth racial and ethnic standards for 2000 Census).

⁸⁸ *Id.* at 58,789.

⁸⁹ 18 U.S.C. § 1093(6) (2000).

⁹⁰ Among the first was Darwin’s cousin, Francis Gaulton, who advocated selective

particular, some have sought to show that races (however defined) differ not just in skin color and physiognomy, but also various other traits.⁹¹ The nineteenth and twentieth centuries saw recurrent efforts to establish the inferiority of races in terms of intelligence,⁹² with recent echoes suggesting that racial intellectual capabilities differ as a result of genetics.⁹³ Such thinking could obviously carry over to medicine as well: some have suggested that various genetic diseases could be more prevalent in some races than in others.⁹⁴

There are strong reasons to believe, however, that there are relatively few important genetic differences between races, even when race serves as a proxy for ancestry.⁹⁵ From the standpoint of genetics, the races are generally indistinguishable. There are, of course, variations, particularly for the physiological characteristics that we generally associate with a particular race, such as skin color and physiognomy.⁹⁶ And as we will note, there are variations in the distribution of some genes between races, although not so great as to make any particular gene associated with a particular race.⁹⁷ But with only a few exceptions, the variation *within* a race for a given trait is much greater than the variation *across* races.⁹⁸

Race, therefore, even when defined solely by ancestry, is generally meaningless as an independent concept in genetics: there is no set of genes that can tell us whether an individual is or is not a member of a particular race.⁹⁹ Race can serve as a proxy for skin color and physiognomy. But, even as a way of searching for the prevalence of other genetic traits, race is generally unlikely to be useful. Most biologists doubt in *most* cases that an individual's ancestry will tell us much, if anything, about the likelihood of another trait in an individual's genome.¹⁰⁰

breeding as a means of "improving" the human species. See MATT RIDLEY, *GENOME* 288 (1999). Others were quick to jump from the idea of "improving" the species to "improving" nations, and in the United States, this became a justification for excluding racial and ethnic groups who were perceived as having inferior genes. See *id.* at 289–90. For a general critique of such views, see *id.* at 290–300.

⁹¹ See *infra* text accompanying note 172.

⁹² See generally GOULD, *supra* note 50.

⁹³ See HERRNSTEIN & MURRAY, *supra* note 8.

⁹⁴ See, e.g., MARK H. SWARTZ, *TEXTBOOK OF PHYSICAL DIAGNOSIS: HISTORY AND EXAMINATION* 54–55 & tbl.3.1 (4th ed. 2002) (noting, in textbook for first-year medical students, ethnic and geographic distribution of diseases, including genetic ones).

⁹⁵ TIMOTHY H. GOLDSMITH & WILLIAM F. ZIMMERMAN, *BIOLOGY, EVOLUTION AND HUMAN NATURE* 290 (2001). When race is defined in other manners, the amount of difference among the races is even smaller.

⁹⁶ LUIGI LUCA CAVALLI-SFORZA, *GENES, PEOPLE AND LANGUAGES* 9–13 (2000).

⁹⁷ As we will explain, what we really mean here is "allele" and not gene. See *infra* text accompanying notes 104–199.

⁹⁸ OLSON, *supra* note 51, at 63.

⁹⁹ A somewhat unusual attempt to use genetics to define not race per se, but membership in a particular Indian tribe is recounted in Johnston, *supra* note 62, at 262.

¹⁰⁰ See PAUL EHRLICH, *HUMAN NATURES: GENES, CULTURES, AND THE HUMAN PROSPECT* 293–94 (2000); GOLDSMITH & ZIMMERMAN, *supra* note 95, 289–90.

Nonetheless, in *particular cases*, members of groups defined by ancestry will have greater or lesser frequencies of certain genes compared to members of other ancestry-based groups. Two common examples are the sickle cell anemia gene in African Americans and the Tay-Sachs gene among Ashkenazi Jews.¹⁰¹ In both cases, the particular gene exists in a higher frequency among members of the group as the result of specific evolutionary, biological and statistical events. Race can, then, help predict the likelihood that an individual will have that gene, if race is coextensive with that group. The link between sickle cell anemia and African Americans is an example of such a situation. More frequently, though, the relevant groups will be a subset of a race, as it is with Tay-Sachs, where the relevant ancestry-based group is Ashkenazi Jews, not whites more generally.

This Section begins by first discussing some basics of genetics and human evolution. It then turns to modern genetic theory to describe both why most genes are unlikely to significantly vary in frequency between races, and why some variation is inevitable. As modern humans have spread across the Earth over the last 100,000 to 200,000 years, there has been little time (in genetic terms) for significant variations between groups to emerge. Nonetheless, under certain conditions, some differences may emerge and be observable on average between members of different races.

1. *Genes and Human Evolution*

a. *Primer on Genes*

Human beings, by current estimates, have between 26,000 and 40,000 separate genes,¹⁰² spread across twenty-three chromosomes, with each human carrying two copies of each chromosome.¹⁰³ Thus, for most genes, each of us actually has two copies, or *alleles*.¹⁰⁴ When a person has two identical alleles of a particular gene (that is, has the same allele on both chromosomes), the person is *homozygous* for that particular trait.¹⁰⁵ When

¹⁰¹ See *infra* notes 203–206 and accompanying text.

¹⁰² Nicholas Wade, *Human Genome Appears More Complicated*, N.Y. TIMES, Aug. 24, 2001, at A12. This number is actually far fewer than previously thought (about 100,000), a result that has provoked some consternation that our genome appears not to be all that more complex than the genome of the common fruit fly. *Id.*: see also RIDLEY, GENOME, *supra* note 90, at 4 (estimating 60,000 to 80,000 genes). That number, though, may mask a far larger number of uses to which these genes are put. See MATT RIDLEY, NATURE VIA NURTURE: GENES, EXPERIENCE AND WHAT MAKES US HUMAN 33 (2003) [hereinafter RIDLEY, NATURE VIA NURTURE]; see also *id.* at 141 (describing how this process may work).

¹⁰³ The major exception is that men contain two copies of twenty-two chromosomes, but one copy of the X chromosome and one copy of the Y chromosome (women contain two copies of the X chromosome, but no Y chromosome). OLSON, *supra* note 51, at 14–15.

¹⁰⁴ See GOLDSMITH & ZIMMERMAN, *supra* note 95, at 76.

¹⁰⁵ *Id.* at 87.

the person has two different alleles of the gene, the person is *heterozygous* for that trait.¹⁰⁶

For example, one of the first genes studied extensively was that for sickle cell anemia, a disease that results from a malfunction in the gene that codes for the hemoglobin protein. We will refer to the allele for the normal hemoglobin molecule as *A* and to the allele for the sickle cell hemoglobin molecule as *S*. Someone who has two copies of the normal hemoglobin allele is denoted as *AA*; such a person will not develop sickle cell anemia. Conversely, someone who is homozygous for the sickle cell hemoglobin allele, denoted *SS*, will develop the disease.

But what happens when someone is heterozygous for the hemoglobin gene, that is, has one copy of the normal allele and one copy of the sickle cell allele? Such a person, denoted *AS*, does *not* usually develop the disease.¹⁰⁷ This is because the normal hemoglobin allele is said to be *dominant* and the allele for the sickle cell hemoglobin molecule is *recessive*. In other words, dominant alleles can be understood as “drowning out” the recessive alleles when a person has one copy of each.¹⁰⁸ This characteristic of the sickle cell hemoglobin allele explains its continued presence in humans.¹⁰⁹

b. Evolution: Genes over Time

Genes are the result of billions of years of evolution, during which genes have in a sense been competing to reproduce themselves. Alleles that promote the reproductive success of the organism will tend to reproduce themselves and spread; those that undermine reproductive success will tend to disappear.¹¹⁰

The result of this ongoing competition is evolution. Stated simply, the theory of evolution supposes that all organisms descend from preexisting organisms, such that all life on Earth today is descended from earlier forms of life.¹¹¹ That thought predates Darwin and Wallace.¹¹² The Darwinian explanation that evolution occurs by natural selection is more controversial:

¹⁰⁶ *Id.*

¹⁰⁷ In fact, the sickle cell genes are properly described as co-dominant, because people who are heterozygous manufacture both the normal and sickle cell forms of the hemoglobin protein. NEIL A. CAMPBELL, *BIOLOGY* 274 (3d ed. 1993). Under certain conditions, people who are heterozygous do develop sickle cell anemia. See Martin H. Steinberg, *Sickle Cell Trait*, in *DISORDERS OF HEMOGLOBIN* 811, 818–19 (Martin H. Steinberg et al. eds., 2001).

¹⁰⁸ See GOLDSMITH & ZIMMERMAN, *supra* note 95, at 86–88.

¹⁰⁹ See *id.* at 69; EHRlich, *supra* note 100, at 41–42.

¹¹⁰ See GOLDSMITH & ZIMMERMAN, *supra* note 95, at 96.

¹¹¹ See CAMPBELL, *supra* note 107, at 420, 427. See generally WILLIAM S. KLUG & MICHAEL R. CUMMINGS, *CONCEPTS OF GENETICS* 526–48 (1983).

¹¹² It dates back to Jean-Baptiste de Lamarck. See JEFFREY K. MCKEE, *THE RIDDLED CHAIN* 192 (2000).

If within a species there is variation among individuals in their hereditary traits, and some traits are more conducive to survival and reproduction than others, then those traits will (obviously) become more widespread within the population. The result (obviously) is that the species' aggregate pool of hereditary traits changes.¹¹³

In other words, "a population of organisms can change over time as a result of individuals with certain heritable traits leaving more offspring than other individuals."¹¹⁴ The heritable traits are, of course, alleles; so, where different alleles lead to different phenotypes,¹¹⁵ one of which is more successful at surviving than others, that allele should tend to predominate over time, if not fully eliminate, the other alleles.¹¹⁶

To illustrate, imagine an allele that causes someone who possesses it to die by age five (denoted allele C),¹¹⁷ and assume that this allele is dominant. Assume there is another allele of the gene, denoted c , that does not lead to infant death and that this c allele is recessive. Finally, assume that both alleles are equally distributed among the population. Absent any other factors, a population of sixteen people would be distributed by: four with the genotype CC ; eight with Cc ; and four with cc .¹¹⁸ No individual would have the C allele in the next generation because everyone with the allele (the four who are CC and the eight who are Cc) would die by the age of five and would not reproduce; the only people contributing alleles to the next generation would be those who are cc .¹¹⁹ Thus, absent some other source for new copies of C , it should be eliminated in one generation.

In reality, of course, things are far more complicated than this illustration suggests, and "less fit" alleles can persist in a population in a number of circumstances. For instance, recessive lethal alleles are far harder

¹¹³ ROBERT WRIGHT, *THE MORAL ANIMAL* 23 (1994).

¹¹⁴ CAMPBELL, *supra* note 107, at 420.

¹¹⁵ "Phenotype" refers to the observable, physical characteristics of an organism; in contrast, a "genotype" refers to the organism's genetic makeup.

¹¹⁶ As we will explain below, we are not arguing that evolution is driven only by natural selection. Indeed, our thesis is that natural selection is not the most important evolutionary force at work in connection with racial disparities in health status. We take no position on the broader controversy in evolutionary biology over the relative importance of natural selection and other forces for evolution generally. Having acknowledged this controversy, however, we want to be clear that these debates are not over the validity of evolution more generally.

¹¹⁷ The gene for cystic fibrosis is just such a gene, although the following discussion does not apply to that gene, which is recessive and not dominant. See RICHARD DAWKINS, *THE SELFISH GENE* 273 (new ed. 1989).

¹¹⁸ This follows from the Hardy-Weinberg equation. See ERIC C. PIANKA, *EVOLUTIONARY ECOLOGY* 117-18 (6th ed. 1999); see also PAUL R. EHRLICH & JONATHAN ROUGHGARDEN, *THE SCIENCE OF ECOLOGY* 110 (1987).

¹¹⁹ See JONATHAN ROUGHGARDEN, *THEORY OF POPULATION GENETICS AND EVOLUTIONARY ECOLOGY: AN INTRODUCTION* 31 (1979). For an illustration, we need to make an assumption about fertility of the individuals who reach mating age. Assuming four "children," the next generation will be made up of eight individuals, all with the genotype cc .

to eliminate from a population than dominant ones.¹²⁰ Assume the same illustration, but instead assume that the dominant *C* allele is normal and the recessive *c* allele leads to our disease. If *c* made up one-half of the alleles in the first generation, then about seventy-five percent of the population carries the allele, either in homozygous or heterozygous form. In the next generation, the allele will be about one-third of the alleles. In the following generation, the *c* allele will continue to decline but will still constitute one-quarter of the overall gene pool. Thus, even after two generations, a significant portion of the population will still carry the *c* allele, even though all of those who were homozygous for it died by the age of five.¹²¹ Instead, as the carrier rate falls, the decline in the carrier rate becomes quite small.¹²²

It is also hard to remove any gene that is less fatal than our example. If the fitness of individuals who are homozygous recessive is merely one-half that of the other individuals, after one generation over sixty-five percent of the population will still carry the *c* allele and almost sixty-one percent will still carry it after two generations. Thus, even in the face of relatively strong selective pressures, it can take many, many generations to remove a widespread recessive allele from a gene pool.¹²³ Similarly, if we assume instead that the dominant gene is selected against but is not fatal at an early age, it can still take a number of generations to remove the allele.¹²⁴

Finally, there is the problem of heterozygote superiority, of which the sickle cell allele is an example. In an environment where malaria from bacterium of the species *Plasmodium falciparum* is present, the genotype *AS* is superior to both *AA* and *SS*: *SS* is obviously bad because it leads to sickle cell anemia, but *AA* is also bad because it provides no protection against malaria, whereas *AS* does. The population will tend to equilibrium at a proportion of *A* and *S* that is a function of the relative selective disadvantages of *AA* and *SS*.¹²⁵

¹²⁰ See DOUGLAS FUTUYAMA, *EVOLUTIONARY BIOLOGY* 379 fig.13.13 (3d ed. 1998) (graphing decline in recessive allele over twelve generations); GOLDSMITH & ZIMMERMAN, *supra* note 95, at 99.

¹²¹ The exact percentage depends upon assumptions about the fertility and survival rates of zygotes.

¹²² See GOLDSMITH & ZIMMERMAN, *supra* note 95, at 99 (noting that with carrier rate of 1:1000, overall rate should only decline to 0.89 in 1000 after 250 generations).

¹²³ See ROUGHGARDEN, *supra* note 119, at 31–35 fig.3.3 (modeling the difficulty of eliminating a less-fit recessive gene). All of this assumes that selection is working on just one gene, not many. As the number of genes increases, the process of natural selection becomes even more difficult and slower.

¹²⁴ See *id.* at 31–32 fig.3.2. Computer modeling suggests that, when the fitness of the dominant allele is one-half that of the recessive allele, it will take over twenty generations to eliminate the dominant allele if its initial proportion is .9. Even if the starting proportion is .5, it may take nearly ten generations to eliminate the allele. *Id.*

¹²⁵ See *id.* at 36–40 for a detailed explanation of the mathematics involved. We have not attempted to catalog all the complications with natural selection as an evolutionary force. For instance, there is also the problem of heterozygote inferiority, which we will

In the absence of strong selection, three other factors can also influence allelic distributions in populations: mutation, genetic drift and gene flow (or migration).¹²⁶ First, mutations occur when the DNA for a particular allele is altered and the alteration results in the coding of an addition and/or deletion of one or more amino acids.¹²⁷ Mutations occur constantly and are a source of some genetic variation in populations.¹²⁸

Second, genetic drift can play a large role in genetic distributions. As Professor Luigi Luca Cavalli-Sforza has stated, genetic drift “is really nothing more than the chance fluctuation of gene frequencies over several generations.”¹²⁹ Consider again our example of alleles *C* and *c*. In the second iteration, we imagined a scenario in which *c* was very unfit and predicted its decline in the population in each succeeding generation. But this was only a statistical prediction; as a matter of statistical chance,¹³⁰ the proportion of *c* could actually increase if carriers of the *c* allele randomly had more offspring. But over time, statistical probabilities should prevail, absent some countervailing reason that causes individuals with the less fit allele to be evolutionarily successful.

The power of genetic drift is a function of both population size and time: a smaller population size will increase genetic drift, as will a longer time-frame.¹³¹ Genetic drift is very unlikely to have a significant effect on short-term genetic variation among population groups.¹³² The exception occurs when a population group is quite small, leading to the “founders’ effect.”¹³³ The smaller a group, the more likely are great fluctuations from the statistical norm in the distribution of genes in that group. If the “found-

ignore. *See id.* at 40–43.

¹²⁶ *See* GOLDSMITH & ZIMMERMAN, *supra* note 95, at 95–96; PIANKA, *supra* note 118, at 124; *see also* MCKEE, *supra* note 112, at 140–42 (detailing mutation, gene flow, and genetic drift). Meiotic drive, or segregation distortion, can also influence genetic distributions, but this rarely occurs. PIANKA, *supra* note 118, at 116. Other difficulties also exist. The present point is that natural selection is not the only evolutionary force.

¹²⁷ Some changes in DNA produce no change in amino acids because there are multiple codons that code for the same amino acid. Thus, the substitution of one base pair for another may have no effect on the gene. Furthermore, even if a new amino acid were substituted in by the mutation, this new amino acid may have no effect on the construction of the protein; if it does not, then no physiological change would occur. *See* MCKEE, *supra* note 112, at 136.

¹²⁸ *See* FUTUYAMA, *supra* note 120, at 275.

¹²⁹ CAVALLI-SFORZA, *supra* note 96, at 42.

¹³⁰ Professor Jake Barnes, our colleague at Seton Hall Law School tells us that “chance” or “random chance” may simply be descriptions for non-random causes that we are not yet able (and may never be able) to identify. For our purposes, however, the term serves to indicate the kinds of fluctuations—whether in mutations, differential reproductive successes of individual members of a species, or other factors—that cannot otherwise be accounted for at our present state of knowledge.

¹³¹ *See* FUTUYAMA, *supra* note 120, at 301 (noting that drift occurs faster in smaller populations); ROUGHGARDEN, *supra* note 119, at 60–63 figs.5.2 & 5.3 (documenting the larger number of generations needed for genetic drift to homogenize a population as population size increases).

¹³² *See* MCKEE, *supra* note 112, at 140–41.

¹³³ CAVALLI-SFORZA, *supra* note 96, at 42–43.

ing group” then expands rapidly (especially if it is endogamous, that is, in-breeding), this initial gene fluctuation may well be preserved in future generations. Within human populations, the founders’ effect appears to explain some variation in genetic distribution of particular alleles, such as the prevalence of a number of genetic defects within the Old Order Amish communities in Pennsylvania and Ohio.¹³⁴

Over longer periods of time, genetic drift will affect the distribution of alleles even between larger population groups. Imagine, for instance, that there is no difference in fitness between the two alleles *C* and *c*, and suppose two population groups completely separated from one another. Over time, simply as a matter of chance, the first population may eliminate the *C* allele, and the other population may eliminate the *c* allele.¹³⁵ This is known as the “tendency to homogenize”: absent other factors, a population will tend to eliminate all but one genetic group.¹³⁶ This homogenization may occur even absent any difference in fitness between the two alleles.

The final cause of evolution, gene flow, works against genetic drift and natural selection: gene flow occurs when genes are exchanged between population groups as a result of exogamy (that is, outbreeding).¹³⁷ Because members of a population group (usually) do not reproduce solely with other members of that group, alleles are exchanged between population groups.¹³⁸ As a result, alleles that might have been eliminated by genetic drift or natural selection can be re-established in a population group.¹³⁹ Indeed, only through limiting gene flow can the genetic mixture of population groups vary.¹⁴⁰ The result is some difference in genetic distributions between different population groups, although this difference is very rarely so great as to eliminate a particular allele (in the absence of complete endogamy).

In sum, a variety of factors—natural selection, mutation, genetic drift, and gene flow—can affect the frequency of particular alleles in populations. Natural selection can increase the variance between particular groups, but only where those groups are subject to different selection pressures. Genetic drift can also lead to differences, but these should be slight so long as there is migration of alleles between the two groups. Finally, mutations can increase genetic variation by placing a particular allele in one

¹³⁴ See Francis X. Clines, *Research Clinic Opens in Ohio for Genetic Maladies that Haunt Amish Families*, N.Y. TIMES, June 20, 2002, at A22.

¹³⁵ See CAVALLI-SFORZA, *supra* note 96, at 43.

¹³⁶ *Id.*; FUTUYAMA, *supra* note 120, at 301–02 (noting that drift tends to eliminate genetic variation).

¹³⁷ See GOLDSMITH & ZIMMERMAN, *supra* note 95, at 95.

¹³⁸ FUTUYAMA, *supra* note 120, at 314.

¹³⁹ See PIANKA, *supra* note 118, at 119.

¹⁴⁰ See FUTUYAMA, *supra* note 120, at 314 (noting that gene flow homogenizes the population groups of a species); Guido Barbujani & Robert R. Sokal, *Zones of Sharp Genetic Change in Europe are Also Linguistic Boundaries*, 87 PROC. NAT’L ACAD. SCI. 1816 (1990) (citing J. A. ENDLER, GEOGRAPHIC VARIATION, SELECTION, AND CLINES (1977)).

population but not the other; but they can also decrease variation because of a recurrent mutation that re-establishes an allele in a population.

c. Human Evolution

As to the effects of evolution on humans, in particular, our ancestors branched off from the chimpanzees and bonobos—our closest relatives—somewhere between five million and nine million years ago.¹⁴¹ The resulting “protohumans” evolved into *Homo sapiens* about 500,000 years ago, although these early humans were different from modern humans in both physiognomy and culture.¹⁴² Differences also arose among the *H. sapiens* populations in Africa, East Asia, and Western Eurasia. By about 100,000 years ago, the skeletons of humans in Africa were far closer to those of fully modern humans than their contemporaries elsewhere, such as the Neanderthals.¹⁴³

It is still uncertain what exactly occurred next. By about 40,000 years ago fully modern humans had spread throughout much of the planet.¹⁴⁴ Under the dominant “Out-of-Africa” theory,¹⁴⁵ somewhere between 100,000 and 50,000 years ago, there was some change, linked either to brain organization or to physical language abilities, that allowed for the evolution of fully modern humans in Africa.¹⁴⁶ These modern humans then left Africa between 40,000 and 50,000 years ago and spread, first to Asia, then to Europe and Australia, displacing the populations of other humans (such as the Neanderthals) that had previously lived in those places.¹⁴⁷

These early modern humans were hunter-gatherers. Such societies tended to be small, between twenty-five to fifty people,¹⁴⁸ and limited in

¹⁴¹ JARED DIAMOND, GUNS, GERMS, AND STEEL 36 (1998); RIDLEY, NATURE VIA NURTURE, *supra* note 102, at 10. The most recent discovery may place the divergence at seven million years ago, although it is not certain that this fossil is a direct ancestor of either chimpanzees or humans. See Michel Brunet, et al., *A New Hominid From the Upper Miocene of Chad, Central Africa*, 418 NATURE 145 (2002).

¹⁴² See, DIAMOND, GUNS, GERMS, AND STEEL, *supra* note 141, at 38.

¹⁴³ See *id.* at 38–39.

¹⁴⁴ By this time, the only unpopulated areas appear to have been the Americas, Antarctica, and the Pacific Islands. See *id.* at 37 fig.1.1.

¹⁴⁵ See, e.g., Leonard Lieberman & Fatimah Linda C. Jackson, *Race and Three Models of Human Origin*, 97 AM. ANTHROPOLOGIST 231 (1995) (labeling the three competing theories as “Out of Africa,” “Multi-regional Continuity,” and “Afro-European Sapiens”). The Multiregional Continuity theory, the main alternative to Out-of-Africa, suggests that modern humans evolved simultaneously in several different locations, a process enabled by gene flow. *Id.* at 236. Under this theory, evolution took place throughout Europe, Africa and Asia in one inter-connected process over a million years. *Id.* A full response to this theory is beyond the scope of this Article, but this theory, even if correct, would not change any of our conclusions. See discussion *infra* note 194.

¹⁴⁶ See, e.g., EHRLICH, *supra* note 100, at 161–62. The exact change has been hotly debated among biologists and anthropologists, among others.

¹⁴⁷ See DIAMOND, GUNS, GERMS, AND STEEL, *supra* note 141, at 40–41.

¹⁴⁸ EHRLICH, *supra* note 100, at 173.

their ability to grow rapidly, particularly compared to later agricultural societies, where women could bear a child once every two years.¹⁴⁹

Sometime in the last 10,000 years, after humans abandoned the hunter-gatherer lifestyle, agricultural societies spread rapidly from their points of origin.¹⁵⁰ Within 4000 years, agriculture spread from the Near East through Europe.¹⁵¹ This expansion may have occurred primarily through the growth of agricultural communities, rather than the transfer of agricultural technologies from such communities to hunter-gatherers.¹⁵² Within 10,000 years, agricultural communities came to dominate the planet.

This human expansion out of Africa, followed by a second agricultural expansion, has important ramifications for human genetic evolution. First, it means that all modern humans share ancestors from a population group that dates back only about 150,000 years.¹⁵³ This translates into approximately 7500 generations,¹⁵⁴ and some have placed it as low as 3000 generations.¹⁵⁵ Second, because most present-day humans are descended from the early agricultural communities of the last 10,000 years, a great deal of our genetic heritage is likely to come from those same agricultural populations.¹⁵⁶ The result is that many humans share origins with others in their population group that span back no more than 500 generations.

Over this period genetic variation did occur. Indeed, the variants which have occurred over the last 100,000 to 200,000 years allow us to trace the

¹⁴⁹ In hunter-gatherer societies, constant movement limits reproduction to something like once every four years. JARED DIAMOND, *THE THIRD CHIMPANZEE* 189–90 (1992).

¹⁵⁰ There are at least five points of origin for agriculture: the Middle East, China, Mesoamerica (or central and southern Mexico and nearby areas in Central America), the Andes and the eastern United States. See DIAMOND, *GUNS, GERMS, AND STEEL*, *supra* note 141, at 98. More recently, there is evidence of independent agricultural origins in the highlands of New Guinea. John Noble Wilford, *An Early Heartland of Agriculture Is Found in New Guinea*, N.Y. TIMES, June 24, 2003, at F2.

¹⁵¹ See CAVALLI-SFORZA, *supra* note 96, at 99.

¹⁵² Cavalli-Sforza has been a principal proponent of this idea, and he points to both more modern examples and genetic studies. See *id.* at 101–13. He admits, however, that not all have agreed with his theory. *Id.* at 103.

¹⁵³ The actual date could be somewhat earlier. There are two people who are the last common ancestors of all modern humans for two particular sets of genes. The first is a woman, usually denoted Eve, from whom all of our mitochondrial DNA (“mtDNA”) is descended (except for rare circumstances, all of our mtDNA comes from our mothers). The second is a man, usually denoted Adam, from whom each modern human male received his Y chromosome. Although this may seem counter-intuitive, Adam and Eve may have lived at different times. See *id.* at 77–82. Furthermore, both Adam and Eve likely lived after our actual last common ancestor; they are just each the last common ancestors for a particular part of our genetic heritage, not all of it. See EHRLICH, *supra* note 100, at 99; OLSON, *supra* note 51, at 26.

¹⁵⁴ Ehrlich, for instance, assumes a generational time-span of about twenty years per generation. EHRLICH, *supra* note 100, at 383 n.8; OLSON, *supra* note 51, at 44 (same). Cavalli-Sforza suggests about twenty-five years. CAVALLI-SFORZA, *supra* note 96, at 45 (equating one thousand generations with 25,000 years).

¹⁵⁵ See RIDLEY, *NATURE VIA NURTURE*, *supra* note 102, at 264.

¹⁵⁶ See CAVALLI-SFORZA, *supra* note 96, at 107 (noting that agricultural peoples “would expand to neighboring areas and their genes would not be completely diluted by their migration into Europe”). For a recent review of this view, see OLSON, *supra* note 51, at 163–74.

likely geographical paths of human expansion.¹⁵⁷ Nonetheless, this variation is probably *not* the product of natural selection, but rather the result of genetic drift and mutations as human population groups branched off from one another.¹⁵⁸ Indeed there is little reason to believe that natural selection has played a major role since the advent of agriculture.¹⁵⁹ However, as we will see, some genetic diseases may be linked to relatively uncommon situations where strong selection pressure has been placed upon human population groups.

2. Races, Genes, and Population Groups

Knowing that all organisms are the result of evolution, and that many differences between species can be explained by natural selection, many late-nineteenth- and early-twentieth-century thinkers suggested that differences between human *races* could also be understood through natural selection.¹⁶⁰ The idea has a certain intuitive appeal: we humans have, at least to our own eyes, a great deal of physical variation. Perhaps this physical variation is mirrored by other variations as well, variations that are determined by our genes.

Modern biologists believe this sort of thinking wrong-headed.¹⁶¹ From the perspective of biology, there is no such thing as a “race,” at least as we commonly view the term.¹⁶² Variations in allelic distribution are generally continuous, not discontinuous, and therefore there are no biological rationales for drawing distinctions based on “racial” classifications.

But while a person’s race cannot be derived from observed biological traits, race viewed as a proxy for geographic origin can provide some important genetic information. Genetic studies suggest that, after the migration out of Africa, the populations of the various other continents “branched off” from one another.¹⁶³ The first break was between the present-day populations of Africa and the other continents, followed in turn by the branching off of the population of Oceania¹⁶⁴ from the other conti-

¹⁵⁷ See CAVALLI-SFORZA, *supra* note 96, at 36–42.

¹⁵⁸ This is because of the relatively limited time span involved and the absence of strong selective pressure. See *infra* text accompanying notes 160–162.

¹⁵⁹ See EHRlich, *supra* note 100, at 166; FUTUYAMA, *supra* note 120, at 740.

¹⁶⁰ See, e.g., GOULD, *supra* note 50, at 47–48 (quoting Rev. Josiah Strong in 1900 as stating that races develop over the course of centuries and that, as a result, some are incapable of self-government); *id.* at 205 (noting that Lewis Terman believed that evolution had led to different mathematical ability between races); DANIEL J. KELVES, IN THE NAME OF EUGENICS: GENETICS AND THE USES OF HUMAN HEREDITY 46–47 (1985) (discussing the views of Charles Davenport); *id.* at 74–76 (discussing the views of others). The idea that humans could be divided into a number of races, of supposedly inferior and superior sorts dates back before Darwin. For a discussion of some of the early attempts, see GOULD, *supra* note 50, at 62–104.

¹⁶¹ The most powerful modern critique remains GOULD, *supra* note 50.

¹⁶² See FUTUYAMA, *supra* note 120, at 737–38.

¹⁶³ See GOLDSMITH & ZIMMERMAN, *supra* note 95, at 286–87.

¹⁶⁴ Oceania refers to the populations of Australia and New Guinea. *Id.* at 40. For an ac-

mental populations, then Europe and finally, the split of Asian and Native American populations.¹⁶⁵ Thus, genetic studies show that the population of Oceania is the most divergent from that of Africa.¹⁶⁶

Accordingly, people sharing ancestors of a common geographic origin tend to be more similar in their genetic make-up than people not sharing such ancestors. Thus, to the extent that individuals in a race share a common ancestry, they too are likely to be more similar in their genetic make-up than other individuals. This difference, however, is still quite small,¹⁶⁷ and furthermore probably has more to do with genetic drift and mutations than natural selection. Genetic drift would have been particularly important in the evolution of human genes prior to the advent of agriculture. In hunter-gatherer societies, population size and density are both low, ensuring a population group in which drift will have a stronger impact since the effect of drift is stronger as population size decreases. Low population density should limit significant exogamy, which in turn ensures that the effect of gene flow will be minimized.¹⁶⁸

As Professor Cavalli-Sforza has noted, genetic drift results in the genetic make-up of even neighboring towns and villages often being quite different; as the distance between locations increases, so too does the genetic variation:

A race is a group of individuals that we can recognize as biologically different from others. To be scientifically “recognized,” the differences between a population that we would like to call a race and neighboring populations must be statistically significant according to some defined criteria. The threshold of statistical significance is arbitrary. The probability of reaching significance for a given distance increases steadily with the number of individuals and genes tested.¹⁶⁹

In sum, we can find differences between the genetic make-up of “races”: because the ancestors of these groups come from different geographic places, there are almost certain to be some differences.

None of this, however, justifies defining race in any particular way, much less the way in which our society does. Other classification systems,

count of the likely history of the population groups in the smaller Pacific islands, see DIAMOND, GUNS, GERMS, AND STEEL, *supra* note 141, at 334–53.

¹⁶⁵ CAVALLI-SFORZA, *supra* note 96, at 39. Despite the early break between Africa and the other continents, the distance between the sub-Saharan African population and the European population is relatively low, reflecting likely gene flow between the two populations. *See id.* at 52.

¹⁶⁶ *See id.* at 37.

¹⁶⁷ *See* FUTUYAMA, *supra* note 120, at 737.

¹⁶⁸ *See id.* at 739–40. The flip side to this is that after the advent of agriculture and the resulting population explosion, genetic drift has probably become a smaller force in human evolution. *See* CAVALLI-SFORZA, *supra* note 96, at 206.

¹⁶⁹ CAVALLI-SFORZA, *supra* note 96, at 25.

working at a lower level of generality, could find equally valid differences using the same basic criterion. Instead, this analysis merely shows that, once we have decided to use race as the level of generality at which to discuss geographic origins, it can indicate genetic differences.

At this point, an example might be helpful. Imagine the numbers in the set from one to ten, laid out as follows:

1 2 3 4 5 6 7 8 9 10

If we wanted to subdivide them, we could arbitrarily divide them into five groups perhaps (1,2) (3,4) (5,6) (7,8) (9,10). This grouping can be justified (and weakly at that) only by the decision to subdivide into five groups in the first place. But why not divide into two groups, or three? The same problem bedevils attempts to divide people into races on the basis of genetics. Although we can find differences, there appears to be no particular *biological* reason to believe that our present racial categories are the proper criteria by which to make this categorization.

It is true that there are discontinuities in the variation of the genetic make-up of groups that might justify particular divisions between groups. So while genetic variation increases with geographical distance between populations, some factors can increase (or decrease) this relationship, particularly geographic and language barriers.¹⁷⁰ Thus, we might re-imagine our hypothetical number set above such that it looks like this:

1 2 3 5 6 7 8 9 10

Now we might conclude that there is some reason to categorize the groups into three: (1,2,3) (5,6,7,8) (10). Perhaps the same thing is true of race: maybe there are discontinuities between, say, the genetic variation of whites and Asians that can justify placing them in different groups. Such discontinuities certainly exist in the real world. For instance, researchers Guido Barbujani and Robert Sokal have shown that there are thirty-three genetic “boundaries” within Europe, which correspond in most cases to either geographic or language barriers.¹⁷¹ Thus, the only reason to pick out the five races we presently use would be some especially significant discontinuity at the edges of these races.

A final response to this analysis might be that race is not based on variation in all genes, but just in particular genes. Thus, race might be defined simply by reference to the gene(s) for skin color. Even defined this way, race is incoherent. The genes for skin color lead to placing Af-

¹⁷⁰ *Id.* at 26–27.

¹⁷¹ *See id.* Barbujani and Sokal’s research suggests that physical and communication barriers prevent gene flow and thereby allow for different genetic distributions in population groups. Barbujani & Sokal, *supra* note 140, at 1816.

ricans and Native Australians together in one group, even though they are the two groups with the *greatest* genetic difference between them.¹⁷² Reliance on other traits is not likely to be any more useful. Hair type turns out to be even less consistent with our general assumptions about race than skin color.¹⁷³

Nonetheless, once our society has created a social construct of race based on ancestry, race has some value because it serves as a proxy for place of geographic origin, and geographic origins can matter when it comes to genes. Furthermore, as Professor Neil Risch and his colleagues have recently noted, racial groups often do coincide with one set (but by no means the only set) of geographic discontinuities in allelic frequency.¹⁷⁴

Despite this *rough* congruity between our social construction of race and the geographic origin of an individual's ancestors, there are some important exceptions. First, Hispanics/Latinos do not fit into any particular geographic location well: the ancestors of the individuals so labeled come from at least three different locations: Africa, the Americas, and Europe. Even more problematic, for any individual, the combination will vary quite considerably—some will only have ancestors from one of these places, while others will have ancestors from two or more locations.¹⁷⁵

Another important exception derives from racial exogamy, which is the tendency of individuals to mate with members of another race. The most widely noted application of this is among the African American population. Although estimates vary, approximately seventeen percent of the ancestors of those classified as African American were European in origin.¹⁷⁶ Racial exogamy is lower among the other races.¹⁷⁷

Finally, there is the problem of imprecise borders.¹⁷⁸ Sharp discontinuities can exist *within* continents. For instance, people whose ancestors are from Northern Africa and the Middle East are frequently categorized as white, rather than as black or Asian.¹⁷⁹ Genetically, this categorization

¹⁷² See CAVALLI-SFORZA, *supra* note 96, at 38–39 (documenting genetic difference between Africans and Australian Aborigines); EHRLICH, *supra* note 100, at 50–51 figs. a–d (showing skin color and other traits in various parts of the world).

¹⁷³ See EHRLICH, *supra* note 100, at 50 fig.b.

¹⁷⁴ Neil Risch et al., *Categorization of Humans in Biomedical Research: Genes, Race and Disease*, 3 *GENOME BIOLOGY* 1, 2–4 (2002).

¹⁷⁵ See Pedro Zubizarreta, Letter, *Is There a Hispanic Race?*, 354 *LANCET* 780 (1999). A similar exception is Native Australians. Under the Census guidelines, it is not clear where they should be grouped. Although many would group them with Pacific Islanders, the genetic studies strongly suggest that most Pacific Islanders have different genetic roots. See CAVALLI-SFORZA, *supra* note 96, at 70–71 (noting separate grouping of Australians and New Guineans); DIAMOND, GUNS, GERMS, AND STEEL, *supra* note 141, at 336 (noting that Polynesians originally came from Mainland China).

¹⁷⁶ The estimates vary from 7% to 50%. See *infra* note 249 and accompanying text.

¹⁷⁷ However, some amount still exists, particularly among Native Americans. See *infra* note 185.

¹⁷⁸ See, e.g., RIDLEY, *NATURE VIA NURTURE*, *supra* note 102, at 264 (“There are no sharp geographic boundaries where one race begins and another ends . . .”).

¹⁷⁹ The Census guidelines seem to require such a result: white is defined as “[a] person

makes sense—the gene flow around the Mediterranean appears to be relatively smooth, with the sharp breaks occurring to the south and the west. But the absence of sharp discontinuities can also create difficulties: for instance, Ethiopians appear to share as much with “Whites” as with “Africans.”¹⁸⁰ The result is that it is difficult to know whether a person of East African descent is more likely to share the genetic make-up of Europeans or Africans.

These differences in genetic make-ups among members of different races are usually the result of genetic drift and the absence of gene flow, not natural selection.¹⁸¹ One significant implication is that, although differences in genetic makeup may emerge, observable differences should generally not affect the fitness of the individual. Natural selection would generally work to remove the less fit allele from the gene pool. But natural selection is not always a strong force, and genetic drift can promote a less fit allele where the founders’ effect comes into play.¹⁸² In this situation, the small population size, perhaps combined with a skewed initial distribution of alleles, can overcome natural selection, at least over the short-term or where an absence of exogamy leads to no gene flow.¹⁸³ In such cases, a more fit allele cannot reestablish itself in the population group. Hence, present-day ethnic groups descending from relative endogamous small population groups, such as Icelanders, Ashkenazi Jews, and Finns, all show disproportionately high (and disproportionately low) levels of some alleles.¹⁸⁴ Importantly, though, the founders’ effect should have relatively little impact on the distribution of alleles among races (as we have defined them) because the necessary conditions do not exist: races, with the possible exception of Native Americans, were not formed from small population groups that were then cut off from each other.¹⁸⁵

That said, natural selection can play a role in explaining some genetic variation between races. For instance, differences in human external physical traits may be linked to natural selection, although even here there is substantial debate. People who trace their ancestry to Scandinavia have a lighter skin color than those whose ancestors came from Central Africa. Two explanations predominate. One is that such external physical differences differ as the direct result of natural selection. On this theory, a darker

having origins in any of the original peoples of Europe, the Middle East, or North Africa.” Revisions to the Standards for the Classification of Federal Data on Race and Ethnicity, 62 Fed. Reg. 58789 (Oct. 30, 1997).

¹⁸⁰ See Risch et al., *supra* note 174, at 4.

¹⁸¹ See CAVALLI-SFORZA, *supra* note 96, at 44 (noting importance of drift); FUTUYAMA, *supra* note 120, at 739–40.

¹⁸² See CAVALLI-SFORZA, *supra* note 96, at 42–43.

¹⁸³ See *id.*; FUTUYAMA, *supra* note 120, at 393.

¹⁸⁴ The initial generations may have been quite small and one or more of them may have had a disproportionately high or low level of a particular less fit allele. Because of endogamy, the alternative allele could not reassert itself in the group’s gene pool.

¹⁸⁵ There is some evidence to suggest a founders’ effect among Native Americans. See CAVALLI-SFORZA, *supra* note 96, at 42–44 (noting this possibility).

skin color is important for people living in the tropics because it protects them from the sun. In colder regions, this selective pressure is removed, and a lighter skin color (really a lack of pigmentation) provides a selective benefit because it allows for the production of Vitamin D, which is otherwise missing from the diet of such peoples.¹⁸⁶ The natural selection explanation, however, is contested since not all of the traits associated with population groups in the tropics are adaptive. Furthermore, it does not explain why some populations living in the tropics have traits normally associated with colder climates and vice versa.¹⁸⁷

A second theory, sexual selection, has been urged: particular traits are selected because those traits either attract potential mates or intimidate rivals of the same sex.¹⁸⁸ Jared Diamond has suggested that humans have a tendency to prefer as sexual partners those persons of the opposite sex who resemble the people we were around when we grew up, although we avoid as sexual partners those particular people themselves.¹⁸⁹ The result is a feedback loop: one raised in an environment with mostly white-skinned and brown-haired people would likely mate with such a person, thereby ensuring that offspring will likely have those traits too. We cannot resolve the debate between these two theories and, in any event, many biologists seem to view them as somewhat complementary.¹⁹⁰ What is important for our purposes, though, is that both theories imply that the likelihood of differences not linked to appearance is low. Under the sexual selection theory, such differences evolve in different groups because they are a signal for mating. This theory, however, does not predict such selection pressure for differences in internal structure, which provide no external signal.¹⁹¹

¹⁸⁶ See CAVALLI-SFORZA, *supra* note 96, at 10–11; GOLDSMITH & ZIMMERMAN, *supra* note 95, at 289–90; MCKEE, *supra* note 112, at 191–92. The darker skin of some groups living in northern climates has been explained by the fact that those groups supplement their diet with other sources of Vitamin D. See EHRLICH, *supra* note 100, at 52. Other features common among people living in the tropics, such as body shape, nose size, and eye shape, have been explained in a similar fashion. See CAVALLI-SFORZA, *supra* note 96, at 11.

¹⁸⁷ See CAVALLI-SFORZA, *supra* note 96, at 11 (noting fitness of slanted eyes in cold environments and the existence of this trait in warmer climates); DIAMOND, *THE THIRD CHIMPANZEE*, *supra* note 149, at 113–15.

¹⁸⁸ See DIAMOND, *THE THIRD CHIMPANZEE*, *supra* note 149, at 117–20; MCKEE, *supra* note 112, at 216–17; MARLENE ZUK, *SEXUAL SELECTIONS: WHAT WE CAN AND CAN'T LEARN ABOUT SEX FROM ANIMALS* 5–10 (2002). The theory grows out of the insight that in many animal species physical traits have evolved that seem to have no selective advantage; the paradigmatic example is the tail of the peacock. Biologists have suggested that these traits evolved as a mechanism for attracting mates: having a vast plumage is a signal from the male to potential peahen mates that he would be a good partner: if he was not exceptionally fit, he could not possibly have such a disadvantage and still survive.

¹⁸⁹ See DIAMOND, *THE THIRD CHIMPANZEE*, *supra* note 149, at 99–109; see also RIDLEY, *NATURE VIA NURTURE*, *supra* note 102, at 172 (noting human female sexual aversion to mates known at age three and younger).

¹⁹⁰ See CAVALLI-SFORZA, *supra* note 96, at 11; DIAMOND, *THE THIRD CHIMPANZEE*, *supra* note 149, at 120–21; EHRLICH, *supra* note 100, at 53.

¹⁹¹ See DIAMOND, *THE THIRD CHIMPANZEE*, *supra* note 149, at 120.

Under the natural selection model, there is slightly more reason to expect other differences, but the effect should still be minimal. Differences in physical appearance have evolved because of environmental pressure, but internal differences are rarely as directly affected by natural selection. The few exceptions include increased lung capacity among Indians living in the Andean highlands¹⁹² and the sickle cell anemia gene, which gives people in areas infected with malaria a selective advantage.¹⁹³ But what is notable about these examples is the strength of the selective pressure involved: genes for internal differences between groups have tended to arise where there is a big selective advantage to them.

This limited evolution of alleles for internal differences should not be surprising.¹⁹⁴ After all, human population groups have had at most 200,000 years (10,000 generations) to evolve differences in allelic frequencies.¹⁹⁵ And not until at least 100,000 years ago (when the early humans left Africa) were human populations subject to significantly different selective environments. Even something that is heavily selected for, such as malarial resistance, appears to take between 1500 to 2000 years to evolve.¹⁹⁶

There is one final important caveat: these examples are not tied directly to particular races, but rather to human population groups in particular places and times. The allele for sickle cell anemia is often associated in the popular press with people of African ancestry, but this is too simple a story.¹⁹⁷ The sickle cell anemia allele is but one of a number of alleles that have evolved because they provide malarial resistance.¹⁹⁸ The most common similar allele is that for Thalessemia, a disease prevalent in both Asian and Southern European populations. Like sickle cell anemia, this allele also provides malarial resistance in heterozygous form, and it is

¹⁹² See *id.* at 114.

¹⁹³ See *id.* Similar genes that “combat” malaria have arisen elsewhere, most notably the Thalessemia genes.

¹⁹⁴ Because Multi-regional Continuity theory, see *supra* note 145, suggests that the common ancestors of humans are far more ancient—perhaps a million years old—it would initially appear to allow for a great deal more locally evolved variation among human populations. While there appears to be evidence for and against this theory, even that theory would recognize that the vast majority of our genetic heritage came from relatively recent African populations. See Ann Gibbons, *First Member of Human Family Uncovered*, 297 SCIENCE 171 (2002).

¹⁹⁵ We are not assuming here that humans evolved from a single individuals, nor from a genetically homogenous group. Instead, we assume that the initial human group from which we all descended had significant internal genetic variation but of course no external variation (because all the relevant individuals were in the group). It was only after that group started to expand that between-group variation could be found. Of course, this initial group may really have been several separate groups, but we assume sufficient gene flow among these groups to make them indistinguishable for our purposes.

¹⁹⁶ See CAVALLI-SFORZA, *supra* note 96, at 49.

¹⁹⁷ It is also present in other groups, particularly Arab and Asian Indian populations. See *id.* at 48.

¹⁹⁸ See Lucio Luzzatto & Rosario Notaro, *Protecting Against Bad Air*, 293 SCIENCE 442 (2001).

therefore no surprise that it occurs where there has been chronic malaria infestations.¹⁹⁹

This points to two important insights. First, if we are looking for widespread genetic differences between humans based on natural selection, generally our focus should be on alleles that provide significant selective advantages in different environments.²⁰⁰

Second, races tell us useful things about genes only because they are each the amalgamation of underlying population groups on which the various forces of evolution work. And when it comes to natural selection as one evolutionary pressure, those underlying population groups are likely to be far more informative than races. Because racial groups have generally lived in a wide variety of environments, they are unlikely to vary in significant ways.²⁰¹ But smaller population groups are likely to have been exposed to a particular environment long enough that, for genes with particularly strong selective advantages, we may see evolved differences. Even when thinking about genetic drift, focusing on smaller population groups, rather than races, makes sense. Since genetic drift is the chance fluctuation of gene frequencies from generation to generation, particularly small communities with large degrees of endogamy may exhibit alleles that are rather unfit; this explains the presence of certain mutations among the Old Order Amish.²⁰² The allele for Tay-Sachs disease may also be an example. This allele is often associated with Ashkenazi Jewish populations, noted for their endogamy and who may have been founded by a rather small group.²⁰³ Recent research suggests that its presence is most likely the result of the founder's effect.²⁰⁴ Other groups believed to exhibit a founder's effect are Icelanders and Finns.²⁰⁵

The core lesson is that smaller population groups, particularly ones that practice endogamy, are more likely to exhibit genetic differences than races.²⁰⁶ Indeed, except for sickle cell anemia, most genetic differences

¹⁹⁹ See RIDLEY, *GENOME*, *supra* note 90, at 141–42; see also CAVALLI-SFORZA, *supra* note 96, at 47–48.

²⁰⁰ Furthermore, the complexity of evolution suggests that, for alleles that provide only a small advantage, there is unlikely to be any significant movement, even over a long period of time. See MCKEE, *supra* note 112, at 165–67.

²⁰¹ So, for example, Africans and Asians both live in jungles and near deserts. Whites have lived in environments not much different from the savannah. Asians and whites have both lived in Arctic environments, and Native Americans have lived over an incredible range of environments.

²⁰² See Clines, *supra* note 134.

²⁰³ Nicholas Wade, *Diseases Common in Ashkenazim May Be Random*, N.Y. TIMES, Mar. 4, 2003, at F2.

²⁰⁴ See Neil Risch et al., *Geographic Distributions of Disease Mutations in the Ashkenazi Jewish Population Supports Genetic Drift Over Selection*, 72 AM. J. HUM. GENETICS 812 (2003).

²⁰⁵ See CAVALLI-SFORZA, *supra* note 96, at 116; Nicholas Wade, *A Genomic Treasure Hunt May Be Striking Gold*, N.Y. TIMES, June 18, 2002, at F1.

²⁰⁶ The overall frequency of such disease-causing alleles in population groups such as Old Order Amish, Ashkenazi Jews, Icelanders, and Finns is no greater than in larger popula-

are really differences between population groups, rather than between races. Nonetheless, different races, at least as we have defined that term, do show different frequencies of at least some alleles. Although other methods, such as genetic testing, may be more accurate for ascertaining the presence of a particular allele in an individual, race does provide some potentially useful genetic information. What we explore next is whether these differences, along with others, can explain the use of race in medicine.

III. USE OF RACE IN MEDICINE

Medicine is concerned with obtaining and maintaining good health for patients. While some of this mission involves treatment of accidents, most medical care attempts to treat and avoid disease.²⁰⁷ First, health care providers attempt to prevent diseases from occurring by taking actions, such as instituting changes in behavior, administering vaccines, or prescribing pharmaceuticals to prevent disease. Second, health care providers address diseases once they have occurred, either by eliminating the cause of the disease or by alleviating its symptoms.²⁰⁸

What relevance can race have for either diagnosing disease or treating it? To analyze this question, we begin this Part by discussing the various causes for diseases and their treatments. Differences in environments and differences in genes can explain disparate outcomes in disease acquisition and treatment for various individuals. We then turn to whether race is helpful in predicting disease acquisition and in treating it once it arises. Our conclusion is a tentative yes, but with the important caveat that other predictors are likely to be even more important.

A. *The Acquisition and Treatment of Disease*

We acquire diseases²⁰⁹ in a number of ways, but there are two root causes of diseases: genes and the environment. Some diseases are caused entirely by genes. Cystic fibrosis and Huntington's disease are examples: one who has the alleles for them (two alleles in the case of cystic fibrosis, just one in the case of Huntington's) will get the disease and eventually

tion groups. While these groups have elevated frequencies of some alleles, they also have decreased frequencies of other alleles.

²⁰⁷ See Mary A. Crossley, *Of Diagnoses and Discrimination: Discriminatory Nontreatment of Infants with HIV Infection*, 93 COLUM. L. REV. 1581, 1619 (1993).

²⁰⁸ See *id.*

²⁰⁹ By "disease," we generally mean any deviation from the normal workings of the body. See Lars Noah, *Pigeonholing Illness: Medical Diagnosis as a Legal Construct*, 50 HASTINGS L.J. 241, 245-46 (1999). An illness, in contrast, is a disease which in some manner incapacitates the patient. See *id.* at 244 n.5.

die from it.²¹⁰ Other diseases are caused entirely by our environment, either as a result of infections (such as the flu²¹¹), our behavior (such as scurvy, which is caused by the absence of Vitamin C in the diet²¹²), or elements to which we are exposed (such as radiation sickness²¹³). Finally, other diseases appear to be the result of psychological stress.²¹⁴

Most diseases, even those that we usually consider as distinctly genetic or environmental, are caused by a complex interaction of genes and environment.²¹⁵ For instance, consider phenylketonuria (“PKU”), which is a classic genetic disease, in which a child is born with two faulty alleles for the gene on Chromosome 12 that codes for the enzyme phenylalanine hydroxylase.²¹⁶ Because of this defect, the child can become mentally retarded and have other physical problems.²¹⁷ The disease can be treated, however, by a special diet that contains very low amounts of the amino acid phenylalanine; without exposure to this amino acid, the child will not develop the disease.²¹⁸ Maple syrup urine disease is quite similar: children with this disease are unable to metabolize the amino acids leucine, isoleucine and valine.²¹⁹ Again, such children are treated with a special diet.²²⁰ Both of these diseases obviously have a strong genetic component, but it is not genes alone that give rise to the invidious symptoms of the disease: they both require an environment where the child is exposed to specific amino acids in her diet.

²¹⁰ See discussion *infra* note 236. This assumes, of course, that something else does not kill the person first. Particularly with Huntington’s disease, where onset is frequently not until after age thirty, see Umberto De Girolami et al., *The Central Nervous System*, in *PATHOLOGIC BASIS OF DISEASE* 1293, 1336 (Ramzi S. Cotran et al. eds., 6th ed. 1999), one might die prior to the onset of the disease.

²¹¹ See John Samuelson, *Infectious Diseases*, in *PATHOLOGIC BASIS OF DISEASE*, *supra* note 210, at 329, 332.

²¹² See Agnes B. Kane & Vinay Kumar, *Environmental and Nutritional Pathology*, in *PATHOLOGIC BASIS OF DISEASE*, *supra* note 210, at 403, 449; James B. Reuler et al., *Adult Scurvy*, 253 *JAMA* 805, 806 (1985). Another example is cirrhosis, which can result from prolonged heavy consumption of alcohol. See Kane & Kumar, *supra*, at 411.

²¹³ Kane & Kumar, *supra* note 212, at 424–31. Another is thalidomide-related birth defects, which resulted from exposure as a fetus to thalidomide. See Charles Marwick, *Thalidomide Back—Under Strict Control*, 278 *JAMA* 1135 (1997); Deborah Schofield & Ramzi S. Cotran, *Diseases at Infancy and Childhood*, in *PATHOLOGIC BASIS OF DISEASE*, *supra* note 210, at 459, 467.

²¹⁴ See generally ROBERT M. SAPOLSKY, *WHY ZEBRAS DON’T GET ULCERS* (1994) (discussing stress-related diseases).

²¹⁵ See RIDLEY, *NATURE VIA NURTURE*, *supra* note 102, at 100.

²¹⁶ See Schofield & Cotran, *supra* note 213, at 475.

²¹⁷ See Rebecca Wappner et al., *Management of Phenylketonuria for Optimal Outcome: A Review of Guidelines for Phenylketonuria Management and a Report of Surveys of Parents, Patients, and Clinic Directors*, 104 *PEDIATRICS* 1377 (1999).

²¹⁸ See *id.*; see also RIDLEY, *NATURE VIA NURTURE*, *supra* note 102, at 100.

²¹⁹ See Latisha D. Love-Gregory et al., *Evidence of Common Ancestry for the Maple Syrup Urine Disease (MSUD) Y438N Allele in Non-Mennonite MSUD Patients*, 75 *MOLECULAR GENETICS & METABOLISM* 79, 79 (2002).

²²⁰ David T. Chuang & Vivian E. Shih, *Maple Syrup Urine Disease (Branched-Chain Ketoaciduria)*, in 2 *THE METABOLIC & MOLECULAR BASES OF INHERITED DISEASE* 1971, 1991–92 (Charles R. Scriver et al. eds., 8th ed. 2001).

Similarly, for other diseases that we think of as environmental, genes can play an important ameliorative role. Malaria is a typical “environmental” disease: by avoiding places where there are female mosquitoes of the genus *Anopheles* infected with the bacterium of the genus *Plasmodium* (Sub-Saharan Africa, much of Central and South America, the Middle East and South Asia²²¹), we can avoid the disease.²²² Furthermore, even if bitten by a mosquito, a victim can avoid malaria by taking a drug regime before, during and after exposure.²²³ But as we have already seen, genes also play a role. Someone who is heterozygous for the sickle cell anemia gene has some protection against malaria infection.²²⁴ In a sense, then, malaria is a genetic disease: those of us who do not carry the correct alleles to protect ourselves suffer from a condition that may well lead to death unless we avoid certain places, much like children with PKU and maple syrup urine disease must avoid certain amino acids.

The cause of most disease lies at this intersection between the environment and the genome.²²⁵ For these diseases, genes are still very important because they either increase the risk of getting the disease or worsen its symptoms. Different alleles protect people against a number of infectious diseases, such as malaria and cholera, although these alleles frequently also have trade-offs associated with them. For people without those alleles, the risk of developing the disease is notably greater if the person is exposed. Similarly, two alleles, *BRCA1* and *BRCA2*, are associated with a higher risk of developing breast cancer—but not all women with these alleles actually develop the disease.²²⁶

²²¹ See Karen P. Day, *The Epidemiology of Malaria*, in *MALARIA: MOLECULAR AND CLINICAL ASPECTS* 57, 58 fig.3.1 (Mats Wahlgren & Peter Perlmann eds., 1999); Samuelson, *supra* note 211, at 389.

²²² See Day, *supra* note 221, at 57–58. The actual symptoms of malaria depend on which of the four species of bacteria a person is infected by, with *P. falciparum* by far the most deadly. See Kevin Marsh, *Clinical Features of Malaria*, in *MALARIA: MOLECULAR AND CLINICAL ASPECTS*, *supra* note 221, at 87. In this Article, we will use the term “malaria” to refer to *P. falciparum* malaria.

²²³ See Centers for Disease Control and Prevention, *Recommendations for the Prevention of Malaria Among Travelers*, 39 *MORBIDITY & MORTALITY WKLY. REP.* 1 (1990), reprinted in 263 *JAMA* 2729 (1990). The drug used depends on the nature of the malaria infestation. See *id.* at 2734–37.

²²⁴ See Johan Carlson, *Inborn Resistance to Malaria*, in *MALARIA: MOLECULAR AND CLINICAL ASPECTS*, *supra* note 221, at 363, 367–68 (suggesting that it protects against *P. falciparum* only, but that the parasite count remains low, so that the infection tends to be shorter and the risk of death much lower).

There are a number of genes that protect against malaria. Beyond the Thalessemia gene, see *supra* text accompanying note 193, some people carry a gene for the enzyme glucose-6-phosphate dehydrogenase (G6PD) that results in a decrease in enzyme activity (there are at least thirty-four such alleles). People carrying such genes are at risk of developing anemia under certain conditions (exposure to certain drugs or fava beans), but seem to be protected against malaria from the *P. falciparum* bacterium. See Luzzatto & Notaro, *supra* note 198.

²²⁵ See generally RIDLEY, *NATURE VIA NURTURE*, *supra* note 102.

²²⁶ See Susan C. Lester & Ramzi S. Cotran, *The Breast*, in *PATHOLOGIC BASIS OF DISEASE*, *supra* note 210, at 1093, 1105; Richard Wooster et al., *Identification of the Breast Can-*

Just as important for our purposes, genes and environment both influence the treatment of diseases. Many diseases are treated with drugs, and different individuals can react to the same drugs in different ways. One example is a genetic deficiency in the enzyme G6PD, which can result in a severe reaction to the antimalarial drug primaquine.²²⁷ But once again, environment can be as important as genes. For instance, properly functioning alleles for the enzyme butyrylcholinesterase (BChE) are necessary to break down many common anesthetics: decreased BChE functioning is associated with decreased ability to metabolize succinylcholine and procaine.²²⁸ But butyrylcholinesterase functioning can also be lowered by environmental factors, such as the consumption of potatoes, tomatoes, and eggplant.²²⁹

Thus, both genes and environment are clearly relevant to health care. The next question, then, is what can race tell us about genes and environments that might be relevant to medicine?

B. Race in Prevention, Diagnosis and Treatment

Race itself will never be the cause of a disease, nor will it treat a disease. Instead, race may be correlated with the occurrence of a particular disease or with the success of a particular treatment for a disease, for either of two reasons. First, those people who are classified as a particular race might have a disproportionately high (or low) distribution of a particular allele associated with developing or treating a disease. Second, those same people might be disproportionately exposed to an environment that is associated with a particular disease or treatment.

1. Race and Genes

If alleles can affect both the acquisition and treatment of disease, can such alleles be correlated with particular races? The answer for at least some alleles has to be yes. For instance, there can be no doubt that, in the United States, race can play a large role in predicting the presence of the sickle cell allele: Americans of African ancestry are far more likely to

cer Susceptibility Gene BRCA2, 378 NATURE 789, 790, 792 (1995) (noting that *BRCA2* appears to give rise to breast cancer susceptibility, but that actual development of disease also turns on environment, lifestyle or other genetic factors).

²²⁷ See *Genetic Disorders*, in PATHOLOGIC BASIS OF DISEASE, *supra* note 210, at 139, 148.

²²⁸ See Werner Kalow & Denis M. Grant, *Pharmacogenetics*, in 1 THE METABOLIC & MOLECULAR BASES OF INHERITED DISEASE, *supra* note 220, at 225, 236–37. Succinylcholine is used for muscle relaxation during surgery and it is (normally) rapidly hydrolyzed to succinic acid and choline. MARY WHITTAKER, CHOLINESTERASE 7–8 (1986). People who do not hydrolyze succinylcholine as rapidly experience much longer effects from that drug. *Id.* at 8–9.

²²⁹ See Rebecca Voelker, *No Potatoes Before Surgery*, 280 JAMA 1735 (1998).

have the allele than those of European ancestry.²³⁰ We can predict that African Americans are more likely to develop sickle cell anemia than whites, and that, conversely, African Americans are less likely to be severely infected by the most dangerous form of malaria after exposure. What is more difficult is how many alleles are like the sickle cell allele: are some (or even many) disease-causing (or treatment-preventing) alleles distributed in a statistically significant way between races? As we noted in Part II, above, there are generally good reasons to suspect some difference in allelic frequencies between races. The question we must pursue, though, is whether genes linked to disease and treatment are likely to have been affected in this way. There are two main reasons that differences in allelic frequencies could emerge. First, natural selection: one allele of a gene might be more fit in the environment of a particular race than in another. Second, random chance: one allele might be more predominant in one race than another not because it has any selective advantage but as the result of statistical aberrations, such as genetic drift and mutations. Our conclusions are that at least an occasional correlation will exist between race and disease and that this is usually the result of such statistical aberrations, and that it will probably be uncommon.

a. Natural Selection

Natural selection can explain some differences in allelic frequencies between races. However, this should occur only in quite limited circumstances. Indeed, natural selection is more likely to create substantial differences among sub-racial groups than between races.

Natural selection should, if anything, generally select *against*, not *for*, alleles that cause diseases. Diseases, after all, generally harm us and, as a result, most disease-causing alleles should decrease our reproductive fitness and therefore tend to disappear from the gene pool.²³¹ Thus, for a disease-causing allele to perpetuate itself, the allele should provide some benefit that offsets the costs of the disease.²³² In addition, this benefit needs to be relatively large, for the time available for natural selection to work on

²³⁰ Approximately 9% of African Americans are carriers, GOLDSMITH & ZIMMERMAN, *supra* note 95, at 69, whereas no more than 0.7% of American whites are carriers, Nadia Ewing et al., *Newborn Diagnosis of Abnormal Hemoglobins from a Large Municipal Hospital in Los Angeles*, 71 AM. J. PUB. HEALTH 629, 629 (1981).

²³¹ This is a general principle, subject to important qualifications. For instance, alleles that cause diseases that occur only after the years of reproduction will have much less of an effect on reproductive success, although they can still decrease fitness by limiting the extent to which individuals past the age of reproduction contribute to the up-bringing of subsequent generations. See DAWKINS, *supra* note 117, at 40–41.

²³² While this is also theoretically true of a treatment-inhibiting allele, treatment is such a relatively new phenomenon, in evolutionary terms, that it is unlikely that much adverse selection has yet occurred.

racess differentially has been relatively short. Here, we can again consider the sickle cell anemia allele. This allele is able to survive, although its reproductive fitness in the homozygous form is low, because its reproductive fitness in the heterozygous form is high when carriers are exposed to malaria.²³³

Reproductive fitness alone is not enough, however, for differences to arise between races. In addition, the allele's fitness must be different in the environments of the two races. The sickle cell anemia allele is again an excellent example. When a population is in an environment where malaria is present, this particular allele has positive reproductive fitness because of the benefits to heterozygous carriers, which outweighs the reproductive costs that the allele imposes on homozygous carriers (who develop anemia). As a result, the allele spreads in such a population.²³⁴ When a population is not in such an environment, however, the allele provides no benefit in the heterozygous form and a large cost in the homozygous form. The result is that the allele is selected against and will generally decline. As sickle cell anemia and malaria illustrate, races may be subject to different selective environments and, as a result, disease-resisting genes evolve in only a particular race. If the members of one race are more frequently exposed to a certain selective pressure than the members of the other race, and if an allele provides a greater benefit in one of those environments than the other, over time the allele's frequency will become greater in one race.

There are three important caveats to these observations. First, offspring must continue to be exposed to the differential selective pressure. For instance, if a population is no longer exposed to malaria, then the sickle cell allele no longer has any selective advantage and the allele's frequency will decline. Of course, for a number of generations after the slackening of the selective pressure, an evolutionary "hangover" will continue, where the allele remains present at a higher, but declining frequency.²³⁵ This is no doubt true of the sickle cell anemia allele among African Americans: absent exposure to malaria, a selective pressure against the sickle cell allele should slowly eliminate it from the population.²³⁶

²³³ See FUTUYAMA, *supra* note 120, at 385; ERNST MAYR, WHAT EVOLUTION IS 122 Box 6.3 (2001).

²³⁴ Where malaria is common in Africa, the carrier rate may be as high as 30%. See *Red Cells and Bleeding Disorders*, in PATHOLOGIC BASIS OF DISEASE, *supra* note 210, at 601, 611; Steinberg, *supra* note 107, at 817.

²³⁵ See MAYR, *supra* note 233, at 122 Box 6.3.

²³⁶ *Id.* Similarly, different frequencies of the cystic fibrosis alleles may also be explained by different exposures to cholera and similar diseases. Different racial and ethnic groups appear to carry the disease in different proportions: 1:29 whites carries the alleles, 1:65 African Americans, 1:46 Hispanics and 1:90 Asians. See JAMES WYNBRANDT & MARK D. LUDMAN, THE ENCYCLOPEDIA OF GENETIC DISORDERS AND BIRTH DEFECTS 24, 160, 308 (2000); Schofield & Cotran, *supra* note 213, at 478; see also Michigan State DNA Diagnostic Program, at http://www.phd.msu.edu/DNA/cf_family3.html (last visited Apr. 12, 2004). There are estimates as high as 1:20, however, for whites. See Schofield &

The second caveat is that pressure from natural selection must be strong. Humans have been exposed to different environments for only a relatively small number of generations: at most 10,000 generations and perhaps far fewer.²³⁷ More importantly, the most common difference in environments that turns a disease-causing allele from reproductively unfit to reproductively fit seems to be other infectious diseases. Infectious diseases generally require high population densities to spread.²³⁸ The advent of agriculture was particularly propitious for such diseases: before agriculture, human population groups were too small and isolated to enable the rapid spread of such diseases. As Jared Diamond has noted, this helps explain why, when Europeans and Native Americans first came into contact, it was (generally) Europeans spreading disease to the Native Americans and not vice versa.²³⁹ But since agriculture developed only in the last 10,000 years, there have only been 500 generations for the evolution of defenses against such diseases.²⁴⁰

A third caveat is that, while differences among races should be observable, there may be even greater observable differences among population groups within any group we socially construct as a race. Because natural selection works primarily on the gene or the individual, not on the race or

Cotran, *supra* note 213, at 478. The alleles for the disease are, like the allele for sickle cell anemia, recessive and fatal when the carrier is homozygous for it. But cystic fibrosis alleles are much less fit than the sickle cell allele, for *none* of their homozygous carriers live to reproduce. Thus, we would expect these alleles to be selected against rather strongly. Nonetheless, a large number of cystic fibrosis alleles are in the gene pool.

Part of the reason for the persistence and spread of these alleles appears to be that individuals who are heterozygous (having only one copy of a cystic fibrosis allele) may have increased immunity to cholera and other diseases that cause diarrhea. *See* DIAMOND, GUNS, GERMS, AND STEEL, *supra* note 141, at 201; ROBERT M. SAPOLSKY, *THE TROUBLE WITH TESTOSTERONE* 247 (1997); Sherif E. Gabriel et al., *Cystic Fibrosis Heterozygote Resistance to Cholera Toxin in the Cystic Fibrosis Mouse Model*, 266 *SCIENCE* 107, 109 (1994); *see also* K. P. Dawson & P. M. Frossard, *A Hypothesis Regarding the Origin and Spread of the Cystic Fibrosis Mutation $\Delta F508$* , 93 *Q.J. MED.* 313, 313 (2000) (noting hypothesis that it originally arose as protection against typhoid fever); Sherif E. Gabriel, *Response*, 267 *SCIENCE* 440, 440 (1995) (noting other possible advantages of allele). Thus, any population group exposed to cholera or other such diseases was likely to have been a better selective environment for the cystic fibrosis allele. Because such infectious diseases require high population densities and locations with unsanitized drinking water, *see* MATT RIDLEY, *THE FUTURE OF DISEASE* 11–12 (1997), descendants of people who lived in such environments would be more likely to carry a cystic fibrosis allele. As a result, it is possible that the relatively higher frequencies of the alleles in the white and African American populations are one result of their increased exposure to cholera and similar diseases. As others have noted, for the allele to have spread quite so far among European populations, the defective alleles possibly had to provide other benefits. *See* Dawson & Frossard, *supra*, at 313. The relative absence of cystic fibrosis alleles in non-white populations may simply be a result of genetic drift: the allele may not have been at sufficiently high levels at the time of initial cholera exposure for the heterozygote advantage to lead to high carrier rates. *See* Gabriel, *Response*, *supra*, at 440 (noting need for high incidence of allele for heterozygote advantage to work).

²³⁷ *See supra* text accompanying note 195.

²³⁸ *See* DIAMOND, GUNS, GERMS, AND STEEL, *supra* note 141, at 202–03.

²³⁹ *See id.* at 210–13.

²⁴⁰ *See supra* text accompanying note 159.

other group,²⁴¹ an allele will thrive when it is exposed to an environment where it is fit, and will diminish when exposed to an environment in which it is not fit. The ancestors of individuals comprising various races have been exposed to different environments, which means their genes have been exposed to different selective pressures. But the difference in selective pressures within races can be even greater than among the races: the difference in average temperatures for Europe and Africa are great, but probably not as great as the difference between Sicily and Lapland.²⁴² So, thinking at the level of race, there is less reason to believe that natural selection will have given rise to an allele that aids in protecting against extremely cold weather than if we think at the level of a population group.

Finally, natural selection should play little to no role in creating differential frequencies of alleles that either enhance or impair treatments; these differences, as we explain in the next section, should generally be the result of genetic drift. Of course, natural selection can give rise to alleles that *resist* disease, such as the sickle cell anemia allele.²⁴³ Modern treatment regimes, though, have been around for only the past century or so, not a sufficient number of generations for significant selection to occur to alleles that positively or negatively affect treatment. Thus, natural selection should have little or no impact on differential allele frequencies for treatment susceptibility.

b. Random Chance

Another explanation for different frequencies of both disease-causing and treatment-affecting alleles in races is random chance and, in particular, genetic drift. Random chance appears to play an important role in creating differential allele distributions for various genes between races.²⁴⁴ As we explained in Part II, genetic drift is particularly important where there are two alleles of a gene, neither of which has a selective advantage. Mutations can also create differences in allele frequencies between groups.²⁴⁵

²⁴¹ See DAWKINS, *supra* note 117, at 11 (advocating theory that selection works on genes); MAYR, *supra* note 233, at 126 (advocating theory that selection works on individuals). We do not mean to suggest that group selection does not occur, or that natural selection occurs only at the level of the gene. See MAYR, *supra* note 233, at 131–32.

²⁴² Note the low level of cystic fibrosis mutations in Finland, for instance. See sources cited *infra* note 253. Furthermore, different groups can have very different alleles causing the same diseases, as is the case with cystic fibrosis. See B. Mercier et al., *Complete Detection of Mutations in Cystic Fibrosis Patients of Native American Origin*, 94 HUM. GENETICS 629, 629 (1994) (noting complete absence of $\Delta F508$ mutation in particular Native American population and existence of one unique mutation, along with three other already known mutations).

²⁴³ DIAMOND, GUNS, GERMS, AND STEEL, *supra* note 141, at 201.

²⁴⁴ See Burchard et al., *supra* note 21, at 1172.

²⁴⁵ For instance, between 15% and 30% of the cases of Marfan Syndrome, a genetic disorder, result from novel mutations. *Genetic Disorders*, *supra* note 227, at 148. Such

Of course, even where geneticists can detect differences, the differences may not be significant enough to alter clinical practice. This may be particularly true in instances where the disease is multifactorial. If multiple genes contribute to disease susceptibility, one allele may be more prevalent among one racial group, while another is among a different group, leading to a genetic stalemate.²⁴⁶

Nonetheless, these differences in allelic frequency appear to be important where a disease-causing allele for a single gene disorder has a relatively low frequency in a particular race because it is then possible that the allele will not be found at all among other races.²⁴⁷ In such circumstances, knowledge of a person's race (again, as a proxy for ancestry) may allow health care practitioners to either consider, or rule out, a particular disease when confronted with a particular set of symptoms.

There are, however, a number of problems with using race as a proxy for ancestry in this context. The most obvious is admixture: many individuals are descended from individuals from multiple places.²⁴⁸ As we noted above, current studies estimate that about seventeen percent of the genome in African Americans is actually from European ancestors.²⁴⁹ Admixture also occurs among Native Americans.²⁵⁰

Furthermore, information about an individual's race will often be far less important than more precise information about the particular population subgroup(s) from which a person is descended.²⁵¹ Tay-Sachs dis-

mutations are constantly occurring, resulting in the formation of new alleles of genes. In the absence of gene flow, the particular allele will only exist in one population, not the other. For instance, suppose that African and European populations were both similarly exposed to malaria. Then imagine that a beneficial allele to prevent malaria, such as the sickle cell anemia allele, arose in an African population as the result of a mutation. Absent gene flow between the African and European populations, the allele would be found only in the African population.

²⁴⁶ See Cooper et al., *supra* note 22, at 1168.

²⁴⁷ Burchard et al., *supra* note 21, at 1172. For examples based on genetic markers, see Michael Dean et al., *Polymorphic Admixture Typing in Human Ethnic Populations*, 55 AM. J. HUM. GENETICS 788 (1994). The very first marker reported, 118G, has two alleles, the second of which is found only in 4% of Caucasians and less than 2% of African Americans. The allele is not found at all among Asian or Native American populations. *Id.* at 791 tbl.1.

²⁴⁸ It is an even bigger factor among Hispanics, who are primarily European in genetic heritage, but can be upwards of 30% Native American and 30% African in ancestry in given populations. Michael W. Smith et al., *Markers for Mapping by Admixture Linkage Disequilibrium in African American and Hispanic Populations*, 69 AM. J. HUM. GENETICS 1080, 1082 (2001).

²⁴⁹ See Burchard et al., *supra* note 21, at 1173; Smith et al., *supra* note 248, at 1082. Other studies have placed the percentage even larger: between 10% and 50%, depending upon geographic location in the United States. See CAVALLI-SFORZA, *supra* note 96, at 74.

²⁵⁰ For instance, in a sample of Pima Indians of the Gila River Indian Community of Arizona, researchers found that the mean amount of admixture with European genes was 9.6%. See Robert C. Williams et al., *Individual Estimates of European Genetic Admixture Associated with Lower Body-Mass Index, Plasma Glucose, and Prevalence of Type 2 Diabetes in Pima Indians*, 66 AM. J. HUM. GENETICS 527, 532 fig.1 (2000).

²⁵¹ An example is the distribution of various cystic fibrosis mutations. See Xavier Estivill et al., *Geographic Distribution and Regional Origin of 272 Cystic Fibrosis Mutations*

ease, which appears to be a disease caused by genetic drift, is not a “racial” disease, but rather a population group disease. What aids practitioners in diagnosing the disease is knowing that the individual is descended from Ashkenazi Jews, not knowing that the individual is white.²⁵² Similar information about population groups might also lead away from other diagnoses. The relatively low rate of cystic fibrosis carriers in Finland is an example.²⁵³

The same is likely to be true of alleles that affect disease treatment. A new field has blossomed in recent years dealing with the interaction of genes and drug resistance: pharmacogenetics.²⁵⁴ Researchers have discovered a number of alleles that can enhance (or decrease) the effectiveness of drugs. As noted above, a properly functioning gene for the enzyme BChE is necessary to metabolize succinylcholine and procaine.²⁵⁵ Yet another example is the variations in the alleles for N-acetyltransferase 2 (NAT2), which affects the metabolization of a widely used drug in leukemia treatment.²⁵⁶

Here again, racial groups sometimes have different frequencies of the various alleles for drug resistance and receptivity. For instance, in the case of NAT2, the alleles leading to slow acetylation of drugs can range from 14% among East Asians to 54% among whites.²⁵⁷ Similarly, different alleles of the *CYP2C9* gene vary in their affinity or intrinsic clearance (that is, their metabolization) of various drugs, and the distribution of these alleles varies by race.²⁵⁸ While 98.4% of Asians and 96.3% of African

in *European Populations*, 10 *HUM. MUTATION* 135 (1997).

²⁵² See Cooper et al., *supra* note 22, at 1167.

²⁵³ See Juha Kere et al., *Cystic Fibrosis in a Low-Incidence Population: Two Major Mutations in Finland*, 93 *HUM. GENETICS* 162, 162 (1994) (noting lower frequency of cystic fibrosis in Finland). Consider also maple syrup urine disease. At least sixty-three different mutations have been shown to cause the disease, but the worldwide frequency of the disease remains low: one in 185,000. See Chuang & Shih, *supra* note 220, at 1983, 1987. While the frequency among European whites is essentially the same, H. Bickel et al., *Neonatal Mass Screening for Metabolic Disorders*, 137 *EUR. J. PEDIATRICS* 133, 139 (1981) (data for Western Europe), in the Old Order Mennonite population the frequency ranges from 1:150 to 1:176, Love-Gregory et al., *supra* note 219, at 79. Thus, knowing that an individual is white will not aid in diagnosing the disease; what might help instead is knowing that a patient has Old Order Mennonite ancestry.

²⁵⁴ See Howard L. McLeod, *Pharmacogenetics: More than Skin Deep*, 29 *NATURE GENETICS* 247 (2001).

²⁵⁵ See *supra* text accompanying note 228. Similarly, a mutation in a single allele for the enzyme thiopurine methyltransferase (TPMT) creates a high risk of adverse reactions to several drugs used in leukemia treatment. See H. L. McLeod et al., *Genetic Polymorphism of Thiopurine Methyltransferase and its Clinical Relevance for Childhood Acute Lymphoblastic Leukemia*, 14 *LEUKEMIA* 567 (2000).

²⁵⁶ See Burchard et al., *supra* note 21, at 1173; Mimi C. Yu et al., *Acetylator Phenotype, Aminobiphenyl-Hemoglobin Adduct Levels, and Bladder Cancer Risk in White, Black and Asian Men in Los Angeles, California*, 86 *J. NAT'L CANCER INST.* 712, 715 (1994) (collecting studies).

²⁵⁷ Burchard et al., *supra* note 21, at 1173.

²⁵⁸ See Hong-Guang Xie et al., *Molecular Basis of Ethnic Differences in Drug Disposition and Response*, 41 *ANN. REV. PHARMACOLOGY & TOXICOLOGY* 815, 816–17 (2001).

Americans appear to carry the *CYP2C9*1* allele, only 80.3% of whites do.²⁵⁹ Research has revealed a number of other genes related to drug response that show similar variation in allelic distributions.²⁶⁰

The same caveats that applied to disease-causing genes apply here as well. Admixture complicates the process of determining an individual's likelihood of carrying a particular allele. In addition, differences between races may not be significant enough to justify choice of drugs *by race*: as one commentator has noted, "such differences are relatively small and there is considerable overlap between groups."²⁶¹ Even if the allelic frequencies variations between races would justify different treatment based on race, focusing on population groups may well be more important: there may be greater variation when comparing population groups within races than the rate variation between races. Consider, the alleles for the *CYP2D6* gene, which, like *CYP2C9*, affects the metabolism of various drugs. The frequency of the *CYP2D6*1* allele in Asian populations varies between 22.7% (one study of a Chinese population) and 49.0% (Korean), while in white populations the variation was only between 33.4% (British) and 37.1% (Turkish).²⁶² Furthermore, modern genetic testing may provide a more accurate means for identifying individuals likely to have various genes for drug susceptibility.²⁶³

2. Race and Environment

Environmental factors can also play an important role in explaining why "races" might have different frequencies of various diseases and why they might also be more or less receptive to various treatments. In particular, the physical and social environment in which a person lives can have a large impact on the occurrence and treatment of disease.

Environmental causes of disease can vary significantly. Some are chemical agents, such as Thalidomide, while others are biological, such as

²⁵⁹ The number for American whites is slightly higher: 82.1%. These percentages are calculated by excluding the frequencies of the two best known variant alleles. *See id.* at 817 tbl.1. However, the discovery of other alleles may drive down calculations of the allelic frequency. *See id.* at 818. Of course, it does not necessarily follow from any of this that treatment distinctions should therefore be made based on race. *See id.* at 817 (noting that the relationship between ethnicity and enzyme expression is unclear).

²⁶⁰ *See id.* at 817; Wood, *supra* note 19.

²⁶¹ *Genes, Drugs and Race*, 29 *NATURE GENETICS* 239, 240 (2001). For instance, in the case of TPMT deficiency there is a great deal of heterogeneity in the particular mutation present in any given race, but the overall difference in the likelihood of suffering the disorder remains relatively low for all races (from 5% to 0.5% chance). Even the carrier rate was relatively constant, varying only between 2% and 14.4%. *See* McLeod et al., *supra* note 255, at 570 tbl.1.

²⁶² *See* Xie et al., *supra* note 258, at 820–23 & tbl.3. Excluded are numbers for the *1 allele that were indirectly calculated from other data, leading to overestimates. *Id.*

²⁶³ *See* McLeod, *supra* note 254, at 248.

the *Plasmodium* bacteria. Still others can relate to social conditions, including cultural and psychological mechanisms. For instance, decisions about food intake and exercise level, which are both related to cardiovascular disease, may be determined in part by cultural factors. More subtly, psychological factors, particularly exposure to acute stress, can have a negative impact on an individual's health.

For race to be relevant in this context, members of races—as we have defined them—must be exposed at different rates to various environmental factors. There are at least two ways in which the environment can differentially affect the health of members of races. The first set of factors is internal, that is, factors that result from the behavior of the members of the race themselves. Most prominent among these are cultural practices that tend toward certain health results. The other set of factors is external, that is, influences from outside the race that affect members of a particular race more often than members of other races. One example might be differential exposure of members of a race to environmental factors.

a. Internal Factors

As we noted in Part II, race in the United States itself is socially constructed, which implies that it is the creation of American society.²⁶⁴ The social construction of race is not just exogenous creation—the decision of others to label individuals as members of a race—but also endogenous creation, the decision of the individuals themselves to adopt the identity of a particular race.²⁶⁵

Because racial identification is at least in part a decision by the individual to adopt a racial identity, members of races often *on average* exhibit different behaviors. We are, of course, *not* claiming that there are distinctive behaviors for members of races; just as there are no distinctive white, black or Asian genes, there are no distinctive white, black, Asian or other racial behaviors. Instead, members of certain races will, on average, partake in certain behaviors at different rates.

For instance, a recent *Sports Illustrated* article noted the decline in black youth participation in baseball over the last two decades.²⁶⁶ It suggested that the reasons are complex—linked to changes in publicity and resources, among other things—but the point for our purposes is simple: baseball, and sports more generally, are a behavior that racial groups may

²⁶⁴ Of course, when it is used in other societies, it is also socially constructed, although not necessarily in the same ways.

²⁶⁵ See Haney López, *supra* note 48, at 10. Perhaps the most dramatic evidence of this is the approach of the Bureau of the Census: all demographic data depends on individual self-identification of race.

²⁶⁶ Tom Verducci, *Blackout: The African-American baseball player is vanishing. Does he have a future?*, SPORTS ILLUSTRATED, July 7, 2003, at 56.

engage in at different rates. Of course, generally participation (or non-participation) in baseball is unlikely to have much of an effect on health outcomes. Instead, things like diet and exercise are far more likely to do so. For instance, it is believed that increased physical activity lowers the risk of coronary and cardiovascular disease.²⁶⁷

Merely knowing that behavior affects health outcomes is not enough to identify racial differences. We also have to show that members of races, on average, differ in their behaviors in ways that could give rise to different health outcomes. Increasingly, such evidence exists. In 2002, a study in the *New England Journal of Medicine* tracked the decline in physical activity in black and white adolescent females. The study demonstrated that, although both groups experienced substantial declines in leisure-time physical activity, the decline was much greater among adolescent black females.²⁶⁸ As a result, African American women (on average) may be at increased risk (as compared to white females) for obesity and other long-term problems, such as Type 2 diabetes and coronary and cardiovascular disease.

In addition, groups may differ in their diets. It is well understood, for example, that high cholesterol is a risk factor for stroke and heart disease. Diet clearly has an influence on both serum cholesterol and low-density lipoprotein cholesterol levels.²⁶⁹ And the diets of members of various racial groups may vary, if only because of their access to healthy food.²⁷⁰

Other behaviors that affect health also demonstrate a racial distinction. Smoking has been long associated with increased risk for a large number of diseases.²⁷¹ Rates of smoking, though, can vary between different racial groups. For instance, in the *New England Journal of Medicine* study, white adolescent females were far more likely to smoke than black adolescent females.²⁷² Indeed, through young adulthood, whites and Hispanics are more likely to be smokers than African Americans and Asians, at

²⁶⁷ JoAnn E. Manson et al., *Walking Compared with Vigorous Exercise for the Prevention of Cardiovascular Events in Women*, 347 *NEW ENG. J. MED.* 716 (2002).

²⁶⁸ Sue Y. S. Kimm et al., *Decline in Physical Activity in Black Girls and White Girls During Adolescence*, 347 *NEW ENG. J. MED.* 709 (2002).

²⁶⁹ James W. Anderson, *Diet First, Then Medication for Hypercholesterolemia*, 290 *JAMA* 531 (2003).

²⁷⁰ See Mary Duenwald, *Good Health Is Linked to Grocer*, *N.Y. TIMES*, Nov. 12, 2002, at F5 (Researchers have found a correlation between the number of supermarkets in a neighborhood and the intake of fresh fruits and vegetables and that African American populations tend to live in neighborhoods with fewer supermarkets.).

²⁷¹ Michael J. Thun et al., *Smoking vs. Other Risk Factors as the Cause of Smoking-Attributable Deaths: Confounding in the Courtroom*, 284 *JAMA* 706, 710–11 tbl.3 (2000).

²⁷² Kimm et al., *supra* note 268, at 711 tbl.1 (showing difference of 27.0% vs. 5.7%).

least in the United States,²⁷³ although the rates change when extended to include all adults.²⁷⁴ Similarly, rates of alcoholism vary between races.²⁷⁵

b. External Factors

It is not just our own behavior that can affect our health, but also the behavior of others in creating our day-to-day surroundings. The resulting environment can cause changes in our health. The best example of this is stress, which without question has negative implications for human health, particularly when experienced at an acute level. For instance, the Adverse Childhood Experiences study looked at the effects of childhood psychological, physical and sexual abuse on the health of individuals as adults.²⁷⁶ The results showed that individuals subjected to abuse were at increased risk for alcoholism, drug use, sexually transmitted disease and heart disease.

In recent years, researchers have increasingly been able to document the ways in which stress can negatively affect health. As Professor Robert Sapolsky explains, stress causes the heart rate and blood pressure to rise and the re-absorption of water from the kidneys.²⁷⁷ These processes contribute to cardiovascular disease, among other disorders.²⁷⁸ In addition, the normal process of breaking down food is hampered, and the body instead uses stored energy.²⁷⁹ This can lead to Type 2 diabetes, which itself can lead to heart disease and blindness.²⁸⁰

²⁷³ Phyllis L. Ellickson et al., *Explaining Racial/Ethnic Difference in Smoking During Transition to Adulthood*, 28 ADDICTIVE BEHAVIORS 915 (2003); Steven H. Kelder et al., *Smoking Differences Among African American, Hispanic and White Middle School Students in an Urban Setting*, 28 ADDICTIVE BEHAVIORS 513 (2003).

²⁷⁴ The Centers for Disease Control and Prevention reported that, in 1999, the rate of smoking among whites and African Americans was the same (24.3%). However, the African American male rate was higher than the white male rate (28.7% vs. 25.5%) while the white female rate was higher than the African American female rate (23.1% vs. 20.8%). The data for other racial and ethnic groups varies widely, with the rate for Hispanics (18.1%) and Asians (15.1%) below the national average, and the rate for Native Americans (40.8%) substantially above the average. Centers for Disease Control and Prevention, *Cigarette Smoking Among Adults—United States, 1999*, 50 MORBIDITY & MORTALITY WKLY. REP. 869, 870 tbl.1 (2001). Some studies, however, suggest that African Americans tend to smoke more mentholated cigarettes than other groups, which some claim have more health risks. Vernellia R. Randall, *Smoking, The African-American Community, and the Proposed National Tobacco Settlement*, 29 U. TOL. L. REV. 677, 688–89 (1998).

²⁷⁵ For a general study of the trends of alcohol intake among races and ethnic groups, see Deborah A. Dawson, *Beyond Black, White and Hispanic: Race, Ethnic Origin and Drinking Patterns in the United States*, 10 J. SUBSTANCE ABUSE 321 (1998).

²⁷⁶ Vincent J. Felitti et al., *Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults*, 14 AM. J. PREVENTIVE MED. 245 (1998).

²⁷⁷ See SAPOLSKY, *supra* note 214, at 38–39.

²⁷⁸ *Id.* at 41–48.

²⁷⁹ *Id.* at 62–64.

²⁸⁰ *Id.* at 65. Recently, researchers have begun demonstrating that stress is not always bad. Researchers have shown that early childhood stress leads to depression and suicidal

While stress can certainly lead to poor health outcomes, it is not immediately clear that the members of a particular racial group suffer more stress (on average) than members of other racial groups. There is reason to believe, however, that individuals in lower socioeconomic positions suffer more stress than persons in higher positions. For instance, the Whitehall study of British civil servants showed that, independent of a number of other causes, individuals in low status jobs had higher rates of ischemia, angina and diabetes.²⁸¹ Similar findings have been made in other contexts.²⁸² As one researcher has noted, “persons of lower socioeconomic status (particularly blacks in these positions) by definition face more difficult psychosocial environmental stressors than more economically privileged individuals.”²⁸³ Because African Americans and Native Americans tend to fall into lower socioeconomic groups more than whites, members of those groups may be at increased risk for certain diseases. Furthermore, it is possible that racism itself creates more stress for members of minority groups.²⁸⁴

Finally, racism may be a cause of disease by causing members of racial minority groups to experience stress. Thus, it may be accurate to say that, in this limited case, the social construction of race, which brings with it racism (at least in the contemporary United States), causes stress to those deemed to be members of certain racial minority groups, and thereby creates long-term negative disease outcomes for those persons.²⁸⁵ Outside of this limited situation, though, the mere fact of race, or membership in a racial group, has no direct effect on health.

In addition to stress, which is the result of emotional and other environments created by others, our physical environment may also affect our health. Again, members of certain racial groups might live in locations where they are more likely to be exposed to toxins. For instance, although genes are believed to play a large role in the development of asthma, genetic factors alone appear insufficient to explain the sharp increase in asthma in the United States over the past twenty years.²⁸⁶ One hypothesis

tendencies only in individuals who have a genetic predisposition. A. Caspi et al., *Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene*, 301 SCIENCE 386 (2003).

²⁸¹ M. G. Marmot et al., *Health Inequalities Among British Civil Servants: The Whitehall II Study*, 337 LANCET 1387, 1389 tbl.1 (1991).

²⁸² See, e.g., Darwin R. Labarthe, *Hypertension*, in PUB. HEALTH & PREVENTIVE MED. 849, 852 (John M. Last & Robert B. Wallace eds., 13th ed. 1992).

²⁸³ Sherman A. James, *John Henryism and the Health of African-Americans*, 18 CULTURE, MED. & PSYCHIATRY 163, 166 (1994) (citing S. Leonard Syme, *Psychosocial Determinants of Hypertension*, in HYPERTENSION DETERMINANTS, COMPLICATIONS AND INTERVENTION 95 (Gaddo Onesti & Christian R. Klimt eds., 1979)).

²⁸⁴ See, e.g., James Y. Nazroo, *The Structuring of Ethnic Inequalities in Health: Economic Position, Racial Discrimination, and Racism*, 93 AM. J. PUB. HEALTH 277, 281–82 (2003).

²⁸⁵ See Terry Smith, *Everyday Indignities: Race, Retaliation, and the Promise of Title VII*, 34 COLUM. HUM. RTS. L. REV. 529, 546–48 (2003) (noting studies).

²⁸⁶ See Prescott G. Woodruff & John V. Fahy, *Asthma: Prevalence, Pathogenesis, and*

for this rise has been increased exposure of children to various allergens, particularly cockroach droppings, dust mites, and cat dander.²⁸⁷ The very high rates of asthma recently reported in Central Harlem, for instance, might be the result of increased exposure to these causes,²⁸⁸ particularly because minority racial groups tend to live in urban areas at higher rates than whites.²⁸⁹

Other problems associated with lower socioeconomic status can also affect health. For instance, low birth weight (as well as low weight at age one) is associated with increased risk of heart disease.²⁹⁰ Children of lower socioeconomic status appear to be at increased risk of low birth weight.²⁹¹ In addition, short stature, which can be the result of poor early nutrition and therefore probably associated with lower socioeconomic status, has also been linked to increased risk of cardiovascular mortality.²⁹² It seems clear that external environmental causes of disease afflict racial group members in different ways.

Prospects for Novel Therapies, 286 JAMA 395, 395 (2001).

²⁸⁷ See *id.* at 396. However, exposure to infections and certain allergens in the first year of life may be negatively correlated with the onset of asthma later in childhood. See *id.* at 396–97; see also Dennis R. Ownby et al., *Exposure to Dogs and Cats in the First Year of Life and Risk of Allergic Sensitization at 6 to 7 Years of Age*, 288 JAMA 963, 971 (2002). But other researchers have noted problems with this research. See Letters, *Exposure to Pets in Childhood and Risk of Atopic Disorders*, 289 JAMA 841, 841–42 (2003).

²⁸⁸ See Richard Pérez-Peña, *Study Finds Asthma in 25% of Children in Central Harlem*, N.Y. TIMES, Apr. 19, 2003, at A1. Obviously, cockroach infestations are more likely in poor, inner-city areas. In addition, dust mites, which thrive in areas with both high temperature and high humidity, might do better in inner cities where apartments are the primary living environment. See Susumu Uchikoshi et al., *A Study of the Ecology of the House Dust Mite in Dwelling Houses*, 7 TOKAI J. EXPERIMENTAL CLINICAL MED. 233, 237 (1982). Occurrence of allergic reactions is not dependent on the mite count, but severity of reaction may be. *Id.*

²⁸⁹ Whites make up only 71.5% of the urban population but 88.9% of the rural population, African Americans are 14.0% of the urban population but only 6.0% of the rural population, and Asians are 4.5% of the urban population and 0.5% of the rural population. U.S. CENSUS BUREAU, U.S. DEP'T OF COMMERCE, 1990 CENSUS OF POPULATION: SOCIAL AND ECONOMIC CHARACTERISTICS 4 tbl.4 (1993), available at <http://www.census.gov/prod/cen1990/cp2/cp-2-1.pdf>. The exception is Native Americans, who make up 0.7% of the urban population, but 1.7% of the rural population. *Id.*

²⁹⁰ See RIDLEY, NATURE VIA NURTURE, *supra* note 102, at 154–57; Nazroo, *supra* note 284, at 281.

²⁹¹ See Olga Basso et al., *Change in Social Status and Risk of Low Birth Weight in Denmark: Population Based Cohort Study*, 315 BRITISH MED. J. 1498 (1997); Maureen Hack & Irwin R. Merkatz, *Preterm Delivery and Low Birth Weight—A Dire Legacy*, 333 NEW ENG. J. MED. 1772, 1772–73 (1995) (noting that economic factors are a partial explanation of low birth weight); Sharon L. Hillier et al., *Association Between Bacterial Vaginosis and Preterm Delivery of a Low-Birth-Weight Infant*, 333 NEW ENG. J. MED. 1737, 1737 (1995).

²⁹² See Nazroo, *supra* note 284, at 281.

IV. THE LEGALITY OF RACIAL PROFILING IN MEDICINE

From a legal standpoint, the constraints on race discrimination in general—let alone in health care—are more limited than might be expected given the strong national consensus against such discrimination. The four major sources of federal rights, the Equal Protection Clause and three federal statutory schemes, collectively leave large areas untouched. In any event, as we shall see, these regimes have not often been applied to health care, and their application to the question of present concern is problematic for several reasons. One overriding difference between racial profiling in health care (as opposed to, say, racial profiling in law enforcement or racial segregation in education) is that most treatment decisions are, at least in theory, made not by the health care provider but by the patients themselves.²⁹³ A second is the supposed purity of purpose of health care providers when they draw distinctions—they are acting from what is generally accepted as an intent to benefit those who are being treated differently on the basis of their race. While this claim has historically been made by those who discriminate,²⁹⁴ it is both more credible and, given information asymmetries, less paternalistic in the medical context. Third, First Amendment concerns with restricting the flow of information are especially strong in this arena, where the information in question may be life-saving.

²⁹³ See Fran Carnerie, *Crisis and Informed Consent: Analysis of a Law-Medicine Malocclusion*, 12 AM. J.L. & MED. 55 (1986) (noting that the principle of individual autonomy lies at the core of the doctrine of informed consent). In actuality, most commentators believe consent in health care situations is rarely “informed.” See Jay Katz, *Informed Consent—A Fairy Tale? Law’s Vision*, 39 U. PITT. L. REV. 137, 148 (1977) (stating that “disclosure and consent . . . are obligations alien to medical practice” because physicians do not disclose all relevant information to their patients); Jay Katz, *Informed Consent—Must It Remain a Fairy Tale?*, 10 J. CONTEMP. HEALTH L. & POL’Y 69, 81 (1994) (asserting that the doctrine of informed consent is “a charade”); Alan Meisel & Mark Kuczewski, *Legal and Ethical Myths About Informed Consent*, 156 ARCHIVES INTERNAL MED. 2521, 2522 (1996) (declaring that “informed consent is often no more than a medical Miranda warning”); William M. Sage, *Regulating Through Information: Disclosure Laws and American Health Care*, 99 COLUM. L. REV. 1701, 1705 n.8 (1999) (questioning whether doctrine furthers the patient’s autonomy or merely provides her with “the illusion of self-determination”).

²⁹⁴ The Southern system of de jure segregation was often justified as in the interests of both the white and black races. See generally 110 CONG. REC. 1511, 1516–2805 (1964). Perhaps more credibly beneficent were the motives of reformers. For example, many feminists supported efforts such as those of Louis Brandeis, whose “Brandeis brief,” in *Muller v. Oregon*, 208 U.S. 412 (1908), justified the Court’s upholding a state law limiting the working hours for women when a similar law applied to men would have been invalidated. See Deborah L. Rhode, *The “No-Problem” Problem: Feminist Challenges and Cultural Change*, 100 YALE L.J. 1731, 1739–44 (1991). However, many such “protective” laws were not so well motivated and, even in the most charitable view, sprang from paternalistic impulses that are anathema today. See, e.g., *Goesaert v. Cleary*, 335 U.S. 464 (1948) (upholding law forbidding females from bartending unless they were the wife or daughter of the owner, despite its transparent purpose to maintain male dominance in the bartending business).

*A. The Legal Regimes**1. The Equal Protection Clause*

The Equal Protection Clause limits discrimination by the government and therefore implicates actions by the FDA and decisions by public hospitals or public research facilities.²⁹⁵ Equal protection claims are generally enforced through 42 U.S.C. § 1983.²⁹⁶ Suits are limited to intentional race discrimination (disparate treatment) by governmental institutions, although sometimes differences in treatment can be stark enough to allow the inference of intent to discriminate.²⁹⁷

The narrowest prohibition, both in terms of reach and in substantive restrictions, is the Equal Protection Clause.²⁹⁸ It applies only to governmental action, and therefore leaves untouched activities by those who do not count as the state or federal government. While much medical research and treatment is conducted and delivered by governmental entities, most is not. Further, the heavy involvement of federal and state governments in aspects of health care delivery does not convert the actual providers of health care into state actors.²⁹⁹

With respect to substance, the Equal Protection Clause subjects racial classifications to “strict scrutiny.”³⁰⁰ To constitute a racial classification, the

²⁹⁵ Technically, the Equal Protection Clause is a Fourteenth Amendment limitation on the states, and therefore does not reach the federal government. The Court has, however, read the Due Process Clause of the Fifth Amendment to embrace an equal protection principle identical to that applicable to the states. *See Adarand Constructors v. Peña*, 515 U.S. 200 (1995). For simplicity, this Article will describe claims against both federal and state actors as equal protection claims although distinctions between enforcement against federal and state defendants will be noted.

²⁹⁶ Since § 1983 does not authorize suits against the federal government, discrimination claims are generally maintained through *Bivens* actions, which pose different but analogous problems. *See Bivens v. Six Unknown Named Agents of Fed. Bureau of Narcotics*, 403 U.S. 388 (1971).

²⁹⁷ *See Village of Arlington Heights v. Metro. Hous. Dev. Corp.*, 429 U.S. 252, 265–67 (1977) (while requiring discriminatory intent, recognizing that differences in treatment can be probative of underlying intent).

²⁹⁸ Equal Protection Clause actions under § 1983 lie against local government entities when a policy or practice or the action of a high-level decision maker is at issue, *Monell v. Dep’t of Soc. Servs. of N.Y.*, 436 U.S. 658 (1978), but not against the state because the state is not a “person” within the meaning of the statute, *Will v. Mich. Dep’t of State Police*, 491 U.S. 58 (1989). They may also be brought against individual state employees and officers, but subject to a complex web of immunities. *See generally* Harold S. Lewis, Jr. & Theodore Y. Blumoff, *Reshaping Section 1983’s Asymmetry*, 140 U. PA. L. REV. 755 (1992).

²⁹⁹ *See Blum v. Yaretsky*, 457 U.S. 991 (1982) (stating that nursing homes are not state actors despite federal and state subsidization and pervasive regulation); *see also* *Cnty. Med. Ctr. v. Emergency Med. Services, Inc.*, 712 F.2d 878 (3d Cir. 1983) (noting that a nonprofit corporation organized to coordinate emergency health care services in northeastern Pennsylvania is not a state actor). While Title VI would permit suit against recipients of federal funds, *see Barbara A. Noah, Racist Health Care?*, 48 FLA. L. REV. 357 (1996), most healthcare providers receiving Medicare reimbursement are not viewed as recipients of federal funds for this purpose. *See infra* text accompanying note 310.

³⁰⁰ *See, e.g., Shaw v. Hunt*, 517 U.S. 899 (1996); *Miller v. Johnson*, 515 U.S. 900

government must treat individuals disparately because of their race, although racial classifications include not only facial discrimination on racial grounds but also facially neutral government action that is motivated by a racial purpose.³⁰¹ Under conventional analysis, racial classifications are not per se impermissible. While they trigger “strict scrutiny,” they can in theory survive such scrutiny where they are justified by a compelling governmental interest. However, in practice very few governmental interests have been found by the Supreme Court to be compelling enough to validate a racial classification.³⁰²

Of particular interest to the present discussion, the Supreme Court has been clear that the purity of the purpose motivating a racial classification is irrelevant to whether such a classification is subject to strict scrutiny. In the affirmative action context, the fact that a racial classification was motivated by efforts to include African Americans and thereby redress generations of legal and societal discrimination was not sufficient reason to apply lower scrutiny.³⁰³ Further, while the Court has reiterated that strict scrutiny is not necessarily “fatal in fact,” it has found very few bases for drawing racial classifications to be compelling.³⁰⁴ Even the Court’s latest approval of affirmative action plans in the academic context was limited to the benefits of diversity in education.³⁰⁵

Finally, strict scrutiny requires a close fit between the racial classification and the compelling state interest: the “narrowly tailored” requirement. In an often forgotten passage of *Regents of University of California v. Bakke*,³⁰⁶ Justice Powell considered the defendants’ claim that the racial preference was justified as part of the University’s attempt to “improv[e] the delivery of health-care services to communities currently underserved.”³⁰⁷ Justice Powell wrote:

It may be assumed that in some situations a State’s interest in facilitating the health care of its citizens is sufficiently compelling to support the use of a suspect classification. But there is virtually

(1995); *Korematsu v. United States*, 323 U.S. 214 (1944).

³⁰¹ Equal protection analysis of facially neutral government action searches for real intent and rejects mere disparate racial impact as triggering strict scrutiny. See *Pers. Adm’r of Mass. v. Feeney*, 442 U.S. 256 (1979); *Washington v. Davis*, 426 U.S. 229 (1976).

³⁰² Some commentators have observed a tendency on the part of lower circuit courts of appeal to avoid this analysis by manipulating the initial question as to whether a classification is “racial.” See Richard A. Primus, *Equal Protection and Disparate Impact: Round 3*, 117 HARV. L. REV. 494, 511–14 (2003).

³⁰³ *Adarand Constructors v. Peña*, 515 U.S. 200 (1995).

³⁰⁴ The Court has repeatedly approved racially premised relief for identified victims of de jure discrimination, e.g., *United States v. Paradise*, 480 U.S. 149 (1987). The case that created the strict scrutiny analysis and approved a racial classification as a security measure in wartime, *Korematsu v. United States*, 323 U.S. 214 (1944), probably remains intact in principle although it is universally criticized in application.

³⁰⁵ *Grutter v. Bollinger*, 539 U.S. 306 (2003). Cf. *Gratz v. Bollinger*, 539 U.S. 244 (2003).

³⁰⁶ 438 U.S. 265 (1978).

³⁰⁷ *Id.* at 310.

no evidence in the record indicating that petitioner's special admissions program is either needed or geared to promote that goal.³⁰⁸

As applied to racial profiling in health care, if, for example, there were sufficient empirical data to support, say, treating African Americans differently than whites, such data might not justify treating, say, Asians the same as African Americans. In short, treating individuals differently on account of race for medical purposes might be permissible, but only if the evidence of racial differences were sufficiently strong and the differences in treatment properly tailored to that evidence.

2. *The Statutory Schemes*

Three statutory schemes expand the protection against racial discrimination beyond the relatively limited constraints of the Equal Protection Clause. The first and oldest of these regimes is 42 U.S.C. § 1981, which bars racial discrimination in contracts even between private individuals. Like equal protection claims, § 1981 actions are limited to intentional disparate treatment.

The second source of protection is Title VI of the Civil Rights Act of 1964, which prohibits race discrimination in federally funded programs, regardless of whether the recipient is a public or private institution.³⁰⁹ This statute is, like § 1981, broader than § 1983 insofar as it reaches beyond public entities, but the coverage in the medical context has been sharply limited by early decisions that doctors receiving Medicare funding are not "programs" within the meaning of the statute.³¹⁰ It may also be broader than both §§ 1981 and 1983 by reaching not merely disparate treatment discrimination but also disparate impact discrimination.³¹¹ However, the Supreme Court recently held that, whether or not Title VI bars disparate impact discrimination, there is no private right of action to enforce disparate impact claims.³¹² Thus, any policy with a disparate impact can be challenged only by the federal government, which has not historically brought such actions.

³⁰⁸ *Id.*

³⁰⁹ See 42 U.S.C. § 2000d (2000).

³¹⁰ See Crossley, *supra* note 31, at 263–68.

³¹¹ *E.g.*, Linton v. Carney, 779 F. Supp. 925 (M.D. Tenn. 1990) (finding unjustified disparate impact against blacks in Medicaid bed certification policy), *rev'd on other grounds sub nom.* Linton v. Comm'r of Health & Env't, 973 F.2d 1311 (6th Cir. 1992).

³¹² Alexander v. Sandoval, 532 U.S. 275, 293 (2001). This decision renders largely irrelevant the literature applying disparate impact to federally funded health care. *E.g.*, Daniel K. Hampton, *Title VI Challenges by Private Parties to the Location of Health Care Facilities: Toward a Just and Effective Action*, 37 B.C. L. REV. 517 (1996); Sidney D. Watson, *Reinvigorating Title VI: Defending Health Care Discrimination—It Shouldn't Be So Easy*, 58 FORDHAM L. REV. 939 (1990).

Finally, Title II of the Civil Rights Act of 1964 prohibits discrimination or segregation in a place of public accommodation.³¹³ This section would seem to reach at least some health care providers, such as hospitals, but even that is not clear,³¹⁴ and other provisions of the statute suggest a focus on places providing meals, lodging, or entertainment;³¹⁵ in any event, the case law is very sparse.³¹⁶ Assuming § 2000(a) can be invoked, it is probably limited to disparate treatment discrimination.

In short, all four legal regimes recognize a private cause of action only for intentional discrimination.³¹⁷ However, there are significant differences among the remedies. In order to understand the legal application of the various tools to these distinct problems, it is useful to review briefly the governing doctrine. Given the very limited reach of both Title II and Title VI, however, the focus will be on § 1981 and the Equal Protection Clause.

Section 1981 is both more restrictive and more sweeping than the Equal Protection Clause. The language of § 1981 has been definitively construed to bar denying on the basis of race the right of individuals to contract.³¹⁸ As amended by the 1991 Civil Rights Act, § 1981 now reaches the “making, performance, modification, and termination of contracts, and the enjoyment of all benefits, privileges, terms, and conditions of the contractual relationship.”³¹⁹ It also reaches both the public and private sectors, although there are some differences in application.³²⁰ While the most important

³¹³ See 42 U.S.C. § 2000a (2000).

³¹⁴ See *United States v. Med. Soc’y of S.C.*, 298 F. Supp. 145 (D.S.C. 1969) (holding a hospital covered, in part because of its cafeteria and snack bar); *but cf.* *Verhagen v. Olarte*, No. 89 CIV. 0300(CSH), 1989 WL 146265, at *5 (S.D.N.Y. Nov. 21, 1989) (finding hospital excluded).

³¹⁵ See, e.g., *Daniel v. Paul*, 395 U.S. 298 (1969). See also Note, *A Public Accommodations Challenge to the Use of Indian Team Names and Mascots in Professional Sports*, 112 HARV. L. REV. 904 (1999); Patrick J. Poff, *Welsh v. Boy Scouts of America: Defining the Scope of a “Place Of Public Accommodation” Under Title II of the Civil Rights Act of 1964*, 45 MERCER L. REV. 1137 (1994).

³¹⁶ See *Sims v. Mercy Hosp. of Monroe*, 451 F.2d 171 (6th Cir. 1971); *Med. Soc’y of S.C.*, 298 F. Supp. at 145.

³¹⁷ The difficulty of proving intent to discriminate will vary substantially depending on the context in which the discrimination arises.

³¹⁸ *Johnson v. Ry. Express Agency*, 421 U.S. 454, 459–60 (1975) (“[I]t is well settled among the federal Courts of Appeals—and we now join them—that § 1981 affords a federal remedy against discrimination in private employment on the basis of race.” (footnote omitted)).

³¹⁹ 42 U.S.C. § 1981(b) (2000).

³²⁰ In practice, § 1981 may be less rigorous where state and local governments are concerned for reasons related to technical doctrines such as immunities and respondeat superior. While § 1981 provides that “[t]he rights protected by this section are protected against impairment by nongovernmental discrimination and impairment under color of State law,” 42 U.S.C. § 1981(c), only one circuit has held that this provision broadly authorizes suit against state actors. Three circuits have rejected this view. Compare *Fed’n of African Am. Contractors v. City of Oakland*, 96 F.3d 1204 (9th Cir. 1996) (concluding 1991 amendments to § 1981 imply a cause of action against state actors), with *Oden v. Oktibbeha County*, 246 F.3d 458 (5th Cir. 2001); *Butts v. County of Volusia*, 222 F.3d 891 (11th Cir. 2000); *Dennis v. County of Fairfax*, 55 F.3d 151, 156 (4th Cir. 1995). The effect of limit-

applications of § 1981 have been to employment, the provision reaches any kind of contractual relationship³²¹ under normal contract principles.³²²

Importantly, § 1981 contains no justification for racial discrimination in contracting; in that sense, § 1981 is more stringent than the Equal Protection Clause because nothing comparable to a compelling state interest has been recognized. Admittedly, there have been few instances where such a defense might have been attempted, but it has been rejected whenever raised.³²³ Indeed, the only exception to the ban of race discrimination recognized is the judicially created one of valid affirmative action plans, which also operates for Title VII.³²⁴ There has been some suggestion of limiting the reach of § 1981 by excluding contracts that are too “personal” to be controlled,³²⁵ but this would not apply in the areas of present concern even if such a construction were adopted.

ing § 1981 suits to those that can be prosecuted under § 1983 could be substantial. Since § 1983 does not reach states (as opposed to municipalities and individual state and local government officers and employees), limiting § 1981 to conduct also prohibited by § 1983 effectively immunizes states from § 1981 suits. *See Will v. Mich. Dep’t of State Police*, 491 U.S. 58 (1989).

³²¹ *See Runyon v. McCrary*, 427 U.S. 160, 170–71 (1976).

³²² In the health context, the statute has sometimes been used to challenge denials or non-renewals of physician privileges at health care facilities, which are normally viewed as beyond the reach of Title VII since an employment relationship is not involved. *See, e.g., Van v. Anderson*, 199 F. Supp. 2d 550 (N.D. Tex. 2002); *see also LeFlore v. Sea Breeze Nursing Home & Rehab. Ctr.*, No. 98-1136-AH-S, 2000 U.S. Dist. LEXIS 8872 (S.D. Ala. Jan. 21, 2000).

³²³ *See, e.g., Ferrill v. Parker Group*, 168 F.3d 468, 473 (11th Cir. 1999) (“Because § 1981 proscribes discrimination solely on the basis of *race*, and the BFOQ defense [in Title VII] does not apply to racial discrimination, the BFOQ [bona fide occupational qualification] defense is never available to the § 1981 defendant.” (emphasis added)); *see also Alexander v. Fulton County*, 207 F.3d 1303, 1338 (11th Cir. 2000) (“The question of race as a bona fide occupational qualification was never suggested.”).

As *Ferrill* states, Title VII permits discrimination when being of one sex or of a particular national origin or religion is “a bona fide occupational qualification” for the job in question. *Ferrill*, 168 F.3d at 473. This exception, however, does not include race. 42 U.S.C. § 2000e-2(e) (2000). The courts have uniformly found Congress’ decision to exclude race from the statutory BFOQ defense was intentional and, therefore, rejected any defenses once racial discrimination is proven. *See, e.g., Knight v. Nassau County Civil Serv. Comm’n*, 649 F.2d 157 (2d Cir. 1981) (noting that benign race-based intent in assigning plaintiff to minority recruitment not a defense); *Miller v. Tex. State Bd. of Barber Exam’rs*, 615 F.2d 650, 652 (5th Cir. 1980) (noting that race is “conspicuously absent” from the statutory language); *Swint v. Pullman-Standard*, 624 F.2d 525, 535 (5th Cir. 1980), *rev’d on other grounds*, 456 U.S. 273 (1982) (stating that since the omission of race as BFOQ was intentional by Congress, the defense is not available in race discrimination cases); *Burwell v. E. Air Lines*, 633 F.2d 361, 370 n.13 (4th Cir. 1980) (finding that statutory BFOQ defense is not available for race discrimination).

³²⁴ The Supreme Court’s approval of affirmative action plans under Title VII, *see, e.g., Johnson v. Transp. Agency*, 480 U.S. 616, 641–42 (1987); *United Steelworkers v. Weber*, 443 U.S. 193, 209 (1979), has been carried over into § 1981. *See, e.g., Schurr v. Resorts Int’l Hotel*, 196 F.3d 486, 498–99 (3d Cir. 1999) (“While a valid affirmative action plan serves as a defense to an action under section 1981, the standard for evaluating the validity of a plan is identical to the standard developed in Title VII cases.”).

³²⁵ In *Runyon*, 427 U.S. 160, where a private school violated § 1981 by excluding black children, Justice Powell wrote that § 1981 might not reach cases “[where] the choice made by the offeror is selective, it reflects a purpose of exclusiveness’ other than the desire

The mere fact that the race discrimination is not motivated by animus, and may even be benevolently motivated, is irrelevant in § 1981, as is the claim that the discrimination is rational a defense. Both points are illustrated by the Title VII cases refusing to allow gender-based discrimination in pensions even though women as a class do outlive men as a class and discrimination in benefits or contributions merely reflected that actuarial reality.³²⁶

3. First Amendment Constraints

Whatever the dictates of the various statutes in the abstract, their application is constrained by the First Amendment. A recent example is the Supreme Court's invalidation, in *Boy Scouts of America v. Dale*,³²⁷ of a state public accommodation law to the extent it would require a nonprofit organization to admit individuals whose presence would affect in a significant way the organization's ability to advocate its viewpoint. While the right of "expressive association" at issue in *Dale* is not likely to apply often in the health care context, the more general right of free speech limits government efforts to restrict private speech.

Much of the speech at issue in this setting—discussing genetic or socially constructed racial differences and the superiority of racially differentiated treatment—will be generally protected under the First Amendment since it will reflect scientific discussion or policy debate. Under this regime, while time, place and manner restrictions are possible,³²⁸ speech cannot be otherwise restricted unless there is a compelling state interest and the restriction is carefully tailored to that interest.³²⁹

In some contexts, however, some speech may be subject to greater regulation. There are three such situations. Most obviously, in *Rust v. Sullivan*³³⁰ the Supreme Court upheld limiting the speech rights of publicly funded speakers in order to enable the government to convey its own message.³³¹ Second, the government, as part of its regulatory power, may be

to bar members of the Negro race. Such a purpose, certainly in most cases, would invoke associational rights long respected." *Id.* at 187–88 (Powell, J., concurring). Powell's opinion has generated little law limiting § 1981's application, but it may have a resurgence in light of the Supreme Court's decision in *Boy Scouts of America v. Dale*, 530 U.S. 640 (2000), limiting state antidiscrimination laws because of freedom of association concerns.

³²⁶ See, e.g., *Los Angeles Dep't of Water & Power v. Manhart*, 435 U.S. 702 (1978).

³²⁷ 530 U.S. 640 (2000).

³²⁸ See, e.g., *Int'l Soc'y for Krishna Consciousness v. Lee*, 505 U.S. 672 (1992).

³²⁹ See, e.g., *Cal. Democratic Party v. Jones*, 530 U.S. 567, 582 (2000) (because of its heavy burden on a political party's associational freedom, "Proposition 198 is therefore unconstitutional unless it is narrowly tailored to serve a compelling state interest."); *United States v. Playboy Entertainment Group Inc.*, 529 U.S. 803, 813 (2000) ("If a statute regulates speech based on its content, it must be narrowly tailored to promote a compelling Government interest.").

³³⁰ 500 U.S. 173 (1991).

³³¹ See Paula Berg, *Toward a First Amendment Theory of Doctor-Patient Discourse and the Right to Receive Unbiased Medical Advice*, 74 B.U. L. REV. 201 (1994); Robert C.

able to require health care providers to provide certain information to patients.³³² Third, and potentially broadest, is the doctrine of “commercial speech,”³³³ which entails lower scrutiny than other First Amendment exercises.³³⁴ A drug company’s advertising a product or even a doctor’s counseling a patient about treatment may be characterized as “commercial speech” under the Supreme Court’s taxonomy, and therefore subject to less exacting scrutiny than when pure speech is concerned.³³⁵ To that extent, restricting physician or pharmaceutical company communications under § 1981 may be permissible.

However, even under the more relaxed review applied to speech categorized as commercial, the Court has struck down restrictions on speech in the health arena. Most recently, *Thompson v. Western States Medical Center*³³⁶ invalidated FDA restrictions on advertising compounded drugs:

[W]e ask as a threshold matter whether the commercial speech concerns unlawful activity or is misleading. If so, then the speech is not protected by the First Amendment. If the speech concerns lawful activity and is not misleading, however, we next ask “whether the asserted governmental interest is substantial.” If it is, then we “determine whether the regulation directly advances the governmental interest asserted,” and, finally, “whether it is not more extensive than is necessary to serve that interest.” Each

Post, *Subsidized Speech*, 106 YALE L.J. 151 (1996).

³³² The plurality so held in *Planned Parenthood v. Casey*, 505 U.S. 833 (1992), although a majority did not go so far. The plurality spoke of “the giving of truthful, non-misleading information,” in that case upholding requirements “about the nature of the procedure, the attendant health risks and those of childbirth, and the ‘probable gestational age’ of the fetus.” *Id.* at 882. The extent to which a physician would be permitted to distance herself from such disclosures by disagreeing with them while making them is unclear. See Berg, *supra* note 330, at 201.

³³³ See generally Alex Kozinski & Stuart Banner, *Who’s Afraid of Commercial Speech?*, 76 VA. L. REV. 627, 628 (1990); Robert Post, *The Constitutional Status of Commercial Speech*, 48 UCLA L. REV. 1 (2000); Frederick Schauer, *Commercial Speech and the Architecture of the First Amendment*, 56 U. CIN. L. REV. 1181 (1988); Rodney A. Smolla, *Information, Imagery, and the First Amendment: A Case for Expansive Protection of Commercial Speech*, 71 TEX. L. REV. 777, 780 (1993). Articles focusing on the doctrine as it relates to health care and science generally include Berg, *supra* note 330; Margaret Gilhooley, *Constitutionalizing Food and Drug Law*, 74 TUL. L. REV. 815 (2000); Lars Noah & Barbara A. Noah, *Liberating Commercial Speech: Product Labeling Controls and the First Amendment*, 47 FLA. L. REV. 63 (1995); Martin H. Redish, *Product Health Claims and the First Amendment: Scientific Expression and the Twilight Zone of Commercial Speech*, 43 VAND. L. REV. 1433 (1990); Charles J. Walsh & Alissa Pyrich, *FDA Efforts to Control the Flow of Information at Pharmaceutical Industry-Sponsored Medical Education Programs: A Regulatory Overdose*, 24 SETON HALL L. REV. 1325 (1994).

³³⁴ While *Planned Parenthood* was decided on other grounds in the Supreme Court, the Third Circuit had upheld the speech requirements under a commercial speech analysis. *Planned Parenthood v. Casey*, 947 F.2d 682, 705–06 (3d Cir. 1991), *aff’d in part and rev’d in part*, 505 U.S. 833 (1992).

³³⁵ See Daniel Halberstam, *Commercial Speech, Professional Speech, and the Constitutional Status of Social Institutions*, 147 U. PA. L. REV. 771 (1999).

³³⁶ 535 U.S. 357 (2002).

of these latter three inquiries must be answered in the affirmative for the regulation to be found constitutional.³³⁷

In the case before the Court, the advertising proposed a legal transaction and was not misleading. While the Court was willing to assume that the regulation directly furthered a substantial government interest, it found that a ban on compounding was more extensive than necessary to advance that interest.³³⁸

The significance of the First Amendment to our inquiry depends in large part on whether the government is attempting to restrict speech (as opposed to conduct) whether the speech is categorized as “pure” or “commercial,” and the strength and fit of the government’s justification for the restriction. Thus, any definitive assessment would require focusing on a concrete case. However, there are serious First Amendment obstacles to any reading of the antidiscrimination laws that would discourage health care organizations from providing information or medical opinions on the race-relatedness of medical screening or treatment.

B. Applying Legal Regimes and First Amendment Limitations to Racial Profiling

Both the application of the antidiscrimination regimes to the health arena and any First Amendment limitations on them are, at this point, largely theoretical. Putting aside decisions challenging employment discrimination or privilege denials, there have been remarkably few cases involving race discrimination in health care, and only a few more dealing with free speech in the health context.³³⁹ The paucity of decisions probably results from the intent requirement for strict scrutiny of governmental classifications: at least since the 1960s,³⁴⁰ few systemic health-related deci-

³³⁷ *Id.* at 367 (quoting *Cent. Hudson Gas & Elec. Corp. v. Pub. Serv. Comm’n*, 447 U.S. 557, 566 (1980) (citations omitted)). *See also* *Bolger v. Youngs Drug Prods. Corp.*, 463 U.S. 60 (1983) (invalidating application of federal ban on sending unsolicited information regarding contraceptives through the mail); *Va. State Bd. of Pharmacy v. Va. Citizens Consumer Council*, 425 U.S. 748 (1976) (invalidating state statute limiting advertising of prescription drugs). *But see* *Friedman v. Rogers*, 440 U.S. 1 (1979) (upholding state law limiting use of trade names for optometrists). *Western States Medical Center* recognized that *Central Hudson* had been much criticized, but found that the case before it could be resolved under that test. *W. States Med. Ctr.*, 535 U.S. at 367–68.

³³⁸ *W. States Med. Ctr.*, 535 U.S. at 373 (“The Government has not offered any reason why [various alternative] possibilities, alone or in combination, would be insufficient to prevent compounding from occurring on such a scale as to undermine the new drug approval process.”).

³³⁹ For example, in the 1960s, the desegregation of hospitals and nursing homes led to a handful of decisions. *E.g.*, *Simkins v. Moses H. Cone Mem’l Hosp.*, 323 F.2d 959 (4th Cir. 1963). But even there the focus was on whether government involvement was sufficient to satisfy the threshold state action requirement. *Rust v. Sullivan*, 500 U.S. 173 (1991), is another notable example.

³⁴⁰ *E.g.*, *Cypress v. Newport News Gen. & Nonsectarian Hosp. Ass’n*, 375 F.2d 648 (4th Cir. 1967) (enjoining hospital from denying African American physicians admission

sions plausibly could be litigated on the basis of intentional discrimination,³⁴¹ and individual cases of discrimination pose serious proof problems.³⁴² As for the First Amendment, almost all the challenged state regulations have related to drugs or optometry, with most of the regulations being struck down.³⁴³

However, the growing interest in “racial profiling” in medicine raises the possibility of new challenges. This section attempts to survey some of the developments in this regard and addresses the legal analysis of various kinds of “profiling” designed to improve the health of African Americans and other racial minorities. It approaches the question by asking how the four legal tools—and particularly the Equal Protection Clause and § 1981—bear on the five potential uses of race we discussed in Part I: (1) race-focused studies; (2) race-related outreach; (3) race-based clinical screening; (4) race-based treatment; and (5) race-based clinical trials. We also examine the extent to which these tools are constrained by the First Amendment.

1. Race-Focused Studies

Race-focused studies³⁴⁴ are perhaps the least problematic use of race from a legal standpoint, and in fact reflect normal practices today.³⁴⁵ “Studies” include surveys that collect information in the course of delivering

to the medical staff and assigning patients to hospital rooms on a segregated basis); *Eaton v. Grubb*, 329 F.2d 710 (4th Cir. 1964) (permitting suit to enjoin North Carolina hospital from continuing to deny staff privileges on a racially discriminatory basis and segregating patients).

³⁴¹ A series of challenges to hospital siting decisions in the early 1980s were rejected because the plaintiffs could not establish disparate treatment, and any disparate impact under Title VI was found justified. *See NAACP v. Med. Ctr., Inc.*, 657 F.2d 1322 (3d Cir. 1981); *Bryan v. Koch*, 627 F.2d 612 (2d Cir. 1980).

³⁴² Professor Mary Crossley addresses the difficulties of proving intentional discrimination in physician treatment decisions using the proof schemes developed in connection with employment discrimination under Title VII and related statutes. Crossley, *supra* note 31, at 280–91. She also addresses “indirect” ways of challenging physician discrimination, that is, using medical malpractice, informed consent and breach of fiduciary duty. *Id.* at 244–57. Her article explores unique difficulties of proving discrimination in this context, especially privileges and privacy rules which may hamper obtaining data necessary for cross-racial comparisons. *Id.* at 258–61.

³⁴³ *See* cases cited *supra* note 337.

³⁴⁴ For our definition, see *supra* text accompanying note 37.

³⁴⁵ *See* U.S. Dep’t of Health & Human Servs., Policy Statement on Inclusion of Race and Ethnicity in DHHS Data Collection Activities (Oct. 24, 1997) (letter from Donna E. Shalala, U.S. Sec’y of Health & Human Servs., to Heads of Operating Divisions and Heads of Staff Divisions), available at <http://www.hhs.gov/oirm/infocollect/nclusion.html> (last modified Sept. 1, 2000); *see also* Sidney D. Watson, *Race, Ethnicity and Quality of Care: Inequalities and Incentives*, 27 AM. J.L. & MED. 203, 221–23 (2001) (noting the need for health care providers to collect and report patient data by race in order to address racial treatment disparities). *But see* Bowser, *Racial Profiling*, *supra* note 34 (arguing that race-based medical research actually promotes racial stereotypes and leads to inferior medical treatment).

services but do not affect the treatment.³⁴⁶ They also include genetic studies, where the genomes of subjects are tested for various alleles. But they do not include clinical trials, which we discuss in another section.

Under current law, studies (at least as we define the term) are almost certain to be permissible, particularly if the data are simply classified by race.³⁴⁷ The initial question is semantic—a study that keeps data by race is, formally, a “classification,” and therefore would seem subject to strict scrutiny under the Equal Protection Clause. However, the word “classification” in equal protection jurisprudence is shorthand for classifying individuals in order to impose a harm or deny a benefit.³⁴⁸ Statistical data, as such, cannot do either, and therefore the creation and retention of such data by the government may not need any justification. Similarly, such activities would scarcely violate the nondiscrimination commands of § 1981 or Titles II or VI.

On the other hand, the creation of such data may suggest its use—indeed, there seems to be little point to collecting data unless some use is possible. Further, data collection may itself have a chilling effect on certain activities. In addition, there is an expressive aspect to such collection that has been decried as a policy matter,³⁴⁹ and which may raise legal

³⁴⁶ See, e.g., INSTITUTE OF MEDICINE, *supra* note 15.

³⁴⁷ If the study is designed to include particular racial groups, it may raise some of the same problems we ascribe to clinical trials. See *infra* Part IV.B.5.

³⁴⁸ See, e.g., *Nordlinger v. Hahn*, 505 U.S. 1, 10 (1992) (“The Equal Protection Clause does not forbid classifications. It simply keeps governmental decision makers from treating differently persons who are in all relevant respects alike.”); see also Michelle Adams, *The Last Wave of Affirmative Action*, 1998 WISC. L. REV. 1395, 1436. Melissa L. Saunders, *Equal Protection, Class Legislation, and Colorblindness*, 96 MICH. L. REV. 245 (1997), writes:

In traditional equal protection jurisprudence, however, the terms “classify” and “discriminate” have much more specialized meanings. To “classify” means to sort people into groups for differing benefits or burdens under the law, and to “discriminate” means to single out for special benefits or burdens not accorded others.

The distinction between the legal and lay usages of these two terms is seldom of any practical importance in equal protection litigation, for most state action *does* have the effect of singling out certain persons or groups of persons for special benefits or burdens. But a state *can* classify persons in the lay sense—that is, divide, sort, or arrange them into classes—without subjecting those classes to differing benefits or burdens. For example, it can sort persons into classes for informational purposes, as it does when it gathers and organizes census or other demographic data. Such a classification does not, in and of itself, implicate the Equal Protection Clause, for it does not single out any class of persons for special benefits or burdens.

Id. at 314–16 (citations omitted).

³⁴⁹ For one application of such an approach to health care, see Bowser, *Racial Profiling*, *supra* note 34:

The biomedical professions are largely responsible for creating, reproducing, and legitimating these race-based expectations. Within the disciplines of science and medicine, biomedical researchers continue the historical practice of attempting to

concerns. While these arguments would not justify the application of § 1981 or Titles II or VI, they may suggest that governmental racial classifications fall within the Equal Protection Clause and require justification by a sufficiently strong governmental interest.³⁵⁰

The case law is sparse and inconclusive. A handful of cases invalidate racial data-keeping by the government³⁵¹ (as opposed to myriad cases in which racial identification was used to prove that a subsequent action was, in fact, race-based). For example, *Anderson v. Martin*³⁵² invalidated racial designations in elections, and, in a case closer to the medical data context, *Hamm v. Virginia State Board of Elections*,³⁵³ a three-judge court struck down a Virginia law that required public records regarding voting and property taxes be maintained with racial designations, though the plaintiffs were not discriminated against in any way. The same court, however, upheld racial designations in divorce records. It wrote: “[T]he securing and chronicling of racial data for identification or statistical use violates no constitutional privilege. If the purpose is legitimate, the reason justifiable, then no infringement results. . . . Vital statistics, obviously, are aided by denotation in the divorce decrees of the race of the parties.”³⁵⁴ Other courts have struck down racial collection of information, but usually in the context of a threatened or intended use against one race.³⁵⁵ Generally speaking, where the data collection has been justified by

attribute health differences to race-based biological and cultural differences between Black and White patients. Whether their motives are to reverse the health disadvantages of ethnic minority groups or curiosity about racial and ethnic differences, this race-based research has not discovered the causes and processes of diseases. Instead, race-based research into health differences may paradoxically help to support institutionalized expectations and suspicions within the medical community that unwittingly produce the disparities in treatment that most researchers rightly decry.

Id. at 81 (citation omitted). See *supra* text accompanying notes 43–48 for a broader discussion of expressive harms in this context.

³⁵⁰ Of course, as Richard Primus, *supra* note 302, has pointed out, a court may reach this result by simply refusing to categorize this as a racial classification in the first place.

³⁵¹ *E.g.*, *Avery v. Georgia*, 345 U.S. 559 (1953) (reversing conviction because color-coded jury cards, together with other evidence, gave rise to an inference of discrimination in jury selection).

³⁵² 375 U.S. 399 (1964).

³⁵³ 230 F. Supp. 156 (E.D. Va. 1964), *aff'd sub nom.* *Tancil v. Woolls*, 379 U.S. 19 (1964) (mem.).

³⁵⁴ *Hamm*, 230 F. Supp. at 158; *see also* *Bryant v. State Bd. of Assessment*, 293 F. Supp. 1379 (E.D.N.C. 1968) (three-judge court) (striking down a similar North Carolina statute).

³⁵⁵ *See* *Hall v. Pa. State Police*, 570 F.2d 86 (3d Cir. 1978) (finding that a police photography program targeted at black bank customers was impermissible). *But see* *Brown v. City of Oneonta*, 221 F.3d 329, 333-34 (2d Cir. 2000) (“[W]here law enforcement officials possessed a description of a criminal suspect, even though that description consisted primarily of the suspect’s race and gender, absent other evidence of discriminatory racial animus, they could act on the basis of that description without violating the Equal Protection Clause.”); *Doe v. State*, 479 So. 2d 369, 374 (La. Ct. App. 1985) (permitting Louisiana to designate plaintiff’s race on her birth certificate).

the need to enforce an antidiscrimination statute³⁵⁶ or would be used only for statistical analysis, as with census data,³⁵⁷ constitutional challenges have been rejected.³⁵⁸

However, statistics are rarely gathered for the joy of the chase: they are gathered to inform decision-making. Race-based statistics undoubtedly provide the basis for race-based decisions—and the use of such information may be unpredictable. For example, racial statistics have been offered as the basis for calculating tort damage awards.³⁵⁹ If the scant extant authority is correct, however, that mere possibility is not enough to trigger an equal protection violation and seems clearly not to violate § 1981 or either Title II or VI. The absence of legal remedies for such activities has generated proposals such as the Racial Privacy Initiative in California,³⁶⁰ which appeared on the ballot in last October's recall election. The Initiative, which failed, would have barred the classification of "any individual by race, ethnicity, color, or national origin in the operation of public education, public contracting, or public employment"³⁶¹ and prohibited racial classifications "in the operation of any other state operations, unless the Legislature specifically determines that said classification serves a compelling state interest" by a two-thirds vote in both houses.³⁶² As written, the Initiative did not apply to the "[o]therwise lawful classification of medical research subjects and patients."³⁶³ However, since the Initiative would have prevented other race-identified data collection, opponents argued that its adoption would have been a major blow to health research in California, if only because "medical" research frequently draws on data that is gathered for non-medical purposes.

³⁵⁶ See *Caulfield v. Bd. of Educ.*, 583 F.2d 605 (2d Cir. 1978) (affirming the denial of a preliminary injunction to prevent collection of racial data in connection with federal enforcement of Title VI against a school district); *United States v. New Hampshire*, 539 F.2d 277 (1st Cir. 1976) (noting that hypothetical misuse of racial data collected by the federal government did not justify finding unconstitutional a statute requiring such data to be provided).

³⁵⁷ See *Morales v. Daley*, 116 F. Supp. 2d 801, 813–15 (S.D. Tex. 2000), *aff'd sub nom. Morales v. Evans*, 275 F.3d 45 (5th Cir. 2001).

³⁵⁸ See *Edwards v. Garrison*, 544 F.2d 514 (4th Cir. 1976) (unpublished table decision) (noting that while gathering information by race is unobjectionable, the prospect of use of such information to segregate or discriminate may make racial identification of prisoners impermissible, absent a compelling state interest).

³⁵⁹ See Martha Chamallas, *Questioning the Use of Race-Specific and Gender-Specific Data In Tort: A Constitutional Argument*, 63 *FORDHAM L. REV.* 73 (1994).

³⁶⁰ See Racial Privacy Initiative, at <http://www.racialprivacy.org/> (last modified Oct. 9, 2003); see also Miguel Bustillo, *Ban on Gathering Racial Data on 2004 Ballot*, *L.A. TIMES*, July 16, 2002, at B7; Glenn C. Loury, *When Color Should Count*, *N.Y. TIMES*, July 28, 2002, § 4, at 13.

³⁶¹ CAL. SEC'Y OF STATE, OFFICIAL VOTER INFORMATION GUIDE 45, § 32(a) (2003); see also Proposition 54 to amend CAL. CONST. art. I (2003), available at <http://vote2003.ss.ca.gov/voterguide/english.pdf>.

³⁶² CAL. SEC'Y OF STATE, OFFICIAL VOTER INFORMATION GUIDE § 32(b).

³⁶³ *Id.* § 32(f).

In sum, there are few legal problems for race-focused studies from the four federal regimes we have studied. If legal difficulties are to arise, they would be caused by efforts such as the now-defeated Racial Privacy Initiative.

2. *Race-Based Outreach*

One obvious use of statistical studies is to determine whether particular racial groups are at greater risk for particular diseases. In turn, a logical response to data indicating demographic susceptibilities is outreach to particular groups; private groups or governmental agencies may seek to educate particular racial groups about these facts.³⁶⁴ For example, if African Americans are at greater risk than the general population for hypertension, efforts to educate this group about the dangers of the condition and appropriate responses is an obvious reaction. Similarly, if cultural factors, for example, dietary preferences, contribute to certain diseases, a public health response might be to target the group whose practices are problematic.

Such outreach can take one of two forms. First, what we will refer to as “race-limited” outreach seeks to educate only one (racial or ethnic) group on various health issues.³⁶⁵ The second approach, which we will refer to as “race-targeted” outreach, seeks instead to communicate with all groups about diseases and treatments, but allows for variation in how each group is reached. There is no clear line delineating these two approaches; some outreach will fall somewhere between them. They are, nonetheless, useful categories for analyzing when the government should and should not engage in race-based outreach.

Section 1981 would seem inapplicable to both types of outreach, since no one is being denied the right to contract on the basis of race. There also seems to be little problem with Title II since it is difficult to describe such efforts as a “public accommodation.” As for Title VI, no one would be excluded from any program on the basis of race. Although an argument could be made that, regardless of the language of the statute, focusing a government-funded program on a particular race is precisely what Title VI is designed to avoid,³⁶⁶ it nevertheless may be hard to say that particular groups are being excluded in practice. Even race-targeted outreach may be aimed at particular geographic areas or media appealing to particular racial or ethnic groups, and necessarily would reach individuals

³⁶⁴ E.g., Jennifer Roemer, *Inner-City Mammography Programs Aim to Make Breast Cancer “A Visible Disease,”* 92 J. NAT’L. CANCER INST. 444 (2000).

³⁶⁵ An example might be Black Women’s Health Imperative, at <http://www.blackwomenshealth.org/site/PageServer> (last visited Mar. 13, 2004).

³⁶⁶ The textual argument is, of course, that a program intended to benefit (in this case, educate) a particular racial group necessarily excludes other groups “on the ground of race” from “the benefits of . . . Federal financial assistance.” 42 U.S.C. § 2000d (2000).

outside the race whose health difficulties justified the outreach in the first place.

The remaining question is the effects of the Equal Protection Clause on race-based outreach when government efforts are concerned. Both race-targeted and race-limited outreach might be constitutionally suspect if the Equal Protection Clause is understood to forbid any governmental action motivated by a racial purpose.³⁶⁷ After all, the purpose of both forms of race-based outreach is to benefit a particular race. In general, though, courts have classified efforts to increase minority participation in employment and housing as race-neutral, and therefore constitutionally permissible.³⁶⁸ Nonetheless, race-limited outreach, because it is designed to reach only one particular race, might not be viewed as race-neutral in this way; since the purpose is not to increase the information available for all persons but instead, to reach members of a particular race.³⁶⁹

Even if race-limited outreach is seen as racially motivated, it nonetheless may survive strict scrutiny. This answer seems obvious to three Supreme Court Justices, who compared the voting rights case before the Court to what they framed as a hypothetical: “Requiring the State to ignore the association between race and party affiliation would be no more logical, and potentially as harmful, as it would be to prohibit the Public Health Service from targeting African American communities in an effort to increase awareness regarding sickle cell anemia.”³⁷⁰ In these Justices’ view, race could legitimately be a proxy for the targeted matter if the correlation were close enough, and therefore strict scrutiny were inapplicable. In the case before the Court, however, the correlation between the trait at issue (tendency to vote Democratic in a particular community) and race was apparently close to perfect.³⁷¹

In the health context, this approach would permit government actors to engage in such outreach if the data were to show a close correlation with race. And, of course, private actors would in no way be constrained.

³⁶⁷ See Adams, *supra* note 348, at 1438 (noting but rejecting argument).

³⁶⁸ Duffy v. Wolle, 123 F.3d 1026, 1038–39 (8th Cir. 1997) (“An employer’s affirmative efforts to recruit minority, female applicants does not constitute discrimination.”); Ensley Branch, NAACP v. Seibels, 31 F.3d 1548, 1571 (11th Cir. 1994) (“True, the City and the Board have engaged in several race-neutral efforts to cure past and present discrimination. In the 1960s, the Board actively encouraged blacks to apply for jobs, and either waived or eliminated certain application fees.”). See also Adams, *supra* note 348, at 1453–54.

³⁶⁹ Cf. Adams, *supra* note 348, at 1438 (arguing that much “soft” affirmative action is race-neutral because it seeks to open up opportunities in general).

³⁷⁰ See Bush v. Vera, 517 U.S. 952, 1032 (1996) (Stevens, J., dissenting, joined by Ginsburg and Breyer, JJ.).

³⁷¹ *Id.* at 1031 (“It is neither irrational, nor invidious, however, to assume that a black resident of a particular community is a Democrat if reliable statistical evidence discloses that 97% of the blacks in that community vote in Democratic primary elections.”). Ironically, the correlation between being black and having sickle cell is far less strong.

3. Race-Based Screening

Race-based clinical screening would seem to create slightly more substantial legal issues. By “screening” we mean the use of certain diagnostic tests on entire groups of people, either the entire population or a population group or subgroup. We distinguish this from “testing,” by which we mean the use of diagnostic tests on an individualized basis.³⁷²

Screening would include such programs as the routine testing of all newborns for PKU, as is mandated in almost all states.³⁷³ An example of testing would be the use of tests for the *BRCA1* and *BRCA2* alleles in individuals who have a family history of breast cancer.³⁷⁴ The line between what we refer to as screening and testing is by no means clear. For instance, should testing of Ashkenazi Jewish women for the Tay-Sachs allele be considered screening? Probably yes, but it could also be understood as testing. Nonetheless, we believe these two labels are useful for our purposes. In this section, we conclude that race-based screening is generally illegal. We view testing as a subcategory of treatment because it (should) involve individualized assessments, and we defer our discussion to the next section.

Screening will run afoul of § 1981 only if the health care provider denies the patient the test without meaningful patient choice. Put another way, informing an African American woman that her greater risk factors indicated earlier mammographies than an otherwise similarly situated white woman³⁷⁵ would not abridge her right to contract (even aside from any First Amendment concerns). Any resultant differences in screening would be the result of patient choice, not provider discrimination.³⁷⁶

Actually delivering a different standard of care on racial grounds in terms of screens administered without informed patient choice, though, seems impermissible under § 1981. For example, offering screening for

³⁷² See, e.g., NEW YORK STATE TASK FORCE ON LIFE AND THE LAW, GENETIC TESTING AND SCREENING IN THE AGE OF GENOMIC MEDICINE 49, 103 (2000) [hereinafter N.Y. STATE TASK FORCE].

³⁷³ See M. Susan Lindee, *Babies' Blood: Fragmentation, Redemption, and Phenylketonuria*, 75 CHI.-KENT. L. REV. 113 (1999).

³⁷⁴ See, e.g., N.Y. STATE TASK FORCE, *supra* note 372.

³⁷⁵ Black women seem to have a higher incidence of breast cancer than white women. See Carrie P. Hunter et al., *Breast Cancer: Factors Associated with Stage at Diagnosis in Blacks and White Women*, 85 J. NAT'L CANCER INST. 1129, 1129–37 (1993) (“[N]o single factor or group of factors can explain more than half of the race-stage differences” between black and white women.).

³⁷⁶ Even if such conduct were within the statute, it would seem to be protected by the First Amendment to the extent that the doctor or other provider was merely providing information for patient choice. See *supra* note 328 and accompanying text. The effectiveness of patient consent, in terms of the ability of patients to rationally process information provided to them, has been a subject of increasing debate in the informed consent literature, see sources cited *supra* note 293, but no cause of action for discrimination will lie against a healthcare provider who simply implements a patient's desires after appropriately informing her of the information available.

sickle cell anemia only to African American newborns—as some states have done historically and one state may do now—would be a plain violation of § 1981.³⁷⁷ Similarly, ordering a mammogram for an African American when the test would not be ordered for a similarly situated white woman, in the context of a contract for medical care, is classic disparate treatment. While it may be benignly motivated, such a practice imposes costs and discomfort on the basis of race.³⁷⁸ In an era of less health insurance and more patient contribution even when there is insurance, as with higher deductibles and co-pays, much of the cost of such a practice will be directly borne by the patients.³⁷⁹ Similarly, a health maintenance organization or other insurer that reimbursed or referred African American women in circumstances when it would not reimburse or refer members of other races³⁸⁰ would seem to fall afoul of § 1981's prohibition of racial discrimination in contracts.³⁸¹ As we have seen, there is no recognized defense to this bar.

Finally, the Equal Protection Clause might well prevent the government from requiring or implementing racially premised screening. Theoretically, a firm medical foundation for the racial distinctions in screening might satisfy the compelling state interest test, but it would rarely pass the narrowly tailored requirement. That is, it would rarely be necessary to require or engage in racial screening as opposed to screening individuals of all races.

In sum, race-based screening poses the risk of legal liability for both private and public actors. When the action is simply the dissemination of information about racial distinctions, the practitioners would seem to be protected by the First Amendment. But in the absence of fully informed patient consent, racial distinctions would seem impermissible, and even

³⁷⁷ For a discussion of past and present practices, see *infra* text accompanying notes 442–447.

³⁷⁸ Proof of disparate treatment can be very difficult. Absent admission by the practitioner, such proof might depend on a comparison of treatment patterns from which an intent to discriminate can be inferred. See CHARLES A. SULLIVAN, MICHAEL J. ZIMMER, & REBECCA HANNER WHITE, *EMPLOYMENT DISCRIMINATION: LAW AND PRACTICE* ch. 2 (2002). As Professor Crossley suggests, privilege and privacy interests may hamper discovery of such information. See *supra* note 207, at 258–59.

³⁷⁹ Of course, a white woman who was denied a mammogram on this basis would also have a cause of action.

³⁸⁰ Some insurers use ethnicity to determine reimbursement for preconception and prenatal screening for Tay-Sachs. N.Y. STATE TASK FORCE, *supra* note 372, at 380.

³⁸¹ However, the § 1981 remedial scheme may be problematic. Some violations may have easily ascertainable damages: for example, an insurance company refusing to pay for a screening procedure on racial grounds. The cost of the procedure is, typically, easily established, and, for someone who paid for the procedure out of her own pocket, should be recoverable. More complicated is determining if consequential damages are available for denial of screening and/or treatment on racial grounds. Obviously, some denials of screening and/or treatment will result in no harm at all—perhaps even a benefit. Even in cases where the failure to screen and/or treat can plausibly be linked to a harm—such as a cancer not detected in its early stages because of a failure to screen—the remedial responses may be limited.

patient consent will not protect racial differences in insurance coverage under § 1981. Such distinctions would pass scrutiny only if a court were willing to conclude that the failure to provide such screening to other groups were medically necessary (and private actors would not even have this defense under existing law).

4. Race-Based Treatment

Discussions of race-related treatments by health care practitioners with patients will typically not constitute discrimination under any of the statutory schemes or, if they do, will be protected by the First Amendment. For example, informing an African American male that prostate cancer was more dangerous for African Americans than for whites and recommending or providing more aggressive treatment would not be actionable.

However, making treatment decisions or offering different treatment options solely on the basis of race (for example, surgery for African Americans when whites are provided less invasive care) seems a plain violation of § 1981.³⁸² Thus, decisions by health care practitioners to provide or deny treatments using the patient's race without full patient consent would be illegal. Similarly, decisions by health insurers to subsidize certain treatment for some patients but not others based on race would also be illegal.

Considering race as one element in determining a treatment regimen, however, poses more difficult legal issues. For instance, Dr. Satel, a leading conservative public intellectual, proclaimed in the *New York Times Magazine* that she and other doctors routinely prescribe Prozac at one-half the dosage to African American patients as compared to white patients.³⁸³ Without consent, such actions appear to violate § 1981. But presumably she and the other doctors later adjust their dosages based on the actual result achieved by Prozac. They would argue that the correct dosage is reached, regardless of whether it started at a lower level for African Americans and was increased (if indicated) or whether it was started at a higher level for whites and was decreased (if indicated). Framing this in legal terms, Dr. Satel and her colleagues might attempt two kinds of defenses. The first is simply that any discrimination is too insignificant to be cognizable. While there is some basis for this under Title VII,³⁸⁴ § 1981 has not

³⁸² It will not always be clear who the victims of race-based distinctions are. While at first it may seem that only white women would be hurt by a policy that covered mammographies for African American women at a younger age, some screening may itself be dangerous. Further, in the case of differential treatment of whites and African Americans with otherwise similar prostate cancer, both whites and blacks may have a claim—whites for being denied possibly life-saving care and blacks for having been subjected to more aggressive surgery.

³⁸³ Satel, *supra* note 2, at 56.

³⁸⁴ In the employment context, some circuits have found certain conduct not actionable because it does not constitute an "adverse employment action." *E.g.*, *Primes v. Reno*, 190 F.3d 765 (6th Cir. 1999) (job evaluation putting plaintiff in mid-range is not actionable);

recognized the defense for discrimination, although the less harm done, the less sweeping the remedy. The second is arguing that, although race was *a* factor in determining the course of treatment, the same decision would have been reached in any event, thus negating any violation.³⁸⁵ Thus, the clinicians would have to show that, while race may have been taken into account, the same actions would have been taken if race were not considered.

We suspect that either defense would often apply in the cases most likely to be litigated—when the treatment had gone seriously awry. “Overdosing” a patient on a drug such as Prozac is likely to cause little harm, and therefore not likely to result in suit—although, obviously there are drugs for which this is not true. But “underdosing” with Prozac might be plausibly traced to subsequent harm to a patient—for example, if the patient commits suicide.³⁸⁶ Consider an African American patient who was a normal metabolizer of Prozac, and was harmed by the clinician’s failure to prescribe him the higher dosage. It is hard to imagine that the clinician could show that she would have prescribed the lower dosage even without reference to the plaintiff’s race. Presumably, for instance, Dr. Satel

Enowmbitang v. Seagate Tech. Inc., 148 F.3d 970, 973 (8th Cir. 1998) (employer’s failure to provide plaintiff with a computer “does not rise above a ‘mere inconvenience’ and therefore does not constitute an adverse employment action”); *Williams v. Bristol-Myers Squibb Co.*, 85 F.3d 270 (7th Cir. 1996) (lateral transfer not actionable when any resulting diminution in pay was “indirect and minor”). *But see* Rebecca Hanner White, *De Minimis Discrimination*, 47 EMORY L.J. 1121 (1998) (critiquing the approach).

³⁸⁵ In *Price Waterhouse v. Hopkins*, 490 U.S. 228 (1989), the Supreme Court recognized that sex (and, by extension, race) could be a factor in an adverse employment decision but that the employer might have made the same decision in any event. If the plaintiff proved that race was a factor, the defendant could nevertheless avoid all liability by proving by a preponderance of the evidence that it would have made the same decision in any event. The Civil Rights Act of 1991 amended Title VII to permit the same decision defense, but made it a limitation of remedies rather than a complete defense. Under current Title VII law, once an employee proves that a prohibited consideration was a motivating factor, liability attaches, although the defendant may limit the plaintiff’s remedies by proving it would have made the same decision had race not been involved. *See, e.g.*, *Desert Palace, Inc. v. Costa*, 123 S. Ct. 2148 (2003). Section 1981 has been interpreted to reach the same result. *See, e.g.*, *Mabra v. United Food & Commercial Workers Local Union No. 1996*, 176 F.3d 1357 (11th Cir. 1999); *Harris v. Shelby County Bd. of Educ.*, 99 F.3d 1078 (11th Cir. 1996). *See generally* Joanna L. Grossman, *The Second Circuit Review: 2000–2001 Term: Making a Federal Case Out of It: Section 1981 and At-Will Employment*, 67 BROOKLYN L. REV. 329 (2001).

³⁸⁶ The concepts of “overdosing” and “underdosing” are obviously problematic in this setting. Presumably, for most drugs there is a standard dose (at least for patients of given weight or clinical condition), which is suitable to most patients. Giving a white the standard dose might be hard to describe as an “overdose,” even though it turns out to have harmful effects from hindsight. But a departure from the standard—whether more or less—should turn on clinical judgments of what is likely to be beneficial for this patient—and how easy or hard it is to use alternative methods to determine whether the individual patient should receive more or less. It is here that physicians like Dr. Satel apparently use race as a proxy for metabolism.

prescribes Prozac at higher levels in the absence of information about the patient's race.³⁸⁷

The Equal Protection Clause analysis is slightly more difficult. Racially differentiated treatment by state actors raises concerns identical to those in any other system of racial differentiation. It seems likely, therefore, that strict scrutiny would apply. While such treatment will be benignly motivated, that will not preclude such scrutiny. As we noted in the previous section, a sufficient degree of scientific certainty or genetic differences that are either "racial" or sufficiently correlated with race is likely to constitute a compelling state interest for such differences in treatment.³⁸⁸ But such decisions by governmental actors would still violate the Equal Protection Clause unless they are narrowly tailored. In normal circumstances other mechanisms—such as questions about social or cultural conditions or genetic testing—will be available.³⁸⁹

In sum, in most circumstances the use of race in treatment decisions will also violate the Equal Protection Clause, either because race is insufficiently predictive of treatment success or because other mechanisms for such prediction exist.

5. *Race-Based Clinical Trials*³⁹⁰

Clinical trials lie at the junction of studies and treatment. While most trials are designed to test the effectiveness of drugs as treatments for diseases, a minority focus on the effectiveness of a diagnostic test or a vaccine as a protective measure.³⁹¹ In the United States a typical clinical trial of a new drug is conducted subject to Food and Drug Administration ap-

³⁸⁷ However, note that this defense would work in a case where a slow-metabolizing white complained that the clinician should have given him a lower dosage. While the plaintiff's race played a role in the decision, the decision would have been the same absent information about race. Critical race theorists would use this as an illustration that the white norm has become the default standard, *see, e.g.*, Darren Lenard Hutchinson, "Unexplainable on Grounds Other than Race": *The Inversion of Privilege and Subordination in Equal Protection Jurisprudence*, 2003 U. ILL. L. REV. 615, but the reality remains that the physician only did what she would have done without regard to race.

³⁸⁸ Scientific evidence usually comes in various degrees of certainty, depending on the number and persuasiveness of the studies undertaken and their relation to the treatment at issue. We would anticipate a large degree of deference to clinical judgments, provided only that these judgments are grounded in research or solid clinical observation.

³⁸⁹ An exception would be for emergency treatment, where the physician may not have an opportunity to obtain such information.

³⁹⁰ By "clinical trials," we mean to include Phase I, Phase II and Phase III drug studies, as well as trials pursuant to an individual device examination for premarketing approvals of medical devices. *See generally* Barbara A. Noah, *The Participation of Underrepresented Minorities in Clinical Research*, 29 AM. J.L. & MED. 221 (2003).

³⁹¹ Vaccines are regulated as drugs. New diagnostic tests are regulated by a similar regime to the one that governs new drugs. Such tests constitute medical devices, which are covered by the Medical Device Amendments of 1976. *See* Steven R. Salbu, *HIV Home Testing and the FDA: The Case for Regulatory Restraint*, 46 HASTINGS L.J. 403, 440–41 (1995).

proval and is preceded by an Investigational New Drug (IND) application to the FDA.³⁹² The main exception is for drugs that are already licensed and approved by the FDA, but this exception applies only where no changes are made in the usage of or claims about the drug.³⁹³ For treatments not involving either drugs or medical devices, such as surgical techniques, FDA rules do not apply, but are subject to Department of Health & Human Services regulations if the research is federally funded.³⁹⁴

Absent approval of the IND application, a drug could not legally be used in the United States, and, therefore, could not be tested on human subjects legally. FDA approval is not a blanket permission to study the drug in question. Rather, the trial must proceed in accordance with strict protocols established by the agency: in particular, the drug usually passes through three stages of testing, known as Phase I, Phase II, and Phase III. Phase I testing is generally conducted only to test the safety of the drug; Phase II trials are limited studies of the effectiveness of the drug; and Phase III trials are expanded trials after evidence of drug effectiveness has been obtained.³⁹⁵ The results of the trials are then considered in the agency's decision to approve or reject the drug. The involvement of both federal and private entities in this process, and in federally funded research conducted by HHS grants and under federal regulations, raises legal questions about racial profiling in clinical trials.

Merely recording data in racial terms, as the FDA has recently required for all clinical trials, poses no problems not considered earlier.³⁹⁶ However, a clinical trial that is race-based in the sense that race is a criterion for admission, as seems to be the protocol with BiDil,³⁹⁷ presents significant legal issues. Normal § 1981 analysis would view admission to the trial as contractual, and, therefore, not able to be conditioned on race.³⁹⁸ Similarly, Title VI bars discrimination in admittance to any federally funded research. Neither of these statutes has recognized a justification defense for

³⁹² See CHARLES G. SMITH, *THE PROCESS OF NEW DRUG DISCOVERY AND DEVELOPMENT* 63–76 (1992).

³⁹³ See Dale E. Hammerschmidt, *Understanding the FDA's IND Process*, in *INSTITUTIONAL REVIEW BOARDS: MANAGEMENT AND FUNCTION*, 323, 323–24 (2002). Off-label use is permitted by physicians without an IND so long as it is not carried out to collect data for a licensing application. *Id.* at 324.

³⁹⁴ See Carl H. Coleman, *Rationalizing Risk Assessment in Human Subject Research*, 46 *ARIZ. L. REV.* 1, 5 (2004).

³⁹⁵ NOAH & NOAH, *supra* note 47, at 145–46.

³⁹⁶ See FDA Investigational New Drug Application, 21 C.F.R. § 312.33 (2003).

³⁹⁷ See NitroMed, *BiDil® for treatment of heart failure in African Americans*, at <http://www.nitromed.com/BiDil.shtml> (last visited Mar. 16, 2004).

³⁹⁸ Arguably, since admission to a clinical trial typically does not involve payment, the arrangement lacks the “bargained for” consideration necessary to establish a contract. Of course, some clinical trials do involve payment, and, in any event, participating in a trial may very well be viewed as “bargained for” consideration under normal contract analysis. The “bargain” is the subject's willingness to undergo risk in return for the possible benefits of the trial. See *RESTATEMENT (SECOND) OF CONTRACTS* §§ 71, 72 (1981).

conduct that would otherwise be prohibited, which strongly suggests that such actions would be impermissible.

Equal protection analysis seems even clearer. The government, through the FDA and HHS, may not discriminate when it authorizes or funds trials. Further, even lesser levels of government involvement may trigger normal constitutional analysis.³⁹⁹ To the extent that racial qualifications are revealed in HHS grant applications, the federal government would be knowingly funding discriminatory trials. To the extent that the FDA is approving INDs using race as a qualification, the agency is legalizing the use of drugs that would otherwise be illegal in order to authorize discrimination. This would seem to constitute a racial classification that could be justified only by the agency in question demonstrating a compelling interest for the discrimination and that the restriction was narrowly tailored.

Given our present state of knowledge, such a showing would appear difficult to make. As we noted in Part III, there are good reasons—both social and genetic—to suspect that racial distinctions in disease susceptibility and disease treatment exist. Such distinctions *might* constitute a compelling state interest, if significant enough, but may run afoul of the “narrowly tailored” requirement, because they are typically proxies for other causes. Consider, for instance, racial distinctions that result from different social conditions to which members of races are exposed. While health care practitioners and researchers might rely on race to predict whether an individual had been exposed to a certain environmental cause, other methods of determining exposure may be just as (if not more) accurate and would not involve using race. For instance, if the concern is that a particular racial group exercises less than other groups (on average), then instead of assuming anything about the patient’s exercise habits based on her race, the clinician should ask the patient directly about her exercise habits. The existence of such an alternative would seem to violate the narrowly tailored rule and require a finding that the use of race in such a circumstance would violate the Equal Protection Clause.

Racial distinctions made on the basis of genetic differences between racial groups would appear to be just as suspect. While the existence of such disparities might constitute a compelling state interest, the use of race again appears not to be “narrowly tailored.” As we noted in Part III, many “racial” distinctions in disease acquisition and treatment are really differences between a particular population group and all other groups. Thus, Tay-Sachs is not a “white” disease, but rather a disease afflicting Ashkenazi Jews. Indeed, as recent research has revealed, it may really be

³⁹⁹ Government financial support for racially segregated private schools has been found unconstitutional. *E.g.*, *Norwood v. Harrison*, 413 U.S. 455 (1973); *Graham v. Evangeline Parish Sch. Bd.*, 484 F.2d 649 (5th Cir. 1973).

a disease of certain subgroups of Ashkenazi Jews.⁴⁰⁰ Obviously, this observation is just as true of diseases like PKU and Maple Syrup Urine disease, which are also found in certain population subgroups, and it is also true of particular alleles for cystic fibrosis.⁴⁰¹ Indeed, even for more widespread diseases like sickle cell anemia, differences within racial groups are likely. Thus an African American, who we have defined as including anyone descended from the original people of sub-Saharan Africa, will have a different probability of being a carrier based on where in Africa her ancestor originated and the content of admixture with other races.⁴⁰² The ability to rely on such subgroups, rather than on race, would make the use of race a probable violation of § 1981 and the Equal Protection Clause.⁴⁰³

A more complicated question is posed by National Institutes of Health (NIH) rules that presumptively require the inclusion of minority groups in all NIH-funded clinical research. While the NIH does not mandate the inclusion of minority groups in *all* trials, it does require that researchers “address” the inclusion of such groups by describing the composition of

⁴⁰⁰ The data in Risch et al., *supra* note 204, suggests that one of the three mutations that cause Tay-Sachs is found almost exclusively among German, Russian, and Lithuanian populations, while a second mutation is found primarily (but not exclusively) among the Austrian population. The third and final mutation is more widespread but is still more heavily concentrated in the Austrian and Czech populations. *Id.* at 817 tbl.4.

⁴⁰¹ See Cooper et al., *supra* note 22.

⁴⁰² For instance, carrier rates in Southern Africa are significantly lower than in Central Africa. See Steinberg, *supra* note 107, at 817 tbl.29.4.

⁴⁰³ This raises the question of whether reliance on population subgroups might be discrimination based on “national origin.” In fact, the use of certain subgroups might constitute “racial” discrimination under § 1981. As we saw above, *supra* text accompanying note 71, in the mid-nineteenth century the term “race” applied to groupings that today we would view as groupings by national origin. To the extent that “Scandinavians” or “Gypsies,” for example, are races for purposes of the statute, even more focused differentiations may be actionable. Theoretically, testing *all* Jews for Tay-Sachs would be problematic under the statute, though perhaps testing all *Ashkenazi* Jews would not.

Given their shared history, the Equal Protection Clause is likely to be interpreted like § 1981 with regard to the meaning of “race.” While it does not explicitly refer to “race,” the primary purpose of the Fourteenth Amendment was to equalize the status of former slaves. Race discrimination, therefore, has been the paradigmatic suspect classification. However, the Court has not always been clear regarding what constitutes “race” for the purposes of a “racial classification.” Ironically, *Korematsu v. United States*, 323 U.S. 214 (1944), which originated the strict scrutiny method of analysis for racial classifications, could more readily be described as a case concerning national origin classification since the targets were descendants of Japanese immigrants. While the Court has sometimes spoken of national origin classifications as requiring strict scrutiny, the cases have typically involved racially premised groups (Japanese, Chinese, or Mexicans) and/or aliens, for whom there are other constitutional considerations. See, e.g., *Graham v. Richardson*, 403 U.S. 365, 371–72 (1971) (“[T]he Court’s decisions have established that classifications based on alienage, like those based on nationality or race, are inherently suspect and subject to close judicial scrutiny.”) (citing *Oyama v. California*, 332 U.S. 633, 644–46 (1948); *Korematsu v. United States*, 323 U.S. 214, 216 (1944); *Hirabayashi v. United States*, 320 U.S. 81, 100 (1943)). See generally Linda Bosniak, *Constitutional Citizenship Through the Prism of Alienage*, 63 OHIO ST. L.J. 1285 (2002); Earl M. Maltz, *Citizenship and the Constitution: A History and Critique of the Supreme Court’s Alienage Jurisprudence*, 28 ARIZ. ST. L.J. 1135 (1996).

the proposed study population in terms of “racial/ethnic group,” and it directs researchers to “provide a rationale for selection of such subjects.”⁴⁰⁴ Although there are exceptions, the policy appears to generally require the inclusion of such groups at some level, an action that seems to border on a quota for representation in clinical trials. The most detailed guidance is given in connection with Phase III drug studies, where inclusion is generally presumed, but not required where no significant differences between races have been demonstrated in prior studies.⁴⁰⁵

This policy may, in limited circumstances, violate the Equal Protection Clause. Because it clearly involves a racial classification, once again the ultimate answer turns on whether the NIH can demonstrate a compelling state interest and whether the guidelines are narrowly tailored. In cases involving drugs already shown to have differential effects on racial groups, there is a compelling state interest, and the forced collection of additional data is probably narrowly tailored to that interest. In cases where no prior racial effect has been shown, NIH policy does not require the inclusion of racial groups, thereby avoiding any constitutional difficulties. The most difficult cases would be those involving drugs where prior trials have neither shown nor negated the possibility of significant differences between races. Here, NIH policy requires the inclusion of minority groups, albeit at lower level than for drugs where an actual effect has been shown.⁴⁰⁶ It is arguable that such cases might involve a compelling state interest such as the lack of evidence for this drug, plus the reality that some other drugs do show such an effect, may be sufficient to justify some racial disparity. The close question for courts will be whether these rules are narrowly tailored. We believe that the NIH’s willingness to weaken the requirement for racial inclusion in such cases most likely would satisfy this standard.

The bottom line is that racial profiling in the admission of individuals to clinical screening is likely to violate the Equal Protection Clause, because there will often be equally (or nearly equally) valid criteria for selecting individuals, and it is virtually certain to raise § 1981 problems under existing law. The one exception is that NIH rules mandating inclusion of racial groups in some circumstances may be drawn narrowly enough to avoid constitutional difficulty.

⁴⁰⁴ National Institutes of Health, Policy and Guidelines on the Inclusion of Women and Minorities as Subjects in Clinical Research, at http://grants.nih.gov/grants/funding/women_min/guidelines_amended_10_2001.htm (last modified Oct. 11, 2001).

⁴⁰⁵ *Id.*

⁴⁰⁶ *Id.* (“If the data from prior studies strongly support no significant differences of clinical or public health importance in intervention effect based on sex/gender, racial/ethnic and/or relevant subpopulation comparisons, then sex/gender and race/ethnicity will not be required as subject selection criteria. However, the inclusion and analysis of sex/gender and/or racial/ethnic subgroups is still strongly encouraged.”).

V. NORMATIVE IMPLICATIONS OF MEDICAL RACIAL PROFILING

The uncertain legality of some forms of racial profiling in medicine naturally leads to the normative question: what ought the law allow? This is by no means a hypothetical issue for, as we showed in the previous section, some present practices could clearly be challenged, perhaps even successfully, under existing law. Furthermore, the existence of the California Racial Privacy Initiative⁴⁰⁷ and the possibility of a similar initiative in Michigan after the Michigan affirmative action cases suggest the real possibility of a state outlawing the collection of racial data in connection with medical research.

Deciding whether racial profiling *ought* to be allowed requires judgments about what to value. As we noted in Part III, race has the potential to be a valuable tool in medicine. But it is often not the only tool that can achieve the same results for race is usually just a proxy for other ultimate causes. Furthermore, the use of race carries with it special risks that we will develop in this section. Thus, in Section A we consider what uses of race, if any, should be permitted in medicine, and in Section B we discuss possible alternatives.

A. *When Race Should Be Allowed*

The actual use of race in medical research and practice has to weigh the concerns we have outlined against the possible benefits. The particular direction to which this balance will tilt depends upon the particular use of race suggested. In this section, we make a proposal as to which uses should—and should not—be allowed.

1. *Race-Focused Studies*

We do not believe that the existing law that permits race-focused studies should be changed, and we believe that legislation such as the California Racial Privacy Initiative will only serve to undermine beneficial research. A great deal of ink has been spilled in recent years in the medical and scientific literature over the propriety of race-based research. While many scholars have suggested that this research is of little or no use,⁴⁰⁸ other schol-

⁴⁰⁷ See *supra* note 360.

⁴⁰⁸ See Cooper et al., *supra* note 22 (questioning relevance); *Genes, Drugs and Race*, *supra* note 261, at 239–40; Jay S. Kaufman & Richard S. Cooper, *Commentary: Consideration for Use of Racial/Ethnic Classification in Etiologic Research*, 154 AM. J. EPIDEMIOLOGY 291 (2001) (suggesting limitation on usefulness); Schwartz, *supra* note 20; Jacqueline Stevens, *Racial Meanings and Scientific Methods: Changing Policies for NIH-Sponsored Publications Reporting Human Variation*, 28 J. HEALTH POL., POL'Y & L. 1033 (2003) (noting harmful consequences).

ars have recently supported such research.⁴⁰⁹ Furthermore, such research is commonplace, whatever the misgivings of some in the academy.⁴¹⁰

The main “scientific” objection to such research is its limited, or even negative utility. Commentators have argued that, if the object is to discover genetic causes of disease or effectiveness of treatments, it is preferable to study those genes directly.⁴¹¹ In addition, the link between race and the genetic cluster of interest is often weak.⁴¹² Furthermore, the vast majority of diseases are multifactorial, where the link between race and health outcomes is small.⁴¹³

Alternatively, some commentators are concerned that race-based research distracts researchers from other causes.⁴¹⁴ This is the concern of those who recognize possible benefits but believe that expended resources are misdirected and that the modest results will potentially be misused. The recent primacy of research into the genetic causes of disease may tempt researchers to suggest genes as the cause of differences between races in health outcomes, thereby overlooking or minimizing other potential causes, including differences in access to health care and the existence of racism.⁴¹⁵

Proponents respond that, even though race may not be an *exact* proxy for genes and/or environment, it remains a useful one for research.⁴¹⁶ Single gene disorders caused by an allele with a low frequency (<2%) in one racial group are unlikely to be found among other racial groups.⁴¹⁷ Even when the allele or alleles are found at higher frequencies (2%–20%) in one race, they can be quite rare in other races.⁴¹⁸ Use of racial data thus allows researchers to target possible causes of disease or differences in response to treatment.⁴¹⁹ Although relying instead on genetic clusters might be more accurate, such research would be more difficult (at least at the present time) because it requires additional time and resources to divide

⁴⁰⁹ Burchard et al., *supra* note 21; Howard L. McLeod, *Letter to the Editor*, 345 *NEW ENG. J. MED.* 766 (2001); Neil Risch et al., *Categorization of Humans in Biomedical Research: Genes, Race and Disease*, *GENOME BIOLOGY*, July 2002, at 1 (arguing for the usefulness of race- and ethnicity-based research); *see also* Wood, *supra* note 19 (reporting favorably on race-based studies).

⁴¹⁰ *See* Sandra Soo-Jin Lee et al., *The Meaning of “Race” in the New Genomics: Implications for Health Disparities Research*, 1 *YALE J. HEALTH POL’Y L. & ETHICS* 33, 54 (2001) (noting that, between 1910 and 1990, race was used in 64% of the articles in the *American Journal of Epidemiology*).

⁴¹¹ McLeod, *supra* note 254, at 248; Schwartz, *supra* note 20, at 1393; Stevens, *supra* note 408, at 1070; *see also* Nicholas Wade, *Race is Seen as Real Guide to Track Roots of Disease*, *N.Y. TIMES*, July 30, 2002 (noting proposal of David Goldstein).

⁴¹² *Genes, Drugs and Race*, *supra* note 261, at 239–40.

⁴¹³ *See* Cooper et al., *supra* note 22, at 1168.

⁴¹⁴ *See, e.g.*, Lee et al., *supra* note 410, at 56; Stevens, *supra* note 408, at 1070.

⁴¹⁵ *See* Cooper et al., *supra* note 22, at 1168.

⁴¹⁶ *See* Burchard et al., *supra* note 21, at 1172.

⁴¹⁷ *Id.*

⁴¹⁸ *Id.*

⁴¹⁹ *Id.* at 1174.

up the research into the appropriate clusters. Relying on self-reported race (which appears to be somewhat accurate⁴²⁰) is therefore simpler and less expensive.

Proponents also respond by noting that, whatever the flaws in the link between genes and race, a race-neutral approach might itself mask potential underlying causes for health-related differences. For instance, Neil Risch and his colleagues pose the following hypothetical: two groups are tested for the effectiveness of a drug to prevent heart attacks, and then are separated through a genotype cluster analysis. Suppose the data indicates the first cluster responded strongly to the drug, but the second cluster did not. The natural inference would be a genetic difference between the first and second clusters. But, in the real world, if the first cluster was primarily whites, and the second cluster were primarily African Americans, we would instantly suspect other possible causes: environmental or behavioral differences. By removing race, and focusing instead on just genetic clusters, we lose possible clues to differences.⁴²¹

In addition, pure racial blindness would lead to research that heavily favors whites, who remain the largest racial group in the United States. To the extent that, for at least some diseases, genetic causes are real, race-neutral studies would tend to uncover only the diseases of whites, while ignoring those of other groups.⁴²² This might be particularly harmful for investigations into pharmacogenetics, where differences in responsiveness to treatment may result from alleles that are quite rare in the white population and could be completely absent in a race-neutral sample.

In sum, while the merits of such research remain hotly debated in the medical and scientific community, there is a place for race-based scientific research, at least for now. While race may be over-used and tools such as genetic clustering will replace race,⁴²³ we have not yet reached the point where we can conclude that such research has no scientific utility.

Some might still contend that race-based research ought to be forbidden. A concern that transcends the scientific debate over such research is the expressive effects we discussed above.⁴²⁴ In addition, research funds might be spent addressing other problems.⁴²⁵ Despite potential harms, though, we believe that the potential benefit from such research is sufficient that an outright prohibition is not yet warranted. In particular, we are

⁴²⁰ See *id.* at 1173–74. “Accuracy,” of course, merely means that the individual’s genetic heritage is from ancestors who predominately came from a particular geographic location; it does not suggest genetic “purity.”

⁴²¹ See Risch et al., *supra* note 174, at 7–9.

⁴²² See Burchard et al., *supra* note 21, at 1174; Risch et al., *supra* note 174, at 11. This phenomenon might explain the comparative ineffectiveness of cystic fibrosis screening for African Americans and Hispanics.

⁴²³ See James F. Wilson et al., *Population Genetic Structure of Variable Drug Response*, 29 NATURE GENETICS 268 (2001).

⁴²⁴ See *supra* notes 43–48, 349 and accompanying text.

⁴²⁵ Stevens, *supra* note 408, at 1070.

concerned that outright prohibitions on race-based research will hinder efforts to uncover the causes of diseases—both genetic and environmental—that harm racial minorities and will only perpetuate the current focus on the diseases of whites. Indeed, even the proponents of the California Racial Privacy Initiative, who appeared to otherwise oppose just about any use of race, attempted to carve out an exception for medical research.⁴²⁶ Until it becomes clear that other methods of researching the true causes of disease are superior, it would be unwise to limit the potential for scientific advancement, particularly when racial-blindness may actually result in more harm to minorities.

2. Race-Based Outreach

In discussing race-based outreach, it is important to bear in mind the distinction between race-limited and race-targeted outreach. Race-limited outreach (that which is designed to reach only the members of a particular race) in general is of dubious value. Such actions presuppose that one group is significantly more likely to be aided by such outreach than others. Outside a few single gene disorders, this is unlikely to be true. Instead, for most ailments (even genetic ones),⁴²⁷ and particularly chronic ailments, there will be only small variations between groups in the prevalence of particular diseases.⁴²⁸ In targeting scarce public health resources, there would seem to be little reason to limit educational efforts on, say, hypertension at one particular race.

Even for a classically race-linked disease such as sickle cell anemia this may be true. While African Americans are far more likely to carry the sickle cell trait than American whites,⁴²⁹ other groups are also at risk for the disease. For instance, the trait is present at elevated frequencies both in the Arabian peninsula and the Indian subcontinent.⁴³⁰ Thus, two population subgroups—one white, the other Asian—are at risk for the disease

⁴²⁶ See Burchard et al., *supra* note 21, at 1174.

⁴²⁷ The number of ailments that can be satisfactorily explained as single gene disorders is now diminishing. See Jose L. Badano & Nicholas Katsanis, *Beyond Mendel: An Evolving View of Human Genetic Disease Transmission*, 3 NATURE REVIEWS GENETICS 779 (2002).

⁴²⁸ WYNBRANDT & LUDMAN, *supra* note 236, at 349–66 app.1.

⁴²⁹ As we have noted, *supra* note 230, the carrier rate in African Americans is 9%, while in white Americans, the carrier rate is 0.7%. If we assume endogamy, perfect randomness in reproductive partners and equal viability of fetuses, approximately 1:500 of African American children will be born with sickle cell, whereas only 1:100,000 white American children will have the disease; in other words, African Americans are 200 times as likely to be afflicted. Nonetheless, there is a risk of the disease in white American children, a risk even higher among children of mixed parents.

⁴³⁰ The exact rates are unknown. The sickle cell allele is widespread in the Middle East and India. Fred W. Lorey et al., *Distribution of Hemoglobinopathy Variants by Ethnicity in Multiethnic State*, 13 GENETIC EPIDEMIOLOGY 501, 502 (1996). Recent screening in California, though, revealed that people of Middle Eastern and Indian subcontinent ancestry showed fewer than expected occurrences of sickle cell disease. *Id.* at 504 tbl.1.

and would be overlooked if public health officials were to use a race approach and target only African Americans for outreach. Increased intermarriage between racial groups also increases the need for across-the-board outreach on sickle cell anemia,⁴³¹ and almost all states now properly require screening of newborns.⁴³² The same is true for cystic fibrosis: although whites are at far higher risk for having a child with the disease,⁴³³ there is a non-trivial risk among other groups and outreach limited to whites would only contribute to further tragedies like the one recounted by Doctor Garcia.⁴³⁴

The negative expressive effects of race-limited outreach are likely to be substantial as well. Outreach, by its very nature, requires communication, and an inevitable part of the message of race-limited outreach is that this particular race is different. Given our history of the short step from racial differences to (white) racial superiority, government communication that races are different will reinforce messages that white groups are superior. This is a particular concern with health care information, we believe, because much of the information likely to be racially targeted will involve the occurrence of disease. For example, governmental actors may wish to inform African Americans about an increased risk for hypertension. In so doing, the government is (perhaps unintentionally) buttressing views that African Americans are inferior to whites, either as a result of genes or culture.⁴³⁵ In the absence of a stronger link between race and a particular disease, there is no reason to risk what may be termed an expressive backlash.

For some particularly rare disorders, where there are stark differences in the rates of disease occurrence, such as maple syrup urine disease and Tay-Sachs, limited outreach does make sense, but at the population group level, not at the racial level. As we noted in Parts II and III, such stark differences will likely only occur in special circumstances; particularly in “founder’s effect” situations where there is little or no gene flow through exogamy to offset genetic drift. As a result, these diseases are usually found among relatively small groups, and outreach that is confined to Old Order Mennonite or Ashkenazi Jewish groups, for example, would appear justified. Requiring governmental actors to educate the population at large

⁴³¹ See *id.* at 508–10. Intermarriage rates have been steadily increasing for the last twenty years. According to the United States Census Bureau, in 1980 there were 651,000 interracial married couples (excluding interracial couples where neither partner was white or African American) out of a total of 49,714,000, or 1.3%. By 2002, there were 1,674,000 interracial couples out of 57,919,000 married couples, or 2.9%. Put another way, the total number of interracial couples had increased by 257%, whereas the total number of married couples had increased only by 16.5%. U.S. Census Bureau, U.S. Dep’t of Commerce, *Interracial Married Couples: 1980 to Present*, Table MS-3 (June 12, 2003), at <http://www.census.gov/population/socdemo/hh-fam/tabMS-3.pdf>.

⁴³² See *infra* text accompanying notes 442–444.

⁴³³ See discussion *supra* note 236.

⁴³⁴ See *supra* text accompanying note 60.

⁴³⁵ See Cooper et al., *supra* note 22, at 1168.

about such disorders would risk diluting the message for the groups that need to be reached. Given prior statements by the Supreme Court noted in Part IV, it is likely that such actions, provided that there is a sufficient correlation between the group and the disease, would be permitted.⁴³⁶ Oddly, what should not be permitted is the very example of such race-limited outreach that the Court supported: limited outreach on sickle cell anemia.

Race-*targeted* outreach makes more sense. Outreach necessarily acts through various social channels: media outlets, community organizations and religious institutions, to name a few. Most of these will not be effective at reaching all races. Religious affiliation remains highly segregated.⁴³⁷ Community groups, because they usually draw membership from the neighborhood, no doubt reflect the residential segregation that continues to plague America.⁴³⁸ Even media outlets often are targeted at particular racial audiences.⁴³⁹ If government actors were to attempt to engage in outreach only through mainstream outlets, they would risk large segments of the American public remaining oblivious to their message.

Furthermore, outreach efforts channeled through media outlets aimed at minority racial groups may help overcome the distrust many minorities have of the health care system. This may be particularly true for immigrant groups, who may be especially cut-off from mainstream outlets. By addressing them in their own language and through their own cultural institutions, the government may be much more effective at getting health-related information to these groups.

Of course, race-targeted outreach also has expressive effects. Although it does not imply that there are racial differences in health outcomes, it still sends the message that racial groups remain segregated from one another. The risk is that the government, by engaging in race-targeted outreach, is seen as supportive of such segregation. While we recognize this risk, it is less problematic than the expressive effects of race-limited outreach.

The discussion thus far has been limited to governmental actors. Private actors remain mostly beyond the reach of the law, given the restraints of the First Amendment on the ability of government to restrict speech by private groups.⁴⁴⁰ Although our remarks about the utility of race-based outreach apply equally to governmental and private actors, limiting private actors is simply not permissible under current views of the

⁴³⁶ See *supra* text accompanying note 308.

⁴³⁷ In part this is because religious affiliation is one of the traits of personality that is most strongly controlled by the parental environment. See STEVEN PINKER, *THE BLANK SLATE: THE MODERN DENIAL OF HUMAN NATURE* 375 (2002); RIDLEY, *NATURE VIA NURTURE*, *supra* note 102, at 88.

⁴³⁸ Michelle Adams, *Separate and [Un]Equal: Housing Choice, Mobility and Equality in the Federally Subsidized Housing Program*, 71 TUL. L. REV. 413, 424–28 (1996).

⁴³⁹ The most visible example of racially targeted media is Black Entertainment Television (BET), but many other such outlets exist.

⁴⁴⁰ See *supra* text accompanying note 329.

First Amendment. Indeed, the Supreme Court's most recent decision at the intersection of discrimination and the First Amendment made clear that the right of expressive association trumped the right to be free of discrimination.⁴⁴¹

3. Race-Based Screening

Race-based screening is also problematic. In its most explicit form, mandated race-based screening has become rare. In the past, for instance, many states mandated sickle cell anemia screening only for African Americans.⁴⁴² Some such statutes remain on the books. New York law, for instance, mandates sickle cell screening for all marriage license applicants "not of the Caucasian, Indian or Oriental race," but it appears that the statute is no longer enforced.⁴⁴³ Racial screening, we believe, nonetheless still occurs. For instance, when one of our research assistants inquired into state-sponsored screening for sickle cell anemia in New Hampshire,⁴⁴⁴ he was told that such screening was performed when the individual was at "high risk." When he pressed further and asked if this meant that persons were selected based on racial or ethnic background, he was told that it was primarily a matter of physician decision-making. While we cannot be sure exactly how such decisions were being made in New Hampshire during the summer of 2003, we strongly suspect that the decision on whether to screen is racially based.

This vignette illustrates two interesting phenomena about such screening: first, many in the medical profession feel it can be justified, and second, they are not sure it is socially and/or legally acceptable. The result has been to limit racially based screening, at least in any publicly visible way. For instance, despite our New Hampshire example, almost all newborn screening these days is performed without any reference to race,⁴⁴⁵ in part because of the declining costs of such activities. Thus, some states now test for a much larger number of disorders, including phenylketonuria, congenital hypothyroidism, galactosemia, maple syrup urine disease,

⁴⁴¹ *Boy Scouts of Am. v. Dale*, 530 U.S. 640 (2001). Although *Dale* involved the right to be free of discrimination on the basis of sexual orientation, not race, the opinion would not justify any distinction. While some private outreach might be subject to lesser protection under the commercial speech doctrine, *see supra* text accompanying notes 333–334, most outreach would seem protected even under the commercial speech doctrine.

⁴⁴² N.Y. STATE TASK FORCE, *supra* note 372, at 114.

⁴⁴³ N.Y. DOM. REL. LAW § 13-aa (Consol. 2003); *see also* N.Y. STATE TASK FORCE, *supra* note 372, at 116 (suggesting that law is not enforced). Under some other circumstances, New York now requires universal screening for sickle cell anemia. *See* N.Y. EDUC. LAW § 903 (McKinney 2003).

⁴⁴⁴ New Hampshire was called because it is one of a few states that screen for sickle cell but does not presently screen all newborns.

⁴⁴⁵ Wertz, *supra* note 442.

homocystinuria, biotinidase, and congenital adrenal hyperplasia, along with sickle cell anemia.⁴⁴⁶

Even in an environment of low costs, such testing still requires resources. California, for instance, recently ended a pilot project for expanded newborn screening, which had cost \$3.9 million over eighteen months.⁴⁴⁷ With limited health care resources, a cost-benefit analysis might suggest limiting screening to a particular group. Consider the following hypothetical. Assume a disease present in one of every 500 white infants, but only in one of every 100,000 African American infants. Assume also that testing costs \$50, and that early detection of the disease can save \$50,000 in “costs,” which could consist only of medical expenses, or could also include other costs, such as the patient’s pain and suffering and the emotional toll on family members. Testing 500 white infants, at a total cost of \$25,000, would be justified by the saving of \$50,000 in medical costs. Testing African American infants, though, is not justified, because the costs of \$5,000,000 to save \$50,000 would seem imprudent. Under this analysis, racially based newborn screening may be justifiable.

There are (at least) two problems with this sort of simplified cost-benefit analysis. First, it assumes that it is possible to quantify in any meaningful way the benefits of screening. Unless we limit costs simply to medical savings, deciding whether to test for any given disorder would turn on how much, if any, savings could be achieved through early detection. In many cases, detection actually will *increase* medical costs, because it sets the patient on a course of expensive treatment for the remainder of his or her life (recall the special diets for PKU and maple syrup urine disease patients).⁴⁴⁸ Thus, decisions about screening turn not just on medical savings, but on the other benefits from having a sure diagnosis of a disease, and quantifying these benefits is problematic.

Even if such quantification were possible, a second problem renders limiting screening unwise. For instance, assume that in the case of our hypothetical disease, the actual medical costs savings were zero. A decision to screen white Americans, but not African Americans, might be justified on the notion that the psychological benefits are, say, \$1,000,000. But viewed from the perspective of the individual, the “message” might be much simpler: society values (and protects against) the suffering of white Americans, but not that of African Americans. The expressive costs of such a decision appear to us to exceed any benefits.

⁴⁴⁶ Nat’l Newborn Screening & Genetics Resource Ctr., National Newborn Screening Status, at <http://genes-r-us.uthscsa.edu/resources/newborn/screenstatus.htm> (last modified Feb. 26, 2004).

⁴⁴⁷ State of California Department of Health Services (May 22, 2003) (letter from George Cunningham, Chief of Genetic Disease Branch to Pediatric Care Providers), available at <http://www.dhs.cahwnet.gov/pcf/h/GDB/html/NBS/Pediatricians.pdf>.

⁴⁴⁸ See Chuang & Shih, *supra* note 220; Wappner et al., *supra* note 217.

On a related note, as with race-limited outreach, race-based screening has real expressive costs.⁴⁴⁹ In particular, it reinforces the notion that there are important biological differences between racial groups. This, we believe, is also a sufficient concern to counsel against routine race-based screening of individuals. Particularly as the costs of screening decline, there is little reason to limit access based on race alone.⁴⁵⁰

4. Race-Based Treatment

We see little reason to relax the limitations on race-based treatment. Once a patient has demonstrated the symptoms of a particular disorder, there would seem to be, as of this date, little reason to alter care based solely upon the individual's race. As we have noted, though, such decisions seem to be commonplace.

To echo themes we have sounded before, the variations between races are *generally* insufficient to justify differential treatment and, in this particular case, there may soon be technology that makes such distinctions unnecessary. The most obvious reason for differential treatment would be differences in sensitivity to drugs. But as we noted in Part III, in most cases the differences appear to be too small to justify differential treatment regimes. For instance, we discussed the *CYP2C9*1* allele, which is present in almost all persons of Asian descent, but "only" about 80% of whites.⁴⁵¹ Although white patients are at an increased risk (perhaps ten times as great) of having lessened ability to metabolize various drugs, access to drugs based on race should not be restricted, because most patients of any race will have normal functioning. Instead, practitioners ought to be aware of the possibility of reduced metabolism in patients of all races.

Even more dramatic examples appear, on closer examination, to be of limited clinical relevance. For instance, one study found that therapy with the drug enalapril was associated with a lower risk of hospitalization, and lower diastolic and systolic blood pressure in white patients than in African American patients.⁴⁵² While such results suggest that

⁴⁴⁹ See *supra* notes 42–48 and accompanying text.

⁴⁵⁰ The contrary argument is that there is little expressive harm because few individuals will focus on the race aspects of mandated screening for newborns. The mystique of medicine, and the arcane nature of much medical testing, arguably cut against any robust "message" from limiting certain tests to particular racial groups. On the other hand, the New Hampshire response, see *supra* text accompanying note 444, and even the New York statute's phraseology (excluding groups, among them "Orientals," rather than including African Americans), see *supra* text accompanying note 443, suggest that state authorities are aware of the possible expressive import of even relatively "technical" decisions, even if the "solution" is a clumsy one that is offensive to, at least, Asians.

⁴⁵¹ See *supra* text accompanying note 259.

⁴⁵² Derek V. Exner et al., *Lesser Response to Angiotensin-Converting Enzyme Inhibitor Therapy in Black as Compared with White Patients with Left Ventricular Dysfunction*, 344 NEW ENG. J. MED. 1351 (2001).

doctors should treat white, but not African American, patients with enalapril, it overlooks two important facts. First, enalapril therapy in both white and African Americans failed to show any significant reduction in the risk of death: in white patients, thirty percent died within four years regardless, and in African American patients forty percent died over the same time period.⁴⁵³ Second, these are statistical comparisons, which ignore the possibility that specific African American patients were aided by enalapril therapy.

Of course, it would be most useful to know why these differences exist, isolating the causes, and then prescribing (or not prescribing) enalapril based on this information. The pursuit of such information is one reason that race-based studies should continue. But what occurs in actual clinical practice? In the long-term, once the underlying biochemical causes are understood, physicians should be able to tailor drug treatments to the patient. In the interim, many will be tempted to rely instead on the limited information available: race. We counsel against much, if any, reliance.

Even if significant enough differences in treatment outcomes between members of racial groups can be documented, they may over-encourage the use of race in medicine. Such fears are not misplaced. Based on anecdotal information, physicians appear to routinely prescribe pharmaceuticals in different doses for African American, white and other patients.⁴⁵⁴ Clinicians justify such decisions by stating that African Americans are poorer metabolizers of drugs than other groups.⁴⁵⁵ The actual evidence, though, is far more limited. Studies have shown, for instance, that there *may* be a higher incident of individuals in African American populations who are poor metabolizers of drugs metabolized by the enzyme CYP2D6.⁴⁵⁶ But the overall number of poor metabolizers is still a minority in both groups (no more than 10% of whites and no more than 19% of African Americans). Furthermore, classification of individuals can mask important differences. For instance, while poor metabolizers are far less common in Asian than white populations (about 1% in Asians), white extensive metabolizers (that is, individuals who are generally normal) appear to clear at least some drugs faster on average than Asians.⁴⁵⁷ Clinical use of this data is complicated: white patients are at greater risk of extremely slow metabolization, while Asians are at a higher risk of normal but slower metabolization. Furthermore, as with CYP2C9, there is significant within-

⁴⁵³ See *id.* at 1355 fig.1. An earlier study suggested that white patients with a history of hypertension did show a reduction in mortality. See Peter Carson et al., *Racial Differences in Response to Therapy for Heart Failure: Analysis of the Vasodilator-Heart Failure Trials*, 5 J. CARDIAC FAILURE 178 (1999).

⁴⁵⁴ See, e.g., Satel, *supra* note 2.

⁴⁵⁵ See *id.*

⁴⁵⁶ See Xie et al., *supra* note 258, at 815, 820–23.

⁴⁵⁷ See *id.* at 821. African American extensive metabolizers also appear to be, on average, slower metabolizers than whites. See Emile D. Risby, *Ethnic Considerations in the Pharmacotherapy of Mood Disorders*, 32 PSYCHOPHARMACOLOGY BULL. 231, 231 (1996).

race variation for metabolization through CYP2D6; for example, while Korean and Japanese extensive metabolizers appear to be quite similar, Chinese extensive metabolizers are slower on average than those two groups, probably due to a higher incidence of the *CYP2D6*10* allele.⁴⁵⁸

These data counsel for far more circumspect use than is likely in the clinical setting where seat-of-the-pants judgments are common—and perhaps inevitable. Simplistic decisions, such routinely under- or over-prescribing drugs for races, can ignore individual variants. In the area of drug treatment, other alternatives may soon be available. Instead of relying on race, some researchers have suggested relying on genetic clusterings of SNPs.⁴⁵⁹ And in the long-term, DNA chips, which could test for the existence of particular alleles (and therefore make predictions about metabolization rates), would be a far more accurate method for deciding appropriate medication for individuals.

In the very short term, some use of race may be inevitable, as alternative measures are not available. But it should be used sparingly and *only* where there is clear evidence that race is the best predictor of treatment success. So, rather than simply make such use illegal, we suggest judicial recognition of a defense in cases of racial treatment, one akin to the bona fide occupational qualification (BFOQ) test used in Title VII. Although such a defense is not in the statutory language of § 1981, nor is it applicable to racial discrimination under Title VII,⁴⁶⁰ our defense, which we will refer to as a BFTR (bona fide treatment rationale) would permit health care practitioners to take race into account in treatment in very limited circumstances.

Under current law, BFOQs can be established in one of two circumstances: first, where substantially all the members of a group possess a characteristic that prevents them from working safely or effectively in a job, and second, where only some members of a group possess such a characteristic but it is impossible to make individual determinations.⁴⁶¹ In the employment context, the first test might be satisfied where, say, privacy was at issue and no individual of the opposite sex could be placed in a particular position without compromising the privacy of the clientele.⁴⁶²

⁴⁵⁸ See Xie et al., *supra* note 258, at 821–23.

⁴⁵⁹ See McLeod, *supra* note 254, at 248.

⁴⁶⁰ See *supra* text accompanying note 323.

⁴⁶¹ See SULLIVAN ET AL., *supra* note 378, § 3.05.

⁴⁶² See EEOC v. Mercy Health Ctr., 29 F.E.P. 159 (W.D. Okla. 1982); Backus v. Baptist Med. Ctr., 510 F. Supp. 1191 (E.D. Ark. 1981), *vacated as moot*, 671 F.2d 1100 (8th Cir. 1982); Fesel v. Masonic Home of Delaware, 447 F. Supp. 1346, 1354 (D. Del. 1978), *aff'd*, 591 F.2d 1334 (3rd Cir. 1979). See generally Jillian B. Berman, Comment, *Defining the "Essence of Business": An Analysis of Title VII's Privacy BFOQ after Johnson Controls*, 67 U. CHI. L. REV. 749 (2000); Carolyn S. Bratt, *Privacy and the Sex BFOQ: An Immodest Proposal*, 48 ALB. L. REV. 923 (1984); Deborah A. Calloway, *Equal Employment and Third Party Privacy Interests: An Analytical Framework for Reconciling Competing Rights*, 54 FORDHAM L. REV. 327 (1985); Amy Kapczynski, Note, *Same-Sex Privacy and the Limits of Antidiscrimination Law*, 112 YALE L.J. 1257 (2003); Sharon M. McGowan, *The Bona*

The second test is more likely to be applicable, say, where safety is concerned. In several cases, the courts have upheld limitations on how long flight attendants may serve during their pregnancies. Given the duties such workers have during emergencies, it would be impractical to determine on an individual basis the point at which a particular woman would no longer be able to perform the functions of her job.⁴⁶³ The first scenario we believe will almost never exist in health care; that is, there will almost never be cases in which all members of a race have a particular trait. It is the second scenario where we would apply the defense: health professionals can take race into account only where there is a (scientifically demonstrated) substantial treatment responsiveness for members of different races *and* there is no better way of determining responsiveness.

The BFTR approach would have three benefits. First, the use of race in treatment would be presumptively illegal, and the BFTR would be an affirmative defense; this would signal that the legitimate uses of race are rare, and should minimize any negative expressive effects. Second, it would ensure that treatments should differ only when there is substantial scientific basis for believing there is differential response. Third, because race has to be the *best available method*, as new technologies and approaches become available in the near future, the use of race will be eliminated.

The notion of “best available method” should take into account not only the state of scientific knowledge at the time but also speed, efficacy, and efficiency. For example, a doctor would be permitted to use race as a diagnosis and treatment proxy both when race is the best indicator and also when the constraints of time and resources make race the best available alternative, if not the best theoretical alternative. Conversely, the test would cut against governments, managed care organizations, or other institutions justifying race-limited screening, reimbursement, or treatment as a means of allocating resources—“available” in this context means more than resource-allocation.⁴⁶⁴ Like all tests, the parameters of the BFTR would have to be worked out on a case-by-case basis, but it represents the best approach to this complicated and sensitive area of medical practice.

Fide Body: Title VII's Last Bastion of Intentional Sex Discrimination, 12 COLUM. J. GENDER & L. 77, (2003).

⁴⁶³ *Levin v. Delta Airlines*, 730 F.2d 994, 998 (5th Cir. 1984) (upholding as BFOQ an airline policy requiring flight attendants to take involuntary leave 24 hours after discovering pregnancy); *Harriss v. Pan Am. World Airlines, Inc.*, 649 F.2d 670 (9th Cir. 1980) (same).

⁴⁶⁴ In the sex BFOQ context under Title VII, the Supreme Court rejected an employer's attempt to justify restricting women from numerous jobs in battery manufacturing because of the dangers to potential fetuses. *Int'l Union v. Johnson Controls, Inc.*, 499 U.S. 187 (1991). The policy in question assumed all women were capable of being pregnant unless they medically demonstrated the contrary. The Court was skeptical of the defendant's justification that such a policy was necessary to avoid potentially heavy liability to the offspring of the women but did suggest that proof of ruinous liability might be a defense: “We, of course, are not presented with, nor do we decide, a case in which costs would be so prohibitive as to threaten the survival of the employer's business.” *Id.* at 210–11.

5. Race-Based Clinical Trials

Clinical trials lie at the intersection of treatment and research. Unlike other treatment decisions, though, race-based exclusions eliminate persons from possible participation in the absence of evidence of racial distinctions. Indeed, in some cases the point of a race-based clinical trial would be to demonstrate such differences. Furthermore, unlike pure research, race-based clinical trials cannot be justified solely as increasing our knowledge, and lending valuable clues to disease causes and the source of treatment response variations. Instead, race-based clinical trials lead to denials of potentially life-saving treatment to individuals on the basis of race.

Complete exclusion of racial groups from trials based on race has little merit. For instance, focusing a trial on one group only does not appear likely to give us much information about the underlying causes of differences in treatment regimens. Thus, trials such as the African American Heart Failure Trial (A-HeFT) to confirm the efficacy of BiDil in the treatment of African American heart failure patients seem unwise.⁴⁶⁵ While there may be preliminary data suggesting that BiDil is more effective in African Americans than whites,⁴⁶⁶ the reasons for these differences are unclear. One report suggests that African Americans may “have a greater deficiency of nitric oxide generation that is restored by” BiDil.⁴⁶⁷ Assuming such a biochemical pathway is the reason, a number of white patients, as well as Asian patients, may share the deficiency that BiDil restores.⁴⁶⁸ Simply because, on average, such deficiencies are less frequent among white (or perhaps Asian) patients does not mean they do not exist. Instead, what we have shown repeatedly throughout this Article is that they almost assuredly *do* exist, just in lower frequencies.

In being skeptical of race-exclusionary studies, we do not mean to deny the importance of efforts to include races. Attempts to broaden the groups included in clinical trials seem wise, particularly given the ten-

⁴⁶⁵ Cf. Stolberg, *supra* note 3. The exclusion of non-minorities from BiDil trials would be especially ironic in light of the history of BiDil’s development. Jonathan D. Kahn argues that BiDil is an “ethnic drug” today because of the interventions of law and commerce rather than because of any biomedical considerations; he concludes that the story implicates federal agencies in inappropriately giving the state’s imprimatur to using race as a biological category. Jonathan Kahn, *How a Drug Becomes “Ethnic”: Law, Commerce, and the Production of Racial Categories in Medicine*, 4 YALE J.HEALTH POL’Y, L. & ETHICS 1 (2004). He traces the development from “a drug for everyone, with no ethnic marketing,” *id.* at 4, to its present focus on African Americans as stemming from a re-analysis of the trial data after the FDA had disapproved its use in the general population. *Id.* at 15–16.

⁴⁶⁶ See Peter Carson et al., *Racial Differences in Response to Therapy for Heart Failure: Analysis of the Vasodilator-Heart Failure Trials*, 5 J. CARDIAC FAILURE 178 (1999).

⁴⁶⁷ *Id.* at 186.

⁴⁶⁸ See Kahn, *supra* note 9, at 481.

gency of some rare alleles to appear only in one particular race.⁴⁶⁹ Studies that are overly reliant on white populations are likely to miss the effects generally absent in those populations but present in other racial groups. Therefore, while we see no reason to exclude race-based clinical trials from existing anti-discrimination laws, we do believe that efforts to *include* races should be protected.

B. The Alternatives to Race

Because race is never the actual cause of racial disparities in health, there are alternative ways to attack those root causes. When the root cause is genes, genetic testing can be used. When the root cause is environmental, medical professionals can work harder to discover environmental causes of disease. Both of these alternatives are necessarily more costly than simply relying on race—particularly the genetic testing alternative, where some tests are still very expensive—but, in addition to avoiding many of the harms of using race, such approaches seem likely to yield more accurate results.

1. Genetic Testing

If the cause of a disease is genetic, the most direct way to test for the disease is simply to test for the presence of the particular allele in the patient's genome. Genetic testing is available for a wide range of disorders and has become common-place in health care.⁴⁷⁰ Not only do all states require screening of newborns for at least some genetic diseases⁴⁷¹ but parents are also routinely offered additional genetic screening at a price.⁴⁷²

There are several limits on genetic testing. First, it is subject to possible false negatives—inaccurate results indicating that the patient does not

⁴⁶⁹ See Burchard et al., *supra* note 21, at 1172.

⁴⁷⁰ N.Y. STATE TASK FORCE, *supra* note 372.

⁴⁷¹ See Muin J. Khoury et al., *Population Screening in the Age of Genomic Medicine*, 348 *NEW ENG. J. MED.* 50 (2003); NATIONAL NEWBORN SCREENING AND GENETICS RESOURCE CENTER, U.S. NATIONAL SCREENING STATUS REPORT, available at <http://genes-r-us.uthscsa.edu/resources/newborn/screenstatus.htm> (last visited July 18, 2003). See *supra* notes 442–444 and accompanying text.

⁴⁷² See Amy Dockser Marcus, *Anxious Parents Pay Labs for Extra Tests on Newborns*, *WALL ST. J.*, Nov. 20, 2002, at D1. Genetic testing can take a number of forms. The most direct method is DNA-based testing, which determines the presence of specific alleles of genes. See N.Y. STATE TASK FORCE, *supra* note 372, at 32–36; see also LORI B. ANDREWS ET AL., *GENETICS: ETHICS, LAW & POLICY* 222–24 (2002). Several different forms of DNA testing exist. The most accurate is DNA sequence analysis, but other variants include PCR analysis and Southern blotting. ANDREWS ET AL., *supra*, at 222–24; N.Y. STATE TASK FORCE, *supra* note 372, at 32–36. In addition to DNA-based testing, investigators can use alternative approaches, including RNA analysis, karyotype analysis, and protein analysis. See N.Y. STATE TASK FORCE, *supra* note 372, at 37–40. These tests rely on either detecting the products of genes (RNA and protein) or counting chromosomes (karyotype testing for Down's Syndrome).

have the allele for the disease. Such results are most likely to occur where there are several disease-causing alleles for the same gene. For instance, there are literally hundreds of different alleles of the cystic fibrosis gene.⁴⁷³ A test that picks up only the most common disease-causing allele would return a false negative for any individual carrying one of the other alleles.⁴⁷⁴

Genetic testing also works best when focused on single-gene disorders for which there are no other causes. Many genetic diseases are multifactorial: they are caused not by the malfunction of one gene but many, often in connection with the environment.⁴⁷⁵ Many cancers, for instance, are believed to be caused not by a single mutation, but by a series of mutations.⁴⁷⁶ Similarly, researchers have suggested that schizophrenia is the result of at least two genetic faults, in connection with an outside trigger, and that there may even be other genes that can minimize or eliminate the disease.⁴⁷⁷ Genetic testing for such multifactorial diseases is of less value because it can give rise to both false positives and false negatives. False positives arise when a test detects the existence of a particular susceptibility gene but cannot detect the existence of other necessary prerequisites for disease occurrence, either genetic or environmental. False negatives can result when the disease develops even in the absence of the susceptibility gene. For instance, testing for the breast cancer susceptibility genes, *BRCA1* and *BRCA2*, could easily lead to false negatives because many women develop the disease in the absence of these genes.⁴⁷⁸

Historically, one limit to genetic testing has been its cost and the length of time needed to obtain results.⁴⁷⁹ In recent years, however, both the time for and the costs associated with genetic testing have declined rapidly.⁴⁸⁰ Today, routine neonatal screening by private companies can cost between \$25 and \$50.⁴⁸¹ In the future, the speed and cost of such tests should improve, as new technologies become more prevalent, particularly

⁴⁷³ For instance, one recent article focused on the distribution of over 270 different alleles for the cystic fibrosis gene. See Xavier Estivill et al., *Geographic Distribution and Regional Origin of 272 Cystic Fibrosis Mutations in European Populations*, 10 HUM. MUTATION 135 (1997).

⁴⁷⁴ See N.Y. STATE TASK FORCE, *supra* note 372, at 42.

⁴⁷⁵ See Cooper et al., *supra* note 22, at 1168; WYNBRANDT & LUDMAN, *supra* note 236, at 143.

⁴⁷⁶ *Neoplasia*, in PATHOLOGIC BASIS OF DISEASE, *supra* note 210, at 260, 297.

⁴⁷⁷ RIDLEY, NATURE VIA NURTURE, *supra* note 102, at 121. Researchers have also recently made similar suggestions about depression. Avshalom Caspi et al., *Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene*, 301 SCIENCE 386 (2003).

⁴⁷⁸ See Stevens, *supra* note 408, at 1043 (noting that many women develop breast cancer from nonhereditary causes).

⁴⁷⁹ See ANDREWS ET AL., *supra* note 472, at 348 (noting that costs for a test can range over \$1,000). For instance, in 1989, the costs for a test for cystic fibrosis were over \$1,000. David Stipp, *Cystic Fibrosis Test May Provide Boost For Emerging Genetic Testing Industry*, WALL ST. J., Sept. 19, 1989, at B6.

⁴⁸⁰ See Laurent Belsie, *Genetic Research Data Will Double Annually*, CHRISTIAN SCI. MONITOR, July 30, 1998, at B4 (documenting decline in costs).

⁴⁸¹ See Marcus, *supra* note 472, at D1.

microarrays or DNA chips and tandem mass spectrometry, which allow for testing of hundreds or even thousands of alleles at one time.⁴⁸² As such testing becomes widely available, the need to rely on race to predict genetic factors will cease.

At present, though, despite declining costs, such testing can be expensive, and insurance coverage is uneven.⁴⁸³ In addition, such testing does not always have benefits. For instance, some have argued that newborn screening for cystic fibrosis does not improve a child's treatment.⁴⁸⁴ As a result, in at least some cases, genetic testing may not make sense, at least in cost-benefit terms.

Despite these limits, genetic testing would generally seem to be a far more accurate predictor of many diseases than the blind use of race. Certainly in the case of classic single-gene disorders, modern genetic testing is more likely to lead to accurate diagnoses. Indeed, in the case of a disease such as cystic fibrosis, where the course of the disease differs depending upon the particular allele involved, genetic testing has the potential to lead to even more accurate treatment.⁴⁸⁵ Even in the case of multifactorial diseases, the information given by genetic testing—the presence or absence of a particular allele—is likely to be more useful in prevention, diagnosis and treatment than an assumption of susceptibility (or non-susceptibility) based on the apparent race of the patient.

2. *Uncovering Patient Histories*

One of the first things that first-year medical students learn is to take a history of their patient. Such histories are an essential part of medical

⁴⁸² See N.Y. STATE TASK FORCE, *supra* note 372, at 164; Maggie Jones, *No Surprises: The Genetic Report Card That Will Tell You If Your Embryo Will Get Prostate Cancer*, N.Y. TIMES, June 11, 2000, § 6 (Magazine), at 80 (discussing DNA chips); see also Emanuel F. Petricola III et al., *Medical Applications of Microarray Technologies: A Regulatory Science Perspective*, 32 NATURE GENETICS 474 (2002).

⁴⁸³ See N.Y. STATE TASK FORCE, *supra* note 372, at 378–81; Michelle Andrews, *Genetic Tests Abound; Why Won't Insurers Pay?*, N.Y. TIMES, May 19, 2002, § 3, at 9 (noting high costs of colon and breast cancer tests); Christine Gorman, *Katie's Crusade*, TIME, Mar. 13, 2000, at 70 (noting cost of \$250 for a test for colon cancer); Christine Gorman, *Radical Surgery*, TIME, Jan. 25, 1999, at 83 (noting high costs of test for *BRCA1* and *BRCA2*).

⁴⁸⁴ See N.Y. STATE TASK FORCE, *supra* note 372, at 144, 159. A recent article reported that Aetna will cover genetic testing only when the test result can be used to prevent or treat a disease. David Wessel, *Wanted: Public Policies to Help Genetic Testing Fulfill Its Promise*, WALL ST. J., June 19, 2003, at A2.

⁴⁸⁵ However, there is, even here, a subtle effect from race. Marketing material from one laboratory, Quest Diagnostics, suggests that its cystic fibrosis screening test is far more accurate for non-Hispanic whites (88% of alleles found) than for African Americans (69%) and Hispanics (57%). See QUEST DIAGNOSTICS, HUMAN GENETICS: CYSTIC FIBROSIS CARRIER SCREENING (2002). To the extent that genetic tests continue to be designed to pick out only alleles common among whites, they will be less useful to members of other races, and practitioners will need to use race to interpret their results. This suggests the need to design tests to identify all alleles, not just those common among whites.

practice.⁴⁸⁶ In the course of taking the history, the doctor obtains such information as the patient's chief complaint and her past medical history (past illnesses, injuries, hospitalizations, surgeries, medications, etc.). In addition, the physician asks about the patient's substance abuse (including alcohol and tobacco), diet, sleep patterns, family history, occupational history, environmental history, and psychosocial history (which includes such information as education, leisure activities, and home situation).⁴⁸⁷

Much like genetic tests, a complete patient history would seem to be far more valuable to disease diagnosis and treatment than information about the patient's race.⁴⁸⁸ Direct questioning of a patient about diet and lifestyle should be more accurate than assumptions based on race. Although there is always risk that patients will be less than forthcoming with physicians, these risks seem to us lower than the errors induced by reliance on race. Patients have a large incentive to provide doctors with accurate information and, although some may be willing to risk their health to avoid disclosing certain information, this number is likely to be small. Of course, members of minority groups may (perhaps justifiably) trust doctors less than do whites, which may lead to less candor between the patient and the physician.⁴⁸⁹

Despite the clear need for taking accurate histories, physicians and other health care professionals often fail to perform them or perform them perfunctorily. Instead, doctors rely unnecessarily on the race of the patient, noting it even when there is no reason to do so.⁴⁹⁰ This no doubt has some benefit to the practitioners themselves: it presents a useful heuristic that allows them to make assumptions about the patient without investing the time or energy in acquiring more detailed information. Indeed, many physicians may find over time that these stereotypical assumptions, being generally true, aid them in making diagnoses. But just because such stereotypes aid the provider of health care does not mean that such heuristics should be encouraged, particularly when they can result in the very sort of harms that Doctor Garcia describes concerning the failure to diagnose cystic fibrosis.⁴⁹¹

One obvious goal for the medical community is to eliminate such shortcuts and instead insist on full histories. Such a focus will allow doctors

⁴⁸⁶ See MARK H. SWARTZ, *TEXTBOOK OF PHYSICAL DIAGNOSIS: HISTORY AND EXAMINATION* 3 (4th ed. 2002); see also J. STANLEY MCQUADE, *MEDICAL PRACTICE FOR TRIAL LAWYERS* §§ 2.3, 2.4 (6th ed. 2000).

⁴⁸⁷ SWARTZ, *supra* note 486, at 16–28.

⁴⁸⁸ As one textbook notes, “[a] medical history must be dynamic. Every history is different. All patients are asked the standard questions, but each patient should be evaluated differently.” *Id.* at 31. Forms for personal histories do not contain a place to indicate race, although it is nonetheless common for physicians to note the race. See Schwartz, *supra* note 20, at 1392.

⁴⁸⁹ On the problem of trust, see INSTITUTE OF MEDICINE, *supra* note 15, at 137–38.

⁴⁹⁰ See Schwartz, *supra* note 20, at 1392.

⁴⁹¹ See *supra* text accompanying note 60.

to diagnose and prescribe treatments based on real information about the patient's environment, rather than on stereotypes.

CONCLUSION

At the risk of oversimplifying, this Article reaches the following conclusions regarding the "science," sociology, and law of race. First, from a genetic perspective, there is no such thing as race; instead, race is socially constructed. One means of socially constructing race is to view it as an indicator of the geographic location of one's ancestors. When viewed this way, race, as population groups, can in some limited circumstances, be characterized by some differences in allelic frequencies when compared to other population groups. These differences in allelic frequencies may have a substantial impact on disease or its treatment, particularly when also linked to environmental or behavioral influences that may also impact races in different ways. Nonetheless, such differences among population groups are generally not very important from a medical perspective, although there are some exceptions where differences can be critical. It is, therefore, sometimes appropriate to use race (in the socially constructed sense) as a proxy for genetic, environmental and behavioral differences.

Despite the limited potential utility of race in medicine, the law currently broadly proscribes the use of race. While there are inherent limitations in the reach of the various legal regimes, and difficult problems of proof of the intentional racial discrimination that each requires, none of the regimes has an exception for using race as a proxy for the kinds of genetic, environmental, and behavioral factors that may relate to health. Furthermore, beyond the legal difficulties posed by current doctrine, the use of race in medicine is deeply problematic because it may validate racism to the extent it confirms genetic differences, a particularly serious problem to the extent the government is seen as recognizing, and therefore, reifying racial differences. It may further result in the inappropriate screening and treatment of patients, particularly but not exclusively members of minority races.

Accordingly, such use of race should be severely circumscribed. It should be permissible where race-focused studies and race-based outreach are concerned, but race-based screening, as we have defined it, and racial exclusion in clinical trials should not be permitted. Racial differences in treatment (including diagnostic screening) should be permissible only in rare circumstances where there is a bona fide treatment rationale, as we have defined that term. Under this test, the key requirement would be a scientific basis for believing not that race was helpful in diagnosis and treatment but rather that race was the *best available method* at the time.